

**NUREG/CR-6571
EUR 16773
SAND98-0119
Vol. 2**

Probabilistic Accident Consequence Uncertainty Analysis

Uncertainty Assessment for Internal Dosimetry

Appendices

Manuscript Completed: February 1998
Date Published: April 1998

Prepared by:

L.H.J. Goossens
Delft University of Technology
The Netherlands

F.T. Harper
Sandia National Laboratories
USA

J.D. Harrison
National Radiological Protection Board
United Kingdom

S.C. Hora
University of Hawaii at Hilo, USA

B.C.P. Kraan
Delft University of Technology
The Netherlands

R.M. Cooke
Delft University of Technology
The Netherlands

Prepared for

**Division of Systems Technology
Office of Nuclear Regulatory Research
U.S. Nuclear Regulatory Commission
Washington, DC 20555-0001
USA
NRC Job Code W6352**

**Commission of the European Communities
DG XII and XI
200, rue de la Loi
B-1049 Brussels
Belgium
EC Contract Numbers F13P-CT92-0023 and
94-ET-018**


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MASTER

Publication No. EUR 16773

Luxembourg: Office for Publications of the European Communities 1997

X11-F-6 Radiation Protection Research

ISBN 92-827-6699-3

Catalogue No. CG-NA-16-773-EN-C

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This report was written under the following contracts:

Contract No. W6352, United States Nuclear Regulatory Commission, Office of Nuclear Regulatory Research, Division of Systems Technology.

Contract No. F13P-CT-92-0023, European Commission, Directorate-General for Science, Research and Development, XII-F-6, Radiation Protection Research Action.

Contract No. 94-ET-018, European Commission, Directorate-General of Environment, Nuclear Safety and Civil Protection, XI-A-1 Radiation Protection.

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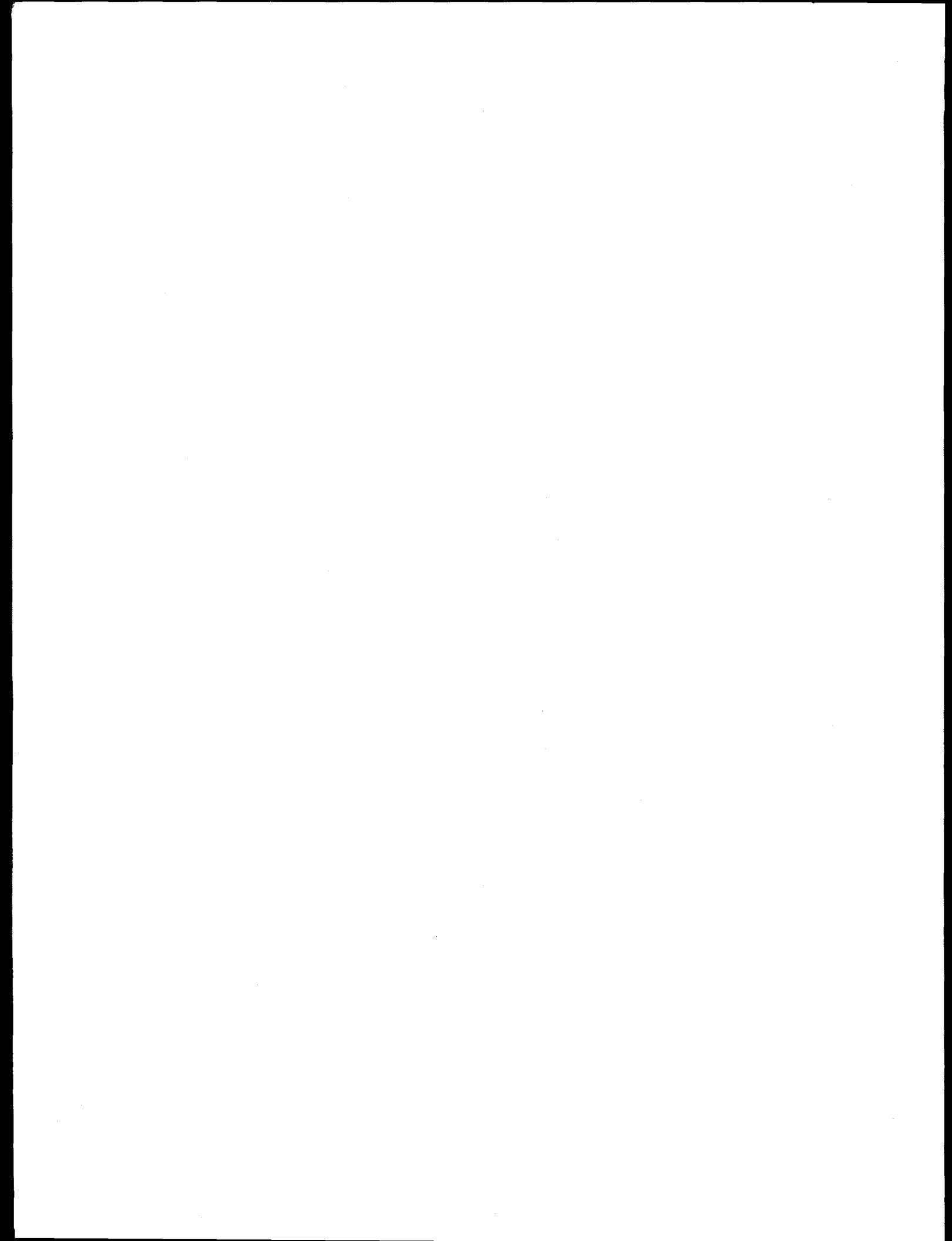
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Abstract

The development of two new probabilistic accident consequence codes, MACCS and COSYMA, was completed in 1990. These codes estimate the risks presented by nuclear installations based on postulated frequencies and magnitudes of potential accidents. In 1991, the US Nuclear Regulatory Commission (NRC) and the European Commission (EC) began a joint uncertainty analysis of the two codes. The ultimate objective was to develop credible and traceable uncertainty distributions for the input variables of the codes.

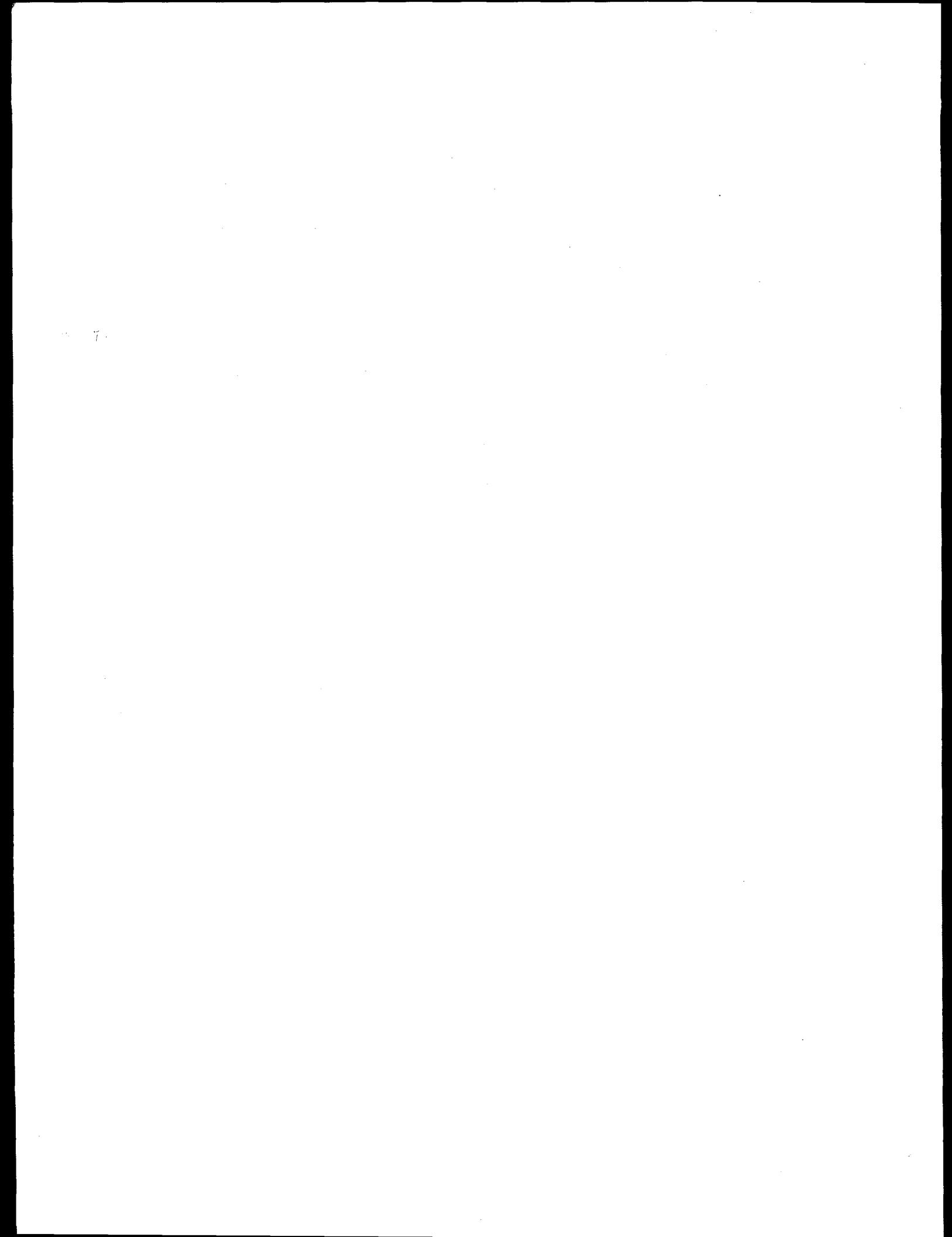
The study was formulated jointly and was limited to the current code models and to physical quantities that could be measured in experiments. An elicitation procedure was devised from previous US and EC studies with refinements based on recent experience. Elicitation questions were developed, tested, and clarified. Internationally recognized experts were selected using a common set of criteria. Probability training exercises were conducted to establish ground rules and set the initial and boundary conditions. Experts developed their distributions independently.

After the first feasibility study on atmospheric dispersion and deposition parameters, a second expert judgment exercise was carried out on food chain and external dose (calculation) parameters. A third expert judgment exercise has been carried out on early and late health effects and internal dosimetry parameters. The goal again was to develop a library of uncertainty distributions for the selected consequence parameters. Nine experts from five countries were selected for an expert panel on internal dosimetry. Their results were processed with an equal-weighting aggregation method, and the aggregated distributions will be used to determine distributions on the code input parameters of the dose per unit intake (DUPI) models used in COSYMA and for MACCS.



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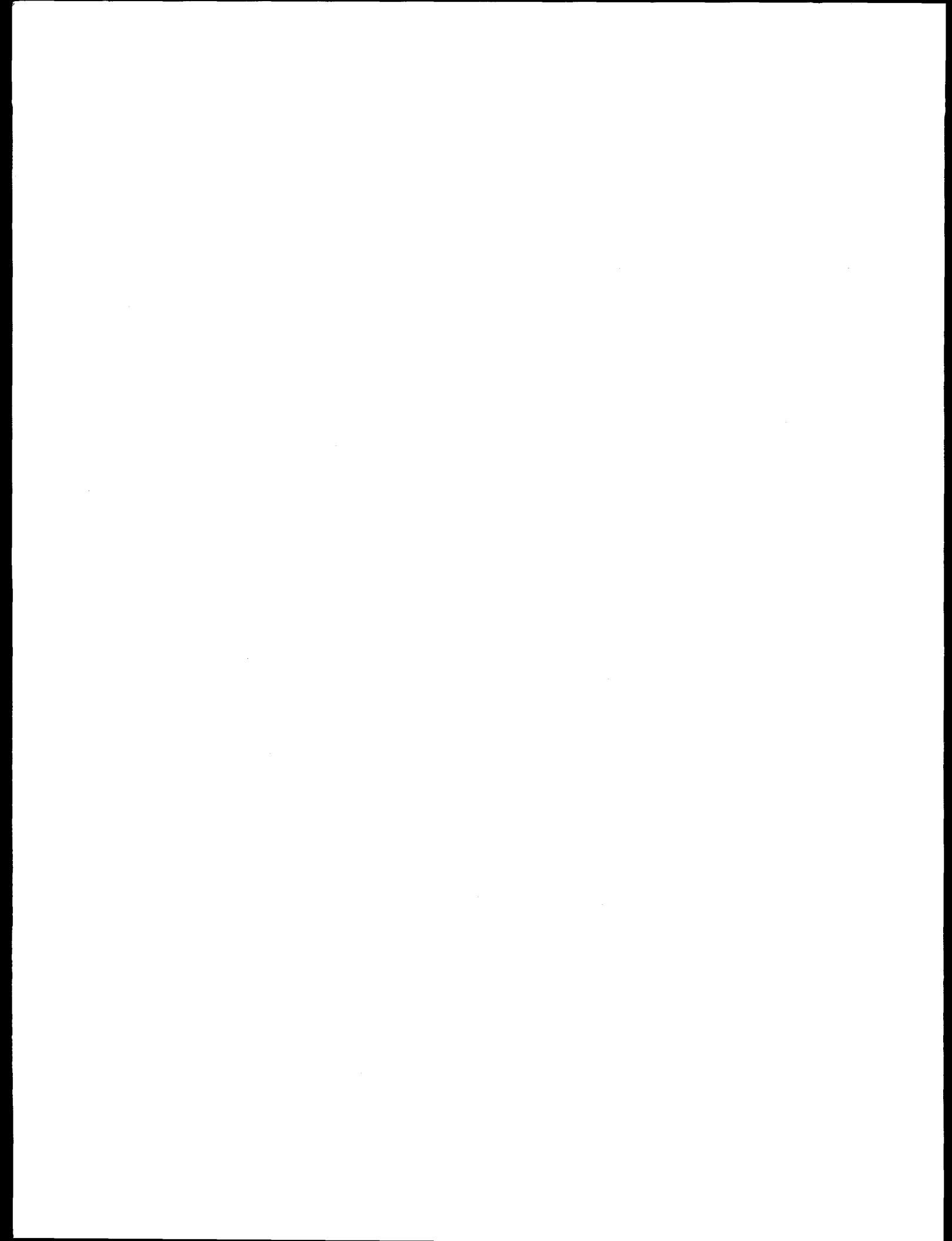
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Preface

This volume is the second of a two-volume document that summarizes a joint project conducted by the US Nuclear Regulatory Commission and the European Commission to assess uncertainties in the MACCS and COSYMA probabilistic accident consequence codes. These codes were developed primarily for estimating the risks presented by nuclear reactors based on postulated frequencies and magnitudes of potential accidents. This document reports on an ongoing project to assess uncertainty in the MACCS and COSYMA calculations for the offsite consequences of radionuclide releases by hypothetical nuclear power plant accidents. A panel of nine experts was formed to compile credible and traceable uncertainty distributions for internal dosimetry input variables that affect calculations of offsite consequences. The expert judgment elicitation procedure and its outcomes are described in these volumes. Other panels were formed to consider uncertainty in other aspects of the codes. Their results are described in companion reports.

Volume 1 contains background information and a complete description of the joint consequence uncertainty study along with a summary of the results of this aspect of the study. Volume 2 contains appendices that include (1) a summary of the MACCS and COSYMA consequence codes, (2) the elicitation questionnaires and case structures, (3) the rationales and results for the panel on internal dosimetry, (4) short biographies of the experts, and (5) the aggregated results of their responses.

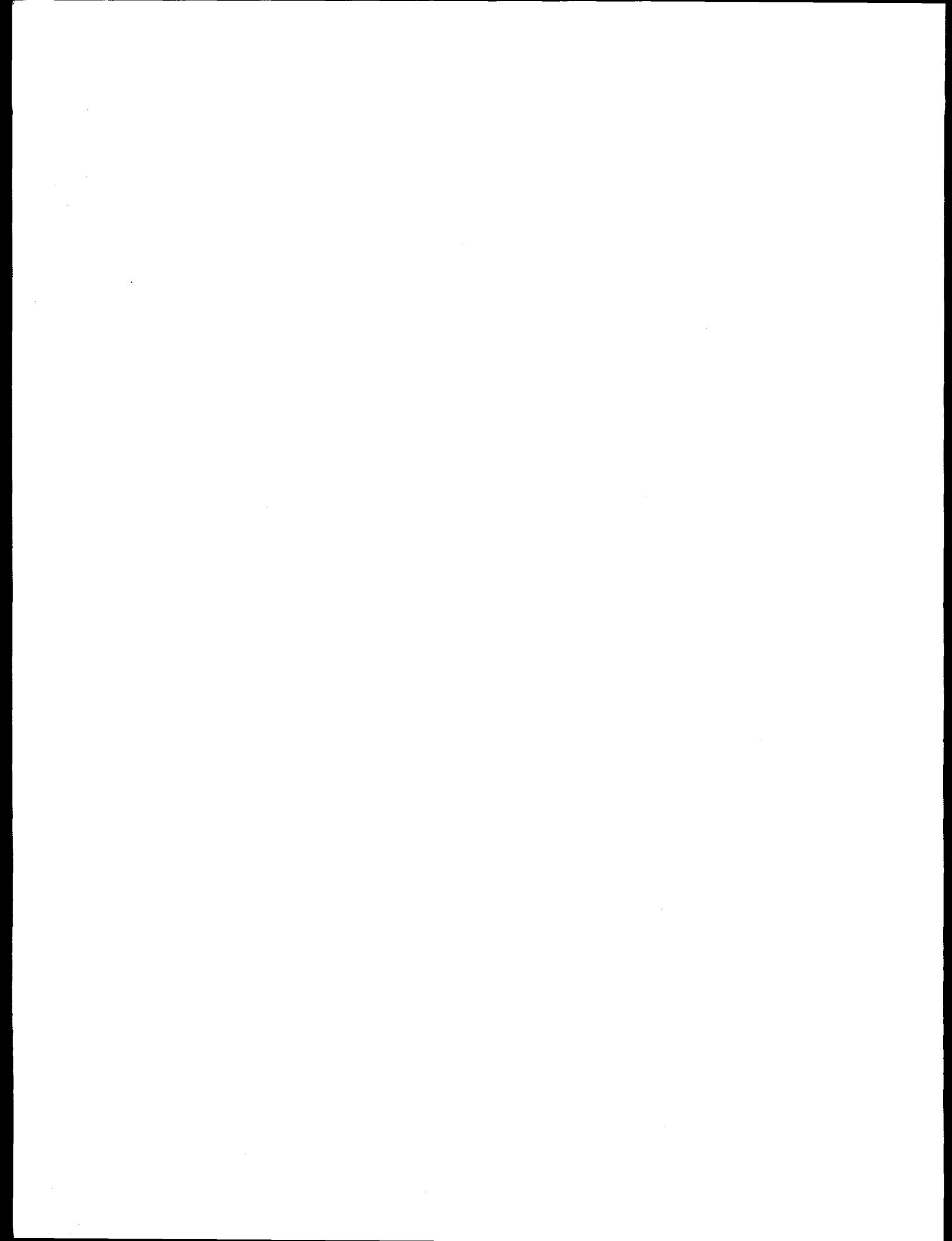


Acknowledgments

The authors would like to acknowledge all the participants in the expert judgment election process, in particular the expert panel on deposited material and external doses. While we organized the process, processed the results, and wrote and edited the report, the experts provided the technical content that is the foundation of this report. Dr. Detlof von Winterfeldt is acknowledged for his contribution as elicitor in several expert sessions. We would also like to express our thanks for the support and fruitful remarks of Dr. G. N. Kelly (EC/DG XII).

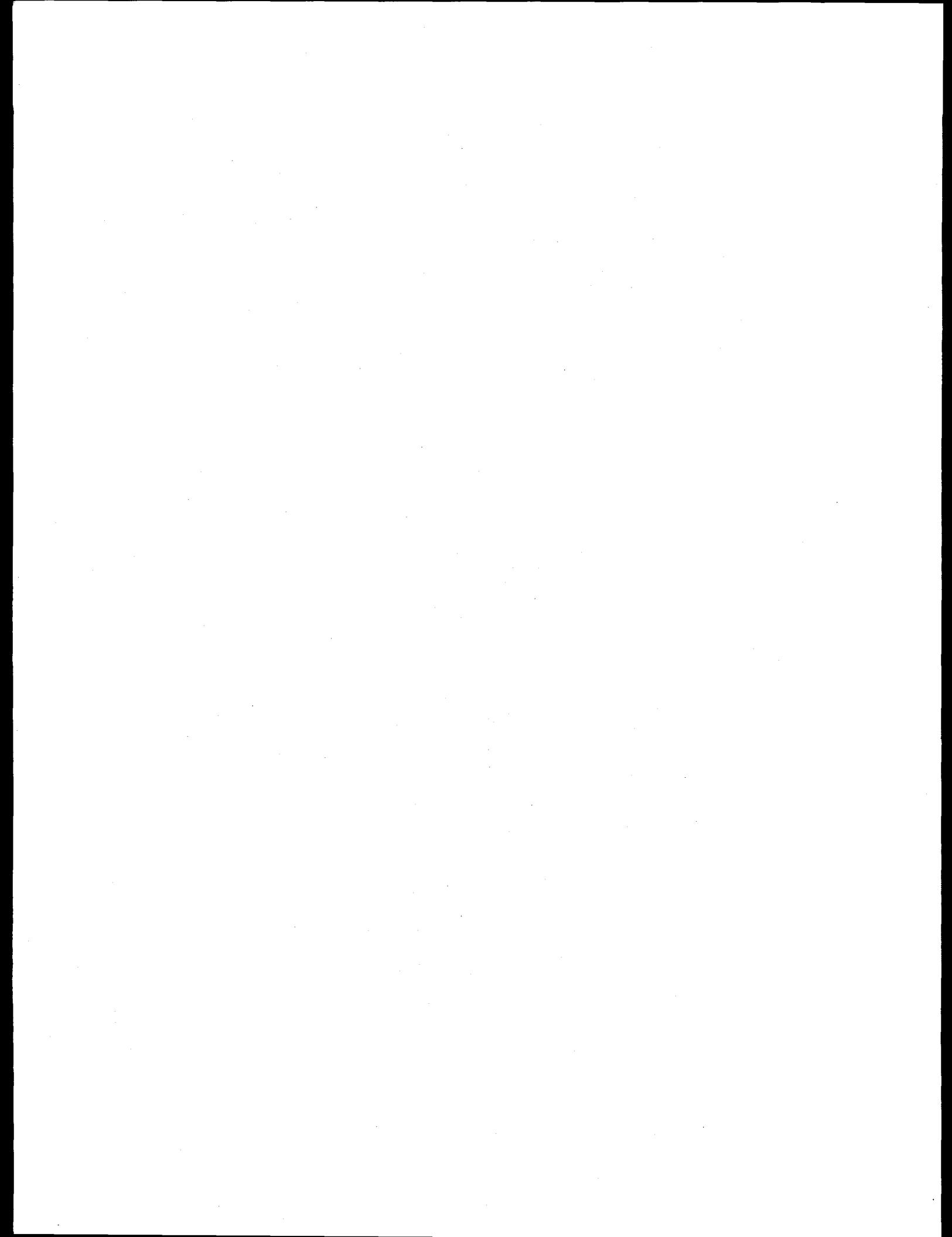
We would like to acknowledge several institutes that facilitated the collection of unpublished experimental information used in the probabilistic training and evaluation of the experts on deposited materials and related doses.

We also greatly appreciate the technical assistance of Ms. Ina Bos of Delft University of Technology, The Netherlands; the editorial help of Ruth Haas and Sally Kmetz at Tech Reps, the support of Judy Jones at Sandia National Laboratories, and the extensive assistance and guidance provided by Ms. Reeta Garber of Sandia National Laboratories in preparing this report.



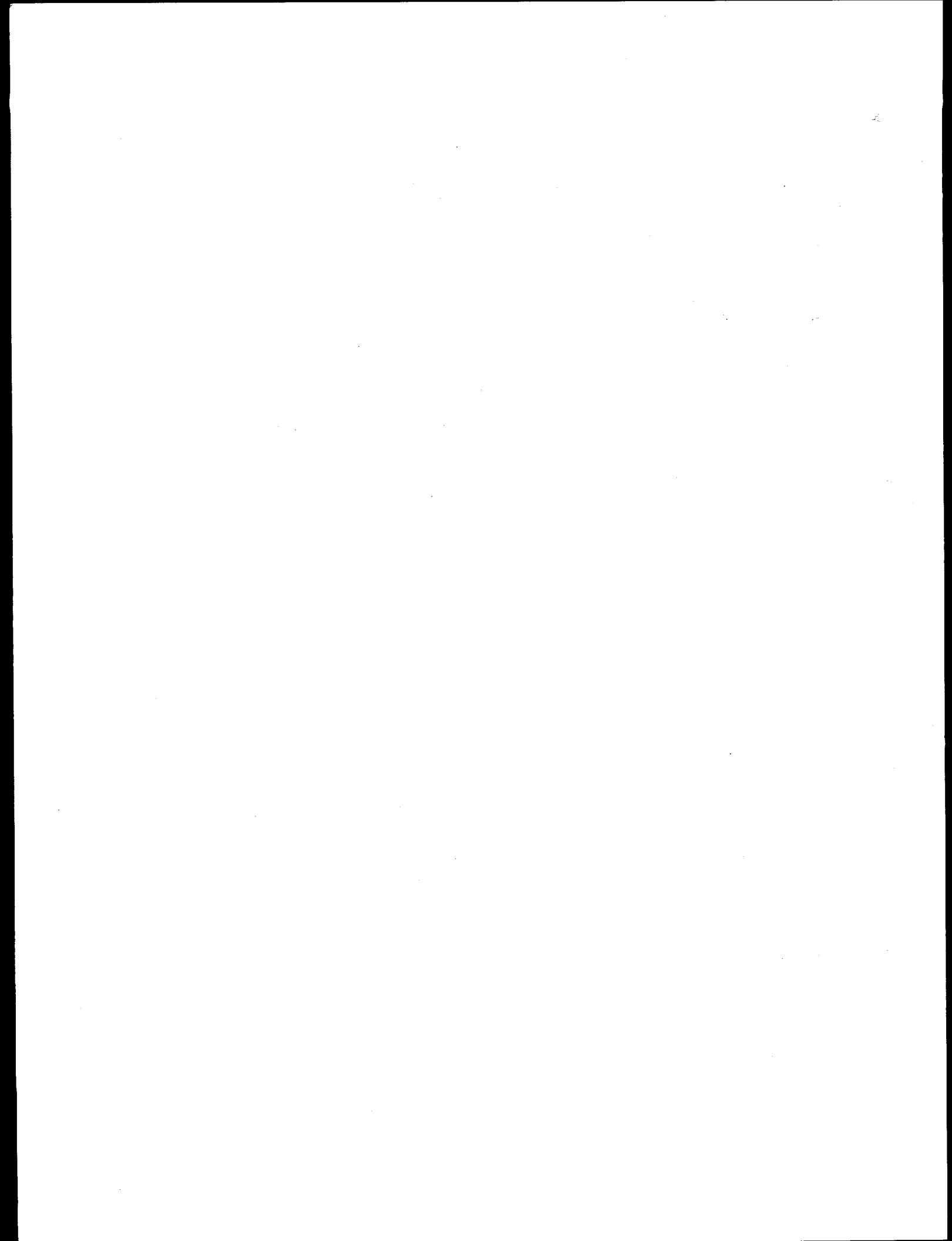
List of Acronyms

ACA	accident consequence analysis
AI	alveolar-interstitial region of the lungs
AMAD	activity median aerodynamic diameter
CDF	cumulative distribution function
COSYMA	code system from MARIA (method for assessing the radiological impact of accidents
cs	cortical surfaces of the bone
DOSAGE	code developed by BfS
DS86	Dosimetry System of 1986
EC	European Commission
ERR	excess relative risk
es	endosteal surfaces of the bone
ET	extrathoracic region of respiratory tract
LUDEP	NRPB code
MACCS	MELCOR accident consequence code system
NRC	Nuclear Regulatory Commission
NRPB	National Radiation Protection Board
TB	tracheobronchial region of lungs
UNSCEAR	United Nations Scientific Committee on the Effects of Atomic Radiation
VR	ventilation rates



APPENDIX A

Summary of the MACCS and COSYMA Consequence Codes



Summary of the MACCS and COSYMA Consequence Codes

Introduction

The information developed in this study will be used to perform uncertainty studies using the European Commission (EC) consequence code COSYMA and the US Nuclear Regulatory Commission (USNRC) code MACCS. COSYMA and MACCS model the offsite consequences of postulated severe reactor accidents that release a plume of radioactive material to the atmosphere. These codes model the transport and deposition of radioactive gases and aerosols into the environment and the potential resulting human health and economic consequences. They calculate the health effects, impact of countermeasures and economic costs of the releases. The processes considered in the calculations, and the routes of exposure following accidental releases to atmosphere, are illustrated in Figure A-1. The calculations are divided into a number of steps, illustrated in Figure A-2. COSYMA and MACCS are modular codes, with different modules addressing the different stages of the calculation. However, while Figure A-1 illustrates the steps in the calculation, the modules of the codes do not correspond exactly with the boxes shown.

