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# Occupational Intakes of Radionuclides Part 1

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Approved by the Commission in XXX

**Abstract**- This report is the first in a series of documents replacing the Publication 30 series and Publication 68 to provide revised dose coefficients for occupational intakes of radionuclides (OIR) by inhalation and ingestion. The revised dose coefficients have been calculated using the Publication 100 Human Alimentary Tract Model (HATM) and a revision of the Publication 66 Human Respiratory Tract Model (HRTM) which takes account of more recent data. In addition, information has been provided on absorption to blood following inhalation and ingestion of different chemical forms of elements and their radioisotopes, in those cases for which it is judged that the data are sufficient to make specific recommendations. Revisions have been made to many models for the systemic biokinetics of radionuclides absorbed to blood, making them more physiologically realistic representations of uptake and retention in organs and tissues and of excretion.

The reports in this series provide data for the interpretation of bioassay measurements as well as giving dose coefficients, replacing Publications 54 and 78. In assessing bioassay data such as measurements of whole-body or organ content or urinary excretion, assumptions have to be made about the exposure scenario, including the pattern and mode of radionuclide intake, physical and chemical characteristics of the material involved and the elapsed time between the exposure(s) and measurement. This report provides some guidance on monitoring programmes and data interpretation.

*Keywords:* Occupational exposure, Internal Dose Assessment, Biokinetic and Dosimetric models, Bioassays interpretation.

## CONTENTS

## PREFACE

The system of protection recommended by the International Commission on Radiological Protection is the basis for standards and working practices throughout the world (ICRP, 1991, 2007; IAEA 1996a). Fundamental to the application of ICRP recommendations are the protection quantities defined by ICRP, equivalent dose and effective dose. While the definition of these quantities remains unchanged in the most recent recommendations (ICRP, 2007), there have been important changes that affect the values calculated per unit radiation exposure. Committee 2 of ICRP is responsible for the provision of these reference dose coefficients for the assessment of internal and external radiation exposure, calculated using reference biokinetic and dosimetric models, and reference data for workers and members of the public. Following from the 2007 Recommendations, Committee 2 and its Task Groups are engaged in a substantial programme of work to provide new dose coefficients for various circumstances of radiation exposure.

The 2007 Recommendations (Publication 103, ICRP, 2007) introduced changes to the radiation weighting factors used in the calculation of equivalent dose to organs and tissues and also changes to the tissue weighting factors used in the calculation of effective dose. In addition, an important development was the adoption of reference anatomical computational phantoms (that is, models of the human body based on medical imaging data), in place of the composite mathematical models that have been used for all previous calculations of organ doses. This process has commenced with the adoption of reference male and female adult models (ICRP, 2009) and will be continued with the adoption of paediatric phantoms. Publication 103 also clarified the need for separate calculation of equivalent dose to males and females and sex-averaging in the calculation of effective dose (ICRP, 2007). In the revision of dose coefficients, the opportunity has also been taken to improve calculations by updating radionuclide decay data (ICRP, 2008) and implementing more sophisticated treatments of radiation transport (ICRP, 2010) using the ICRP reference anatomical phantoms of the human body (ICRP, 2009). These improvements impact on dose calculations for external exposures as well as for internal emitters.

This report is the first in a series of documents replacing the Publication 30 series (ICRP, 1979, 1980, 1981, 1988b) and Publication 68 (ICRP, 1994b) to provide revised dose coefficients for occupational intakes of radionuclides (OIR) by inhalation and ingestion. The revised dose coefficients have been calculated using the Publication 100 (ICRP, 2006) Human Alimentary Tract Model (HATM) and a revision of the Publication 66 (ICRP, 1994a) Human Respiratory Tract Model (HRTM) which takes account of more recent data. In addition, information has been provided on absorption to blood following inhalation and ingestion of different chemical forms of elements and their radioisotopes, in those cases for which it is judged that the data are sufficient to make specific recommendations. Revisions have been made to many models for the systemic biokinetics of radionuclides absorbed to blood, making them more physiologically realistic representations of uptake and retention in organs and tissues and of excretion.

The reports in this series provide data for the interpretation of bioassay measurements as well as giving dose coefficients, replacing Publications 54 and 78 (ICRP, 1988a,

1997b). In assessing bioassay data such as measurements of whole-body or organ content or urinary excretion, assumptions have to be made about the exposure scenario, including the pattern and mode of radionuclide intake, physical and chemical characteristics of the material involved and the elapsed time between the exposure(s) and measurement. This report provides some guidance on monitoring programmes and data interpretation.

This first report in the series provides an introduction to the report series and includes chapters on control of occupational exposures, biokinetic and dosimetric models, monitoring methods, monitoring programmes and retrospective dose assessment. Subsequent reports provide data on individual elements and their radioisotopes, including biokinetic data and models, dose coefficients and data for bioassay interpretation. CD-ROMs accompanying this series give extensive additional information.

The membership of the Task Group on Internal Dosimetry (INDOS) at the time of the completion of this report was:

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# GLOSSARY

For convenience, this glossary has been structured under the subheadings of terms for: General Dosimetry and Radiological Protection, the Biokinetic Models and Bioassay Interpretation.

## Terms for General Dosimetry and Radiological Protection

Absorbed dose, $D$

The absorbed dose is given by

$$D = \frac{\mathrm{d}\bar{\varepsilon}}{\mathrm{d}m}$$

where $\mathrm{d}\bar{\varepsilon}$ is the mean energy imparted by ionising radiation to matter of mass d$m$. The unit of absorbed dose is joule per kilogram (J kg$^{-1}$), and its special name is gray (Gy).

Absorbed Fraction, $\phi(r_T \leftarrow r_S, E_{R,i})$

Fraction of radiation energy $E_i$ emitted within the source region $r_S$ that is absorbed in the target tissue $r_T$.

Active (bone) marrow

Active marrow is haematopoietically active and gets its red colour from the large numbers of erythrocytes (red blood cells) being produced. Active bone marrow serves as a target tissue for radiogenic risk of leukaemia.

Annual Limit on Intake (ALI)

The *ALI* was defined in Publication 60 (ICRP, 1991, para S30) as an intake (in Bq) of a radionuclide in a year which would lead to a committed effective dose of 20 mSv. ALIs are calculated separately for each intake pathway. The annual limit on intake for workers is thus:

$$ALI_j = \frac{0.02}{e_j(50)}$$

where j denotes the intake pathway (either inhalation or ingestion).

Becquerel (Bq)

The special name for the SI unit of activity, 1 Bq = 1 s$^{-1}$.

Biological half-time

The time required for a biological system to eliminate, by natural processes not including radioactive decay, and in the absence of additional input, half the amount of a substance, (*e.g.* radioactive material) that has entered it.

Bone marrow. See also 'Active (bone) marrow'; 'Inactive (bone) marrow'.

Bone marrow is a soft, highly cellular tissue that occupies the cylindrical cavities of long bones and the cavities defined by the bone trabeculae of the axial and appendicular skeleton. Total bone marrow consists of a sponge-like, reticular, connective tissue framework called stroma, myeloid (blood-cell-forming) tissue, fat cells (adipocytes), small accumulations of lymphatic tissue, and numerous blood vessels and sinusoids. There are two types of bone marrow, red (active) and yellow (inactive).

Committed Effective Dose ($E(50)$). See also 'Effective Dose'.

In this report series: effective dose calculated with the use of committed equivalent doses.

$$E(50) = \sum_T w_T \cdot \left[ \frac{H(r_T,50)^{Male} + H(r_T,50)^{Female}}{2} \right]$$

Committed Equivalent Dose ($H(r_T 50)$). See also 'Equivalent Dose'.

In this report series: Equivalent dose calculated using a 50-year commitment period. It is the time integral of the equivalent dose rate in a tissue or organ $r_T$ of the Reference Adult Male or the Reference Adult Female that is predicted by the reference biokinetic and dosimetric models following intake of radioactive material into the body of the Reference Worker. The integration period is 50 years following the intake.

$$H(r_T,50) = \int_0^{50} \dot{H}(r_T,t)dt$$

For both sexes the equivalent dose rate $\dot{H}(r_T,t)$ in target tissue $r_T$ at time t after an acute intake is expressed as

$$\dot{H}(r_T,t) = \sum_{r_S} A(r_S,t) \cdot S_w(r_T \leftarrow r_S)$$

where:

$A(r_S,t)$ is the activity of the radionuclide in source region $r_S$ at time t after intake, Bq, predicted by the reference biokinetic models for Reference Worker,

$S_w(r_S \leftarrow r_T)$ is the radiation-weighted S value (Bolch *et al*, 2009); *i.e.* the equivalent dose in target tissue $r_T$ per nuclear transformation in source region $r_S$, Sv $(\text{Bq s})^{-1}$, for the Reference Adult Male and Reference Adult Female.

Derived Air Concentration (DAC)

The *DAC* is the activity concentration in air in $\text{Bq/m}^3$ of the radionuclide considered which would lead to an intake of an *ALI* assuming a breathing rate

of the Reference Worker of 1.2 $m^3$ $h^{-1}$ and an annual working time of 2000 h. Then the *DAC* is given by:

$$DAC_j = \frac{ALI_j}{2400}$$

Dose Coefficient

Committed tissue equivalent dose per unit intake at age $t_o$, $h_T(\tau)$, or committed effective dose per unit intake, $e(\tau_D)$, where $\tau_D$ is the dose-commitment period in years over which the dose is calculated *i.e.* 50 y for adults and $(70–t_o)$ y for children. Note that elsewhere the term 'dose per unit intake (DPUI)' is sometimes used for dose coefficient.

Dose constraint

A prospective and source-related restriction on the individual dose from a source, which provides a basic level of protection for the most highly exposed individuals from a source, and serves as an upper bound on the dose in optimisation of protection for that source. For occupational exposures, the dose constraint is a value of individual dose used to limit the range of options considered in the process of optimisation (ICRP, 2007).

Dose limit

Recommended value of the effective dose or the organ- or tissue-equivalent dose to an individual that shall not be exceeded in planned exposure situations (ICRP, 2007).

Dose of record

In this document the dose of record refers to the effective dose, assessed by summing the measured personal dose equivalent $H_P(10)$ and the committed effective dose retrospectively determined for the Reference Worker using results of individual monitoring of the worker and ICRP reference biokinetic and dosimetric computational models. Dose of record may be assessed using site-specific parameters of exposure such as the absorption Type of the material and the AMAD/AMTD of the inhaled aerosol, but the parameters of the Reference Worker shall be fixed as defined by the ICRP in this report series. Dose of record is assigned to the worker and required to be kept for purposes of reporting and retrospective demonstration of compliance with regulatory requirements.

Dose Per Unit Content (DPUC)

In this report series: A set of tabulated values $z(t)=e(50)/m(t)$ $zh_T(t)=h_T(50)/m(t)$, where e(50) is DPUI and $z(t)$ is the bioassay function. DPUC represent the committed effective dose or committed equivalent dose in an organ *T* per unit predicted activity content in the body, a given organ (organ *T* or other organ) or per unit daily excretion.

Dose Per Unit Intake (DPUI). See also Dose Coefficient

In this report series: The committed effective dose per unit radionuclide intake, $e(50)$, or committed tissue equivalent dose to the tissue or organ $r_T$ per unit radionuclide intake, $h_T(r_T,50)$, where the dose-commitment period over which the dose is calculated is 50 years.

Dose–response function (DRF)

A particular function used in this publication to represent the absorbed dose in a target region per particle fluence in that region, derived using models of the microscopic structure of the target region geometry and the transport of the secondary ionising radiations in those regions.

Effective Dose, $E$

In this report series, in accordance with the generic definition of effective dose in ICRP Publication 103 (ICRP, 2006), the effective dose is calculated as:

$$E = \sum_T w_T \left[ \frac{H(r_T)^{Male} + H(r_T)^{Female}}{2} \right]$$

where $\boldsymbol{H(r_T)^{Male}}$ and $\boldsymbol{H(r_T)^{Female}}$ are the equivalent doses to the tissue or organs $r_T$ of the Reference Adult Male and Reference Adult Female respectively, and $w_T$ is the tissue weighting factor for tissue $r_T$ , with $\sum_T w_T = 1$. The sum is performed over all organs and tissues of the human body considered to be sensitive to the induction of stochastic effects. Since $w_R$ and $w_T$ are dimensionless, the unit for the effective dose is the same as for absorbed dose, J kg$^{-1}$, and its special name is sievert (Sv).

Equivalent Dose ($H(r_T)$)

In this report series: The equivalent dose is defined as:

$$H(r_T) = \sum_R w_R D_R(r_T)$$

where $w_R$ is the radiation weighting factor for radiation type R, and $D_R(r_T)$ is the organ dose from radiation type $R$ to in a tissue or organ $r_T$ of the Reference Adult Male or Reference Adult Female.. Since $w_R$ is dimensionless, the unit for the equivalent dose is the same as for absorbed dose, J kg$^{-1}$, and its special name is sievert (Sv).

Exposure

The state or condition of being subject to irradiation.

- External Exposure: exposure to radiation from a source outside the body.
- Internal Exposure: exposure to radiation from a source within the body.

Gray (Gy)

The special name for the SI unit of absorbed dose: 1 Gy = 1 J kg$^{-1}$.

Inactive (bone) marrow

In contrast to the active marrow, the inactive marrow is haematopoietically inactive (i.e. does not directly support haematopoiesis). It gets its yellow colour from fat cells (adipocytes) which occupy most of the space of the yellow bone marrow framework.

Marrow cellularity

The fraction of bone marrow volume in a given bone that is haematopoietically active. Age- and bone-site-dependent reference values for marrow cellularity are given in Table 41 of ICRP Publication 70 (ICRP, 1995). As a first approximation, marrow cellularity may be thought of as 1 minus the fat fraction of bone marrow.

Mean absorbed dose in an organ or tissue , $D_T$

The mean absorbed dose in a specified organ or tissue T is given by

$D_T = 1/m_T \int D \, dm$, where $m_T$ is the mass of the organ or tissue, and $D$ is the absorbed dose in the mass element d$m$. The unit of mean absorbed dose is joule per kilogram (J kg$^{-1}$), and its special name is gray (Gy).

Occupational exposure

The radiation exposure of workers incurred as a result of their work. The ICRP limits its use of ‘occupational exposures’ to radiation exposures incurred at work as a result of situations that can reasonably be regarded as being the responsibility of the operating management.

Organ absorbed dose. See ‘Mean absorbed dose’

Short phrase for “mean absorbed dose in an organ or tissue”.

Organ equivalent dose. See ‘Equivalent Dose’.

Short phrase for “equivalent dose in an organ or tissue”.

Protection Quantities

Values that ICRP has developed for radiological protection that allow quantification of the extent of exposure to ionising radiation from both whole and partial body external irradiation and from intakes of radionuclides.

Radiation weighting factor, $w_R$

A dimensionless factor by which the organ or tissue absorbed dose is multiplied to reflect the relative biological effectiveness of high-LET radiations compared to photon radiations. It is used to derive the equivalent dose from the mean absorbed dose in an organ or tissue.

Red (bone) marrow

See ‘Active (bone) marrow’.

Response function

See ‘Dose–response function’.

Reference level

In emergency or existing controllable exposure situations, this represents the level of dose or risk, above which it is judged to be inappropriate to plan to allow exposures to occur, and below which optimisation of protection should be implemented. The chosen value for a reference level will depend upon the prevailing circumstances of the exposure under consideration.

Reference male and reference female (reference individual)

An idealised male or female with characteristics defined by the ICRP for the purpose of radiological protection, and with the anatomical and physiological characteristics defined in ICRP Publication 89 (ICRP, 2002).

Reference person

An idealised person, for whom the equivalent doses in organs and tissues are calculated by averaging the corresponding doses of the Reference Male and Reference Female. The equivalent doses of the Reference person are used for the calculation of the effective dose.

Reference phantom

The computational phantom of the human body (male or female voxel phantom based on medical imaging data, defined in ICRP Publication 110 (ICRP, 2009) with the anatomical and physiological characteristics defined in ICRP Publication 89 (ICRP, 2002).

Reference Worker

An adult Reference Person associated with the reference biokinetic, anatomical and physiological characteristics assigned in this report series. The definition of the Reference Worker includes the structure and parameter values of the reference systemic biokinetic models, HATM, HATM, and dosimetric models and is invariant on sex, age and other individual-specific characteristics. The average breathing rate of the Reference Worker is 1.2 $m^3$ $h^{-1}$ during the 8 h working day, which corresponds to the daily intake of 9.6 $m^3$ (Publication 66, ICRP, 1994a).

Reference value

The value of a parameter, factor or quantity that is regarded as valid for use in dosimetric calculations and recommended by ICRP. To prevent accumulation of error in successive calculations, the reference values are sometimes expressed with higher precision than data would support..

Sievert (Sv)

The special name for the SI unit (J $kg^{-1}$) of equivalent dose and effective dose.

Source Region ($r_s$)

Region of the body containing the radionuclide. The region may be an organ, a tissue, the contents of the alimentary tract or urinary bladder, or the surfaces of tissues as in the skeleton and the respiratory tract.

Spongiosa

Term referring to the combined tissues of the bone trabeculae and marrow tissues (both active and inactive) located within cortical bone cortices across regions of the axial and appendicular skeleton. Spongiosa is one of three bone regions defined in the ICRP Publication 110 (ICRP, 2009) reference phantoms, the other two being cortical bone and medullary marrow of the long bone shafts. As the relative proportions of trabecular bone, active marrow, and inactive marrow vary with skeletal site, the homogeneous elemental composition and mass density of spongiosa is not constant but varies with skeletal site [see Annex B of ICRP Publication 110 (ICRP, 2009)].

S-value (radiation-weighted) $S_w(r_T \leftarrow r_S)$

The equivalent dose in target tissue or organ $r_T$ per nuclear transformation of a given radionuclide in source region $r_S$, Sv (Bq s)$^{-1}$, for the Reference Adult Male and Reference Adult Female.

$$S_w(r_T \leftarrow r_S) = \sum_R w_R \sum_i \frac{E_{R,i} \cdot Y_{R,i} \cdot \phi(r_T \leftarrow r_S, E_{R,i})}{M(r_T)}$$

where:

$E_{R,i}$ is the energy of the i$^{th}$ radiation of type R with the unit joule (J),

$Y_{R,i}$ is the yield of i$^{th}$ radiation of type R per nuclear transformation, (Bq s)$^{-1}$,

$w_R$ is the radiation weighting factor for radiation type R, Table 1,

$\phi(r_T \leftarrow r_S, E_{R,i})$ is the absorbed fraction,

$M(r_T)$ is the mass of target tissue $r_T$, kg.

Target Tissue ($r_T$)

Tissue or organ of the body in which a radiation dose is received.

Tissue weighting factor, $w_T$. See also 'Effective Dose'.

The factor by which the equivalent dose in an organ or tissue T is weighted to represent the relative contribution of that organ or tissue to overall radiation detriment from stochastic effects (ICRP, 1991). It is defined such that

$$\sum_T w_T = 1$$

Worker

In this text any person who works, whether full time, part time or temporarily, for an employer and who has recognised rights and duties in relation to occupational radiation protection (ICRP, 2007).

**Terms for the biokinetic models**

Absorption

Transfer of material to body fluids regardless of mechanism. Generally applies to dissociation of particles and the uptake into body fluids of soluble substances and material dissociated from particles.

Aerodynamic diameter ($d_{ae}$)

Diameter (μm) of a unit density (1 g $cm^{-3}$) sphere that has same terminal settling velocity in air as the particle of interest.

Alimentary tract

The tube from mouth to anus in which food is digested.

Alimentary tract transfer factor ($f_A$)

The fraction of activity entering the alimentary tract that is absorbed to blood, taking no account of losses due to radioactive decay or endogenous input of activity into the tract.

Alveolar-Interstitial Region (AI)

Part of the respiratory tract, consisting of the respiratory bronchioles, alveolar ducts and sacs with their alveoli, and the interstitial connective tissue; airway generations 16 and beyond.

AMAD

Activity Median Aerodynamic Diameter. Fifty percent of the activity in the aerosol is associated with particles of aerodynamic diameter ($d_{ae}$) greater than the AMAD. Used when deposition depends principally on inertial impaction and sedimentation, typically when the AMAD is greater than about 0.5 μm.

AMTD

Activity Median Thermodynamic Diameter. Fifty percent of the activity in the aerosol is associated with particles of thermodynamic diameter ($d_{th}$) greater than the AMTD. Used when deposition depends principally on diffusion, typically when the AMAD is less than about 0.5 μm.

Basal cells

Cuboidal epithelial cells attached to the basement membrane of extrathoracic and bronchial epithelium and not extending to the surface.

Biokinetic model

A mathematical model adopted in this report for the Reference Worker. Reference biokinetic model describes the intake, uptake and retention of a radionuclide in various organs or tissues of the body and the subsequent excretion from the body by various pathways.

Bronchial Region (BB)

Part of the respiratory tract, consisting of the trachea (generation 0) and bronchi, airway generations 1 through 8.

Bronchiolar Region (bb)

Part of the respiratory tract, consisting of the bronchioles and terminal bronchioles; airway generations 9 through 15.

Bone surfaces

See 'Endosteum'.

Class SR-0

Insoluble and nonreactive. Negligible deposition in the respiratory tract.

Class SR-1

Soluble or reactive. Deposition throughout the respiratory tract, which may be complete or incomplete.

Class SR-2

Highly soluble or reactive. Complete deposition in the respiratory tract with instantaneous uptake to body fluids.

Clearance

The removal of material from the respiratory tract by particle transport and by absorption into body fluids.

Compartment

In this report series: Mathematical pool of radioactive materials in the body which can be characterised by first order kinetics; a compartment can be associated with an organ (as for example the liver), a part of an organ (as for example the bronchial region of the lungs), a tissue (as for example the bone), a part of a tissue (as for example the bone surface) or another substance of the body (as for example the body fluids). Activity is considered to be uniformly distributed in a compartment.

Compartments in the particle transport model representing retention of material in each region defined in the Human Respiratory Tract Model:

*Original HRTM*

$AI_1$ relatively short-term retention (half-time, $t_{½}$ about 35 d) of a fraction, taken to be 0.3, of the deposit in the alveolar-interstitial region.

$AI_2$ long-term retention ($t_{½}$ about 700 d) of a fraction, taken to be 0.6, of the deposit in the alveolar-interstitial region.

$AI_3$ very long-term retention ($t_{½}$ about 6000 d) of a fraction, taken to be 0.1, of the deposit in the alveolar-interstitial region.

$BB_1$ short-term retention ($t_{½}$ about 100 minutes) of particles in the bronchial region: the particles are removed by rapid mucociliary clearance.

$bb_1$ short-term retention ($t_{½}$ about 8 hours) of particles in the bronchiolar region: the particles are removed by rapid mucociliary clearance.

$BB_2$ intermediate retention ($t_{½}$ about 20 d) of particles in the bronchial region.

$bb_2$ intermediate retention ($t_{½}$ about 20 d) of particles in the bronchiolar region.

$BB_{seq}$ long-term retention ($t_{½}$ about 70 d) in airway walls of a small fraction of the particles deposited in the bronchial region.

$bb_{seq}$ long-term retention ($t_{½}$ about 70 d) in airway walls of a small fraction of the particles deposited in the bronchiolar region.

$ET_1$ retention of material deposited in the anterior nose (region $ET_1$, which is not subdivided).

$ET_2'$ short-term retention ($t_{½}$ about 10 minutes) of the material deposited in the posterior nasal passage, larynx, pharynx and mouth (region $ET_2$), except for the small fraction, taken to be 0.0005, retained in $ET_{seq}$. (In *Publication 66* this *compartment* was labelled $ET_2$. It is here, as in *Publication 71*, labelled $ET_2'$ to distinguish it from the *region* $ET_2$ which also includes *compartment* $ET_{seq}$.)

$ET_{seq}$ long-term retention ($t_{½}$ about 700 d) in airway tissue of a small fraction of particles deposited in the nasal passages.

$LN_{ET}$ lymphatics and lymph nodes that drain the extrathoracic region.

$LN_{TH}$ lymphatics and lymph nodes that drain the thoracic regions.

*Revised HRTM*

$ET_2'$ short-term ($t_{½}$ about 10 minutes) of the material deposited in the posterior nasal passage, larynx and pharynx ($ET_2$ region) except for the small fraction (taken to be 0.002) retained in $ET_{seq}$.

$BB'$ retention ($t_{½}$ about 100 minutes) of particles in the bronchial region, with particle transport to $ET_2'$.

$bb'$ retention ($t_{½}$ about 3.5 days) of particles in the bronchiolar region, with particle transport to $BB'$.

$BB_{seq}$ long-term retention ($t_{½}$ about 700 d) in airway walls of a small fraction of the particles deposited in the bronchial region.

$bb_{seq}$ long-term retention ($t_{½}$ about 700 d) in airway walls of a small fraction of the particles deposited in the bronchiolar region.

ALV retention ($t_{½}$ about 200 d) of particles deposited in the alveoli. A fraction (0.67) of the deposit is removed by particle transport to the ciliated airways (bb′), while the remainder penetrates to the interstitium (INT).

INT very long-term retention ($t_{½}$ about 60 y) of the particles deposited in the alveoli that penetrate to the interstitium: the particles are removed slowly to the lymph nodes.

Deposition

Refers to the initial processes determining how much of the material in the inspired air remains behind in the respiratory tract after exhalation. Deposition of material occurs during both inspiration and exhalation.

Endogenous excretion

Term used to specify the excretion of materials from body fluids to the alimentary tract, applying to biliary excretion and passage of materials through the alimentary tract wall.

Endosteum (or endosteal layer)

A 50 µm-thick layer covering the surfaces of the bone trabeculae in regions of trabecular spongiosa and those of the cortical surfaces of the medullary cavities within the shafts of all long bones. It is assumed to be the target tissue for radiogenic bone cancer. This target region replaces that previously introduced in ICRP Publications 26 and 30 (ICRP, 1977, 1979) – the bone surfaces – which had been defined as a single-cell layer, 10 µm in thickness, covering the surfaces of both the bone trabeculae and the Haversian canals of cortical bone.

Exogenous excretion

Term used to specify the (faecal) excretion of material that passes through the alimentary tract without absorption.

Extrathoracic (ET) Airways

Part of the respiratory tract, consisting of the anterior nose (the $ET_1$ region) and the posterior nasal passages, pharynx and larynx (the $ET_2$ region). Note that the oral part of the pharynx is no longer part of $ET_2$ because it is included in the HATM.

Exposure (in the context of inhalation)

The product of the air concentration of a radionuclide to which a person is exposed (Bq $m^{-3}$) and the time of exposure. More generally, when the air concentration varies with time, the time integral of the air concentration of a radionuclide to which a person is exposed, integrated over the time of exposure.

Fractional absorption in the gastrointestinal tract ($f_1$)

The fraction of an element directly absorbed from the gut to body fluids, used in the Publication 30 gastrointestinal tract model. See also 'Alimentary tract transfer factor'.

Habitual Mouth Breather

A person who breathes oro-nasally (partly through the nose and partly through the mouth) at all levels of exercise: "sleep", "sitting" "light exercise" and "heavy exercise". At "heavy exercise" such a person inhales a greater fraction of air through the mouth than a Nasal Augmenter.

Habitual Nose Breather

A person who breathes entirely through the nose at the exercise level of "heavy exercise" as well as at "sleep", "sitting" and "light exercise". Such a person may switch to breathing oro-nasally (partly through the nose and partly through the mouth), but at a ventilation rate greater than the reference value for heavy exercise (3 $m^3$ $h^{-1}$).

Human Alimentary Tract Model (HATM)

Biokinetic model for describing the movement of ingested materials through the human alimentary tract; published in Publication 100 (ICRP, 2006).

Human Respiratory Tract Model (HRTM)

Biokinetic model for describing the deposition, translocation and absorption of inhaled materials in the human respiratory tract; published in Publication 66 (ICRP, 1994a).

Inhalability

Fraction of particles that enters the nose and mouth, of those present in the volume of ambient air before inspiration.

Intake. See also 'Uptake'

Activity that enters the respiratory tract or gastrointestinal tract from the environment.

Acute intake - a single intake by inhalation or ingestion, taken to occur instantaneously.

Chronic intake - a protracted intake over a specified period of time.

Nasal Augmenter

A person who breathes entirely through the nose at the exercise levels of "sleep", "sitting" and "light exercise", but oro-nasally (partly through the nose and partly through the mouth) during "heavy exercise". Also known as a "normal nose breather", because most people breathe according to this pattern. All reference subjects, including the Reference Worker are assumed to be Nasal Augmenters.

Normal Nose Breather
: See ‘Nasal Augmenter’.

Particle transport
: Processes that clear material from the respiratory tract to the alimentary tract and to the lymph nodes, and move material from one part of the respiratory tract to another.

Secretory cells
: Nonciliated epithelial cells that have mucous or serous secretions.

Subcutaneous tissue
: Loose fibrous tissue situated directly below the skin. It includes blood vessels, connective tissue, muscle, fat and glands. In the context of intake through wounds, it represents tissue at the wound site in which radionuclides could be retained prior to removal of soluble or dissolved material to blood or insoluble material via lymphatic vessels.

Target tissues in the bronchial region of the Human Respiratory Tract Model:
: (See Table 8. For each of the other regions only one target tissue is specified and hence no special symbol is required.)

: $BB_{bas}$ tissue in bronchial region through which basal cell nuclei are distributed.

: $BB_{sec}$ tissue in bronchial region through which secretory cell nuclei are distributed.

Thermodynamic diameter ($d_{th}$)
: Diameter (μm) of a spherical particle that has the same diffusion coefficient in air as the particle of interest.

Thoracic (TH) Airways
: Combined bronchial, bronchiolar and alveolar-interstitial regions.

Transfer compartment
: The compartment introduced for mathematical convenience into many of the biokinetic models previously used by ICRP to account for the translocation of the radioactive material through the body fluids from where they are deposited in tissues.

Types of materials, classified according to their rates of absorption from the respiratory tract to body fluids:

: Type F deposited materials that are readily absorbed into body fluids from the respiratory tract. (Fast absorption)

: Type M deposited materials that have intermediate rates of absorption into body fluids from the respiratory tract. (Moderate absorption)

Type S deposited materials that are relatively insoluble in the respiratory tract. (Slow absorption.)

Type V deposited materials that, for dosimetric purposes, are assumed to be instantaneously absorbed into body fluids from the respiratory tract: only certain gases and vapours. (Very fast absorption)

Uptake. See also 'Intake'

Activity that enters body fluids from the respiratory or alimentary tract or through the skin.

**Terms for Bioassay Interpretation**

Action level

A pre-set level above which some remedial action should be considered.

Activity

The number of nuclear transformations per unit time (s) of a radioactive material. The SI unit of the activity is the becquerel (Bq): 1 Bq = 1 $s^{-1}$

Bioassay

Any procedure used to determine the nature, activity, location or retention of radionuclides in the body by direct (*in vivo*) measurement or by indirect (*in vitro*) analysis of material excreted or otherwise removed from the body.

Bioassay function

In this report series: A set of tabulated values *m(t)* predicted by the reference biokinetic models describing the time course of the activity in the body ("retention function") or the activity excreted via urine or faeces ("excretion function") following a single intake at time t = 0. In general, the retention functions represent the body or organ activity at the time t after the intake, whereas the excretion functions represent the daily excretion: the integral of the instantaneous excretion rate from (t – 1) until t, where t is the number of days after a single intake (integer).

Decision Threshold

Fixed value of a measured quantity that, when exceeded by the result of an actual measurement quantifying a physical effect (e.g. the presence of a radionuclide in a sample), may be taken to indicate that the physical effect is present. The decision threshold is the critical value of a statistical test for the decision between the hypothesis that the physical effect is not present and the alternative hypothesis that it is present. When the critical value is exceeded by the result of an actual measurement, this is taken to indicate that the hypothesis should be rejected. The statistical test is designed in such a way that the probability of wrongly rejecting the hypothesis (Type I error) is at most equal to a given value, $\alpha$. The decision threshold is an *a posteriori*

quantity, evaluated after a particular measurement in order to decide whether the result of the measurement is significant. The decision threshold is also referred as the critical level or the minimum significant activity.

Direct measurement

Generic term for any kind of *in vivo* measurement of incorporated radionuclides (*i.e.* whole body counting, lung counting, thyroid counting, etc.).

Excretion function.

See 'Bioassay function'.

Excretion rate

In general, the excretion rate is the amount of activity which is excreted via urine or faeces during a 24 hour period, with the decay of the radionuclide having been corrected for the end of the 24 hour sampling period.

Investigation level

A pre-set level above which the cause or the implications of an intake should be examined (ICRP, 1997b). Investigation levels can be set for any operational parameter related to the individual or to the working environment. For individual monitoring of exposure to intakes of radionuclides, they are most likely to relate to a measured body or organ/tissue content, an activity level in excreta, or an air concentration measured by a personal air sampler.

Measured quantity ($M$)

Primary result of incorporation monitoring; the measured quantity represents in the case of *in vivo* measurements the whole body, organ or tissue activity (Bq) and in the case of *in vitro* measurements the daily excretion rate (Bq $d^{-1}$, Bq $l^{-1}$, or Bq $kg^{-1}$).

Minimum Detectable Amount (MDA)

The smallest true value of a measured quantity that is detectable by the measuring method. The MDA is the smallest true value that is associated with the statistical test and hypothesis in accordance with the Decision Threshold, as follows: if in reality the true value is equal to or exceeds the MDA, the probability of wrongly not rejecting the hypothesis (Type II error) is at most equal to a given value, $\beta$. The MDA is an *a priori* quantity, evaluated for a particular measurement method in advance of the performance of a measurement. The MDA is also referred as the detection limit or the lower limit of detection; the term 'MDA' is also used as an abbreviation for minimum detectable activity.

Recording level

A pre-set level above which a result should be recorded, lower values being ignored.

Retention function.

See 'Bioassay function'.

Threshold levels

Values of measured quantities above which some specified action or decision should be taken. They include:

Recording levels, above which a result should be recorded, lower values being ignored;

Investigation levels, above which the cause or the implication of the result should be examined;

Action levels, above which some remedial action should be considered.

# 1 INTRODUCTION

## 1.1 Purpose of this report series

(1) Occupational intakes of radionuclides may occur during routine operations in a range of industrial, medical, educational and research facilities. They may also occur as a result of a nuclear accident, after an incident involving radioactive material, during post-accident remediation work at a nuclear installation, or during environmental remediation activities.

(2) An adequate assessment of occupational internal exposure resulting from intakes of radionuclides is essential for the design, planning and authorisation of a facility or activity, for the optimisation of radiation protection of workers, for operational radiation protection and for the retrospective demonstration of compliance with regulatory requirements.

(3) After intake of radionuclides, doses received by organs and tissues are protracted over time and so equivalent and effective doses are accumulated over time. The resulting quantities are referred to as committed doses.

(4) Internal exposure of workers should be assessed in terms of the protection quantity *committed effective dose* The individual exposure of a worker should be assessed and recorded in terms of *dose of record*, which takes into account both internal and external exposures.

(5) This report series provides a comprehensive set of dose coefficients (i.e. committed effective dose and committed equivalent dose per unit intake (DPUI)) and also provides values for committed effective dose and committed equivalent dose per unit content (DPUC).

(6) These data may be used for both prospective assessments and retrospective assessments. Prospective assessments provide estimates of intakes and resulting doses for workers engaged in specific activities using information on potential exposures to radionuclides obtained at the design and planning stage of a facility or practice. Retrospective assessments use the results of individual monitoring and workplace monitoring to assess doses in order to maintain individual dose records and demonstrate compliance with regulatory requirements. Prospective assessments generally make use of default assumptions about exposure conditions and default values for parameters describing material-specific properties such as the particle size distribution of an inhaled aerosol or the absorption characteristics of a material after inhalation or ingestion. Retrospective assessments may in some circumstances make use of specific information relating to the exposure, as discussed in Chapter 6.

(7) The report series contains detailed information on the ICRP reference models used for the derivation of dose coefficients. The information provided in this first report of the series includes a description of revisions made to the ICRP reference Human Respiratory Tract Model (ICRP, 1994a) and an overview of the ICRP reference Human Alimentary Tract Model (ICRP, 2006). Subsequent reports in the series present descriptions of the structures and parameter values of the reference systemic biokinetic models,

(8) This report also presents an overview of monitoring methods and programmes, and generic guidance on the interpretation of the bioassay data.

Subsequent reports in the series present radionuclide-specific information for the design and planning of monitoring programmes and retrospective assessment of occupational internal doses.

(9) The material presented in this report series is not intended for applications beyond the scope of occupational radiation protection. An example of such an application is the assessment of a case of substantial radionuclide intake, where organ doses can approach or exceed the thresholds for tissue reactions, and where medical treatment may require an individual-specific reconstruction of the magnitude of absorbed doses and associated parameters characterising the exposure.

### 1.2 Protection quantities and dose coefficients in this report series

(10) The protection quantities defined by ICRP, equivalent dose and effective dose, are fundamental to the application of ICRP recommendations. The concept of effective dose provides a single quantity that may be used to characterise both internal and external individual exposures in a manner that is independent of the individual's body-related parameters, such as sex, age (for adults), anatomy, physiology and race. In order to achieve wide applicability, the protection quantities (effective dose and equivalent dose) are defined using computational models with broad averaging of physiological parameter values. Specifically, Publication 89 (ICRP 1975, 2002) defines the key parameters of the Reference Individuals (the mass, geometry and composition of human organs and tissues), while this report series provides relevant parameters for the Reference Worker (ICRP 1994) together with an associated set of ICRP reference biokinetic models.

(11) Effective dose is not an individual-specific dose quantity, but rather the dose to a Reference Person under specified exposure conditions. In the general case, the Reference Person can be either a Reference Worker (see Glossary) or a Reference Member of the Public of a specified age.

(12) The protection quantities for internal exposure (committed effective dose and committed equivalent dose) are derived using models and are not directly measurable. For retrospective assessments of internal exposure, the dose can be assessed from measurements of the amounts of radionuclides in the human body, their rates of excretion or their concentrations in the ambient air. In contrast, the operational quantities for exposure to external radiation fields are directly measurable.

(13) The dose coefficients and dose per unit content values presented in this report series are given for a Reference Worker with an average breathing rate of 1.2 $m^3$ $h^{-1}$ during an 8 h working day.. These data are provided for a range of physico-chemical forms for each radionuclide and for a range of aerosol particle size distributions. Data for ingestion and injection (i.e. direct entry to the blood) are provided to allow the interpretation of bioassay data for cases of inadvertent ingestion (e.g. of material on contaminated skin) or rapid absorption through intact or damaged skin (injection).

(14) While the generic definition of protection quantities remains unchanged in the most recent recommendations (ICRP, 2007), there have been changes that affect calculated values of dose per unit radiation exposure, including changes to radiation and tissue weighting factors, adoption of reference computational phantoms (ICRP, 2009), and the development of the new generation of reference biokinetic models.

(15) This report series provides revised dose coefficients for occupational intakes of radionuclides (OIR) replacing the Publication 30 series (ICRP, 1979, 1980, 1981, 1988b) and Publication 68 (ICRP, 1994b).

(16) Data for the interpretation of bioassay measurements are also provided, replacing Publications 54 and 78 (ICRP, 1988a, 1997b) and consolidating all of the information needed to interpret the results of bioassay measurements for a particular radionuclide in a single ICRP publication.

(17) The full data set of the report series is provided as an electronic annex on the attached CD-ROMs. The printed documents contain a selected set of data and materials.

(18) Data are presented in a standard format for each element and its radioisotopes. Tabulated dose coefficients may be used to determine committed effective dose and committed equivalent doses from a known intake of a radionuclide. Tabulated values for dose per unit content may be used to assess committed doses directly from measurements of appropriate bioassay quantities (e.g. radionuclide activity in whole body or lungs, or daily excretion of a radionuclide in urine or faeces). Similarly, values of radionuclide activities per unit intake in the body or in daily excreta samples, presented in tabular and graphical formats, may be used to assess the intake corresponding to a single bioassay measurement. Committed doses may then be assessed from the intake using the tabulated dose coefficients. A full description of the information provided for each element and radioisotope is given in Chapter 7.

(19) The revised dose coefficients, dose per unit content values and reference bioassay functions have been calculated using the Publication 100 (ICRP, 2006) Human Alimentary Tract Model (HATM) and a revision of the Publication 66 (ICRP, 1994a) Human Respiratory Tract Model (HRTM) which takes account of more recent data. The revisions made to the HRTM are described in Section 3.2 of this report. In addition, information is provided in this report series on absorption to blood following inhalation and ingestion of different chemical forms of elements and their radioisotopes, in those cases for which it is currently judged that the data are sufficient to make specific recommendations. Revisions have been made to many models for the systemic biokinetics of radionuclides, making them more physiologically realistic representations of uptake and retention in organs and tissues and of excretion.

(20) Biokinetic models, reference physiological data, computational phantoms and radiation transport calculation codes are used for the calculation of dose coefficients (ICRP, 2007). ICRP publishes dose coefficients for the inhalation or ingestion of individual radionuclides by workers, giving both equivalent doses to organs and tissues, and effective dose (ICRP, 1991, 2007). The steps in the calculation (Figure 1) can be summarised as follows:

- By use of the reference biokinetic models, the distribution and retention of radionuclides in body organs and tissues of the Reference Worker are determined as a function of time after intake by inhalation or ingestion For radiation protection purposes, it assumed that all biokinetic parameters of the Reference Worker are invariant on sex, anatomy, physiology, race and other

individual-related factors.

- The total number of nuclear transformations (radioactive decays) occurring within a defined time period in each source region is calculated
- The dosimetric models based on reference computational phantoms and Monte Carlo radiation transport codes are used to calculate the mean absorbed dose to each target organ or tissue resulting from a nuclear disintegration in each source organ.
- The radiation weighting factors are applied to determine sex-specific committed equivalent doses.
- The tissue weighting factors are then applied to determine the sex-averaged committed effective dose.

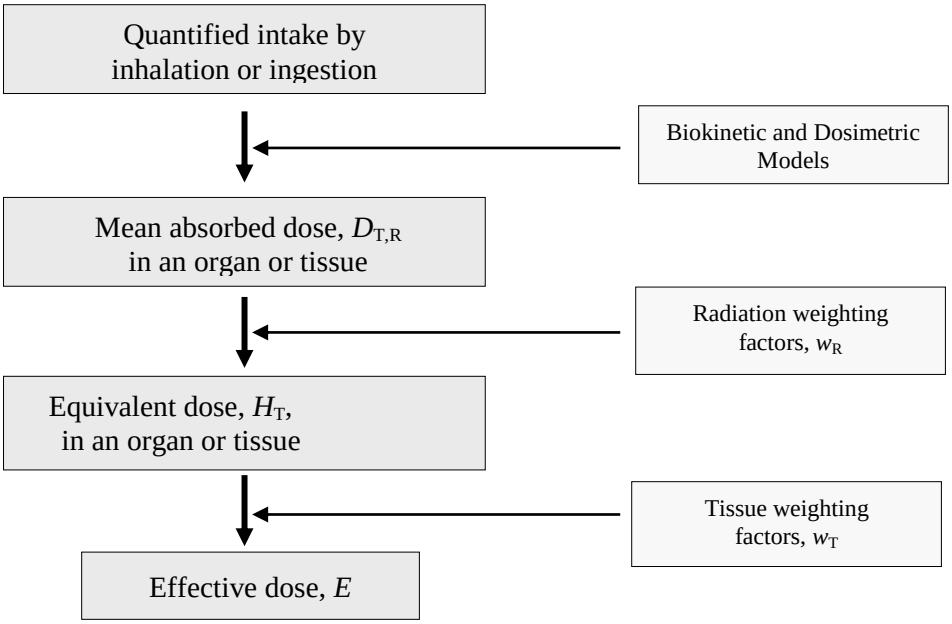



Figure 1. Calculation of absorbed dose and the ICRP protection quantities, equivalent and effective dose, for intakes of radionuclides

(21) The detailed computational procedure used in this report series is described in section 3.7.

### 1.3 Previous reports on occupational intakes of radionuclides

(22) Publication 30 (ICRP, 1979, 1980, 1981, 1988b) and its Supplements gave dose coefficients and values of Annual Limits on Intake (ALI) for workers, for intakes of radionuclides by inhalation and ingestion, referencing the recommendations issued in Publication 26 (ICRP, 1977) and the anatomical and physiological data in Reference Man (ICRP, 1975). Publication 68 (ICRP, 1994b) provided updated dose coefficients for workers following the 1990 Recommendations issued in Publication 60 (ICRP, 1991). It applied the Publication 66 HRTM (ICRP, 1994a) for inhaled radionuclides, the updated basic anatomical and physiological data for the skeleton in

Publication 70 (ICRP, 1995b) and revised systemic biokinetic models for selected isotopes of 31 elements given in Publications 56, 67, 69 and 71 (ICRP, 1989, 1993b, 1995a,c). Biokinetic models for other elements were taken from Publication 30 and modified by addition of explicit excretion pathways to improve dose estimates for the urinary bladder and colon walls. Publication 68 did not give ALIs, as ICRP wished to emphasise the need to take account of all exposures to ionising radiation in the workplace, from external radiation and intakes of all radionuclides.

(23) Publications 54 and 78 gave guidance on the design of monitoring programmes and the interpretation of results to estimate doses to workers following radionuclide inhalation or ingestion (ICRP, 1988a, 1997b). The guidance was supported by numerical data to enable the assessment of intakes and doses from bioassay data (that is, measurements of body and organ content, and daily urinary and faecal excretion). These data were provided for a number of radionuclides selected as those most likely to be encountered in the workplace. Predicted values of the measured quantities for various times after a single intake or for routine monitoring were given in terms of the activity of the intake per unit activity measured. Standard dose coefficients would then be used to calculate effective dose from the assessed intake.

## 1.4 Changes in Publication 103 (ICRP, 2007) that affect the calculation of equivalent and effective dose

(24) In the 2007 Recommendations issued in Publication 103 (ICRP, 2007), the concept and use of equivalent and effective dose remain unchanged, but a number of revisions were made to the methods used in their calculation. Changes were introduced in the radiation and tissue weighting factors, from the values previously recommended in Publication 60 (ICRP, 1991). Since radiation weighting factors ($w_R$) for photons, electron and alpha particles are unchanged, the only difference of potential importance to internally deposited radionuclides is for neutrons (Table 1). The changes made do not reflect the availability of additional data but rather a reconsideration of the appropriate treatment of radiation weighting for protection purposes. The abandonment of a step function for neutron $w_R$ as a function of energy is a reflection of the fact that in practice only a continuous function has been used. The major change in the continuous function is a lower $w_R$ value at low energies which more properly reflects the low LET contribution from secondary photons. In addition, there are good theoretical reasons for assuming that $w_R$ values at high energies will converge with that for protons.

Table 1. ICRP radiation weighting factors

| Radiation Type | Radiation Weighting Factor, $w_R$ | |
|---|---|---|
| | Publication 103 | Publication 60 |
| Photons | 1 | 1 |
| Electrons and muons | 1 | 1 |
| Protons and charged pions | 2 | 5* |
| Alpha particles, fission fragments, heavy ions | 20 | 20 |
| Neutrons | Revised continuous function of neutron energy | Step and continuous functions of neutron energy |

*Pions were not considered

(25) The values of tissue weighting factors ($w_T$) recommended in Publication 103 (ICRP, 2007) are shown in Table 2. Changes from values given in Publication 60 (ICRP, 1991) reflect improved knowledge of radiation risks. The main sources of data on cancer risks were the follow-up studies of the Japanese atomic bomb survivors, used to derive risk coefficients averaged over seven Western and Asian populations with different background cancer rates (ICRP, 2007). The new $w_T$ values are based on cancer incidence rather than fatality data, adjusted for lethality, loss of quality of life and years of life lost. Weighting for hereditary effects is now based on estimates of disease in the first two generations rather than at theoretical equilibrium. The main changes in $w_T$ values in the 2007 Recommendations are an increase for breast (from 0.05 to 0.12), a decrease for gonads (from 0.2 to 0.08) and inclusion of more organs and tissues in a larger 'Remainder' (from 0.05 to 0.12). The remainder dose is now calculated as the arithmetic mean of the doses to the thirteen organs and tissues for each sex (Table 2). Tissue weighting factors continue to represent averages across the sexes and across all ages.

Table 2. Publication 103 (ICRP, 2007) tissue weighting factors

| Tissue | $w_T$ | $\sum w_T$ |
|---|---|---|
| Bone-marrow, breast, colon, lung, stomach, remainder tissues *(13*)* | 0.12 | 0.72 |
| Gonads | 0.08 | 0.08 |
| Urinary bladder, oesophagus, liver, thyroid | 0.04 | 0.16 |
| Bone surface, brain, salivary glands, skin | 0.01 | 0.04 |

*Remainder Tissues: adrenals, extrathoracic (ET) regions of the respiratory tract, gall bladder, heart, kidneys, lymphatic nodes, muscle, oral mucosa, pancreas, prostate (male), small intestine, spleen, thymus, uterus/cervix (female).

(26) A further important change introduced in the 2007 Recommendations is that doses from external and internal sources are calculated using reference computational phantoms of the human body ICRP, 2009). In the past, the Commission did not specify a particular phantom, and in fact various mathematical phantoms such as hermaphrodite MIRD-type phantoms (Snyder *et al*, 1969), the sex-specific models of Kramer *et al* (1982), or the age-specific phantoms of Cristy and Eckerman (1987) have been used. Voxel models, constructed from medical imaging data of real people,

give a more realistic description of the human body than afforded in mathematical (or stylised) phantoms. Thus, the ICRP decided to use voxel models to define the reference phantoms to be used in the calculations of dose distribution in the body for both internal and external exposures. These models (or computational phantoms), described in Publication 110 (ICRP, 2009), represent the Reference Male and Female, and have organ masses in compliance with the reference anatomical values compiled in Publication 89 (ICRP, 2002). These phantoms are designed specifically for the calculation of the radiological protection quantities corresponding to the effective dose concept of the 2007 Recommendations.

(27) Equivalent doses to organs and tissues, $H_T$, are calculated separately for the Reference Male and Reference Female and then averaged in the calculation of effective dose, $E$:

$$E = \sum_T w_T \left[ \frac{H_T^M + H_T^F}{2} \right]$$

Where :

$$H_T^M = \sum_T w_R D_{T,R} \qquad \text{(male)}$$

$$H_T^F = \sum_T w_R D_{T,R} \qquad \text{(female)}$$

(28) It is made clear in Publication 103 (ICRP, 2007) that effective dose is intended for use as a protection quantity on the basis of reference values and relates to reference persons rather than specific individuals. The main uses of effective dose are in prospective dose assessment for planning and optimisation in radiological protection, and retrospective demonstration of compliance for regulatory purposes. Sex-averaging in the calculation of equivalent and effective doses, implicit in the past use of hermaphrodite mathematical phantoms, is now explicit in the averaging of equivalent doses to adult male and female phantoms. Sex- and age-averaging in the derivation of tissue weighting factors can be seen to obscure differences in estimates of absolute radiation detriment between men and women and between adults and children. However, practical protection would not be improved by calculating effective dose separately for males and females and to do so might give a misleading impression of the precision of these quantities.

## 1.5 Biokinetic models implemented in this report

(29) Biokinetic models for individual elements and their radioisotopes are used to calculate the total number of transformations occurring within specific tissues, organs or body regions (source regions) during a given period of time (usually 50 y for adults, or to age 70 y for children) by determining the time-integrated activity in each source region. Dosimetric models are used to calculate the deposition of energy in all important organs/tissues (targets) for transformations occurring in each source region, taking account of the energies and yields of all emissions (Eckerman, 1994).

Committed absorbed dose in grays can then be calculated, knowing the number of decays occurring in source regions and energy deposition in target regions.

(30) Biokinetic models of the alimentary and respiratory tracts are used to define the movement of radionuclides within these systems, resulting in absorption to blood and/or loss from the body. The behaviour of radionuclides absorbed to blood is described by element-specific systemic models that range in complexity. These models are intended both for the derivation of dose coefficients and the interpretation of bioassay data. The models used in this report are as given below, with more information provided in Chapter 3.

### 1.5.1 Human Respiratory Tract Model

(31) The Human Respiratory Tract Model (HRTM) described in Publication 66 (ICRP, 1994a) has been updated in this report to take account of data accumulated since its publication, although the basic features of the model remain unchanged. Inhaled particles containing radionuclides deposit in the extrathoracic airways (nose, larynx, etc.), the bronchial and bronchiolar airways of the lung and the alveolar interstitial region, with deposition in the different regions being mainly dependent on particle size (ICRP, 1994a, 2002b). Removal from the respiratory tract occurs mainly by dissolution and absorption to blood and the competing process of transport of particles to the throat followed by their entry into the alimentary tract. The proportions absorbed to blood or cleared by particle transport depend on the speciation and the solubility of the material, and on the radioactive half-life of the radionuclide. The ICRP model for the respiratory tract is also applied here to gases and vapours and to inhalation of radon and its radioactive progeny.

(32) For absorption to blood, the main changes introduced in this report are:

- Redefinition of the Type F, M and S absorption defaults: larger $f_r$ values for M and S of 0.2 and 0.01, rather than 0.1 and 0.001, respectively, with lower $s_r$ values of 3 $d^{-1}$ for M and S, and 30 $d^{-1}$ for F, rather than 100 $d^{-1}$.
- Material-specific parameter values for the rapid dissolution fraction ($f_r$) and the rapid and slow dissolution rates ($s_r$ and $s_s$) in selected cases where sufficient information is available (*e.g.* forms of uranium).
- Element-specific values of $s_r$ and the bound state parameters, $f_b$ and $s_b$, where sufficient information is available.
- Revised treatment of gases and vapours in which solubility and reactivity are defined in terms of the proportion deposited in the respiratory tract. The default assumption is 100% deposition (20% $ET_2$, 10% BB, 20% bb and 50% AI), and Type F absorption. The SR-0, -1, -2 classification has not been found to be helpful and is not used.

(33) For clearance by particle transport the main changes are:

- More realistic clearance from the nasal passage, including transfer from the anterior to the posterior region, based on recent human experimental studies.
- Revised characteristics of slow particle clearance from the bronchial tree based on recent human experimental studies; it is now assumed that it occurs only in the bronchioles rather than as a particle size dependent phenomenon throughout the bronchial tree.

- Longer retention in the alveolar-interstitial region of the lung, with a revised model structure, based on recent data including long-term follow-up of workers exposed to insoluble $^{60}$Co particles, and plutonium dioxide.

### 1.5.2 Human Alimentary Tract Model (HATM), Publication 100 (ICRP, 2006)

(34) The Publication 30 (ICRP, 1979) model of the gastrointestinal tract has been replaced by the Human Alimentary Tract Model (HATM) described in Publication 100 (ICRP, 2006). The main features of the HATM can be summarised as follows:

- Inclusion of all alimentary tract regions: oral cavity, oesophagus, stomach, small intestine, right colon, left colon and rectosigmoid (the sigmoid colon and rectum).
- A default assumption that absorption of an element and its radioisotopes to blood occurs exclusively in the small intestine, *i.e.* the total fractional absorption, $f_A$ equals the fractional absorption from the small intestine, $f_{SI}$. Model structure to allow for absorption in other regions, where information is available.
- A model structure that allows for retention in the mucosal tissues of the walls of alimentary tract regions, and on teeth, where information is available.
- Explicit specification of the location of target regions for cancer induction within each alimentary tract region.

### 1.5.3 Systemic models

(35) A systemic model describes the time-dependent distribution and retention of a radionuclide in the body after it reaches the systemic circulation, and its excretion from the body. In contrast to ICRP's current and past biokinetic models describing the behaviour of radionuclides in the respiratory and alimentary tracts, ICRP's systemic models have generally been element-specific with regard to model structure as well as parameter values. A single generic model structure that depicts all potentially important systemic repositories and paths of transfer of all elements of interest in radiation protection would be too complex to be of much practical use. However, generic model structures have been used in previous ICRP documents to address the systemic biokinetics of groups of elements, typically chemical families, known (or expected to have) qualitatively similar behaviour in the body. For example, Publication 20 (ICRP, 1973) introduced a generic model formulation for the alkaline earth elements calcium, strontium, barium and radium, but provided element-specific values for most model parameters. In Parts 1-3 of Publication 30 (ICRP, 1979, 1980, 1981) a model developed for plutonium, including parameter values as well as model structure, was applied to most actinide elements. The use of generic systemic model structures was increased in ICRP's reports on doses to members of the public from intake of radionuclides (ICRP, 1993b, 1995a, 1995c) and is further expanded in this report because it facilitates the development, description, and application of systemic biokinetic models. An important development is that, as the availability of data allows, models have been made to be physiologically realistic with regard to the

dynamics of organ retention and excretion so that they are applicable to the interpretation of bioassay data as well as the calculation of dose coefficients.

## 1.6 Dosimetry implemented in this report

(36) Dose calculations involve the use of nuclear decay data, anthropomorphic phantoms that describe the human anatomy and codes that simulate radiation transport and energy deposition in the body. The data provided in this report are calculated using revised decay data (Publication 107, ICRP, 2008), the ICRP reference computational phantoms of the adult male and female based on medical imaging data (Publication 110, ICRP, 2009) and well-established Monte Carlo codes (Kawrakow et al, 2009), (Pelowitz, 2008), Niita et al, 2010. .

(37) For all dose calculations, radionuclides are assumed to be uniformly distributed throughout source regions, although these can be whole organs (*e.g.* liver) or a thin layer within a tissue (*e.g.* bone surfaces). Similarly, target cells are assumed to be uniformly distributed throughout target regions that vary in size from whole organs to layers of cells. Doses from 'cross-fire' radiation between source and target tissues are important for penetrating photon radiation. For 'non-penetrating' alpha and beta particle radiations, energy will in most cases be largely deposited in the tissue in which the radionuclide is deposited. Photon and electron transport is followed for most source and target combinations. Additionally special considerations are taken into account for alpha and beta emissions in a number of important cases. These include:

- Doses to target cells in the walls of the respiratory tract airways from radionuclides in the airways (ICRP, 1994a).
- Doses to target regions in the alimentary tract from radionuclides in the lumen (ICRP, 2006).
- Doses to cells adjacent to inner bone surfaces (50 μm layer; see below) and all red marrow from radionuclides on bone surfaces and within bone mineral.

### 1.6.1 Nuclear Decay Data, Publication 107 (ICRP, 2008)

(38) A fundamental requirement for dose calculations is reliable information on half-life, modes of decay, and the energies and yields of the various radiations emitted by nuclides and their progeny (Eckerman *et al*, 1994; Endo *et al* 2003, 2004). The calculations in this report use the nuclear decay data provided in Publication 107 (ICRP, 2008). This publication replaces Publication 38 (ICRP, 1983) and consists of an explanatory text, with an accompanying CD-ROM, providing data on the radiation emissions of 1252 radioisotopes of 97 elements. Radioisotopes of elements of atomic number less than 101 were included in Publication 107 if their half-lives exceed one minute or if they are the progeny of a selected radionuclide and if the basic nuclear structure data enabled a meaningful analysis of their emissions. Presentation using CD-ROM has enabled the complete listing of emitted radiations, and more details of Auger cascades and spontaneous fission data. The data given include: energies and intensities of emitted radiations; beta, neutron and Auger-CK spectra; spontaneous fission radiations and alpha recoil; half-lives, branching decay and chains; and no cut-off on the number of emissions.

### 1.6.2 Adult Reference Computational Phantoms, Publication 110 (ICRP, 2009)

(39) Traditionally, stylised computational phantoms of human anatomy have been utilised for assembling dose coefficients for both external and internal radiation protection. These phantoms are constructed using mathematical surface equations to describe internal organ anatomy and exterior body surfaces of reference individuals (Cristy, 1980; Cristy and Eckerman, 1987), and as such, are limited in their ability to capture true anatomic realism completely. As an alternative format for radiation transport simulation, voxel phantoms are based on segmented tomographic data of real individuals obtained from computed tomography or magnetic resonance imaging (Zankl *et al*, 2002, 2003, 2007). As outlined above, the 2007 Recommendations adopted the use of realistic anatomical models for the revision of dose coefficients for both internal and external radiation sources. Publication 110 (ICRP, 2009) describes the development and intended use of the computational phantoms of the ICRP adult Reference Male and Reference Female. The reference phantoms were constructed after modifying the voxel models of two individuals whose body height and mass closely matched reference values. Organ volumes of both models were adjusted to yield organ masses consistent with ICRP reference data given in Publication 89 (ICRP, 2002a) without compromising their anatomic realism regarding organ shape, depth, and position in the body. The report describes the methods used for this process and the anatomical and computational characteristics of the resulting phantoms.

(40) The computational phantoms of adult Reference Male and Female may be used, together with codes that simulate radiation transport and energy deposition, for the assessment of the mean absorbed dose, $D_T$, in an organ or tissue T, from which equivalent doses and the effective dose may be successively calculated.

### 1.6.3 Advances in skeletal dosimetry

(41) In this report, the skeletal dosimetry models of Publication 30 (ICRP, 1979) have been substantially updated for all radiations emitted from internalised radionuclides – alpha particles, electrons, beta particles, photons, and neutrons (e.g. from spontaneous fission). Improvements over the Publication 30 model include a more refined treatment of the dependence of the absorbed fraction on particle energy, marrow cellularity, and bone-specific spongiosa micro-architecture. Two reference sets of skeletal images were established for radiation transport simulation. The first included 1-mm *ex vivo* CT images of some 38 skeletal sites harvested from a 40-year male cadaver (Hough *et al*, 2011). These images were used to establish fractional volumes of cortical bone, trabecular spongiosa, and medullary cavities by skeletal site, and to serve as the *macroscopic* geometric model for particle transport. The second included 30-µm microCT images of cored samples of trabecular spongiosa to establish fractional volumes of trabecular bone and marrow tissues, and to serve as the *microscopic* geometric model for particle transport. Both image sets were then combined during paired-image radiation transport (PIRT) of internally emitted electrons (Shah *et al*, 2005). Source tissues were: bone marrow (active and inactive),

mineral bone surfaces (trabecular and cortical), and mineral bone volumes (trabecular and cortical). Target tissues considered were: active marrow (surrogate tissue for the hematopoietic stem and progenitor cells), and a revised 50-µm model of the skeletal endosteum (surrogate tissue for the osteoprogenitor cells) (see 'Endosteum' in the Glossary). Absorbed fractions for internalised alpha particles and neutron-generated recoil protons were established based on path length-based transport algorithms given in Jokisch *et al* (2011a, 2011b). Values of absorbed fractions to active marrow and endosteum for internally-emitted photons and neutrons were obtained by first tallying energy-dependent particle fluences within the spongiosa and medullary cavity regions of the Publication 110 reference adult male and female voxel phantoms (ICRP, 2009) and then applying fluence-to-absorbed dose response functions (DRFs). Further details on the derivations of these photon and neutron skeletal dose-response functions are given in Johnson *et al* (2011) and Bahadori *et al* (2011), respectively, as well as in Annexes D and E of Publication 116 (ICRP, 2010).

## 1.7 Interpretation of bioassay data

(42) The system of dose assessment from bioassay data that is generally applied relies first on the evaluation of the intake of a radionuclide either from direct measurements (*e.g.* external monitoring of the whole body or of specific organs and tissues) or indirect measurements (*e.g.* of urine, faeces or environmental samples). Predicted values of these measured quantities for unit intake of a radionuclide are recommended by ICRP and these values can be used to estimate the intake (ICRP, 1997b). The committed effective dose resulting from any intake is then calculated using the appropriate dose coefficient recommended by ICRP or determined using ICRP's recommended methodology. In some cases national authorities require the assessment of the intake of a radionuclide as well as formal assessment of dose. The data provided also serve this purpose.

(43) It is possible, as discussed by Berkovski *et al* (2003a), to calculate committed effective dose directly from bioassay measurements using functions that relate them to the time of the intake. The main advantage of this approach is that the user does not perform the intermediate step of calculating the intake in order to evaluate the dose. This eliminates the risk of using bioassay functions calculated with a particular biokinetic model and dose coefficients derived from a different (earlier or more recent) version of that model. This has been shown to be a rather frequent cause of miscalculations in intercomparison exercises (IAEA, 2007).

(44) Whichever approach is adopted, the assessed dose is in many cases less sensitive to the choice of parameter values than is the assessed intake. Berkovski *et al* (2003a) showed that for a number of chemical forms of radionuclides the 'dose per unit content' is largely insensitive to the choice of inhaled particle size for a wide range of measurement times following an intake. In such circumstances the need for specific information on the appropriate activity median aerodynamic diameter (AMAD) of an aerosol may not therefore arise. Similarly, dose per unit content may be insensitive to the choice of absorption Type for the specific chemical form involved, for specific ranges of measurement times after the intake.) Care is still

needed in the choice of the most appropriate measurement data and in defining the time of the intake.

(45) Effective dose assessed from bioassay measurements is relatively insensitive to choice of parameter values when the measured quantity is directly related to an organ dose that makes a dominant contribution to the effective dose, *e.g.* in the case of lung retention measurements after inhalation of an insoluble $^{60}$Co compound, where lung dose dominates the effective dose. However, sensitivity to parameter values may be much higher when the measured quantity is not so closely related to the effective dose, for instance when lung dose makes a dominant contribution to effective dose and urine monitoring is employed. For such a case, the results of urine monitoring can provide a reliable measure of doses to systemic organs, but assessed lung dose is sensitive to choice of absorption parameter values. An example is the assessment of effective dose from urine monitoring data after inhalation of an insoluble $^{239}$Pu compound.

## 1.8 Structure of the Report

(46) This report series provides revised dose coefficients for occupational intakes of radionuclides (OIR) by inhalation and ingestion, replacing the Publication 30 series (ICRP, 1979, 1980, 1981, 1988b) and Publication 68 (ICRP, 1994b). It also provides data for the interpretation of bioassay measurements, replacing Publications 54 and 78 (ICRP, 1988a, 1997b).

(47) Chapter 2 of this report discusses the application of dose limits and constraints to the control of occupational exposures to radionuclides. It also outlines the objectives and requirements of monitoring programmes designed to ensure compliance with regulatory requirements. Chapter 3 gives an overview of the biokinetic and dosimetric models used to calculate dose coefficients and bioassay data. It explains the changes made to the Publication 66 Human Respiratory Tract Model (HRTM) (ICRP, 1994a) and describes the main features of the Publication 100 Human Alimentary Tract Model (HATM) (ICRP, 2006). Chapter 3 also provides an introduction to the models used in this series of reports to describe the systemic biokinetics of elements and their radioisotopes. Dosimetric models and methodology are also explained.

(48) Routes of intake other than inhalation and ingestion are not considered in this series of reports for the reasons discussed in Section 3.1. However, a summary of a biokinetic model for radionuclide contaminated wounds, prepared by the U.S. National Council on Radiation Protection and Measurements (NCRP, 2007), is included in Chapter 3.

(49) A description of methods for individual monitoring is given in Chapter 4. The Chapter covers *in vivo* measurements and the analysis of excreta and other biological materials as well as workplace monitoring. The general principles for design of monitoring programmes, types of programmes and monitoring requirements are summarised in Chapter 5. Also covered briefly are wound monitoring and the potential effects of medical intervention. General aspects of retrospective dose assessment are considered in Chapter 6. The Chapter examines the need to understand the exposure situation and radionuclide(s) being handled as well as their physico-

chemical form. It also stresses the need for any assessment to be proportionate to the expected exposure. It discusses the requirements for an effective monitoring programme and summarises approaches to data handling for single or multiple measurements. Uncertainties associated with the use of biokinetic models for the interpretation of the results of bioassay measurements are considered.

(50) Chapter 7 provides a brief outline of the types of information included in subsequent parts of this series of reports: biokinetic data, dose coefficients and data for bioassay interpretation for individual elements and their radioisotopes. Each element section provides tables of dose coefficients (committed effective dose, Sv per Bq intake) for inhalation and ingestion of all relevant radioisotopes and tables of bioassay data, giving values of activity (Bq) retained in the body or specific organs, or excreted in urine or faeces, at various times after unit intake by inhalation or ingestion (*i.e.* 1 Bq). The bioassay data are also presented in graphical form. In addition tables are provided of committed effective dose (Sv) per unit activity measurements (Bq). In cases for which sufficient information is available (principally for actinide elements), lung absorption is specified for different chemical forms and dose coefficients and bioassay data are calculated accordingly.

(51) The CD-ROMs that accompany this series of reports contain a comprehensive set of dose coefficients, dose per unit content (DPUC) values, and bioassay functions for a range of physico-chemical forms and aerosol AMADs. (The printed reports in this series contain data only for the 5 μm AMAD default). In addition to the data in the printed reports, the CD-ROMs provide values for activity retained in the body and daily excretion after unit intake, DPUC values, and reference bioassay functions tabulated at additional times after intake. The dose coefficients and other radionuclide-specific data are provided as a set of data files which may be accessed by the user directly or by using the accompanying Data Viewer. The Viewer permits rapid navigation of the dataset and visualisation of the data in tabulated and graphical formats, such as graphs of the time series of DPUC values or predicted activity content per unit dose (Bq $Sv^{-1}$) as a function of time after intake. Graphical presentations of decay chains and nuclear decay data from Publication 107 (ICRP, 2008) are also included.

# 2 CONTROL OF OCCUPATIONAL EXPOSURES TO RADIONUCLIDES

## 2.1 Limits, Constraints, Reference Levels and Investigation Levels

(52) For occupational exposure to ionising radiation, the Commission continues to recommend that the primary annual limit relating to stochastic effects should be expressed as an effective dose of 20 mSv, averaged over defined 5 year periods (100 mSv in 5 years), with the further provision that the annual effective dose should not exceed 50 mSv in any single year (ICRP, 2007). To prevent deterministic effects, there are additional annual limits on equivalent dose to the lens of the eye (20 mSv averaged over defined 5 year periods, with no single year exceeding 50 mSv), the skin (500 mSv), and the hands and feet (500 mSv), but these are generally not likely to be relevant in the context of intakes of radionuclides. Where workers may be exposed to both external radiation and intakes of radionuclides, the annual dose limit applies to the sum of the effective doses from external radiations and the committed effective dose from intakes of radionuclides occurring within the year.

(53) In the 2007 Recommendations (ICRP, 2007), emphasis was placed on the use of dose constraints and reference levels. Dose constraints were included in the system of radiological protection given in Publication 60 (ICRP, 1991) and their use is developed further in the 2007 Recommendations. A dose constraint is a prospective and source related restriction on the individual dose from a source in planned exposure conditions (except in planned exposure of patients), which serves as an upper bound on the predicted dose in the optimisation of protection for that source. It is a level of dose above which it is unlikely that protection is optimised for a given source of exposure, and for which, therefore, action must almost always be taken. Dose constraints for planned situations represent a basic level of protection and will always be lower than the pertinent dose limit. During planning it must be ensured that the source concerned does not imply doses exceeding the dose constraint. Optimisation of protection will establish an acceptable level of dose below the constraint. This optimised level then becomes the expected outcome of the planned protective actions (ICRP, 2007). The Commission has emphasised that dose constraints are not to be used or understood as prescriptive regulatory limits.

(54) In an emergency or existing controllable exposure situation, the reference levels are taken to represent the level of dose or risk above which it is judged to be inappropriate to plan to allow exposures to occur, and for which therefore protective actions should be planned and their extent be decided through optimisation. The chosen value for a reference level will depend upon the prevailing circumstances of the exposure situation under consideration (ICRP, 2007).

(55) The Commission's constraints and reference levels apply across occupational, public and medical exposures (ICRP, 2007) and three defined bands are recommended. These are: ≤1 mSv; >1 - ≤20 mSv and >20-100 mSv. Doses greater than 100 mSv are only considered in the context of life-saving actions. The first band, ≤1 mSv, applies to exposure situations where individuals receive exposures – usually

planned – that may be of no direct benefit to them but the exposure situation may be of benefit to society. The exposure of members of the public as a result of the planned operation of practices is a prime example of this type of situation. The second band, from 1 mSv to 20 mSv, is of greatest relevance in the context of this report, applying in circumstances where individuals receive direct benefits from an exposure situation. Constraints and reference levels in this band will often be set in circumstances where there is individual surveillance or dose monitoring or assessment, and where individuals benefit from training or information. Examples are the constraints set for occupational exposure in planned exposure situations, or the reference levels for some protective actions in emergency exposure situations (ICRP, 2007). Exposure situations involving abnormally high levels of natural background radiation, or stages in post-accident rehabilitation may also be in this band. The third band, from 20 mSv to 100 mSv, applies in unusual, and often extreme, situations where actions taken to reduce exposures would be disruptive. Reference levels and, occasionally, constraints could also be set in this range in circumstances where benefits from the exposure situation are commensurately high. Action taken to reduce exposures in a radiological emergency is the main example of this type of situation.

(56) The Commission considers that it will usually be appropriate for dose constraints to be fixed by an operator at the operational level or by expert bodies or regulatory authorities. The overall responsibility should be with those who are responsible for worker exposure.

(57) As described in Publications 75 and 78 (ICRP, 1997a,b), investigation levels are set to trigger assessment of the conditions giving rise to the exposure. They are therefore used retrospectively. Investigation levels can be set for any operational parameter related to monitoring of individuals or of the working environment. Investigation levels set for individual radionuclides should take account of the presence of other radionuclides in the working environment.

## 2.2 Control of Worker Doses

(58) In occupational exposure, doses are often received from both external and internal radiation sources. For external exposure, individual monitoring is usually performed by measuring the personal dose equivalent using personal dosemeters and taking this measured value as an acceptable estimate of the value of effective dose. For internal exposure, committed effective dose values are determined from measurements of radionuclide activities in the body, in bioassay samples or in the workplace.

(59) For practical purposes, the annual effective dose, $E$, can in most situations of occupational exposure be estimated as:

$$E \cong H_p(10) + E(50)$$

where $H_P(10)$ is the personal dose equivalent from external exposure, normally defined by the dose equivalent at a depth of 10 mm in the body below the

position where the dosemeter is worn, and $E(50)$ is the committed effective dose from internal exposure as assessed by:

$$E(50) = \sum_{j} e_{j,inh}(50) \cdot I_{j,inh} + \sum_{j} e_{j,ing}(50) \cdot I_{j,ing}$$

where $e_j(50)$ is the dose coefficient (committed effective dose per unit intake, Sv $Bq^{-1}$) of a radionuclide, integrated over 50 years after intake by inhalation (inh) and/or ingestion (ing). The intakes, $I_j$ (Bq), may be for one or a number of radionuclides.

(60) The dose coefficient for intakes of radionuclides is the fundamental quantity recommended by ICRP for protection purposes. The Annual Limit on Intake (*ALI*) and the Derived Air Concentration (*DAC*) are derived parameters that can be useful in the control of exposures.

(61) The *ALI* was defined in Publication 60 (ICRP, 1991, paragraph S30) as an intake (in Bq) of a radionuclide in a year which would lead to a committed effective dose of 20 mSv (0.02 Sv). The average annual limit on intake for workers would thus be:

$$ALI_j = \frac{0.02}{e_j(50)}$$

(62) The *DAC* is the activity concentration in air (in Bq $m^{-3}$) of the radionuclide considered which would lead to an intake of an *ALI* assuming a sex-averaged breathing rate of 1.1 $m^3$ $h^{-1}$ and an annual working time of 2000 h. Then the *DAC* is given by:

$$DAC_j = \frac{ALI_j}{2200}$$

(63) ICRP does not now give ALI values because it considers that for compliance with dose limits it is the total dose from external radiation as well as from intakes of radionuclides that must be taken into account, as indicated above. It is, however, noted that the ALI concept can be useful in various practical situations, characterising the relative hazard of radiation sources to ensure that appropriate administrative controls are in place. ALI values can be easily calculated using the equations given in the previous paragraphs.

## 2.3 Objectives of Monitoring

(64) The purpose of monitoring for internal exposure to radionuclides is to verify and document that the worker is protected adequately against radiological risks, and that the protection afforded complies with legal requirements. Two types of monitoring of internal exposures of workers can be identified: *workplace monitoring* and *individual monitoring.*

(65) Workplace monitoring of internal exposures makes use of measurements made in the working environment. An example is the measurement of radionuclide concentration(s) in air using static air samplers. In general, workplace monitoring complements individual monitoring. It may be used for monitoring internal exposures in place of individual monitoring when the latter is not justified, or where the sensitivity of individual monitoring is inadequate. It can be used to provide an assessment of exposure for groups of workers, but this requires assumptions to be made about exposure conditions. It is also of value in demonstrating that working conditions meet safe working criteria and have not changed. It can indicate the release of radionuclides into the working environment and so trigger subsequent individual monitoring measurements.

(66) Individual monitoring of internal exposure uses measurements made for individual workers for the assessment of their *dose of record*, together with other dosimetric quantities if required. The principal objectives of individual monitoring in planned and existing situations are:

- to assess the worker's dose of record and to demonstrate compliance with regulatory requirements.
- to contribute to the safety management and control of the operation of the facility.

(67) The principal objectives of individual monitoring of workers in emergency situations are:

- to document the worker's exposure in terms of dose of record and, if appropriate, in terms of absorbed doses in significantly exposed tissues.
- to provide information for the initiation and support of any appropriate health surveillance and treatment.

(68) Usually it is necessary to carry out only a simple assessment of dose to demonstrate compliance with regulatory requirements when annual doses are expected to be only small fractions of the dose limits. In some countries it may be unnecessary to make an assessment of individual dose, the measured value being compared with an appropriate threshold or recording level. At higher doses more emphasis will need to be placed upon specific dose assessments and the circumstances of any exposure.

(69) Measurements, together with information about the workplace, should enable each radionuclide to be identified, its activity quantified, and the measurement result interpreted in terms of intake and/or committed effective dose. There may be some circumstances where individual monitoring techniques are not adequate to assess doses and it may be necessary to combine individual and workplace monitoring techniques.

## 2.4 Categories of Individual Monitoring Programme

(70) Routine monitoring is performed under conditions of essentially continuous risk of contamination of the workplace as a result of normal operations, or where undetected accidental intakes may occur. Measurements in a routine monitoring programme are made at pre-determined times not related to known intakes, and

therefore it is necessary to make some assumptions about the pattern of intakes. National or local legislation or regulations may also set the requirements for systematic routine monitoring that may be needed if exposures could exceed a specified fraction of the dose limit or a dose constraint.

(71) Other monitoring programmes may be conducted in relation to a particular task, or to determine intakes in actual or suspected abnormal conditions. In these circumstances, the time of intake, or potential intake, is likely to be known and workplace monitoring programmes may provide some information on the physical and chemical nature of any contamination. Special monitoring is performed to quantify significant exposures following actual or suspected abnormal events. Confirmatory monitoring is performed where there is a need to check assumptions made about exposure conditions, for example in order to confirm the effectiveness of protection measures. Task-related monitoring is carried out for workers engaged on specific operations.

## 2.5 Needs for Individual Monitoring

(72) An important function of an employer and/or licensee is that of maintaining control over sources of exposure and ensuring the protection of workers who are occupationally exposed. In order to achieve this, the Commission continues to recommend the classification of controlled and supervised areas (ICRP, 2007). A controlled area requires consideration of specific protection measures and safety provisions for controlling normal exposures or preventing the spread of contamination during normal operations, and preventing or limiting the extent of accidental exposures. A supervised area is one in which the radiological conditions are kept under review but special procedures are not normally needed.

(73) It is necessary to identify groups of workers for whom individual monitoring is needed. The decision to provide individual monitoring depends on many factors. Routine individual monitoring for intakes of radioactive material should be used for workers in areas that are designated as controlled areas specifically in relation to the control of contamination and in which significant intakes cannot be excluded.

(74) Workers in controlled areas are the group who are most often monitored for radiation exposures incurred in the workplace, and may also receive special medical surveillance. They should be well informed and specially trained, and form a readily identifiable group.

(75) The use of individual monitoring for workers whose annual doses could exceed 1 mSv is common practice in many organisations although it may not be required by legislation. Regulatory, technical and managerial considerations may support arguments for the assessment of individual dose at these lower levels, at least for those radionuclides for which assessment is straightforward and practical.

(76) The following examples indicate the type of operations where experience has shown that it is necessary to give consideration to routine individual monitoring for internal exposure of workers:

- the handling of 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;

- maintenance of reactor facilities;
- handling of radioactive waste, for example from nuclear facilities and hospitals;
- the processing of plutonium and other transuranic elements;
- the processing of thorium ores and the use of thorium and its compounds (these activities can lead to internal exposure from both radioactive dusts and thoron [$^{220}$Rn] and its progeny);
- the mining, milling and refining of uranium ores;
- natural and enriched uranium processing and fuel fabrication;
- work with large quantities of naturally occurring radioactive materials (NORM);
- the production of radiopharmaceuticals;
- the handling of large quantities of $^{131}$I for medical applications.

(77) The results of monitoring of the workplace may also indicate a need for a temporary programme of special individual monitoring aimed at identifying any need for a routine programme of workplace monitoring.

## 2.6 Female Workers: pregnancy and breast-feeding

(78) It is the Commission's policy (ICRP, 2007) that the methods of protection at work for women who are pregnant should provide a level of protection for the embryo/fetus broadly similar to that provided for members of the public. The Commission considers that this policy will be adequately applied if the mother is exposed, prior to her declaration of pregnancy, under the system of protection recommended by the Commission. Once pregnancy has been declared, and the employer notified, additional protection of the embryo/fetus should be considered. The working conditions of a pregnant worker, after declaration of pregnancy, should be such as to make it unlikely that the additional external dose to the fetus, together with the committed effective dose to the fetus and newborn child from intakes of radionuclides before or during the pregnancy, would exceed about 1 mSv.

(79) ICRP has provided information in Publications 88 and 95 (ICRP, 2001, 2004) on doses to the embryo, fetus and newborn child following intake of radionuclides by female workers either before or during pregnancy or during lactation. Comparisons of fetal dose coefficients given in Publication 88 with corresponding adult dose coefficients showed that doses received by a woman from intakes before or during pregnancy will in most cases be substantially greater than doses to her fetus. However, doses to the offspring can exceed doses to the mother for a number of radionuclides. In particular, the requirements of skeletal development during fetal growth, particularly in late pregnancy, can lead to significant uptake of radioisotopes of phosphorus and of calcium and, to a lesser extent, other alkaline earth elements. Thus, offspring:adult dose ratios were up to factors of about 10 – 20 for isotopes of P and Ca and 2 – 6 for isotopes of Sr (Stather *et al*, 2003; ICRP 2004). Uptake of radioisotopes of iodine by the fetal thyroid can also lead to greater doses to the fetus than to the mother following intakes late in pregnancy (dose ratios of up to about 3) (Berkovski *et al*, 2003b). Other radionuclides for which doses to the fetus can exceed

doses to the mother include tritium as tritiated water, $^{14}C$ and $^{35}S$. Offspring:adult dose ratios are greatest following ingestion or inhalation of soluble (Type F) forms. Values of offspring:adult ratios may change as a result of future calculations following from Publication 103 (ICRP, 2007) and associated changes. Offspring doses may also be of concern when the dose ratio is <1 since a dose of 1 mSv might be reached at otherwise acceptable levels of occupational dose (Phipps *et al*, 2001).

(80) When a worker has declared pregnancy, possible doses to her child will be taken into account in measures taken to limit exposures. Thus, offspring doses resulting from intakes later in pregnancy may in practice be of less importance than doses resulting from intakes before the declaration of pregnancy**.** A number of radionuclides of potential significance in this category have been identified, including $^{63}Ni$ and $^{55}Fe$ (Phipps *et al*, 2001; Nosske and Karcher, 2003).

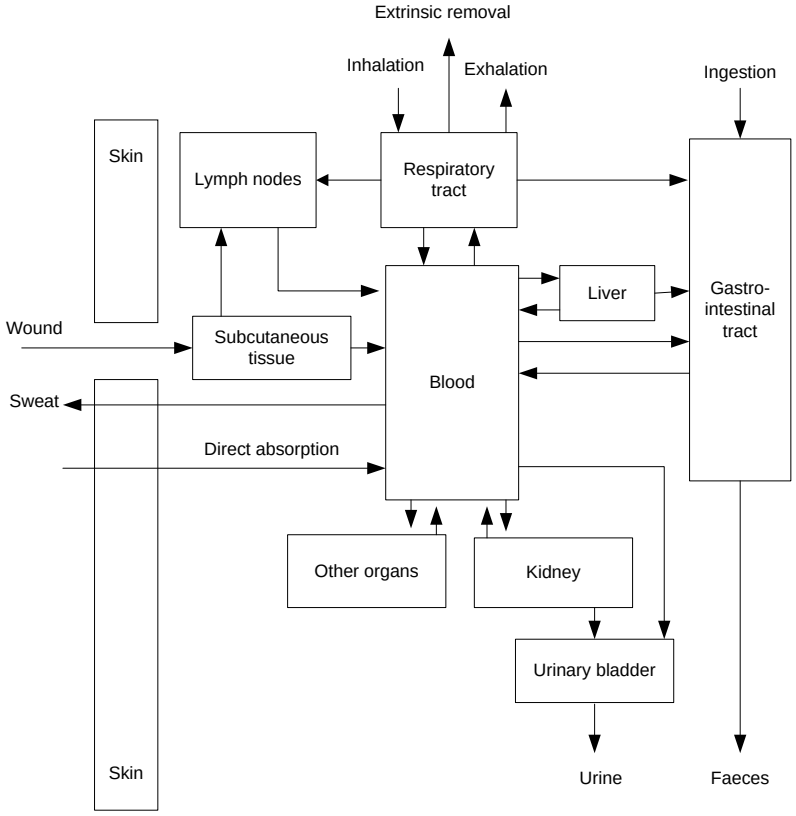
(81) In general, doses to the infant from radionuclides ingested in breast-milk are estimated to be small in comparison with doses to the reference adult (ICRP, 2004). On the basis of the models developed in Publication 95 (ICRP, 2004), it is only in the cases of tritiated water, $^{45}Ca$, $^{75}Se$ and $^{131}I$ that infant doses may exceed adult doses, by factors of between 1 and 3. Infant doses are highest when maternal intakes by ingestion occur shortly after birth because maximum transfer occurs under these conditions. Ratios of infant to adult doses are generally lower for intakes by inhalation than for ingestion. Comparisons with Publication 88 (ICRP, 2001) doses to the offspring due to *in utero* exposures show that in most cases these are more important than doses that may result from breast feeding; exceptions include $^{60}Co$, $^{131}I$ and $^{210}Po$.

# 3 BIOKINETIC AND DOSIMETRIC MODELS

## 3.1 Introduction

(82) This chapter gives an overview of the biokinetic and dosimetric models used to calculate dose coefficients and bioassay data. It explains the changes made here to the Human Respiratory Tract Model (HRTM) (ICRP, 1994a) and describes the main features of the Human Alimentary Tract Model (HATM) (ICRP, 2006). It also provides an introduction to the models used in this series of reports to describe the systemic biokinetics of elements and their radioisotopes. Dosimetric models and methodology are also explained.

(83) Radionuclide exposures in the workplace can lead to intakes by a number of routes: inhalation, ingestion, entry through intact skin and wounds. Figure 2 summarises the routes of intake, internal transfers, and routes of excretion.




(84)

Figure 2. Summary of the main routes of intake, transfer and excretion of radionuclides in the body

(85) For inhalation, the HRTM (ICRP, 1994a) was applied in Publication 68 (ICRP, 1994b) and in subsequent publications on dose coefficients (ICRP, 1995c, 1996). For these implementations of the HRTM, chemical forms of radionuclides that

had been assigned to Publication 30 inhalation Classes D, W, and Y were assigned to HRTM absorption Types F, M, and S respectively. In the element sections of this series of reports, information is reviewed on the lung clearance characteristics of different chemical forms of each element, within the framework of the HRTM. The opportunity has been taken to update some aspects of the HRTM in the light of information that has become available since Publication 66 was issued, as summarised in Section 1.5.2 above, and described in Section 3.2 below.

(86) For ingestion of radionuclides, the HATM (ICRP, 2006) is applied. The model is also used for radionuclides in particles cleared to the throat from the respiratory tract after inhalation. In the HATM, fractional absorption of radionuclides is specified by the alimentary tract transfer factor, $f_A$, instead of the $f_1$ value as given for the gastrointestinal tract (GIT) model described in Publication 30 (ICRP, 1979). The $f_A$ value describes total absorption from all regions of the alimentary tract, although the default assumption is that all absorption takes place in the small intestine.

(87) ICRP has generally not given advice on assessing doses from intakes of radionuclides transferred from wound sites to blood and other organs and tissues. Internal exposure resulting from wounds almost always arises because of accidents in the workplace, rather than as a result of routine operations that are subject to the normal environmental controls. Uptake from wounds can vary greatly depending on the circumstances of a particular incident and in practice the assessment of internal contamination is treated on a case-by-case basis. As a result, provision of generic dose coefficients or bioassay data would be of limited value. Information on the transfer of radionuclides from wound sites has, however, been reviewed by a Scientific Committee of NCRP and these data have been used to develop a model to describe the transfer of material from wounds after intakes in different physico-chemical forms (NCRP, 2007). Section 3.4 summarises the main features of the NCRP model, since this information may be of use in the prospective assessment of doses and the interpretation of bioassay data for individual cases of wound contamination.

(88) For each route of intake, a proportion of the radionuclide entering the body is absorbed to blood and distributed systemically. The systemic distribution of radionuclides in the body can be diffuse and relatively homogeneous, as for the examples of tritiated water and radioisotopes of potassium and caesium, or may be localised in certain organs or tissues, as for the examples of radioisotopes of iodine (thyroid), alkaline earth elements (bone), and plutonium (bone and liver). Systemic biokinetic models are used to describe the distribution and excretion of radionuclides absorbed to blood. The systemic models for the elements have been reviewed and revised as necessary to take account of more recent information and provide models that are appropriate for both dosimetry and bioassay interpretation.

(89) Removal of deposited material from the body occurs principally by urinary and faecal excretion although radionuclides may also be lost by exhalation or through the skin (*e.g.* tritiated water (HTO)). Urinary excretion is the removal in urine of radionuclides from blood following filtration by the kidneys. Faecal excretion has two components: systemic (endogenous) faecal excretion which represents removal of systemic material via the alimentary tract, due to biliary secretion from the liver and secretions at other sites along the alimentary tract; and direct (exogenous) faecal

excretion, strictly elimination, of the material passing unabsorbed through the alimentary tract after ingestion or clearance to the throat from the respiratory system after inhalation.

(90) The reference models outlined in this Chapter are assigned reference parameter values and used to calculate body or organ content and daily urinary or faecal excretion at specified times after acute or chronic intake. They are used to calculate reference bioassay functions and, together with dosimetric data, reference dose coefficients.

## 3.2 Revised Human Respiratory Tract Model (HRTM)

(91) The Human Respiratory Tract Model (HRTM) described in Publication 66 (ICRP, 1994a) was applied to calculate inhalation dose coefficients for workers and members of the public in Publications 68, 71 and 72 (ICRP, 1994b, 1995c, 1996), and bioassay functions in Publication 78 (ICRP, 1997b). A revised version of the HRTM is used in this series of reports and is described below.