The following sections give brief descriptions of the COSYMA and MACCS codes.

Brief Description of MACCS and COSYMA Dispersion and Deposition Models

COSYMA and MACCS both employ a Gaussian plume model (GPM) for atmospheric dispersion. At a given downwind distance and given atmospheric conditions, the Gaussian model predicts the time-integrated concentration at various horizontal and vertical displacements from the center-line of the plume. When the plume is not constrained by the ground or the inversion layer, the basic Gaussian plume equation for determining the concentration relative to the release rate is:

$$\frac{\chi}{Q} = \frac{1}{2\pi\sigma_y\sigma_z\bar{U}} \exp\left(-\frac{y^2}{2\sigma_y^2}\right) \exp\left(-\frac{(z-h)^2}{2\sigma_z^2}\right)$$

where:

χ = time-integrated air concentration,
 Q = the source strength,

y = the horizontal displacement relative to the plume centerline,

z = the vertical displacement,

h = the vertical height of the plume centerline,

\bar{U} = the average wind velocity, and

σ_y and σ_z are plume expansion parameters.

In MACCS and COSYMA, the plume expansion parameters, σ_y and σ_z , are modeled by the following power law:

$$\sigma_y = a_y x^{b_y}; \sigma_z = a_z x^{b_z}$$

where x = the downwind distance from the plume release point.

Currently, constant values for a_y , b_y and a_z , b_z are provided in the codes. The values for the parameters are determined by the atmospheric stability class and the roughness length of the terrain.

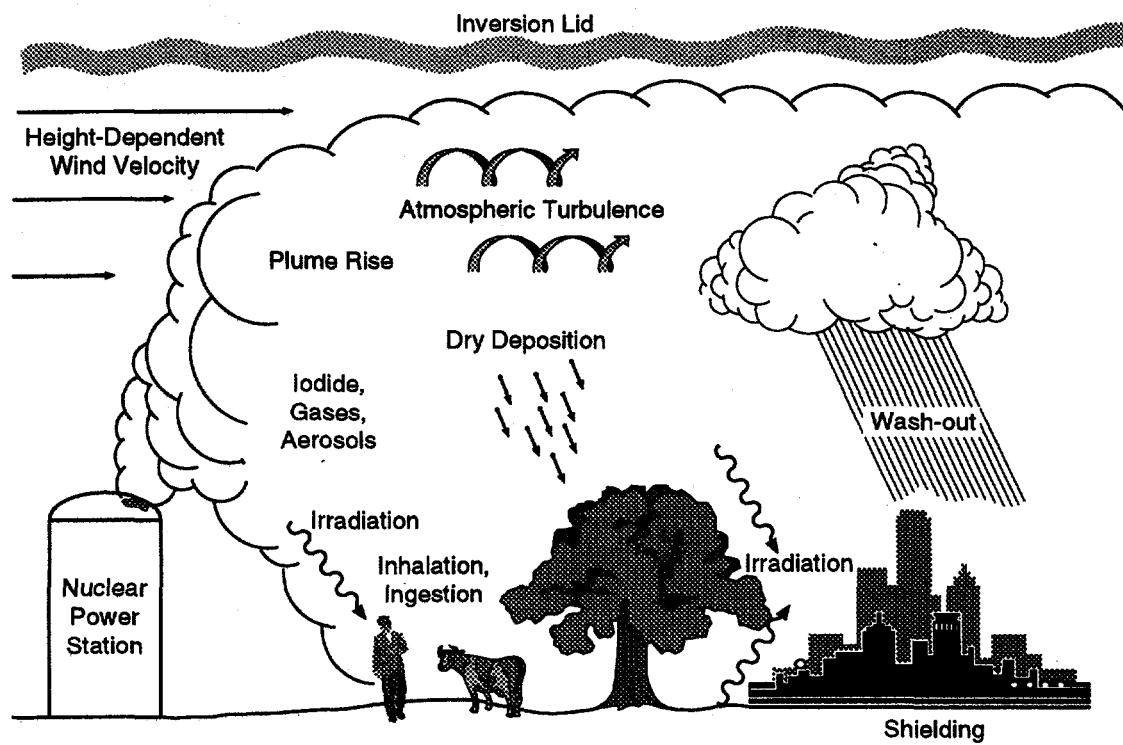
Two types of deposition are modeled in the MACCS and COSYMA codes: wet and dry. Dry deposition incorporates removal from the plume by diffusion, impaction, and settling; it is modeled through a dry deposition velocity, which is a user input. The dry deposition velocity depends on particle size; therefore, if the aerosol size distribution is divided into ranges, a dry deposition velocity must be specified for each range. The washout of radioactive material from the plume, wet deposition, is modeled as dependent on the rain intensity. The fraction of material, f_w , that remains in the plume is given by:

$$f_w = \exp\{-a I^b \Delta t\}$$

where I is the rain intensity and Δt is the amount of time the plume is exposed to the rain. The parameters a and b are the user-specified parameters that determine the amount of material washed from the plume as a result of rain intensity. Rainout, in which droplets nucleate on the aerosol particles, is not modeled.

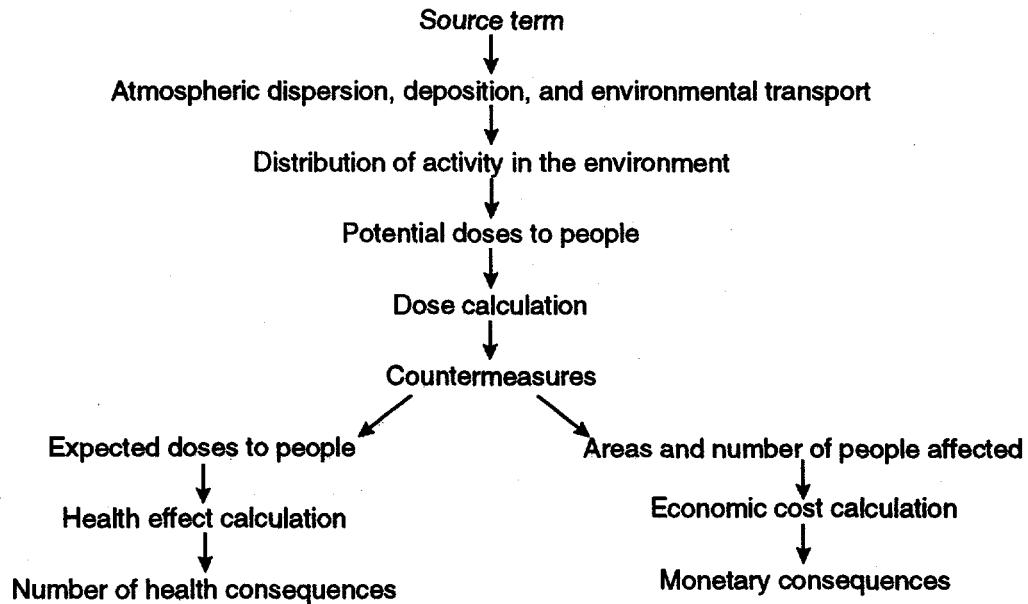
Summary of the MACCS Radiological Consequence Code

The MACCS code was originally developed under NRC sponsorship to estimate the offsite consequences of



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Figure A-1. Dispersion and deposition phenomena considered in an accident consequence analysis.



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Figure A-2. Basic features and relationships of an accident consequence analysis.

potential severe accidents at nuclear power plants by using meteorological data that vary on an hourly basis. The code models the transport and dispersion of plumes of radioactive material released from the facility to the atmosphere. As the plumes travel through the atmosphere, material may be deposited on the ground via wet and dry deposition processes. There are seven pathways through which the general population can be exposed: cloudshine, groundshine, direct inhalation, resuspension inhalation, ingestion of contaminated food, ingestion of contaminated water, and deposition on skin. Emergency response and protective action guides for both the short and long term are also considered as means for mitigating the extent of the exposures. As a final step, the economic costs that would result from the mitigative actions are estimated. Variability in consequences as a result of weather may be obtained in the form of a complementary cumulative distribution function.

MACCS is organized into three modules. The ATMOS module performs the atmospheric transport and deposition portion of the calculation. The EARLY module estimates the consequences of the accident immediately following the incident (usually within the first week), and the CHRONC module estimates the long-term consequences of the accident. A schematic representation of these modules and the input files that provide information to them is shown in Figure A-3. The following sections describe the phenomena modeled in MACCS in more detail.

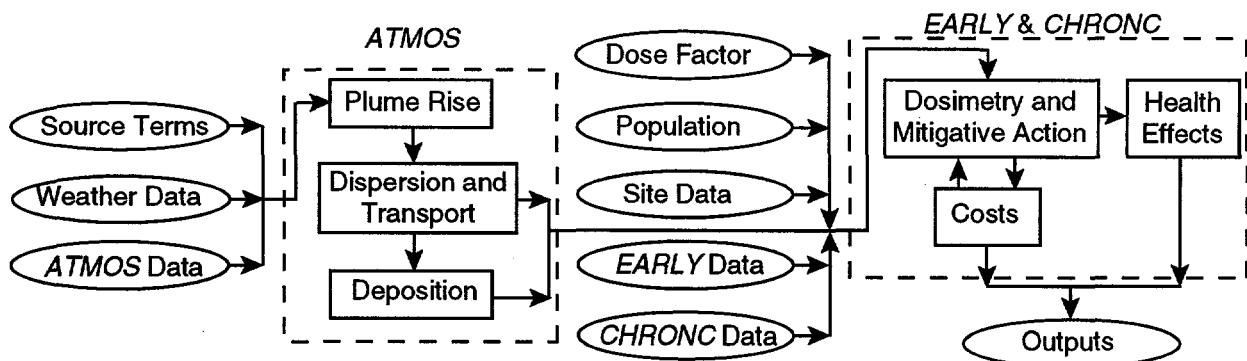
Atmospheric Dispersion and Transport

The release of radioactive materials to the atmosphere can be divided into successive plume segments, which can have different compositions, release times, durations, release

heights, and amounts of sensible heats. The plume segment lengths are determined by the product of the segment's release duration and the average windspeed during release. The initial vertical and horizontal dimensions of each plume segment are user-specified.

A lift-off criterion based on a critical windspeed determines whether or not a plume is subject to buoyant plume rise. Momentum plume rise is not modeled. If the windspeed at release is greater than the critical windspeed, plume rise is prevented.

After release from the facility, windspeed determines the rates at which plume segments transport in the downwind direction, and the wind direction at the time of release determines the direction of travel. MACCS neglects wind trajectories, as do most other consequence codes. Sixteen compass-sector population distributions are assumed to constitute a representative set of downwind exposed populations. The exposure probability of each of the 16 compass-sector population distributions is assumed to be given by the frequency with which the wind blows from the site into the sector. During transport, dispersion of the plume in the vertical and horizontal directions is estimated using an empirical model, the GPM. In this model, dispersion depends on atmospheric stability and windspeed. Horizontal dispersion of the plume segments is unconstrained. However, vertical dispersion is bounded by the ground and by the mixing layer, which are both modeled as totally reflecting layers. A single value for the mixing layer is specified by the user for each season of the year and is constant during a calculation. Eventually the vertical distribution of each plume segment becomes uniform and is so modeled.



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Figure A-3. Progression of a MACCS consequence calculation.

Deposition, Weathering, Resuspension, and Decay

As noted earlier, two types of deposition are modeled in MACCS: wet deposition and dry deposition. Weathering, resuspension, washoff, and radioactive decay decrease the deposited concentrations of radioactive materials. Radioactive decay treats only first generation daughter products.

Weather

Plume rise, dispersion, downwind transport, and deposition depend on the prevailing meteorological conditions. These conditions can be modeled as time-invariant or as varying hour-by-hour. If they are modeled as variable, the user may specify them directly or through an input file.

Dosimetry

The MACCS dosimetry model consists of three interacting processes: (1) the projection of individual exposures to radioactive contamination for each of the seven exposure pathways modeled over a user-specified time, (2) mitigation of these exposures by protective-measure actions, and (3) calculation of the actual exposures incurred after mitigation by protective-measure actions. For each exposure pathway, MACCS models the radiological burden for the pathway as reduced by the actions taken to mitigate that pathway dose. The total dose to an organ is obtained by summing the doses delivered by each of the individual pathways.

Dose Mitigation

The time after accident initiation is divided into three phases: (1) an emergency phase, (2) an optional intermediate phase, and (3) a long-term phase. During the emergency phase, which can last up to seven days, doses are reduced by evacuation, sheltering, and temporary relocation of people. During the intermediate phase, doses may be avoided by temporary relocation of people. During the long-term phase, doses are reduced by decontamination of property that is not habitable, by temporary interdiction of property that cannot be restored to habitability by decontamination alone, by condemnation of property that cannot be restored to habitability at a cost below or equal to the worth of the property, by disposal of contaminated crops, and by banning farming on contaminated farmland.

Exposure Pathways

MACCS models seven exposure pathways: (1) exposure to the passing plume (cloudshine), (2) exposure to materials

deposited on the ground (groundshine), (3) exposure to materials deposited on skin, (4) inhalation of materials directly from the passing plume (inhalation), (5) inhalation of materials resuspended from the ground by natural and mechanical process (resuspension inhalation), (6) ingestion of contaminated foodstuffs (food ingestion), and (7) ingestion of contaminated water (water ingestion). Ingestion doses do not contribute to the doses calculated for the emergency phase of the accident. Only groundshine and inhalation of resuspended materials produce doses during the optional intermediate phase of the accident. Long-term doses are caused by groundshine, resuspension inhalation, water ingestion, and food ingestion. Ingestion of contaminated food or water generates doses to people who reside at unknown locations both on and off of the computational grid.

Population Cohorts

People on the computational grid are assigned to three groups: (1) evacuees, (2) people actively taking shelter, and (3) people who continue normal activities. Shielding factors for each of the groups are specified by the user.

Health Effects

Health effects are calculated from doses to specific organs using dose conversion factors. Early injuries and fatalities (those occurring within one year of the accident) are estimated using nonlinear dose-response models. Latent cancers are estimated using a piecewise linear dose-response model that is discontinuous. Two equations are implemented in the code, one for high exposures and one for low exposures.

Economic Effects

Economic consequences result from the implementation of mitigative actions. The following costs are considered in this estimate: (1) evacuation costs, (2) temporary relocation costs, (3) costs of decontaminating land and buildings, (4) lost return-on-investments from temporarily interdicted properties, (5) value of crops destroyed or not grown, and (6) value of condemned property. Costs associated with damage to the reactor, the purchase of replacement power, medical care, life-shortening, and litigation are not considered.

Summary of COSYMA Radiological Consequence Code

COSYMA was developed by the National Radiological Protection Board (NRPB) of the UK and Forschun-

gszentrum Karlsruhe (FZK) of Germany, as part of the European Commission's MARIA project (FZK and NRPB, 1991). It represents a fusion of ideas from the NRPB program MARC (Hill et al., 1988), the FZK program system UFOMOD (Ehrhardt et al., 1988) and input from other MARIA contractors. The program package was first made available in 1990 for use on mainframe computers, and several updates have been released since then. A PC version was first released in 1993 and has since been updated (Jones et al., 1995).

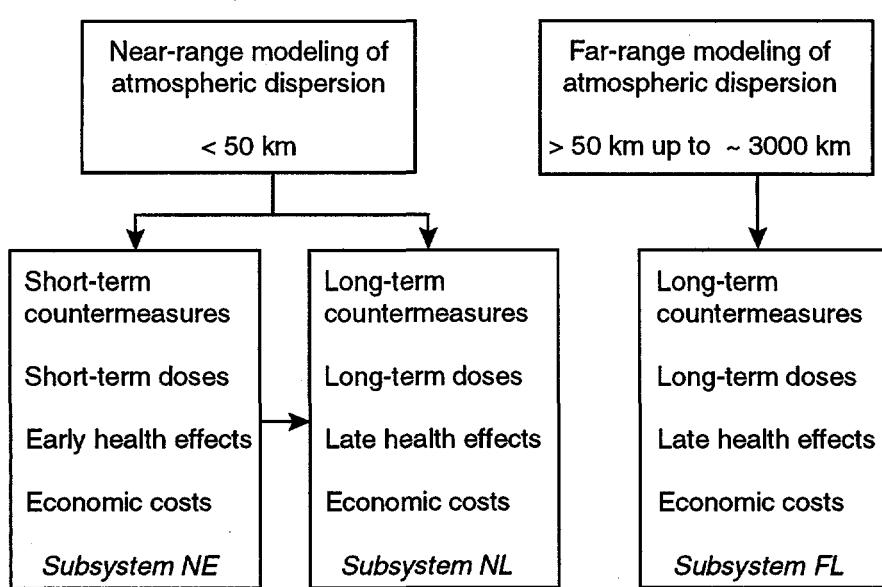
COSYMA is a system of programs and data bases, rather than a single program. The mainframe version contains three main accident consequence assessment programs together with a number of preprocessing and evaluation programs. The three main sub-systems of COSYMA are known as the NE, NL, and FL sub-systems (Figure A-4). The NE (near, early) sub-system is limited to calculating early health effects and the influence of emergency actions to reduce those effects and applies to the region near the accident site. The NL (near, late) subsystem is limited to calculating late health effects and the associated countermeasures, and applies mainly to the region near the site. The FL (far, late) sub-system calculates late health effects and appropriate countermeasures at greater distances from the site. Each of these programs is subdivided into a series of modules for the various steps in the calculation.

PC COSYMA incorporates the NE and NL sub-systems of the mainframe version.

The main endpoints of COSYMA are the numbers of health effects, the impact of countermeasures, and the economic costs resulting from the accidental release. A large number of intermediate results are obtained in the process of calculating the major endpoints; these results include activity concentrations, individual and collective doses, and the countermeasures assumed at different locations. COSYMA contains a series of evaluation programs that allow these results to be presented in a variety of ways.

Following an accidental release to atmosphere, people can be irradiated by a number of exposure paths. Those considered in COSYMA are cloudshine, groundshine, exposure to materials deposited on skin, direct inhalation of plume material, inhalation of resuspended materials, and ingestion of contaminated foods.

COSYMA includes some models directly within the various modules or subsidiary programs, such as atmospheric dispersion models. In other cases, COSYMA uses data libraries giving the results of other models which are not part of COSYMA itself, but whose uncertainty is considered within the current study.



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Figure A-4. General structure of the COSYMA program system.

Atmospheric dispersion and deposition

Mainframe COSYMA contains five different models of atmospheric dispersion that are appropriate for different applications or are based on different assumptions and approximations (Panitz et al., 1989).

The NE and NL sub-system include the MUSEMET (Straka et al., 1981) model, originally written at Forschungsanlage Jülich and extensively modified at FZK for use with COSYMA. This is a segmented Gaussian plume model allowing for changes of atmospheric conditions and wind direction during plume travel. This model derives the sequences of atmospheric conditions affecting the plume from hourly averages for wind speed and direction, stability category, precipitation intensity and mixing layer depth. It allows for the effects on the subsequent dispersion of plume rise and buildings near the release point. It also includes the effects of wet and dry deposition of the dispersing material. This model is also included in PC COSYMA.

The NE and NL sub-systems can also be used with the COSGAP or RIMPUFF dispersion models, which are provided as separate programs. COSGAP (Jones and Charles, 1982) is a Gaussian plume dispersion model, which is similar to MUSEMET but does not consider changes of wind direction during plume travel. It is based on the dispersion model in MARC. RIMPUFF (Mikkelsen et al., 1984), developed by Risø National Laboratory, Denmark, is a Gaussian puff trajectory model which derives the atmospheric conditions affecting the plume by interpolating between data from a number of meteorological stations in the region of interest.

The NL sub-system also contains the ISOLA (Hübschmann and Raskob) model for very long release durations. This uses statistics of atmospheric conditions and is only appropriate for releases that are sufficiently small that no countermeasures and no early health effects would be expected.

The FL sub-system is linked to the Mesos model (ApSimon and Goddard, 1983), developed by Imperial College, UK. This is a trajectory model for dispersion over long distances using meteorological data for a large area, such as the whole of Europe.

Accident consequence assessment programs need to consider that the accident could occur in any of a wide range of atmospheric conditions. It is not possible to calculate the consequences for every sequence of conditions that might arise, so a method of sampling a representative set of

conditions from those possible is needed. Both the mainframe and PC versions of COSYMA include a flexible program to conduct this sampling.

Dose calculations

As stated earlier, COSYMA does not include dosimetric models but uses information from data libraries which are calculated with these models. The libraries include information on doses from 197 nuclides.

The data library used for calculating external exposure from activity deposited on the ground contains outdoor doses per unit deposit for a series of times. These doses are mitigated by location factors describing the reduction in exposure due to shielding by buildings. The library is drawn from a number of sources, using results of models developed at NRPB (Charles et al., 1982; Crick and Brown, 1990) and Forschungszentrum für Umwelt und Gesundheit (GSF) (Jacob et al., 1988), Germany. The doses for major contributing nuclides in a fission reactor accident are derived from a model describing the deposition patterns in urban areas and the subsequent transfer of material between the different surfaces.

The doses from internal irradiation following ingestion or inhalation are calculated using data libraries of dose per unit intake derived using models which are consistent with those in International Commission on Radiological Protection (ICRP) publications 56, 67 and 69 (ICRP, 1990, 1994, 1995). COSYMA requires information on the dose received during different periods after the accident, which is included in the data libraries. Because the method used for calculating doses and risks of health effects in the mainframe version of COSYMA allows for the variation of dose per unit intake with age at intake, the libraries contain information on doses for different age groups in the population. The PC version, however, uses a simpler method which considers only the doses to adults.

Food chain models

COSYMA requires information on the concentration of material in foods as a function of time after the accident. It does not include a food chain model, but uses the results of such models through data libraries which give concentrations for a range of radionuclides in a number of foods at a series of times following unit deposition. The concentration of material in foods depends on the time of year at which the deposition occurs. COSYMA uses two data libraries for deposition in summer and in winter.

COSYMA uses libraries derived from the NRPB model FARMLAND (Brown and Simmonds, 1995) and the GSF model ECOSYS (Matthies et al., 1982). The libraries were created using accepted values for the food chain parameters for application within the EC, but differences exist because of other modeling assumptions made and because of the foods considered in each. The foods which can be considered with FARMLAND are: milk; meat and liver from cattle; pork; meat and liver from sheep; green vegetables; grain products; and potatoes and other root vegetables. The foods which can be considered with ECOSYS are: milk; beef; pork; grain products; potatoes and other root vegetables; and leafy and non-leafy green vegetables.

The intakes of these foods are calculated within COSYMA using one of two assumptions about the distribution of food between harvest and consumption. One method assumes that all food consumed is produced locally, and is used in calculating individual ingestion doses. The other method uses information on the amount of food produced in the area of interest, and calculates collective doses on the assumption that all food produced is consumed somewhere.

Countermeasures

COSYMA allows the user to consider the effects of a wide range of countermeasures in reducing the exposure of the population, and gives the user considerable freedom in specifying the criteria at which the actions will be imposed or withdrawn (Hasemann and Ehrhardt, 1994).

Sheltering alone or combined with evacuation may be implemented automatically or on the basis of dose. The distribution of iodine tablets, automatically or on the basis of dose, can also be considered. These actions are assumed to be implemented sufficiently rapidly to reduce the risks of both early and late health effects. Relocation is considered as an action to reduce doses and risks over longer time periods. It can be implemented on a dose criterion, as can return from evacuation or relocation. The effects of decontamination in reducing the period of relocation can be considered. If these actions are initiated on the basis of dose, the user can specify the intervention levels, organs and pathways to be considered, and the time over which the dose is to be integrated. The behavior of the population considered in the dose criteria can also be described using location factors.

Food bans can also be considered (Steinhauer, 1992). They can be implemented or withdrawn on the basis of doses

received within specified time periods or on the basis of the instantaneous concentration of radionuclides in foods.

Health effects

COSYMA considers both early and late health effects in the population, using methods recommended by NRPB (Edwards;¹ NRPB, 1993), the USNRC (Evans et al., 1990) and GSF (Paretzke et al., 1991).

The risk of early health effects is calculated using "hazard functions." The method allows for the variation of risk with the rate at which dose is accumulated over the first few days following the accident. Ten different fatal and non-fatal effects are considered.

The risk of late health effects is calculated using the linear dose response relationship. COSYMA considers the risk of fatal and non-fatal cancers in ten organs, as well as the risk of leukemia. It also considers the risk of hereditary effects. The method adopted in the mainframe version of COSYMA allows for the variation of risk with age at exposure (Ehrhardt et al., 1995). PC COSYMA uses a simpler method which only considers the doses and risks to adults. The mainframe version of COSYMA can provide information on the numbers of cancers in the people alive at the time of the accident, and in their descendants. It also gives information on the times at which the cancers occur.

Economic effects

COSYMA can calculate the off-site economic effects of the accident, considering the costs arising from the countermeasures and the costs of health effects. The assumptions and models are described in Haywood et al. (1991) and Faude (1992). The countermeasures for which costs are considered are movement of the population, food restrictions, and decontamination. The costs arising from lost production in the area from which people are moved can be assessed in terms of the per capita contribution of the relocated population to gross domestic product (GDP) or in terms of the value of the land affected. For longer periods of relocation, the lost capital value of the land and its assets may be calculated. The costs of food bans include contributions to GDP as well as the lost capital value and the disposal costs of the food affected. The cost arising from health effects may be calculated in terms of the treatment costs and the lost economic productivity of the affected individuals, or an estimation of the cost of health effects may be obtained using a more subjective approach to the valuation of life.