(92) As in the original version of the HRTM, the respiratory tract is treated as two tissues: the extrathoracic regions (ET) and the thoracic regions (TH). The sub-division of these tissues into regions was based mainly on differences in sensitivity to radiation. The thoracic regions are bronchial, (BB: trachea, generation 0, and bronchi, airway generations 1 – 8), bronchiolar (bb: airway generations 9 – 15), alveolar-interstitial (AI: the gas exchange region, airway generations 16 and beyond); and the thoracic lymph nodes, $LN_{TH}$. The extrathoracic regions are the anterior nasal passage, $ET_1$; the posterior nasal passages, pharynx and larynx, $ET_2$; and the extrathoracic lymph nodes $LN_{ET}$ (Figure 3). For consistency with the HATM, the oral passage is not now included in region $ET_2$ as it was in Publication 66. This does not affect results obtained with the model, because deposition in ET from air entering the mouth was taken to occur only in the larynx.

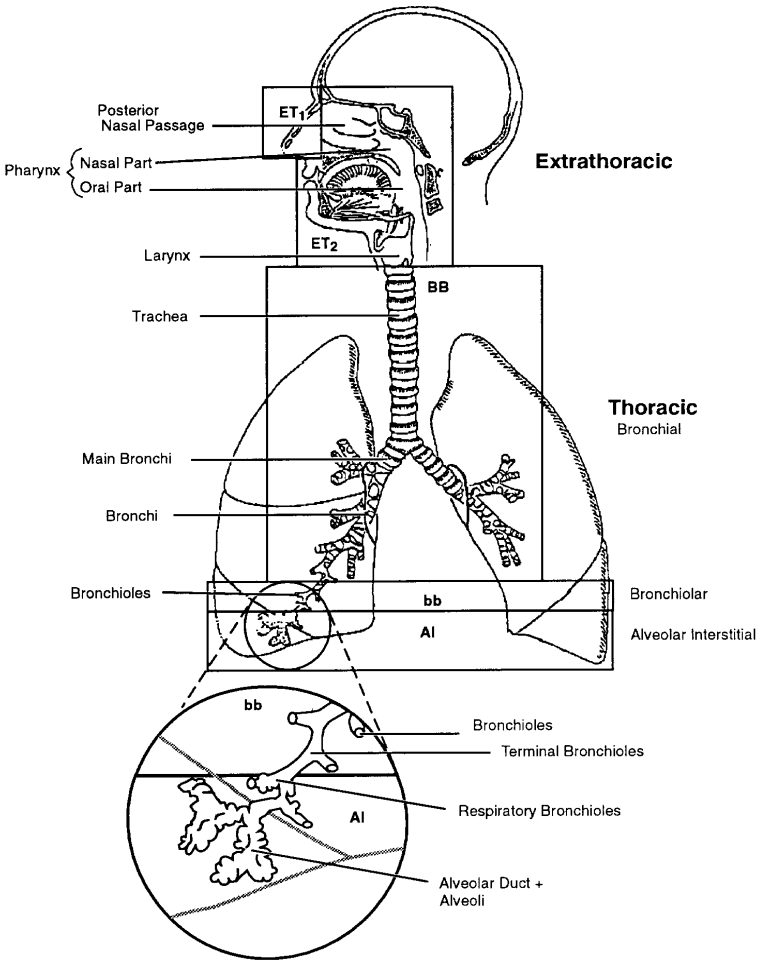



Figure 3. Respiratory tract regions defined in the Human Respiratory Tract Model (HRTM). Note that the oral part of the pharynx is no longer part of $ET_2$.

### 3.2.1 Deposition

***Aerosols of (solid or liquid) particulate materials***

(93) The deposition model described in Publication 66 (ICRP, 1994a) evaluates fractional deposition of an aerosol in each region, for all aerosol sizes of practical interest (0.6 nm – 100 μm). For the ET regions, measured deposition efficiencies were related to characteristic parameters of particle size and airflow, and were scaled by anatomical dimensions to predict deposition under other conditions (*e.g.* sex, ethnic group). For the thoracic airways, a theoretical model of gas transport and particle deposition was used to calculate particle deposition in each of the BB, bb, and AI regions, and to quantify the effects of the subject's lung size and breathing rate. To model particle deposition, the regions were treated as a series of filters, during both inhalation and exhalation. The efficiency of each was evaluated by considering aerodynamic (gravitational settling, inertial impaction) and thermodynamic

(diffusion) processes acting competitively. Regional deposition fractions were calculated for aerosols having lognormal particle size distributions, with geometric standard deviations taken to be a function of the median particle diameter, increasing from a value of 1.0 at 0.6 nm to a value of 2.5 above about 1 μm.

(94) No changes are made here to the Publication 66 implementation of the deposition model for aerosols, except for the distribution of the deposit in the ET airways between regions $ET_1$ and $ET_2$. In Publication 66 (ICRP, 1994a) it was assessed, on the basis of the available information, that deposition in $ET_1$ is somewhat higher than in $ET_2$ during inhalation through the nose, and that most of the particles deposited in $ET_1$ are cleared by nose-blowing, but some clear to $ET_2$ and hence to the alimentary tract on a time scale of hours. However, because of the lack of quantitative information, these judgements were applied in a simplified form in the original HRTM. It was assumed that particles deposited in the nasal passage during inhalation are partitioned equally between $ET_1$ and the posterior nasal passage, which is part of $ET_2$. (However, because of the way the deposition efficiencies were calculated for polydisperse aerosols during inhalation and exhalation, for most aerosol sizes of interest in radiation protection the deposition fractions given in Publication 66 are somewhat higher for $ET_2$ than for $ET_1$.) As described in the section below on particle transport from the ET airways, recent experimental studies (Smith *et al*, 2011) enable a more accurate representation of ET deposition and clearance to be implemented here. Results for a group of subjects indicated that the distribution of the deposit in the ET airways can be characterised by mean deposition fractions of 65% to $ET_1$ and 35% to $ET_2$. To calculate the fractions of inhaled material deposited in $ET_1$ and $ET_2$, the fractions deposited in $ET_1$ and $ET_2$ (calculated using the original HRTM) were summed to give the total deposit in the ET airways, and then re-partitioned 65% to $ET_1$ and 35% to $ET_2$. (For mouth breathing there is no deposition in $ET_1$ and the fraction deposited in $ET_2$ remains as calculated using the original HRTM.)

(95) For inhalation of radionuclides by workers, the reference subjects are taken to be normal nose-breathing adult males and females at light work. However, for simplicity, deposition in (and clearance from) the respiratory tract are calculated for the reference adult male only. For occupational exposure, the default value recommended for the Activity Median Aerodynamic Diameter (AMAD) is 5 μm (ICRP, 1994b), consistent with the review of data by Dorrian and Bailey (1995) and Ansoborlo *et al* (1997). Fractional deposition in each region of the respiratory tract of the reference worker is given in Table 3 for aerosols of 5 μm AMAD.

Table 3 Regional deposition of inhaled 5 μm AMAD aerosols in Reference Workers engaged in light work (% of inhaled activity)

| Region | Deposition (%)[a,b,c] |
|---|---|
| | Male |
| $ET_1$ | 47.94 |
| $ET_2$ | 25.82 |
| BB | 1.78 |
| bb | 1.10 |
| AI | 5.32 |
| Total | 81.96 |

[a]Reference values are given to a greater degree of precision than would be chosen to reflect the certainty with which the average value of each parameter is known.

[b]The particles are assumed to have density 3.00 g $cm^{-3}$, and shape factor 1.5. The particle aerodynamic diameters are assumed to be log-normally distributed with geometric standard deviation, $\sigma_g$ of approximately 2.50. (The value of $\sigma_g$ is not a reference value, but is derived from the corresponding Activity Median Thermodynamic Diameter, AMTD (ICRP, 1994a)).

[c]Light work is defined on the following basis: 2.5 h sitting, at which the amount inhaled is 0.54 $m^3$ $h^{-1}$; and 5.5 h light exercise, at which the amount inhaled is 1.5 $m^3$ $h^{-1}$. For both levels of activity all the inhaled air enters through the nose. The deposition fractions are therefore volume-weighted average values for the two levels of activity given for normal nose-breathing adult males sitting and at light exercise in Publication 66, Annex F (ICRP, 1994a). However, as described in the text, the fractions deposited in $ET_1$ and $ET_2$ from Publication 66 were summed to give the total deposit in the ET airways, and partitioned 65% to $ET_1$ and 35% to $ET_2$.

### *Gases and Vapours*

(96) For radionuclides inhaled as aerosols, the HRTM assumes that total and regional deposits in the respiratory tract are determined only by the size distribution of the inhaled particles. The situation is different for gases and vapours, for which deposition in the respiratory tract depends entirely on the chemical form. In this context, *deposition* refers to how much of the material in the inhaled air remains in the body after exhalation. Almost all inhaled gas 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.

(97) As for particulate forms of radionuclides, default parameter values are provided for use in the absence of more specific information. The general defaults for gases and vapours are 100% total deposition in the respiratory tract (regional deposition: 20% $ET_2$, 10% BB, 20% bb and 50% AI) with Type F absorption (Section 3.2.3). This classification is somewhat different from that recommended in Publication 66, but simpler to apply. In particular, it is assumed by default that there is no deposition in $ET_1$. The SR-0, -1, -2, classification described in Publication 66 was not found to be helpful and is not used here.

(98) In this series of reports, parameter values are adopted for gaseous and vapour forms of compounds of a number of elements, including hydrogen, carbon, sulphur and iodine. In each case, values are given for total deposition, regional deposition and absorption.

### 3.2.2 Clearance: particle transport

(99) The model describes several routes of clearance from the respiratory tract (Figure 4). Some material deposited in $ET_1$ is removed by extrinsic means such as nose-blowing. In other regions, clearance is competitive between the movement of particles towards the alimentary tract and lymph nodes (particle transport), and the absorption into blood of material from the particles in the respiratory tract. Removal rates due to particle transport and absorption to blood are taken to be independent. It is assumed that all clearance rates are independent of age and sex.

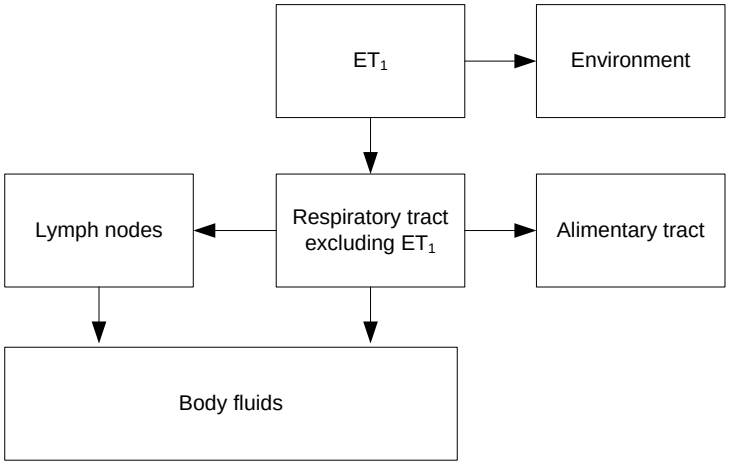



Figure 4. Routes of clearance from the respiratory tract

(100) As in the original HRTM, it is assumed that particle transport rates are the same for all materials. A generic compartment model is therefore provided to describe particle transport of all materials. The original model is shown in Figure 5. Reference values of rate constants were derived, as far as possible, from human studies, since particle transport rates are known to vary greatly among mammalian species. Figure 5 as it stands would describe the retention and clearance of an insoluble material. However, as noted above, there is in general simultaneous absorption to blood. New studies enable more reliable particle transport parameter values to be chosen for the extrathoracic regions (ET); bronchial (BB); bronchiolar (bb) and alveolar-interstitial (AI) regions, than was possible when Publication 66 was issued in 1994.

(101) The revised particle transport model adopted here is shown in Figure 6. Region $ET_2$ is described in the model by two compartments, $ET_{seq}$ and $ET_2'$. Because the oral passage is no longer included in Region $ET_2$ (see above), compartment $ET_2'$ is redefined as consisting of the posterior nasal passage, pharynx and larynx. The compartments used to represent the retention of particles deposited in the BB and bb regions that are cleared slowly (compartments $BB_2$ and $bb_2$ in Figure 5) are no longer

included, and bronchial and bronchiolar retention is represented by the BB' and bb' compartments, respectively. The three AI compartments of the original HRTM have been replaced by the ALV compartment, from which particles either clear to the ciliated airways or penetrate to the interstitium (the INT compartment). Particles clear very slowly from the INT compartment to the lymph nodes.

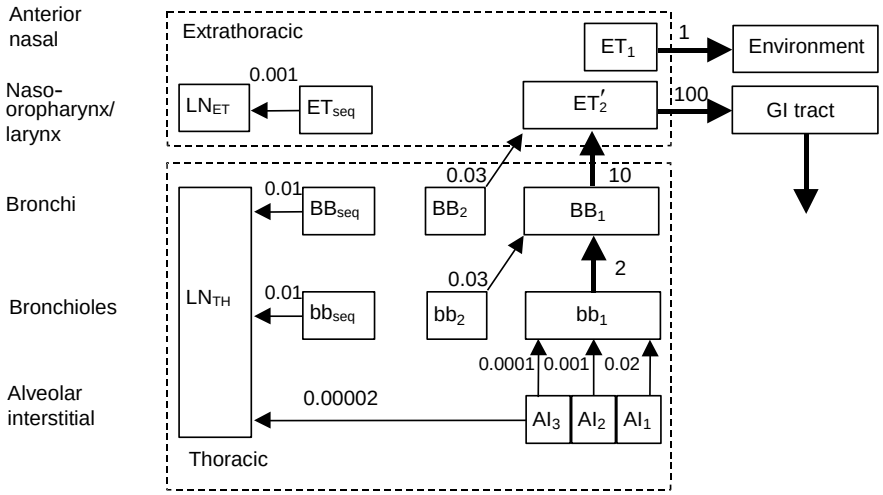



Figure 5. Compartment model representing time-dependent particle transport from each respiratory tract region in the original HRTM. Rates shown alongside arrows are reference values in units of $d^{-1}$. It was assumed that: (i) the AI deposit is divided between $AI_1$, $AI_2$ and $AI_3$ in the ratio 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.

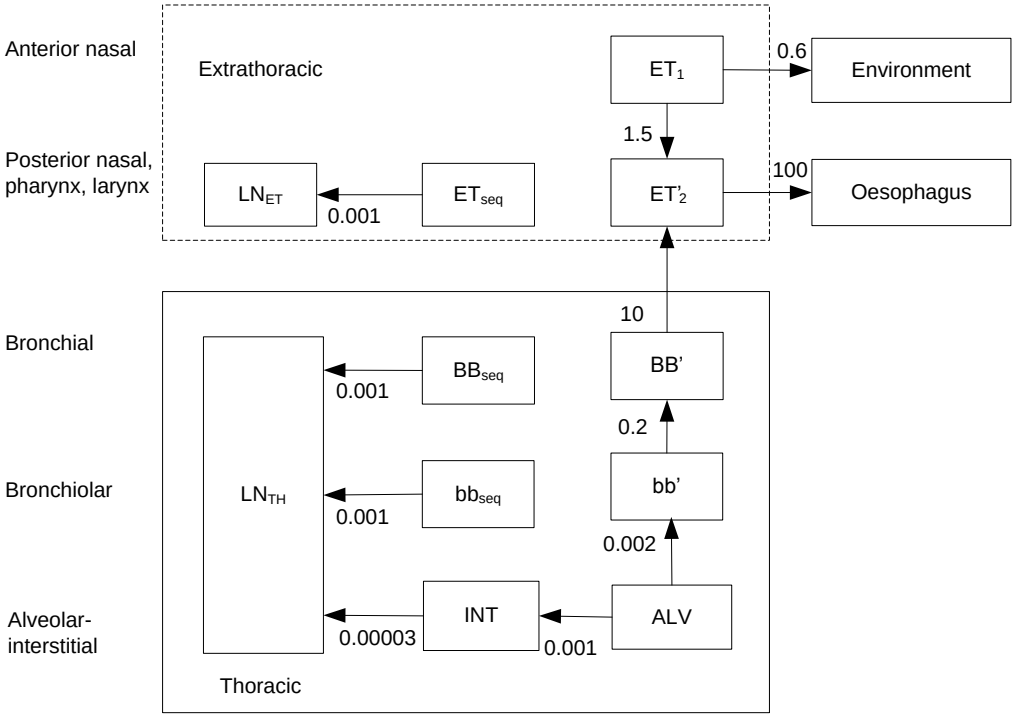



Figure 6. Revised compartment model representing time-dependent particle transport from each respiratory tract region. Rates shown alongside arrows are reference values in units of $d^{-1}$. It is assumed that 0.2% of material deposited in regions $ET_2$, BB and bb is retained in the airway wall ($ET_{seq}$, $BB_{seq}$ and $bb_{seq}$ respectively).

***Particle transport: extrathoracic airways***

(102) The Publication 66 model assumed that of material deposited in the ET airways, about 50% deposits in $ET_1$ (Figure 5), which is cleared by nose blowing at a rate of 1 $d^{-1}$, and the rest deposits in $ET_2$, which clears to the GI tract at a rate of 100 $d^{-1}$. However, there was little information available to quantify clearance from $ET_1$. It was recognised that the fraction deposited in $ET_1$ was generally greater than that in $ET_2$ and that there was slow transfer from $ET_1$ to $ET_2$, but insufficient information was available to quantify these factors and transfer rates. In experiments intended to address this deficiency, subjects inhaled 1.5-, 3- or 6-µm aerodynamic diameter ($d_{ae}$) radiolabelled insoluble particles through the nose while sitting at rest or performing light exercise (Smith *et al*, 2002, 2011). Retention in the nasal airways and clearance by voluntary nose blowing were followed until at least 95% of the initial ET deposit (IETD) had cleared (typically about 2 days). On average, 19% IETD was cleared by nose blowing (geometric mean time for 50% clearance was 8 hours), and the rest was cleared to the alimentary tract: 15% within a few minutes, 21% between a few minutes and an hour, and 45% on a similar time-scale to the fraction cleared by nose-blowing. Measurements in this study, and the previous studies on which the original model was based, indicate that most particles that have not cleared within an hour are retained in the anterior nasal passage.

(103) On the basis of these data, it is assumed here that material deposited in $ET_1$ (now taken to be 65% of the deposit in ET, as described in Section 3.2.1) is cleared at a rate of 2.1 $d^{-1}$ (half-time about 8 hours): about one-third, by nose blowing and two-thirds by transfer to $ET_2$. This is implemented with particle transport rates of 0.6 $d^{-1}$ from $ET_1$ to the Environment and 1.5 $d^{-1}$ from $ET_1$ to $ET_2'$. Clearance from $ET_2'$ is unchanged, with a rate to the alimentary tract of 100 $d^{-1}$ (half-time about 10 minutes). As in the original HRTM, a small fraction of particles deposited in $ET_2$ (but not cleared to it from $ET_1$) is sequestered in the airway wall ($ET_{seq}$) and transferred to lymph nodes. However, the fraction sequestered is increased from 0.05% of the deposit in $ET_2$ in the original HRTM, to 0.2% here, partly because of the smaller fractional deposition in $ET_2$, but also from reconsideration of the experimental data relating to long-term retention of inhaled particles in the nasal passages, which were reviewed in Publication 66.

(104) The changes from the original HRTM treatment of ET will in many cases increase dose coefficients because of the transfer from $ET_1$ to $ET_2$ and hence greater systemic uptake in $ET_2$ and the alimentary tract. The changes will also affect interpretation of measurements of radionuclides in faecal samples: a larger fraction of the material deposited in the nose (which is typically about 50% of the material inhaled) is cleared through the alimentary tract.

***Particle transport: bronchial and bronchiolar airways***

*Slow clearance*

(105) The original HRTM includes a slow phase of clearance of particles deposited in the BB and bb regions (compartments $BB_2$ and $bb_2$ in Figure 5), with a half-time of 23 days. It was based mainly on the results of experiments in which volunteers inhaled a 'shallow bolus' of radio-labelled particles *i.e.*, a small volume of aerosol at the end of each breath, designed to deposit particles in the major airways. A 'slow-cleared' fraction was observed, which was considered to show a better correlation with particle geometric diameter, $d_p$ than with $d_{ae}$ (ICRP, 1994a). The original HRTM assumes that the slow-cleared fraction of particles deposited in BB and in bb ($f_s$) is 0.5 for $d_p \leq 2.5$ μm, and decreases exponentially for larger particles.

(106) In the revised HRTM, a different approach has been taken to slow clearance from the bronchial tree based on more recent human volunteer experiments. In particular, in a series of studies, large particles (6-μm $d_{ae}$) were inhaled extremely slowly, which theoretically should result in most deposition occurring in the bronchioles (*e.g.* Anderson *et al*, 1995; Camner *et al*, 1997; Falk *et al*, 1997, 1999; Philipson *et al*, 2000; Svartengren *et al*, 2001). Retention at 24 hours was much greater than the predicted AI deposition, supporting the concept of slow clearance in the bronchial tree.

(107) Falk *et al* (1997, 1999) compared lung retention of 6 μm $d_{ae}$ Teflon particles inhaled slowly (~45 $cm^3$ $s^{-1}$) with retention of similar particles inhaled at a normal flow-rate (~450 $cm^3$ $s^{-1}$) for up to 6 months. About 50% of the initial lung deposit (ILD) cleared in the first 24 hours following both modes of inhalation. Retention after 24 hours was well described by a two-component exponential function, the clearance rates having half-times of about 3.7 days ('intermediate' phase) and 200 days (attributed to clearance from the AI region). The fractions associated with the

intermediate phase were about 18% ILD after slow inhalation and 6% ILD after normal inhalation. Deposition in the BB, bb and AI regions calculated using three different models showed good agreement with, on average, 17%, 63% and 18% ILD, respectively, after slow inhalation and 30%, 26% and 43% after normal inhalation. Thus, there was a strong correlation between predicted bronchiolar deposition and the amount cleared in the intermediate phase, suggesting that the intermediate phase was associated with about 25% of particles deposited in the bronchioles.

(108) Svartengren *et al* (2001) found very similar retention in each subject when 6 µm $d_{ae}$ particles were inhaled as a shallow bolus and by slow inhalation on separate occasions. One interpretation was that slow clearance is a characteristic of the bronchioles, and the pattern of deposition was very similar, even though the techniques were so different, a view supported by complementary deposition modelling. However, the possibility could not be excluded that the deposition patterns were different, with more bronchial deposition following bolus inhalation than following slow inhalation, and as assumed in the HRTM, slow clearance occurring to a similar extent in both large and small airways.

(109) Philipson *et al* (2000) investigated the effect of $d_p$ directly by administering particles with the same $d_{ae}$, and hence the same lung deposition pattern, but different densities and so different values of $d_p$ ($d_{ae} \approx d_p\sqrt{\rho}$ where $\rho$ is the particle density). Volunteers inhaled 6 µm $d_{ae}$ particles of polystyrene (PSL, density 1.05 g cm$^{-3}$) and Teflon (density 2.13 g cm$^{-3}$). The geometric diameter, $d_p$, of the Teflon was smaller (4.5 µm) than that of the PSL (6.1 µm), and the HRTM predicts $f_s$ to be greater (14% versus 5%). However, retention of the two particles was similar in each subject.

(110) Smith *et al* (2007, 2008) tested these alternative hypotheses more critically, also administering two particles of the same $d_{ae}$, but with a greater difference in densities, and as shallow boluses to minimise alveolar deposition. In one study, volunteers inhaled 5 µm $d_{ae}$ PSL and gold ($\rho$ = 19.3 g cm$^{-3}$) particles; corresponding $d_p$ values were 5 and 1.2 µm and values of $f_s$ were about 10% and 50%, respectively. Hence, according to the HRTM, lung retention of the gold should have been much greater than that of the PSL. However, no significant difference was observed between them in any subject. In another study, 8 µm $d_{ae}$ PSL and gold particles were used and broadly similar results were obtained.

(111) These results are thus inconsistent with the dependence of $f_s$ on $d_p$ assumed in the HRTM. However, the apparent discrepancy with the results of the bolus experiments on which the Publication 66 assumptions were based has not been resolved. A possible explanation may be that the inferred dependence on $d_p$ was fortuitous. It was based mainly on measurements made with relatively large particles ($d_p$ or $d_{ae}$ > 4 µm), and there were relatively few such measurements available at the time.

(112) Another recent study showed inconsistencies with the original HRTM's assumptions on slow particle clearance from the bronchial tree. Gregoratto *et al* (2010), in analysing alveolar retention in the study by Philipson *et al* (1996) (see below), observed that there was far less lung clearance between 7 and 50 days after inhalation than predicted by the HRTM as a result of slow clearance from the BB and bb regions, even assuming no clearance from the AI region over that period.

(113) Most of the relevant recent human studies thus suggest that slow clearance in the conducting airways is associated with particles deposited in the bronchioles: a simpler explanation than the particle-size dependent clearance mechanism assumed in Publication 66. In the revised HRTM, it is assumed that slow clearance in the conducting airways occurs only in the bb region, and following Falk *et al* (1997, 1999) as described above, particles are taken to be cleared from the bb region to the BB region at a rate of 0.2 $d^{-1}$ ($t_{½}$ ~3.5 d) (except for the small sequestered fraction, see below). The rate of rapid clearance from the BB region to the ET region is unchanged at 10 $d^{-1}$.

(114) The results of Falk *et al* (1997, 1999) suggest that only a fraction of particles deposited in bb is cleared slowly, perhaps 25% for the conditions of their experiments. If so, it is reasonable to suppose that it occurs mainly in the smaller bronchioles, as proposed by Camner *et al* (1997). However, given the remaining uncertainties, (and the lack of deposition fractions available for subdivisions of the bb region) it is assumed here for simplicity that it applies to all particles deposited in the bb region. It is also assumed that it applies to all particles cleared from the AI region to the bb region, unlike the Publication 66 implementation of the HRTM which assumed that slow clearance applied only to particles deposited directly in the BB and bb regions. These changes result in a simplification of the model: a single compartment BB′ replaces $BB_1$ and $BB_2$ and a single compartment bb′ replaces $bb_1$ and $bb_2$ (Figure 6). Associated changes to the dosimetric model are described in Section 3.2.4.

*Sequestration in the airway walls*

(115) The original HRTM assumes that the fraction of particles deposited in the BB and bb regions retained in the airway wall ($BB_{seq}$ and $bb_{seq}$) is 0.7% at all sizes, and that this material clears to lymph nodes at a rate of 0.01 $d^{-1}$. When the original HRTM was finalised the phenomenon had only been well quantified by Patrick and colleagues (*e.g.* Takahashi and Patrick, 1987), who followed retention of activity after deposition of radio-labelled particles onto the distal trachea of rats. Subsequently, Takahashi *et al* (1993) conducted similar experiments, instilling $^{133}$Ba-labelled $BaSO_4$ onto the distal trachea of rabbits, dogs and monkeys. The amounts retained 1 week after injection were 0.145%, 0.044% and 0.043% of the injected amount, respectively. These values are far lower than found in rats, suggesting inter-species differences. The value chosen above for retention of particles in the wall of the nasal epithelium, $ET_{seq}$, 0.2%, which was based on results for several different materials in several species, is within the range observed for the trachea. On that basis it is assumed here that values for both the retained fractions and clearance rates from BB and bb to $LN_{TH}$ are the same as those for $ET_{seq}$, *i.e.*, 0.2% and 0.001 $d^{-1}$. The revised model thus assumes less transfer to $LN_{TH}$ from BB and bb, but more transfer from the AI region (see below), maintaining consistency with the ratio of lung to $LN_{TH}$ contents observed in autopsy studies.

(116) The changes from the treatment of slow clearance from the bronchial tree in the original HRTM will in many cases decrease dose coefficients. The decreases will be considerable for Type M alpha-emitting radionuclides with half-lives of weeks or more, for which slow clearance gave the largest component of the effective dose

coefficient. Changes to parameter values relating to sequestration have little effect on effective dose coefficients because it only ever makes a small contribution to them.

***Particle transport: alveolar-interstitial (AI) region***

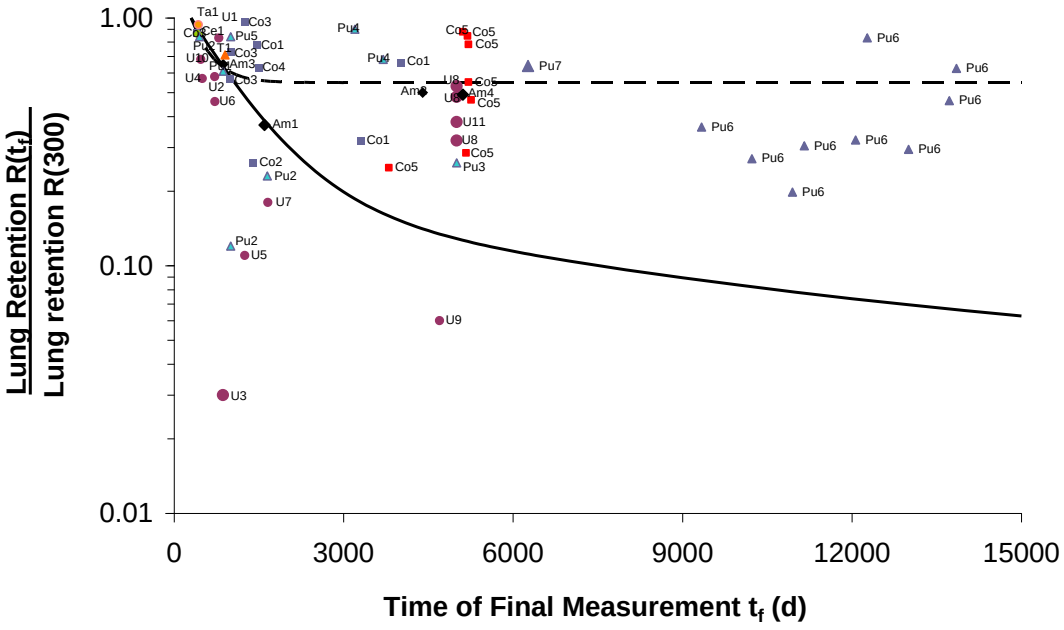
(117) In the original HRTM, the AI region was represented by three compartments: $AI_1$, $AI_2$ and $AI_3$, which mainly clear to the GI tract via the bronchial tree at rates of 0.02, 0.001 and 0.0001 $d^{-1}$, respectively (approximate half-times 35, 700 and 7000 d) (Figure 5). Human lung clearance had been quantified in experimental studies up to about a year after inhalation (ICRP, 1994a). It was considered that lung retention of insoluble particles over this time typically follows a two-component exponential function: about 30% with a half-time of about 30 d, and the rest with a half-time of several hundred days, giving about 50% retention of the initial alveolar deposit (IAD) at 300 d. This information was used to define the parameter values for $AI_1$.

(118) Measurements of activity in the chest after occupational exposure, and of activity in the lungs at autopsy, indicate that some material can be retained in the lungs for decades. Information on thoracic retention in humans following accidental inhalation, based on *in vivo* measurements of radionuclides, was reviewed in Publication 66 (ICRP, 1994a). Because retention up to 300 d after intake had been characterised in controlled experiments, only studies of accidental intakes in which retention was followed for at least 400 d were included. Since the aim was to obtain guidance on the likely fate of the approximately 50% IAD that remains at 300 d after intake, thoracic retention $R(t_f)$ at $t_f$, the time of the final measurement, was expressed as a fraction of $R(300)$, retention at 300 d. This also facilitated the inclusion of information in cases where the first measurement was made some time after intake, and avoided the effects of differences in early clearance due to factors such as aerosol size, breathing patterns, and soluble components. In Figure E.10 of Publication 66, thoracic retention $R(t_f)$, as a fraction of $R(300)$, was plotted against $t_f$: the information is shown here in Figure 7. Evidence for very long term retention of a significant fraction (> 10%) of the material remaining in the thorax at 300 d was seen for each of the elements (cobalt, uranium, plutonium, and americium) for which measurements extended to 10 y after acute intake of the oxide.

(119) The results were not used to set parameter values for $AI_2$ and $AI_3$ quantitatively because it was considered possible that the published *in vivo* studies represented unusually slow lung clearance. It was noted (ICRP, 1994a) that: "The fraction of the AI deposit that goes to $AI_3$ ($a_3$) is not easily quantified. Since only 50% IAD is retained at 300 d, $a_3$ is less than 0.5. Since there is measurable thoracic retention at 5000 d after intake in some subjects (Figure 7), $a_3$ is likely to be at least a few percent of the IAD. As a rounded value it is assumed that $a_3 = 0.1$, and, hence, by difference, that $a_2 = 0.6$." Figure 7 also shows retention of insoluble particles as predicted by the original HRTM: it fits quite well to results where the final measurement was made less than 2000 days after intake, but underestimates those with later measurements.

(120) In the revised model, account has been taken of additional human studies published since the original HRTM was adopted, which all show greater long term retention in the AI region than was assumed.

(121) A recent study by Davis *et al* (2007) provides better *in vivo* information on long-term lung retention than any available when Publication 66 was adopted. A group of workers had a simultaneous brief inhalation exposure to particles containing cobalt-60, and most (seven) had been followed for about 15 years. It is reasonable to assume that they are representative of nuclear industry workers. They all showed much slower clearance than the HRTM predicts, consistent with the few data on retention beyond 2000 days available at the time that the HRTM was published (Figure 7).




(122) Figure 7 Long term thoracic retention following accidental inhalation. References for data included in the figure are listed in Table 4. Separate symbols are used for each element. The solid and dashed curves show retention of insoluble particles as predicted by the original HRTM and the revised HRTM, respectively. Thoracic retention $R(t_f)$ at $t_f$, the time of the final measurement, is expressed as a fraction of $R(300)$, retention at 300 d.

2093 Table 4 Sources of data on thoracic retention used in Figure 7

| COBALT | URANIUM | PLUTONIUM |
|---|---|---|
| $Co_1$ Newton and Rundo (1971) | $U_1$ Ronen (1969) | $Pu_1$ Newton *et al* (1983) |
| $Co_2$ Gupton and Brown (1972) | $U_2$ Saxby *et al* (1964) | $Pu_2$ Ramsden (1976) |
| $Co_3$ Raghavendran *et al* (1978) | $U_3$ Rundo (1965) | $Pu_3$ Ramsden *et al* (1978); Ramsden (1984) |
| $Co_4$ Ramsden (1984) | $U_4$ Schultz (1966) | $Pu_4$ Bihl *et al* (1988a,b,c) |
| $Co_5$ Davis *et al* (2007) | $U_5$ Scott and West (1967) | $Pu_5$ Foster (1991) |
| | $U_6$ West and Scott (1966) | $Pu_6$ ORAUT (2007) |
| CERIUM | $U_7$ West and Scott (1969) | $Pu_7$ Carbaugh and La Bone (2003) |
| $Ce_1$ Tyler and Lister (1973) | $U_8$ West *et al* (1979) | |
| | $U_9$ Crawford-Brown and Wilson (1984) | AMERICIUM |
| TANTALUM | $U_{10}$ Kvasnicka (1987) | $Am_1$ Fry (1976) |
| $Ta_1$ Newton (1977) | $U_{11}$ Price (1989) | $Am_2$ Toohey and Essling (1980) |
| | | $Am_3$ Newton *et al* (1983) |
| $^{195}$Au-LABELLED TEFLON | | $Am_4$ Wernli and Eikenberg (2007) |
| $T_1$ Philipson *et al* (1996) | | |

2094
2095
2096 (123) A review of long-term lung retention data has therefore been conducted
2097 (Gregoratto *et al*, 2010). Three other major relevant studies were identified that were
2098 published since the HRTM was finalised. Their results, together with those on which
2099 the HRTM was based, were used to develop a new compartment model of particle
2100 transport from the AI region.
2101 (124) Philipson *et al* (1996) followed lung retention in 10 volunteers for about 3
2102 years after inhalation of $^{195}$Au-labelled Teflon particles. The duration of this study
2103 was about three times longer than for the experiments available when the HRTM was
2104 developed, and it seems likely that there was less leakage of the radioactive label from
2105 the test particles. Lung retention has been followed for over thirty years in workers
2106 who inhaled plutonium oxide during a fire at the Rocky Flats Plant (RFP) in October
2107 1965 (Mann and Kirchner, 1967; ORAUT 2007): another group who should be
2108 representative of nuclear industry workers (Gregoratto *et al*, 2010). Kuempel *et al*
2109 (2001) developed a model of particle retention in the AI region that is both
2110 physiologically more realistic and simpler than that in the original HRTM. Instead of
2111 the three AI compartments in the HRTM, it has an alveolar compartment which clears
2112 both to the bronchial tree and to an interstitial compartment which clears to lymph
2113 nodes. This model was applied to a group of U.S. coal miners with exposure histories
2114 from which particle mass deposition rates could be assessed, and autopsy
2115 measurements of dust concentration in lung (and also for lymph nodes in about 50%
2116 of cases). The model was considered to be the simplest consistent with the data and
2117 no evidence was found for impaired clearance at high lung loadings over the range

observed. The optimised parameter values derived by Kuempel *et al* (2001) were a rate $m_T$ = 0.001 $d^{-1}$ for clearance from the alveolar compartment to the bronchiolar region, a rate $m_I$ = 0.00047 $d^{-1}$ for clearance from the alveolar compartment to the interstitium, and a rate $m_{LN}$ = $10^{-5}$ $d^{-1}$ for clearance from the interstitium to lymph nodes. The main difference from the original HRTM AI model is that a significant fraction of the AI deposit is sequestered in the interstitium [$m_I/(m_I+m_T)$ = 0.32]. Kuempel *et al* (2001) noted that the HRTM underestimated lung retention in the miners by about a factor of four.

(125) Gregoratto *et al* (2010) showed that the Kuempel *et al* (2001) model provides an adequate representation of AI retention for the data in the other three studies outlined above. They developed a new model using the Kuempel *et al* model structure but fitted to both the experimental datasets on which the HRTM parameter values were based, and the more recent long-term studies (Figure 8). They obtained particle transport rates from the alveolar compartment of $m_T$ = 0.0017 $d^{-1}$ and $m_I$ = 0.0010 $d^{-1}$. These values are adopted here, but the value of $m_T$ is rounded to 0.002 $d^{-1}$, reflecting the underlying uncertainty in the model (Figure 6). These rates give a clearance half-time from the alveolar compartment of about 250 days ($m_I+m_T$ = 0.003 $d^{-1}$), and about 33% of the alveolar deposit of insoluble particles is sequestered in the interstitium. The greater AI retention than in the original HRTM is likely to result in lung doses per unit intake that are 50-100% higher for Type S long-lived alpha-emitters, but will have little, if any effect on more soluble forms.

(126) No clear difference was observed by Gregoratto *et al* (2010) between smokers and non-smokers in the long-term studies they analyzed. This contrasts with the greater retention in smokers than in non-smokers observed in those studies reviewed in Publication 66 in which the comparison could be made, although it is noted that the earlier studies were of relatively short duration. It also contrasts with the much greater retention in smokers than in non-smokers observed in studies of alveolar retention of iron oxide followed using magnetopneumography (see section on iron in Part 2), but for which absorption to blood rather than particle transport is considered to be the dominant clearance mechanism.The modifying functions proposed in Table 19 of Publication 66 relating to the effect of cigarette smoking on particle transport from the AI region are therefore not considered applicable to the revised model. Furthermore, it is not recommended that the other modifying factors in that table are applied in individual dose assessments.

(127) In the original HRTM, the transport rate from the AI region to the thoracic lymph nodes, $LN_{TH}$, was set at 2x$10^{-5}$ $d^{-1}$ to give the ratio of material concentration in lymph nodes and lungs equal to that estimated from autopsy data: for non–smokers [LN]/[L]≈20 after 10,000 days after inhalation of Pu (Kathren *et al*, 1993). Because of the smaller fraction of the deposit in the BB and bb regions cleared to $LN_{TH}$ via the airway walls ($BB_{seq}$ and $bb_{seq}$), and the longer AI retention in the model adopted here than in the Publication 66 model, the amount cleared to $LN_{TH}$ from the BB and bb is now negligible compared to that from the AI region. The ratio [LN]/[L]≈20 is obtained with a transport rate from the interstitium to $LN_{TH}$ of 3x$10^{-5}$ $d^{-1}$ (Gregoratto *et al*, 2010).

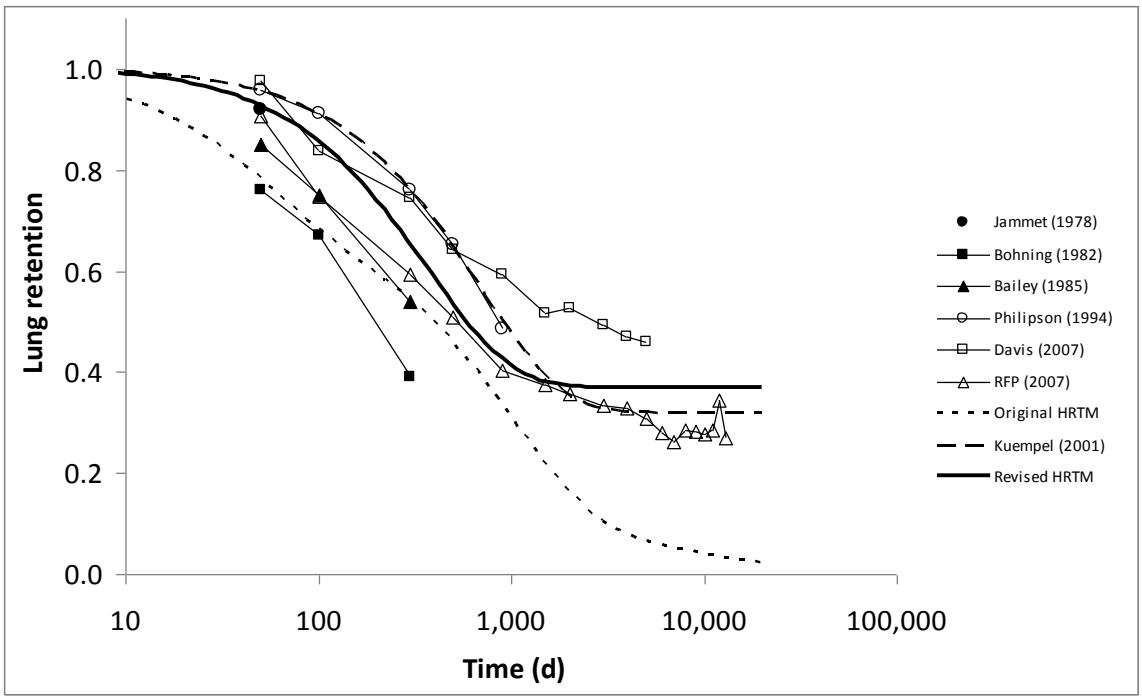



Figure 8. Measured lung retention data (Philipson *et al*, 1996; Davis *et al*, 2007; (ORAUT, 2007) and studies reported in Publication 66 Annex E, (ICRP, 1994a) are shown together with the model predictions by assuming initial deposition in the alveolar region only. Predictions of both the original HRTM and the Kuempel *et al* (2001) model with default parameter values are shown. The 'Revised HRTM' curve was obtained with optimised AI particle transport parameters $AI_{seq}$ = 0.37 and m = 0.0027 $d^{-1}$ (from Gregoratto *et al*, 2010).

### 3.2.3 Clearance: Absorption to blood

(128) Absorption to blood (body fluids) depends on the physical and chemical form of the deposited material. In both the original and revised HRTM it is assumed (by default) to occur at the same rate in all regions (including the lymph nodes) except $ET_1$ for which it is assumed that no absorption takes place. It is recognised that absorption is likely to be faster in the AI region where the air-blood barrier is thinner than in the conducting airways (ET, BB and bb regions), but there is insufficient information available to provide a general systematic basis for taking this into account, such as a scaling factor for different rates in different regions.

(129) In the HRTM absorption is treated as a two-stage process: dissociation of the particles into material that can be absorbed into body fluids (dissolution); and absorption into body fluids of soluble material and of material dissociated from particles (uptake). The clearance rates associated with both stages can be time-dependent.

(130) *Dissolution:* both the original and revised HRTM use the same simple compartment model to represent time-dependent dissolution. It is assumed that a fraction ($f_r$) dissolves relatively rapidly, at a rate $s_r$, and the remaining fraction ($1 - f_r$) dissolves more slowly, at a rate $s_s$ (Figure 9(a)).

(131) A limitation of this system is that it can only represent an overall dissolution rate that decreases with time. To overcome this, Publication 66 also describes a more flexible system, shown in Figure 9 (b). In this system, the material deposited in the respiratory tract is assigned to compartments labelled 'Particles in initial state' in which it dissolves at a constant rate $s_p$. Material is simultaneously transferred (at a constant rate $s_{pt}$) to a corresponding compartment labelled 'Particles in transformed state' in which it has a different dissolution rate, $s_t$. With this system, the initial dissolution rate is approximately $s_p$ and the final dissolution rate is approximately $s_t$. Thus with a suitable choice of parameters, including $s_t > s_p$, an increasing dissolution rate can be represented. The ratio of $s_p$ to $s_{pt}$ approximates to the fraction that dissolves rapidly. It may be noted that any time-dependent dissolution behaviour that can be represented using the model shown in Figure 9(a) can also be represented by the model shown in Figure 9(b) with a suitable choice of parameter values. However, the reverse is not true, as noted above.

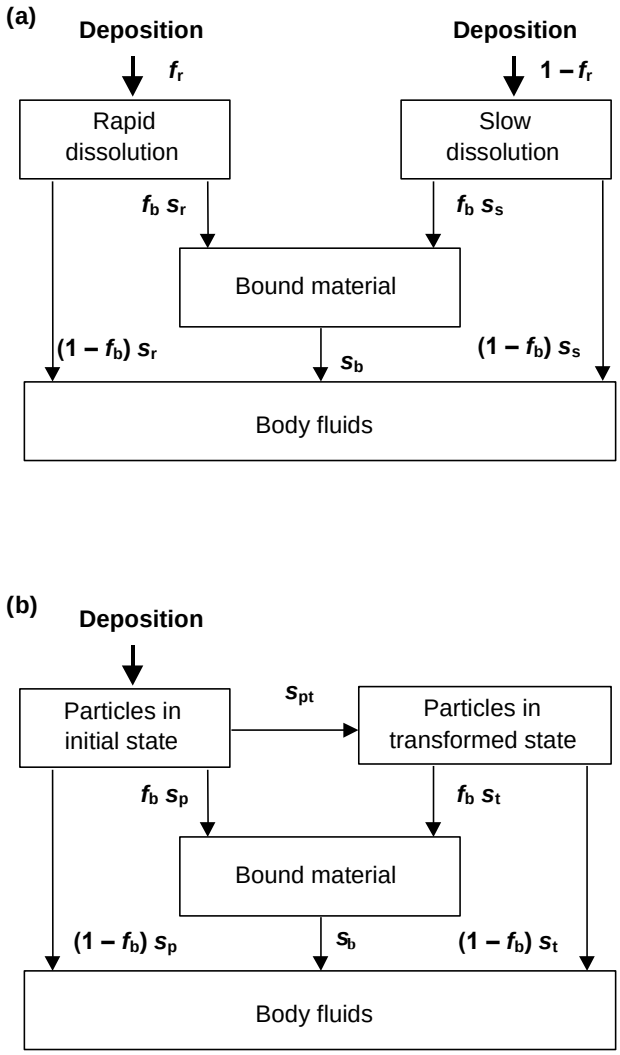


Figure 9. Alternative compartment models representing time-dependent absorption to body fluids (dissolution and uptake). In the model shown in Figure 9(a) a fraction $f_r$ of the deposit is initially assigned to the compartment labelled 'Rapid dissolution', and the rest of the deposit $(1 - f_r)$ is initially assigned to the compartment labelled 'Slow dissolution'. In the model shown in Figure 9(b) all the deposit is initially assigned to the compartment labelled 'Particles in initial state', and material in the compartment labelled 'Particles in transformed state' is subject to particle transport at the same rate as material in the compartment labelled 'Particles in initial state'. Material in the compartment labelled 'Bound material' is not subject to particle transport and is cleared only by uptake into body fluids. For definition of symbols, see text.

(132) If the dissolution rate decreases with time, as is usually the case, either system could be used, and would give the same results, with the following values:

$$s_p = s_s + f_r\,(s_r - s_s)$$
$$s_{pt} = (1 - f_r)\,(s_r - s_s)$$
$$s_t = s_s$$

(133) The system shown in Figure 9(b) was applied by default in earlier Publications (ICRP, 1994b, 1995c, 1997b). The additional flexibility it provides is, however, rarely required in practice, and it is more complex (and less intuitive) to present. The simpler approach is therefore adopted now as the default, with the more flexible approach retained as an alternative. Examples of materials that show dissolution rates that increase with time, which have been represented by 'particles in initial state' and 'particles in transformed state', including uranium aluminide, are given in the element sections in subsequent reports of this series.

(134) *Uptake:* uptake to body fluids of dissolved material is usually assumed to be instantaneous. For some elements, however, part of the dissolved material is absorbed rapidly into body fluids, but a significant fraction is absorbed more slowly because of binding to respiratory tract components. To represent time-dependent uptake, it is assumed that a fraction ($f_b$) of the dissolved material is retained in the 'bound' state, from which it goes into body fluids at a rate $s_b$, while the remaining fraction $(1 - f_b)$ goes to body fluids instantaneously (Figure 9). In the model, material in the 'bound' state is <u>not</u> cleared by particle transport processes, but only by uptake to body fluids. Thus, only one 'bound' compartment is required for each region.

(135) The system shown in Figure 9 applies to each of the compartments in the particle transport model shown in Figure 6. It is assumed that no absorption takes place from $ET_1$, but if the model in Figure 9 (a) is used the $ET_1$ deposition still has to be partitioned between fast and slow compartments because material is cleared from $ET_1$ to $ET_2$, from which absorption does take place.

(136) For all elements, default values of parameters are recommended, according to whether the absorption is considered to be fast (Type F), moderate (M) or slow (S). The original reference values, given in Publication 66 (ICRP, 1994a) and reproduced in Table 5, were specified in terms of the parameters initial dissolution rate, $s_p$; transformation rate, $s_{pt}$; and final dissolution rate, $s_t$ (Figure 9 (b)), rather than $f_r$, $s_r$ and $s_s$ (Figure 9 (a)), for which approximate values were given. For gases or vapours, instantaneous uptake to body fluids has also been recommended, as in Publication 68 (ICRP, 1994b), and defined as Type V (very fast), in Publication 71 (ICRP, 1995).

Table 5. Original HRTM default absorption parameter values for Type F, M, and S materials (based on Publication 66, ICRP 1994a, Table 18)[a]

| Type | | F(fast) | M (moderate) | S (slow) |
|---|---|---|---|---|
| Model parameters: | | | | |
| Initial dissolution rate ($d^{-1}$) | $s_p$ | 100 | 10 | 0.1 |
| Transformation rate ($d^{-1}$) | $s_{pt}$ | 0 | 90 | 100 |
| Final dissolution rate ($d^{-1}$) | $s_t$ | - | 0.005 | 0.0001 |
| Fraction dissolved rapidly | $f_r$ | 1 | 0.1 | 0.001 |
| Approximate dissolution rates: | | | | |
| Rapid ($d^{-1}$) | $s_r$ | 100 | 100 | 100 |
| Slow ($d^{-1}$) | $s_s$ | - | 0.005 | 0.0001 |
| Fraction to bound state | $f_b$ | 0 | 0 | 0 |
| Uptake rate from bound state ($d^{-1}$) | $s_b$ | - | - | - |

[a]The model values $s_p$, $s_{pt}$ and $s_t$ in this table are the original HRTM *reference values i.e.*, the recommended default values for use in the model. No 'bound' state was assumed for default Types.

(137) The original default values for Types F, M and S (ICRP, 1994a,b, Table 5) were not based on reviews of experimental data but on comparison with particle transport rates. The value of 100 $d^{-1}$ for the rapid dissolution rate, $s_r$, was chosen to equal the particle clearance rate from the nose ($ET_2$) to the throat. Hence for Type F about half the material deposited in $ET_2$ is absorbed into blood and the rest swallowed. The slow dissolution rate for Type S of $10^{-4}$ $d^{-1}$ was chosen to equal the slowest particle transport rate from the AI region to the GI tract, to ensure that there was some long term lung retention. Type M values were chosen to be intermediate between the two. It has, however, been recognised that the parameter values for default Type F and Type S represent extremes of 'fast' and 'slow' dissolution rather than being representative of these classes of materials.

*Review of absorption characteristics of inhaled materials*

(138) In developing the subsequent parts of this document, detailed reviews were conducted of the absorption characteristics of inhaled materials relevant to radiological protection. They are summarised in the inhalation sections of each element.

(139) Where information was available, specific parameter values were derived from experimental data from both *in vivo* and *in vitro* studies. As described below, these provided a database to give guidance on selecting values that are representative of materials that are generally considered to clear at 'fast', 'moderate' or 'slow' rates. Values selected on that basis for default Type F, M and S have been adopted in the revised HRTM used in this series of documents.

(140) Material-specific rates of absorption have been adopted in the element sections (and dose coefficients and bioassay functions provided for them on the accompanying CD-ROM) for a limited number of selected materials, *i.e.*, those for which:

- There are *in vivo* data from which specific parameter values can be derived;

- Results from different studies are consistent;
- It was considered that occupational exposure to the material is possible;
- The specific parameter values are sufficiently different from default Type F, M or S parameter values to justify providing specific dose coefficients and bioassay functions.

(141) Other materials were assigned to default Types using current information. Publication 66 did not give criteria for assigning materials to absorption Types on the basis of experimental results. Criteria were developed in Publication 71 (ICRP, 1995c) and their application was discussed further in Guidance Document 3 (ICRP, 2002b). Type M is assumed for all particulate forms of most elements in the absence of information. A material is assigned to Type F if the amount absorbed into body fluids by 30 d after an acute intake is *greater* than the amount that would be absorbed over the same period from a hypothetical material with a constant rate of absorption of 0.069 $d^{-1}$ (corresponding to a half time of 10 d) under identical conditions. Similarly, a material is assigned to Type S if the amount absorbed into body fluids by 180 d after an acute intake is *less* than the amount that would be absorbed over the same period from a hypothetical material with a constant rate of absorption to body fluids of 0.001 $d^{-1}$ (corresponding to a half-time of about 700 d) under identical conditions.

(142) Particulate forms of each element were assigned to the HRTM default absorption Types using these criteria. However, strict application of the criterion for assigning materials to Type S requires experiments of at least 180 days duration, and since this would exclude much useful information, extrapolation has been used in some cases, as indicated in the text. For studies where it was possible to apply the criteria, a statement is made to the effect that results “are consistent with” (or “give”) assignment to Type F (M or S). For studies where the results point towards a particular Type, but there was insufficient information to apply the criteria, a statement is made to the effect that the results "indicate" or "suggest" Type F (M or S) behaviour. For some elements, for which there is little or no experimental data on absorption from the respiratory tract, some materials have been assigned to default Types based on chemical analogy.

(143) For soluble (Type F) forms of each element, estimates are made of the overall rate of absorption from the respiratory tract to blood (where information is available). In general this might result from a combination of processes including: (i) dissolution of the deposited material (if not inhaled as droplets and so already in solution); (ii) transfer through the lining fluid to the epithelium, especially in the conducting airways; (iii) transfer across the epithelium. Strictly, in terms of the model structure, the first two of these would be described as ‘dissolution’ and be represented by the rapid dissolution rate, $s_r$, because the material is subject to particle transport, whereas transfer across the epithelium, unless extremely rapid, should be represented by a bound fraction. In practice it would often be difficult to assess how much of the overall rate should be assigned to each process, and for simplicity $s_r$ is used to represent the overall absorption. However, it is assumed that $s_r$ is a characteristic of the element, and this would be expected for transfers through the lining fluid and epithelium. Wide variation in values of $s_r$ was found between elements, ranging from about 1 $d^{-1}$ (*e.g.* yttrium) to 100 $d^{-1}$ (*e.g.* caesium). Some justification for this

approach comes from the fact that the value of $s_r$ tends to have more effect on the overall biokinetics of an inhaled material deposited in the conducting airways (where the lining fluid is relatively thick) than on material deposited in the alveolar region, because it competes with particle transport rates of similar magnitude (10 $d^{-1}$ from BB' to $ET_2'$ and 100 $d^{-1}$ from $ET_2'$ to oesophagus). Because of the wide variation between elements in the estimated value of $s_r$, element-specific values are adopted in this series of documents for those elements for which an estimate of the value could be made.

(144) For soluble (Type F) forms of some elements, however, part of the dissolved material is absorbed rapidly into body fluids, but a significant fraction is absorbed more slowly. To represent this time-dependent uptake, it is assumed that a fraction ($f_b$) of the dissolved material is retained in the 'bound' state, from which it goes into body fluids at a rate $s_b$, while the remaining fraction (1 – $f_b$) goes to body fluids instantaneously (Figure 9). Evidence for retention in the bound state, rather than by transformation into particulate material may be in one or more forms: e.g. systemic uptake rather than faecal clearance of the retained material, or autoradiography showing diffuse rather than focal retention of activity. In Part 2, bound state parameter values are used for cobalt, ruthenium and lead.

*Revision to default absorption parameter values*

(145) As noted above, the specific parameter values derived from experimental data (from both *in vivo* and *in vitro* studies) provided a database to give guidance on selecting values that are representative of materials that are generally considered to clear at 'fast', 'moderate' or 'slow' rates.

(146) When about 100 sets of parameter values were available (*i.e.* when most of the reviews for Part 2 elements were completed) the results were collated and analyzed. It is emphasised that this was not a representative survey from which central values could be derived by some objective statistical means. Rather it provided a basis for informing judgements as described below.

(147) Parameter values given in the text of the current draft element sections were sorted into Types F, M and S according to the Publication 71 criteria given above, and tabulated. Some selection was made. A few values noted to be particularly uncertain were excluded. Where there was more than one set of results for a material (or very similar materials) they were merged, and central values taken, to avoid giving too much weight to a few compounds. Note that for some sets of parameter values, because of limitations in data fitting, the value of $s_r$ was fixed and only the values of $f_r$ and $s_s$ were assessed. In such cases the assumed value of $s_r$ was not included in the derivation of central values.

(148) Medians, geometric means, and geometric standard deviations (GSD) of the assessed values of $f_r$, $s_r$ and $s_s$ are given in Table 6. Except for the value of $f_r$ for Type F materials, GSDs are very large (4 – 14) reflecting the wide ranges of estimated values, and hence indicating large uncertainties in the central values.

Table 6 Central values of dissolution parameters for Type F, M, and S material from a review of experimental data[a]

| Type | | F(fast) | M (moderate) | S (slow) |
|---|---|---|---|---|
| Fraction dissolved rapidly | fr | 0.95 (0.84) [1.4] | 0.20 (0.18) [4] | 0.007 (0.003) [9] |
| Dissolution rates: | | | | |
| Rapid (d-1) | sr | 12 (9) [8] | 1.7 (1.5) [9] | 2.0 (3.8) [14] |
| Slow (d-1) | ss | 0.02 (0.02) [8] | 0.003 (0.003) [4] | 0.00018 (0.00008) [9] |

[a]Median value, with geometric mean in parentheses, and geometric standard deviation in brackets.

(149) Updated default values, given in Table 7, were based mainly on the following considerations, but also take account of the large uncertainties in the central values and the need for simple rounded numbers.

*Rapid fraction $f_r$*:

(150) For Type F, the median value (0.95) is close to the current default value of 1.0. For simplicity in implementation it is preferable not to change to two-phase dissolution. The default value remains 1.0.

For Type M, the median value is higher (0.20) than the current default (0.1). The updated default value is taken to be 0.2.

For Type S, the median value is higher (0.007) than the current default (0.001). The updated default value is rounded to 0.01.

*Rapid dissolution rate, $s_r$*:

(151) *Type F:* the median of values of $s_r$ estimated from experimental data for materials that would be assigned to Type F, is 12 $d^{-1}$ (Table 5), (much lower than the original HRTM default value of 100 $d^{-1}$). However, this outcome is heavily influenced by results for a few elements: about half of the results are from only four elements. To include information from a wider range of elements in choosing the default value, consideration was also given to the element-specific values of $s_r$ for soluble (Type F) forms of the element, which were assessed where suitable experimental information was available (see above). There are element-specific values for several elements for which no material-specific values were assessed. Hence the distribution of element-specific values covers a wider range of elements, and in it each element makes the same contribution (one entry): its median value is 50 $d^{-1}$. Taking both medians into account, the updated default value of $s_r$ for Type F is taken to be 30 $d^{-1}$.

(152) *Types M and Type S:* The medians of estimated values of $s_r$ for materials that would be assigned to Types M and S, are 1.7 $d^{-1}$, and 2 $d^{-1}$, respectively (Table 5), (very much lower than the original HRTM default of 100 $d^{-1}$). As for Type F, the distributions are heavily influenced by results for a few elements. For Type F, consideration of element-specific values of $s_r$ involved a wider range of elements, and led to the choice of a somewhat higher value than the material-specific values. However, whereas for Type F materials the rapid dissolution rate, $s_r$, represents overall absorption, and is assumed to be element-specific, for Type M and S materials $s_r$ is more likely to be determined by dissolution of the particle matrix, and so less

characteristic of the element. Thus element-specific values of $s_r$, were not assessed for Type M and S materials. Taking account of these factors and the overall large variation in estimated values of $s_r$, the updated default values for Types M and S were taken to be the same and rounded up to 3 $d^{-1}$. It is assumed here that the default $s_r$ value of 3 $d^{-1}$ for Type M and S materials applies to all elements, unless the Type F element-specific value is itself less than 3 $d^{-1}$, in which case the Type F element-specific value is also applied to Types M and S. For example, for silver, default values are used for all three Types, as in Table 7; for barium, the element-specific value of $s_r$ is 20 $d^{-1}$ for Type F, but the default value of 3 $d^{-1}$ is used for Types M and S; for yttrium, the element-specific value of $s_r$ is 1 $d^{-1}$ for Type F, and so 1 $d^{-1}$ is also used for Types M and S.

*Slow dissolution rate, $s_s$*:

(153) For Types M and S, median values are 0.003 $d^{-1}$ and 0.00018 $d^{-1}$, similar to the current default values of 0.005 $d^{-1}$ and 0.0001 $d^{-1}$. The default values remain 0.005 $d^{-1}$ and 0.0001 $d^{-1}$, respectively.

(154) Thus the data currently available suggest larger typical rapid fractions for Types M and S materials, but with lower rapid dissolution rates than original default values for all three Types. This has the effect of reducing rapid absorption in the extrathoracic airways and increasing it in the lungs.

Table 7. Updated default absorption parameter values for Type F, M, and S materials[a,b]

| Type | | F(fast) | M (moderate) | S (slow) |
|---|---|---|---|---|
| Fraction dissolved rapidly | $f_r$ | 1 | 0.2 | 0.01 |
| Dissolution rates: | | | | |
| Rapid ($d^{-1}$) | $s_r$ | 30[c] | 3[d] | 3[d] |
| Slow ($d^{-1}$) | $s_s$ | - | 0.005 | 0.0001 |

[a]Reference values (see footnote to Table 3).
[b]The bound state is also used for default Types of some elements.
[c]Element-specific rapid dissolution rates are adopted for Type F forms of many elements
[d]The element-specific value for Type F is used if it is less than 3 $d^{-1}$

(155) The default absorption rates, expressed as *approximate* half-times, and the corresponding amounts of material deposited in each region *that reach body fluids* (from the respiratory tract) can be summarised as follows:

*Type V*: 100% absorbed instantaneously. Regional deposition does not need to be assessed for such materials, because in dose calculations they can be treated as if they were injected directly into body fluids.

*Type F*: 100% absorbed with a half-time of ~30 minutes. There is rapid absorption of almost all material deposited in bb and AI, ~80% of material deposited in BB, and ~25% of material deposited in $ET_2$. The

other material deposited in BB and $ET_2$ is cleared to the alimentary tract by particle transport.

*Type M:* 20% absorbed with a half-time of ~6 hours and 80% with a half-time of ~140 d. There is rapid absorption of ~20%, 5% and 0.5% of material deposited in bb, BB and $ET_2$, respectively. About 80% of the deposit in AI eventually reaches body fluids.

*Type S:* 1% absorbed with a half-time of ~6 hours and 99% with a half-time of ~7000 d. There is rapid absorption of ~1%, 0.25% and 0.03% of material deposited in bb, BB and $ET_2$, respectively. About 30% of the deposit in AI eventually reaches body fluids.

(156) For absorption Types F, M, and S, some the material deposited in $ET_1$ is removed by extrinsic means. Most of the material deposited in the respiratory tract that is not absorbed is cleared to the alimentary tract by particle transport. The small amounts transferred to lymph nodes continue to be absorbed into body fluids at the same rate as in the respiratory tract.

*Decay products formed in the respiratory tract*

(157) Note that the following applies specifically to decay products formed in the respiratory tract after inhalation of the parent radionuclide. Decay products formed before inhalation and inhaled with the parent are generally treated as separate intakes, and so each decay product is assumed to adopt the biokinetics appropriate to the element of which it is an isotope. Many issues relating to the behaviour of decay products in the respiratory tract arise in connection with the natural decay series, which are therefore shown in Figures 10 (uranium-238 series), 11 (uranium-235 series) and 12 (thorium-232 series).

(158) Publication 66 (ICRP, 1994a, Paragraph 272) noted that it would be expected that:

- the rate at which a particle dissociates is determined by the particle matrix and therefore the dissolution parameter values of the inhaled material would be applied to decay products formed within particles in the respiratory tract ('shared kinetics');
- decay products formed as noble gases, including radon, would be exceptions because they would diffuse from the particles;
- the behaviour of dissociated material would depend on its elemental form, and so, for example, bound fraction parameter values for a decay product would not be those of the parent ('independent kinetics').

(159) These points are considered in turn below. However, it should be noted that in previous applications of the HRTM (*e.g.* Publications 68, 71, 72 and 78), with the exception of noble gases, the absorption parameters of the parent were applied to all members of the decay chain formed in the respiratory tract (shared kinetics). After consideration (see below) the same approach is taken in this series of documents.

*Retention in the particle matrix*

(160) Generally the assumption applied to the slowly-dissolving fractions of Type M and Type S materials is that the dissolution of a decay product is determined by that of the particle matrix in which it is formed. Thus its dissolution parameter values should be those of the inhaled material.

*Emanation of radon and alpha recoil*

(161) In applying the HRTM, general exceptions have been made for noble gases formed as decay products (ICRP, 1994b, 1995c). Radioisotopes of xenon formed from the decay of iodine are assumed to escape from the body without decay, as in Publication 30 Part 1 (ICRP, 1979). This includes xenon formed in the respiratory tract. For calculation purposes it is assumed that radon formed as a decay product within the respiratory tract escapes from the body at a rate of 100 $d^{-1}$, in addition to other routes of removal (ICRP, 1995c). This rate was set as a convenient, arbitrary, rapid rate. The underlying assumption is that loss of radon (for example) is a continuous process such as diffusion. The three radon isotopes in the natural decay series: $^{222}$Rn (radon), $^{220}$Rn (thoron), and $^{219}$Rn (actinon) have half-lives of about 3.8 days, 56 seconds and 4 seconds, and therefore decay rates of about 0.18, 1100 and 15,000 $d^{-1}$, respectively. Hence the assumption of a rate of loss of 100 $d^{-1}$ implies that nearly all $^{222}$Rn escapes from the particles before it decays, about 10% of $^{220}$Rn escapes, and nearly all $^{219}$Rn decays within the particles. As described in the thorium inhalation section, studies which have compared thorium lung contents with exhaled thoron seem broadly consistent with the assumption that about 10% of thoron formed within particles in the lungs escapes, but measurements of emanation of radon ($^{222}$Rn) from uranium ore dust give values much lower than 100%.

(162) Griffith *et al* (1980) developed a model to describe the retention of $^{232}$U and its decay products (which include $^{228}$Th) in the lungs following inhalation of $ThO_2$ or $UO_2$ particles. In addition to chemical dissolution, they considered emanation of $^{220}$Rn from particles by diffusion, and emanation of decay products, including $^{220}$Rn, as a result of the recoil of nuclei formed in alpha-particle decay. They presented equations to calculate fractional losses by diffusion and recoil as functions of particle size (but only for spherical particles). They calculated recoil ranges of about 0.05 µm for the decay products, (assuming a particle density of 10 g $cm^{-3}$) and fractional losses by recoil emanation in the range 0.3 – 0.1, for aerosols with AMAD in the range 1 – 10 µm. The calculated loss of $^{220}$Rn from particles by diffusion emanation was difficult to predict, ranging from 0.03 to 0.7 depending on the assumed diffusion coefficient ($10^{-15}$ - $10^{-11}$ $cm^2$ $s^{-1}$).

(163) Coombs and Cuddihy (1983) measured the fraction of $^{228}$Th escaping by recoil, and the fraction of $^{220}$Rn escaping by diffusion, from size-fractionated samples of $ThO_2$ and uranium oxide (mixture of $UO_{2.2}$ and $U_3O_8$) containing 1% $^{232}$U. The fraction of $^{228}$Th escaping increased from ~0.07 for particles with AMAD 2.5 µm (count median diameter, CMD, ~1 µm) to ~0.3 for particles with AMAD 0.65 µm (CMD ~0.1 µm). This was in reasonable agreement with the model of Griffith *et al* (1980). Calculated recoil range was expressed in terms of recoil range multiplied by density, with values of ~20 µg $cm^{-2}$. The fraction of $^{220}$Rn escaping by diffusion increased from ~0.07 for particles with AMAD 2.5 µm, to ~0.35 for particles with AMAD 0.65 µm, and gave a diffusion coefficient of ~$3x10^{-14}$ $cm^2$ $s^{-1}$. This was

similar to the fraction of $^{228}$Th escaping by recoil, and therefore presumably similar to the fraction of $^{220}$Rn escaping by recoil, since the recoil ranges of $^{220}$Rn and $^{228}$Th are similar (Griffith *et al*, 1980).

(164) Johnson and Peterman (1984) developed a model to describe the emanation of $^{220}$Rn from $ThO_2$ particles by alpha-particle recoil, and its exhalation from the lungs. They calculated that the fraction of $^{220}$Rn atoms produced that escaped from particles (density 10 g cm$^{-3}$) by recoil decreased from ~1.0 at 1 nm to ~0.5 at 10 nm and ~0.1 at 0.5 µm diameter. The average fraction for an aerosol of AMAD 1 µm was calculated to be 0.2, which seems to be broadly consistent with the results derived by Griffith *et al* (1980).

(165) Thus it seems that recoil is a mechanism that is at least as important as diffusion for emanation of radon from particles. It seems possible that it is the dominant mechanism, in which case for aerosols of AMAD about 1 µm there would be a release to lung air of ~10% of $^{222}$Rn, $^{220}$Rn or $^{219}$Rn formed in particles in the lungs. Furthermore, alpha-particle recoil applies not only to radon formed as a decay product, but also to other decay products formed by alpha emission. In the case of decay chains, this will result in successively lower activities of members of the chain compared to the parent retained in relatively insoluble particles. There is some experimental evidence confirming this (see thorium inhalation section). However, it was considered impractical to implement loss of decay products by alpha recoil in the calculation of dose coefficients and bioassay functions in this series of documents. Assessment of the fractional loss for representative workplace aerosols would be complex, because it depends on the alpha decay energy, the size distribution of deposited particles, and their shape and density: simplifying assumptions would be needed for practical implementation. Investigations conducted at by the Task Group into the effect of recoil on doses for inhaled $^{232}$U and its decay products following inhalation in relatively insoluble particles found, as expected, that doses to the respiratory tract decreased and doses to tissues resulting from systemic uptake increased. However, there was little impact on effective dose in this example. The computational effort involved in identifying radionuclides formed by alpha decay, and partitioning the decay product atoms between a fraction remaining in the particle and a fraction escaping to lung fluids would be considerable, and was considered disproportionate to the benefit gained on a routine basis as in these documents. For calculation purposes the assumption that radon formed as a decay product within the respiratory tract escapes from the body at a rate of 100 d$^{-1}$ is retained in this series of documents. Nevertheless, this phenomenon should be borne in mind, especially when using decay products to monitor intakes and doses of the parent radionuclide.

*Soluble (dissociated) material*

(166) The behaviour of soluble or dissolved material (specifically the rate of uptake to blood) of decay products formed in the respiratory tract can be expected to depend on the element of which the decay product formed is an isotope. As discussed above, for soluble (Type F) materials the rapid dissolution rate, $s_r$, represents the overall absorption from the respiratory tract to blood and is element-specific for many elements. Hence, when a Type F material is deposited in the respiratory tract, the value of $s_r$ for a decay product formed would be expected to be that of the element formed ('independent kinetics'), rather than following that of the parent ('shared

kinetics'). Similarly, element-specific bound state parameter values would be expected to apply to decay products formed in the respiratory tract. However, analysis carried out by the Task Group showed that application of independent kinetics rather than shared kinetics within the respiratory tract, to decay products of Type F radionuclides, would make little difference to respiratory tract tissue dose coefficients (up to a factor of two, but in most cases much less), and less difference to effective dose coefficients. For Type F materials absorption to blood is rapid and doses from deposition in systemic tissues will often make greater contributions to effective dose than doses to respiratory tract tissues. The additional complexity involved in application of independent kinetics was therefore considered to be unjustified. Furthermore, in many practical exposure situations, an intake of a parent nuclide will often be accompanied by simultaneous intakes of its decay products. Their activities (which, being treated as separate intakes will be given absorption kinetics appropriate to the element) will often be considerably greater than the activities of decay products formed within the respiratory tract, because very little decay of the parent takes place before a Type F material is absorbed into blood.

(167) Thus, in this series of documents, radioactive decay products formed within the respiratory tract (with the exception of noble gases) are assumed by default to follow the absorption behaviour of the parent nuclide, and are given the same dissolution and uptake parameter values as the parent (shared kinetics). Following absorption to blood, they are assumed to behave according to the systemic model applied to the element as a daughter of the parent radionuclide.

(168) Nevertheless, where experimental results are available which allow direct comparison between the absorption behaviour of a parent radionuclide, and that of its radioactive decay products, they are summarised in the inhalation section of the parent element (*e.g.* uranium, thorium). Such information may be of use to those carrying out individual monitoring, especially if intakes of a parent are being assessed by means of measurements on one or more of its decay products. The behaviour of thorium and its decay products can be of particular importance in this context, because there is generally significant long-term retention of thorium in the lungs following its deposition in soluble form, whereas soluble forms of important decay products, notably radium and lead, are absorbed relatively readily.

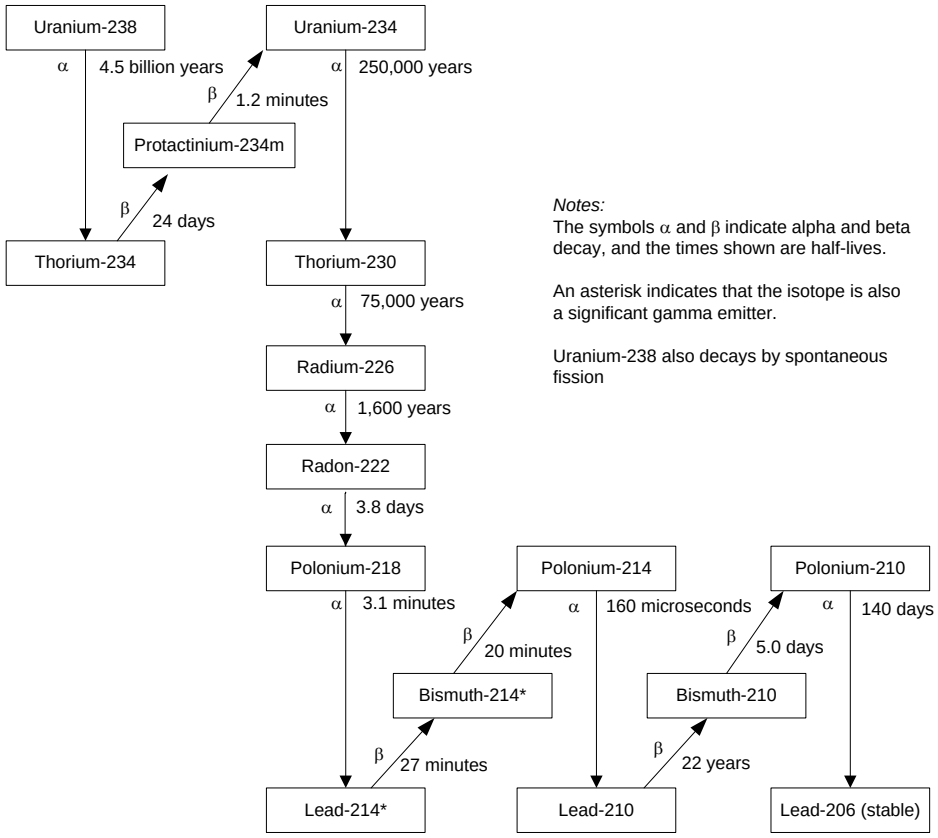



Figure 10 Natural decay series: Uranium-238

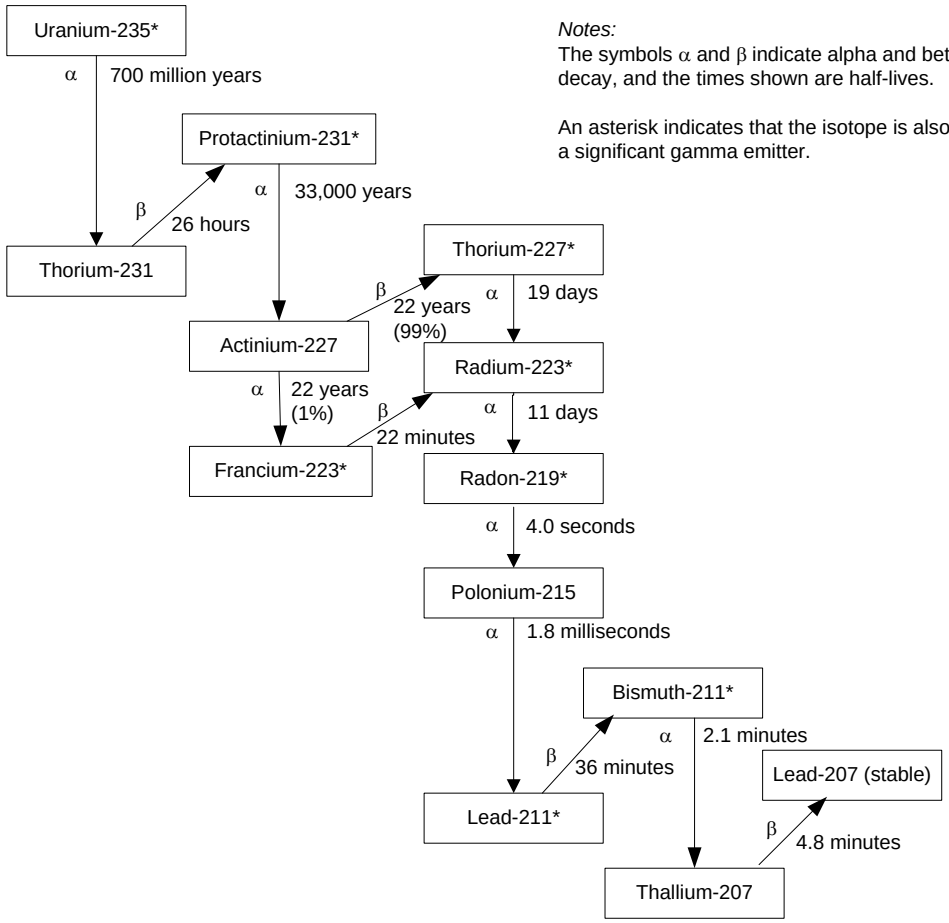



Figure 11 Natural decay series: Uranium-235

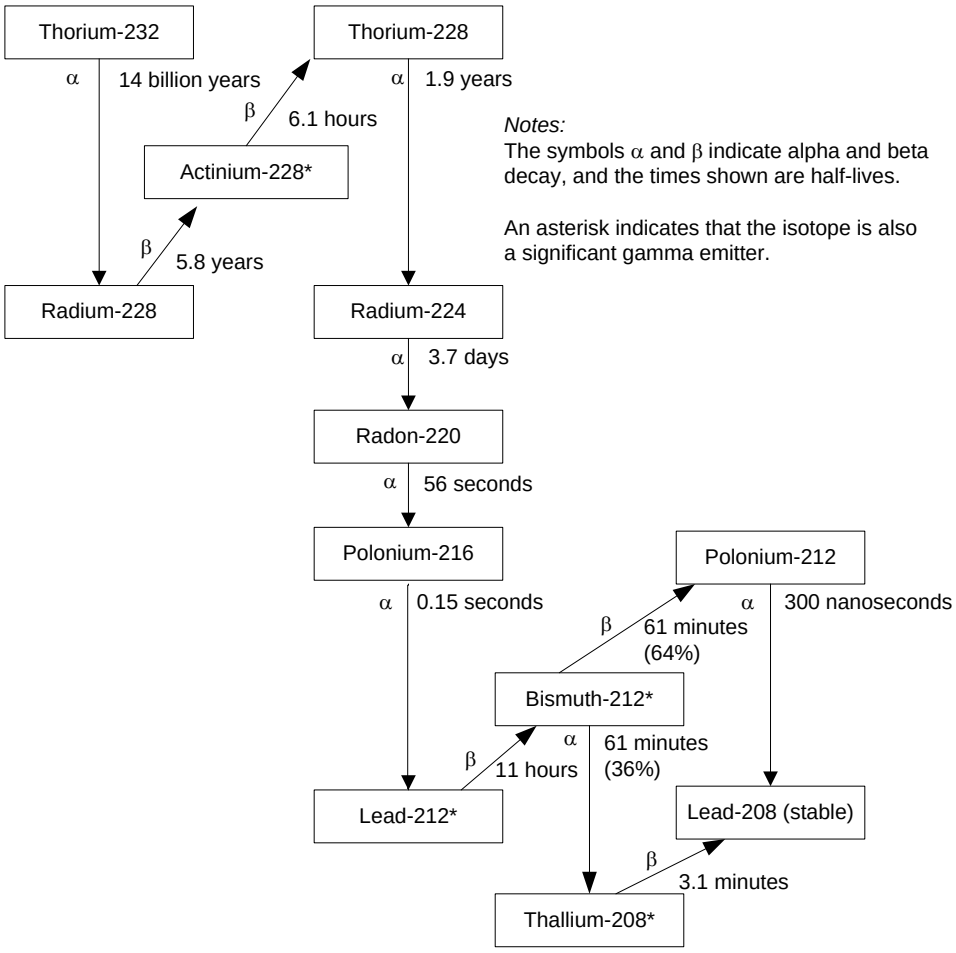



Figure 12 Natural decay series: Thorium-232

### 3.2.4 Respiratory Tract Dosimetry

(169) The HRTM dosimetric model is described in Publication 66 (ICRP, 1994a) Chapter 8. For dosimetric purposes, the respiratory tract is treated as two tissues: the thoracic airways (TH) and the extrathoracic airways (ET). These are sub-divided into regions, primarily based on considerations of differences in sensitivity to radiation. The thoracic regions are bronchial, BB; bronchiolar, bb; alveolar-interstitial, AI; and the thoracic lymph nodes, $LN_{TH}$. The extrathoracic regions are the anterior nose, $ET_1$; the posterior nasal passages, pharynx and larynx, $ET_2$; and the extrathoracic lymph nodes $LN_{ET}$ (Figure 3).

(170) The dose to each respiratory tract region is calculated as the average dose to the target tissue which contains the target cells at risk. In the alveolar region (AI) and lymph nodes ($LN_{TH}$ and $LN_{ET}$), the cells at risk are thought to be distributed throughout the region, and the average dose to the whole lung and the lymph nodes, respectively, is calculated. For the regions making up the conducting airways ($ET_1$, $ET_2$, BB and bb), the target cells are considered to lie in a layer of tissue at a certain range of depths from the airway surface and the average dose to this layer is

calculated. The target cells identified in $ET_1$, $ET_2$, BB and bb, and the masses of tissue containing target cells in each region for dose calculations, are given in Table 8.

(171) In each of these regions there are also several possible source regions. In the bronchiolar region (bb), particles retained in the airway wall ($bb_{seq}$) are taken to be in a macrophage layer at a depth of 20-25 μm (*i.e.* below the target cells); activity 'bound' to the epithelium is uniformly distributed in it; and account is also taken of irradiation from activity present in the AI region. In the original HRTM, activity in the fast phase of clearance (compartment $bb_1$, Figure 5) was taken to be in the mucus layer above the cilia; activity in the slow phase of clearance ($bb_2$) was taken to be in the mucus between the cilia. In the revised HRTM, there is only one phase of clearance.

(172) For each source/target combination, Publication 66 provides absorbed fractions for non-penetrating radiations: α, β and electrons; in each case as a function of energy. Since these absorbed fractions are not represented in the voxel phantoms because of inadequate spatial resolution, the values were derived in Publication 66 using a single cylindrical geometry to represent each region of the conducting airways ($ET_1$, $ET_2$, BB, bb): the representative bronchus for BB being 5 mm diameter and the representative bronchiole for bb being 1 mm diameter. The absorbed fractions for the BB and bb source regions were derived as the thickness-weighted sum of the slow and fast clearing source regions, as tabulated in Publication 66.

(173) To take account of differences in sensitivity between tissues, the equivalent dose, $H_i$, to each region, $i$, is multiplied by an apportionment factor, $A_i$, representing the region's estimated sensitivity relative to that of the whole organ. The recommended values of $A_i$ are also given in Table 8. In Publication 103 (ICRP, 2007) the extrathoracic and thoracic lymph nodes were included in the tissue 'lymphatic nodes', which is itself included in the list of remainder tissues and organs (Table 2), and so are no longer included in the extrathoracic and thoracic airways respectively as they were in the original HRTM. The fractions, $A_i$, of $w_T$ that they were assigned in Publication 66 are reassigned to other regions in Table 8. The weighted sum of the equivalent dose, $H_i$, to each region, is the equivalent dose to the extrathoracic or thoracic airways respectively:

$$H_{ET} = H_{ET_1} A_{ET_1} + H_{ET_2} A_{ET_2}$$

$$H_{TH} = H_{BB} A_{BB} + H_{bb} A_{bb} + H_{AI} A_{AI}$$

(174) The tissue weighting factor, $w_T$ of 0.12 specified for lung in Publication 103 (ICRP, 2007) is applied to the equivalent dose to the thoracic region, $H_{TH}$. The extrathoracic airways are included in the list of remainder tissues and organs (Table 2).

Table 8. Target tissues of the respiratory tract

| Tissue | Region | Target cells | Depth of target cell nuclei[a], μm | Mass of target tissue[a,b], kg | Assigned fraction[a,c] $A_i$ of $w_T$ |
|---|---|---|---|---|---|

| | | | | Male | Female | |
|---|---|---|---|---|---|---|
| Extrathoracic | $ET_1$ (anterior nose) | Basal | 40-50 | $2.000 \times 10^{-5}$ | $1.729 \times 10^{-5}$ | 0.001 |
| | $ET_2$ (posterior nose, larynx, pharynx) | Basal | 40-50 | $4.500 \times 10^{-4}$ | $3.890 \times 10^{-4}$ | 0.999 |
| Thoracic (lungs) | BB (bronchial) | Secretory ($BB_{sec}$) | 10-40 | $8.648 \times 10^{-4}$ | $7.771 \times 10^{-4}$ | 1/3[c] |
| | | | 35-50 | $4.324 \times 10^{-4}$ | | |
| | | Basal ($BB_{bas}$) | | | $3.885 \times 10^{-4}$ | |
| | bb (bronchiolar) | Secretory | 4-12 | $1.949 \times 10^{-3}$ | $1.874 \times 10^{-3}$ | 1/3 |
| | AI (alveolar-interstitial) | | d | 1.100 | 0.904 | 1/3 |

[a]Reference values (see footnote a to Table **3**). For the BB, bb and AI regions each value of $A_i$ is exactly one-third.

[b] Male values were taken from Publication 68, Table 3 (ICRP, 1994b). Female values for ET and AI were taken from Publication 66, Table 5. Female values for BB were calculated here using information from Publication 66, Tables 2, 4 and B6 (ICRP, 1994a). Masses for $BB_{sec}$ and $BB_{bas}$ are the masses of bronchial epithelium through which the nuclei of secretory cells and basal cells respectively are distributed and are based on reference values of airway dimensions. The mass of AI includes blood, but excludes lymph nodes.

[c]The dose to BB ($H_{BB}$) is calculated as the arithmetic mean of the doses to $BB_{sec}$ and $BB_{bas}$.

[d]Average dose to region calculated.

## 3.3 Human Alimentary Tract Model (HATM)

(175) The Publication 30 (ICRP, 1979) model of the gastrointestinal tract has been replaced by the Human Alimentary Tract Model (HATM) described in Publication 100 (ICRP, 2006). This replacement was motivated by a number of developments, including the availability of improved information on the gut transit of materials, and developments in our understanding of the location of sensitive cells. The model structure is shown in Figure 13, and parameter values are shown in Table 9. As for the HRTM, an important feature of the HATM is the specific calculation of doses to target regions containing sensitive cells for cancer induction, and the consideration of specific absorption and/or retention values, where information is available. The HATM and the HRTM are compatible and inter-connected, as shown in Figure 13.

### 3.3.1 Structure

(176) The HATM depicts the entry of a radionuclide into the oral cavity by ingestion or into the oesophagus after particle transport from the respiratory tract. It describes the sequential transfer through all alimentary tract regions, including the oral cavity, oesophagus, stomach, small intestine, and segments of the colon, followed

by emptying in faeces. Doses are calculated for all these regions. The colon is partitioned, for the purposes of dose calculations, into right colon, left colon and rectosigmoid (the sigmoid colon and rectum) based on the availability of transit time data. The rectum is included with the sigmoid colon, as the rectosigmoid, because of difficulties in determining transit times separately and because the rectum does not have a specific $w_T$ value. Total colon doses are combined as a mass-weighted mean to include the right colon, left colon and rectosigmoid.

### 3.3.2 Model parameters

(177) The HATM presents different transit times for solid foods, liquids, and total diet, in the mouth, oesophagus and stomach. First-order kinetics is assumed for all transfers in the HAT. This is a considerable simplification of the complex processes involved in transfer of material through the lumen of the alimentary tract but is expected to provide a reasonably accurate representation of the mean residence time of a radionuclide in each segment of the tract.