1. Edwards, A.A. 1995. Personal communication.

References

ApSimon, H.M. and Goddard, A.J.H. 1983. *Atmospheric transport of radioisotopes and the assessment of population doses on a European scale*. CEC Luxembourg EUR-9128.

Brown, J. and Simmonds, J.R. 1995. *FARMLAND a dynamic model for the transfer of radionuclides through terrestrial foodchains*. Chilton NRPB-R273.

Charles, D., Crick, M.J., Fell, T.P. and Greenhalgh, J.R. 1982. *DOSE-MARC: The dosimetric module in the methodology for assessing the radiological consequences of accidental releases*. Chilton NRPB-M74.

Crick, M.J. and Brown, J. 1990. *EXPURT: A model for evaluating exposure from radioactive material deposited in the urban environment*. Chilton NRPB-R235.

Ehrhardt, J., Burkart, K., Hasemann, I., Matzerath, C., Panitz, H.-J. and Steinhauer, C. 1988. *The program system UFOMOD for assessing the consequences of nuclear accidents*. FZK 4330.

Ehrhardt, J., Hasemann, I., Matzerath-Boccaccini, C., Steinhauer, C. and Raicevic, J. 1995. *COSYMA: health effects models*. Karlsruhe FZK 5567.

Evans, J.S., Moeller, D.W. and Cooper, D.W. 1990. *Health effects models for nuclear power plant accident consequence analysis*. NUREG/CR-4214, Rev. 1.

Faude, D. 1992. *COSYMA: Modelling of economic consequences*. Karlsruhe, FZK Report 4336.

Hasemann, I. and Ehrhardt, J. 1994. *COSYMA: dose models and countermeasures for external exposure and inhalation*. Karlsruhe FZK 4333.

Haywood, S.M., Robinson, C.A. and Heady, C. 1991. *COCO-1: model for assessing the cost of offsite consequences of accidental releases of radioactivity*. Chilton NRPB-R243.

Hill, M.D., Simmonds, J.R. and Jones, J.A. 1988. *NRPB methodology for assessing the radiological consequences of accidental releases of radionuclides to atmosphere - MARC-1*. Chilton NRPB-R224. London HMSO.

Hübschmann, W. and Raskob, W. *ISOLA V: A Fortran-77 code for the calculation of the long-term concentration distribution in the environment of nuclear installations*.

International Commission in Radiological Protection (ICRP). 1990. *Age-Dependent Doses to Members of the Public from Intakes of Radionuclides: Part 1*. ICRP Publication 56, Pergamon Press, Oxford.

ICRP. 1994. *Age-Dependent Doses to Members of the Public from Intakes of Radionuclides: Part 2 Ingestion Dose Coefficients*. ICRP Publication 67, Elsevier Science Ltd., Oxford.

ICRP. 1995. *Age-Dependent Doses to Members of the Public from Intakes of Radionuclides: Part 3 Ingestion Dose Coefficients*, ICRP Publication 69, Elsevier Science Ltd., Oxford.

Jacob, P., Paretzke, H.G., Rosenbaum, H., Zankl, M. 1988. "Organ doses from radionuclides on the ground. Part 1: Simple time dependencies," *Health Physics* 54, 617-633.

Jones, J.A., Mansfield, P.A., Haywood, S.M., Hasemann, I., Steinhauer, C., Ehrhardt, J. and Faude, D. 1995. *PC COSYMA (Version 2): An accident consequence assessment package for use on a PC*. EUR report 16239.

Jones, J.A. and Charles, D. 1982. *AD-MARC: The atmospheric dispersion module in the methodology for assessing the radiological consequences of accidental releases*. Chilton NRPB-M72.

FZK and NRPB. 1991. *COSYMA: A new program package for accident consequence assessment*. CEC. Brussels, EUR-13028.

Matthies, M., Eisfeld, K., Müller, H., Paretzke, H.G., Pröhl, G. and Wirth, G. 1982. *Simulation des Transfers von Radionukliden in landwirtschaftlichen Nahrungsketten*. GSF Bericht S-882.

Mikkelsen, T., Larsen, S.E. and Thykier-Nielsen, S. 1984. "Description of the Risø puff model," *Nuclear Technol.* 76, 56-65.

NRPB. 1993. "Estimates of late radiation risks to the UK population," *Documents of the NRPB* 4 (4) 15-157.

Panitz, H.-J., Päslar-Sauer, J. and Matzerath, C. 1989. *UFOMOD: Atmospheric dispersion and deposition*. FZK-4332.

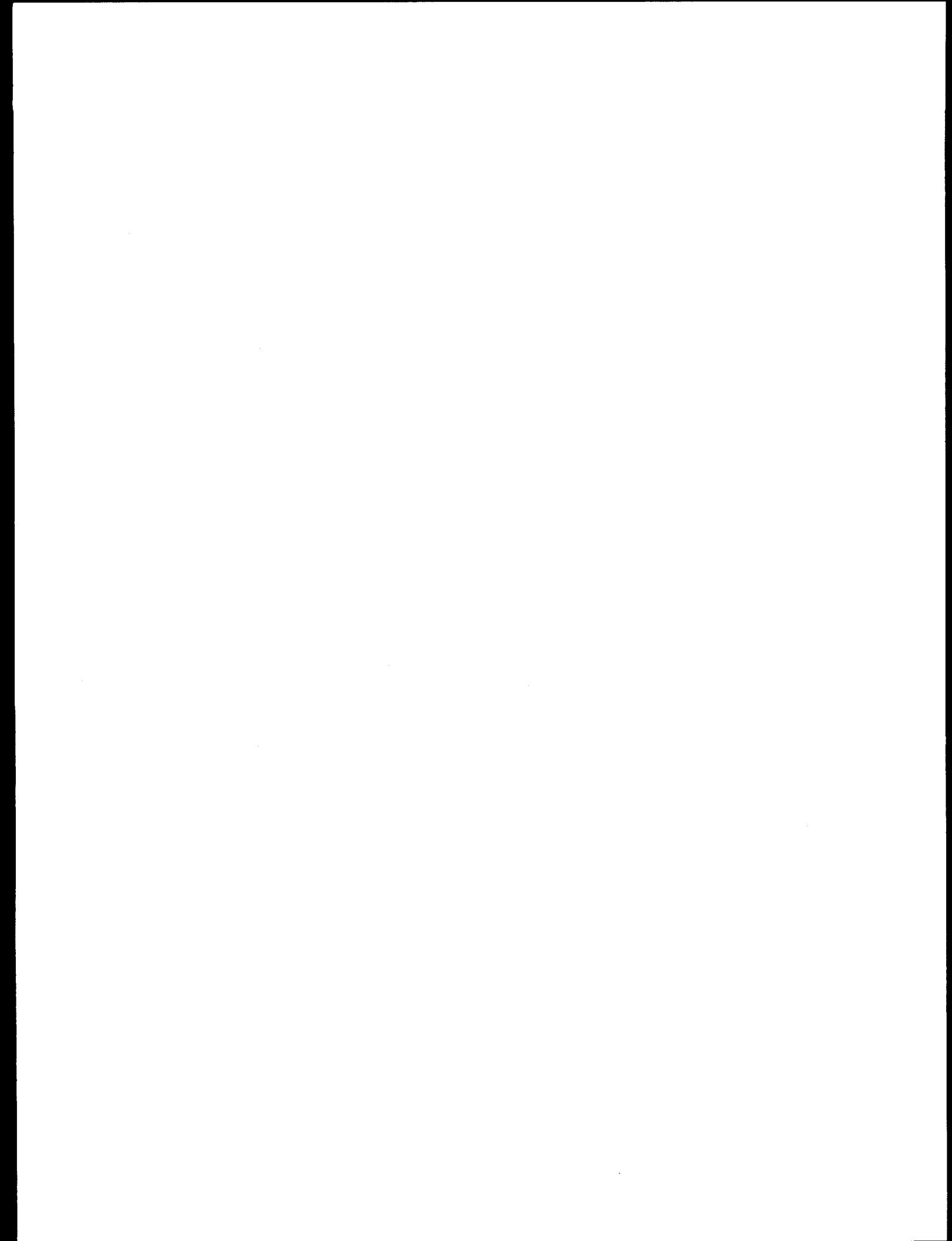
Paretzke, H.G., Stather, J.W. and Muirhead, C.R. 1991. "Risk factors for late somatic effects," In *Proceedings of the CEC Seminar on methods and codes for assessing the off-site consequences of nuclear accidents*, Athens 1990, Luxembourg EUR 13013.

Steinhauer, C. 1992. *COSYMA: Ingestion pathways and foodbans*. Karlsruhe. FZK 4334.

Straka, J., Geiß, H. and Vogt, K.J. 1981. "Diffusion of waste air puffs and plumes under changing weather conditions," *Contr. Atmos. Phys.* 54, 207-221.

APPENDIX B

Structure Document and Elicitation Questionnaire for the Expert Panel on Internal Dosimetry



ELICITATION QUESTIONS

Expert Panel on Internal Dosimetry

CEC/USNRC Joint Project on
Uncertainty Analysis of Consequence Assessment Programs

1. Introduction

The EC/USNRC Joint Study has been initiated to apply expert judgement elicitation techniques to estimate uncertainties associated with the predictions of probabilistic risk assessment (PRA) codes. The uncertainties in the various aspects of consequence assessment modeling are being considered separately by several expert panels. These panels are being formed jointly, where possible, between members of the European Community and the United States of America. This document provides introductory information for members of the internal dosimetry panel and elicitation questions.

2. Objectives of the Study

The overall aim is to assess uncertainties associated with consequences calculations for accidental releases of radionuclides from commercial nuclear power plants. It is envisaged that the uncertainty analyses of at least two consequence assessments programs will make use of the information derived in this project (i.e., COSYMA from the EC and MACCS from the US). However, the main objective of the exercise is to develop a library of uncertainty distributions that can be used for different uncertainty studies in the future. Questions are largely concerned with measurable/observable quantities that provide input to models, rather than details of the models themselves. Uncertainty distributions suggested by experts are used to determine uncertainties in model parameters, in this case inhalation and ingestion dose coefficients for selected radionuclides.

3. Choice of Experts and Elicitation Process

The experts have been chosen with the intention of providing a diversity of expertise and experience. Alternative points of view are encouraged and the experts will have the opportunity to discuss the issues together at the initial training meeting to be held in December 1995. Following this meeting the experts will be given time to assess the problems contained in the elicitation questions. They will be free to answer the questions in whatever manner they choose. They are encouraged, however, to record all assumptions made and methods used during this process, in a report (rationale) that they will be asked to provide. Elicitation sessions will be carried out some weeks later in private meetings between the individual experts and two analysts, one specializing in probability assessment and the other in the specific aspect of consequence modeling under consideration.

4. Formal expert elicitation

Expert judgement applicable to uncertainty analysis must be cast in the form of subjective probability distributions. Subjective probability measures degree of belief with respect to possible observations. In this study, experts are asked only about observable quantities.

Degree of belief is elicited in the form of 5%, 50% and 95% quantiles of subjective probability distributions. The 5% quantile of the distribution for an uncertain quantity X is the number x_{05} such that

$$\text{Prob}(X > x_{05}) = 5\%$$

For each assessment, certain background information is supplied. It is not the intention to provide all relevant information; rather to set the context of the questions to be asked.

5. Combining expert judgements

There are two reasons why panels of expert are used in this study. Firstly, eliciting differing viewpoints gives a better representation of the true uncertainty about the parameters under consideration. In contrast, a single expert would normally offer only one viewpoint. Secondly, empirical evidence shows that when the judgements of a number of experts, expressed in the form of probabilities, are combined using some reasonable aggregation procedure, the resulting probability distributions are more reliable. Such aggregated distributions better express the true uncertainty than the probability distribution of a single expert. Two concepts are important when evaluating the goodness of probability distributions:

Calibration

Calibration refers to the faithfulness of probabilities. In principle, events that are assigned a given probability should occur with a relative frequency equal to that probability. For example, an expert who assigns probability distributions to a set of uncertain quantities should find 5% of the quantities fall in the lower 5% tails of the distribution, 50% in the lower one-half of the distribution, etc.

Calibration is a concept that applies to sets of distributions, not to individual probabilities. An expert is said to be well calibrated if, over a large number of assessments, the probabilities assigned are correctly reflected in the relative frequencies. Of course, the measurement of calibration can occur only when the true values of the uncertain quantities become known. Calibration can be measured, in a statistical sense, through goodness-of-fit statistics and relative entropy.

Informativeness

Informativeness refers to how well probabilities define the value of a variable or the likelihood of an event. Probabilities near zero and one better resolve uncertainty than probabilities near one-half. Similarly, sharp or peaked density functions better resolve uncertainty than flat or diffused densities.

Calibration and high informativeness may not be compatible, however. A set of probability distributions may be very peaked, but very wrong. In fact, there is a common tendency for elicited probability distributions to be more "informative" than is warranted. Combined judgements tend to be better calibrated but less informative (more diffuse).

Many forms of combining judgements have been suggested. The simplest rule for combining expert judgements is to take a simple average of their probabilities. Another method is to weight the experts on the basis of how well they perform on questions for which the true answers are known. This approach is known as performance based weighting.

6. Scope of the Internal Dosimetry Panel

While COSYMA and other ACA codes include doses from a comprehensive list of radionuclides, uncertainty analysis will be undertaken only for the most important radionuclides. Parameters for infants and children as well as adults will be considered where it is considered that sufficient data will be available.

An Annex to this document gives background information on the calculation of doses from intakes of radionuclides, intended as information for those involved in processing data from the expert elicitations rather than for the experts themselves. The biokinetic and dosimetric approaches summarized in the Annex are largely those developed and used by the International Commission on Radiological Protection (ICRP). However, the Elicitation questions, as formulated, address experimentally observable parameters and are not constrained by any existing models.

Elicitation Questions

For each question, a median estimate is required for an average individual, representative of the group under consideration. The uncertainty associated with the median estimate should be given as 5th and 95th percentiles.

Inhalation

These questions cover important contributions to uncertainty in calculating doses from inhaled radionuclides, considering the behavior of materials in the respiratory tract. Factors omitted that might also contribute significantly to uncertainties are the location of sensitive cells in different regions, the relative radiosensitivity of the different regions and tissue mass and geometric consideration. Intake can be considered to be due to exposure to air concentrations of radionuclides of say, 1 Bq m⁻³, for short duration of say, 1 minute.

Question 1. Average ventilation rates, L min⁻¹, assuming a normal daily (24h) mix of activities (combined male, female average)?

	5 year old children			Mature Adults		
	5%	50%	95%	5%	50%	95%
L min ⁻¹						

Question 2. Total initial deposition in the respiratory tract, % of total amount inhaled, normal daily (24 hr) mix of activities?

	0.1 µm AMAD			1 µm AMAD			10 µm AMAD		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Mature Adults									
5 year old children									

Question 3. Initial deposition in the extrathoracic region, % of total deposition in the respiratory tract? (This question also gives the initial deposition in the lung (tracheobronchial (TB) + pulmonary (AI) regions), since lung = total - ET)

	0.1 µm AMAD			1 µm AMAD			10 µm AMAD		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Mature Adults									
5 year old children									

Question 4. Initial deposition in the tracheobronchial (TB) region, % of the total deposition in the lung (TB + AI regions)?

	0.1 µm AMAD			1 µm AMAD			10 µm AMAD		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Mature Adults									
5 year old children									

Question 5. Assuming completely insoluble particles (1 μm AMAD), retention in each respiratory tract region in mature adults as a % of the total initial deposition in the respiratory tract, as a function of time after deposition?

	10 min			1 hr			1 day		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Tracheobronchial airways									
Pulmonary (AI) region									
	1 month			1 year			10 years		
Tracheobronchial airways									
Pulmonary (AI) region									

Question 6. By what factors would you expect the median values to be different in 5 year old children (1=no difference)?

	10 min	1 hr	1 day	1 month	1 year	10 years
Tracheobronchial airways						
Pulmonary (AI) region						

Question 7. Absorption (dissolution and transfer) to blood in adults, % of the total initial deposition (1 μm AMAD particles) in the respiratory tract, considering the range of chemical forms which you judge most likely to be inhaled after an accident? (To include absorption to blood from the gastrointestinal tract for material cleared from the respiratory tract via the mucociliary escalator and swallowed.) The usual assumption has been that elements will be inhaled in oxide form, apart from I in elemental form.

	1 hr			1 day			1 week		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Sr									
I									
Cs									
Pu									
Ru									
Ce									
Te									
	1 month			1 year			10 years		
Sr									
I									
Cs									
Pu									
Ru									
Ce									
Te									

Question 8. By what factors would you expect the median values to be different in 5 year old children (1=no difference)?

5 year old children	1 hr	1 day	1 week	1 month	1 year	10 years
Sr						
I						
Cs						
Pu						
Ru						
Ce						
Te						

Ingestion

Factors omitted that might also contribute significantly to uncertainties are doses to sensitive cells from activity in gut contents, particularly for alpha emitters, retention in intestinal tissue, and tissue mass and geometric considerations. Consider a single intake involving ingestion of 1 Bq.

Question 9. Absorption to blood as a fraction (f_1) of activity ingested, (considering chemical forms most likely to be ingested after an accident)?

	3 month old infants			5 year old children			Adults		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Sr									
I									
Cs									
PuO ₂ *									
Pu biol†									

* Refractory oxide
† "Biologically incorporated"

Systemic distribution and retention

These questions cover important contributions to uncertainty in calculating doses from radionuclides reaching blood. Factors omitted that might also contribute significantly to uncertainties are the location of sensitive cells in bone and absorbed fractions for alpha- and beta- emitting bone-seekers, and tissue mass and geometric considerations. Consider the behavior of the elements, taking no account of radioactive half-lives of isotopes.

Strontium, Plutonium, Cerium, Tellurium

Question 10. Considering the total amount reaching blood (as if administered intravenously as a single injection). % retained in liver and skeleton (bone + bone marrow), as a function of time after entry into blood?

5 year old children			Adults			
	5%	50%	95%	5%	50%	95%
Skeleton and liver, 1 day						
Sr						
Pu						
Ce						
Te						
Skeleton and liver, 1 week						
Sr						
Pu						
Ce						
Te						
Skeleton and liver, 1 month						
Sr						
Pu						
Ce						
Te						
Sb						
Skeleton and liver, 1 year						
Sr						
Pu						
Ce						
Skeleton and liver, 10 years						
Sr						
Pu						
Ce						
Skeleton and liver, 50 years						
Sr						
Pu						
Ce						

Question 11. Retention in the skeleton, % total retention in liver + skeleton (bone + marrow), as a function of time after entry into blood?

	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
Skeleton, 1 day						
Sr						
Pu						
Ce						
Te						
Skeleton, 1 week						
Sr						
Pu						
Ce						
Te						
Skeleton, 1 month						
Sr						
Pu						
Ce						
Te						
Skeleton, 1 year						
Sr						
Pu						
Ce						
Skeleton, 10 years						
Sr						
Pu						
Ce						
Skeleton, 50 years						
Sr						
Pu						
Ce						

Question 12. Retention of plutonium on endosteal bone surfaces (considering a 10 μm depth of bone mineral) as a % of total skeletal retention, as a function of time after entry into blood?

		5 year old children			Adults		
		5%	50%	95%	5%	50%	95%
Endosteal bone surface	1 day						
	1 week						
	1 month						
	1 year						
	10 years						
	50 years						

Question 13. Retention of plutonium on trabecular surfaces, % total endosteal surface retention as a function of time after entry into blood?

		5 year old children			Adults		
		5%	50%	95%	5%	50%	95%
Trabecular bone surface	1 day						
	1 week						
	1 month						
	1 year						
	10 years						
	50 years						

Question 14. Retention of plutonium in red bone marrow as a % total skeletal retention, as a function of time after entry into blood?

		5 year old children			Adults		
		5%	50%	95%	5%	50%	95%
Red bone marrow	1 day						
	1 week						
	1 month						
	1 year						
	10 years						
	50 years						

Ruthenium, cesium

Question 15. Considering the total amount reaching blood (as if administered intravenously as a single injection), % total retention (whole body or systemic) as a function of time after entry into blood?

	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
1 day						
Ru						
Cs						
1 week						
Ru						
Cs						
1 month						
Ru						
Cs						
1 year						
Ru						
Cs						
5 years						
Ru						
Cs						

Iodine

Question 16. Considering the total amount of I reaching blood (as if administered intravenously as a single injection), % retained in the thyroid as a function of time after entry into blood?

	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
1 day						
1 week						
1 month						
3 months						

Dose coefficients

Inhalation and ingestion dose coefficients represent ACA code inputs. Uncertainties will include dosimetric modeling considerations as well as the parameters considered above.

Question 17. Intake dose coefficients, absorbed dose to specified organs or tissue per unit activity inhaled or ingested (committed equivalent doses), Gy Bq⁻¹?

	5 year old children			Adult		
	5%	50%	95%	5%	50%	95%
⁹⁰Sr, inhalation, 1 μm AMAD						
lung						
bone marrow						
bone surface						
⁹⁰Sr, ingestion						
colon						
bone marrow						
bone surface						
¹³¹I, inhalation, 1 μm AMAD + vapor (decide proportions)						
thyroid						
¹³¹I, ingestion						
thyroid						
¹³²Te, inhalation, 1 μm AMAD + vapor (decide proportions)						
lung						
thyroid						
colon						
¹³⁷Cs, inhalation, 1 μm AMAD						
lung						
colon						
stomach						
bone marrow						
¹³⁷Cs, ingestion						
lung						
colon						
stomach						
bone marrow						
¹⁴⁴Ce, inhalation, 1 μm AMAD						
bone surface						
lung						
bone marrow						
liver						

	5 year old children			Adult		
	5%	50%	95%	5%	50%	95%
^{239}Pu, inhalation, 1 μm AMAD						
bone surface						
bone marrow						
liver						
lung						
^{239}Pu, ingestion						
bone surface						
bone marrow						
liver						
colon						

(xi) Joint dosimetry/late effects question:

The number of radiation-induced cancer deaths up to 40 years following exposure in a population of a hundred million persons (5×10^7 male, 5×10^7 female) each of whom inhales 10 K Bq of the radionuclides specified.

Nuclide	Physical Form	Chemical Form	Cancer Type	Number of Cancers		
				5%	50%	95%
Pu-239	1 μm AMAD	Oxide	Lung			
			Bone			
			Liver			
			Leukemia			
			All cancers			
Sr-90	1 μm AMAD	Oxide	Lung			
			Bone			
			Leukemia			
			All cancers			

APPENDIX C

Rationales and Responses of the Expert Panel on Internal Dosimetry

Note: Tables without data indicate that the expert had no response.

EXPERT A

General Approach

Consistent with my experience I have concentrated on the questions related to inhalation (1 – 8). Indeed, within that area I would regard myself as an expert on retention and clearance (Questions 5 – 8) rather than ventilation and deposition, which are specialist topics in themselves.

The report describing the new ICRP (Publication 66) respiratory tract model, IRTM (ICRP, 1994a) provides recent comprehensive reviews of those aspects of inhalation covered by Questions 1–6: ventilation; particle and vapor deposition in the respiratory tract and clearance of material from the respiratory tract by particle transport. *ICRP Publication 66* also addresses uncertainties in each aspect of the model, but to varying degrees, and in various ways. As a member of the ICRP Task Group on Human Respiratory Tract Models for Radiological Protection I was directly involved in production of the IRTM. However, the various aspects of the model were distributed between members of the Task Group: I had a leading role with respect to clearance, but had little input into the ventilation and deposition aspects. The general approach I have taken here has been to use the IRTM as a starting point.

For consideration of absorption of specific elements (Questions 7 – 8) I have drawn mainly on *ICRP Publication 71* (ICRP, 1996), with which I was also heavily involved, and which provides up to date relevant reviews.

This work was partially supported by the EC.

1. Inhalation: Ventilation Rates

1. Average ventilation rates, $L \text{ min}^{-1}$, assuming a normal daily mix of activities (combined male, female average)?

In Section 1.1 I have summarized the treatment of ventilation rates in *ICRP Publication 66*, Annex B, Respiratory Physiology (Roy et al., 1994). I have used its average values for my median estimates, since it is based on a thorough, up-to-date review which I could not improve upon, especially within the short timescale of this exercise. Similarly, consideration of uncertainties did not reveal any specific information that would justify revising them. However, the adult values taken are different from those used in *ICRP Publication 71* (ICRP, 1996: inhalation dose coefficients for members of the public) since the adult

member of the public selected by ICRP is male, and here we have taken a male-female average.

In Section 1.2 I have summarized the approach to estimating inhalation rates based on energy expenditure by Layton (1993). I have given some detail partly because the approach is indirect and somewhat complicated, but also because Layton concludes that the IRTM approach leads to significant overestimation of daily average values. This led to a recent exchange of correspondence in *Health Physics Journal*.

My colleague, Ms. K.A. Jones investigated uncertainties in the values by going back to the references cited in *ICRP Publication 66*, and a few more recent ones (Section 1.3). She has for some time had responsibility for advising an appropriate ventilation rates for use in environmental assessments at NRPB. We discussed together the approach to estimating x_5 and x_{95} .

Note that Section 1.1 gives ventilation rates in $\text{m}^3 \text{ h}^{-1}$, since these are the units used for reference values in *ICRP Publication 66*. Section 1.3 uses $L \text{ min}^{-1}$, consistent with Question 1, and much of the discussion in *ICRP Publication 66*, Annex B.

1.1 Summary of IRTM treatment

The ventilation rate (i.e., the volume of air inhaled per unit time) is the more important of the two main factors that relate exposure (time-integrated air concentration, $\text{Bq} \cdot \text{s m}^{-3}$), to intake in Bq . The other factor is inhalability (also known as aspiration efficiency), which is the ratio of the particle concentration in the air entering the respiratory tract to that in the ambient air. The inertia of particles larger than a few microns increases the concentration in the air entering the nose or mouth when facing into a wind, and reduces it otherwise, the average net effect being to reduce it to about half that in the ambient air. Inhalability is here considered with deposition (Section 2.2), as it is in the IRTM.

Ventilation, the breathing frequency and tidal volume, is the main factor in the IRTM that depends on age and level of exercise. It is also the aspect for which there are comprehensive data relating to women and children as well as men.

The approach taken in the IRTM is described in detail in Annex B (Respiratory Physiology) of *ICRP Publication 66* (Roy et al., 1994). It follows that taken previously by ICRP in its report on Reference Man (ICRP, 1975). Reference

levels of physical exercise are defined, in this case four: sleep, sitting, light exercise (LE), and heavy exercise (HE). For each reference subject, representing each age group, reference values were determined from review of the literature, for the primary quantities: breathing frequency (f_R) (breaths per minute) and ventilation rate (B) ($\text{m}^3 \text{ h}^{-1}$) for the four reference levels of exercise. (The breathing frequency is required as an input to the deposition model, as is the ventilation rate). The results of habit surveys were also compiled, to obtain typical distributions of time spent by reference subjects at each of the reference levels of exercise. By combining these, the total volume of air inhaled in a typical day, and hence the daily average ventilation rate (DAVR), are obtained.