(178)

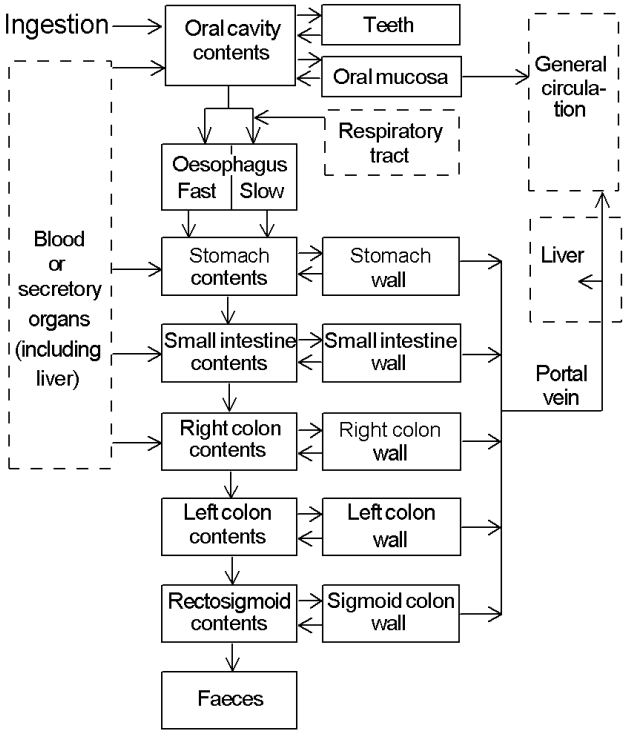



Figure 13 Structure of the HAT model. The dashed boxes are included to show connections between the HATM and the HRTM and systemic biokinetic models. $f_A$ gives net transfer to blood and replaces the $f_1$ value of the gastrointestinal tract model. In general, uptake of radionuclides is assumed to occur from the small intestine.

Table 9. Default generic HAT model transfer coefficients (per day) for total diet for the reference worker[a,b]

| From | To | Transfer coefficient [c] ($d^{-1}$) |
|---|---|---|
| Oral cavity contents | Oesophagus Fast | 6480 |
| Oral cavity contents | Oesophagus slow | 720 |
| Oesophagus Fast | Stomach contents | 12343 |
| Oesophagus Slow | Stomach contents | 2160 |
| Stomach contents | Small intestine contents | 20.57 |
| SI contents | Right colon contents | 6 |
| RC contents | Left colon contents | 2 |
| LC contents | Rectosigmoid contents | 2 |
| RS contents | Faeces | 2 |

[a] The transfer rates of ICRP Publication 100 for the adult male have been assumed for the reference worker.

[b] Other transfer coefficients not given here are assumed to be zero unless specified in the relevant element section. In most cases uptake to blood from the alimentary tract is taken to occur from the small intestine (SI) contents, without retention in SI wall. The corresponding transfer coefficient is $\frac{f_A \lambda_{SI,RC}}{1-f_A}$, where $\lambda_{SI,RC}$ is the transfer coefficient from SI

contents to right colon contents.

[c] The degree of precision of the values given is for computational purposes and does not reflect the certainty with which they are known.

***Modifying factors***

(179) The default regional transit times given in the HATM are central estimates based on collected data for a given sex, age group, and type of material (*e.g.*, solids, liquids, caloric liquids, or non-caloric liquids). As extensively illustrated in Publication 100 (ICRP, 2006), transit of material through each of the major segments of the tract shows considerable inter- and intra-subject variability even under normal conditions. Extremely large deviations from the norm may result from constipation, diarrhoea, unusual diet, pharmaceuticals, and a variety of diseases that affect the nervous system or increase energy requirements, for example.

***Sex specific values***

(180) The HATM provides sex-specific parameter values for adults for dimensions and transit times of contents through the regions. Transit times and dimensions of the stomach and intestines are generally greater and lower respectively in females compared to males. In adults, mean transit times for the stomach and colon are about one-third greater in females than males. However, for simplicity, parameter values for the reference adult male are used in this report series.

***Material entering from the respiratory tract***

(181) Mucus and associated materials cleared from the respiratory tract enter the oesophagus via the oropharynx. For ingested food and liquids, the HATM specifies two components of oesophageal transit, representing relatively fast transfer of 90% of the swallowed material (mean transit time of 7 seconds for total diet) and relatively slow transit of the residual 10% (40 seconds for total diet). It is assumed that the slower oesophageal transit times apply to all material cleared from the respiratory tract.

### 3.3.3 Absorption from the alimentary tract

(182) Radionuclides may enter the alimentary tract directly as a result of ingestion, or indirectly after inhalation and mucociliary escalation of particles from the respiratory tract to the oropharynx and oesophagus. The absorption of radionuclides to blood is specified in the HATM as a fraction of the amount entering the alimentary tract, with total absorption denoted as $f_A$ (ICRP, 2006). The model structure allows for the use of data on absorption in any region, where information is available. In most cases, no information will be available on the regional absorption of radionuclides and the default assumption is that all absorption takes place in the small intestine, *i.e.* $f_{SI} = f_A$. As a default, it is also assumed there is no recycling from the wall to the contents of the alimentary tract.

(183) Some $f_A$ values recommended in this report are the same as the $f_1$ values given previously for use with the Publication 30 model, since there is not sufficient new

information to warrant a revision in the value. Specific data of absorption from other regions are considered in the small number of cases for which they were available, although in some cases (*e.g.* isotopes of iodine) doses to alimentary tract regions and other tissues are insensitive to assumptions regarding the site of absorption (ICRP, 2006).

(184) The extent of absorption of radionuclides will depend on the element and its chemical forms. Changes in chemical forms are likely to occur during digestive processes, beginning in the mouth, but principally occurring in the stomach and the small intestine. These changes in chemical form or speciation will determine the availability of the radionuclide for absorption and hence the extent of uptake through the intestinal epithelium to bloodstream (ICRP, 2006).

(185) Radionuclides entering the alimentary tract in the form of an insoluble matrix represent a specific case since absorption is likely to be controlled by the biokinetics of the matrix rather than that of the radionuclide. In the absence of material-specific data, it is proposed that the fractional absorption of the radionuclide should be taken to be that of the particle matrix.

(186) A further specific case arises for ingestion of insoluble particulate material containing radionuclides with decay chains, where the decay products formed in the stomach or the intestine may be more soluble than their parents. In the absence of material-specific data, it is proposed (as for the respiratory tract) that the fractional absorption should be taken to be that of the particle matrix, which could be predominantly made up of a compound containing the parent radionuclide, or another material in which the parent radionuclide is a minor constituent.

(187) For inhaled particles reaching the alimentary tract after clearance from the respiratory tract, it is appropriate to take account of solubility in the lungs in specifying $f_A$ values. For some elements exhibiting a range in solubility according to their physicochemical form, there is evidence that the reduced solubility of Type M or S materials is also associated with reduced intestinal absorption. For inhaled and then ingested Types M and S materials, the default $f_A$ value is determined here as the product of $f_r$ for the absorption Type and the $f_A$ value for soluble forms of the element. However, because of the need for realism in estimates of absorption for application to bioassay interpretation, attempts have been made wherever possible to use available data to specify $f_A$ values for different forms rather than rely on defaults.

### 3.3.4 Retention in the alimentary tract regions

(188) The model structure allows, where information is available, for the use of data on retention of radionuclides in different compartments. Human and animal data suggesting or showing retention of ingested radionuclides on teeth or in mucosal tissues of the walls of alimentary tract regions, principally the small intestine, can be used to refine calculation of doses to the alimentary tract. An example given in Publication 100 (ICRP, 2006) for cadmium shows that retention of $^{115}$Cd on teeth increases the estimated dose to the oral mucosa by almost two orders of magnitude compared to that calculated using the Publication 30 model. Similarly, retention of $^{59}$Fe in the wall of the small intestine may increase the dose by about a factor two, compared to that calculated with the ICRP 30 model. However, in both examples, these increases in organ doses do not lead to significant changes in the committed

effective doses, which are dominated by contributions from other tissues (ICRP, 2006). Information on retention in alimentary tract tissues is given, where available, in individual element sections of this report series.

### 3.3.5 Alimentary Tract Dosimetry

(189) The HATM allows explicit calculations of dose to target regions for cancer induction within each alimentary tract region, considering doses from radionuclides in the contents of the regions, and considering mucosal retention of radionuclides when appropriate.

(190) The oesophagus and oral cavity will receive very low doses from ingested radionuclides because of short transit times in these regions (ICRP, 2006). However, they were included because a specific $w_T$ is assigned to the oesophagus (ICRP, 2007), and because retention in the mouth, on teeth for example, can result in a substantial increase in dose to the oral mucosa (which was added to the organs and tissues constituting the remainder in Publication 103).

(191) In general, the alimentary tract regions of greater importance in terms of doses and cancer risk are the stomach and particularly the colon. While the small intestine may receive greater doses than the stomach, it is not sensitive to radiation-induced cancer and is not assigned a specific $w_T$ value (ICRP, 2007), but is included in the remainder.

(192) An important refinement in the HATM is the methodology used to calculate doses in the various regions from non-penetrating alpha and electron radiations. Thus, while the Publication 30 approach was to assume that the dose to the wall was one half of that to contents of the region, with an additional factor of 0.01 included for alpha particles to allow for their short range (ICRP, 1979), the HATM takes explicit account of the location of the target tissue in the mucosal layer of the wall of each region. The targets relating to cancer induction are taken in each case to be the epithelial stem cells, located in the basal layers of the stratified epithelia of the oral cavity and oesophagus and within the crypts that replenish the single cell layer epithelium of the stomach and small and large intestines.

(193) This new methodology generally results in substantially lower estimates of doses to the colon from alpha and beta-emitting radionuclides than obtained using the Publication 30 model. This is because of the loss of the alpha particles and electrons energies in the colon contents and in the mucosal tissue overlying the target stem cells (at a depth of 280 – 300 μm). This reduces energy deposition in the target tissue for electrons and results in zero contribution to dose in the target tissue from alpha particles emitted within the contents. In the absence of retention of radionuclides in the alimentary tract wall, doses from ingested alpha emitters to all regions of the alimentary tract will be solely due to their absorption to blood and subsequent irradiation from systemic activity in soft tissues.

(194) The consequences of this decrease in local colon dose on the total committed effective dose will vary according to the radionuclide. Examples given in Publication 100 (ICRP, 2006) for $^{55}$Fe, $^{90}$Sr and $^{239}$Pu show that this decrease of local dose to the colon has little or no impact on the effective dose since the dominating contributions are from equivalent doses to organs and tissues from activity absorbed to blood. In general, the effect on effective dose is small for radionuclides with large $f_A$ values or

long-lived radionuclides with long term retention in the body. However, for the example of $^{106}$Ru, there is a decrease in committed effective dose as well as colon dose, by about a factor two and five respectively, due to the major contribution to effective dose from equivalent doses to alimentary tract regions for this radionuclide.

## 3.4 Intact Skin and Wounds

### 3.4.1 Intact skin

(195) Intact skin is an effective barrier against entry of most substances into the body, and few radionuclides cross it to any significant extent. Exceptions of practical importance are tritiated water in liquid or vapour form, organic carbon compounds and iodine in vapour form or in solution.

(196) There is no general model for absorption of radionuclides through the skin because of the wide range of possible exposure scenarios. Skin can become contaminated by contact with, for example, aerosols, liquids, contaminated surfaces or contaminated clothing. The physical and chemical form of the contaminant (including pH) and the physiological condition of the skin are important factors in any dose assessment.

(197) Both the radiation dose to the area of skin contaminated and the dose to the whole body as a result of absorption should be considered. ICRP (ICRP, 1991, 2007) recommends that skin doses should be calculated to sensitive cells, assumed to be at a depth of 70 μm, or averaged over the layer of tissue 50 to 100 μm below the skin surface and averaged over the most exposed 1 cm$^2$ of skin tissue. This applies to activity either distributed over the skin surface or aggregated in particles. No dosimetric models are recommended by ICRP for calculating doses from radionuclides deposited on the skin and no dose coefficients are given.

### 3.4.2 Wounds

(198) Radionuclides may be transferred from the site of a contaminated wound to blood and to other organs and tissues, and the NCRP has developed a model to describe this transfer for materials in different physico-chemical forms (NCRP, 2007 and Figure 14). Because of the lack of adequate human data, parameter values for the model were based on experimental animal data. When coupled with an element-specific systemic biokinetic model, the model can be used to calculate committed doses to organs and tissues and committed effective doses following transfer of the radionuclide to the blood and systemic circulation, as well as to predict urinary and faecal excretion.

(199) This model was designed to be applicable for both soluble and insoluble radioactive materials. Five compartments are used were designated to describe physical or chemical states of the radionuclide within the wound site. These comprise: Soluble (S) material; Colloidal and Intermediate State (CIS) material; Particles, Aggregates and Bound State (PABS); Trapped Particles and Aggregates (TPA); and Fragments. In some cases, the compartments contain the radionuclide in its original physico-chemical form. In others, the originally deposited material changes state and moves from one compartment to another with time. In most cases the model

simplifies to two or three compartments depending on the physical and chemical form of the radionuclide specified.

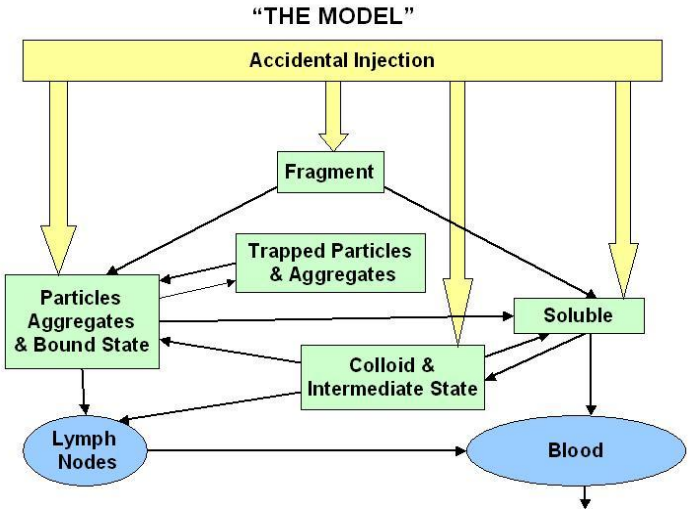
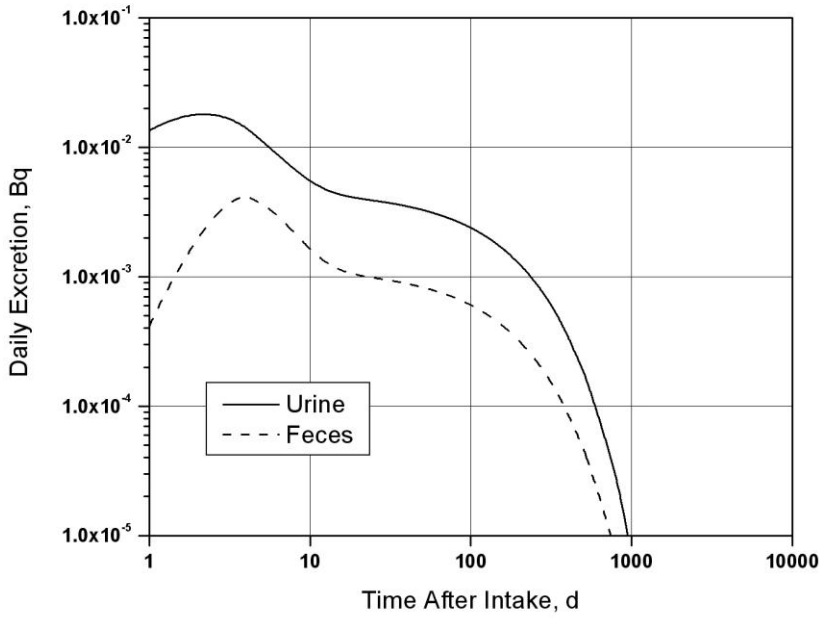



Figure 14. Diagram illustrating the NCRP Model for Wounds

(200) Four retention categories are defined for radionuclides present initially in soluble form in a wound: Weak, Moderate, Strong and Avid, which refer generally to the magnitude of persistent retention at the wound site. The criteria for categorisation are based on: (a) the fraction of the injected radioactive material remaining 1 d after deposition and (b) the rate(s) at which the initially retained fraction was cleared.

(201) Release of the radionuclide from the wound site occurs via the blood for soluble materials and via lymph nodes (LN) for particulates. Further dissolution of particles in LN also results in radionuclide transfer to the blood. The blood is the central compartment that links the wound model with the respective radioelement-specific systemic biokinetic model. Once the radionuclide reaches the blood, it behaves as if it had been injected directly into blood in a soluble form. This is the same approach as is taken in the HRTM and HATM.

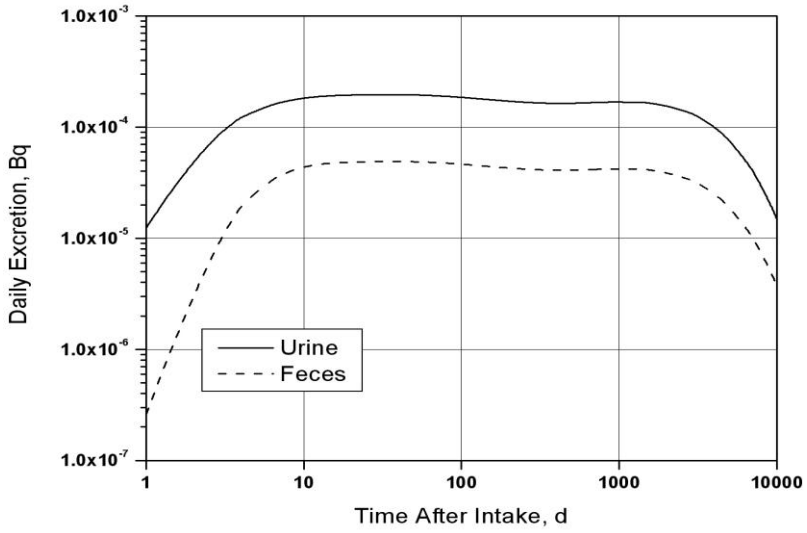
(202) To illustrate the application of the model for bioassay interpretation, the wound model was coupled to the systemic biokinetic model for $^{137}$Cs (ICRP, 1979, 1989, 1997b). The principal default for Cs in the wound model is the Weak Category. Accordingly, the parameters for this category were applied to the wound model, and urine and faecal excretion patterns predicted (Figure 15). The patterns show peak excretion of $^{137}$Cs in urine at 2-3 days after intake, and for faeces at about 5 days. Both patterns reflect the rapid movement of $^{137}$Cs from the wound site, and its distribution in and excretion from the systemic organ sites.




(203)

Figure 15 $^{137}$Cs Wound, Weak Category; predicted values (Bq per Bq intake) following acute intake.

(204) In comparison, if the $^{137}$Cs in the contaminated wound site is assumed to be present in particles of irradiated power reactor fuel, then it can be given parameter values of the Particle Category. In this case, dissolution and absorption to blood are much slower than for the Weak Category, and the urine and faecal excretion patterns exhibit a pseudo-equilibrium pattern after about 10 days, lasting for several years (Figure 16).




(205)

Figure 16 $^{137}$Cs Wound, Particle Category; predicted values (Bq per Bq intake) following acute intake.

(206) The presence of wounds, 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.

(207) As noted in Section 3.1, the assessment of internal contamination resulting from wounds is in practice treated on a case-by-case basis using expert judgement. In many cases, the amount of a radionuclide transferred from a wound site to blood may be assessed directly from urine bioassay data. No dosimetric models are recommended by ICRP for calculating doses from radionuclides transferred from wound sites to blood and to other organs and tissues, and no dose coefficients are given.

## 3.5 Biokinetic Models for Systemic Radionuclides

### 3.5.1 General patterns of behaviour of systemic radionuclides

(208) Radionuclides entering blood may distribute nearly uniformly throughout the body (*e.g.*, $^{3}$H as tritiated water), they may selectively deposit in a particular organ (*e.g.* $^{131}$I in the thyroid), or they may show elevated uptake in a few different organs (*e.g.*, $^{239}$Pu or $^{241}$Am in liver and bone). If a radionuclide that enters blood is an isotope of an essential element (*e.g.*, $^{45}$Ca or $^{55}$Fe), it is expected to follow the normal metabolic pathways for that element. If it is chemically similar to an essential element (*e.g.*, $^{137}$Cs as a chemical analogue of potassium, and $^{90}$Sr as a chemical analogue of calcium), it may follow the movement of the essential element in a qualitative manner but may show different rates of transfer across membranes. The behaviour of a radioisotope of a non-essential element after its uptake to blood (*e.g.*, $^{106}$Ru, $^{125}$Sb, $^{232}$Th, $^{239}$Pu, or $^{241}$Am) depends on such factors as the extent to which it can be sequestered by the reticuloendothelial (RE) system, its affinity for specific biological ligands, its filterability by the kidneys, and the ability of the body to eliminate it in liver bile or other secretions into the gastrointestinal tract. In some cases, the biokinetics of an isotope of a non-essential element may resemble that of an essential element to some extent due to common affinities for some but not all components of tissues and fluids. For example, the behaviour of plutonium in blood and liver is related to that of iron due to an affinity of plutonium for certain proteins that transport or store iron (*e.g.* transferrin), but as a whole the biokinetic behaviour of plutonium in the body differs greatly from that of iron. Also, the behaviours of lead and uranium in the skeleton bear some resemblance to that of calcium because these elements can replace calcium to some extent in bone crystal, although the biokinetic behaviours of lead and uranium in other parts of the body show greater differences compared with calcium. Nevertheless, it is important to emphasise that the use of chemical or biological anologues has its limits (Ansoborlo *et al*, 2006).

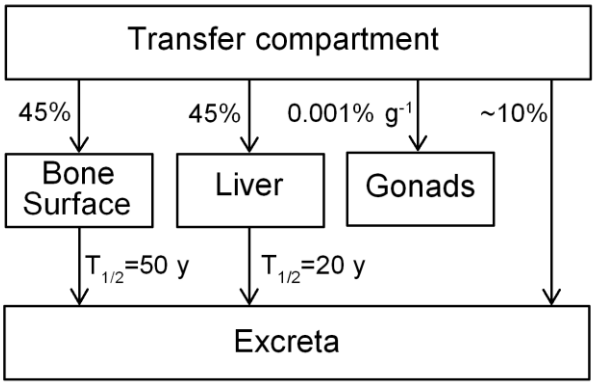
(209) A model that describes the time-dependent distribution and excretion of a radionuclide in the body after it reaches the systemic circulation is referred to here as a systemic biokinetic model. In contrast to ICRP's current and past biokinetic models describing the behaviour of radionuclides in the respiratory and alimentary tracts, ICRP's systemic biokinetic models generally have been element-specific models with regard to model structure as well as parameter values. A generic model structure that depicts all potentially important systemic repositories and paths of transfer of all elements of interest in radiation protection would be too complex to be of much practical use. However, generic model structures have been used in previous ICRP documents to describe the systemic biokinetics of small groups of elements, typically chemical families, known or expected to have qualitatively similar behaviour in the body. For example, Publication 20 (ICRP, 1973) introduced a generic model formulation for the alkaline earth elements calcium, strontium, barium, and radium, but provided element-specific values for most model parameters. In Parts 1-3 of Publication 30 (ICRP, 1979, 1980, 1981) a model developed for plutonium, including parameter values as well as model structure, was applied to most actinide elements. The biokinetic models for several of these actinide elements were modified in Part 4 of Publication 30 (ICRP, 1988), where the model structure for plutonium was used as a generic structure; a common set of parameter values was applied to plutonium, americium, and curium; and element-specific values were applied to selected parameters in the models for other elements. The use of generic systemic model structures was increased in ICRP's reports on doses to members of the public from intake of radionuclides (ICRP, 1993, 1995a, 1995b) and is further expanded in the present document because it facilitates the development, description, and application of systemic biokinetic models.

### 3.5.2 Formulation of systemic models in modern ICRP reports

(210) Publication 30 (ICRP, 1979, 1980, 1981, 1988) provided a comprehensive set of systemic biokinetic models for radionuclides commonly encountered in occupational settings. The models were generally in the form of retention functions (*e.g.*, sums of exponential terms) that may be interpreted as first-order compartmental models with one-directional flow. These models were designed mainly to estimate the cumulative activities of each radionuclide in its main repositories in the body. They do not depict realistic paths of movement of radionuclides in the body but describe only the initial distribution of elements after uptake to blood and the net biological half-times of elements in source organs. Activity absorbed from the gastrointestinal or respiratory tract or through wounds was assumed to enter a transfer compartment, from which it transfers to source organs with a specified half-time, typically 0.25 d or longer. Retention in a source organ was usually described in terms of 1 - 3 first-order retention components, with multiple biological half-times representing retention in multiple hypothetical compartments within a source organ. Feedback of activity from tissues to blood was not treated explicitly in Publication 30 with the exception of the model for iodine. It was generally assumed that activity leaving an organ moves directly to a collective excretion compartment, *i.e.*, radioactive decay along actual routes of excretion is not assessed. Relatively short-lived radionuclides (half-lives up to 15 d) depositing in bone were generally assigned to bone surface and longer-lived

radionuclides were assigned either to bone surface or bone volume, depending on their main sites of retention in bone as indicated by available data.

(211) The systemic biokinetic models of Publication 30 were intended primarily for calculation of dose per unit intake for planning purposes rather than for retrospective evaluation of doses. For some elements these systemic biokinetic models were developed separately from ICRP's concurrent bioassay models. For example, urinary and faecal excretion models for plutonium, americium, and curium recommended in Publication 54 (ICRP, 1988) were derived independently of the concurrent systemic biokinetic model for these elements shown in Figure 17.




(212)

Figure 17. Systemic biokinetic model for plutonium, americium, and curium recommended in Publication 30, Part 4 (ICRP, 1988). This illustrates the one-directional flow of systemic activity depicted in models of Publication 30 and, for many radionuclides, in later ICRP documents on occupational or environmental exposure to radionuclides.

(213) A series of ICRP reports on doses to members of the public from intake of radionuclides (ICRP, 1989, 1993b, 1995a,c, 1996) provided age-specific systemic biokinetic models for selected radioisotopes of 31 elements: hydrogen, carbon, sulphur, calcium, iron, cobalt, nickel, zinc, selenium, strontium, zirconium, niobium, molybdenum, technetium, ruthenium, silver, antimony, tellurium, iodine, caesium, barium, cerium, lead, polonium, radium, thorium, uranium, neptunium, plutonium, americium, and curium. Those reports are referred to here as the Publication 72 series, after the summary document that concluded the series (ICRP, 1996). Most of the systemic biokinetic models in the Publication 72 series followed the same modelling scheme as applied in Publication 30 and illustrated in Figure 17, except that explicit excretion pathways were included in reports completed after the issue of Publication 60. These pathways were included to allow the assessment of doses to the urinary bladder and colon, both of which were assigned tissue weighting factors in Publication 60. A different modelling scheme involving more realistic paths of movement of systemic radionuclides was applied in the Publication 72 series to iron

and the following ‘bone-seeking’ elements: calcium, strontium, barium, lead, radium, thorium, uranium, neptunium, plutonium, americium, and curium. The model structures for these elements and the structure for iodine, carried over from Publication 30, depict feedback of material from organs to blood and, where feasible, physiological processes that determine the biokinetics of radionuclides. Examples of such physiological processes are bone remodelling, which results in removal of plutonium or americium from bone surface, and phagocytosis of aging erythrocytes by reticuloendothelial cells, which results in transfer of iron from blood to iron storage sites.

(214) The physiologically based modelling scheme applied in the Publication 72 series is illustrated in Figure 18, which shows the generic model structure used for the actinide elements thorium, neptunium, plutonium, americium and curium. The systemic tissues and fluids are divided into five main components: blood, skeleton, liver, kidneys, and other soft tissues. Blood is treated as a uniformly mixed pool. Each of the other main components is further divided into a minimal number of compartments needed to model the available biokinetic data on these five elements or, more generally, ‘bone-surface-seeking’ elements. The liver is divided into compartments representing short- and long-term retention. Activity entering the liver is assigned to the short-term compartment (Liver 1), from which it may transfer back to blood, to the intestines via biliary secretion, or to the long-term compartment from which activity slowly returns to blood. The kidneys are divided into two compartments, one that loses activity to urine over a period of hours or days (Urinary path) and another that slowly returns activity to blood (other kidney tissue). The remaining soft tissue other than bone marrow is divided into compartments ST0, ST1, and ST2 representing rapid, intermediate, and slow return of activity to blood, respectively. ST0 is used to account for a rapid build-up of activity in soft tissues and rapid feedback to blood after acute input of activity to blood and is regarded as part of the activity circulating in body fluids. The skeleton is divided into cortical and trabecular fractions, and each of these fractions is subdivided into bone surface, bone volume, and bone marrow. Activity entering the skeleton is assigned to bone surface, from which it is transferred gradually to bone marrow and bone volume by bone remodelling processes. Activity in bone volume is transferred gradually to bone marrow by bone remodelling. Activity is lost from bone marrow to blood over a period of months and is subsequently redistributed in the same pattern as the original input to blood. The rates of transfer from cortical and trabecular bone compartments to all destinations are functions of the turnover rate of cortical and trabecular bone, assumed to be 3% and 18% per year, respectively. Other parameter values in the model are element-specific.

(215) A variation of the model structure shown in Figure 18 was applied in the Publication 72 series to calcium, strontium, barium, radium, lead and uranium (Figure 19). These elements behave differently from the bone-surface seekers addressed above in that they diffuse throughout bone volume within hours or days after depositing in bone. After reaching bone volume, these elements may migrate back to plasma (via bone surface in the model) or they may become fixed in bone volume and are then gradually removed to blood at the rate of bone remodelling. The compartments in Figure 18 representing bone-marrow and gonads are omitted from

the model for bone-volume seekers because these generally are not sites of elevated accumulation of these elements. Some of the compartments shown in Figure 19 are not applicable to all bone-volume seekers. For example, the liver, kidneys, and red blood cells are not important sites of accumulation of calcium and strontium but are important repositories for lead. If a particular compartment or pathway shown in Figure 19 is not important for a given element, it is not considered separately in the model for that element. For example, in the model for calcium, blood is treated as a single well-mixed pool, and the liver and kidneys are assumed to be part of 'Other soft tissues'.

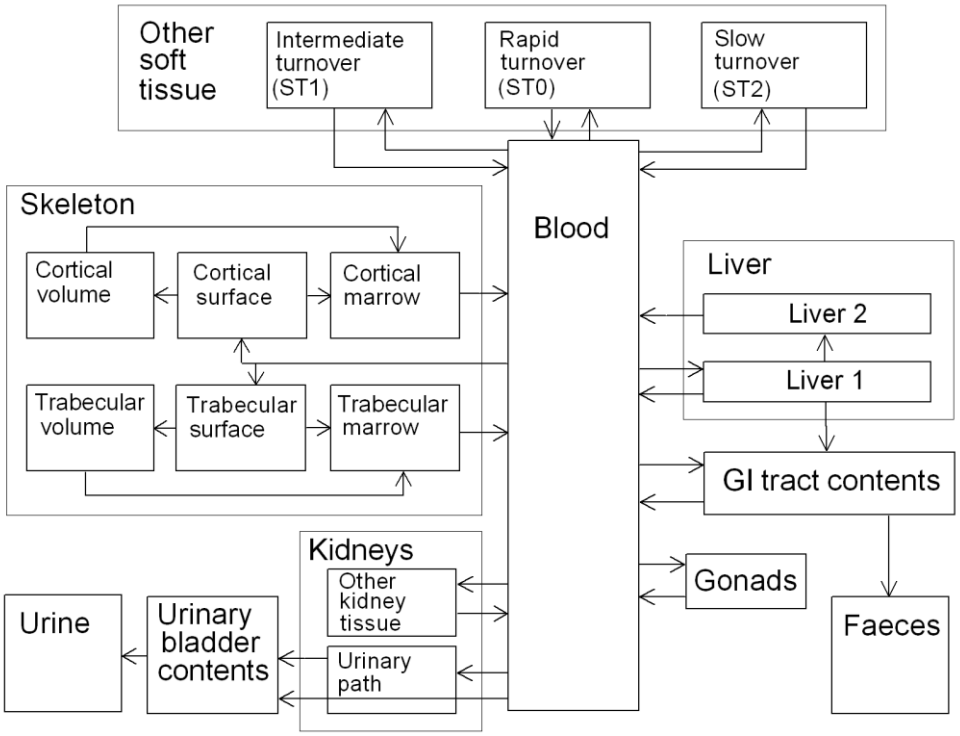



Figure 18. Model structure applied in the Publication 72 series to the bone-surface seekers thorium, neptunium, plutonium, americium, and curium. This structure (or modest variations of it) are applied to a number of elements in this report series, including elements not regarded as bone-seekers.

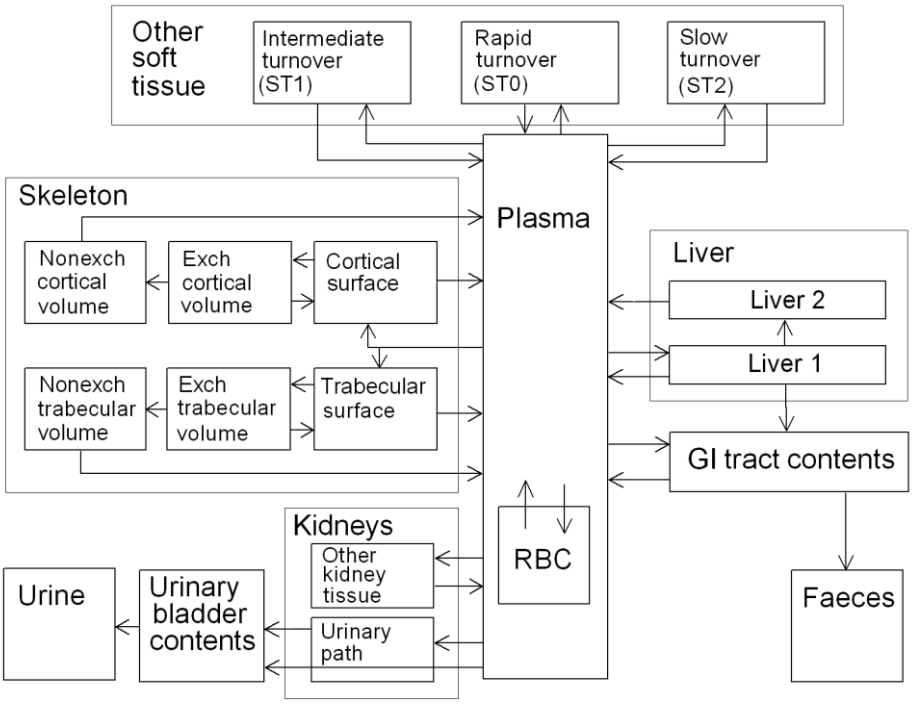



Figure 19. Model structure applied in the Publication 72 series to calcium, strontium, barium, lead, radium, and uranium. This structure (or modest variations of it) are applied to a number of elements in this report series, including elements not regarded as bone-seekers. Abbreviations: Exch = exchangeable, Nonexch = nonexchangeable, RBC = red blood cells.

(216) The systemic models used in the Publication 72 series were applied in Publication 68 (ICRP, 1994b), along with ICRP's Human Respiratory Tract Model (ICRP, 1994a), to update dose coefficients for occupational intake of radionuclides based on recommendations in Publication 60 (ICRP, 1991). For elements not addressed in the Publication 72 series, the systemic biokinetic models applied in Publication 68 were taken from Publication 30 and modified to include specific excretion pathways to address doses to the urinary bladder and colon.

(217) The biokinetic models applied in Publication 68 were used in Publication 78 (ICRP, 1997) to provide new recommendations concerning interpretation of bioassay data for workers for selected radioisotopes of 15 elements. The systemic models for nine of the 15 elements addressed in Publication 78 were physiological based models adopted in the Publication 72 series.

### 3.5.3 Systemic model structures used in this report series

(218) It is now generally recognised that the physiologically descriptive model structures introduced for selected elements in the Publication 72 series have a number of potential advantages over the retention-function models traditionally used in radiation protection. For example, a physiological descriptive model structure facilitates the use of physiological information and physiologically reasonable assumptions as a supplement to radiobiological data in the development of model parameter values; provides a basis for extrapolating beyond the radiobiological

database to different subgroups of the population and to times outside the period of observation (for example, a parameter value found to depend on the rate of bone remodelling can be varied with age on the basis of age-specific data on bone remodelling rates); facilitates the extrapolation of biokinetic data from laboratory animals to man, in that it helps to focus interspecies comparisons on specific physiological processes and specific subsystems of the body for which extrapolation may be valid, even if whole-body extrapolations are not; facilitates the extrapolation of biokinetic data from an element to its chemical analogues, in that the degree of physiological similarity of chemical analogues may vary from one physiological process to another (for example, the alkaline earth elements show similar rates of transfer from blood to bone but much different rates of transfer to non-exchangeable sites in bone); links excretion with exchanges of activity among body tissues and fluids, so that the same model can be used for dose calculation and bioassay interpretation; allows modelling of the differential biokinetics of parent radionuclides and their radioactive progeny produced in the body; and allows the addition of compartments and pathways to the model for purposes of extending the model to new applications, as was demonstrated in the ICRP documents on doses to the embryo and fetus (ICRP, 2001) and to the nursing infant (ICRP, 2004) from intakes of radionuclides by the mother.

(219) On the other hand, the level of physiological realism in the systemic biokinetic models currently used in radiation protection, including those recommended in the present report, should not be overstated. Even the most sophisticated models represent a compromise between biological realism and practical considerations regarding the quantity and quality of information available to determine parameter values. For example, the recycling models applied to bone-seeking radionuclides in the Publication 72 series all include soft-tissue compartments representing fast, intermediate, and slow exchange with blood for all soft tissues not explicitly identified in the models. These soft tissue compartments typically are defined on a kinetic basis rather than a physiological basis, *i.e.*, the compartment sizes and turnover rates are set for reasonable consistency with data on accumulation and loss of elements by soft tissues. For some elements, these soft tissue compartments appear to be associated with specific sites or processes, but the associations generally are not confirmed by available information. For example, biokinetic studies of calcium suggest, but do not establish, that the rapid-turnover pool in soft tissues may correspond roughly to interstitial fluids plus some rapidly exchangeable cellular calcium (Heaney, 1964 Harrison *et al*, 1967;Hart and Spencer 1976); the intermediate turnover rate may stem from a composite of several pools with slower exchange rates, including mitochondrial calcium, cartilage calcium, and exchangeable dystrophic calcium (*e.g.*, arterial plaque and calcified nodes) (Heaney, 1964; Borle, 1981); and long-term retention in soft tissues may be associated with relatively nonexchangeable dystrophic calcium that gradually accumulates in the human body (Heaney, 1964).

(220) For many elements it is not feasible to develop genuine physiological system models due to inadequate information on the processes that determine the systemic behaviour of these elements. Even for relatively well understood elements the model components are often intended only to represent the net result of multiple processes. For example, in the model for bone-surface-seeking radionuclides shown in Figure 18

and its precursors (Leggett, 1985, 1992), the depiction of burial of activity in bone volume is intended to approximate the net result over time of a number of known or suspected burial processes occurring at different rates. Activity depositing in bone remodelling units, either in the formation period or in the transitional period between resorption and formation, may be buried relatively quickly. Delayed burial of surface activity may result from 'local recycling' during bone restructuring processes; that is, some of the surface activity removed by osteoclasts during bone remodelling may be redeposited almost immediately at closely adjacent sites of new bone formation that are supplied by the same blood vessels. Such local redeposition of mineral ions is thought to occur, particularly in cortical bone (Parfitt and Kleerekoper, 1980). Burial of surface deposits may also occur as a result of 'bone drift', a phenomenon in which new bone is deposited on previously formed bone without any prior resorption process. Bone drift occurs on a larger scale in immature bone than in mature bone, but drift within bones and expansion of bone volume via periostial-endosteal drift continues throughout life in humans (Epker and Frost, 1965a,b; Frost 1986; Priest *et al*, 1992). 'Drifting osteons' are observed at all ages within human cortical bone, and their count is used in forensics for age-at-death estimation.

(221) The systemic biokinetic models used in this series of reports generally follow the physiologically descriptive modelling scheme applied on a more limited scale in the Publication 72 series. That is, the model structures include one or more compartments representing blood, depict feedback of activity from extravascular repositories to blood (*i.e.*, they are recycling models), and, as far as practical, depict the main physiological processes thought to determine the systemic biokinetics of individual elements.

(222) The systemic biokinetic models for some elements, such as iodine and iron, are developed within model structures specifically designed to describe the unique behaviour of these elements in the body. The models for most elements, however, have been constructed within one of the two generic model structures applied in the Publication 72 series to bone-seeking radionuclides (Figure 18 and 19), or variations of those structures. This was done not only for bone-seeking elements but for a number of elements that show relatively low deposition in bone (*e.g.*, cobalt and ruthenium) because the main repositories and paths of movement of those elements in the body are included in one or the other of these two structures. In some cases, the model structure as applied in the Publication 72 series has been modified slightly to accommodate specific characteristics of an element or to reflect the limited information on certain aspects of the biokinetics of an element. This is illustrated in Figure 20, which shows the model applied in this series to cobalt. The structure shown in Figure 20 is a variation of the structure for bone-surface seekers (Figure 18), although it could also be viewed as a variation of the structure for bone-volume seekers (Figure 19). In either case, the model for the skeleton has been simplified because of the limited information on the skeletal behaviour of cobalt, and two non-specific blood pools are used to represent two components of retention of cobalt in blood.

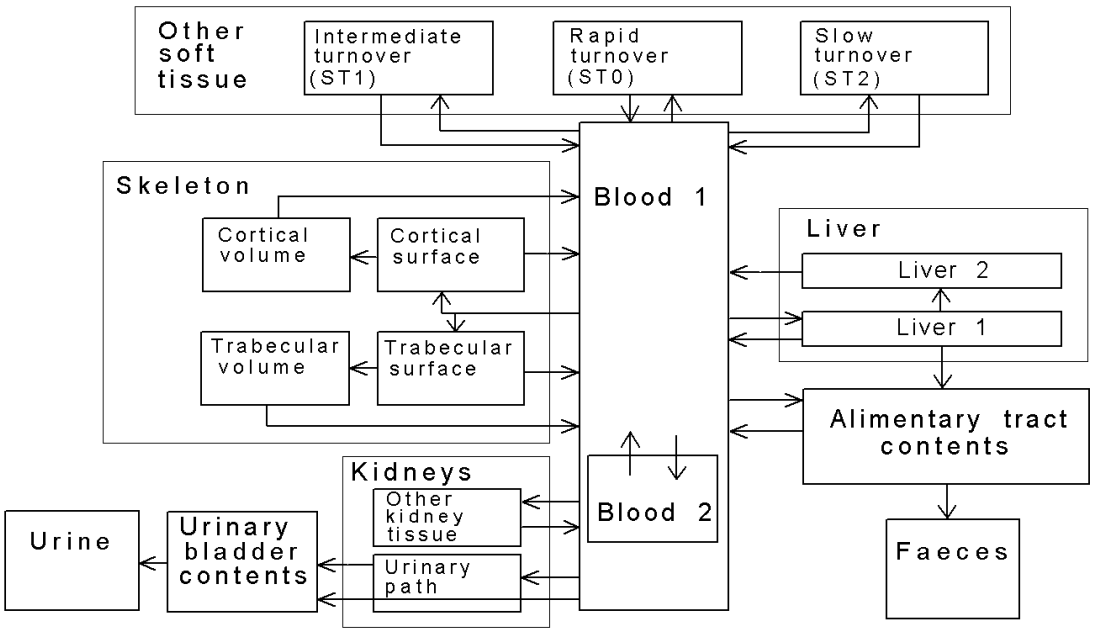



Figure 20. Structure of the systemic biokinetic model for cobalt used here

(223) The systemic biokinetic models used in this report include explicit routes of biological removal of systemic activity in urine and faeces. Additional excretion pathways such as sweat are also depicted in the models for some elements.

(224) The biokinetic model adopted for the urinary bladder is described in Publication 67 (ICRP, 1993b) and Publication 68 (ICRP, l994b). The number of voids per day is taken to be six for workers. To represent the kinetics of the bladder in terms of first-order processes, the rate of elimination from the bladder is taken to be 12 $d^{-1}$.

(225) Activity is assumed to be removed in faeces after transfer from systemic compartments into specified segments of the alimentary tract representing element-specific endogenous secretion pathways. The rates of transfer of secreted material through different segments of the alimentary tract are element-independent rates specified in the HATM. Activity transferred from systemic compartments into the contents of the small intestine or higher segments of the tract is assumed to be reabsorbed in part to blood, with fractional absorption usually but not always assumed to be the same as that for swallowed activity. Activity assigned to the contents of the right colon or lower sections of the tract is not subject to reabsorption.

### 3.5.4 Treatment of radioactive progeny produced in systemic compartments

(226) In Publication 30 (1979) and Publication 68 (1994b) the general assumption was made that chain members produced in systemic compartments following intake of a parent radionuclide adopt the biokinetics of the parent. This is referred to as the assumption of 'shared kinetics'. The alternate assumption of 'independent kinetics' of chain members was made in Publication 68 when the parent was an isotope of lead, radium, thorium, and uranium, and also for iodine progeny of tellurium and for noble gas isotopes arising in various chains. The implementation of independent kinetics of progeny was based on a general pattern of behaviour of systemically produced progeny radionuclides suggested by a review of experimental and occupational studies (Leggett, 1985). That is, the data suggested that most radioactive progeny produced in soft tissue or bone surface tended to migrate from the parent and begin to follow their characteristic biological behavior, while radionuclides produced in bone volume tended to remain with the parent radionuclide in bone over the period of observation.

(227) The assumption of independent kinetics is generally applied here to progeny radionuclides produced in systemic compartments or absorbed to blood after production in the respiratory or alimentary tract. The basic assumption is that a progeny radionuclide will follow its characteristic behaviour after it first reaches blood. The rate at which a progeny radionuclide is estimated to migrate from its place of birth to blood is based on reported data where available. In the absence of specific information the default assumption is that the progeny radionuclide immediately begins to follow its characteristic behaviour from the time of birth. The implementation of this default assumption is essentially a matter of assigning progeny atoms produced by decay of the preceding chain member(s) to appropriate compartments of the progeny radionuclide's characteristic biokinetic model, which predicts the subsequent fate of these atoms. This is not always a straightforward exercise due to structural differences in the systemic models for many parent and progeny combinations. For example, a radionuclide may be born in an explicitly designated tissue T in the parent's model that is not an explicitly designated tissue in the progeny radionuclide's characteristic model. When this happens, the rate of removal of the progeny radionuclide from T and the destination of the removed activity must be defined before the model can be solved. For a number of chains addressed in this series of reports, this problem has been resolved by expanding the chain members' characteristic models to include all explicitly designated tissues in the models for preceding chain members, based on available biokinetic data on the progeny radionuclide and its chemical or physiological analogues. An alternate 'automated' default treatment of this problem and other issues regarding differences in model structures for parent and progeny radionuclides is described in Section 3.7.2, which addresses the contribution of radioactive progeny to dose.

(228) Even if the progeny radionuclide is produced in a tissue that is an explicitly designated source organ in the progeny radionuclide's characteristic model, implementation of the default treatment of independent kinetics becomes somewhat arbitrary if the progeny radionuclide's model divides the tissue into compartments that are not identifiable with compartments in the parent's model. For example,

suppose the liver is depicted in the parent's model as two compartments and also in the progeny radionuclide's model as two compartments. If the compartments represent the same physically identifiable portions of the liver in both models, *e.g.*, hepatocytes and Kuppfer cells, then decays of the parent in the two liver compartments in the parent's model would be assigned to the corresponding liver compartments in the progeny radionuclide's model. However, if the compartments are defined on a kinetic basis in both models and have no obvious physical interpretation, it will generally not be evident how the progeny atoms produced in the parent's liver compartments should be divided between the progeny's liver compartments. In such cases the convention used here is to assign all of the progeny atoms to the compartment with the highest turnover rate, and assume that the progeny radionuclide is removed from that compartment to the central blood compartment at that rate.

## 3.6 Medical Intervention

(229) If medical treatment to prevent uptake or enhance excretion is administered, then the data provided in the models summarised above cannot be used directly to assess committed effective doses from monitoring information (NCRP, 1980; Gerber and Thomas 1992; IAEA 1996). In such circumstances a programme of special monitoring (Section 5.5) should be undertaken to follow the retention of the particular contaminant in the person, and these data should be used to make a specific assessment of committed dose.

## 3.7 Methodology for dose calculations

(230) The general method of dose calculation described here is similar as in earlier ICRP Publications (ICRP, 1979, 1994b), but some changes were introduced to accord with ICRP Publications (ICRP, , 2008) which used MIRD terminology for radiopharmaceutical dosimetry. In order to provide a consistent internal dosimetry framework for both radiation protection and nuclear medicine, the standardised nomenclature and symbols of MIRD Pamphlet No. 21 (Bolch *et al* 2009) used for protection quantities, and their conventions are followed in this section.

(231) The Commission defines effective dose, E, for adults as:

$$E = \sum_T w_T \cdot \left[ \frac{H(r_T,50)^{Male} + H(r_T,50)^{Female}}{2} \right] \qquad (1)$$

where $w_T$ is the tissue weighting factor (Table 2) for the target tissue $r_T$ and $H(r_T,50)^{Male}$ and $H(r_T,50)^{Female}$ are the committed equivalent doses for the target tissue $r_T$ for the reference male and female, respectively, integrated over 50 years. The committed equivalent dose in the target tissue for the reference male or female is:

$$H(r_T,50) = \int_0^{50} \dot{H}(r_T,t)dt \qquad (2)$$

For both sexes the equivalent dose rate $\dot{H}(r_T,t)$ in target tissue $r_T$ at time $t$ after an acute intake is expressed as

$$\dot{H}(r_T,t) = \sum_{r_S} A(r_S,t) \cdot S_w(r_T \leftarrow r_S) \qquad (3)$$

where

$A(r_S,t)$ is the activity, Bq, of the radionuclide in source region $r_S$ at time $t$ after intake for the reference male or female; in this report series, only male values are used;

$S_w(r_T \leftarrow r_S)$ is the radiation-weighted S value (Bolch *et al*, 2009); *i.e.* the equivalent dose in target tissue $r_T$ per nuclear transformation in source region $r_S$, Sv (Bq s)$^{-1}$, for the reference male or female.

(232) The first factor in equation (3) is derived with biokinetic models which describe the uptake of activity into the body, its distribution and retention within body regions, and its excretion from the body. The second factor is derived with dosimetric models which are used to calculate the dose to target tissues arising from transformations in source regions.

### 3.7.1 Dosimetric models

(233) The equivalent dose in target tissue $r_T$ per nuclear transformation in source region $r_S$, is calculated by:

$$S_w(r_T \leftarrow r_S) = \sum_R w_R \sum_i \frac{E_{R,i} \cdot Y_{R,i} \cdot \phi(r_T \leftarrow r_S, E_{R,i})}{M(r_T)} \qquad (4)$$

where

$E_{R,i}$ is the energy of the $i^{th}$ nuclear transition of radiation type R, in Mev,

$Y_{R,i}$ is the yield of $i^{th}$ radiation of type R per nuclear transformation, (Bq s)$^{-1}$,

$w_R$ is the radiation weighting factor for radiation type R, Table 1

$\phi(r_T \leftarrow r_S, E_{R,i})$ is the absorbed fraction, defined as the fraction of energy $E_{R,i}$ of radiation type R emitted within the source region $r_S$ that is absorbed in the target tissue $r_T$,

$M(r_T)$ is the mass of target tissue $r_T$, kg.

(234) The energies and yields of the emitted radiations, $E_{R,i}$ and $Y_{R,i}$, are taken from Publication 107 (ICRP, 2008), which supersedes Publication 38 (ICRP, 1983). For β radiation and neutrons accompanying spontaneous fission, the spectral data are used in the calculation of $S_w$ rather than mean values. The sex-dependent target tissue

masses $M(r_T)$ are given in Publication 89 (ICRP, 2002a), which supersedes Publication 23 (ICRP, 1975).

(235) For both sexes, the values of the specific absorbed fractions $\Phi(r_T \leftarrow r_S, E_R) = \frac{\phi(r_T \leftarrow r_S, E_R)}{M(r_T)}$ for alpha particles, electrons, photons and neutrons are taken from Publication 125 (ICRP, 2012). For most source and target combinations, the absorbed fractions for photons, electrons and neutrons are based on Monte Carlo radiation transport calculations performed using the voxel phantoms for the ICRP reference adult male and adult female described in Publication 110 (ICRP, 2009). These voxel phantoms are constructed from tomographic images of real persons, with the height and organ masses adjusted to the values given in Publication 89 (ICRP, 2002).

(236) For α particles the absorbed fractions are taken to be 1 for $r_S = r_T$ and 0 for $r_S \neq r_T$ in most cases. Exceptions are combinations of source regions and target tissues in the respiratory and alimentary tracts and in the skeleton. In these cases some regions are small enough for alpha particles to escape.

(237) In the human alimentary tract and the human respiratory tract, absorbed fractions for photons are derived using the reference voxel models. For electrons and α particles, absorbed fractions given for the alimentary tract in Publication 100 (ICRP, 2006) have been updated with supplementary calculations in Publication 125 (ICRP, 2012). The absorbed fractions for electrons and α particles in the respiratory tract given in Publication 66 (ICRP, 1994a) were adopted in Publication 125 (ICRP, 2012).

(238) In the skeleton, biokinetic models consider the source regions to be:

- trabecular bone surfaces and volumes
- cortical bone surfaces (CBS) and volumes. In the new skeletal models, CBS can be
    - haversian canal surfaces within the cortical bone cortex surrounding all regions of trabecular spongiosa
    - haversian canal surfaces within the cortical bone of the long-bone shafts
    - surfaces separating medullary marrow cavities and cortical bone shafts of the long bones
- trabecular bone marrow, corresponding to all bone marrow within regions of trabecular spongiosa – both active and inactive marrow
- cortical bone marrow, corresponding to all bone marrow within the medullary marrow shafts of the long bones, as well as the fluids within the Haversian canals of all regions of cortical bone. In the adult, the marrow of the long bone shafts is 100% inactive marrow.

and the target tissues to be:

- 50 µm endosteal region and
- active (red) marrow.

Endosteum is not considered as all marrow (both active and inactive) within 50 µm of a bone surface, but is thought to be the surrogate tissue for the osteoprogenitor cells, which are present along all bone surfaces regardless of the marrow cellularity (mixture or percentages of active / inactive marrow). The biokinetic models presented in this report may therefore have either a "active marrow source" or a "trabecular marrow source" and specific alpha/electron AFs will be produced for these two skeletal regions.

(239) Values of specific absorbed fractions at fixed energies are tabulated in Publication 125 (ICRP, 2012). Specific absorbed fractions at the particular energies of radionuclide emissions are found by interpolating the tabulated values using a mathematical technique such as cubic splines.

### 3.7.2 Contribution of decay products to dose

(240) The dose coefficients given in this report series take into account the contribution to dose from radionuclides produced in the body by ingrowth. However, as in the past, it is assumed that no radioactive progeny are present in the initial intake of the radionuclide for which the dose coefficient is determined, except for radon and its progeny. Nuclear decay data are taken from Publication 107 (ICRP, 2008).

(241) The source region 'Other tissues' is commonly used in systemic models when uptake is specified in particular organs and tissues and any remaining activity is taken to be distributed in these other tissues. 'Other tissues' is the complement of the explicitly designated tissues; that is, it is the set of all systemic tissues other than those specified in the model. If independent kinetics are assumed for decay products, each member of the decay chain may have different sets of specified source tissues, and as a result the anatomic identity of 'Other tissues' varies among the chain members. This can lead to anomalies when the biokinetic models are solved, such as excess activity growing into one compartment at the expense of another. Annexe C.3 of Publication 71 (ICRP, 1995) outlines two alternative computational procedures that seek to minimise these anomalies.

(242) To explain these approaches, it is useful to distinguish between local and global sources. Local source tissues and local 'Other tissues' are both specific to each chain member. Global sources incorporate all the chain's local sources and global 'Other tissues' is the set of all systemic tissues other than the global sources. Thus for the simple example of a two member chain where the parent's local source is liver and the decay product's local source is kidneys, the corresponding global sources are liver and kidneys and global 'Other tissues' includes all systemic tissues other than liver and kidneys.

(243) The aim of the two approaches of Annexe C.3 (ICRP, 1995) is to redistribute transformations from each chain member's 'Other tissues' compartment to sources $r_S'$ which are in the global set of source compartments but not the local set. For each such source region, $r_S'$, a mass fraction $\frac{m(r_S')}{m(OT)}$ of the chain member's 'Other tissues' (OT) transformations is deducted from OT and transferred to this source $r_S'$. Sources

present in the kinetics of a chain member, but not in the kinetics of the chain parent, also receive a transfer of transformations from the chain member's 'Other tissues' based on a mass fraction computed using the parents $m(OT)$.

(244) The first approach of Annexe C.3 (ICRP, 1995) redistributes transformations after the given biokinetic models are solved, whilst the second effectively 'automates' the process by amending the biokinetic models before solving them. In the latter approach any global sources not included in a chain member's local sources are added to the chain member's model and represented by the same number of compartments specified for their 'Other tissues', each with the same kinetic transfer pathways. The rates of loss from these compartments are the same as the corresponding 'Other tissues' compartments but the transfer rates to them are mass fractions of those of the corresponding 'Other tissues' compartments. The transfer rates to the 'Other tissues' compartments are decremented accordingly. Although both approaches give similar results, the latter is considered to be more rigorous and is used here.

### 3.7.3 Bioassay data

(245) A number of issues should be noted regarding the use of biokinetic models for the retrospective assessment of doses from bioassay data:

(a) As explained in Section 1.4, equivalent dose coefficients for organs and tissues are calculated separately for the Reference Male and Reference Female and then averaged in the calculation of effective dose. Some biokinetic models have sex-specific parameter values, and so a number of possible methods could be implemented to determine effective dose from bioassay measurements:

i. Equivalent doses to organs per unit content of a bioassay quantity could be calculated separately for males and females. Equation 1 (Section 3.7) would then be applied to determine effective dose per unit content.

ii. Intakes could be calculated separately for males and females. The dose coefficient would then be applied to the average intake.

iii. The intake could be calculated with sex-averaged biokinetic data, and the dose coefficient would then be applied to this intake. Biokinetic model parameters could be averaged, or predicted retention/excretion functions could be averaged.

iv. The intake could be determined only with the male (or female) biokinetic model. The dose coefficient would then be applied to this intake.

Since effective dose is a protection quantity that provides a dose for a Reference Person rather than an individual-specific dose, significant advantages arise from adopting a simple approach to retrospective dose assessment. For this reason, method (iv) has been adopted in this series of reports, with the intake determined using the male biokinetic model where sex-specific models are provided. It is recommended that this method should be adopted for the interpretation of bioassay data.

(b) In the dose per unit content functions for retained activity presented in subsequent reports of this series, all activity within the body (including contents of the urinary bladder and the alimentary tract) is included. For the lungs, all activity in the thoracic region of the respiratory tract, including the thoracic lymph nodes, is

included. For the skeleton, all activity in trabecular and cortical bone (both surface and volume) and in bone marrow is included.

(c) In the dose per unit content functions for bioassay samples, the dose per unit content for a 24 h sample of urine or faeces is provided. The sample activity is decay-corrected to the time of the end of the sampling period.

# 4 METHODS OF INDIVIDUAL AND WORKPLACE MONITORING

## 4.1 Introduction

(246) This Chapter briefly describes the main measurement techniques, their advantages and their limitations for individual monitoring. In most cases, assessment of intakes of radionuclides may be achieved by body activity measurements, excreta monitoring, air sampling with personal air samplers, workplace measurements or a combination of these techniques. The choice of measurement technique will be determined by a number of factors including the radiation emitted by the radionuclide, the availability of equipment, the biokinetic behaviour of the contaminant and the likely radiation dose.

## 4.2 Body Activity Measurements (In Vivo Measurements)

(247) *In vivo* measurement of body or organ content provides a quick and convenient estimate of activity in the body. It is performed with one or more photon detectors placed at specific positions in relation to the subject being measured. It is feasible only for those radionuclides emitting radiation that can be detected outside the body. In principle, the technique can be used for radionuclides that emit: X or $\gamma$ radiation: positrons, since they can be detected by measurement of annihilation radiation; or energetic $\beta$ particles that can be detected by measurement of Bremsstrahlung radiation (*e.g.* $^{90}$Y, produced by the decay of its $^{90}$Sr parent).

(248) The detectors used for *in vivo* measurements are usually partially shielded and the individual to be measured can be placed in a shielded, low background room to reduce the interference from ambient sources of radiation.

(249) Direct (*in vivo*) bioassay is likely to be the monitoring method of choice if the radionuclide is a high yield, high energy gamma-ray emitter or decays by positron emission (with emission of annihilation radiation), unless the material is excreted rapidly from the body. The gamma-radiation emitted by such radionuclides is strongly penetrating, and so is readily detected using scintillation or semiconductor detectors positioned close to the body. If the material is absorbed rapidly from the respiratory tract, and is then either distributed uniformly in body tissues (*e.g.* $^{137}$Cs in most common chemical forms), or is distributed preferentially among a number of organs, (*e.g.* $^{59}$Fe) then whole body monitoring should be chosen. If the radionuclide deposits preferentially in a single organ such as the thyroid (*e.g.* $^{125}$I, $^{131}$I), then partial body monitoring of the relevant organ should be chosen. In the case of materials that are absorbed less rapidly from the respiratory tract (*e.g.* insoluble forms of $^{60}$Co oxide), lung monitoring is preferable to whole body monitoring soon after the intake, as it gives a more accurate measure of lung deposition and retention than a whole body measurement.

(250) Direct bioassay is also useful for some radionuclides that emit photons (X- or $\gamma$-rays) at lower energies and/or with lower yields (*e.g.* $^{241}$Am, $^{210}$Pb, $^{144}$Ce). However, in the case of radionuclides that mainly emit X-rays below 25 keV with low yields (notably, the alpha-emitting isotopes of plutonium and curium) direct bioassay may not achieve the sensitivity required for radiological protection purposes.

(251) The activity present in a wound can be detected with conventional $\gamma$ detectors if the contaminant emits energetic $\gamma$-rays. In the case of contamination with $\alpha$-emitting radionuclides, detection is much more difficult since the low energy X-rays that follow the $\alpha$-decay will be strongly 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 must have an energy discrimination capability if a good estimate is to be made of contamination with mixtures of radionuclides. If whole body measurements are made, it may be necessary to shield any activity remaining at the wound site.

(252) For activity calibrations of *in vivo* monitoring systems, laboratories generally use physical phantoms, either commercially available or handcrafted (*e.g.* the Bottle-Mannikin-ABsorption (BOMAB) phantom, the Lawrence Livermore thorax phantom (Griffith *et al*, 1986; Snyder *et al*, 2010)). This approach has some limitations with respect to the body size, body shape, and radionuclide distribution. The distribution of the radionuclide in the calibration phantom should match that expected in the human subject as far as possible. Alternatively, numerical calibration techniques may be applied. Mathematical software combines voxel phantoms and Monte-Carlo statistical simulations to model photon transport from the phantom and the detection of photons by a simulated detector (Franck *et al*, 2003; Hunt *et al*, 2003; Kramer, 2005; Gómez-Ros *et al* 2007; Lopez *et al* 2011a).

(253) The IAEA (1996) and the ICRU (2002a) have given guidance on the direct measurement of body content of radionuclides.

## 4.3 Analysis of Excreta and Other Biological Materials

(254) Excreta monitoring programmes usually involve analysis of urine, although faecal analysis may also be required if the material is relatively insoluble. Other samples may be analyzed for specific investigations. Examples are the use of nose blow or nasal smears as routine screening techniques.

(255) 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 or estimate the total activity excreted in urine per unit time from measurements on 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. Where a 24 h sample is not easily collected then the first morning voiding is preferable for analysis (IAEA, 2000). Measurement of creatinine concentration in urine has frequently been used to estimate 24 h excretion of radionuclides from urine samples collected over part of a day. Tritium is an exceptional 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 (*e.g.* Duke, 1998).

(256) The interpretation of faecal samples for routine monitoring involves uncertainty 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 investigations, particularly following a known or suspected intake by inhalation of moderately soluble or insoluble 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.

(257) Radionuclides that emit photons may be determined in biological samples by direct measurement with scintillation or semiconductor detectors. Analysis of $\alpha$- and $\beta$-emitting radionuclides usually requires chemical separation followed by appropriate measurement techniques, including alpha spectrometry and liquid scintillation counting. Measurement of so-called total $\alpha$ or $\beta$ activity may occasionally be useful as a simple screening technique.

(258) Increasing use is being made of mass spectrometric techniques for the analysis of excreta samples. Examples are Inductively Coupled Plasma - Mass Spectrometry (ICP-MS) that can achieve much lower detection limits for long-lived radionuclides than is possible with alpha spectrometry and Thermal Ionization Mass Spectrometry (TIMS), used to monitor very low activities of $^{239}$Pu in urine (Inkret *et al*, 1998; LaMont *et al*, 2005; Elliot *et al*, 2006).

(259) 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 (Youngman *et al*, 1994; Sathyabama *et al*, 2005). It can also be used to monitor $^{14}CO_2$ formed *in vivo* from the metabolism of $^{14}$C-labelled compounds (Leide-Svegborn *et al*, 1999; Gunnarsson *et al*, 2003).

(260) Nasal smears may be employed as a useful screening technique. A positive nasal swab gives an indication that an unexpected situation might have occurred. Excreta measurements or lung monitoring should follow, to confirm the intake and to provide a quantitative assessment.

### 4.4 Exposure Monitoring of the Workplace

(261) Workplace monitoring is useful for triggering bioassay measurements. In addition, workplace characterization may be used as a complement to bioassay monitoring as it provides useful information on physical and chemical composition of the radionuclides present in the working environment (*e.g.* information on the particle sizes (AMAD)).

(262) Two workplace monitoring methods may be used for monitoring individual exposures: personal air sampling (PAS) and static air sampling (SAS). A Personal Air Sampler is a portable device specifically designed for the estimation of intake by an individual worker from a measurement of concentration of activity in air in the breathing zone of the worker. A sampling head containing a filter is worn on the upper torso within the breathing zone. Air is drawn through the filter by a calibrated air pump carried by the worker. Ideally, sampling rates would be similar to typical breathing rates for a worker (~1.2 $m^3$ $h^{-1}$). However, sampling rates of current devices are only about 1/5$^{th}$ 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

difficulties in assessing intakes from PAS measurements were considered by Whicker (2004). Breathing zone measurements can vary significantly as they can be affected by measurement conditions such as orientation of the sampler with respect to source, on which lapel (right or left) the sampler is worn, design of the air sampling head, particle size, local air velocities and directions, and sharp gradients in and around the breathing zone of workers.

(263) Britcher and Strong (1994) reviewed the use of PAS as part of the internal dosimetry monitoring programmes for the Calder Hall reactors and the Sellafield nuclear fuel reprocessing facility in the U.K. It was concluded that samplers can be used to obtain satisfactory estimates of intake for groups of workers. However, for individuals, the correlation between assessments using PAS and biological samples was poor and the authors cast doubt on the adequacy of PAS for estimating annual intakes of individual employees at the levels of exposure encountered in operational environments. The authors also questioned whether, for environmental monitoring, PAS offered any advantages over static air sampling programmes. The same lack of correlation between PAS and bioassay sample-based intake estimates was also seen for known acute exposures (Britcher *et al*, 1998).

(264) A uranium exposure study was conducted by Eckerman and Kerr (1999) to determine the correlation between uranium intakes predicted by PASs and intakes predicted by bioassay at the Y12 uranium enrichment plant in Oak Ridge, USA. This study concluded that there was poor correlation between the two measurements.

(265) Static air samplers are commonly used to monitor workplace conditions, but can underestimate concentrations in air in the breathing zone of a worker. Marshall and Stevens (1980) reported that PAS:SAS air concentration ratios can vary from less than 1 up to 50, depending on the nature of the work. Britcher and Strong ( 1994) concluded from their review of monitoring data for Magnox plant workers in the U.K. that intakes assessed from PAS data were about an order of magnitude greater than those implied by SAS data. SAS devices, however, can provide useful information on radionuclide composition, and on particle size, if used with a size analyzer such as a cascade impactor.

(266) Overall, the use of PASs and SASs can be an important part of a comprehensive workplace monitoring programme and is able to provide an early indication of risk of exposure. Experience of the use of PASs and SASs indicates that body activity measurements and/or excreta analysis are to be preferred for the assessment of individual intakes of airborne radionuclides and doses.

(267) However, for some transuranic radionuclides, body activity measurements and urine analysis can only quantify exposures sufficiently reliably above a few mSv unless sensitive mass spectrometric techniques for the analysis of bioassay samples are available. For the detection of lower exposures, a combination of monitoring methods is then likely to be needed, which could include air sampling and faecal analysis.

# 5 MONITORING PROGRAMMES

## 5.1 Introduction

(268) The design and management of monitoring programmes is considered in this chapter. It is recommended 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.

(269) In general, the assignment of an internal exposure monitoring programme to an individual should be based on the likelihood that the individual could receive an intake of radioactive material exceeding a predetermined level, as a result of normal operations or in the event of an accident. The use of individual monitoring for workers whose effective doses from annual intakes could exceed 1 mSv is common practice in many organisations, although it may not be required by legislation.

(270) It is important to consider both the monitoring programme design and the dose assessment process as integral parts of the overall radiation protection programme. An appropriately designed monitoring programme should provide the data necessary to enable a dose assessment to meet the specified need; even the most sophisticated dose assessment calculations cannot compensate for inadequate monitoring data.

(271) Where assessed doses could be significant, there is much to be gained from using a combination of different monitoring methods (*e.g.* lung, urine, faecal monitoring and exposure monitoring in the workplace), since they provide complementary information. For instance, direct bioassay measurements provide information on deposition and retention in organs, urine measurements can provide a measure of systemic uptake, while workplace monitoring can provide information on airborne activity, particle size and chemical form.

(272) The assessment of intakes and/or doses using those measured activities (bioassay monitoring results and measurements of the workplace) may be complex and often needs professional judgment, on a case by case analysis. Responsibilities for dose assessment should only be assigned to professionals with adequate expertise and skill, acquired through appropriate education, training and practical experience.

## 5.2 General Principles for the Design of Individual Monitoring Programmes

(273) A specification for an individual monitoring programme includes the monitoring method (or methods) to be employed (*e.g.* measurement of activity in the body, in excreta samples, and exposure monitoring in the workplace), the measurement technique used (*e.g.* photon spectrometry, alpha spectrometry, mass spectrometry), monitoring intervals for routine monitoring, and measurement or sample collection times for special monitoring.

(274) Many factors need to be taken into consideration when designing an individual monitoring programme. These include the purpose of the monitoring (*e.g.* whether it is carried out to demonstrate compliance with regulatory requirements, or simply to confirm that doses are very low), local factors such as the number of workers to be monitored and the availability of particular measurement methods, and

economic factors. The main factors that determine the dosimetric performance of the monitoring programme relate to the characteristics of the material to which a worker may potentially be exposed (normally by inhalation). These are:

- the radiations emitted by the radionuclide and its progeny;
- the effective half-life of the radionuclide;
- the respiratory tract deposition characteristics of the aerosol;
- the respiratory tract and alimentary tract absorption characteristics of the material;
- the retention in the body or the excretion rate from the body as a function of the time between intake and measurement;
- any preferential deposition in particular body organs and tissues and subsequent retention in those organs;
- any significant differences between the biokinetic behaviour of a parent radionuclide and its progeny;
- the excretion pathway (*e.g.* urine, faeces);
- the technical feasibility of the measurement.

(275) The dosimetric performance of the monitoring programme may be assessed by considering the effect of these factors on the accuracy of assessed doses and on the sensitivity associated with the monitoring programme, which can be quantified in terms of the assessed minimum detectable dose (Carbaugh, 2003; Etherington *et al*, 2004a, 2004b). One approach to optimising the design of a monitoring programme is to assess how different choices for the type, number and time period of measurements affect uncertainties in assessed dose.

## 5.3 Categories of Monitoring Programmes

(276) Four categories of monitoring programmes can (generally) be defined:

*Routine monitoring* is performed where intakes by workers are probable in anytime during normal operations, or where accidental intakes could otherwise remain undetected.

*Special monitoring* is performed after actual or suspected abnormal events.

*Confirmatory monitoring* is carried out to demonstrate that working conditions are satisfactory, and that there is no need for routine individual monitoring. It could consist of occasional individual monitoring measurements.

*Task-related monitoring* is carried out to provide information about a particular operation.

(277) The four categories of monitoring are not mutually exclusive; in fact there can be considerable overlap. For example, an effective routine monitoring programme not only provides reliable data on individual worker exposures and doses, but can also be used to demonstrate that the work environment and work procedures are under satisfactory control.

### 5.3.1 Routine Monitoring

(278) Routine monitoring programmes may involve only one type of measurement or a combination of techniques, depending on the sensitivity that can be achieved. For some radionuclides, only one measurement technique is practical, *e.g.* urine monitoring for assessment of intakes of tritium. For radionuclides such as plutonium isotopes that present difficulties for both measurement and interpretation, various techniques may have to be employed. If different methods of adequate sensitivity are available, the general order of preference (highest first) in terms of accuracy of interpretation is:

- body activity measurements;
- excreta analysis;
- exposure monitoring in the workplace.

(279) These techniques are, in general, complementary and not mutually exclusive. For example, results of monitoring of the working environment (area monitoring) can provide early indication of worker exposure, and can therefore be used to trigger special bioassay monitoring, or they may provide information that assists in interpreting the results of individual monitoring, *e.g.* information on airborne activity, particle size, chemical form and solubility, and time of intake.

(280) Urine monitoring provides a measure of systemic uptake to organs and tissues after inhalation and ingestion for those elements for which urine excretion rates are sufficiently high. It can also be used to determine the fraction of activity deposited in a wound site that transfers to the systemic circulation.

(281) Caution should be exercised in using urine monitoring for materials that are absorbed relatively slowly from the respiratory tract (*i.e.* 'insoluble' materials). In these circumstances, it is usually the lung dose that makes the greatest contribution to effective dose, and uncertainties on the knowledge of the absorption characteristics of the material can result in significant errors in assessed dose. For insoluble materials, significant improvements in sensitivity can be achieved by using faecal monitoring in addition to urine monitoring. This is because significant fractions of insoluble material deposited in both the extrathoracic airways and the lungs are cleared via the gastro-intestinal tract to faeces.

(282) Interpretation of faecal monitoring data needs to take account of a number of factors that are specific to the faecal excretion pathway. Excretion of faeces is a discrete process (even though it is usually modelled using first-order kinetics), and so it is advisable to sum the amounts excreted over a 3-day period to obtain a daily excretion rate.

(283) In the workplace, individuals may be exposed to a variety of radionuclides, such as those that occur in fuel reprocessing or manufacturing plants. In such circumstances it may be feasible to use a radionuclide that is readily detectable to assess the potential for exposure to other radionuclides in the plant. For example screening for $^{144}$Ce could be used to assess the potential for exposure to actinides (Doerfel et al, 2008).

(284) The results of workplace monitoring for air contamination may sometimes be used to estimate individual intakes if individual monitoring is not feasible. However the interpretation of the results of air sampling measurements in terms of intake is subject to much greater uncertainty and bias.

(285) The probability of exposure and the likely time pattern of intake are often dependent on the tasks being performed. For example, exposures may be chronic for workers in the mining industry. On the other hand, workers in nuclear power plants are not expected to receive significant intakes except in the rare event of an accident.

(286) The required frequency of measurements in a routine monitoring programme depends upon the retention and excretion of the radionuclide and the sensitivity of the measurement techniques available. Selection of monitoring intervals should also take into account the probability of occurrence of an intake; where the risk of intake is high, the frequency of monitoring may need to be increased to reduce the uncertainty in the time of intake. The measurement technique should be selected so that uncertainties in the measured value are small in relation to the major sources of uncertainty.

(287) For situations where an acute exposure situation may be expected, Publication 78 (ICRP, 1997b) provides a simple rule that limits the possible error on the estimate of intake arising from the unknown time of exposure. Monitoring intervals are selected so that any underestimation introduced by the unknown time of intake is no more than a factor of three. In practice, this is a maximum underestimate because the actual distribution of the exposure in time is unknown. The error in assessed intake can take on both positive and negative values, depending on the probability distribution of the exposure over the monitoring interval, with the result that the mean value of any underestimate is less than a factor of three. However, if a substantial part of the intake occurs just before sampling or measurement, the intake could be overestimated by more than a factor of three. This may be particularly important in the case of excreta monitoring, since the fraction excreted each day may change rapidly with time in the period immediately following the intake.

(288) An alternative, graphical approach has been developed by Stradling *et al* (2004), which takes into account uncertainties in material-specific parameters such as those describing absorption and particle size distribution, as well as time of intake. Information on the minimum detectable amount for a particular measurement technique is used to determine a monitoring interval appropriate for the dose level of interest.

(289) When chronic exposures are expected, the monitoring programme should be chosen taking into consideration that the amount present in the body and in excreta will increase in time until equilibrium is reached. In each monitoring interval, measurements will reflect the activity accumulated in body organs as a result of chronic intakes received in earlier years. The monitoring programme should take into account the workers' assignment of duties. For certain radionuclides there may be a significant difference between measurements taken before and after the weekend, or before and after an absence from work.

### 5.3.2 Confirmatory Monitoring

(290) One method of confirming that working conditions are satisfactory (typically for annual effective doses less than 1 mSv) is to carry out occasional individual monitoring. 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; occasional measurements may be made to confirm the absence of build-up of activity within the body.

### 5.3.3 Special or Task-Related Monitoring

(291) 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 a novel or unusual situation. 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 specific information about the physical and chemical form of the contaminant.

(292) 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 in the nose can be used to confirm that an intake has occurred and to give a rough estimate of the intake. Positive nasal swabs should trigger special bioassay measurements (Guilmette *et al*, 2007).

(293) If therapeutic procedures have been applied to enhance the rate of elimination of a radionuclide from the body then special monitoring may be needed to follow its retention in the body and to provide the basis for a dose assessment. In cases where treatment has been given, care must be taken in selecting the monitoring methods because normal biokinetics of the radionuclides can be altered significantly. For example Prussian Blue enhances the faecal elimination of radioisotopes of caesium and therefore faeces bioassay, although not used routinely, should be implemented in addition to *in vivo* and urine monitoring.

(294) Following a cut or wound, some radioactive material may penetrate to subcutaneous tissue and hence be taken up by body fluids and distributed around the body. Depending upon the radionuclide(s) 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 the need for excision. If an attempt is made to remove material from the wound, measurements should be made of the activity recovered and remaining at the wound site, so as to maintain an activity balance. The excised material can also provide information on the isotopic ratios and physico-chemical composition which can inform the dose assessment. A series of further measurements may also be needed to determine any further uptake to blood and body tissues from which any additional committed effective dose can be calculated.

## 5.4 Derived Investigation Levels

(295) In many situations of potential exposure to radionuclides, it is convenient to set derived investigation levels (DIL) for the quantities that are measured in monitoring programmes, *i.e.* whole body content, organ content, daily urinary or faecal excretion, activity concentration in air. The chosen value for the DIL may be

directly related to the dose or to the intake. For example, an investigation could be based on an intake of a radionuclide that would give a committed effective dose of 1 mSv. Thus in a routine monitoring programme for a single radionuclide and with a period of T days, a DIL could be based on the body content that would give a committed dose of 1 mSv. This would be appropriate where the probability of more than one intake occurring within a year is considered to be low. Where this probability is higher, and the probability of intake through the year is considered to be uniform, the DIL could be derived from a committed effective dose of (T/365) mSv.

(296) The value corresponding to the investigation level can be obtained directly from the relevant graphs or calculated from the tables of dose per unit content in the data sets given in this report series or the accompanying CD-ROMs. The use of constraints as described in Publication 103 (ICRP, 2007) could be used as a basis for setting investigation levels. In setting such investigation levels, due attention must be given to other sources of exposure, *i.e.* other radionuclides and external irradiation. In situations where intakes and doses are known to be low and there is considerable experience of the processes being undertaken, it may be possible simply to set investigation levels for the measured quantities on the basis of experience. A measurement result in excess of the investigation level would indicate a departure from normal conditions and the need to investigate further.

## 5.5 Record Keeping and Reporting

(297) Dose record keeping is the making and keeping of individual dose records for radiation workers. It is an essential part of the process of monitoring the exposures of individuals to both external radiation and to intakes of radionuclides and for demonstrating compliance with dose limits and constraints. Formal procedures should be established for dose record keeping and these have been described in publications by the IAEA (IAEA, 1999b, 2004). The procedures and criteria for reporting individual and workplace monitoring results should be clearly specified by the management and/or regulatory authority. Information reported should be clearly identifiable and understandable and sufficient for the dose to be recalculated from the measurements at a later time if necessary. Included in the information to be documented must be a specification of the models, assumptions and computational codes used. In accident situations interim information will be needed to judge the need for management actions and the need for follow-up monitoring.

## 5.6 Quality Management System

(298) The need for a quality management system (QMS) within an overall radiation protection programme has been discussed in an ISO standard (ISO, 2006). Reference should be made to the ISO standard for a complete account, but some of the more important issues are:

- in deciding on the nature and extent of the quality assurance programme, consideration should be given to the number of workers monitored, and the magnitude and probability of exposures expected
- assumptions on factors such as radionuclide composition, inhaled particle size,

identity of chemical compounds, absorption behaviour, etc., should be verified by appropriate measurements

- reviews or audits should be conducted at appropriate times (*e.g.* when a new monitoring programme is implemented, or when a significant change to a programme is made)

(299) Laboratories should participate in national or international intercomparisons of measurements and dose assessments at appropriate intervals. Such participation enables the determination of the accuracy of measurement and dose assessment procedures, improves reliability, and facilitates harmonisation of methods.

# 6 GENERAL ASPECTS OF RETROSPECTIVE DOSE ASSESSMENT

## 6.1 Introduction

(300) The effective dose calculated for protection purposes is determined from the equivalent doses to organs and tissues of the human body, which are in turn calculated from the mean absorbed doses to those organs and tissues (Section 1.2). Effective dose provides a value which takes account of the given exposure conditions but not of the characteristics of a specific individual. In particular, the tissue weighting factors that are used to determine effective dose are selected, rounded values representing averages over many individuals of different ages and both sexes. The equivalent doses to each organ or tissue of the Reference Male and the Reference Female are averaged, and these averaged doses are each multiplied with the corresponding tissue weighting factor to determine the sex-averaged effective dose for the Reference Person (ICRP, 2007). It follows that effective dose does not provide an individual-specific dose but rather that for a Reference Person under given exposure conditions (ICRP, 2007).

(301) There are two alternative approaches that may be applied for retrospective dose assessment:

a) The calculation of the intake of a radionuclide either from direct measurements (*e.g.*, measuring the activity of radionuclides in the whole body or in specific organs and tissues by external counting) and/or from indirect measurements (*e.g.*, measuring the activity of radionuclides in urine or faeces, or exposure monitoring in the workplace). Biokinetic models are used to interpret the measurements and the effective dose is calculated from the intake using reference dose coefficients (doses per unit intake, Sv $Bq^{-1}$) recommended by ICRP or determined using ICRP's recommended methodology (ICRP, 2007).

b) Calculation of the committed effective dose directly from the measurements using functions that relate them to the time of the intake. The measurements could be of whole body or organ content, activity in 24 hour urine or faecal samples, or concentration of radionuclides in air in the workplace. For the interpretation of bioassay data, this approach requires the use of tables of 'dose per unit content' as a function of time after the intake (ICRP, 2007).

The two approaches are equivalent and should produce identical results provided the same biokinetic models, parameter values and assumptions are used.

(302) 'Dose per unit content' tables for selected radionuclides are given in this report series and on the accompanying CD-ROMs. They provide data on the committed effective dose corresponding to values of bioassay quantities measured at specified times after an acute intake of the radionuclide. A more detailed description of the data provided is given in Section 7.3. The tables provide a simple and easy-to-use tool, which should promote harmonisation in the interpretation of bioassay data.

(303) There may be some circumstances in which parameter values may be changed from the reference values in the calculation of effective dose. It is, therefore,

important to distinguish between those reference parameter values that might be changed in the calculation of effective dose under particular circumstances of exposure and those values that cannot be changed under the definition of effective dose. As effective dose applies to a reference person, individual-specific parameter values should not be changed whereas material-specific parameter values may be changed. Examples of material-specific parameters include lung-to-blood absorption parameters, alimentary tract transfer factors and aerosol parameters such as the activity median aerodynamic diameter (AMAD) of the inhaled aerosol.

(304) In the majority of cases, assessed doses are low in comparison to dose limits, and for such cases it is likely that dose assessments will make use of the recommended default values for material-specific parameters, the tabulated dose coefficients and the 'dose per unit content' tables that accompany this report series. Where assessed doses are likely to be greater, or where more than one monitoring method has been used and a number of monitoring measurements have been made, material-specific parameter values other than the recommended defaults may be used.

(305) In carrying out retrospective assessments of doses from monitoring data, the assessor may need to make assumptions about factors such as the pattern of intake and properties of the material because of lack of specific information on these factors. A European project in the EC 5th Framework Programme was established to give general guidelines for the estimation of committed dose from incorporation monitoring data (Project IDEAS). The project developed a structured approach to the interpretation of individual monitoring data (Doerfel *et al*, 2006, 2007), building on the proposals made by the ICRP Working Party on Dose Assessment (Fry *et al*, 2003). This guidance has been developed further by the European Radiation Dosimetry Group (EURADOS) (Lopez *et al*, 2011b; Marsh *et al*, 2008).

(306) In addition to this guidance, the International Organization for Standardization (ISO) has published an International Standard, ISO 27048:2011 (ISO, 2011) that specifies the minimum requirements for internal dose assessment for the monitoring of workers. The IDEAS guidelines and ISO 27048 both adopt the principle that the effort needed for dose assessment should broadly correspond to the anticipated level of exposure.

(307) In unusual cases where doses to specified individuals may substantially exceed dose limits, the committed effective dose can only provide a first approximate measure of the overall detriment. If radiation dose and risk need to be assessed in a more accurate way, further specific estimates of organ or tissue doses are necessary, especially if organ-specific risks for the specified individuals are needed. In such cases, absorbed dose to organs should be calculated and used with the most appropriate biological effectiveness and risk factor data (ICRP, 2007). This retrospective individual dose assessment should only be performed by professionals with recognised expertise, skills and practical experience. It is beyond the scope of this publication to give advice on how to perform individualized retrospective dose and risk assessments.

(308) This Chapter discusses the information that should be collected on the exposure, summarises approaches to data handling for single or multiple measurements, and discusses uncertainties associated with internal dose assessments,

including measurement uncertainties. Two types of analysis are discussed: reference evaluation and site-specific evaluation.

## 6.2 Types of Analysis

### 6.2.1 Basic evaluation with ICRP default biokinetic and dosimetric computational models

(309) For installations and tasks where the annual committed effective doses to workers from intakes of radionuclides assessed prospectively are low (not likely to exceed 1 mSv), the half-life of the radionuclides that are handled are short and the quantity of material present is limited, internal monitoring might be carried out to demonstrate compliance or may be established for other purposes. For workers in those installations, there is *generally* no need to evaluate the results of monitoring measurements using site-specific or material-specific parameters. A typical example is a nuclear medicine service. If required by the Authorities, the bioassay monitoring of the technical staff, medical doctors and nurses will be accomplished, using ICRP standard models, without the need for workplace characterization (*e.g.* the determination of AMAD). Other examples might include university or research laboratories using trace quantities of radioisotopes.

(310) For such routine operations, where a new intake has been confirmed, a reference evaluation may be carried out with the following default assumptions:

- The intake was an acute event at the mid-point of the monitoring interval.
- The exposure was via inhalation of material with an AMAD of 5 μm.
- Absorption and $f_A$ values: the absorption Type or the default specific absorption parameter values for the known material are as described in this document. If the compound is unknown, then for those elements where there is a choice of absorption Types, the Type for 'unspecified compounds' should be used.

(311) Alternatively, where site-specific or material-specific default values are available and documented, these may be used provided that they are shown to be appropriate for the process in which the worker was engaged.

(312) If the value of committed effective dose is confirmed to be less than a previous established low value (*e.g.*1 mSv), no further evaluation is necessary.

### 6.2.2 Detailed evaluation of doses

(313) At installations where workers have the potential to be exposed to doses higher than 1 mSv, or higher than the derived investigation level (*e.g.* in situations such as the loss of control of the source), information should be gathered on the physical and chemical characteristics of the inhaled or ingested radionuclide, as part of a workplace monitoring programme, and on the time and pattern of intake. This information may be used to refine the assessment and reduce uncertainties in the assessed dose. The types of information that may be used in such an assessment are discussed in section 6.4.

## 6.3 Understanding Exposure Situations

(314) Workplace information should be gathered in order to understand the exposure situations, *e.g.* radionuclides that may have been incorporated (including equilibrium assumptions for the natural series), chemical form, presumed particle size, likely time, pattern and pathway of any intake.

### 6.3.1 Time(s) and Pattern of Intake

(315) A principal source of uncertainty in the interpretation of bioassay data is the assignment of the time(s) and pattern of intake. Since the bioassay function that gives the predicted measurement depends on the time since the intake it follows that the estimate of intake will vary, depending on when it is assumed the intake took place. Consideration should be given to different possible patterns of intake, such as a single contamination event, several individual events during the monitoring period, intakes lasting a short period of time or chronic intakes.

(316) Where chronic intakes are expected, an assessment should be made as to whether the working schedule should be taken into account when selecting the time of measurement (or sampling), and interpreting the results from bioassay monitoring. For elements where a fraction of the intake is rapidly excreted, the times and duration of periods when no exposure could take place such as the weekend, days off or vacations could strongly influence the assessed intake. With the exception of short half-life radionuclides, the selection of a measurement or sampling time immediately following such a period will reduce the uncertainty in assessed intake associated with rapid excretion.

(317) For routine monitoring, when chronic exposures are not expected, it is necessary to estimate an intake from a measurement made at the end of a monitoring interval, often without knowing the time of intake.

(318) When a positive measurement appears from a routine bioassay programme, a the review of workplace monitoring data, such as airborne or surface contamination levels, can indicate a likely time for the intake to have occurred. Similarly, if other workers in the same workplace have exhibited positive routine bioassay samples, a review of the data and monitoring schedules for those individual workers will help determine the time of intake for all. Workers interviews should elucidate whether if an incident, an unusual procedure or equipment failure could have led to the intake. Follow-up bioassay should be scheduled to confirm the positive measurement. When several bioassay results are available, perhaps including different types of measurement, a comparison of these results with the intake retention fractions tables may help in narrowing the choice of the time the intake occurred.

(319) Another approach has been described by Miller *et al* (2002) in which Bayesian-based dosimetry calculations are performed using a Markov chain Monte Carlo algorithm. This method, which analyzes all available bioassay data simultaneously, determines probabilistically the number, magnitude and times for N possible intakes using a previously agreed set of biokinetic models. The Weighted Likelihood Monte Carlo Sampling (WeLMoS) method is another Bayesian technique (Puncher and Birchall, 2008). In this approach, biokinetic model parameters and times of intake are sampled from probability distributions that express the state of

knowledge about the exposure before bioassay data are obtained. Each sample is weighted by the appropriate likelihood function for a given intake to produce a quantity termed the 'weighted likelihood'. The probability of each intake and time of intake, given the observed measurement data, is calculated from the weighted likelihoods using simple numerical integration techniques. These methods, although computationally intensive, obviate the need to assume intake times when other types of circumstantial information are absent.