1.1.1 Ventilation rates

Estimates were made of respiratory frequencies (f_R), tidal volume (V_T), and ventilation minute volumes (\dot{V}_E , L min^{-1}) according to age and level of exercise. Values of work load and minute ventilation, for untrained men at the four exercise levels were obtained from the literature (ICRP, 1975; Dejours, 1982; Scherrer, 1981) (Table 1.1).

Exercise levels for adult males were chosen as follows:

- Light exercise: one-third of highest work load completed (W_{\max} measured by Godfrey et al., 1971). This corresponds to working in laboratories and workshops; active house cleaning; painting, woodworking, etc.
- Heavy exercise: two-thirds of W_{\max} . Firemen, construction workers, farmers, athletes, etc. might spend up to 2 h d^{-1} at this level.

These levels of exercise are consistent with *ICRP Publication 23* (1975), and slightly lower than those reported by Anderson et al., 1985.

To obtain V_T and f_R for 5-y-old children and women, the values for males were scaled according to published experimental data. Results were given in Table B.15. Data relevant to this project (5-y-old children and adults) are given in Table 1.2.

1.1.2 Time budgets

Information on time budgets was collated. It was noted that values had been published previously in *ICRP Publications 23 and 50* (ICRP, 1975; 1987), and UNSCEAR (1982; 1988). UNSCEAR (1988) used data from Brown (1983),

which were based on a survey made in Great Britain in 1978. Similar data were also collected in the Multinational Comparative Time Budget Research Project, in which about 30,000 people from 12 countries in Europe, the United States, Peru, and the Soviet Union participated. These data were discussed by Szalai (1972), and used by Moschandreas (1981) to evaluate exposure to airborne pollutants. More recent information on time budgets activities was obtained from economic surveys (INSEE, 1988, 1989a,b,c,d). They gave values of the time spent indoors higher by 5 – 10% than those of Anderson et al. (1985) for the population of the USA. Two groups of workers were not included in the INSEE surveys: outdoor workers (farmers, construction workers, etc.) and miners. For outdoor workers ICRP (1994a) assumed the same time budget as for sedentary workers, but a higher level of exercise.

1.1.3 Volumes of air breathed daily

The four standard exercise levels, with associated ventilation rates, were assigned to defined periods of the day. *ICRP Publication 66* gives results for children and adult members of the public in Tables B.16A and B.16B.

Tables 1.3 and 1.4 give data for 5-y-old children and adults respectively. For the workers listed these values are for an “average” day including both working days and weekends etc. In Table B.17 (Table 1.5 below) workers were divided into “sedentary” and “heavy”; an average working day was divided into three periods of 8 h each, following previous ICRP practice.

1.1.4 Comparison with Previous Standards

ICRP Publication 23 (ICRP, 1975) divided the day into three periods of 8 h (Table 1.6). Values for men from UNSCEAR (1982) and *ICRP Publication 50* (ICRP, 1987) are given in Table 1.7.

UNSCEAR (1988) assumed the same mean breathing rate of $0.8 \text{ m}^3 \text{ h}^{-1}$ for indoors and outdoors, giving a daily volume inhaled of 19.20 m^3 .

General Population

Compared with *ICRP Publication 23*, the total volume of air breathed per day calculated in *ICRP Publication 66* is slightly lower for adult females, almost equal for adult males and 10-y-old children, and higher for 1-y-olds and newborns. The values for adults are similar to those in *ICRP Publication 50*, which are average values for males and females.

Workers

The values obtained for workers (Table B.17) are similar to those in *ICRP Publication 23* (ICRP, 1975). However, to take into account higher ventilation rates required by some workers (miners, firemen, construction workers, farmers) *ICRP Publication 66* added a "heavy worker" class, so as not to underestimate their exposure under critical situations.

Uncertainties

It is considered in *ICRP Publication 66* that with the method used of estimating DAVR from minute volumes required for physical activity levels, uncertainty arises more from the evaluation of daily activity levels than from the associated breathing rates derived from experimental data. Experimental ventilation data for individuals performing various daily activities would be of value to verify these estimates. Preliminary data were cited from a study in South African gold mines, in which 620 subjects wore expired air analyzers while working, and the oxygen consumption was related to heart frequency. The results gave a mean ventilatory rate for underground heavy workers of $\sim 1.3 \text{ m}^3 \text{ h}^{-1}$, with 70% of the miners performing heavy work having a ventilation rate $>1.2 \text{ m}^3 \text{ h}^{-1}$, and 15% $>1.5 \text{ m}^3 \text{ h}^{-1}$. This is not much higher than that assumed for sedentary workers (Table 1.4).

Parameter values relevant to this project are brought together in Table 1.8. A comparison for the adult male is made with *ICRP Publication 71*, which is also based on Annex B. The difference arises because for *ICRP Publication 71* the time asleep was rounded down to 8 h, and the time sitting correspondingly increased. The total volumes per day in the penultimate column (m^3) were converted to ventilation rates (L min^{-1}) to use as median values in answer to Question 1.

1.2 Estimation of breathing rates from energy expenditure

1.2.1 Methodology

Layton (1993) proposed a different approach to evaluating ventilation rates, based on energy expenditure. In justification for taking such an apparently indirect approach, he pointed out weaknesses in the traditional approach, as used by ICRP and others. This he termed the "time-activity-ventilation" (TAV) approach, expressed as:

$$\dot{V}_E = \frac{1}{T} \sum_{i=1}^k \dot{V}_{E,i} t_i$$

Where $\dot{V}_{E,i}$ is the average minute volume (L min^{-1}), t_i is the duration of exercise level i , $\dot{V}_{E,i}$ is the corresponding minute volume, k is the number of periods of exercise, and T is the total duration. He noted that there had been no studies that systematically measured inhalation rates associated with the various activities of daily life. Moreover, available methods for measuring breathing rates were cumbersome and would themselves cause changes in behavior, so that such a study would be difficult to carry out. The TAV approach relied on short term measurements on selected individuals working at selected levels of energy expenditure, and there are limited data on the distribution of time spent by different sub-groups of the population at different rates of energy expenditure (as opposed to different activities). Furthermore, he considered that the rates derived in this way were inconsistent with breathing rates required for metabolic conversion of food, and this could lead to inconsistencies in intake assessments involving both inhalation and ingestion.

Layton stated that there was some evidence indicating that the TAV approach overestimated actual respiration. However, the only example cited (^{239}Pu from weapons fallout measured in lungs at autopsy) indicating 15 m^3 per day rather than the ICRP model value of 22 m^3 per day, does not to me suggest a problem, rather that agreement is as good as could be expected. The difference could be due to errors in any of a number of factors: the mean air concentration; particle size distribution; regional deposition; lung retention, as well as DAVR.

Layton proposed that inhalation rates be calculated from the oxygen requirement to support energy expenditure:

$$\dot{V}_E = E \times H \times VQ$$

Where E is energy expenditure (kJ min^{-1}); H is the volume of oxygen (L) consumed in the production of 1 kJ of energy; and VQ is the "ventilatory equivalent", the ratio of the minute volume to the oxygen uptake rate. He then reviewed information on each of these parameters.

Energy expenditure, E

Layton took several approaches to estimating E . He proposed that ventilation rates over short periods be assessed from energy expenditures obtained by multiplying

the basic metabolic rate, BMR (the minimum energy requirement to support cellular respiration at rest) by the Metabolic Equivalent, MET, which depends on the activity. From information in the literature he obtained MET values giving energy expenditures of:

- sedentary: $1.2 \times \text{BMR}$
- light: $1.5 - 2 \times \text{BMR}$
- moderate: $3 - 5 \times \text{BMR}$
- heavy: $10 - 20 \times \text{BMR}$

For longer periods he proposed that energy requirement could be equated with food energy intakes (EFD) derived from dietary surveys, and compared with BMRs. There was however a problem of systematic bias (under-reporting) in dietary surveys. Further information on EFD/BMR ratios were obtained from other dietary surveys and metabolic studies with doubly-labelled water ($^2\text{H}_2^{18}\text{O}$): in which carbon dioxide production and energy expenditure were estimated from turnover rates of the isotopes.

Oxygen uptake factor, H

Layton estimated H by considering the amount of oxygen to produce 1 kJ energy from each type of food: fat, protein and carbohydrate, and the proportions of these in the diet in the USA based on two large surveys. This gave an average of 20 kJ per liter O_2 .

Ventilatory equivalent, VQ

Layton collated measurements of the ratio of minute volume to oxygen uptake from the literature. He concluded that there was a strong correlation between them, with most values of VQ in the range 23 – 32. He does not elaborate on the age-distribution of the data, but referred to adults and newborns and noted that much of the data on the former related to athletes.

1.2.2 Inhalation rates for different subjects

Inhalation rates derived by Layton from MET and BMR for the various age groups and levels of exercise are similar to those presented elsewhere (e.g., Roy and Courtay, 1991).

Because of the bias noted above in dietary surveys, Layton proposed three methods for estimating chronic ventilation rates:

- Breathing rates based on dietary surveys adjusted upwards ($\times 1.2$) to compensate for bias.
- Breathing rates from multiples of the average BMR for each age-gender cohort.
- Breathing rates for energy expenditures for estimated times at each level of expenditure.

The daily average ventilation rates (DAVR) obtained from the first two methods were very similar, and that from the third somewhat higher. The main conclusion was that DAVRs based on the TAV approach, as in the IRTM, are considerably higher for adults: by a factor of 1.4 for men and 1.6 for women. The main source of the discrepancy is the estimated ventilation rate during “light” exercise, which is about 3 times higher than at rest (Table 1.2), compared to an average MET of 1.5, and whether this should be applied to so many of the activities in a modern lifestyle. Layton’s conclusion was that the ICRP approach leads to overestimates of inhalation intakes.

In written comments, Bertholon and Roy (1994) pointed out that the three factors involved in Layton’s approach E , H , and VQ , all have to be estimated indirectly, and that there are uncertainties in each which accumulate in the uncertainties in the inhalation rates. Furthermore they considered that inhalation rates are determined by other factors as well as oxygen requirement for metabolism, and so are not directly related to it. Roy et al. (1994) similarly commented that evaluating breathing rates from food intakes involves several assumptions, some of which lead to underestimation because of nonlinearity in relationships and high individual variability of some factors; for example, the ratio of food intake to energy expenditure and the amount of oxygen required for oxidation. They considered that the major uncertainty lies in the value of VQ , which has a nonlinear relationship to activity level among individuals, and also varies markedly with dietary habits and metabolic characteristics.

In response, Layton (1994) put forward the view that the main source of uncertainty in his approach was variation between individuals not methodological or experimental error, whereas DAVRs estimated with the TAV approach were very sensitive to judgements made about the ventilation rates chosen to represent light activity, which makes the largest contribution to DAVR.

1.3 Consideration of uncertainty (K.A. Jones)

As a first stage I collected together the available information on ventilation rates according to various exercise levels, and the time spent at each level of activity. Tables 1.9 and 1.10

show the information on ventilation rates for different activity levels, and the time spent at the different activity levels, respectively.

The uncertainty in the daily average ventilation rate (DAVR) can be considered to result from uncertainties in (i) the time spent at each exercise level and (ii) the ventilation rate at each exercise level. To avoid overestimating uncertainties we considered (i) varying the time distribution, while holding the ventilation rates constant at the median levels (given in Table 1.8), (ii) varying the ventilation rate at each exercise level, while holding the distribution of time constant at the median values (given in Table 1.8).

1.3.1 5-yr-olds

	Ventilation rate, L min^{-1}			Time, h d^{-1}		
	x_5	x_{50}	x_{95}	x_5	x_{50}	x_{95}
Sleep	3	4	7.5	11	12	13
Sitting	4	5.3	15	3	4	9
LE	8	9.5	35	3	8	8.5

The values for x_{95} for ventilation rates for light exercise (LE) are high due to the data given by Anderson et al. (1985).

- 1) Take time values which lead to low and high DAVR, and median ventilation rates for exercise levels.

	Time, h d^{-1}		Ventilation rate, L min^{-1}
	low DAVR	high DAVR	
Sleep	13	11	4
Sitting	8	4.5	5.3
LE	3	8.5	9.5
DAVR, L min^{-1}	5.1	6.2	

- 2) Take median distribution of time and ventilation rates for exercise levels which lead to low and high DAVR.

	Time, h d^{-1}	Ventilation rate, L min^{-1}	
		low DAVR	high DAVR
Sleep	12	3	7.5
Sitting	4	4	15
LE	8	8	35
DAVR, L min^{-1}		4.8	18

I think that this ventilation rates is too high i.e., that it is too extreme to assume that the average child has such high ventilation rates at every exercise level.

- 3) Consider low and high values of DAVR from the two approaches:

	Low DAVR	Median	High DAVR
Vary time	5.1	6.1	6.2
Vary ventilation rate	4.8	6.1	18
Mean	5	6.1	12

I think that 12 L min^{-1} is too high a value to use for the x_{95} . However Layton (1993) quotes values of 9 L min^{-1} for the 3 - 10 year old age range so I think that 9 L min^{-1} is a good upper value.

Hence estimated uncertainty in daily average ventilation rate for 5 yr olds is:

DAVR, L min^{-1}		
x_5	x_{50}	x_{95}
5	6.1	9

1.3.2 Adult males

	Ventilation rate, L min^{-1}			Time, h d^{-1}		
	x_5	x_{50}	x_{95}	x_5	x_{50}	x_{95}
Sleep	6	7.5	13	7	8.5	11
Sitting	8	9	15	4	5.5	9
LE	22	25	45	4.5	9.75	10
HE	30	50	80	0.2	0.25	1

The values for x_{95} for ventilation rates for light exercise (LE), and heavy exercise (HE) are high due to the data given by Anderson et al. (1985).

1) Take time values which lead to low and high DAVR, and median ventilation rates for exercise levels.

	Time, $h d^{-1}$		Ventilation rate, $L min^{-1}$
	low DAVR	high DAVR	
Sleep	11	7	7.5
Sitting	8.3	6	9
LE	4.5	10	25
HE	0.2	1	50
DAVR, $L min^{-1}$	11.7	16.9	

2) Take median distribution of time and ventilation rates for exercise levels which lead to low and high DAVR.

	Time, $h d^{-1}$	Ventilation rate, $L min^{-1}$	
		low DAVR	high DAVR
Sleep	8.5	6	13
Sitting	5.5	8	15
LE	9.75	22	45
HE	0.25	30	80
DAVR, $L min^{-1}$		13.2	27.2

I think that this ventilation rate is too high i.e., that it is too extreme to assume that the average man has such high ventilation rates at every exercise level.

3) Consider low and high values of DAVR from the two approaches:

	Low DAVR	Median	High DAVR
Vary time	11.7	15.4	16.9
Vary ventilation rate	13.2	15.4	27.2
Mean	12.5	15.4	22

Layton (1993) quotes a value of $10 L min^{-1}$ for an adult male, so I have lowered x_5 to encompass this value. I think that $22 L min^{-1}$ for the x_{95} is too extreme, so I have chosen $18 L min^{-1}$ as a more representative value.

Hence estimated uncertainty in DAVR for adult males is:

DAVR, $L min^{-1}$		
x_5	x_{50}	x_{95}
10	15.4	18

1.3.3 Adult females

	Ventilation rate, $L min^{-1}$			Time, $h d^{-1}$		
	x_5	x_{50}	x_{95}	x_5	x_{50}	x_{95}
Sleep	4.5	5.3	9	7	8.5	11
Sitting	5.5	6.5	11	4	5.42	8.5
LE	14	21	30	4	9.89	10
HE	30	45	50	0.15	0.19	0.25

1) Take time values which lead to low and high DAVR, and median ventilation rates for exercise levels.

	Time, $h d^{-1}$		Ventilation rate, $L min^{-1}$
	low DAVR	high DAVR	
Sleep	11	7	5.3
Sitting	8.5	6.75	6.5
LE	4.4	10	21
HE	0.15	0.25	45
DAVR, $L min^{-1}$	8.8	12.6	

2) Take median distribution of time and ventilation rates for exercise levels which lead to low and high DAVR.

	Time, $h d^{-1}$	Ventilation rate, $L min^{-1}$	
		low DAVR	high DAVR
Sleep	8.5	4.5	9
Sitting	5.42	5.5	11
LE	9.89	14	30
HE	0.19	30	50
DAVR, $L min^{-1}$	8.8	12.6	18.4

I think that this ventilation rates is too high i.e., that it is too extreme to assume that the average woman has such high ventilation rates.

3) Consider low and high values of DAVR from the two approaches:

	Low DAVR	Median	High DAVR
Vary time	8.8	12.3	12.6
Vary ventilation rate	8.8	12.3	18.4
Mean	8.8	12.3	15.5

Layton quotes a value of 7.1 L min^{-1} for an adult female so I have lowered x_5 to encompass this value. I have taken an approximate average of the two high DAVR values

Hence estimated uncertainty in DAVR for adult females is:

DAVR, L min^{-1}		
x_5	x_{50}	x_{95}
6.5	12.4	15

In summary, Table 1.11 shows the daily average ventilation rate (DAVR) for 5 yr olds, adult females and adult males taken from the various references. The estimates of x_5 , x_{50} and x_{95} are also given for comparison.

Table 1.1. Relationship between workload and minute ventilation in adult men (from ICRP Publication 66, Table B.14, Roy et al., 1994)

Activity	Workload		L min^{-1} (\dot{V}_E)	$\text{m}^3 \text{ h}^{-1}$ (B)
	Watts	% Maximum Workload (W_{max}) Completed ^(a)		
Sleep	20	8	7.5	0.45
Sitting	30	12	9	0.54
Light exercise	80	32	25	1.5
Heavy exercise	160	64	50	3
(a) Maximum workload = 250 W				
(\dot{V}_E) = ventilation minute volume; B = ventilation rate				

Table 1.2. Ventilation parameters by age and gender (based on ICRP Publication 66, Table B.15, Roy et al., 1994)

Activity						
	Max, %	Age	Gender	f_R	V_T	(\dot{V}_E)
Sleep	8	5 y		23 ^(c)	0.174 ^(c)	0.24 ^(c)
		30 y	Male	12 ^(f)	0.625 ^(f)	0.45 ^(f)
			Female	12 ^(f)	0.444 ^(f)	0.32 ^(f)
Sitting	12	5 y		25 ^(c)	0.213 ^(c)	0.32 ^(c)
		30 y	Male	12 ^(f)	0.750 ^(f)	0.54 ^(f)
			Female	14 ^(f)	0.464 ^(f)	0.39 ^(f)
Light Exercise	32	5 y		39 ^(d)	0.244 ^(d)	0.57 ^(d)
		30 y	Male	20 ^(e,f)	1.25 ^(e,f)	1.5 ^(e,f)
			Female	21 ^(e,f)	0.992 ^(e,f)	1.25 ^(e,f)
Heavy Exercise	64	5 y		-	-	-
		30 y	Male	26 ^(e,f)	1.923 ^(e,f)	3.0 ^(e,f)
			Female	33 ^(e,f)	1.364 ^(e,f)	2.7 ^(e,f)

(a) ICRP (1975)
 (b) Scherrer (1981)
 (c) Taussig et al. (1977)
 (d) Godfrey et al. (1971)
 (e) Cooper and Weiler-Ravell (1984)
 (f) Cotes (1979)
 f_R = frequency, V_T = tidal volume, (\dot{V}_E) = ventilation minute volume

Table 1.3. Recommended values of daily ventilation rates for dosimetric modeling: children in general population (based on ICRP Publication 66, Table B.16A, Roy et al., 1994)

Age	Indoors				Outdoors		Total	ICRP (1975)		
	Asleep		Awake		Travel, etc. ^(e)					
	$h d^{-1}$	$m^3 h^{-1}$	$h d^{-1}$	$m^3 h^{-1}$	$h d^{-1}$	$m^3 h^{-1}$				
3 mo	17	0.09	7	0.19				0.780		
1 y	14	0.15	9	0.31 ^(a)	1	0.31 ^(a)	5.2	3.780		
5 y	12	0.25	9	0.49 ^(a)	3	0.49 ^(a)	8.76			
10 y	10	0.31	11	0.87 ^(a)	3	0.87 ^(a)	15.28	14.784		
15 y (Male)	10	0.42	11	0.93 ^(c)	3	1.89 ^(d)	20.10			
15 y (Female)	10	0.35	12	0.85 ^(c)	2	1.01 ^(b)	15.72			

(a) Ventilation rate: 1/3 sitting + 2/3 light exercise
 (b) Ventilation rate: 1/2 sitting + 3/8 light exercise + 1/8 heavy exercise
 (c) Ventilation rate: 1/2 sitting + 1/2 light exercise
 (d) Ventilation rate: 2/3 light exercise + 1/3 heavy exercise
 (e) Includes travel by car, train, bus, etc. and time in the open air, sports, etc.

Table 1.4. Values of daily ventilation rates for dosimetric modeling: adults over 17 y in general population (based on ICRP Publication 66, Table B.16B, Roy et al., 1994)

Location and Activity	Housewife	Sedentary workers				Outdoor workers		
		Male		Female				
	Duration of activity and ventilation rates							
		h d ⁻¹	m ³ h ⁻¹	h d ⁻¹	m ³ h ⁻¹	h d ⁻¹	m ³ h ⁻¹	h d ⁻¹
Indoors								
At home								
Sleep	8.5	0.32	8.5	0.45	8.5	0.32	8.5	0.45
Housework, free time, etc.	13.5	0.96 ^(a)	7	1.18 ^(a)	9.5	0.96 ^(a)	7	1.18 ^(a)
Total air breathed, m ³ d ⁻¹	15.68		12.09		11.84		12.09	
Elsewhere								
Miscellaneous	1	0.96 ^(a)	6.5	1.18 ^(a)	4	0.96 ^(a)	1	1.18 ^(a)
Total air breathed, m ³ d ⁻¹	0.96		7.67		3.84		1.18	
Total air breathed indoors, m ³ d ⁻¹	16.64		19.76		15.68		13.27	
Outdoors								
Heavy work ^(d)							6	1.69 ^(b)
Travel, sports, etc. ^(e)	1	1.00 ^(c)	2	1.21 ^(c)	2	1.00 ^(c)	1.5	1.21 ^(c)
Total air breathed outdoors, m ³ d ⁻¹	1.00		2.42		2.00		11.96	
Grand total, m ³ d ⁻¹	17.64		22.18		17.68		25.23	
ICRP (1975), m ³ d ⁻¹	21.12	22.80		21.12				
ICRP (1987), UNSCEAR (1982, 1988)				18.975				

(a) Ventilation rate: 1/3 sitting + 2/3 light exercise
 (b) Ventilation rate: 7/8 light exercise + 1/8 heavy exercise (Monod and Flandrois, 1985)
 (c) Ventilation rate: 1/2 sitting + 3/8 light exercise + 1/8 heavy exercise
 (d) The mean ventilation rate estimated for underground miners engaged in heavy manual work is not significantly higher than the 1.18 m³ h⁻¹ assumed for sedentary workers (see ICRP Publication 66 Section B.76).
 (e) Includes travel by car, train, bus, etc. and time in the open air, sports, etc.

Table 1.5. Recommended values of daily ventilation rates^(a) for dosimetric modeling: adult workers (based on ICRP Publication 66, Table B.17, Roy et al., 1994)

Activity	Sedentary Worker		Heavy Worker
	Male	Female	Male
Sleeping (8 h)	3.6 (3.6) ^(b)	2.6 (2.9)	3.6 (3.6)
Occupational (8 h)			
1/3 sitting	9.6 (9.6)	7.9 (9.1)	
2/3 light exercise			
7/8 light exercise			13.5 (9.6)
1/8 heavy exercise			
Nonoccupational (8 h)			
1/2 sitting			
3/8 light exercise	9.7 (9.6)	8.0 (9.1)	9.7 (9.6)
1/8 heavy exercise			
Total air breathed, m ³	22.9 (22.8)	18.5 (21.1)	26.8 (22.8)

(a) Volumes of air (m³) breathed in a working day.
 (b) Values in parentheses from ICRP Publication 23 (ICRP, 1975).

Table 1.6. Ventilation minute volumes and total daily air (ICRP Publication 23, ICRP, 1975)

	Male	Female	10 y	1 y	Newborn
8 h working, L min ⁻¹	20	19	13		
8 h nonoccupational, L min ⁻¹	20	19	13	4.2 (10h)	1.5 (1h)
8 h resting, L min ⁻¹	7.5	6	4.8	1.5 (14h)	0.5 (23h)
Total air, m ³ d ⁻¹	22.8	21.12	14.78	3.78	0.78

Table 1.7. Ventilation parameters from UNSCEAR (1982) and ICRP Publication 50 (ICRP, 1987)

	UNSCEAR (1982)	ICRP (1987)
Indoors 19 h	5.5 h at 20 L min ⁻¹	19 h at 0.75 m ³ h ⁻¹
	8 h at 7.5 L min ⁻¹	
	5.5 h at 12.5 L min ⁻¹	
Outdoors 5 h	2 h at 20 L min ⁻¹	1.0 m ³ h ⁻¹
	3 h at 12.5 L min ⁻¹	
Total air, m ³ d ⁻¹	18.975	19.25

Table 1.8. Daily time budget and ventilation parameters at each exercise level

		Sleep	Sit	LE ^(a)	HE ^(a)	Total	L min ⁻¹
5-year old							
Time budget ^(b)	12 h @ sleep	h	12				
	12 h @ 1/3 sit, 2/3 LE	h		4	8		
	Total	h	12	4	8		24
	Fraction (time)		0.5	0.17	0.33		1
Breathing rate ^(c)	<i>B</i>	m ³ h ⁻¹	0.24	0.32	0.57		
	volume	m ³	2.88	1.28	4.56		8.72
	Fraction (volume)		0.33	0.147	0.523		1
Adult male							
Time budget ^(d)	8.5 h @ sleep	h	8.5				
	13.5 h @ 1/3 sit, 2/3 LE	h		4.5	9		
	2 h @ 1/2 sit, 3/8 LE, 1/8 HE	h		1	0.75	0.25	
	Total	h	8.5	5.5	9.75	0.25	24
	Fraction (time)		0.354	0.229	0.406	0.010	1
Breathing rate ^(c)	<i>B</i>	m ³ h ⁻¹	0.45	0.54	1.5	3.0	
	Volume	m ³	3.825	2.97	14.625	0.75	22.17
	Fraction (volume)		0.172	0.134	0.660	0.034	1
ICRP Publication 71		m ³	3.60	3.24	14.63	0.75	22.2
Adult female							
Time budget ^(d)	8.5 h @ sleep	h	8.5				
(e)	14 h @ 1/3 sit, 2/3 LE	h		4.67	9.33		
(e)	1.5 h @ 1/2 sit, 3/8 LE, 1/8 HE	h		0.75	0.5625	0.1875	
	Total	h	8.5	5.42	9.89	0.19	24
	Fraction (time)		0.354	0.226	0.412	0.008	1
Breathing-rate ^(c)	<i>B</i>	m ³ h ⁻¹	0.32	0.39	1.25	2.7	
	volume	m ³	2.72	2.11	12.36	0.51	17.70
	Fraction (volume)		0.154	0.119	0.698	0.029	1

a LE = light exercise, HE = heavy exercise

b Table 3

c Table 2

d Table 4

e In Table 4 'housewife' has 14.5 h and 1 h respectively at these two combinations of exercise levels. The 'female sedentary worker' has 13.5 h and 2 h respectively. I have taken the average.

Table 1.9. Ventilation rates (L min⁻¹) according to exercise level

Study	Ventilation rates (L min ⁻¹) according to exercise level							
<i>ICRP Publication 66</i> (ICRP, 1994a)	Sleep	Sitting	Light exercise (LE)	Heavy exercise				
Workload (W)	20	30	80	160				
Age 5 yrs	4	5.3	9.5					
Male	7.5	9	25	50				
Female	5.3	6.5	21	45				
Anderson et al. (1985)	Resting	Light	Moderate	Heavy				
Workload (adult male) W		<50	50-100	>100				
Age 5 yr (M)	6.5	13.9	33.3	33				
Male	12	14	41	80				
Female	6	8	27	48				
Samet et al. (1993)	Resting	Reading	Bicycling					
Male		17.4	87.8					
Female	12.5	13.9						
Johnson et al. (1995)	Slow - Sleeping		Slow - Awake		Medium		Fast	
	GM	GSD	GM	GSD	GM	GSD	GM	GSD
Children	6.5	1.2	7.5	1.1	9.2	1.1	11.1	1.2
Adults	9.2	0.9	12.1	1.0	14.6	1.0	32.1	1.4

Table 1.10. Number of hours per day spent at different exercise levels

Study	No. of hours spent at each exercise level				
<i>ICRP Publication 66</i> (ICRP, 1994a)	Sleep	Sitting	Light exercise (LE)	Heavy exercise	
Workload (W)	20	30	80	160	
5-yr-old	12	4	8		
Adult male	8.5	5.5	9.75	0.25	
Adult female	8.5	5.4	9.89	0.19	
INSEE (1989)					
5-yr-old (M)	10.8	8.9	3.1	0.7	
5-yr-old (F)	10.8	9.4	3.8	0.2	
Adult (M)	9.8	5.0	8.2	1.0	
Adult (F)	10.3	4.6	9.4	0.2	
Anderson et al. (1985)		Low	Medium	High	
Workload (W) (adult male)		<50	50-100	>100	
All age groups		22.4	1.4	0.22	
Layton (1993)	Sleep	Light	Moderate	Heavy	V Heavy
	7.2	14.8	1.2	0.6	0.2

Table 1.11. Daily average ventilation rates (L min^{-1})

Reference	Ventilation rates (averaged over a day) L min^{-1}							
	5-yr-old			Adult male			Adult female	
<i>ICRP Publication 23</i> (ICRP, 1975)				15.8			14.7	
<i>ICRP Publication 30</i> , UNSCEAR (1982, 1988)				13.2				
Layton								
Dietary ^a	8.30			10.4			7.1	
BMR ^a	9.0 (3 to <10 yr old)			11.5			7.6	
Activity-time budget ^a	--			12			9.7	
<i>ICRP Publication 66</i> (ICRP, 1994a)	6.1			15.4			12.3	
Final estimates	x_5	x_{50}	x_{95}	x_5	x_{50}	x_{95}	x_5	x_{50}
	5	6.1	9	10	15.4	18	6.5	12.3
								15

a) Layton (1993) calculated ventilation rates based on daily energy expenditure in 3 ways;
 1) using average daily intakes of food energy from dietary surveys (Dietary)
 2) average daily energy expenditure calculated from ratios of total daily expenditure to basal metabolism. (BMR)
 3) daily energy expenditures determined from a time-activity survey (Activity-time budget)

2. Questions 2-4. Deposition in the Respiratory Tract

Again the approach used was to take the IRTM as a starting point. It is particularly appropriate for this task in that it is age-specific, and because PC implementations are available which enable deposition to be determined under a wide range of user-specified conditions. LUDEP (Jarvis et al., 1993), which is published, enables this to be done for adult males and subsequently tissue doses to be calculated. AGEDEPX developed by the authors of LUDEP allows age-specific deposition fractions to be determined, but not doses. Thus it is relatively straightforward to determine the effects on deposition of selecting parameter values different from the ICRP reference values. With regard to deposition, *ICRP Publication 66* considers in detail inter-subject variability, and gives formulae to derive confidence intervals for it, but does not address uncertainty directly.

Deposition refers to the initial processes that determine how much of the material in the inspired air remains behind after expiration. The deposition model evaluates the fraction of the inhaled particles deposited in each region for aerosol sizes of practical interest (0.6 nm – 100 μm). The model has been developed in detail for particulate materials whose size does not change within the respiratory tract, i.e., materials that are non-volatile and insoluble: here termed “stable”. This is partly a reflection of the state-of-the-art:

most experimental studies of total and regional deposition in the human respiratory tract have been conducted with stable materials for practical reasons, e.g., to provide reproducible results. Similarly, most theoretical modeling of respiratory tract deposition relates to stable materials: it is more straightforward and so more advanced than for materials which change in size. From a practical stand-point it also provides deposition fractions that are of wide application: the deposition fractions apply to all radionuclides in all chemical forms, provided it can be assumed that entry and regional deposition in the respiratory tract of a given subject are governed only by the size distribution of the aerosol particles. Thus *ICRP Publication 66* in Annex F provides regional deposition fractions for selected subjects and aerosol sizes only for stable particles. Similarly, LUDEP and AGEDEPX only apply to stable particles.

The most important exceptions to stable materials are hygroscopic particles, as may well be the case here. Water-soluble materials, such as many salts, absorb water in the very high relative humidity within the respiratory tract and as a result grow in size and change deposition characteristics.

Deposition fractions of gases and vapors are determined entirely by their chemical forms: see *ICRP Publication 66*, Section 6 for general approach and *ICRP Publications 68*

and 71 for specific materials, notably iodine (ICRP, 1994a, 1994b, 1996).

A summary description of the treatment of deposition in the IRTM follows. Its application to the questions here is then considered.

2.1 The IRTM deposition model

A full description is given in *ICRP Publication 66*, Annex D (James et al., 1994). Relevant aspects are summarized here. Particle deposition in the respiratory tract, and the mechanisms that determine regional deposition have been, and continue to be, extensively studied. However, most experimental data are for adult Caucasian males, and for stable particles with aerodynamic diameter, d_{ae} , between 1 and 10 μm . Furthermore, all that can be measured directly is total deposition, and its sub-division between extrathoracic airways, ET (head and larynx), and thoracic airways (lungs).

Deposition in the ET regions was determined empirically. Deposition measurements in men have been related to characteristic parameters of particle size and airflow. The resulting deposition efficiencies are scaled by anatomical dimensions to predict deposition in women and children.

Experimental determination of regional deposition within the lungs, i.e., between the tracheo-bronchiolar (TB) airways, and the pulmonary (AI) region, have relied on the assumption that material deposited in the two regions can be distinguished by different clearance rates. Following deposition of insoluble radiolabelled particles in the human lung, two distinct phases of clearance are usually observed. It was long generally assumed that the fast phase, which is completed within about a day, represents the mucociliary clearance of all the particles deposited in the airways, and the second, much slower phase, clearance of particles deposited in the alveolar region (Albert and Arnett, 1955). This assumed one-to-one correspondence has been used as the basis for measurements of regional deposition in the TB and AI regions (Lippmann et al., 1980; Stahlhofen et al., 1980). However, as discussed below, while recent studies support the view that all material deposited in the AI region is cleared slowly, there is a strong possibility that a substantial fraction of the TB deposit is not cleared quickly. Furthermore, it is not currently feasible to distinguish bronchial (BB) from bronchiolar (bb) deposition experimentally. The IRTM therefore relies on a theoretical approach to estimate deposition in each region, and to take account of the subject's lung size and breathing rate: in particular to estimate deposition fractions for children.

For the thoracic airways the IRTM uses a theoretical model of gas transport and particle deposition (Pack et al., 1977; Egan and Nixon, 1985, 1987; Nixon and Egan, 1987; Egan et al., 1989) to calculate activity deposition in each of the BB, bb, and AI regions, and to quantify the effects of the subject's lung size and breathing rate. Regional deposition fractions are calculated for aerosols having log-normal particle size distributions, with geometric standard deviations (σ_g) 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 (*ICRP Publication 66*, Paragraph 170). Values of the deposition fractions are given in *ICRP Publication 66* as functions of particle size, for each age of subject, for sleep, sitting, light exercise and heavy exercise.

2.2 Application to Questions 2-4

Subjects

It is first necessary to specify the input parameters relating to both the subject and the aerosol. The distribution of time spent between the four levels of exercise are as determined in Question 1 above. We consider the subject at each age (5-year-old and adult) to be a male-female average. Habitual mouth breathers are included (see breathing mode below).

Aerosol

We were instructed to consider aerosols with Activity Median Aerodynamic Diameters (AMAD) of 1 μm (core question) and also 0.1 and 10 μm (subsidiary questions), and that the material is hygroscopic. As in *ICRP Publication 66*, I have assumed an initial particle density, ρ , of 3 g cm^{-3} , and shape factor χ , of 1.5. Changes to these parameters within the respiratory tract are considered under the heading of hygroscopic growth, below. I recognize that the subjects will spend much of their time indoors. It seems likely that the concentration indoors will be lower than outdoors to an extent depending on particle size, and the ventilation characteristics of the building. I would expect losses entering the dwelling and/or other buildings to be highest, and hence the reduction in concentration greatest, at those sizes for which deposition mechanisms are most effective: below 0.1 μm , and increasing with decreasing size; and above 1 μm , and increasing with increasing size. I would expect this to have most effect on the 10 μm AMAD aerosol, for which I would expect the concentration to be markedly lower indoors, and also the AMAD to be lower. Time constraints prevent my attempting any quantitative evaluation of this, however.

Inhalability

Moving air

Inhalability (aspiration efficiency) is defined in the IRTM as the ratio of the particle concentration in the air entering the nose or mouth to that in the inspired volume of ambient air. Particles of d_{ae} less than a few microns follow the airstream into the nose or mouth, so the inhalability is ~1.0. When an airstream changes direction the inertia of larger particles makes them continue to travel in the direction in which they were going. Thus when facing into the wind the concentration entering the respiratory tract is increased, and in other directions the concentration is decreased. As the effect increases, we move to a situation where the particles are blown in, rather than sucked in. Hence inhalability tends to increase with increasing wind speed, so that it can be >1.0. Similarly, inhalability tends to decrease with increasing ventilation rate, since the amount of material in the inspired volume increases, although this is not treated as a variable in the IRTM.

There have been several studies of the inhalability of particles as functions of d_{ae} , windspeed and orientation to the wind (Ogden and Birkett, 1977; Armbruster and Breuer, 1982, 1984; Vincent and Mark, 1982; Vincent et al., 1990). These studies involved sampling aerosols through the nose or mouth orifices of a life-size model of a person (head and torso) in a wind tunnel. Vincent et al. (1990) reviewed the published data and summarized the measured inhalability, averaged over 360° relative to the wind direction. At low windspeeds, inhalability decreases with increasing particle size, reaching approximately 0.5 for d_{ae} larger than about 20 μm . For very large (>50 μm d_{ae}) particles at high windspeeds, inhalability increases with particle size, and may even exceed unity.

The IRTM uses a function based on the review of Vincent et al. (1990), which decreases from unity for d_{ae} less than about 5 μm , to about 0.5 at about 30 μm and above. (It takes account of the increase in inhalability for larger particles at high wind speeds, but that is outside the scope of the situations considered here.) The IRTM assumes inhalability is independent of age and breathing rate.

Still air

Breysse and Swift (1990) published the only study of inhalability in still air. They measured nasal aspiration efficiency of radiolabelled particles in four subjects at normal resting breathing rates. The measured aspiration efficiency fell from about 0.7 at 20 μm d_{ae} to about 0.3 at

30 μm . The IRTM function, which is based on the moving air measurements, gives good agreement at 20 μm , but overestimates the aspiration efficiency measured at 30 μm d_{ae} .

Thus for this exercise, the IRTM model should give a good estimate of inhalability, which is only likely to be an important issue for the 10 μm AMAD aerosol. Uncertainty in inhalability is unlikely to have any significant effect at 0.1 or 1 μm , but at 10 μm could have a significant input into uncertainty in both total and extrathoracic (ET) deposition. It is unlikely to affect lung deposition, since particles that penetrate through ET to the lungs, are mainly the smaller particles ($d_{ae} < 5 \mu\text{m}$), which are unaffected by inhalability.

Breathing habit

The head warms, humidifies and filters the inspired air, thus protecting the thoracic airways. Breathing at rest usually occurs through the nose, at least in adults. However, during physical exercise and speech, oral breathing supplements airflow through the nose.

Since the filtration efficiencies of the nose and mouth are different, a subject's breathing habit affects the amount of inhaled material that deposits in ET and in the lungs. Niinimaa et al. (1980, 1981) found that, in studies of 30 healthy young adults, 20 subjects switched to oro-nasal breathing, typically at a ventilation rate of about 2.1 $\text{m}^3 \text{ h}^{-1}$. Five subjects continued to breathe through the nose even when exercising vigorously. Four subjects, who were habitual "mouth breathers", breathed oro-nasally (through the nose and mouth together) at all levels of exercise. The remaining subject showed no consistent pattern!

In accordance with a review by Miller et al. (1988), the IRTM uses the distribution of air between nose and mouth measured by Niinimaa et al. for "normal augmenters," nose-breathers who switch to oro-nasal breathing at moderate exercise, and "mouth-breathers," as given in Table 2.1.

The proportion of air breathed through the nose during oro-nasal breathing in Table 2.1 seems surprisingly high, since the resistance of the mouth appears to be so much lower than the nose. Recently, however, Malarbet et al. (1994) have conducted studies similar to those of Niinimaa et al., and found broadly similar results in adults. ICRP Publication 66 assumed that, in the absence of specific data, the same breathing habits apply to healthy young subjects as apply to normal, healthy adults. Roy et al. (1995) also studied 10 children (aged 7 – 17 y). The main finding of significance here is that they observed that the proportion of

habitual mouth breathers was higher in children than in adults (9/10). I have therefore calculated deposition patterns for nose- and mouth-breathers separately below.

2.3 Total Deposition

There have been a considerable number of measurements of total deposition of particles in the human respiratory tract. Such measurements can be made rapidly with trace amounts of non-toxic materials and hence total deposition has been determined as functions of particle size and various breathing parameters. Some data are even available for children (Becquemin et al., 1991; Schiller-Scotland et al., 1992; 1994). These measurements are reviewed in Annex D of *ICRP Publication 66*, which in turn refers to a series of reviews of total and regional respiratory tract deposition in the literature. Notably, Heyder and Rudolf (1984) presented a review of deposition models, and noted that all current deposition models gave close agreement with current experimental data. Thus total deposition is reasonably well characterized, but the database is somewhat limited in the context of this study. In particular, most measurements have been made with the subject inhaling the aerosol through a mouthpiece. This reduces experimental variability, and is convenient for studying lung deposition and relating the results to models, but means that the results cannot be applied directly to practical exposure situations. Furthermore most results are for stable (non-hygroscopic) particles, with d_{ae} in the range 1–10 μm , inhaled by healthy adult males. Their main value in this context is thus to validate models over a range of conditions, so that they can be applied with confidence over the much wider range required. Hence the approach I have adopted is to use the IRTM as a basis.

At 0.1 and 1 μm AMAD, deposition is incomplete. Using the IRTM, total deposition at 0.1 μm is typically about 40%, largely due to diffusion, and at 1 μm about 50%, but mainly due to sedimentation and impaction. Deposition in each region is determined mainly by the deposition efficiency of that region. At 10 μm , however, the deposition efficiency of each region is high; total deposition is close to 100% of the activity inhaled, but is reduced by inhalability. Thus deposition occurs predominantly in ET, and the fraction deposited in each successive region is largely determined by the fraction of inhaled activity that penetrates to it.

2.4 Regional deposition

Experimental regional deposition data were recently reviewed by Stahlhofen et al. (1989) as part of the development of the IRTM. There have been many

measurements of nasal deposition of particles with $d_{ae} > 0.1 \mu\text{m}$. These show considerable variation between subjects, but good agreement between study averages. This is reflected in the good agreement between the IRTM prediction of nasal deposition, and that of the TGLD (1966). It is noted by Stahlhofen et al. (1989) that natural nasal breathing consistently gave somewhat higher deposition than the artificial technique (constant flow, in through nose and out through mouth), used in the early studies. Because of the experimental difficulties, there have been few measurements of deposition in the head during natural mouth breathing. In most studies subjects inhale through a mouthpiece.

Measurements of extrathoracic deposition in the various studies are not rigorously comparable since different experimental procedures, in particular different mouthpieces have been used, and the dimensions and position of the tube can influence the measured deposition. Generally, deposition is much lower than for nose-breathing. Most of the ET deposition of particles $< 10 \mu\text{m}$ d_{ae} inhaled through a mouthpiece occurs in the larynx (Emmett and Aitken, 1982; Rudolf et al., 1983; Stahlhofen et al., 1980, 1983, 1984). Dennis (1961) and Swift (1985) observed that normal mouth breathing tends to give higher ET deposition than when the same subjects breathe through a mouthpiece. Their results indicate that deposition in the oral cavity during natural mouth breathing depends on the position of the tongue and the degree to which the mouth is open. Hence, *ICRP Publication 66* concludes that the experimental data are not sufficient to model quantitatively deposition in the mouth and oropharynx itself. The uncertainty in ET deposition for mouth breathing is clearly much greater than for nose breathing.

Again, regional deposition within the lungs has been studied quite extensively, by several groups, notably GSF, Frankfurt (e.g., Stahlhofen et al., 1980) and New York University Medical Center (NYU) (e.g., Lippmann et al., 1980). As noted above, experimental determinations of regional deposition within the lungs, i.e., between the tracheo-bronchiolar (TB) airways, and the pulmonary (AI) region, have relied on the assumption that material deposited in the two regions can be distinguished by different clearance rates. Imaging techniques cannot currently distinguish the two, especially at the bronchiolar level. The same general approach has been used by all groups. Following inhalation of insoluble radiolabelled particles, retention of activity in the chest is followed for a few days. Usually two distinct phases of clearance are observed. It is assumed that the fast phase, which is completed within about a day, represents the mucociliary clearance of all the particles deposited in the

airways (TB), and the second, much slower phase, clearance of particles deposited in the AI region. The slow phase may be determined by the fraction of the initial lung deposit remaining at e.g., 6 or 24 hours, or by taking the intercept of the slow phase.

Experimental regional deposition data were recently reviewed by Stahlhofen et al. (1989). Because of the requirement to use radiolabelled particles, the database has similar limitations as that for total deposition, but is more restricted. Furthermore, there appear to be systematic differences between datasets from different laboratories. In particular, the NYU dataset, which is perhaps the most comprehensive in terms of numbers of subjects, and hence provides the best indication of inter-subject variation, gives consistently higher fast-cleared fractions and hence TB deposition, than the GSF dataset, which is the most comprehensive with regard to relating deposition to breathing parameters. The slow-cleared fraction and hence alveolar deposition, is correspondingly lower in the NYU dataset than in the GSF data. The IRTM is broadly consistent with the GSF data. Stahlhofen et al. (1989) and *ICRP Publication 66* discuss several possible reasons for the difference, but the latter considers it is most likely to be due to hygroscopic growth of the iron oxide particles used at NYU, as a result of the presence of residual chloride. As noted elsewhere, a major problem with interpreting the experimental data, which applies to all studies, is the possibility of a substantial, size-dependent slow phase of TB clearance. This is included in the IRTM, and its inclusion results in higher TB deposition and lower AI deposition than would be inferred from the experimental data without it.

2.5 Hygroscopic growth

The main problem with evaluating the deposition values (total and regional) required here is that the material is taken to be hygroscopic. This is a reasonable assumption, given the presence of metal salts, and likely association with ambient environmental aerosol, some of which is hygroscopic (e.g., sulfate). However, estimation of the deposition of hygroscopic aerosols is not straightforward. The topic is covered in *ICRP Publication 66* (Section 5.6.2, and Annex D, Section D.12), and recently reviewed by Hiller (1991). Hygroscopic materials absorb water vapor in the warm, humid (close to 100% relative humidity) conditions in the respiratory tract. As a result, they can grow rapidly to several times their original size. The resulting deposition in the respiratory tract is then complicated by this growth (Morrow, 1986), the rate of which depends on several parameters, including relative humidity, temperature, particle size, and particle

composition. In the absence of hygroscopic growth, 1 mm diameter particles have a relatively low deposition probability in the lungs (10 – 20%). During a breath, however, hygroscopic particles of 1 μm initial diameter may grow to approximately 4 μm diameter, resulting in much greater lung deposition. On the other hand, 0.1 μm diameter particles that grow to 0.5 μm will have lower deposition by diffusion.

Total lung deposition of hygroscopic aerosols has been measured in humans (Hänel and Heyder, 1980; Blanchard and Willeke, 1984; Tu and Knutson, 1984; Hicks et al., 1986). It is generally found that dry particles in the size range 0.2 – 2.0 μm grow enough to increase deposition substantially, while growth of smaller particles tends to decrease deposition. Growth factors have been found to be in the range 3 – 7.

The only published measurements of regional deposition of hygroscopic aerosols in human subjects were made recently (Pritchard et al., 1994). Monodisperse sodium chloride particles of diameter 0.25 and 0.48 μm , in which an insoluble radiolabel (^{123}I -iodohexadecane) was incorporated, were inhaled by 5 healthy men. Results are given in Table 2.2, as presented by the authors, assuming all TB clearance to be rapid. Estimates are also given on the assumption as in the IRTM for these sizes, that only 50% of the TB deposit clears rapidly, i.e., the TB deposit is twice that given by Pritchard et al., and the AI deposit correspondingly reduced. Also given are deposits predicted by the model of Xu and Yu (1985), as given by Pritchard et al., and predictions of the IRTM (see below). Deposition is far higher than would be expected for stable particles of those sizes, and indeed ET and TB deposition are considerably higher than the model predictions assuming a growth factor of 6.

Several authors have developed models for predicting respiratory tract deposition of hygroscopic particles (Ferron, 1977; Martonen et al., 1982; Xu and Yu, 1985; Ferron et al. 1993). *ICRP Publication 66* (Section D.12) suggests an approach for including hygroscopic growth within the IRTM. However, I could not see any way of applying any of these rigorously within the short time-scale of this exercise, especially given the wide range of conditions (aerosol sizes, ages, exercise levels). (Hygroscopic growth is not implemented in LUDEP or AGEDEPX). Furthermore because the inhaled material is of such uncertain composition, it would be difficult to estimate the input parameters required to apply any model: rate of growth and final size (growth factor). Inspection of Figures D34–D37 in *ICRP Publication 66*, Annex D, shows that hygroscopic

growth can have a marked effect on deposition, in particular increasing BB and bb deposition of 1 μm AMAD aerosols (the central question) by up to about a factor of three (These figures are for NaCl, which might be an extreme case). It has less effect on 0.1 or 10 μm aerosols.

In order to calculate deposition fractions for hygroscopic aerosols, without a model, my approach was been to assume hygroscopic growth takes place between ET and TB. I calculated deposition in ET for a 1 μm AMAD aerosol (as in *ICRP Publication 66*, I assumed an initial particle density, ρ , of 3 g cm^{-3} , and shape factor χ , of 1.5.) I assumed a growth factor of three, following the discussion in *ICRP Publication 66* (Section D.12) on growth of iron oxide particles coated with ferric chloride solution. This seemed a reasonable analogue for a mixed composition aerosol. However, it is the physical (geometric) size of the particles which increases by the growth factor. The aerodynamic diameter may change differently because of changes in density (typically down to about 1 g cm^{-3} , for water) and shape factor (down to 1.0, for spherical droplets). The thermodynamic diameter d_{th} approximates to the physical diameter, and for the aerosol specified above the AMTD (activity median thermodynamic diameter) is 0.68 μm . Applying a growth factor 3 increases the AMTD to 2.0 μm , but taking account of the changes in density and shape factor, the AMAD also becomes 2.0 μm . I therefore calculated deposition in TB (BB and bb) and AI regions for an aerosol with AMAD 2.0 μm , density 1 g cm^{-3} , and shape factor 1.0. I used AGEDEPX to obtain deposition fractions for an aerosol inhaled with these characteristics, and then corrected them for the different penetration (1 – deposition) in ET for a 1 μm rather than 2 μm AMAD aerosol. Thus if $DE_{ET}(1)$ and $DE_{ET}(2)$ are the fractions of inhaled 1 and 2 μm AMAD aerosols as specified above deposited in ET, then the deposition fractions in BB, bb and AI regions for a 2 μm inhaled aerosol were multiplied by $(1 - DE_{ET}(1))/(1 - DE_{ET}(2))$. Similar calculations were performed for 0.1 μm AMAD aerosols. For 10 μm AMAD aerosols, however, a different approach was used, because nearly all particles that enter the nose and mouth deposit: total deposition is determined by inhalability. Thus deposition in ET and lung were taken to be as for a 10 μm AMAD aerosol, but the distribution between BB, bb, and AI was that for droplets assuming a growth factor of 3 (21 μm AMAD aerosol).

A spreadsheet was set up to perform these calculations, since values were required for each subject (5-year-old, adult male, adult female; nose- or mouth-breather) at each exercise level (sleep, sitting, light exercise, and for adults, heavy exercise). The spreadsheet was then used to combine the results at each age, first for a weighted average of nose-

and mouth-breathers. This was assumed to be 10% and 75% nose-breathers for children and adults respectively. The results for 1 μm AMAD aerosols are shown in Tables 2.3 – 2.5. Simplified versions are given as Tables 2.6 – 2.8. These tables also provide useful indications of how deposition (according to the IRTM) varies with size, age, exercise level, breathing mode, and hygroscopic growth.

A weighted average for each subject was then obtained according to the distribution of time spent at each exercise level (Table 1.8). Note however that the deposition fractions needed to be weighted according to the volume fraction inhaled at each exercise level, also given in Table 1.8. The weighted daily averages are given in Table 2.9, for all three sizes, and with the adult male-female averages. (A simplified version for 1 μm aerosols is given in Table 2.10.) These provide the central (50%) values for Questions 2 – 4. In assessing the 5% and 95% values I considered both uncertainty due to model input parameters, which could be assessed from the spreadsheets, since they give results for a variety of input parameter values, and how uncertain the model itself might be. In this context the comparison of model predictions with the one set of experimental measurements (Table 2.2) is instructive. Measured deposition in ET and TB were much higher than predicted, suggestion that significant particle growth occurred within ET; that the growth factor was greater than expected; and/or that the model underestimates deposition in these regions.

Question 2. Total initial deposition in the respiratory tract, % of total amount inhaled, normal daily (24 hr) mix of activities?

At 0.1 μm , total deposition for the four exercise levels, nose- and mouth-breathers, adult male and female, ranged only from 19 – 27%. Deposition in the child is also similar, even though predominant mouth-breathing is assumed. However, hygroscopic growth made a large difference. Total deposition of a stable 0.1 μm AMAD aerosol was calculated to be about 45%. Thus uncertainty about the extent and effect of hygroscopic growth seems to dominate. There is a minimum in total respiratory tract deposition of about 10% that occurs at an AMAD of about 0.5 μm for stable particles, and around 0.1 μm for NaCl (Tu and Knutson, 1984). It is feasible that hygroscopic growth could move total deposition to this minimum. Hence the 5% value is set at 10%. Conversely the experiment summarized in Table 2.2 suggests that 0.25 μm NaCl particles can grow sufficiently to give total deposition of about 70%, which is more than would be predicted. Our aerosol starts smaller, and is unlikely to grow as much as NaCl, so I have taken 60% to be the 95% value.