(320) In Publications 54 and 78 (ICRP, 1988a, 1997b), it is argued that in the absence of any information, the time of intake is equally likely to have occurred before the mid-point of the monitoring interval, than after it, and therefore suggests that in these situations, a value of $t$=T/2 should be used, *i.e.* the intake is assumed to have occurred at the mid-point of the monitoring interval. Alternative approaches have been suggested (Strom 2003; Puncher *et al*, 2006; Birchall *et al*, 2007; Marsh *et al*, 2008). However the results of these alternative approaches, in most circumstances, differ very little from the mid-point method. The mid-point method is recommended here for reference evaluations (Section 6.2.1).

### 6.3.2 Route of Intake

(321) Although intakes by inhalation alone are the most frequent in the workplace, intakes by ingestion and uptake through wounds and intact skin cannot be excluded. If the route of intake is not known and several bioassay results are available, including different types of bioassay measurements, a comparison of these results may help in determining it. In some facilities simultaneous intakes by several routes can occur.

(322) If the radionuclide activity can be assessed by direct measurements, lung counting can be used to differentiate between inhaled and ingested material. However, if this is not possible and the radionuclide is in an insoluble form, interpretation of activities excreted in faecal and urine samples in terms of intake is quite problematic. Both the ingested material and the inhaled material deposited in the upper respiratory tract will clear through the faeces in the first few days after intake. Consequently, it is important to initiate excreta sampling as soon as possible after an acute intake, continuing for an extended period. Material in the faeces after the second week will originate mainly from the respiratory tract, and so later measurements can be used to correct the earlier faecal sample measurements for this component. In the monitoring of workers chronically exposed to long-lived, insoluble radionuclides, activities in the faeces after a 15 days absence from work will mostly reflect the delayed clearance from inhaled material, which dominates the dose (IAEA, 1999, 2004). Intakes of radioactive materials through wounds may occur as a result of accidents. A summary of the wound model developed by NCRP (NCRP, 2007) is presented in Section 3.4.

### 6.3.3 Particle Size

(323) Radionuclides can become airborne through numerous processes and can be present in various physical forms such as gases, vapours, and particles with a wide range of sizes, shapes and densities. Most aerosols are composed of particles with complex shapes and varying particle sizes (NCRP, 2010). For modelling purposes in dose calculations, ICRP advises the use of the activity median aerodynamic diameter

(AMAD) which, together with the geometric standard deviation, describes the particle size distribution of the inhaled aerosol (ICRP, 2002b). The AMAD influences deposition in the respiratory tract and as a consequence the transfer of unabsorbed particles to the GI tract.

(324) The AMAD of the airborne contamination in the workplace may be characterised as part of a workplace monitoring programme. In some working environments more than one particle size distribution mode may be detected. In cases of accidental releases of material, information on the particle size distribution of the airborne fraction of the release should be obtained whenever possible. When the size distribution of the radioactive aerosol is not known, then default value of 5 μm AMAD for occupational exposures should be used (ICRP, 1994a, 2002a).

### 6.3.4 Chemical Composition

(325) The chemical form of the intake can have a significant effect on the behaviour of the radionuclide that has entered the body. Chemical forms commonly encountered in the working environment are given in subsequent reports in this series for selected radionuclides. Where there is adequate experimental data, chemical forms are assigned to one of the default absorption Types (F, M or S), and a value for the alimentary tract transfer factor, $f_A$, is assigned. In some special cases, material-specific values for the parameters describing absorption to blood are provided (Section 3.2.3).

(326) A compound might have absorption characteristics slightly or considerably different from those of the default. The interpretation of bioassay measurements is sensitive to the choice of absorption parameter values of the inhaled radioactive material. In cases of significant intakes of radionuclides, and in an accident situation, it may be necessary to obtain specific data on the chemical form of the radionuclide(s) involved to obtain a more realistic assessment of the intake and committed effective dose. However the gathering of additional source-term information takes time, and often will not be available soon after the incident/accident. The specific/reference ICRP lung absorption parameter and $f_A$ of the chemical form that most closely describes the release material should be used in the first dose calculations, following the first monitoring results. Follow-up bioassay monitoring and further investigations of the accident should be used to confirm or modify the results of the first dose calculations.

(327) In many situations the worker is exposed to several chemical forms of the same radionuclide. Workers exposed in different areas of a uranium enrichment facility, for example, might be exposed to different chemical forms of uranium. Interpretation of bioassay results, excreta results in particular, will rely heavily on the assumptions related to the contributions of the different chemical forms to these results.

### 6.3.5 Influence of Background

(328) Radionuclides from the three natural radioactive decay series and other natural and anthropogenic sources are present in all environmental media, and thus are also contained in foodstuffs, drinking water and in the air, leading to intakes by human populations. Their presence should be taken into account when interpreting bioassay

measurements. The *in vivo* detection capability and minimum detection levels of *in vivo* counting are strongly influenced by the presence of $^{40}$K in the body.

(329) Excretion data from uranium and thorium series radionuclides may need correction for dietary intakes. A 'blank' bioassay sample should be obtained from the workers, prior to the commencement of work. When not possible, bioassay samples from family members or from the population living in the same area should be taken and analyzed, to allow natural or non-occupational intakes and occupational intakes to be distinguished (Lipsztein *et al*, 2003; Lipsztein *et al*, 2001; Eckerman and Kerr, 1999). In cases of positive excreta results resulting from occupational exposures, the background values should be subtracted from the monitoring results, before dose calculations. This might not be simple, especially when dealing with faeces monitoring results. Little *et al* (2007) describe a Bayesian method to identify a typical excretion rate of uranium for each individual in the absence of occupational intakes.

(330) In addition it is important to evaluate the influence of radiopharmaceuticals that may have been administered for diagnostic or therapeutic purposes.

(331) For long lived radionuclides, bioassay monitoring results might carry the influence of intakes identified in preceding monitoring intervals. The retained activity in the body from previous intakes should be taken into account.

### 6.3.6 Special monitoring situations

(332) In many situations exposure will be to a single radionuclide or a limited number of radionuclides. For some elements, however, exposures may involve a number of isotopes with different decay properties. Uranium and plutonium illustrate the potential for exposure to complex mixtures. Various plutonium isotopes are present in the nuclear industry. Studies have shown a significant difference in isotopic behaviour of plutonium, due to differences in specific activity (Guilmette *et al*, 1992). Workers exposed to uranium are always exposed to a mix of isotopes, in different proportions depending on the enrichment level. Knowledge of the enrichment is essential for the correct interpretation of bioassay monitoring results.

(333) Special considerations apply when direct bioassay measurements of radioactive progeny are used to determine the body content of the parent radionuclide (Section 3.2.3). Significant errors can arise if it is assumed that the progeny are always in secular equilibrium. For example, the activity of $^{232}$Th in the lungs can be underestimated when determined from direct measurements of its $^{228}$Ac, $^{212}$Pb, $^{212}$Bi and $^{208}$Tl progeny. Differences in lung retention among the measured element and the radionuclide of concern contribute to the uncertainty of results. For the same reasons, activity of $^{232}$Th in the lungs can be underestimated when determined from measurements of $^{220}$Rn in breath.

(334) There are also situations when one radionuclide is used as a surrogate for another, for example for *in vivo* bioassay monitoring. One example is the determination of the level of internally deposited Pu in the lung which is often estimated on the basis of $^{241}$Am external monitoring of the chest. $^{241}$Am generally accompanies Pu in the work place or is produced in the body by decay of $^{241}$Pu. This procedure is often appropriate but depending on the solubility characteristics and isotopic composition of the aerosols, the relative clearance rates from the lung might

be different and $^{241}$Am lung results may underestimate Pu activity in the lung (*e.g.* nitrate aerosols).

## 6.4 Measurements

### 6.4.1 Data Collection and Processing

(335) Some types of measurement data may need processing before use. Examples include:

- Lung. Generally, the combined activity in lungs and thoracic lymph nodes is referred to as 'lung' activity, and it is this quantity that is calculated by internal dosimetry software. Where estimates of lung and lymph activity are given separately, they should be summed. 'Chest' measurements may also include counts from activity in liver and skeleton for radionuclides that concentrate in these tissues and their contributions will be needed to be subtracted.
- Urine and faecal samples collected over periods less than 24 hours should in general be normalized to an equivalent 24 hour value. This can be achieved by multiplying by the ratio of the reference 24 hour excretion volume or mass to the volume or mass of the sample. The reference volumes, for males and females respectively, are: for urine 1.6 litres and 1.2 litres; and for faeces 150 g and 120 g (ICRP, 2002a). For urine sampling, another widely used method is to normalise to the amount of creatinine excreted per day; 1.7 g and 1.0 g for males and females respectively (ICRP, 2002a). If the 24 hour sample is less than 500 ml for urine or less than 60 g for faeces, then it is doubtful that it has been collected over a full 24 hour period and normalization should be considered. Collection of faecal samples should preferentially cover a period of about three days, as the transit time through the alimentary tract is subject to large inter (and intra-) subject variations.

(336) For some radionuclides the collection of spot samples are sufficient for routine sampling, *e.g.* the monitoring of intakes of tritiated water.

### 6.4.2 Single Measurements, Acute Intakes

***Special monitoring***

(337) For special or task-related monitoring when the time of intake is known, the intake can be estimated from the measured results using the $m(t)$ values given in subsequent reports of this series. An $m(t)$ value is a value of a bioassay quantity measured at time t after a unit intake of a specified radionuclide, sometimes known as a retention or excretion function. If only a single measurement is made, the intake, $I$, can be determined from the measured quantity, $M$, if the contribution of previous intakes to the measured quantity $M$ is negligible.

$$I = \frac{M}{m(t)} \qquad (6.1)$$

(338) The intake should be multiplied by the dose coefficient ($e_{ij}$, for pathway i and radionuclide j) to obtain the committed effective dose E:

$$E(50) = e_{ij} \times I \qquad (6.2)$$

(339) Care must be taken to ensure that the measurement result, *M*, and *m(t)* are comparable; for example, in the case of urinalysis, the bioassay result must be expressed as the total activity in a 24 hour urine sample at the end of collection (not at analysis). Alternatively, the tabulated values of '*Dose per unit content*' for a range of radionuclides and types of materials should be used. Dose per unit content, *z(t)*, is given by:

$$z(t) = e(50) / m(t) \qquad (6.3)$$

(340) If only a single measurement is made, and the contribution of previous intakes to the measured quantity *M* is negligible, the committed effective dose *E*(50), associated with the intake, *I*, can be determined by:

$$E(50) = M \times z(t) \qquad (6.4)$$

***Routine monitoring***

(341) For routine monitoring, an intake during the monitoring period is assessed from the measurement made at the end of the monitoring interval. When the time of intake is not known (or cannot easily be determined) and a reference evaluation is being performed (Section 6.2.1), it should be assumed that the intake occurred at the mid-point of the monitoring interval of T days. For a given measured quantity, *M*, obtained at the end of the monitoring interval, the intake is:

$$I = \frac{M}{m(T/2)} \qquad (6.5)$$

where *m*(T/2) is the predicted value of the measured quantity for a unit intake assumed to have occurred at the mid-point of the monitoring interval. The dose from the intake in the monitoring interval is obtained by multiplying the intake by the dose coefficient. The assessed dose or intake can be compared with the pro-rata fraction of the dose limit or of the intake corresponding to that limit, respectively. Alternatively, the dose or intake can be compared with predetermined investigation levels.

(342) An intake in a preceding monitoring interval may influence the measurement result obtained. For a series of measurements in a routine monitoring programme, the following procedure may be followed:

- Determine the magnitude of the intake in the first monitoring interval.
- Predict the contribution to each of the subsequent measurements from this intake.
- Subtract the corresponding contributions from all subsequent data if the contributions are judged to be significant (ISO, 2011)
- Repeat above for the next monitoring interval.

(343) Alternatively, using the tables of dose per unit content, for a given measured quantity *M*, obtained at the end of the monitoring interval, the *mid-point dose E* associated with intake *I* is:

$$E = M \times z(T/2) \qquad (6.6)$$

(344) It is convenient to assume that for each monitoring interval $n$ the associated effective dose $E_n$ could be equal to 0 or positive. For a given measured quantity, $M(t_k)$, obtained at the end of the last monitoring interval $k$, the associated effective dose $E_k$ is:

$$E_k = \left( M(t_k) - \sum_{n=1}^{k-1} \frac{E_n}{z(t_k - \tau_n)} \right) z(t_k - \tau_k) \qquad (6.7)$$

where $t_k$ is the time of measurement k (end of the last monitoring interval k); $\tau_n$ and $\tau_k$ are the time at mid-points of monitoring intervals n and k, respectively. If M(tk) is below the decision threshold (ISO, 2011) or the result of background subtraction is negative, then $E_k = 0$.

### 6.4.3 Multiple Measurements

(345) Usually, the bioassay data for an intake estimate will consist of results for different measurements performed at different times, and even from different monitoring techniques, *e.g.* direct and indirect measurements.

(346) To determine the best estimate of a single intake, when the time of intake is known, it is first necessary to calculate the predicted values, $m(t_i)$, for unit intake of the measured quantities. It is then required to determine the best estimate of the intake, $I$, such that the product $I\ m(t_i)$ 'best fits' the measurement data $(t_i, M_i)$. In cases where multiple types of bioassay data sets are available, it is recommended to assess the intake and dose by fitting predicted values to the different types of measurement data simultaneously. For example, if urine and faecal data sets are available then, the intake is assessed by fitting appropriately-weighted predicted values to both data sets simultaneously (ISO, 2011; Doerfel *et al*, 2006, 2007).

(347) Numerous statistical methods for data fitting are available (IAEA, 2004a,b). The two methods that are most widely applicable are the maximum likelihood method (ISO, 2011; Doerfel *et al*, 2006) and the Bayesian approach (Miller *et al*, 2002; Puncher and Birchall, 2008). Other methods such as the mean of the point estimates and the least-squares fit can be justified on the basis of the maximum likelihood method for certain assumptions on the error associated with the data. For example, the least squares method can be derived from the maximum likelihood method if it is assumed that the uncertainty on the data can be characterised by a normal distribution. The assumed distribution (*e.g.* normal or lognormal) can have a dramatic influence on the assessed intake and dose if the model is a poor fit to the data. However, as the fit of the model to the data improves, the influence of the data uncertainties on the assessed intake and dose reduces.

### 6.4.4 Chronic Exposures

(348) The amount of activity present in the body and the amount excreted daily depend on the period of time over which the individual has been exposed. The bioassay result obtained, *e.g.* the amount present in the body, in body organs, or in excreta, will reflect the super-position of all the intakes. Intake retention functions for

chronic intakes are not given in this publication, but equilibrium values of bioassay quantities for continuous chronic exposure are provided for some radionuclides.

### 6.4.5 Influence of decorporation therapy

(349) In cases involving internal contamination, blocking, dilution, or chelating agents may be used to enhance the clearance of the activity from the body and reduce committed doses. The use of interventional techniques to enhance the body's natural elimination rate of the compound, or possibly to block the uptake of the radionuclide in sites where high uptake may occur (*e.g.*, radioiodine in the thyroid), may partially or completely invalidate the use of standardized model approaches described above to estimate the intake and dose (NCRP, 2009).

(350) The use of chelating agents such as DTPA, for example, may influence excretion rates for weeks or months after cessation of treatment.

(351) It is not feasible to give specific advice as the treatment of any bioassay data depends upon the circumstances of the exposure and the need and timescale required for the dose assessment.

### 6.4.6 Wounds

(352) Because of their nature, intakes of radionuclides resulting from contaminated cuts or wounds typically account for an appreciable proportion of high dose exposures. Radionuclides may be transferred from the wound site to blood and to other organs and tissues, and the NCRP has developed a model to describe this transfer for various chemical forms of selected radionuclides (NCRP, 2007). Coupled with an element-specific systemic biokinetic model, the NCRP model can be used to calculate committed doses to organs and tissues and committed effective doses following transfer of the radionuclide to the blood and systemic circulation, as well as to predict urinary and faecal excretion.

(353) As noted in Section 3.1, the assessment of internal contamination resulting from wounds is in practice treated on a case-by-case basis using expert judgement. In many cases, the amount of a radionuclide transferred from a wound site to blood may be assessed directly from urine bioassay data. Section 3.4 summarises the main features of the NCRP model, since this information may be of use in the interpretation of bioassay data for individual cases of wound contamination.

## 6.5 Uncertainties in Internal Dose Assessment Based on Bioassay

(354) Publication 103 (ICRP, 2007) makes the following statement with respect to the assessment of uncertainties:

> *In order to assess radiation doses, models are necessary to simulate the geometry of the external exposure, the biokinetics of incorporated radionuclides, and the human body. The reference models and necessary reference parameter values are established and selected from a range of experimental investigations and human studies through judgements. For regulatory purposes, these models and parameter values are fixed by*

*convention and are not subject to uncertainty.*

(355) It follows that there is no requirement to assess or record the uncertainty associated with an individual dose assessment performed to demonstrate compliance with regulatory requirements. Nevertheless, the assessment of uncertainties associated with a specified monitoring procedure (including the dose assessment procedure) provides important information for optimising the design of a monitoring programme (Etherington *et al*, 2004a; Etherington *et al*, 2004b; ISO, 2011). Where uncertainties in assessed effective dose are evaluated, uncertainties in material-specific model parameter values should be considered, but individual-specific model parameter values should be taken to be fixed at their reference values (Section 6.1).

(356) This section describes and discusses the important sources of uncertainty in retrospective assessments of dose. The uncertainty in an internal dose assessment based on bioassay data depends on the uncertainties associated with measurements used to determine the activity of a radionuclide *in vivo* or in a biological sample, uncertainties in the exposure scenario used to interpret the bioassay results, and uncertainties in the biokinetic and dosimetric models used to interpret the bioassay results. The exposure scenario includes factors such as the route of intake, the time pattern of intake, the specific radionuclide(s) taken into the body, and the chemical and physical form of the deposited radionuclide(s).

### 6.5.1 Uncertainties in Measurements

(357) Uncertainties in measurements of activity in the body or in biological samples have been discussed in IAEA publications (IAEA, 1996a, 2000). There are no standard procedures for indirect or direct bioassay measurements, although some examples of bioassay methods are given in these reports and elsewhere. The choice of the procedure, detector or facility will depend on the specific needs such as the nuclides of interest, minimum detectable activities, and budget. All procedures used to quantify the activity of a radionuclide are sources of both random and systematic errors. Uncertainties in measurements are typically due mainly to counting statistics, validity of the calibration procedures, possible contamination of the source or the measurement system, and random fluctuations in background. A committee of the U.S. National Council on Radiation Protection and Measurements (NCRP) has developed a comprehensive report on uncertainties in internal radiation dose assessment that addresses measurement uncertainties in great detail (NCRP, 2010).

(358) The total uncertainty associated with a measurement is generally expressed as an interval within which the value of the measure and is believed to lie with a specified level of confidence (EURACHEM/CITAC, 2000). In estimating the overall uncertainty in a measurement, it may be necessary to take each source of uncertainty and treat it separately to obtain the contribution from that source. Each of the separate contributions to uncertainty is referred to as an uncertainty component.

(359) The components of uncertainty in a quantity may be divided into two main categories referred to as Type A and Type B uncertainties (BIPM *et al*, 2010; EURACHEM/CITAC, 2000; Cox and Harris, 2004; NCRP, 2010). Essentially, a Type A component is one that is evaluated by a statistical analysis of the variability in a set of observations, and a Type B component is one that is evaluated by other

means, generally by scientific judgment using all relevant information available. In the case of a measurement of activity in the total body or in a biological sample, Type A uncertainties are generally taken as those that arise only from counting statistics and can be described by the Poisson distribution, while Type B components of uncertainty are taken as those associated with all other sources of uncertainty.

(360) Examples of Type B components for *in vitro* measurements include the quantification of the sample volume or weight; errors in dilution and pipetting; evaporation of solution in storage; stability and activity of standards used for calibration; similarity of chemical yield between tracer and radioelement of interest; blank corrections; background radionuclide excretion contributions and fluctuations; electronic stability; spectroscopy resolution and peak overlap; contamination of sample and impurities; source positioning for counting; density and shape variation from calibration model and assumptions about homogeneity in calibration (Skrable *et al*, 1994). These uncertainties apply to the measurement of activity in the sample. With excretion measurements, the activity in the sample is used to provide an estimate of the subject's average excretion rate over 24 hours for comparison with the model predictions. If the samples are collected over periods less than 24 hours then they should be normalised to an equivalent 24 hour value. This introduces additional sources of Type B uncertainty relating to biological (inter-and intra-subject) variability and sampling procedures, which may well be greater than the uncertainty in the measured sample activity. Sampling protocols can be designed to minimize the sampling uncertainty, as shown by Sun *et al* (1993) for plutonium urinalysis and Moeller and Sun (2006) for indoor radon exposure.

(361) *In vivo* measurements can be performed in different geometries (whole body measurements, and organ or site-specific measurement such as measurement over the lung, thyroid, skull, or liver, or over a wound. Each type of geometry needs specialized detector systems and calibration methods. The IAEA (1996a) and the ICRU (2003) have published reviews of direct bioassay methods that include discussions of sensitivity and accuracy of the measurements.

(362) Examples of Type B components for *in vivo* monitoring include counting geometry errors; positioning of the individual in relation to the detector and movement of the person during counting; chest wall thickness determination; differences between the phantom and the individual or organ being measured, including geometric characteristics, density, distribution of the radionuclide within the body and organ and linear attenuation coefficient; interference from radioactive material deposits in adjacent body regions; spectroscopy resolution and peak overlap; electronic stability; interference from other radionuclides; variation in background radiation; activity of the standard radionuclide used for calibration; surface external contamination of the person; interference from natural radioactive elements present in the body; and calibration source uncertainties (IAEA, 1996a; Skrable *et al*, 1994).

(363) For partial body measurements it is generally difficult to interpret the result in terms of activity in a specific organ because radiation from other regions of the body may be detected. Interpretation of such measurements requires assumptions concerning the biokinetics of the radionuclide and any radioactive progeny produced *in vivo*. An illustration using $^{241}$Am is given in the IAEA Safety Series Report on Direct Methods for Measuring Radionuclides in the Human Body (IAEA, 1996a). A

fundamental assumption made in calibrating a lung measurement system is that the deposition of radioactivity in the lung is homogeneous, but depositions rarely follow this pattern. The distribution of the particles in the lung is a function of particle size, breathing rate, and health of the subject (Kramer and Hauck, 1999; Kramer *et al*, 2000).

(364) Measurement errors associated with counting statistics (Type A uncertainties) decrease with increasing activity or with increasing counting time, whereas the Type B components of measurement uncertainty may be largely independent of the activity or the counting time. Therefore, when activity levels are low and close to the limit of detection, the total uncertainty is often dominated by the Type A component (*i.e.* by counting statistics). For radionuclides that are easily detected and present in sufficient quantity, the total uncertainty is often dominated by the Type B components (*i.e.* by uncertainties other than counting statistics).

### 6.5.2 Uncertainty in the Exposure Scenario

***Time of Intake***

(365) The uncertainty in the time pattern of intake can be the dominant source of uncertainty in the estimated dose, or it can make or little or no contribution to it. For example, if an intake is not recognised for some time after an incident and total body retention and urinary and faecal excretion rates diminish quickly, the assumed time pattern of intake could be the dominant uncertainty in the dose estimate. On the other hand, if a worker is exposed in the vicinity of an immediately recognised accidental release, or total body retention and excretion rates are fairly constant, the time pattern of intake may be a negligible source of uncertainty in the dose estimate.

(366) In the case of routine monitoring, the intake can be assigned as being at the mid-point of the monitoring interval, or the intakes corresponding to each possible intake time can be calculated and then averaged. Either method may result in a large uncertainty in the dose estimate. Puncher *et al* (2006) and Birchall *et al* (2007) argued that intakes estimated from either of these methods have a tendency to overestimate the true intake and showed that an intake obtained assuming a constant intake rate throughout the monitoring interval (*i.e.*, constant-chronic method) is an unbiased estimate of the true intake when the measurement and the excretion/retention function are accurately known or when they are uncertain but unbiased (*i.e.*, the mean of the distribution describing the uncertainty is the true value). If the uncertainties in the measurement or in the excretion/retention function are affected by a bias, the constant-chronic method produces a biased result, but the bias in the result can be eliminated by the use of appropriate adjustment factors (Birchall *et al*, 2007).

***Route of Intake***

(367) In practice one may encounter situations when the mode of intake is unknown and cannot be easily discerned on the basis of health physics records or available bioassay data. For example, it may not be known if the intake took place by inhalation only, by ingestion only, or by a combination of inhalation and ingestion. Even if it is known that a combination of inhalation and ingestion occurred it may be impossible to determine what fraction of activity was inhaled and what fraction was ingested. In

the absence of specific information, it would be appropriate to assume that intake was by inhalation for an occupational exposure. The effect of assumed route of intake on assessed doses can be large and should be investigated when assessed doses are significant.

***Source term***

(368) Assumptions regarding the source term (*i.e.* the identity of the radionuclides and their relative abundances) may represent major sources of uncertainty when monitoring does not include the measurement of all the radioisotopes present in the working environment. In many situations a worker is exposed to several isotopes of the same radionuclide, but monitoring is accomplished through the measurement of one of the isotopes. For example, lung monitoring of uranium through the measurement of $^{235}$U relies on assumptions on the level of enrichment. In other circumstances, assessments of exposure to certain radionuclides are based on the monitoring results of a progeny radionuclide in the lungs. For example, monitoring of $^{232}$Th by measurement of a progeny radionuclide relies on assumptions about the equilibrium of radionuclides in the $^{232}$Th decay chain in the material to which the worker is exposed. Also, exposure to some radionuclides may be based on measurement of a surrogate radionuclide known to be present in the working environment. For example, lung monitoring of $^{239}$Pu may be based on the measurement of $^{241}$Am, using assumptions about the fraction of $^{241}$Am, which grows from $^{241}$Pu.

(369) Information on the chemical form, or mixture of forms, of an inhaled radionuclide is needed to help determine an appropriate dissolution model for activity deposited in the lungs. The dissolution rate in the lungs can represent a major source of uncertainty in a dose assessment, particularly when dose estimates are based on excretion data alone. For example, if dose estimates are based on urinary excretion data, then the dose to lungs can sometimes be underestimated by several orders of magnitude if the material is incorrectly assumed to be highly soluble or overestimated by several orders of magnitude if the material is incorrectly assumed to have low solubility. When no direct information is available on the inhaled form of a radionuclide, a combination of urinary and faecal data and, where feasible, *in vivo* lung measurements may greatly reduce the uncertainty in dose estimates associated with the chemical form of the radionuclide.

***Particle size***

(370) The particle size can be an important source of uncertainty because it influences the assumed deposition in the respiratory tract. The urinary and faecal excretion rates depend of the particle size because the size influences the transfer of unabsorbed particles to the alimentary tract. In some working environments multimodal aerosols exist within the respirable size range.

### 6.5.3 Uncertainties in Biokinetic Models

(371) Biokinetic models are used in radiation protection to predict the transfer and bioaccumulation of a radionuclide in various organs and the rate of excretion of the

radionuclide in urine and faeces. These models are used in this document to derive dose coefficients for inhalation or ingestion or radionuclides and to provide reference rates of urinary and faecal excretion following intake of a radionuclide for use in interpretation of bioassay data.

(372) The following categorization of the main types of information used to develop biokinetic models and summary of uncertainties associated with each type of information is taken from a paper by Leggett (2001). Additional investigations of the sources and extent of uncertainties in biokinetic models for radionuclides can be found in the following papers and reports: Apostoaei *et al* 1998, Leggett *et al* 1998, 2001, 2007, 2008, Harrison *et al* 2001 2002, Bolch *et al* 2001, 2003, Skrable *et al* 2002, Likhtarev *et al* 2003, Apostoaei and Miller 2004, Sánchez 2007, Pawel *et al* 2007, NCRP, 2010.

***Uncertainties associated with the formulation (structure) of a biokinetic model***

(373) The confidence that can be placed in predictions of a biokinetic model for an element depends not only on uncertainties associated with parameter values of the model but also on uncertainties associated with the model structure. Such uncertainties may arise because the structure provides an oversimplified representation of the known processes, because unknown processes have been omitted from the model, or because part or all of the model formulation is based on mathematical convenience rather than consideration of processes. Some combination of these limitations in model structure is associated with each of the biokinetic models used in this document. These limitations hamper the assignment of meaningful uncertainty statements to the parameter values of a model because they cast doubt on the interpretation of the parameter values. For purposes of assessing the uncertainties associated with predictions of a biokinetic model for an element, it is often more illuminating to examine the range of values generated by a limited number of alternative modelling approaches than to produce large numbers of predictions based on variation of parameter values within a fixed but uncertain model structure.

***Types of information used to construct biokinetic models for elements***

(374) Regardless of the model formulation or modelling approach, a biokinetic model for an element usually is based on some combination of the following sources of information:

H1: direct information on humans, *i.e.*, quantitative measurements of the element in human subjects;

H2: observations of the behaviour of chemically similar elements in human subjects;

A1: observations of the behaviour of the element in non-human species;

A2: observations of the behaviour of one or more chemically similar elements in non-human species.

Data types H2, A1, and A2 serve as surrogates for H1, which is the preferred type of information on which to base a biokinetic model.

(375) The sources H1, H2, A1, and A2 are sometimes supplemented with various other types of information or constraints, such as quantitative physiological

information (*e.g.*, rates of bone restructuring); considerations of mass balance; predictions of theoretical models based on fundamental physical, chemical, and mathematical principles (*e.g.*, a theoretical model of deposition of inhaled particles in the different segments of the lung); experimental data derived with anatomically realistic physical models (*e.g.*, hollow casts of portions of the respiratory tract used to measure deposition of inhaled particles); and *in vitro* data (*e.g.*, dissolution of compounds in simulated lung fluid). Among these supplemental sources of information, mass balance and quantitative physiological data (P) have particularly wide use.

***Sources of uncertainty in applications of human data***

(376) It is desirable to base a biokinetic model for an element on observations of the time-dependent distribution and excretion of that element in human subjects (H1 data). Some degree of this type of direct information is available for most essential elements, as well as for some important non-essential elements, such as caesium, lead, radium, uranium, americium, and plutonium. Depending on the degree of biological realism in the model formulation, it may be possible to supplement element-specific information for human subjects with quantitative physiological information for humans on the important processes controlling the biokinetics of the element of interest. For example, in ICRP Publications 67 (1993), 69 (1995a), and 71 (1995b), long-term removal of certain radionuclides from bone volume is identified with bone turnover.

(377) Although it is the preferred type of information for purposes of model construction, H1 data often have one or more of the following limitations: small study groups, coupled with potentially large inter-subject variability in the biokinetics of an element; short observation periods, coupled with potentially large intra-subject variability; use of unhealthy subjects whose diseases may alter the biokinetics of the element; paucity of observations for women and children; collection of small, potentially non-representative samples of tissue; inaccuracies in measurement techniques; uncertainty in the pattern or level of intake of the element; atypical study conditions; and inconsistency in reported values. In some cases, inconsistency in reported values may provide some of the best evidence of the uncertain nature of the data.

(378) An important tool in the development of biokinetic models for radionuclides has been the use of reference organ contents of stable elements, as estimated from autopsy measurements on subjects chronically exposed at environmental levels or at elevated levels encountered in occupational exposures (ICRP, 1975). Such data are commonly used to adjust parameter values of biokinetic models or introduce new model components to achieve balance between reported values of intake, total-body content, and excretion of stable elements. Such balance considerations can provide useful constraints on model parameters, provided the data have been collected under carefully controlled conditions. However, such balance considerations often have been based on data from disparate sources of information and unreliable measurement techniques and in some cases may have led to erroneous models or parameter values.

(379) A confidence statement based on H1 data would reflect a variety of factors, such as the reliability of the measurement technique(s), the number and state of health

of the subjects, representativeness of the subjects and biological samples, consistency in data from different studies, knowledge concerning the level and pattern of intake, and the relevance of the information to the situation being modelled. For example, confidence in a parameter value based on H1 data would be reduced if the data were determined in a study on any of the following study populations: several seriously ill subjects with known intakes, several healthy subjects with poorly characterized intakes, or one healthy subject with known intake.

***Uncertainty in interspecies extrapolation of biokinetic data***

(380) Interspecies extrapolation of biokinetic data is based on the concept of a general biological regularity across the different species with regard to cellular structure, organ structure, and biochemistry. Mammalian species, with cell structure, organ structure, biochemistry, and body temperature regulation particularly close to those of man, are expected to provide better analogies to man that do non-mammalian species with regard to biokinetics of contaminants.

(381) Despite the broad structural, functional, and biochemical similarities among mammalian species, interspecies extrapolation of biokinetic data has proven to be an uncertain process. Similarities across species often are more of a qualitative than quantitative nature, in that two species that handle an internally deposited radionuclide in the same qualitative manner may exhibit dissimilar kinetics with regard to that substance. Moreover, there are important structural, functional, and biochemical differences among the mammalian species, including differences in specialized organs, hepatic bile formation and composition, level of biliary secretion, urine volume and acidity, the amount of fat in the body, the magnitude of absorption or secretion in various regions of the digestive tract, types of bacteria in the digestive tract, and microstructure and patterns of remodelling of bones.

(382) In general, the choice of an animal model will depend strongly on the processes and subsystems of the body thought to be most important in the biokinetics of the radionuclide in humans, because a given species may resemble humans with regard to certain processes and subsystems and not others. For example, data on monkeys or baboons may be given relatively high weight for purposes of modelling the distribution of a radionuclide in the skeleton due to the close similarities in the skeletons of non-human primates and humans. Data on dogs may be given relatively high weight for purposes of modelling the rate of loss of a radionuclide from the liver due to broad quantitative similarities between dogs and humans with regard to hepatic handling of many radionuclides.

(383) A physiologically based model provides the proper setting in which to extrapolate data from laboratory animals to man, in that it helps to focus interspecies comparisons on specific physiological processes and specific subsystems of the body for which extrapolation may be valid, even if whole-body extrapolations are invalid. Depending on the process being modelled, it may be preferable to limit attention to data for a single species or small number of species, or to appeal to average or scaled data for a collection of species.

(384) The degree of confidence that can be placed in a model value based on animal data depends on the quality and completeness of the data and the expected strength of the animal analogy for the given situation. Thus, one must consider potential

experimental and statistical problems in the data as well as the logical basis for extrapolation of those particular data to humans. Relatively high confidence might be placed in a model value based on animal data if fairly extensive interspecies comparisons have been made and include observations on the species expected to be most human-like; these comparisons suggest a strong basis for interspecies extrapolation, either because the data are species-invariant or because the physiological processes governing the biokinetics of the element in different species have been reasonably well established; the model structure allows meaningful extrapolation to man, usually on the basis of physiological processes; and such processes have been well quantified in humans (*i.e.*, the central value for humans has been reasonably well established). A fairly wide uncertainty interval is indicated if data are available only for species that frequently exhibit qualitative differences from man (*e.g.*, if data were available only for rats) or if no meaningful basis for extrapolation to man has been established with regard to the quantity of interest. Whatever the quality of the animal data, the uncertainty interval should reflect the fact that some confidence in the predictive strength of the data is lost when the data are extrapolated across species.

***Uncertainty in inter-element extrapolation of biokinetic data***

(385) Biokinetic models for elements often are constructed partly or wholly from data for chemically similar elements, on the basis of empirical evidence that chemical analogues often exhibit close physiological similarities. For example, the alkaline earth elements, calcium, strontium, barium, and radium, exhibit many physiological as well as chemical similarities (ICRP, 1993, 1995a), and the alkali metals rubidium and caesium closely follow the movement of their chemical analogue, potassium.

(386) There are, however, counterexamples to the premise that chemical analogues are also physiological analogues. For example, the alkali metals potassium and sodium share close physical and chemical similarities but exhibit diametrically opposite behaviours in the body, with potassium being primarily an intracellular element and sodium being primarily an extracellular element.

(387) Moreover, chemically similar elements that behave in a qualitatively similar fashion in the body may exhibit quite different kinetics. For example, caesium appears to follow the behaviour of potassium in the body in a qualitative sense but is distributed somewhat differently from potassium at early times after intake and exhibits a substantially longer whole-body retention time.

(388) The level of confidence that can be placed in a model value based on human data for a chemically similar element depends on the quality and completeness of the data for the analogue, as well as the expected strength of the analogy for the given situation. Whatever the quality of the data for the chemical analogue, the confidence interval should reflect the fact that some confidence in the predictive strength of the data is lost when the data are extrapolated across elements.

(389) The strength of the chemical analogy for a given element depends largely on the extent to which the chemically similar elements have also been found to be physiologically similar. That is, the analogy would be considered strong for a pair of elements if a relatively large set of experimental data indicate that these elements have essentially the same qualitative behaviour in the body and their quantitative

behaviour either is similar or differs in a predictable fashion. In view of counterexamples to the premise that chemically similar elements are necessarily physiologically similar, the chemical analogy does not provide high confidence if the elements in question have not been compared in animals or man.

(390) If a chemical analogue has been shown to be a good physiological analogue, then application of human data on the chemical analogue (H2 data) may be preferable to application of animal data on the element of interest (A1 data). For example, for purposes of constructing or evaluating a biokinetic model for americium in humans, use of quantitative human data on the physiological analogue curium seems preferable to use of the best quantitative animal data on americium. Similar statements can be made for radium and barium, rubidium and potassium, or other pairs of close physiological analogues. On the other hand, if two chemically similar elements show only broad physiological similarities, the animal analogy may be preferred to the chemical analogy, particularly if element-specific data are available for a variety of animal species (as is the case, for example, for uranium and calcium). In general, lower confidence would be placed in animal data for a chemical analogue than in animal data for the element of interest.

***Uncertainty in central estimates stemming from variability in the population***

(391) 'Uncertainty' refers here to lack of knowledge of a central value for a population, and 'variability' refers to quantitative differences between different members of a population. Although uncertainty and variability are distinct concepts, the variability in biokinetic characteristics within a population is often an important factor contributing to the uncertainty in a central estimate of a biokinetic quantity. This is because such variability complicates the problem of identifying the central tendency of these characteristics in the population due to the small number of observations generally available and the fact that subjects usually are not randomly selected.

(392) Variability in the biokinetics of radionuclides, pharmaceuticals, or chemicals in human populations appears to result from many different physiological factors or modulating host factors of an environmental nature, including age, sex, pregnancy, lactation, exercise, disease, stress, smoking, and diet. Large inter-individual biokinetic variations sometimes persist in the absence of appreciable environmental differences and suggest that these variations may be genetically controlled. In real-world situations, genetic and environmental factors may interact dynamically, producing sizable variations in the behaviour of substances taken into the human body.

### 6.5.4 Uncertainties in Dosimetric Models

(393) Dosimetric models are used to estimate the mean absorbed dose resulting from radiations emitted by nuclear transformations of radionuclides present in the body. The absorbed dose is computed for target regions (organs, tissues, or regions of tissues) considered to be radiosensitive. Radiation and tissue weighting factors are applied to the mean absorbed dose to determine the equivalent and effective dose. The weighting factors are assigned reference values and as such are not regarded as uncertain quantities. Thus, the uncertainties associated with an estimated equivalent

dose to an organ, for example, are considered to be those associated with the underlying mean absorbed dose.

(394) The physical and anatomical parameters contributing to uncertainties in the mean absorbed dose for internal emitters are:

- Energy and intensity of the nuclear and atomic radiations emitted by the radionuclide and by any radioactive progeny;
- Interaction coefficients of the emitted radiations in tissues;
- Elemental composition of the tissues of the body;
- Volume, shape, density of the organs of the body; and
- Parameters describing the spatial relationship of the source regions (regions containing the radionuclide) and the target regions (radiosensitive organs and tissues for which dose values are desired).

(395) Limitations are present in the computational model representing the anatomy and in the numerical procedures used to calculate the energy absorbed in the target tissues. The magnitudes of these uncertainties vary with radiation type, the energy of the radiation, and the specific source-target pair. The adoption of computational phantoms based upon medical imaging data (often referred to as voxel phantoms) has reduced the uncertainties associated with cross-irradiation of tissues by photon and neutron radiations to some extent by providing more realistic spatial relationships of some source and target regions (ICRP, 2009). However the absorbed dose is frequently dominated by the contributions from non-penetrating radiations. For source and target regions that cannot be resolved in the medical image data, *e.g.*, source and target regions in the respiratory and alimentary tracts and in the skeleton, uncertainties are associated with the computational models used to represent these regions.

(396) The anatomical models are static and thus do not address uncertainties in the spatial position of the organs due to breathing and posture other than reclining. Reference values for the masses and elemental composition of the organs of the body have been defined in Publication 89 (ICRP, 2002a) and used in the reference computational models of the anatomy (computational phantoms) noted above.

(397) The parameters of the dosimetric model contributing to uncertainties in the absorbed dose are those physical parameters associated with the nuclear transformation processes that determine the energy and intensity of the emitted radiation and parameters which govern the transport radiations in the body. An uncertainty less than 10% has been assigned to attenuation and absorption coefficients for photons with somewhat higher uncertainties ascribed to soft tissue stopping power values for alpha and electron particles. Improvements in the basic nuclear data have reduced the uncertainties in the physical half-lives of radionuclides and the branching fractions of decay modes. The simplified procedures used in the dosimetric calculations to address the delayed beta and gamma radiations of spontaneous fission can contribute substantial uncertainties in the mean absorbed dose in some tissues.

(398) The dosimetric calculations must associate an anatomical region (source region) with each biokinetic compartment. Many biokinetic models partition the systemic activity among a few identified organs/tissues and include a compartment referred to as ‘Other tissue’ which represents the residual. The dosimetric procedure distributes the activity in the ‘Other tissue’ compartment uniformly among all tissues not explicitly noted in the model. Substantial uncertainty may be associated with the

mean absorbed dose for tissues that are members of ‘Other tissue’. Frequently ‘Other tissue’ includes tissues assigned an explicit tissue weighting factor. For example, breast tissue is rarely if ever explicitly noted in biokinetic models and thus its mean absorbed dose is often based on its membership of ‘Other tissue’.

(399) Some uncertainties also arise in the manner in which the biokinetic models are implemented in the dosimetric calculations. The biokinetic models are presented as compartment models which in a dosimetric evaluation are further extended to include the kinetics of radioactive decay and ingrowth of radioactive progeny. A number of numerical methods are capable of solving the set of potentially large numbers (100s) of coupled differential ‘stiff’ equations that describe the kinetics, although frequently the demands of numerical accuracy have to be balanced with computational time. Compartment-model issues contributing to uncertainties in the mean absorbed dose include the assumed biokinetics of members of a decay chain (independent or shared kinetics), and the representation of ‘Other’ tissues when their anatomical identity varies among the decay chain members. (Section 3.7.2 and Annex C of Publication 71 (ICRP, 1995c)).

# 7 DATA PROVIDED FOR ELEMENTS AND RADIOISOTOPES

## 7.1 Introduction

(400) This Chapter describes the types of information provided in subsequent reports in this series for individual elements and their radioisotopes.

(401) The elements included in Part 2 are: Hydrogen (H), Carbon ( C), Phosphorus (P), Sulphur (S), Calcium (Ca), Iron (Fe), Cobalt (Co), Zinc (Zn), Strontium (Sr), Yttrium (Y), Zirconium (Zr), Niobium (Nb), Ruthenium (Ru), Antimony (Sb), Tellurium (Te), Iodine (I), Caesium (Cs), Barium (Ba), Iridium (Ir), Lead (Pb), Bismuth (Bi), Polonium (Po), Radon (Rn), Radium (Ra), Thorium (Th) and Uranium (U). Part 3 will include elements in the lanthanide series, Actinium (Ac), Protactinium (Pa) and transuranic elements. Part 4 will include Fluorine (F), Sodium (Na), Magnesium (Mg), Potassium (K), Manganese (Mn), Nickel (Ni), Selenium (Se), Molybdenum (Mo), Technetium (Tc) and Silver (Ag).

(402) Each element section provides information on chemical forms encountered in the workplace; principal radioisotopes, their physical half-lives and decay modes; reviews of data on inhalation, ingestion and systemic biokinetics; the structure and parameter values for the systemic biokinetic model; and information on the interpretation of individual monitoring data. Each section includes tables of:

- Dose coefficients (committed effective dose, Sv, per Bq intake) for inhalation of 5 μm AMAD aerosols with the default absorption Types appropriate for the element, for all relevant radioisotopes;
- Principal emissions of selected radioisotopes;
- Measurement techniques, detection limits typically achieved in a practical monitoring programme, and improved detection limits that could be achieved by suitable choice of measurement parameter values (*e.g.* sample and background count times), for selected radioisotopes;
- Committed effective dose (Sv) per unit measurement (Bq) at various times after an acute intake by inhalation of a 5 μm AMAD aerosol with the default absorption Types appropriate for the element, for selected radioisotopes;
- Bioassay data (*i.e.* whole body and/or organ retention, and daily urinary and faecal excretion, Bq per Bq intake), at various times after an acute intake by inhalation of a 5 μm AMAD aerosol with the default absorption Types appropriate for the element;
- Equilibrium values of bioassay quantities for continuous chronic intake of selected radioisotopes.

(403) Bioassay data are also presented graphically.

(404) In cases for which sufficient information is available (principally for actinide elements), lung absorption is specified for different chemical forms and dose coefficients and bioassay data are calculated accordingly.

## 7.2 Dose coefficients

(405) For inhalation, dose coefficients are calculated using the revised HRTM described in Section 3.2. Particle sizes are assumed to be log-normally distributed with an AMAD of 5 μm and geometric standard deviation $\sigma_g$ of approximately 2.5 (ICRP, 1994a, *Paragraph 170*) inhaled by a male Reference Worker at Light Work. They are assumed to have a density of 3.00 g $cm^{-3}$, and a shape factor of 1.5 (ICRP, 1994a, *Paragraph 181*).