At 1 μm , as shown in Tables 2.3 – 2.8 from the spreadsheet, total deposition at 1 μm for adults under various conditions ranged from 33 – 57%, and for children 35 – 60%. Hygroscopic growth was calculated to increase total deposition moderately: typically 10 – 25%. Given that most models are able to predict total deposition satisfactorily, it is unlikely that it would be much less than predicted for stable aerosols i.e., about 40% in adults and 35% in children. Again the 95% value is dominated by uncertainty about the extent and effect of hygroscopic growth. The experiment summarized in Table 2.2 suggests that 0.5 μm NaCl particles can grow sufficiently to give total deposition of about 80%, which is more than would be predicted. Our aerosol starts larger, but is unlikely to grow as much as NaCl, so I have taken 80% to be the 95% value.

At 10 μm AMAD most particles that enter the respiratory tract deposit, and total deposition (~75%) is determined more by inhalability than by deposition within the respiratory tract. There are very few measurements of inhalability in still air, which is the most relevant situation for much of the time, and these are for particles in the range d_{ae} 17 – 30 μm . Thus there is quite a wide range of uncertainty (50% – 90%).

Question 3. Initial deposition in the extrathoracic region, % of total deposition in the respiratory tract? (This question also gives the initial deposition in the lung (tracheobronchial (TB) + pulmonary (AI) regions), since lung = total – ET).

Central values (50%) are taken from Table 2.9: (Deposition in $ET_1 + ET_2$) / Total deposition.

At 0.1 μm , as noted above, uncertainty in deposition is greatly influenced by uncertainty in the effect of hygroscopic growth. This could affect the distribution between ET and lungs. If there is little growth, lung deposition is higher, and ET deposition forms a lower proportion of the total than the central value (25%), around 15%. Conversely, if there was significant growth within ET there could be much higher ET deposition, although lung deposition would also tend to increase. Thus in the experiment (Table 2.2) ET deposition is about 30% of total. Given the lack of information the 5% and 95% values are estimated to be somewhat higher and lower than these (10%, 50%).

At 1 μm , hygroscopic growth has less effect on the distribution between ET and lung than at 0.1 μm . ET was estimated to be about 60% of total for a hygroscopic aerosol and about 50% for a stable 1 μm AMAD aerosol. In the

experiment, it was about 30%, but this was mouth-breathing through a tube. There are reasonably good experimental data for stable aerosols inhaled by adults. Hence this seems relatively well defined. I have taken about 50% uncertainty to derive the 5% and 95% values in adults, and somewhat greater in children.

At 10 μm most of the deposition is in ET (central value 90% of total). Thus small changes in this will have a major impact on lung deposition (central value 10% of total) and it is better to consider the uncertainty in that. From the spreadsheet, there is considerable variation according to the conditions considered. If hygroscopic growth occurred in the nose, this would reduce lung deposition substantially (to 5% of total). Conversely, for mouth-breathers lung deposition can be up to 20% of total.

Question 4. Initial deposition in the tracheobronchial (TB) region, % of total deposition in the lung? (This question also gives the initial deposition in the AI region, since AI = lung – TB).

Central values (50%) are taken from Table 2.9: (Deposition in BB + bb) / (Deposition in BB + bb + AI).

At 0.1 μm , as noted above, uncertainty in deposition is greatly influenced by uncertainty in the effect of hygroscopic growth. This could affect the distribution between TB and AI. However, calculated values decrease in a similar way with growth. In the experiment (Table 2.2) TB deposition was estimated by the authors, assuming no slow TB clearance, at about 40% of lung. However, if it is assumed that 0.5 of the TB deposit clears quickly, then TB deposition becomes about 80% total lung. This illustrates the particularly great uncertainty associated with TB deposition and clearance. There are relatively few measurements of regional deposition of stable particles at this size. Given these uncertainties the 5% and 95% values are estimated to be a factor 3 higher and lower than the central value of 23% (7%, 70%).

At 1 μm , hygroscopic growth increases deposition in both TB and AI. As at 0.1 μm the experiment suggests a higher proportion of TB to lung than the calculated values, possibly much higher. At this size there have been a reasonable number of measurements of regional deposition of stable particles, but there are differences between results from different laboratories, and the problem of possible slow TB clearance affecting interpretation of results. Given these uncertainties the 5% and 95% values are estimated to be a factor 2 higher and lower than the central value of 29% (15%, 60%), for adults, and a factor 3 for children, since

their results have to be extrapolated from adults and there is more uncertainty about the extent of mouthbreathing.

At 10 μm hygroscopic growth increases TB deposition at the expense of AI. Without growth, TB deposition is calculated to be about 40% of lung, compared to the central value of 63%. More rapid growth would increase it further. At this size there have been a few measurements of regional deposition of stable particles, and there is less uncertainty

about possible slow TB clearance affecting interpretation of results, since it is considered that the extent of slow clearance decreases with increasing size and would have relatively little effect here. Given these uncertainties, the 5% and 95% values for deposition in AI are estimated to be a factor 2 higher and lower than the central value of 37% (20%, 80%), and hence the 5% and 95% values for deposition in TB are estimated to be 20% and 80%.

Table 2.1. Percentage of total ventilatory airflow passing through the nose in normal nasal augmenters (nose breathers) and in mouth breathers^(a) (ICRP Publication 66, Table 11)

Level of exercise	$F_n, \%$	
	Normal nasal augmenter	Mouth breather
Sleep	100	70
Rest (sitting)	100	70
Light exercise	100	40
Heavy exercise	50	30

(a) Derived from Miller et al. (1988).

Table 2.2. Regional deposition of hygroscopic aerosols: comparison with experiment

	Particle size, μm	Growth factor	ET	TB		AI		Total	Reference
				a	b	a	b		
Measured	0.25		24	18	36	28	10	70	Pritchard et al., 1994
	0.48		25	23	46	31	8	79	
Predicted	0.25 ^c	1	3	5		22		30	Xu and Yu, 1985
	0.48 ^c	1	3	13		35		51	
	0.25 ^d	6	1	3		10		14	IRTM
	0.48 ^d	6	1	2		10		13	
	1.5 ^d	6	1	4		24		29	
	3.0 ^d	6	6	12		40		57	
	6.0 ^e		24	24		32		80	
	7.0 ^e		31	26		25		81	
	8.0 ^e		37	26		18		81	

a) As given by Pritchard et al., based on fast and slow phases being equivalent to TB and AI.
 b) Assuming only 0.5 of TB cleared quickly.
 c) $\rho = 2.17$, $\chi = 1.5$ i.e., no growth.
 d) For 0.25 and 0.48 d_{th} respectively, assuming growth of d_{th} by factor 6, $\rho = 1.0$, $\chi = 1.0$ (i.e., water droplets).
 e) $\rho = 1.0$, $\chi = 1.0$.

Table 2.3. Deposition (% inhaled) of 1 μm AMAD hygroscopic aerosol. 5-y-old child

	Nose-breather			Mouth-breather			Average ^a
	1 μm ^b	2 μm ^b	1 μm hyg	1 μm ^b	2 μm ^b	1 μm hyg	
Sleep							
ET ₁	13.71	22.36	13.71	8.01	13.66	8.01	8.58
ET ₂	17.07	28.02	17.07	11.08	20.49	11.08	11.68
BB	0.91	1.11	1.55	1.31	2.14	2.63	2.52
bb	2.43	1.86	2.59	2.94	2.91	3.58	3.48
AI	12.00	10.69	14.91	14.52	15.07	18.52	18.16
Total	46.12	64.04	49.84	37.86	54.27	43.81	44.41
ET	30.78	50.38		19.09	34.15		
1-ET	0.69	0.50		0.81	0.66		
Sitting							
ET ₁	15.71	24.67	15.71	9.29	15.26	9.29	9.93
ET ₂	19.86	31.07	19.86	13.01	23.01	13.01	13.70
BB	0.97	1.21	1.76	1.51	2.56	3.22	3.08
bb	2.05	1.48	2.15	2.57	2.50	3.15	3.05
AI	11.72	9.83	14.31	14.63	14.61	18.39	17.98
Total	50.31	68.26	53.80	41.01	57.94	47.06	47.73
ET	35.57	55.74		22.30	38.27		
1-ET	0.64	0.44		0.78	0.62		
Light Exercise							
ET ₁	20.18	29.35	20.18	5.43	8.85	5.43	6.91
ET ₂	26.33	37.28	26.33	10.82	21.21	10.82	12.37
BB	1.13	1.41	2.26	3.06	5.69	6.81	6.36
bb	1.43	0.95	1.52	2.46	2.87	3.44	3.25
AI	7.99	5.88	9.43	13.04	13.56	16.24	15.56
Total	57.06	74.87	59.72	34.81	52.18	42.74	44.44
ET	46.51	66.63		16.25	30.06		
1-ET	0.53	0.33		0.84	0.70		

a 1 μm AMAD hygroscopic: 10% nose-breathers, 90% mouth-breathers.

b Non-hygroscopic aerosols.

Table 2.4. Deposition (% inhaled) of 1 μm AMAD hygroscopic aerosol. Adult male

	Nose-breather			Mouth-breather			Average ^a
	1 μm ^b	2 μm ^b	1 μm hyg	1 μm ^b	2 μm ^b	1 μm hyg	
Sleep							
ET ₁	9.50	16.90	9.50	5.39	10.00	5.39	8.47
ET ₂	11.27	20.77	11.27	7.09	14.43	7.09	10.23
BB	0.82	1.15	1.46	1.07	1.92	2.22	1.65
bb	3.17	3.16	4.02	3.69	4.39	5.08	4.28
AI	14.49	14.70	18.69	16.51	18.67	21.62	19.42
Total	39.25	56.68	44.93	33.75	49.41	41.41	44.05
ET	20.77	37.67		12.48	24.43		
1-ET	0.79	0.62		0.88	0.76		
Sitting							
ET ₁	10.49	18.28	10.49	6.00	10.90	6.00	9.37
ET ₂	12.62	22.58	12.62	7.93	15.76	7.93	11.45
BB	0.87	1.25	1.63	1.19	2.19	2.57	1.86
bb	2.80	2.68	3.48	3.32	3.88	4.55	3.75
AI	15.10	14.83	19.28	17.46	19.32	22.67	20.13
Total	41.88	59.62	47.50	35.90	52.05	43.73	46.56
ET	23.11	40.86		13.93	26.66		
1-ET	0.77	0.59		0.86	0.73		
Light Exercise							
ET ₁	17.50	26.61	17.50	4.52	7.72	4.52	14.26
ET ₂	22.51	33.79	22.51	8.40	17.22	8.40	18.98
BB	1.30	1.80	2.73	3.26	6.49	7.53	3.93
bb	1.47	1.19	1.80	2.51	3.29	3.82	2.31
AI	9.94	8.01	12.13	15.23	16.57	19.22	13.91
Total	52.72	71.40	56.67	33.92	51.29	43.49	53.38
ET	40.01	60.40		12.92	24.94		
1-ET	0.60	0.40		0.87	0.75		
Heavy Exercise							
ET ₁	8.78	13.32	8.78	4.18	6.76	4.18	7.63
ET ₂	14.56	25.11	14.56	9.81	20.03	9.81	13.37
BB	4.97	8.75	10.89	6.16	11.26	13.23	11.48
bb	1.93	2.47	3.08	2.28	3.13	3.68	3.23
AI	10.46	9.94	12.38	11.79	11.90	13.98	12.78
Total	40.70	59.59	49.69	34.22	53.08	44.88	48.48
ET	23.34	38.43		13.99	26.79		
1-ET	0.77	0.62		0.86	0.73		

a 1 μm AMAD hygroscopic: 10% nose-breathers, 90% mouth-breathers.

b Non-hygroscopic aerosols.

Table 2.5. Deposition of 1 μm AMAD hygroscopic aerosol. Adult female

	Nose-breather			Mouth-breather			Average ^a
	1 μm ^b	2 μm ^b	1 μm hyg	1 μm ^b	2 μm ^b	1 μm hyg	
Sleep							
ET ₁	8.81	15.92	8.81	4.98	9.36	4.98	7.85
ET ₂	10.34	19.43	10.34	6.52	13.50	6.52	9.39
BB	0.79	1.04	1.30	1.00	1.67	1.92	1.45
bb	3.90	3.94	4.93	4.47	5.29	6.07	5.21
AI	14.00	14.43	18.05	15.78	17.98	20.63	18.69
Total	37.84	54.76	43.42	32.75	47.80	40.11	42.60
ET	19.15	35.35		11.50	22.86		
1-ET	0.81	0.65		0.89	0.77		
Sitting							
ET ₁	9.95	17.54	9.95	5.67	10.41	5.67	8.88
ET ₂	11.91	21.65	11.91	7.53	15.17	7.53	10.82
BB	0.83	1.13	1.45	1.09	1.91	2.23	1.65
bb	3.31	3.20	4.11	3.86	4.46	5.20	4.38
AI	12.82	12.92	16.60	14.71	16.62	19.38	17.30
Total	38.82	56.44	44.03	32.86	48.57	40.01	43.02
ET	21.86	39.19		13.20	25.58		
1-ET	0.78	0.61		0.87	0.74		
Light Exercise							
ET ₁	17.75	26.87	17.75	4.60	7.82	4.60	14.46
ET ₂	22.85	34.10	22.85	8.61	17.59	8.61	19.29
BB	1.26	1.72	2.62	3.18	6.29	7.32	3.79
bb	1.54	1.18	1.80	2.57	3.21	3.74	2.28
AI	9.93	7.95	12.10	15.33	16.66	19.38	13.92
Total	53.33	71.82	57.11	34.29	51.57	43.65	53.75
ET	40.60	60.97		13.21	25.41		
1-ET	0.59	0.39		0.87	0.75		
Heavy Exercise							
ET ₁	9.20	13.75	9.20	4.40	7.03	4.40	8.00
ET ₂	15.38	26.13	15.38	10.46	21.04	10.46	14.15
BB	5.11	8.85	11.10	6.36	11.44	13.54	11.71
bb	1.89	2.29	2.87	2.22	2.91	3.44	3.02
AI	10.17	9.53	11.96	11.52	11.49	13.60	12.37
Total	41.75	60.55	50.51	34.96	53.91	45.45	49.24
ET	24.58	39.88		14.86	28.07		
1-ET	0.75	0.60		0.85	0.72		

a 1 μm AMAD hygroscopic: 10% nose-breathers, 90% mouth-breathers.

b Non-hygroscopic aerosols.

Table 2.6. Regional deposition (% inhaled) of 1 μm AMAD hygroscopic aerosol in 5-year-old

	Nose-breather			Mouth-breather			Nose/Mouth Average ^a
	1 μm	2 μm	1 μm hyg	1 μm	2 μm	1 μm hyg	
Sleep							
ET	31	50	31	19	34	19	20
TB	3.3	3.0	4.1	4.2	5.1	6.2	6.0
AI	12	11	15	15	15	19	18
Total	46	64	50	38	54	44	44
Sitting							
ET	36	56	36	22	38	22	24
TB	3.0	2.7	3.9	4.1	5.1	6.4	6.1
AI	12	10	14	15	15	18	18
Total	50	68	54	41	58	47	48
Light Exercise							
ET	47	67	47	16	30	16	19
TB	2.5	2.4	3.8	5.5	8.5	10.3	9.6
AI	8	6	9	13	14	16	16
Total	57	75	60	35	52	43	44

Table 2.7. Regional deposition (% inhaled) of 1 μm AMAD hygroscopic aerosol in adult male

	Nose-breather			Mouth-breather			Nose/Mouth Average ^a
	1 μm	2 μm	1 μm hyg	1 μm	2 μm	1 μm hyg	
Sleep							
ET	21	38	21	12	24	12	19
TB	4.0	4.3	5.5	4.8	6.3	7.3	5.9
AI	14	15	19	17	19	22	19
Total	39	57	45	34	49	41	44
Sitting							
ET	23	41	23	14	27	14	21
TB	3.7	3.9	5.1	4.5	6.1	7.1	5.6
AI	15	15	19	17	19	23	20
Total	42	60	48	36	52	44	47
Light Exercise							
ET	40	60	40	13	25	13	33
TB	2.8	3.0	4.5	5.8	9.8	11.4	6.2
AI	10	8	12	15	17	19	14
Total	53	71	57	34	51	44	53
Heavy Exercise							
ET	23	38	23	14	27	14	21
TB	6.9	11.3	14.0	8.4	14.4	16.9	14.7
AI	10	10	12	12	12	14	13
Total	41	60	50	34	53	45	48

Table 2.8. Regional deposition (% inhaled) of 1 μm AMAD hygroscopic aerosol in adult female

	Nose-breather			Mouth-breather			Nose/Mouth Average ^a
	1 μm	2 μm	1 μm hyg	1 μm	2 μm	1 μm hyg	
Sleep							
ET	19	35	19	12	23	12	17
TB	4.7	5.0	6.2	5.5	7.0	8.0	6.7
AI	14	14	18	16	18	21	19
Total	38	55	43	33	48	40	43
Sitting							
ET	22	39	22	13	26	13	20
TB	4.1	4.3	5.6	5.0	6.4	7.4	6.0
AI	13	13	17	15	17	19	17
Total	39	56	44	33	49	40	43
Light Exercise							
ET	41	61	41	13	25	13	34
TB	2.8	2.9	4.4	5.8	9.5	11.1	6.1
AI	10	8	12	15	17	19	14
Total	53	72	57	34	52	44	54
Heavy Exercise							
ET	25	40	25	15	28	15	22
TB	7.0	11.1	14.0	8.6	14.4	17.0	14.7
AI	10	10	12	12	11	14	12
Total	42	61	51	35	54	45	49

Table 2.9. Regional deposition fractions (% inhaled) for 0.1, 1 and 10 μm AMAD hygroscopic aerosols, normal (24 h) mix of activities

AMAD (μm)	Region	Child	Adult Male	Adult Female	Adult Average
0.1	ET1	1.96	2.67	2.70	2.69
	ET2	3.37	3.23	3.27	3.25
	BB	0.86	0.60	0.65	0.63
	bb	4.11	3.43	3.80	3.62
	Al	13.91	14.28	13.81	14.05
	Total	24.22	24.21	24.23	24.22
1	ET1	7.90	12.38	12.59	12.49
	ET2	12.34	16.28	16.61	16.45
	BB	4.61	3.52	3.41	3.47
	bb	3.29	2.87	3.00	2.94
	Al	16.77	15.65	15.01	15.33
	Total	44.91	50.69	50.62	50.66
10	ET1	19.50	28.69	28.73	28.71
	ET2	41.89	38.27	38.37	38.32
	BB	6.00	3.94	3.77	3.86
	bb	1.77	1.36	1.38	1.37
	Al	3.97	3.20	3.06	3.13
	Total	73.13	75.45	75.31	75.38

Table 2.10. Daily average regional deposition of 1 μm AMAD hygroscopic aerosols

AMAD (μm)	Region	Child	Adult Male	Adult Female	Adult Average
	BB	6.00	3.94	3.77	3.86
	bb	1.77	1.36	1.38	1.37
	Al	3.97	3.20	3.06	3.13
	Total	73.13	75.45	75.31	75.38

3. Questions 5-6 Particle Retention in the Respiratory Tract

Question 5. Assuming completely insoluble particles (1 μm AMAD), retention in each respiratory tract region in mature adults as a % of the total initial deposition in the respiratory tract, as a function of time after deposition?

The general approach taken has been to use the IRTM as a starting point. This is the aspect of the IRTM on which I took the lead, and the aspect of this exercise on which I consider myself to be better informed than on any other. The IRTM clearance model is summarized in *ICRP Publication 66*, Section 7 and in detail in *ICRP Publication 66*, Annex E (Bailey and Roy, 1994). Annex E is almost exclusively concerned with particle transport, which is the subject of this question, as opposed to absorption to blood, the subject of Question 6, which is considered in more detail in *ICRP Publication 71* (ICRP, 1996). The following is therefore largely taken from (an advanced draft) of Annex E. However, I have largely left out material relating to the ET regions and lymph nodes, and most of the material relating to modifying factors, other than cigarette smoking. The IRTM reference parameter values were chosen for healthy non-smokers. Cigarette smoking is a modifying factor which applies to a significant fraction of the population and has a significant effect on clearance. Annex E considers uncertainty, variability and modifying factors, as outlined below. Since I was largely involved in selecting the central values and uncertainties there, I have largely drawn on that material here, with two exceptions: (i) I consider here a population containing habitual smokers. I estimate 25% to be the proportion who have smoked enough to have impaired lung clearance. (ii) In a few cases I have taken into account relevant information that has become available since the IRTM was finalized, and which affects the outcome of the questions. In such cases, the item starts "RECENTLY,..." in order to identify it.

3.1 Introduction

Annex E does not provide a complete review of all aspects of clearance of inhaled materials from the respiratory tract, since that was too great a task, and comprehensive reviews of specific aspects were available in the literature. The objective was to review the information on which the IRTM clearance model is based. Section E.1 is an introduction. Section E.2 outlines the clearance mechanisms involved. Section E.3 aims to provide support for, and indicate the limitations of, the general approach used in the model. Sections E.4 to E.7 review the information on which the individual parameters in the particle transport model for the

human respiratory tract were chosen, with information relating to the uncertainty and variability associated with each value, and the effects of factors that might alter it significantly for specific groups of the population.

Selection of reference values for particle transport

As far as possible the parameters in the model are based on observations on humans, since particle transport rates are known to vary markedly between species (Felicetti et al., 1981; Snipes et al., 1983; Bailey, 1989; Bailey et al., 1989; Kreyling, 1990). Ideally each reference value would be based on carefully conducted studies designed to measure that parameter. The uncertainty in the reference value would then be given by the 95% confidence interval on the mean and the variability by the 95% limits on intersubject variation. However, few human lung clearance parameters have been measured directly in this way. It is noted that the particle transport model is not intended to be used for fibres or other materials, such as quartz, whose physicochemical characteristics might interfere with normal clearance mechanisms.

Variability

Three clearance rates for which the distributions have been obtained in humans are described in the relevant sections: mucociliary transport in the posterior nasal passage; mucociliary transport in the trachea; and alveolar clearance at 200 d after inhalation. In each case most measurements conformed well to a log-normal distribution, with similar geometric standard deviations (σ_g): 1.6, 1.8, and 1.7, respectively. However, for nasal and alveolar clearance, about 20% of cases showed clearance slower than predicted by such a distribution. It is not surprising that a similar degree of variation should be seen in mucociliary clearance rates in the nasal passage and trachea, since similar mechanisms are involved. It is interesting, however, that a similar distribution occurs for alveolar clearance, which is determined by the behavior of alveolar macrophages. This result gives support to the proposition, made below, that a similar distribution be assumed for those clearance rates that have not been measured directly.

For a log-normal distribution with median x_{50} , approximately 95% of values lie between x_{50}/σ_g^2 and $x_{50} \cdot \sigma_g^2$. The observed distributions of human particle transport rates suggest a typical value for σ_g of 1.7, and hence a value for σ_g^2 of about 3. It is therefore proposed that, in the absence of specific information, intersubject variation in any clearance rate be represented by a log-normal distribution with x_{50} equal to the reference value,

and $\sigma_g = 1.7$. This gives 95% confidence limits at $x_{50}/3$ and $3x_{50}$.

Uncertainty

In general, reference values are based on indirect information and therefore involve a considerable element of judgement. Some rates are based entirely on data from animal studies, and extrapolating results to humans involves a possible systematic error, which could be large. For example, the alveolar clearance rate at 100 d after inhalation in humans is about 5 times greater than in dogs, and 5 times lower than in mice (Bailey 1989; Kreyling, 1990). Maximum mucociliary transport rates in the trachea ranged from 2 mm min^{-1} in rats to 10 mm min^{-1} in dogs (Felicetti et al., 1981). These are all, however, within a factor of 2.5 of the human value (5 mm min^{-1} , Section E.5.2.3). Thus the combination of a high degree of intersubject variation and possible systematic errors means that the uncertainties in the reference values are large and difficult to quantify. It is therefore assumed that the uncertainty in the reference value is log-normally distributed, and thus that there is a 95% probability that the true mean value lies within a factor Φ_u of the chosen reference value.

The values selected for Φ_u depend on the quality of information available, and are in general derived in the relevant sections. However, the two examples of interspecies differences described above suggest that a factor of 3 is reasonable whenever a rate is based entirely on animal data.

Modifying factors

While it would be expected that many of the parameters in the clearance model will be altered by factors such as age, smoking, and disease, only in a few cases have the effects of such factors been measured. Moreover, it would not be expected that a particular agent or condition would affect all clearance rates equally, and therefore they are discussed for each parameter individually in the relevant section. In cases where it is possible to quantify an effect on a parameter, a modifying factor Φ_m is recommended by which the parameter should be multiplied in order to calculate doses for specific individuals or groups. However, it should not be applied to the parameter(s) describing absorption into blood, since any effect on absorption could be quite different.

It is also necessary to distinguish between two general types of modifying factors: (1) transient factors, such as exercise, which apply only during exposure to the factor, with a time-

scale of days or less, and are relevant only to acute exposures; (2) permanent factors, such as sex or a chronic disease, which are relevant to chronic exposures. As noted above, only cigarette smoking is considered here.

3.2 Clearance Mechanisms

Inhaled substances deposited in the airways are cleared by several mechanisms and pathways, which lead mainly to the GI tract, the lymphatic system, and the blood. These mechanisms have been, and continue to be, intensively studied. Only an outline description is given in Annex E since a number of comprehensive reviews of the literature exist (e.g., Brain et al., 1977; Lauweryns and Baert, 1977; Green et al., 1977; Lippmann et al., 1980; Pavia et al., 1980; Camner, 1980, 1984; Raabe, 1982; Stuart, 1984; Brain, 1985, 1986; Schlesinger, 1985; Sturgess, 1985; Bowden, 1987; Cuddihy and Yeh, 1988; Morrow, 1988; Oberdörster, 1988; Peterson, 1989; Snipes, 1989). These clearance mechanisms can be grouped into two general processes, which in the IRTM are assumed to be independent and competitive:

Particle transport mechanisms transport material to the GI tract and lymph nodes, and from one part of the respiratory tract to another. Particles are removed from ET and TB by secretions and ciliary action, and from the AI region by free phagocytic cells. The mechanisms operate primarily on particulate material, but in the IRTM the term includes the transport of dissociated material along these routes, which can arise through binding to fluids or uptake by cells.

The term absorption is used to refer to the transport of material to the blood, regardless of the mechanism involved. Generally it applies to the dissociation of particles, and the uptake into blood of soluble substances and material dissociated from particles.

Particle transport in the conducting airways

Mucociliary clearance is the most important clearance mechanism throughout the conducting airways, moving particles from the nasal passage and tracheobronchiolar (TB) region (trachea, bronchi, and bronchioles, down to the terminal bronchioles) to the pharynx, where they are swallowed. It may be assisted at times by coughing and sniffing. However, not all particles deposited on the ciliated airways are removed promptly by mucus. There is growing evidence that some are taken up by airway macrophages, and that a very small fraction is retained in the epithelium itself. Clearance of insoluble particles from the oral passage occurs by swallowing, and it is thought that particles are

cleared from the anterior nasal passage (ET₁) mainly by nose blowing and wiping (Fry and Black, 1973; Swift and Proctor, 1988).

Mucociliary clearance

In parts of the extrathoracic airways (ET₂, posterior nasal passage and larynx) and throughout the TB, the ciliated epithelium moves airway secretions, prevents most particles from reaching airway surface cells, and is the main mechanism for their removal to the GI tract.

While a traditional view has developed of a continuous mucous sheath covering the airways and moving toward the pharynx, it has long been recognized that the flow is not uniform around the circumference of each airway, particularly at bifurcations. Hilding (1932, 1957a,b, 1959, 1963) has described mucociliary streaming in the nasal passage, larynx, trachea, and bronchi. Van As and coworkers observed, using *in vitro* preparations of rat airways, that, in the small airways, mucus consisted of sparse, discrete particles (4 μm to 10 μm). In the bronchi it formed flakes 100 μm across, which aggregated to form streams in the main bronchi. Even in the trachea, the streams did not form a continuous layer (Iravani and van As, 1972; van As and Webster, 1974; van As, 1977, 1980). Other workers could not confirm the discontinuous nature of the mucous layer. Luchtel (1976, 1978) reported that it forms a continuous layer in the rabbit and the rat, from the trachea to the bronchioles, but becomes progressively thinner distally. Sturgess (1977a,b, 1985) also reported it to be continuous in the human and rabbit trachea, but with a more open network in the bronchi. Wolff (1986), in discussing the question, reported his own observation that on 25% of the surface area of the trachea of normal rats there was little or no mucous glycoprotein.

Coughing

Coughing aids mucociliary clearance that is impaired by abnormal or pathological conditions, although the mechanism is only effective when secretions are increased (Camner, 1984). To expel secretions effectively from the walls of airways, coughing must produce a linear air velocity of 5 m s^{-1} . After the 7th and 8th generations, the airflow is theoretically too low to produce effective coughing (Leith, 1968). There are probably no cough receptors at this level (Camner, 1981). In healthy dogs, repeated coughing impaired normal tracheal clearance markedly for several hours (Smaldone et al., 1979).

Airway macrophages

Recent observations indicating that a significant fraction of particles deposited in the TB may be subject to delayed clearance have focused attention on the airway macrophage as a possible mechanism involved in this pathway (Brain et al., 1984a; Geiser et al., 1990a,b). Sorokin and Brain (1975) observed many macrophages in the bronchial tree of mice. While some were suspended in the mucus, others were beneath it, attached to the bronchial epithelium. The concentration of macrophages per unit surface area in the airways is similar to that in the alveoli (Lehnert and Sanz-Rodrigues, 1988). Oberdörster (1988) considered that particle phagocytosis by airway macrophages might therefore be a more likely explanation for a slow phase of TB clearance than endocytosis by epithelial cells, discontinuities in the mucous blanket, or lack of epiphase in the bronchioles. Brain (1988) has discussed the possible protective functions of airway macrophages.

Stirling and Patrick (1980) reported that at 2 h after instillation of BaSO₄ particles onto the trachea of rats, most particles remaining on the epithelial surface were within macrophages. Geiser et al. (1990a,b) found that in the intrapulmonary airways of hamsters that inhaled 6- μm -diameter polystyrene latex (PSL) particles (a size expected to deposit mainly in the airways), 40% of retained particles were in macrophages at 20 min after inhalation, and 90% at 24 h. Many other particles were in close contact with epithelial cells, and it was suggested that surface tension forces may draw particles through the mucus onto the epithelial surface (Schürch et al., 1990; Gehr et al., 1990a,b).

Retention in airway walls

There is evidence that a small fraction of particles deposited in the nasal passage, and the TB, are retained in the airway wall, from studies on several species, including humans, and for a variety of materials. At 1 – 7 d after instillation onto the rat trachea, BaSO₄ particles retained in the airway wall were within macrophages (Stirling and Patrick, 1980; Patrick, 1983) and were mainly concentrated just beneath the basement membrane (Gore and Patrick, 1982). There is also evidence for phagocytosis of inhaled iron oxide particles by tracheal epithelial cells in the mouse (Watson and Brain, 1979).

Takahashi and Patrick (1987a) found that between 1 week and 6 months after instillation of ¹³³Ba-labelled BaSO₄ particles onto the rat trachea, the ¹³³Ba cleared from the tracheal wall with a half-time of 88 d. By injecting colloidal

carbon into the tracheal wall, they determined that it drains into the internal jugular and cervical lymph nodes (Takahashi and Patrick, 1987b). Bryant et al. (1953) and Berg et al. (1954) measured the uptake of ^{198}Au -labelled colloidal gold (3- to 4-nm diameter) into the hilar lymph nodes of dogs following instillation into the lumen, or injection into the submucosa of a bronchus. Masse et al. (1974), following observations of the retention of tantalum and iron oxide particles by monkeys, considered that particles phagocytosed in the bronchial lumen were ultimately removed by lymphatic clearance.

Particle transport in the alveolar-interstitial region

In the respiratory bronchioles and alveoli many structures contribute to clearance: airway secretions, alveolar surfactant, and epithelial cells (Green, et al., 1977; Lauweryns and Baert, 1977; Camner, 1980; Brain, 1985). Deposited substances may be moved toward the mucociliary escalator, the interstitium, lymphatics, and blood capillaries, or may be retained in the tissues and lymph nodes.

The most important clearance mechanism involves the alveolar macrophage (AM), whose main function is phagocytosis, which keeps the alveolar surface free of foreign substances (see Brain, 1985, 1986, 1988). Alveolar macrophages are large mononuclear cells that make up more than 95% of the free cell population of the alveolar region. They originate from bone marrow precursor cells and arrive as monocytes in the lungs, where they can mature and multiply in the interstitium or in the alveolar space (Oberdörster, 1988).

AM phagocytosis is the intake of large particles, including bacteria and cell fragments. It is one of the processes by which a cell internalizes material; the others are endocytosis and pinocytosis. In endocytosis, a small region of the plasma membrane folds inward until it has formed a new intracellular, membrane-limited vesicle about 0.1 mm in diameter. This mechanism is used for the specific internalization of macromolecules that are bound to cell surface receptors. Pinocytosis refers to the nonspecific uptake of small droplets of extracellular fluid to form pinocytic vesicles. By these processes, bacteria and particulate matter can be absorbed very rapidly (in 2 – 20 min) (Sorokin, 1977; Brain, et al., 1984b).

The rate of phagocytosis of particles by AM depends upon properties such as size, surface composition, and density. Hahn et al. (1977) reported that rabbit AM phagocytized 2.2- μm FAP in vitro more rapidly than 0.3- μm FAP, and denser PuO_2 particles more rapidly than FAP of the same

size. Camner and Lundborg (1977) demonstrated, in vitro, a higher rate of phagocytosis for carbon-coated than for silver-coated Teflon particles. They also showed a toxic effect of beryllium-coated particles that resulted in inactivation and death of the macrophages. However, Camner et al. (1977) found similar lung clearance of carbon, silver, and beryllium-coated particles between 1 d and 8 d after inhalation by rabbits, suggesting that the differences in rate of phagocytosis observed in vitro did not affect clearance in vivo.

After ingestion of particles, and lysosomal digestion (Lundborg et al., 1984), the macrophage may store the particles or release them into intercellular fluids. The loaded macrophages may be removed from the alveoli via the mucociliary escalator, a pathway that may be oriented by chemotaxis to the bronchial lumen (Lauweryns and Baert, 1977; Oberdörster, 1988). Some researchers consider that AM may penetrate into the interstitium, and hence reach the lymphatics and the lymph nodes (Corry et al., 1984; Harmsen et al., 1985; Gillett et al., 1989). However, others believe that it is mainly free particles that follow this route (Lauweryns and Baert, 1977; Lehnert et al., 1986). Particles may reach the interstitial space by endocytosis and subsequent exocytosis by type I, and to a lesser extent type II, epithelial cells (Sorokin and Brain, 1975; Lauweryns and Baert, 1977; Oberdörster, 1988).

Movement to the interstitium may be greater for very small particles. Takenaka et al. (1986) found that, after chronic inhalation by rats, fly ash particles (1 – 2 μm diameter) were mostly in AM, while TiO_2 (primary particles 0.02 – 0.03 μm) was mostly in interstitial macrophages. Ferin et al. (1990, 1991) found with both titanium and aluminum oxides greater lung retention in rats for particles with diameters of 0.02 – 0.03 μm , than for particles of 0.2 – 0.5 μm . They suggested that this might be due to greater penetration of the ultrafine particles to the interstitium, and that one function of AM phagocytosis is to reduce toxic effects resulting from particles entering the interstitial space.

Particles may move through the interstitium under the influence of concentration gradients, and of the flow of tissue fluids, which is normally toward lymphatics (LeBouffant, 1971; Lauweryns and Baert, 1977; Leak, 1977, 1980). Large molecules and particles enter lymphatics through the lymphatic endothelium by means of its discontinuous basement membrane. The open intercellular junctions act as one-way flap valves, allowing fluids and particulate matter to enter but not leave the lymphatic capillary lumen (Lauweryns and Baert, 1977).

Interstitial macrophages may take up particles that have entered the interstitium, and transport them toward, and possibly into, lymphatics (Lauweryns and Baert, 1977).

3.3 The IRTM Clearance Model

For convenience, both particle transport and absorption are covered here, although the latter relates to Question 7. The following is based partly on Annex E and partly on an advanced draft of *ICRP Publication 71*, since this is the most up-to-date description of the IRTM clearance model I have helped to draft.

The general approach used in the IRTM is based on that developed by Cuddihy and his colleagues (Cuddihy, 1976, 1984; Cuddihy et al., 1979; Cuddihy and Yeh, 1988). The model describes several routes of clearance from the respiratory tract, which involve three general processes. Material deposited in ET₁ (the anterior nasal passage) is removed by extrinsic means such as nose-blowing. In other regions clearance is competitive between the movement of particles towards the GI tract and lymph nodes (particle transport), and absorption into body fluids of material from the particles in the respiratory tract. In the IRTM clearance kinetics are expressed in terms of fractional clearance rates, i.e.:

$$\frac{dR_i(t)}{dt} = -\lambda_i(t)R_i(t) + m_{ji}(t)R_j(t)$$

where $R_i(t)$ is the amount of material retained in region i (ET₂, BB, bb or AI) at time t after intake, $\lambda_i(t)$ is the overall instantaneous rate of clearance of material from region i , and $m_{ji}(t)$ is the rate of clearance of material from any region j into region i . Note, however, that as described below, $\lambda_i(t)$ is not a fixed function for the region, but depends on the initial pattern of respiratory tract deposition and the time course of intake.

Several simplifying assumptions are made to provide a systematic basis for predicting human lung clearance kinetics for the wide range of materials required, on the basis of the limited information available, much of which necessarily comes from animal experiments. Supporting evidence is discussed in Section E.3.3. It is assumed in the model that:

1. All the clearance rates are independent of age and sex. (Essentially by default: there is little information available, but what there is suggests that neither factor has a major effect).

2. The clearance rates due to particle transport and absorption to body fluids are independent. Thus the overall rate of clearance from a region is the sum of the rates due to the individual processes:

$$\begin{aligned}\lambda_i(t) &= m_i(t) + s_i(t) \\ &= g_i(t) + l_i(t) + s_i(t)\end{aligned}$$

where $m_i(t)$ and $s_i(t)$ are the clearance rates from region i due to particle transport and absorption, respectively; $g_i(t)$ and $l_i(t)$ are particle transport rates towards the GI tract and regional lymph nodes. In general the rates of clearance from each region, by each route, will change with time after intake, and will be different for material deposited directly in the region during inhalation, and for material cleared into a region following deposition in another region. Indeed, for the latter, the rate of particle transport out of region i depends on the time since the material was transported into the region, but its rate of absorption to body fluids depends on the time since the material originally deposited in the respiratory tract. Thus, $\lambda_i(t)$ and its components in the equations above are themselves dependent on the initial pattern of deposition, the time course of intake and the time course of transport from other regions into region i . To take account of this and to simplify calculations, clearance from each region is represented in the model by a combination of compartments. Each compartment clears at a constant fractional rate, such that the overall clearance approximates the required time-dependent behavior.

3. It is assumed by default that particle transport rates are the same for all materials. A single compartment model is therefore provided to describe particle transport of all materials (Fig. 1). Reference values of parameters used in the IRTM are listed in Table 3.1. Values of rate constants were derived, so far as possible, from human studies, since particle transport rates are known to vary greatly between mammalian species. Fig. 1 as it stands would describe the retention and clearance of a completely insoluble material. However, as noted above, there is in general simultaneous absorption to body fluids of material from all the compartments except ET₁, where it is assumed that none occurs.
4. Absorption into body fluids, which depends on the physical and chemical form of the deposited material, is assumed, by default, to occur at the same rate in all regions (including the lymph nodes), except ET₁.

Absorption is 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.

The simplest compartment model representation of time-dependent dissolution is to assume 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 . Provision is made for two such states, to avoid undue complexity, as it is considered that there would rarely in practice be sufficient information available to justify more. A limitation of this system, however, is that it can only readily represent an overall fractional dissolution rate that decreases with time. To overcome this the *ICRP Publication 66* model uses an equivalent system with the same number of variables, but which gives greater flexibility, shown in Figure 2. In this, 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 . This provides the same results as the former system with the following values:

$$s_p s_s + f_r(s_r - s_s)$$

$$s_{pt} = (1 - f_r)(s_r - s_s)$$

$$s_t = s_s$$

With this system, the initial dissolution rate is approximately s_p and the final dissolution rate is approximately s_t . Thus with 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. In different situations, the 'Particles in transformed state' may represent the residual material following dissolution of a relatively soluble component or surface layer, or material taken up by macrophages. The essential feature is that it remains subject to particle transport. Activity retained in the respiratory tract that is not subject to particle transport is represented by 'bound' material compartments, see below.

The system shown in Fig. 2 applies to each of the compartments in the particle transport compartment model shown in Fig. 1 (except ET_1). Thus from each of the 13 compartments containing 'Particles in initial state', material

moves at a rate s_{pt} to a corresponding compartment containing 'Particles in transformed state'. The 'Particles in transformed state' are cleared by particle transport at the same rates as 'Particles in initial state'. Thus if $m_{j,k}$ is the rate of particle transport from compartment j to compartment k containing 'Particles in initial state' and $m_{j,T,PT}$ is the corresponding particle transport rate for 'Particles in transformed state', then $m_{j,k} = m_{j,T,PT}$ for all j and k .

Uptake to body fluids of dissociated material can usually be treated as instantaneous, but in some situations a significant fraction of the dissociated material is absorbed slowly into body fluids as a result 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 a 'bound' state (Fig. 2), from which it goes into body fluids at a rate s_b , while the remaining fraction ($1 - f_b$) goes to body fluids instantaneously. In the model, material in the 'bound' state is not cleared by particle transport processes, but only by uptake to body fluids. Thus, only one 'bound' compartment is required for each region.

It is recommended that material-specific rates of absorption should be used in the model for compounds for which reliable human or animal experimental data exist. For other compounds, default values of parameters are recommended, according to whether the absorption is considered to be fast (Type F), moderate (M) or slow (S) (corresponding broadly to inhalation Classes D, W and Y in the *ICRP Publication 30* system). Reference values for each are specified in terms of the parameters s_p , s_{pt} and s_t , and are given in Table 3.2. The 'bound' state is not invoked for the default values, i.e., $f_b = 0$ for all three Types.

These absorption rates, expressed as approximate half-times, and the corresponding amounts of material deposited in each region that reach body fluids can be summarized as follows:

Type F: 100% absorbed with a half-time of 10 minutes. There is rapid absorption of almost all material deposited in BB, bb, and AI, and 50% of material deposited in ET_2 . The other 50% of material deposited in ET_2 is cleared to the GI tract by particle transport.

Type M: 10% absorbed with a half-time of 10 minutes and 90% with a half-time of 140 d. There is rapid absorption of about 10% of the deposit in BB and bb; and 5% of material deposited in ET_2 . About 70% of the deposit in AI reaches body fluids eventually.

Type S: 0.1% absorbed with a half-time of 10 minutes and 99.9% with a half-time of 7000 d. There is little absorption from ET, BB, or bb, and about 10% of the deposit in AI reaches body fluids eventually.

For all three absorption Types, all the material deposited in ET₁ is removed by extrinsic means. Most of the deposited material that is not absorbed is cleared to the GI 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.

As an alternative to any of the three default Types defined in *ICRP Publication 66*, instantaneous uptake to body fluids (Type V) may be recommended. This was employed for several compounds in *ICRP Publication 68*, following the descriptions of behavior given in *ICRP Publication 30*. Although consideration has to be given to the total respiratory tract deposition, regional deposition does not need to be assessed for such materials, since for the purposes of dose calculation they can be treated as if they were directly injected into body fluids.

3.4 Tracheobronchial Airways

The "tracheobronchial airways" are taken to consist of the conducting airways within the thorax, i.e., the trachea, bronchi and bronchioles. This corresponds to the tracheobronchial (TB) region, in the model of the ICRP Task Group on Lung Dynamics (TGLD, 1966) on which that of *ICRP Publication 30* (ICRP, 1979) was based. The IRTM divides the tracheobronchial airways into two regions: the Bronchial (BB) (the trachea, generation 0, and the bronchi, generations 1-8) and the Bronchiolar (bb) (generations 9-15). The bronchioles are less than 2 mm diameter and do not have cartilage support. This division was made principally on the basis of consideration of risk. The Task Group that developed the IRTM considered that the bronchi were considerably more sensitive to radiation-induced cancer than the bronchioles. However, there is no clear distinction between the two with respect to deposition and clearance. Thus for the purpose of this exercise it is convenient to consider the two separately only where this is done in *ICRP Publication 66*, that is with respect to model parameters.

Annex E notes that it is convenient in discussing aspects of particle transport to consider the tracheobronchiolar (TB) region as a unit. Particle clearance from TB has been studied extensively. (See, for example, reviews by Wanner, 1977; Pavia et al., 1980, 1983; Yeates et al., 1981a; Lippmann and Schlesinger, 1984; Pavia, 1984; Wolff,

1986.) However, it has not been well characterized and is the subject of some controversy (Foster, 1988). There is general agreement that mucociliary clearance is the principal transport mechanism, that the main flow is toward the pharynx, and that mucus velocities decrease distally, but there is not agreement about the rate at which the decrease occurs, or the pattern of mucus flow, such as the degree of streaming. The region is inhomogeneous: from the trachea to the terminal bronchioles the airways decrease in length and diameter, and the structure of the epithelium and its lining fluid change. It has proved very difficult even to distinguish TB clearance completely from alveolar clearance, and measurements of transport velocity in an individual airway generation are almost exclusively confined to the trachea.

Following deposition of insoluble radiolabelled particles in the thorax, two distinct phases of clearance are usually observed. It has been generally assumed that the fast phase, which is complete within about a day, represents mucociliary clearance of particles deposited in TB, and the slow phase represents clearance of the alveolar deposit (Albert and Arnett, 1955). This assumption forms the basis of experimental measurements of regional deposition (e.g., Lippmann et al., 1980; Stahlhofen et al., 1980). It is supported by observations that the magnitude and speed of the fast phase increase with increasing particle size (Lippmann and Albert, 1969), and with increasing inhalation flow rate (Heyder et al., 1980; Foster, 1988).

There is also, however, increasing evidence to suggest that there may be a significant slow phase of clearance from part, at least, of TB and that, as in the nasal passage, a small fraction of particles deposited in TB may be sequestered in the airway wall. Although representing minor pathways of clearance, these mechanisms can have significant effects on the doses to the sensitive cells in the epithelium, and are therefore included in the model. They are considered in detail below. The fast phase of particle clearance from the lung is here termed lung mucociliary clearance (LMC).

Lung mucociliary clearance (fast phase)

Measurements of whole-lung clearance

In most studies of bronchial clearance, total lung retention of inhaled gamma-tagged particles may be followed in one lung or both. The lung clearance pattern depends greatly on exposure parameters (particle size, breathing pattern), and individual parameters (lung size, age, disease) (Agnew et al., 1986a; Gerrard et al., 1986). Considerable intersubject variation is observed, but clearance in an individual is

relatively constant, as it is in pairs of monozygotic twins (Albert et al., 1973; Camner et al., 1972). Lung mucociliary clearance (LMC) shows several phases: a rapid phase during the first hour; most of the remaining clearance during the next 6 h; but some detectable up to 2 d (Stahlhofen et al., 1981). It cannot in general be well represented by a single exponential function (ICRP, 1979); the use of three components (Yeates et al., 1982) or a power function (Morrow, 1969) is more appropriate, but clearance following an acute intake is often observed to be a discontinuous process, which is not well represented by any simple function (Lippmann, 1969). This behavior has been utilized to measure tracheal mucus velocities, by following the movement of boluses of particles up the trachea.

Since the mucociliary clearance pattern depends so much on exposure parameters and individual characteristics, and is not easily quantified (see below), no attempt was made in Annex E to collate measurements in normal healthy subjects. Instead, results of earlier models of TB mucociliary clearance were used to quantify it. (A large number of studies are however cited in relation to modifying factors.)

A wide variety of endpoints has been used to measure LMC. This may be simply lung retention as a fraction of the initial lung deposit: $R(\tau) = L(\tau)/L(0)$ at time τ . $R(\tau)$ may be given as a function of time, usually up to a few hours after inhalation, since most of the mucociliary clearance is complete in that period; or at one or more specific times (typically 2 h or 3 h). Often the activity associated with the slow phase (assumed to be alveolar or pulmonary) is subtracted (e.g., by deducting 24-h retention), to give "bronchial" retention. This is clearly preferable, especially when investigating factors that can lead to differences in regional deposition as well as clearance. However, if large particles are used so that alveolar deposition is small, total lung retention may still be useful. Alternatively, clearance (1 minus retention) may be presented, and this is used in Annex E, where $CT(\tau)$ and $CB(\tau)$, respectively, represent total and bronchial clearance up to time τ . Characteristic (lung or bronchial) retention times are also employed:

- T_{avg} the average retention time based on the area under the retention versus time plot
- $t_{1/2}$ the retention half-time based on fitting a single exponential function to the retention over some specified time
- T_{50} the time for retention to reach 50% of its initial value (without fitting any function).

Measurements of zonal lung clearance

Retention of inhaled gamma-tagged particles in different parts of the lung has been followed using collimated detectors and gamma-cameras. The fields viewed do not, however, correspond to morphologically distinct regions, except in the largest airways, and observed retention is affected by material clearing from more distal airways. Morrow et al. (1967a) observed clearance half-times of 2.5 – 3 min, and 20 – 30 min with detectors over the trachea and main bifurcation, respectively, and three phases ($t_{1/2}$ 1 – 2 h, 5 h, and 1 – 2 months) with detectors over the rest of the lungs.

In studies with gamma-cameras, the lungs are generally divided into concentric zones about the helium, but different workers have made different divisions and use different terminology. Most often each lung has been divided into three zones: perihilar (inner), intermediate, peripheral (outer) (Sanchis et al., 1972); central (I), intermediate (II), and peripheral (III) (Short et al., 1979); I, II, III (Wilkey et al., 1980). Würtemberger et al. (1987), however, divided each lung into two: central and peripheral, but designated the trachea and main bronchi as a third zone. Agnew et al. (1984) divided each lung into three zones: inner, intermediate, and peripheral, with the trachea and main bronchi as a fourth (central). Since this terminology is the most comprehensive, it is used in Annex E. (In Table 3.10 zonal clearance parameters are distinguished by the suffixes CZ, IZ, MZ, PZ for central, inner, intermediate, and peripheral zone, respectively.).

All lung zones contain some small bronchi, bronchioles, and alveoli, but some of the largest airways are only included in the inner zone; the intermediate zone includes some airways larger than those in the peripheral zone. There are, however, large differences in the zonal clearance patterns of healthy non-smokers reported by different investigators, because of differences in initial deposition patterns, definition of zones and possibly materials (Wilkey et al., 1980).

Measurement of local mucus velocities

Measurements of tracheal mucociliary transport rate (TMTR) have been made by following the movement of individual particles or boluses. Several techniques have been used, but all tend to measure the maximum, rather than the average, clearance rate. Tracer particles have been deposited via a bronchoscope, and their movement measured by cine-photography, or externally by radiography or gamma-camera. TMTR has also been

measured by following the clearance of boluses of inhaled gamma-tagged particles along the trachea with a gamma-camera or collimated detectors (Yeates et al., 1981b). Since the results should not depend on the initial deposition pattern, and there is a simple, common endpoint, mean velocities measured in healthy subjects are given in Table 3.3. Measurements involving bronchoscopes have given results which are more variable and higher than those involving inhalation, perhaps because of the effects of the trauma and anesthetics (Yeates et al., 1981a; Pavia, 1984).

Yeates et al. (1982) found that TMTRs in 74 healthy subjects measured with inhaled particles were log-normally distributed with a median of 4.2 mm min^{-1} and $\sigma_g = 1.8$. Yeates et al. (1975) found that for TMTR the intra-individual short-term coefficient of variation (25%) was considerably less than that between subjects (75%). The latter could result from a combination of genetic and environmental factors.

The study by Foster et al. (1980, 1982), who followed clearance of inhaled particles with a gamma-camera, is unique in that mucus velocities were also measured in the main bronchus. For healthy non-smokers, these averaged $2.4 \pm 0.5 \text{ mm min}^{-1}$ compared to $5.5 \pm 0.4 \text{ mm min}^{-1}$ in the trachea. Tracheal and bronchial velocities measured in the same trial were correlated. The ratio was greater than would be predicted from the ratio of the circumferences of the airways, assuming continuity of flow and mucus of constant depth.

Radiographic measurements of tracheobronchiolar clearance

Gamsu et al. (1973) insufflated tantalum powder into the TB tree of 26 patients. In 18 it was deposited in a sample of all airways (bronchography), and in the others only in the trachea and main bronchi (tracheography). Radiographs were taken immediately and at times up to 15 months. The specified TB tree was divided into regions, and the amount in each was estimated as a fraction of that on the first radiograph. The clearance time determined for each region of the TB (Table 3.4) is greater than that of the region itself, because of the movement into it of material originally deposited in more distal airways, as shown by the shorter times measured by tracheography. Aspects of the technique may have led to results that are not representative of normal clearance. Furthermore the measurements were only semiquantitative. This study is, however, the only one in which clearance in individual airways distal to the main bronchi was directly observed in humans. It supports the view that clearance rates decrease distally and indicates

residence times in the terminal and/or respiratory bronchioles of about a week.

Mucociliary clearance models

Models of bronchial clearance have been developed to estimate mucus velocities throughout TB. Morrow (1974) associated three observed clearance phases (Morrow et al., 1967b) with hilar, intermediate, and peripheral airways, and deduced a mucus velocity in the terminal bronchioles, v_{tb} , of 0.1 mm min^{-1} .