(406) For ingestion, dose coefficients are calculated using the HATM (ICRP, 2006) with parameter values for the reference adult male, and are given for specified values of $f_A$.

(407) Extensive additional information for all relevant isotopes of each element is given on an accompanying CD-ROM, including:

- Committed equivalent dose coefficients for organs and tissues, for males and females;
- Dose coefficients for all chemical forms considered;
- Dose coefficients for inhaled aerosols with median sizes ranging from an AMTD of 0.001 μm to an AMAD of 20 μm;
- Committed doses to 7 d, 30 d, 1 and 10 years after acute intake as well as 50 years. These data illustrate the build-up of dose with time;
- Dose coefficients for intake by ingestion, with the default $f_A$ values appropriate for the element, for all relevant radioisotopes;
- Dose coefficients for radioisotopes not given in the printed reports in this series.

## 7.3 Interpretation of Individual Monitoring Data

(408) The information provided in subsequent reports in this series on the interpretation of bioassay monitoring data updates that given in Publications 54 and 78 (ICRP, 1988a, 1997b), and also includes data related to the calculation of doses per unit content. These additional data are provided to facilitate the interpretation of monitoring data.

(409) Methods of individual monitoring are given with typical detection limits that can readily be achieved. Comments on preferred measurement techniques and the adequacy of the detection limits are given where appropriate.

(410) Predicted values of the measured quantity (body content, organ content, or daily excretion) are given as a function, $m(t)$, at time t after an acute intake of 1 Bq.

(411) If only a single measurement is made, the intake, $I$, can be determined from the measured quantity, $M$, by:

$$I = \frac{M}{m(t)} \qquad (7.1)$$

The intake can be multiplied by the dose coefficient, $e(50)$, to give the committed effective dose, $E(50)$. Hence:

$$E(50) = I \times e(50) = M \times e(50)/m(t) \quad \ldots\ldots\ldots\ldots (7.2)$$

If the time of the intake during a monitoring period is unknown, the intake is generally assumed to have occurred at the mid-point of the period (Section 6.3).

(412) Dose per unit content, *z(t)*, represents the committed effective dose per unit organ (body) radionuclide content or per unit radionuclide content in the 24 hour excreta sample at time *t* after an acute intake. The use of *z*(t) simplifies the dose evaluation to a single step, instead of the traditional method of first applying the retention or excretion function *m(t)* to calculate the intake (equation 7.1), and then the dose coefficient *e(50)* to calculate the resulting effective dose (equation 7.2).

$$E(50) = M \times z(t) \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (7.3)$$

(413) Values of dose per unit content, *z(t)*, are provided to allow a more straightforward assessment of committed dose from bioassay measurements without the need to first determine the intake. For measurements of activity in body tissues and excreta, predicted values of committed effective dose are tabulated for various times after radionuclide intake following inhalation, ingestion, entry through wounds or uptake to blood.

(414) Graphs of predicted activity of the radionuclide in selected body tissues, urine (daily excretion) and faeces (daily excretion), at various times after intake, are given for an acute intake of 1 Bq of the radionuclide (unit intake). These values correspond to *m*(t). Figures are given for intakes by inhalation, ingestion, and direct transfer to blood. Data are given in the form of fractional activity related to the intake, *i.e.* Bq per Bq intake for retention and daily excretion. One exception to this is for intake of tritiated water where data are given in Bq $l^{-1}$ per Bq intake since this is directly related to the dose rate.

(415) Data are given for time periods up to $10^4$ days after intake or until the fractional activity is less than $10^{-10}$ of the intake.

(416) For each radionuclide, the monitoring periods have been selected (as in Publication 78, paragraph 91) for intake by inhalation for all absorption Types so that any underestimation introduced by an unknown time of intake is no more than a factor of three when an acute intake in the middle of the monitoring interval is assumed. The frequency of monitoring, determined using the models that have been applied, is determined both by the behaviour of the radionuclide in the body and its physical half-life. Within any workplace, the probability of occurrence of an intake should also be taken into account.

(417) The accompanying CD-ROM gives extensive additional information for all relevant isotopes of each element, including:

- Committed effective dose (Sv) per unit measurement (Bq) for an acute intake by inhalation of aerosols with median sizes ranging from an AMTD of 0.001 µm to an AMAD of 20 µm;
- Committed effective dose (Sv) per unit measurement (Bq) for an acute intake by ingestion, with default $f_A$ values appropriate for the element;
- Bioassay data (*i.e.* whole body and/or organ retention, and daily urinary and faecal excretion, Bq per Bq intake), for an acute intake by inhalation of aerosols with median sizes ranging from an AMTD of 0.001 µm to an AMAD

of 20 μm;

- Similar bioassay data for an acute intake by ingestion;
- Figures giving measured activity content per unit dose (Bq $Sv^{-1}$) in selected body tissues, urine (daily excretion) or faeces (daily excretion), at various times after intake by inhalation or ingestion. These values correspond to $0.001/z(t)$. These data can also be used to facilitate decisions about the design of monitoring programmes and the extent of the assessment required, as described in Chapter 5.

## 7.4 Quality Assurance

(418) The Commission attaches particular importance to quality assurance. The Task Group of Committee 2 on Dose Calculations arranged for the quantities given in this series of reports to be calculated independently at different laboratories, using different computer codes. Any discrepancies in these calculations were investigated and resolved before publication.

# REFERENCES


Anderson, M., Philipson, K., Svartengren, M. and Camner, P., 1995. Human deposition and clearance of 6 μm particles inhaled with an extremely low flow rate. Exp. Lung Res. 21, 187–195.

Ansoborlo, E., Boulard, D., LeGuen, B., 1997. Particle size distribution of uranium aerosols in the French nuclear fuel cycle. Radioprotection 32, 319–330.

Ansoborlo, E., Prat, O., Moisy, P., *et al*., 2006. Actinide speciation in relation to biological processes. Biochimie 88 (11), 1605-1618.

Apostoaei, A.I., Lewis, C.J., Hammonds, J.H., Hoffman, F.O., 1998. Uncertainties in doses from ingestion of Cs-137, Sr-90, Co-60, Ru-106, and I-131. Health Phys. 74, S14-S15.

Apostoaei, A. I., Miller, L F., 2004. Uncertainties in dose coefficients from ingestion of $^{131}$I, $^{137}$Cs, and $^{90}$Sr. Health Phys. 86, 460-482.

Bahadori, A.A., Johnson, P.B., Jokisch, D.W., Eckerman, K.F., Bolch, W.E., 2011. Response functions for computing absorbed dose to skeletal tissues from neutron irradiation. Phys Med Biol 56, 6873-6897.

Berkovski, V., Bonchuk, Y., Ratia, G., 2003a. Dose per unit content functions: a robust tool for the interpretation of bioassay data. Radiat. Prot. Dosim. 105(1-4), 399-402.

Berkovski, V., Eckerman, K.F., Phipps, A.W., Noβke, D., 2003b. Dosimetry of radioiodine for embryo and fetus. Radiat. Prot. Dosim. 105(1-4), 265-268.

Bihl, D.E., Lynch, T.P., Carbaugh, E.H., Sula, M.J., 1988a. Problems with Detection of Intakes of Very Insoluble Plutonium. Presented at the Thirty Fourth Annual Conference on Bioassay, Analytical, and Environmental Radiochemistry, Las Vegas, Nevada, October 17-21, 1988. PNL-SA-15981. National Technical Information Service, Springfield, Virginia.

Bihl, D.E., Carbaugh, E.H., Sula, M.J., Aldridge, T.L., 1988b. Human data supporting a super Class Y form of plutonium (abstract). Health Phys. 54(Suppl. 1), S4.

Bihl, D.E., Lynch, T.P., Carbaugh, E.H., and Sula, M.J., 1988c. Methods to Improve Routine Bioassay Monitoring for Freshly Separated, Poorly Transported Plutonium. PNL-6695, Pacific Northwest Laboratory Richland, Washington. National Technical Information Service, Springfield, Virginia.

BIPM, IEC, IFCC, ISO, IUPAC, IUPAP, OIML (2010). Guide to the Expression of Uncertainty in Measurement. JGCM 100:2008. First edition 2008, corrected version 2010.

Birchall, A., Puncher, M., Marsh, J.W., 2007. Avoiding biased estimates of dose when nothing is known about the time of intake. Radiat. Prot. Dosim. 127, 343-346.

Bolch, W.E., Farfan, E.B., Huh, C.H., *et al*., 2001. Influences of parameter uncertainties within the ICRP-66 respiratory tract model: Particle deposition. Health Phys. 81, 378-394.

Bolch, W.E., Huston, T.E., Farfan, E.B., Vernetson, W.G., Bolch, W.E., 2003. Influences of parameter uncertainties within the ICRP-66 respiratory tract model: Particle clearance. Health Phys. 84, 421-435.

5146 Bolch, W.E., Eckerman, K.F., Sgouros, Thomas, S.R. (2009). MIRD Pamphlet No.
5147 21: A generalized schema for radiopharmaceutical dosimetry - Standardization
5148 of Nomenclature. J Nucl Med 50, 477-484.
5149 Britcher, A.R., Strong, R., 1994. Personal air sampling – a technique for the
5150 assessment of chronic low level exposure?. Radiat. Prot. Dosim. 53, 59-62.
5151 Britcher, A.R., Battersby, W.P., Peace, M.S., 1998. The practical application of
5152 models for assessing intakes of radionuclides by workers. Radiat. Protect.
5153 Dosim. 79, 71-74.
5154 Camner, P., Anderson, M., Philipson, K., *et al*., 1997. Human bronchiolar deposition
5155 and retention of 6-, 8- and 10-µm particles, Exp. Lung Res. 23, 517-535.
5156 Carbaugh. E.H., La Bone, T.R., 2003. Two case studies of highly insoluble plutonium
5157 inhalation with implications for bioassay. Radiat. Prot. Dosim. 105, 133-138.
5158 Carbaugh, E.H., 2003. Minimum Detectable Dose as a Measure of Bioassay
5159 Programme Capability. Radiat. Prot. Dosim. 105, 391-394.
5160 Cox, M.G., Harris, P.M., 2004. Best Practice Guide No. 6. Uncertainty evaluation.
5161 Technical report, National Physical Laboratory, Teddington, UK. Available
5162 for download from the SSfM website at www.npl.co.uk.
5163 Coombs, M.A., Cuddihy, R.G. 1983. Emanation of $^{232}$U daughter products from
5164 submicrometer particles of uranium oxide and thorium dioxide by nuclear
5165 recoil and inert gas diffusion. J. Aerosol Sci. 14, 75-86.
5166 Crawford Brown, D.J., Wilson, J., 1984. Observations on very long term removal of
5167 uranium compounds. Health Phys. 47, 443-446.
5168 Cristy, M., 1980. Mathematical phantoms representing children of various ages for
5169 use in estimates of internal dose. Oak Ridge National Laboratory Report
5170 ORNL/NUREG/TM-367.
5171 Cristy, M., Eckerman, K.F., 1987. Specific absorbed fractions of energy at various
5172 ages for internal photon sources. Oak Ridge National Laboratory Report
5173 ORNL/NUREG/TM-8381, Vol. 1-7.
5174 Davis, K., Marsh, J.W., Gerondal, M., *et al*., 2007. Assessment of intakes and doses
5175 to workers followed for 15 years after accidental inhalation of $^{60}$Co. Health
5176 Phys. 92, 332–344.
5177 Doerfel, H., Andrasi, A., Bailey, M.R., *et al*., 2006. General Guidelines for the
5178 Estimation of Committed Effective Dose from Incorporation Monitoring Data
5179 (Project IDEAS – EU Contract No. FIKR-CT2001-00160), Research Centre
5180 Karlsruhe. Research Report FZKA 7243, Karlsruhe. ISSN 0947-8620.
5181 Doerfel, H., Andrasi, A., Bailey, M.R., *et al*., 2007. General guidelines for the
5182 assessment of internal dose from monitoring data: Progress of the IDEAS
5183 Project. European Workshop on Individual Monitoring of Ionising Radiation.
5184 11-15 April, 2005. Vienna, Austria. Radiat. Prot. Dosim. 125, 19-22.
5185 Doerfel, H., Andrasi, A., Bailey, M.R., *et al*., 2008 Internal Dosimetry : The science
5186 and art of internal dose assessment. In: proceed. 12th International Congress
5187 of the International Radiation Protection Association. IRPA12 RC-6: 1-63;..
5188 Dorrian, M.D., Bailey, M.R., 1995. Particle size distribution of radioactive aerosols
5189 measured in the workplace. Radiat. Prot. Dosim. 60, 119–133.
5190 Duke, K., 1998. Use of the urinary excretion of creatinine in plutonium in urine
5191 bioassay. Radiat. Prot. Dosim. 79, 125-128.

5192 Eckerman, K.F., 1994. Dosimetric methodology of the ICRP. In: Internal Radiation
5193 Dosimetry (edited by O.G. Raabe). Medical Physics Publishing, Wisconsin.
5194 pp239-270.
5195 Eckerman, K.F., Westfal., RJ, Ryman, JC., Cristy, M., 1994. Availability of nuclear
5196 decay data in electronic form, including beta spectra not previously published.
5197 Health Phys. 67, 338-45.
5198 Eckerman, K.F., Kerr, G.D., 1999. Y12 uranium exposure study. Oak Ridge, TN: Oak
5199 Ridge National laboratory ORNL/TM-1999-114.
5200 Elliot, N.L., Bickel, G.A., Linauskas, S.H., Paterson, L.M., 2006. Determination of
5201 femtogram quantities of $^{239}$Pu and $^{240}$Pu in bioassay samples by thermal
5202 ionization mass spectrometry. J. Radioanal. Nucl. Chem. 267, 637-650.
5203 Endo, A., Yamaguchi, Y., Eckerman, K.F., 2003. Development and assessment of a
5204 new radioactive decay database use for dosimetry calculation. Radiat. Prot.
5205 Dosim. 105, 565-9.
5206 Endo, A., Yamaguchi, Y., Eckerman, K.F., 2004. Nuclear decay for dosimetry
5207 calculation. Revised Data of ICRP Publication 38 *Japanese Atomic Energy*
5208 *Research Institute (JAERI) Report*.
5209 Epker, B.N., Frost, H.M., 1965a. Correlation of bone resorption and formation with
5210 the physical behavior of loaded bone. J Dental Research 44, 33-41.
5211 Epker, B.N., Frost, H.M., 1965b. The direction of transverse drift of actively forming
5212 osteons in human rib cortex. J Bone and Joint Surgery 47, 1211-1215.
5213 http://www.jbjs.org/data/Journals/JBJS/460/1211.pdf
5214 Etherington. G., Cossonnet, C., Franck, D., *et al*., 2004a. Optimisation of Monitoring
5215 for Internal Exposure (OMINEX). Chilton, NRPB-W60.
5216 Etherington, G., Ansoborlo, E., Bérard, P., *et al*., 2004b. OMINEX: Development of
5217 Guidance on Monitoring for Internal Exposure. Proceedings on the 11th IRPA
5218 International Congress, Madrid, 23-28 May 2004 (published on CD-ROM).
5219 EURACHEM/CITAC, (2000). EURACHEM/CITAC Guide, Quantifying Uncertainty
5220 in Analytical Measurement, 2nd Ed., EURACHEM, Berlin.
5221 http://www.eurachem.org/guides/pdf/QUAM2000-1.pdf
5222 European Union, 1996. Council of the European Union: Council Directive on laying
5223 down the Basic Safety Standards for the protection of the health of workers
5224 and the general public against the dangers arising from ionizing radiation.
5225 Official. J. Eur. Community 39, No. L, 159.
5226 Falk, R., Philipson, K., Svartengren, M., *et al*., 1997. Clearance of particles from
5227 small ciliated airways. Exp. Lung Res. 23, 495-515.
5228 Falk, R., Philipson, K., Svartengren, M., 1999. Assessment of long-term bronchiolar
5229 clearance of particles from measurements of lung retention and theoretical
5230 estimates of regional deposition. Exp. Lung Res. 25, 495-516.
5231 Foster, P.P., 1991. Study of a plutonium oxide fuel inhalation case. Radiat. Prot.
5232 Dosim. 38, 141-146.
5233 Franck, D., Borissov, N., de Carlan, L., *et al*., 2003. Application of Monte Carlo
5234 calculations to calibration of anthropomorphic phantoms used for activity
5235 assessment of actinides in lungs. Radiat. Prot. Dosim. 105, 403-408.
5236 Frost, H. M., (1986). Intermediary Organization of the Skeleton, Volumes I & II. CRC
5237 Press, Boca Raton, FL, USA.

5238 Fry, F.A., 1976. Long term retention of americium-241 following accidental
5239 inhalation. Health Phys. 31, 13-20.
5240 Fry, F.A., Lipsztein, J.L., Birchall, A., 2003. The ICRP working party on bioassay
5241 interpretation. Radiat. Prot. Dosim. 105, 297–302.
5242 Gómez-Ros, J.M., Moraleda, M., López, M.A., Navarro, T. and Navarro, J.F. Monte
5243 Carlo based voxel phantoms for *in vivo* internal dosimetry, Radiat. Prot.
5244 Dosim. 125, 161-165.
5245 Gössner, W., 2003. Target cells in internal dosimetry. Radiat. Prot. Dosim. 40, 245-
5246 257.
5247 Gregoratto, D., Bailey M.R., Marsh J.W., 2010. Modelling particle retention in the
5248 alveolar–interstitial region of the lungs. J. Radiol. Prot. 30, 491-512.
5249 Griffith, R.V., Anderson, S.L., Dean, P.N., *et al*., 1986. Tissue-equivalent torso
5250 phantom for calibration of transuranic nuclide counting facilities. Lawrence
5251 Livermore National Laboratory, Preprint UCRL-93776, Livermore, CA, USA.
5252 http://www.osti.gov/bridge/servlets/purl/5796207-idwX6y/5796207.pdf
5253 Griffith, W.C., Cuddihy, R.G., Hoover, M.D., Stalnaker, N.D., 1980. Simulation of
5254 the retention and dosimetry of $^{232}$U and its daughters after inhalation of $ThO_2$
5255 and $UO_2$ particles. In: Pulmonary Toxicology of Respirable Particles,
5256 Proceedings of the 19th Annual Hanford Life Sciences Symposium, Richland,
5257 Washington, October 1979, National Technical Information Service,
5258 Springfield, VA, USA, pp 193-208.
5259 Guilmette, R.A., Hickman, A.W., Griffith, W.C., 1992. The effect of isotope on the
5260 dosimetry of inhaled plutonium oxide. In: Proceedings of the 8th International
5261 Congress of the International Radiation Protection Association, IRPA,
5262 Montreal, 1992, pp. 900–903.
5263 http://www.irpa.net/irpa8/cdrom/VOL.1/M1_221A.PDF
5264 Guilmette, R.A., Bertelli, L., Miller, G., Little, T.T., 2007. Technical basis for using
5265 nose swab bioassay data for early internal dose assessment. Radiat. Prot.
5266 Dosim. 127(1-4), 356-360.
5267 Gunnarsson, M. Stenström, K., Leide-Svegborn, S., *et al*., 2003. Biokinetics and
5268 radiation dosimetry for patients undergoing a glycerol tri-[14C]oleate fat
5269 malabsorption test. Appl. Radiat. Isot. 58, 517-526.
5270 Gupton, E.D., Brown, P.E., 1972. Chest clearance of inhaled cobalt-60 oxide. Health
5271 Phys. 23, 767-769.
5272 Harrison, J.D., Leggett, R.W., Nosske, D., *et al*., 2001. Reliability of the ICRP's dose
5273 coefficients for members of the public II. Uncertainties in the absorption of
5274 ingested radionuclides and the effect on dose estimates. Radiat. Prot. Dosim.
5275 95(4), 295-308.
5276 Hough, M., Johnson, P.B., Rajon, D., Jokisch, D., Lee, C., Bolch, W.E., 2011. An
5277 image-based skeletal dosimetry model for the ICRP reference adult male –
5278 internal electron sources. Phys Med Biol 56, 2309-2346.
5279 Hunt, J.G., Dantas, B.M., Lourenco, M.C., Azeredo, A.M.G., 2003. Voxel phantoms
5280 and Monte Carlo methods applied to *in vivo* measurements for simultaneous
5281 $^{241}$Am contamination in four body regions. Radiat. Prot. Dosim. 105, 549-552.

IAEA, 1996a. Basic Safety Standards for Direct Methods for Measuring Radionuclides in the Human Body. Safety Series 114. International Atomic Energy Agency, Vienna, Austria.

IAEA, 1999a. Occupational radiation protection. Safety Guide RS-G-1.1. International Atomic Energy Agency, Vienna, Austria.

IAEA, 1999b. Occupational exposure due to intakes of radionuclides. Safety Guide RS-G-1.2. International Atomic Energy Agency, Vienna, Austria.

IAEA, 2000. Basic Safety Standards for Indirect Methods for Assessing Intakes of Radionuclides Causing Occupational Exposure. Safety Series 18. International Atomic Energy Agency, Vienna.

IAEA, 2004a. Basic Safety Standards for Methods for Assessing Occupational Radiation Doses Due to Intakes of Radionuclides. Safety Series 37. International Atomic Energy Agency, Vienna, Austria.

IAEA, 2004b. Assessment and treatment of external and internal radionuclide contamination. TECDOC-869. International Atomic Energy Agency, Vienna, Austria.

IAEA, 2007. Intercomparison Exercise on Internal Dose Assessment, Final report of a Joint IAEA-IDEAS Project. TECDOC-1568. International Atomic Energy Agency, Vienna, Austria.

ICRP, 1973. Alkaline earth metabolism in adult man. ICRP Publication 20. Pergamon Press, Oxford, UK.

ICRP, 1975. Report on the Task Group on Reference Man. ICRP Publication 23. Pergamon Press, Oxford, UK.

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

ICRP, 1979. Limits for intake of radionuclides by workers. ICRP Publication 30, Part 1. Ann. ICRP 2(3/4).

ICRP, 1980. Limits for intakes of radionuclides by workers. ICRP Publication 30, Part 2. Ann. ICRP 4(3/4).

ICRP, 1981. Limits for intakes of radionuclides by workers. ICRP Publication 30, Part 3. Ann. ICRP 6 (2/3).

ICRP, 1983. Radionuclide transformations: energy and intensity of emissions. ICRP Publication 38. Ann. ICRP 11-13.

ICRP, 1986. The metabolism of plutonium and related elements. ICRP Publication 48, Ann. ICRP 16 (2/3).

ICRP, 1988a. Individual monitoring for intakes of radionuclides by workers: design and interpretation. ICRP Publication 54, Ann. ICRP 19 (1-3).

ICRP, 1988b. Limits for intakes of radionuclides by workers: An Addendum. ICRP Publication 30, Part 4. Ann. ICRP 19 (4).

ICRP, 1989. Age-dependent doses to members of the public from intake of radionuclides. ICRP Publication 56, Part 1. Ann. ICRP 20 (2).

ICRP, 1991. 1990 Recommendations of the ICRP. ICRP Publication 60. Ann. ICRP 21 (1-3).

ICRP, 1993a. Protection against radon-222 at home and work. ICRP Publication 65. Ann. ICRP 23 (2).

ICRP, 1993b. Age-dependent doses to members of the public from intake of radionuclides: Part 2, Ingestion dose coefficients. ICRP Publication 67. Ann. ICRP 23 (3/4).

ICRP, 1994a. Human respiratory tract model for radiological protection. ICRP Publication 66. Ann. ICRP 24 (1-3).

ICRP, 1994b. Dose coefficients for intake of radionuclides by workers. ICRP Publication 68. Ann. ICRP 24 (4).

ICRP, 1995a. Age-dependent doses to members of the public from intake of radionuclides: Part 3, Ingestion dose coefficients. ICRP Publication 69. Ann. ICRP 25 (1).

ICRP, 1995b. Basic anatomical and physiological data for use in radiological protection: The skeleton. ICRP Publication 70. Ann ICRP 25 (2).

ICRP, 1995c. Age-dependent doses to members of the public from intake of radionuclides: Part 4, Inhalation dose coefficients. ICRP Publication 71. Ann. ICRP 25 (3-4).

ICRP, 1996. Age-dependent doses to members of the public from intake of radionuclides: Part 5 Compilation of ingestion and inhalation dose coefficients. ICRP Publication 72. Ann. ICRP 26 (1).

ICRP, 1997. Individual monitoring for internal exposure of workers – Replacement of ICRP Publication 54. ICRP Publication 78. Ann. ICRP 27 (3/4).

ICRP, 2001. Dose to the embryo and fetus from intakes of radionuclides by the mother. ICRP Publication 88. Ann. ICRP 31 (1-3).

ICRP, 2002a. Basic anatomical and physiological data for use in radiological protection: reference values. ICRP Publication 89. Ann. ICRP 32 (3-4).

ICRP, 2002b. Guide for the practical applications of the ICRP Human Respiratory Tract Model. Supporting Guidance 3. Ann. ICRP 32 (1-2).

ICRP, 2004. Doses to the infant from radionuclides ingested in mothers' milk. ICRP Publication 95, Ann. ICRP 34 (3-4).

ICRP, 2005. Basis for dosimetric quantities used in radiological protection. Committee 2 Consultation Draft. www.icrp.org (May 2004).

ICRP, 2006. Human alimentary tract model for radiological protection. ICRP Publication 100. Ann. ICRP 36 (1-2).

ICRP, 2007. The 2007 Recommendations of the International Commission on Radiological Protection. ICRP Publication 103. Ann. ICRP 37 (2-4).

ICRP, 2008. Nuclear decay data for dosimetric calculations. ICRP Publication 107. Ann. ICRP 38(3).

ICRP, 2009. Adult reference computational phantoms. ICRP Publication 110. Ann. ICRP 39 (2).

ICRP, 2010. Conversion coefficients for radiological protection quantities for external radiation exposures. ICRP Publication 116. Ann. ICRP 40(1).

ICRP Publication xxx (ICRP 2012). (The forthcoming "SAF" Publication – reference to be added).

ICRU, 2002a. Retrospective assessment of exposure to ionising radiation. ICRU Report 67. Journal of the ICRU 2 (2).

ICRU, 2002b. Absorbed-doses specification in nuclear medicine. ICRU Report 68. Journal of the ICRU 2 (1).

ICRU, 2003. Direct determination of the body content of radionuclides. ICRU Report 69. Journal of the ICRU 3 (1).

ISO, 2006. Radiation protection — Monitoring of workers occupationally exposed to a risk of internal contamination with radioactive material. ISO 205553:2006. International Organization for Standardization, Geneva, Switzerland.

ISO, 2011. Radiation protection — Dose assessment for the monitoring of workers for internal radiation exposure. ISO 27048:2011. International Organization for Standardization, Geneva, Switzerland.

Johnson, J. R., Peterman, B. F., 1984. A model to describe thoron exhalation following an inhalation exposure to thoria powders. In: Lung Modelling for Inhalation of Radioactive Materials, EUR Report 9384, Commission of the European Communities, Luxembourg (1984) pp. 193-196.

Johnson, P.B., Bahadori, A.A., Eckerman, K.F., Lee, C., Bolch, W.E., 2011. Response functions for computing absorbed dose to skeletal tissues from photon irradiation – an update. Phys Med Biol 56, 2347-2366.

Jokisch, D.W., Rajon, D.A., Patton, P.W., Bolch, W.E., 2011a. Methods for inclusion of shallow marrow and adipose tissue in pathlength-based skeletal dosimetry. Phys Med Biol 56, 2699–2713.

Jokisch, D.W., Rajon, D.A., Bolch, W.E., 2011b. An image-based skeletal dosimetry model for the ICRP reference adult male – Specific absorbed fractions for neutron-generated recoil protons. Phys Med Biol 56, 6857-6872.

Kawrakow, I., Mainegra-Hing, E., Rogers, D.W.O., et al., 2009. The EGSnrc code system: Monte Carlo simulation of electron and photon transport. PIRS Report 701, National Research Council of Canada (NRCC), Ottawa.

Kramer, G.H., Hauck, B.M., 1999. The effect of lung deposition patterns on the activity estimate obtained from a large area germanium detector lung counter. Health Phys. 77, 24-32.

Kramer, G.H., Lopez, M.A., Webb, J., 2000. A joint HML-CIEMAT-CEMRC project: testing a function to the counting efficiency of a lung counting germanium detector array to muscle-equivalent chest wall thickness and photon energy using a realistic torso phantom over an extended energy range. Radiat. Prot. Dosim. 92, 324-327.

Kramer, G.H., Hauck B.M., Chamberlain M.J., 2002. Biological half-life of iodine in adults with intact thyroid function and in athyreotic persons. Rad. Prot. Dosim. 102(2) 129-135.

Kuempel, E.D., O'Flaherty E.J., Stayner L.T., 2001. A biomathematical model of particle clearance and retention in the lungs of coal miners. Regul. Toxicol. Pharmacol. 34, 69-87.

Kvasnicka, J. 1987. Assessing dose equivalent from intensive short term U product inhalation. Health Phys. 53, 673-678.

La Bone, T.R., 1994. Evaluation of intakes of transuranics influenced by chelation therapy. In: Internal Radiation Dosimetry. O.G. Raabe, editor. (Madison, Wisconsin: Medical Physics Publishing). http://www.osti.gov/energycitations/product.biblio.jsp?osti_id=10123790

LaMont, S.P., Schick, C.R., Cable-Dunlap. P., 2005. Plutonium determination in bioassay sample using radiochemical thermal ionization mass spectrometry. J. Radioanal. Nucl. Chem. 263, 477-481.

Leggett, R.W., Bouville, A., Eckerman, K.F., 1998. Reliability of the ICRP's systemic biokinetic models. Radiat. Prot. Dosim. 79, 335–342.

Leggett, R.W., 2001. Reliability of ICRP's dose coefficients for members of the public I. Sources of uncertainty in the biokinetic models. Radiat. Prot. Dosim. 95(3).

Leggett, R.W., 2003. Reliability of ICRP's dose coefficients for members of the public III. Plutonium as a case study of uncertainties in the systemic biokinetics of radionuclides. Radiat. Prot. Dosim. 106 (2), 103–120.

Leggett, R.W., 2005.

Leggett, R. W.; Harrison, J. D.; Phipps, A., 2007. Reliability of the ICRP's dose coefficients for members of the public: IV. Basis of the human alimentary tract model and uncertainties in model predictions. Radiat. Prot. Dosim. 123, 156-170.

Leggett, R.W., Eckerman, K.F., Meck, R.A., 2008. Reliability of Current Biokinetic and Dosimetric Models for Radionuclides: A Pilot Study. Oak Ridge National Laboratory: Oak Ridge, TN. ORNL/TM-2008/131.

Leide-Svegborn, S., Stenström, K., Olofsson, M., 1999. Biokinetics and radiation doses for 14C-urea in adults and children undergoing the Helicobacter pylori breath test. Eur. J. Nucl. Med. 26, 573-580.

Likhtarev, I., Minenko, V., Khrouch, V., *et al.*, 2003. Uncertainties in thyroid dose reconstruction after Chernobyl. Radiat. Prot. Dosim. 105 (1-4), 601–608.

Lipsztein, J.L., Dias da Cunha, K.M., Azeredo, A.M.G., Julião, L., Santos, M., Melo D.R., Simões Filho, F.F.L., 2001. Exposure of workers in mineral processing industries in Brazil. J. Environ. Radioactivity 54(1), 189-99.

Lipsztein, J. L., Melo, D. R., Sousa, W., Dias da Cunha, K. M., Azeredo, A. M. G., Julião, L., Santos, M., 2003. NORM workers: a challenge for internal dosimetry programmes. Radiat. Prot. Dosim. 105(1-4), 317-20

Little, T.T., Miller, G., Guilmette, R., Bertelli, L., *et al.*, 2007. Uranium dose assessment: a Bayesian approach to the problem of dietary background**.** Radiat. Prot. Dosim. 127(1-4), 333-338.

Lopez, M.A., Broggio, D., Capello, K., *et al.*, 2011a. EURADOS intercomparison on measurements and Monte Carlo modelling for the assessment of americium in a USTUR leg phantom. Radiat. Prot. Dosim. 144, 295-299.

Lopez, M.A., Balásházy, I., Bérard, P., 2011b. EURADOS coordinated action on research, quality assurance and training of internal dose assessments. Radiat. Prot. Dosim. 144, 349-352.

Mann, J.R., Kirchner, R.A., 1967. Evaluation of lung burden following acute inhalation exposure to highly insoluble $PuO_2$. Health Phys. 13, 877-882.

Marsh, J.W., Castellani, C.M., Hurtgen, C., 2008. Internal Dose Assessments: Uncertainty studies and update of IDEAS Guidelines and Databases within CONRAD Project. Radiat. Prot. Dosim. 131, 34-39.

Marshall, M., Stevens, D.C., 1980. The purposes, methods and accuracy of sampling for airborne particulate radioactive materials. Health Phys. 39, 409–423.

Miller, G., Martz, H.F., Little, T., Guilmette, R., 2002. Using exact Poisson likelihood functions in Bayesian interpretation of counting measurements. Health Phys. 83(4), 512-518.

Miller, G., Martz, H.F., Little, T.T., Guilmette, R., 2002. Bayesian internal dosimetry calculations using Markov chain Monte Carlo. Radiat. Prot. Dosim. 98, 191-198.

Moeller, D.W., Sun, L.S., 2006. Comparison of natural background dose rates for residents of the Amargosa Valley, NV, to those in Leadville, CO, and the states of Colorado and Nevada. Health Phys. 91(4), 338-53.

Moody, J.C., Stradling, G.N., Wilson, I., *et al*., 1993. Biokinetics of plutonium in the rat after the pulmonary deposition of three nitrate bearing materials: implications for human exposure. NRPB-M427. Chilton, Didcot, UK.

NCRP, 1998. Evaluating the Reliability of Biokinetic and Dosimetric Models and parameters Used to Assess Individual Doses for Risk Assessment Purposes. NCRP Commentary 15. National Council on Radiation Protection and Measurements, Bethesda MD.

NCRP, 2007. Development of a Biokinetic Model for Radionuclide-Contaminated Wounds and Procedures for their Assessment, Dosimetry and Treatment. Report No. 156. National Council on Radiation Protection and Measurements, Bethesda MD.

NCRP, 2010. Uncertainties in Internal Radiation Dose Assessment. Report No. 164. National Council on Radiation Protection and Measurements, Bethesda MD.

Newton, D., 1977. Clearance of radioactive tantalum from the human lung after accidental inhalation. Am. J. Roentgenol. Radium Ther. Nucl. Med. 129, 327–328.

Newton, D., Rundo, J., 1971. The long term retention of inhaled cobalt-60. Health Phys. 21, 377–384.

Newton, D., Taylor, B.T., Eakins, J.D., 1983. Differential clearance of plutonium and americium oxides from the human lung. Health Phys., 44 (Suppl. 1), 431 – 439.

Niita, K., Matsuda, N., Iwamoto, Y., et al., 2010. PHITS - Particle and Heavy Ion Transport code System, Version 2.23. Japan Atomic Energy Agency, Tokai-mura, Japan.

Noßke, D., Karcher, K., 2003. Is radiation protection for the unborn child guaranteed by radiation protection for female workers? Radiat. Prot. Dosim. 105, 269-272.

ORAUT, 2007. Estimating doses for plutonium strongly retained in the Lung. ORAUT–OTIB–0049. Oak Ridge Associated Universities Team.

Parfitt, A.M., Kleerekoper, M., 1980. The divalent ion homeostatic system: Physiology and metabolism of calcium, phosphorus, magnesium and bone. In: Clinical Disorders of Fluid and Electrolyte Metabolism, 3rd ed. (Eds. Maxwell, M. and Kleeman, C.R.) McGraw Hill, New York, USA, pp. 269–398.

Pawel, D.J., Leggett, R.W., Eckerman, K. F., Nelson, C.B., 2007. Uncertainties in Cancer Risk Coefficients for Environmental Exposure to Radionuclides. An Uncertainty Analysis for Risk Coefficients Reported in Federal Guidance

Report No. 13. ORNL/TM-2006/583. Oak Ridge National Laboratory. Oak Ridge.

Pelowitz, D.B., 2008. MCNPX User's Manual, Version 2.6.0. LA-CP-07-1473, Los Alamos National Laboratory Los Alamos, NM, USA.

Philipson K., Falk R., Gustafsson J., Camner P., 1996. Long–term lung clearance of $^{195}$Au–labelled teflon particles in humans. Env. Lung Res. 22, 65–83.

Philipson, K., Falk, R., Svartengren, M., 2000. Does lung retention of inhaled particles depend on their geometric diameter? Exp. Lung Res. 26, 437-455.

Phipps, A.W., Smith, T.J., Fell, T.P., Harrison, J.D., 2001. Doses to the embryo/fetus and neonate from intakes of radionulcides by the mother. Contract Research Report to the Health and Safety Executive. CRR 397. HSE Information Services, Caerphilly.

Priest, N.D., Haines, J.W., Humphreys, J.A.M., Metivier, H., Kathren, R.L., 1992. The bone volume effect on the dosimetry of plutonium-239 and americium-241 in the skeleton of man and baboon. J. Radioanal. Nucl. Chem. 156(1), 33-53.

Price, A., 1989. Review of methods for the assessment of intake of uranium by workers at BNFL Springfields. Radiat. Prot. Dosim., 26, 35-42.

Puncher, M., Marsh, J.W., Birchall, A. 2006. Obtaining an unbiased estimate of intake in routine monitoring when the time of intake is unknown. Radiat. Prot. Dosim. 118(3), 280-289.

Puncher, M., Birchall, A., 2008. A Monte Carlo method for calculating Bayesian uncertainties in internal dosimetry. Radiat. Prot. Dosim. 132, 1-12

Raghavendran, K.V., Satbhai, P.D., Abhyankar, B., *et al*., 1978. Long term retention studies of $^{131}$I, $^{137}$Cs and $^{60}$Co in Indian workers. Health Phys. 34, 185–188.

Ramsden, D., 1976. Assessment of Plutonium in Lung for Both Chronic and Acute Exposure Conditions. In: Diagnosis and Treatment of Incorporated Radionuclides. International Atomic Energy Agency, Vienna, Austria, pp. 139-161.

Ramsden, D. 1984. A Modified Lung Model to Match Observed Lung and Urinary Data Following the Inhalation of Plutonium Oxide the Problems of Long Term Retention in the Pulmonary Lymph Nodes. In: Lung Modelling for Inhalation of Radioactive Materials. EUR 9384. (Eds. Smith, H. and Gerber, G.) CEC, Brussels, Belgium, pp. 281–286.

Ramsden, D., Bains, M.E.D., Frazer, D.C., 1978. A case study of multiple low level exposure to plutonium oxide. Health Phys. 34, 649–659.

Ronen, M., 1969. A Case of Insoluble Natural Uranium Exposure (a Two Year Follow Up Study). In: Handling of Radiation Accidents. Proceedings of a symposium on the handling of radiation accidents organized with IAEA in collaboration with the WHO and held in Vienna, Austria, May 19 23, 1969, pp. 451 457.

Rundo, J., 1965. A case of accidental inhalation of irradiated uranium. Br. J. Radiol., 38, 39-50.

Sánchez, G., 2007. Fitting bioassay data and performing uncertainty analysis with BIOKMOD. Health Phys. 92(1), 64-72.

5554 Sathyabama, N., Eappen, K.P., Mayya, Y.S., 2005. Calibration of an electrostatic
5555 chamber for thoron measurements in exhaled breath. Radiat. Prot. Dosim. 118,
5556 61-69.
5557 Saxby, W.N., Taylor, N.A., Garland, J., *et al*., 1964. A Case of Inhalation of Enriched
5558 Uranium Dust. In: Assessment of Radioactivity in Man, Volume II,
5559 International Atomic Energy Agency, Vienna, Austria, pp. 535-547.
5560 Scott, L.M., West, C.M., 1967. An evaluation of $U_3O_8$ exposure with an estimate of
5561 systemic body burden. Health Phys. 13, 21-26.
5562 Schultz, N.B., 1966. Inhalation Cases of Enriched Insoluble Uranium Oxides. In:
5563 Proceedings of First International Congress of Radiation Protection IRPA,
5564 Rome, Italy, September 5 10, 1966, Pergamon Press, Oxford, United
5565 Kingdom, pp. 1205 1213.
5566 Shah, A.P., Bolch, W.E., Rajon, D.A., Patton, P.W., Jokisch, D.W., 2005. A paired-
5567 image radiation transport (PIRT) model for skeletal dosimetry. J Nucl Med 46
5568 (2) 344-353.
5569 Skrable, K., Chabot, G., French, C.M., 1994. Estimation of intakes from repetitive
5570 bioassay measurements, in Internal Radiation Dosimetry (RAABE, O.G., Ed.).
5571 Medical Physics Publishing, Madison, WI, USA.
5572 Skrable, K., French, C.M., Chabot, G., *et al*., 2002. Variance models for estimating
5573 intakes from repetitive bioassay measurements, in Practical Applications of
5574 Internal Dosimetry (BOLCH, W.E., Ed.). Medical Phys. Publishing, Madison,
5575 WI, USA.
5576 Smith, J.R.H., Etherington G., Shutt A.L., Youngman, M.J., 2002. A study of aerosol
5577 deposition and clearance from the human nasal passage, Ann. Occup. Hyg.
5578 46(Suppl. 1) 309–313.
5579 Smith, J.R.H., Bailey, M.R., Etherington, G., *et al*., 2007. Further study of the effect
5580 of particle size on slow particle clearance from the bronchial tree. Radiat. Prot.
5581 Dosim. 127, 35-39.
5582 Smith, J.R.H, Bailey, M.R., Etherington, G., *et al*., 2008. Effect of particle size on
5583 slow particle clearance from the bronchial tree, (Exp. Lung Res. 34, 287-312.
5584 Smith, J.R.H., Bailey, M.R., Etherington, G., *et al*., 2011. An experimental study of
5585 clearance of inhaled particles from the human nose. Exp. Lung Res. 37(2),
5586 109–129.
5587 Snyder, S.F., Traub, R.J., 2010. The Livermore phantom history and supplementation.
5588 Health Phys. 98, 459-465.
5589 Stradling, N., Hodgson, A., Phipps, A.W., *et al*., 2005. Can low doses from inhaled
5590 thorium be confirmed by personal monitoring ?. Proc. 9th Int. Conf. on Health
5591 Effects of Incorporated Radionuclides (HEIR). Nov 29 - Dec 1, 2004, GSF-
5592 Report 06/05, GSF Neuherberg, pp261-268.
5593 Sun, L.C., Clinton, J.H., McDonald, J., Moorthy, A.R., Kaplan, E., Meinhold, C.B.,
5594 1993. Urine collection protocol in the Republic of the Marshall Islands.
5595 Radiat. Prot. Management, 10 64-72.
5596 Svartengren, M., Sommerer, K., Scheuch, G., 2001. Comparison of clearance of
5597 particles inhaled with bolus and extremely slow inhalation techniques. Exp.
5598 Lung Res. 27, 367-386.

5599 Takahashi, S., Patrick, G., 1987. Long-term retention of $^{133}$Ba in the rat trachea
5600 following local administration as barium sulfate particles. Radiat. Res. 110,
5601 321–328.
5602 Takahashi, S., Kubota Y., Sato H., Matsuoka, O., 1993. Retention of $^{133}$Ba in the
5603 trachea of rabbits, dogs and monkeys following local administration of
5604 $^{133}BaSO_4$ particles. Inhal. Tox. 5, 265–273.
5605 Tyler, G.R., Lister, B.A.J., 1973. The Biological Half Life of $^{144}$Ce in the Human
5606 Chest as Determined From *in vivo* Measurements Following an Accidental
5607 Inhalation. In: Proceedings of the IRPA 2nd European Congress on Radiation
5608 Protection, pp. 249–253.
5609 Webb, J.L., Gadd, M., Bronsen, F., Tench, O., 2000. An evaluation of recent lung
5610 counting technology. Radiat. Prot. Dosim. 89, 325-332.
5611 Wernli, C., Eikenberg, J., 2007. Twenty-year follow-up of a Pu/Am inhalation case.
5612 Radiat. Prot. Dosim. 125, 506-512.
5613 West, C.M., Scott, L.M., 1966. A comparison of uranium cases showing long chest
5614 burden retentions. Health Phys. 12, 1545–1555.
5615 West, C.M., Scott, L.M., 1969. Uranium cases showing long chest burden retention
5616 an updating. Health Phys. 17, 781– 791.
5617 West, C.M., Scott, L.M., Schultz, N.B., 1979. Sixteen years of uranium personnel
5618 monitoring experience – in retrospect. Health Phys. 36, 665–669.
5619 Whicker, J.L., 2004. Relationship of air sampling measurements to internal dose: a
5620 review. In: Proceedings of 37th Midyear Health Physics Society Meeting on
5621 Air Monitoring and Internal Dosimetry, Augusta, GA., USA. 8-11 February
5622 2004, 73-77. http://hps.org/meetings/midyear/abstract508.html.
5623 Youngman, M.J., Smith, J.R.H., Kovari, M., 1994. The determination of thorium lung
5624 burden by measurements of thoron in exhaled air. Radiat. Prot. Dosim. 53, 99-
5625 102.
5626 Zankl, M., Fill, U., Petoussi-Henss, N., Regulla, D. 2002. Organ dose conversion
5627 coefficients for external photon irradiation of male and female voxel models.
5628 Phys. Med. Biol. 47, 2367-85.
5629 Zankl, M., Petoussi-Henss, N., Fill, U., Regulla, D., 2003. The application of voxel
5630 phantoms to the internal dosimetry of radionuclides. Radiat. Prot. Dosim. 105,
5631 539-48.
5632 Zankl, M., Eckerman, K., Bolch, W.E., 2007. Voxel-based models representing the
5633 male and female ICRP reference adult – the skeleton *Radiat.* Prot. Dosim.
5634 127, 174-86.