Lee et al. (1979) assumed that all mucus was produced in the terminal bronchioles and was of constant thickness, and hence predicted the velocity (v_α) in generation α , with diameter D_α to be given by:

$$v_\alpha = v_0 D_o / 2^\alpha D_\alpha$$

For a velocity in the trachea of 5.5 mm min^{-1} this gives v_{tb} (generation 15) of $0.005 \text{ mm min}^{-1}$ (Table 3.5).

Yeates et al. (1982) refined this model to fit observed retention curves by introducing an individual-specific factor K ($1 < K < 10$):

$$v_\alpha = K^{1/15} v_0 D_o / 2^\alpha D_\alpha$$

This gave v_{tb} ranging from $0.001 \text{ mm min}^{-1}$ to $0.020 \text{ mm min}^{-1}$.

Yu (1981) treated the TB region as a series of "escalators," one representing each generation i , with an associated velocity v_i . To determine their values, he used a retention curve measured by Lourenço et al. (1971) for $7.7\text{-}\mu\text{m}$ - d_{ae} particles inhaled by a healthy subject. He calculated deposition in each generation and then solved the set of equations for the time to clear each generation. This gave v_{tb} of 0.01 mm min^{-1} . Yu et al. (1986) extended the model calculations to 18 healthy subjects who inhaled particles with d_{ae} in the ranges $4.2 - 4.6 \mu\text{m}$ or $7.4 - 8.1 \mu\text{m}$, to obtain mean mucus velocities for a population (Table 3.5).

Cuddihy and Yeh (1988) used a similar approach to estimate mucus velocities in each airway generation. They calculated deposition in each airway generation for a series of deposition and clearance studies using particles of d_{ae} $1.1 - 9.5 \mu\text{m}$, two breathing patterns, and three subjects (Stahlhofen et al., 1980). On the assumption that the most

proximally deposited material cleared first, they derived clearance times for each generation and hence mucus velocities (Table 3.5). These three models were used to estimate representative clearance rates for the bronchial and bronchiolar regions for the IRTM.

Slow tracheobronchiolar clearance

The main sources of evidence for a significant slow phase of clearance from TB are (1) animal experiments in which particle clearance was measured following deposition at well-defined sites in TB; (2) human inhalation studies in which the fraction of the thoracic deposit clearing in the slow phase was significantly greater than the fraction expected to deposit in the alveolar region.

Animal studies indicating slow tracheobronchiolar clearance

Patrick and Stirling (1977) measured the clearance kinetics of BaSO₄ particles (CMD, 0.35 mm) deposited on the distal trachea of the rat by intratracheal instillation. They found that retention over the first 2 d could be represented by a two-component exponential function: 83% with $t_{1/2}$ 5 min, and 17% with $t_{1/2}$ 16 h. They also found that about 1% of the deposited material was retained for at least 30 d (see below). Similar results were obtained for BaSO₄ administered in suspension in mannitol, or mucus, and with other materials: UO₂ (CMD 0.35 μ m); BaSO₄ (CMD 1.0 μ m) and monodisperse FAP (CMD 1.1 μ m or 5.7 μ m) (Patrick, 1979, 1983). RECENTLY, Patrick has also obtained similar results with colloidal gold (0.02–0.03 μ m).

Svartengren et al. (1981) found a significant slow phase of clearance following intratracheal instillation of Teflon particles (CMD 6 μ m) into rabbits. For particles deposited in the trachea, 39% cleared with $t_{1/2}$ 19 h; and for particles deposited at the first bifurcation, 71% cleared with $t_{1/2}$ 39 h. In a second study, Svartengren and Camner (1984) found a smaller fraction (37%) of particles deposited on the first bifurcation of rabbits was cleared slowly. They also found that the retention half-time of the slow phase was reduced from 30 h to 10 h by administration of the cholinergic drug bethanechloride.

Felicetti et al. (1981) instilled ^{99m}Tc-macroaggregated albumin onto the distal trachea of several species, to measure tracheal mucus velocities. In dogs, an average of 14% of the initial activity remained at the deposition site after 25 min, but in rats, guinea pigs, and rabbits 70–83% remained at an hour.

Wolff et al. (1989) administered a mixed aerosol of 3- μ m and 9- μ m PSL microspheres via a catheter to give deposition primarily in the 6th–10th generation bronchi of dogs. They found similar retention (80% fast clearance; 20% with a half-time of about 20 d) for both sizes.

Spoo et al. (1991) used a similar technique to administer 3-mm-diameter radiolabelled microspheres into the conducting airways of eight dogs, but on separate occasions deposited them in 15-mm, 8-mm, and 4-mm diameter airways. Retention was followed for up to 40 d. Some long-term (>3 d) retention was usually observed, but there was considerable variation between animals exposed in the same size of airway, and also between different airways in the same dog. On average, the fraction retained, and the retention half-time appeared to be greater in the smaller airways. Three dogs exposed in 15-mm airways and one exposed in 8-mm airways apparently cleared all microspheres within 3 d. In other cases the fraction retained varied from 2–84%. The retention half-time was typically of the order of 100 d, but was <30 d in three cases, while in two others no clearance after 3 d could be measured. In a second study, 1.5- μ m mass median diameter polydisperse radiolabelled FAP was administered to 15-mm and 4-mm airways of eight dogs (Snipes et al., 1993). The deposited particles cleared within 3 d from 15-mm sites in seven dogs; and from 4-mm sites in two dogs, with 40–50% of particles retained with half-times of the order of months in the other six dogs. Histological examination of the lungs of three dogs sacrificed 6 d after administration of 3–4 μ m PSL to 15-mm- and 4-mm-diameter airways showed that some particles had moved to the alveoli and were retained there. One possibility is that the microspheres were displaced by peripheral movement of mucus, a phenomenon ("alveolarization") previously proposed to account for slow clearance of tantalum dust deposited in the conducting airways of dogs (Friedman and Tisi, 1972), but which may be related to the high surface concentrations of particles employed in these studies. RECENTLY, Snipes et al. (1995) administered a smaller particle mass as ¹¹¹In-oxide, into 15-mm and 4-mm airways of dogs (four dogs at each size). In seven dogs all radioactivity was below the limit of detection (<3% of deposited particles) at 2 d. In one of the dogs dosed in 4-mm airways, 15% remained more than 3 d. Similarly, Lay et al. (1995) sprayed ^{99m}Tc sulphur colloid radially into 5-mm airways of dogs (segmental bronchus), and found that clearance was ~99% complete at 24 h.

There is some evidence supporting slow TB clearance from inhalation studies, but the results are more difficult to quantify because the initial regional deposition pattern is not well defined. In addition, material measured in the airways

some time after inhalation will result partly from retention at the site and partly from material clearing in transit. Geiser et al. (1990a,b) estimated that, following inhalation by hamsters of 6-mm-diameter PSL (a size expected to deposit mainly in the airways), 14% were retained in the intrapulmonary airways at 24 h. Velasquez and Morrow (1984), however, reported no evidence for prolonged bronchial retention following inhalation of 8- μm - d_{ae} PSL by guinea pigs.

Gore and Patrick (1978) found that, at 14 d after inhalation of UO_2 by rats, the mass in the trachea was 10 times the amount estimated to be in transit from the lung lobes. Gore (1983) reported that at 2 d to 35 d after inhalation of UO_2 by rats, the mass concentration in intrapulmonary airway tissue was about 60% of that in pulmonary tissue, a much higher ratio than would be predicted on the basis of complete TB clearance in 24 h. Similarly, Briant and Sanders (1987), who followed lung clearance of a chain aggregate mixed U/Pu oxide for 85 d after inhalation in rats, considered that the amounts found in extrapulmonary airways were much greater than the expected amounts in transit from the pulmonary region. However, in these experiments, particles sequestered in the airway walls (see below) may well have contributed to the excess. RECENTLY, Patrick et al. (1996) found that following inhalation of 4 μm AMAD ^{57}Co -labelled FAP by rats, the amount of activity in tracheal washings at all times (7, 28 and 112 d) was greater than expected from fast mucociliary clearance.

Human studies indicating slow tracheobronchiolar clearance

The assumption that TB is completely cleared in the rapid phase was challenged by Davies (1980). Following a comparison of experimental results obtained under controlled breathing conditions, with theoretical predictions, he concluded that "the assumption that particles remaining in the lungs after about 30 h are in the alveolated regions" led to "impossibly high values" of alveolar deposition. Bailey et al. (1982) reported that, following inhalation of 1.9- μm - d_{ae} and 6.1- μm - d_{ae} particles under controlled conditions, the fractions cleared within 6 d corresponded only to those expected to have deposited in the first 14 airway generations. For both sizes the fractions cleared in the intermediate phase ($t_{1/2}$ about 20 d) were consistent with the predicted deposits in terminal bronchioles. Becquemin et al. (1987) and Roy et al. (1988) described lung retention in healthy nonsmokers up to 35 d after inhalation, by a three-component exponential function. About 30% cleared with $t_{1/2}$ 1 h, and 40% with $t_{1/2}$ 3 d, while clearance of the remainder was too slow to measure.

They attributed the first two phases to proximal and distal airways, respectively. The intermediate (3-d) component was found in patients with sarcoidosis, but in those with silicosis it could not be distinguished from the third phase.

The main evidence for slow TB clearance in humans comes from a series of experiments conducted by Stahlhofen et al. (1986a,b, 1987a,b, 1990, 1994), Stahlhofen (1989), Scheuch (1991) and Scheuch et al. (1993a). RECENTLY, they have been reviewed by Stahlhofen et al. (1995). In most of these experiments (Table 3.6) the subject inhaled clean air at a constant rate ($250 \text{ cm}^3 \text{ s}^{-1}$), and tidal volume (1000 cm^3). Radiolabelled particles were administered as a small bolus ($\sim 50 \text{ cm}^3$) at a predetermined point in the breathing cycle. The breath was completed, a breath-hold imposed to promote deposition by sedimentation at the chosen lung depth, and retention in the thorax measured by external counting for about a week. In one experiment (Table 3.6), the aerosol was inhaled rapidly ($700 \text{ cm}^3 \text{ s}^{-1}$) and was followed by a minimal breath-hold to promote deposition by inertial impaction.

When the bolus was injected early on in the breath, to deposit it in the alveolar region, the fraction of material deposited in the thorax which cleared slowly, A, was close to unity, as expected. However, when the bolus was injected at the end of the breath, A was surprisingly large: it was still about 0.5 when the bolus front depth, V_F , was only 45 cm^3 , about half the volume of the anatomical dead space.

The experiments were initiated to investigate the assumption that the fast and slow phases of lung clearance corresponded to TB and alveolar deposition, respectively (Stahlhofen, 1989), and the variation of mucociliary clearance velocity with lung depth (Stahlhofen et al., 1986a). However, in view of the unexpected initial results, they have continued with the objective of investigating the phenomenon, in particular to try to establish whether it results from slow TB clearance, rather than greater than expected penetration of the bolus to the alveolar region (Stahlhofen et al., 1986a; Sweeney et al., 1988).

Most of the studies were conducted with iron oxide particles (Fe_2O_3), but similar results were obtained with Teflon or FAP, confirming that the phenomenon is not related to the composition of the particles. Similarly, the results are consistent between subjects.

In most experiments the subject was seated during inhalation, but in some the subjects inhaled the particles lying down, and took a longer breath-hold after inhaling the bolus, maneuvers intended to increase the fraction of the

bolus deposited in large airways. However, A was even greater, about 0.75 for V_F between 30 cm³ and 130 cm³.

Experiments were carried out to investigate the possibility that a significant fraction of the bolus might reach the alveoli either during inhalation, or during the breath-hold imposed at the end of inhalation.

Measurements were made of the total particle deposition of inhaled aerosol boluses, as functions of V_F and breath-holding time t_b (Scheuch and Stahlhofen, 1988, 1994). For boluses of 3-μm- d_{ae} particles, which have a gravitational settling velocity of about 0.3 mm s⁻¹, administered at end-inhalation ($V_F < 55$ cm³), there was <20% deposition during the first 5 s of breath-holding. Since in this time the particles would have fallen more than 1 mm under the action of gravity, all particles that reached alveolar structures, which have dimensions <1 mm, would have deposited. Since there was some deposition of particles in the TB tree, <20% of the bolus could have reached alveoli during inhalation. In an alternative analysis, the bolus deposition as a function of t_b was used to determine the "effective" diameter of the airways (EAD) reached by the bolus (Scheuch and Stahlhofen, 1988). Results indicated that for $V_F < 80$ cm³, EAD was >2 mm, i.e., most of the bolus had not reached the alveolar region.

RECENTLY, further indirect evidence that shallow boluses do not penetrate to alveoli has come from experiments in which aerosol bolus retention was measured in a hollow dog airway cast (Scheuch et al., 1993b). The cast, complete from trachea to 1 mm airways, was connected to the bolus inhalation apparatus and ventilated mechanically. Aerosol was injected as 20 cm³ boluses at various lung depths. Particles that penetrated through the cast left the open-ended airways and were lost in the chamber. There was more than 90% recovery of particles administered to depths of <90 cm³. Similar results have since been obtained with a human airway cast (Scheuch, personal communication).

Scheuch and Stahlhofen (1991, 1994) investigated the possibility that particles might be transported to alveolar structures during the breath-hold at the end of inhalation, as a result of movement produced by the heart beat. It was shown that such an effect did occur, but only for particles with $d_{ae} < 2$ mm. It was therefore concluded that this phenomenon is not significant for the 3-μm- d_{ae} particles with which most of the clearance studies were conducted.

It is notable that there is no obvious effect of front depth on A until $V_F > 145$ cm³, beyond which A increases; nor does A vary noticeably with t_b (Table 3.6). If the observed

retention were mainly due to alveolar deposition, then a continuous decrease in the value of A with decreasing V_F would be expected.

Gamma-camera images following bolus inhalation in both supine and sitting positions, with $V_F = 55$ cm³ and 70 cm³, $d_{ae} = 3$ mm, and inhalation flow rate $Q = 250$ cm³ s⁻¹, clearly showed particle deposition in the central airways but did not demonstrate peripheral deposition (Scheuch, 1991). However, according to Bennett (1991), gamma-camera analysis has indicated that shallow boluses of radiolabelled particles can penetrate to peripheral airways in humans. He showed an image obtained following inhalation of a 70-cm³ bolus of 2-μm MMAD particles at 90% total lung capacity, which indicated that deposition was asymmetric and not confined to the conducting airways.

A high value of A (0.5) was also observed when a bolus of 3.8-μm- d_{ae} particles was inhaled at the end of a rapid breath ($Q = 700$ cm³ s⁻¹) (Stahlhofen et al., 1994; Table 3.6). In this experiment a greater proportion of deposition is expected to occur by impaction than in any of the other experiments, but the value of A was similar. Since deposition by impaction occurs predominantly in the first five bronchial generations, this result is also consistent with the view that the high values of A are due to a slow phase of TB clearance, and indeed indicates that the phenomenon occurs in the bronchi, not only in the bronchioles, as had been proposed.

It has therefore not been possible to account for the high retention values (A) on the basis of penetration of a large fraction of the bolus beyond the ciliated airways, either during inhalation or during breath-hold. Taking all the observations above, together with those from animal studies, such an explanation appears improbable (Stahlhofen et al., 1995).

Most experiments were conducted with 3-μm- d_{ae} particles. The few trials with smaller particles (d_{ae} 1.2 – 1.8 μm) suggest that A may be somewhat greater (about 0.6). Measurements have also been made of lung retention following inhalation by human subjects of ¹¹¹In-labelled indium oxide, with a mass modal diameter of 27 nm (Roth et al., 1994). For such particles, which are deposited mainly by diffusion, it was calculated that TB deposition should be about 20% of thoracic deposition. However, only about 7% of the thoracic deposit was cleared in the rapid phase, suggesting that about two-thirds of the TB deposit is cleared slowly. In this experiment the aerosol was inhaled throughout each breath, not as boluses.

Initial experiments with 6- μm - d_{ae} particles, however, indicate a significantly lower fraction retained in the slow phase, about 0.25 (Stahlhofen et al., 1990). Furthermore, it has long been known that large ($>10\text{-}\mu\text{m}$ - d_{ae}) particles inhaled at moderate flow rates are practically all cleared in the rapid phase (Lippmann and Albert 1969; Stahlhofen et al., 1983). It was hypothesized (Stahlhofen et al., 1987b) that the apparent decrease in the fraction of the TB deposit cleared slowly, f_s , with increasing size might result from deposition of the larger particles by impaction at sites with efficient clearance. The experiment in which 3.8- μm - d_{ae} particles were inhaled rapidly (Stahlhofen et al., 1994) was designed to test this, but did not show the lower value of A expected. It has therefore been proposed that f_s depends on the physical size of the particles. This hypothesis was tested in a recent experiment in which subjects inhaled boluses of PSL particles with a nominal geometric diameter, d_p , of 4 μm (Scheuch et al., 1993b). This diameter was chosen to be similar to that of the 6- μm - d_{ae} Fe_2O_3 ($\rho = 3.2 \text{ g cm}^{-3}$; $d_p = 3.4 \mu\text{m}$) and FAP ($\rho = 2.2 \text{ g cm}^{-3}$; $d_p = 4.0 \text{ mm}$), for which A was previously found to be about 0.25. However, since for the PSL, $\rho = 1.05 \text{ g cm}^{-3}$; d_{ae} would be about 4 μm , similar to that of the FAP and Fe_2O_3 particles for which A was about 0.5. As detailed in Table 3.6, values of A were 0.23 to 0.33, for $V_F < 100 \text{ cm}^3$ (the measured value of d_{ae} was 3.7 μm , and hence d_p was 3.6 μm), supporting the view that f_s decreases with increasing geometric diameter. RECENTLY, Scheuch (1995) found that following shallow bolus inhalation of particles with diameters $>6 \mu\text{m}$, less than 5% was retained more than 24 h.

Stahlhofen (1989) reported the half-time of the slow phase observed in the bolus clearance studies to be $20 \pm 10 \text{ d}$. However, since measurements were only made for up to about a week after inhalation, this might only be representative of part of the retained material.

Some gamma-camera studies have indicated that TB clearance is not complete within 24 h. Smaldone et al. (1988) reported "significant but quantitatively small" retention in central airways at 24 h, notably in those normal subjects with relatively high central deposition, and in patients with "flow-limitation." Foster et al. (1982), however, reported that "asthmatic subjects, except only one, had cleared all particles by 24 h, suggesting not only more centralized deposition, i.e., anatomically close to the lung roots, in these subjects, but deposition that could be entirely cleared by mucociliary transport." In the discussion following the paper (page 244) he further noted that "for some subjects, within 2 h after the initiation of the clearance measurements, the central region of the lung can be virtually free of labelled mucus (<5%)...."

Bennett et al. (1993) found that administration of a beta-adrenergic agent (albuterol) significantly enhanced the rapid phase of particle clearance but not of clearance between 24 h and 48 h. An enhancement of clearance at 24 h would have been strong support for the view that there is substantial particle retention in the airways at 24 h. The authors note, however, that the absence of such enhancement does not rule out the possibility of such retention. One possibility is that the particles are mainly retained in small airways, while albuterol might stimulate clearance mainly in large airways. Another possibility is that the retained particles are in the sol phase or in macrophages and so their retention would not necessarily be affected by an agent that altered mucociliary clearance.

RECENTLY, a different technique for depositing particles selectively in the TB region has been applied (Anderson et al., 1995; Svartengren et al., 1996). This involves inhalation of large (6–7 μm d_{ae}) particles extremely slowly. Theoretically, most should be deposited in the bronchioles. The authors found that the amount remaining after 24 h was much greater than the expected AI deposit, but also that there was a significant amount of clearance between 24 and 72 h, much more than is seen when similar particle are inhaled at a normal flowrate, which gives rise to greater BB and AI deposition. The results suggest that a substantial proportion of particles deposited in the bronchioles (bb region) are cleared on a time scale of days to weeks. Further studies are in progress.

Particle retention in the airway wall

Particle retention in the airway wall of the TB region has been observed qualitatively for a variety of materials and several species (Table 3.7). However, there may well be interspecies differences in the extent to which it occurs. Masse et al. (1973) reported that, following inhalation of tantalum powder, retention of particles in bronchial cells was considerably less in cats than in rats or monkeys.

Little et al. (1965) reported high concentrations of ^{210}Po at segmental bronchi bifurcations of smokers. Radford and Martell (1977) inferred long-term retention of ^{210}Po -enriched particles at bifurcations from observed ^{210}Po : ^{210}Po ratios. However, these results were not confirmed by other investigators (e.g., Robertson and Rogers, 1980; Henshaw et al., 1988). Cohen et al. (1980) generally found much lower surface concentrations than Little et al., but a similar level in one sample from a smoker.

The studies by Churg and his colleagues are of special relevance here, since they demonstrate that particle retention in the airway wall occurs in humans, under normal environmental conditions of exposure, and throughout the airways from the main bronchus (Churg et al., 1990) to the respiratory bronchioles (Churg and Wright, 1988). Churg et al. (1990) measured particle number concentration in the mucosa of bronchi (not at bifurcations), and in the parenchyma tissue supplied by those bronchi, in 11 nonsmokers without lung disease or occupational dust exposure. The concentration of mineral dust particles in the mucosa of most bronchi measured (2×10^8 particles g^{-1} dry tissue) was very similar to that in parenchyma tissue. Differences in the size distribution and composition of particles at different locations suggest that the retained particles result from sequestration of locally deposited particles. Concentrations of larger particles ($>1 \mu\text{m}$) were highest in segmental bronchi, where highest deposition would be expected. Silica accounted for 47% of particles in large airways, but only 24% of particles in the parenchyma. Conversely, kaolin and mica accounted for 44% of particles in parenchyma but only 15% of particles in the large airways. In a recently reported similar study on cigarette smokers without emphysema, Churg et al. (1992) found considerably lower concentrations in the airways of many, but not all, smokers than had been found in non-smokers.

However, when the IRTM was finalized the phenomenon had only been well quantified by Patrick (1989) and his colleagues (Table 3.8), who followed retention of activity after deposition of radiolabelled particles onto the distal trachea of rats. Between 0.4% and 1.1% of the deposited activity was retained at 1 d to 7 d after instillation. Takahashi and Patrick (1987a; Table 3.8) found that between 1 week and 6 months after instillation of ^{133}Ba -labelled BaSO_4 the ^{133}Ba cleared from the tracheal wall with a half-time of 88 d. There is evidence that particles are cleared from the TB mucosa to regional lymph nodes.

RECENTLY, Takahashi et al. (1993) conducted experiments similar to those of Patrick et al., instilling ^{133}Ba -labelled BaSO_4 onto the distal trachea, but of rabbits, dogs and monkeys. The amounts of ^{133}Ba retained 1 week after injection in the caudal region of the trachea were 0.145, 0.044 and 0.043% of the injected amount respectively. These are far lower than found in rats (on which the IRTM value is based), suggesting inter-species differences. The values for dogs and monkeys are close to the value chosen in the IRTM for retention of particles in the wall of the nasal epithelium (Table 3.1), which was based on results for several different materials in several species.

Retention of particles in transit

Consideration of a slow phase of TB clearance and of airway wall retention of particles deposited onto the surfaces of the conducting airways, raises the question of the extent to which particles cleared into a region may be subject to similar retention. For example, are particles that were initially deposited in AI, and are being transported toward the GI tract, subject to slow clearance or airway wall retention in BB and/or bb, and if so how much? Information on this is sparse and inconclusive, and it is difficult to predict what might occur from mechanistic considerations.

Morrow (1972), for example, noted that: "It is logical to assume that materials in transit through the tracheobronchial tree (including mucus) experience absorption, at least to some extent. If this were to occur, the materials would thereafter appear in the peribronchial and perivascular lymphatic drainage and the tracheobronchial nodes." There is also some experimental evidence to support this. Patrick and Stirling (1992, 1994) administered ^{195}Au -labelled colloidal gold particles to rats by microinjection into subpleural alveoli, to confine the initial deposition to alveolar tissue. They followed retention and distribution of the material for 15 months and found that most of the material that was not cleared from the lung remained close to the deposition site. However, they did find some ^{195}Au beneath the bronchial epithelium and also observed it on the luminal surface of the conducting airways more frequently than expected and, remarkably, at times up to 15 months. Filipi et al. (1985) studied by autoradiography the distribution of ^{239}Pu in dogs following inhalation of $^{239}\text{PuO}_2$. They drew attention to "the high concentrations of plutonium particles observed on bronchial surfaces 400 days after a single exposure...."

Conversely, it seems reasonable to assume that material once in a moving mucus stream, keeps going. The very speed of the rapid clearance phase suggests that it is probably not subject to slow clearance. For example, in the experiments by Patrick and Stirling (1977) in which particles were deposited on the distal trachea of rats, about 80% cleared with $t_{1/2}$ of 5 min and about 20% cleared with $t_{1/2}$ of 16 h. There was a very distinct change in the slope of the retention curve between the two phases. The fast phase, $t_{1/2}$ of 5 min, is consistent with mucociliary transport rates of a few mm min^{-1} and a tracheal length of about 3 cm (Gore and Patrick, 1978). If material leaving the deposition site were subject to slow clearance "downstream," then a more gradual transition between the two phases might be expected. Similarly the use of boluses of labelled mucus to

measure tracheal mucociliary transport rates, and the consistency of the results (Table 3.3), also suggest that material that is being transported by mucociliary transport generally keeps moving. Furthermore, Churg et al. (1990) inferred from measurements of the size distribution and composition of particles in human bronchial mucosa, and differences from those found in the parenchyma, that they resulted from local deposition.

Conclusions for modeling

Reference values and uncertainties

Mucociliary clearance (fast phase) (BB₁ and bb₁). Representative values of the clearance rates from the bronchioles (bb) to bronchi (BB), and from the bronchi to the extrathoracic region (ET) are required. Any single value chosen can only be typical, because the regions are not homogeneous; the clearance time is likely to be different for particles cleared into bb from AI, from that for particles deposited directly within bb; and different for particles cleared into BB from bb from that for particles deposited directly within BB. While there are numerous measurements of the overall pattern of lung clearance, direct measurements of clearance rates are confined to the trachea and main bronchi. Thus a model of mucociliary clearance must be used to obtain estimated clearance rates for the bronchi and bronchioles separately. Clearance from the two regions cannot be distinguished experimentally at present.

Similar mucus velocities were calculated for the first nine airway generations (BB) by the three models for which results are given in Table 3.5 (Lee et al., 1979; Yu et al., 1986; Cuddihy and Yeh, 1988). For the example shown in Figure E.9 (Cuddihy and Yeh, 1988), cumulative deposition in the first nine generations is 22% of total TB deposition, and this fraction is cleared in 170 min. From the airway clearance times calculated by Yu et al. (1986, Table 3.5), the first nine generations would be cleared in 236 min. On this basis a rounded value of 10 d⁻¹ was chosen for the IRTM, which corresponds to a *t*_{1/2} of about 100 min. Given the many observations that most mucociliary clearance is complete in a few hours, and the agreement between the clearance models, it is considered that this value is relatively well known and proposed that $\Phi_u = 1.5$.

For the bronchioles, there are greater differences between the model estimates of mucus velocities, especially in the terminal bronchioles, for which Yu et al. (1986) predict a rate 40 times faster than Cuddihy and Yeh (1988). Similarly, according to the model of Yu et al., the bronchioles are completely cleared in 643 min, and,

according to Cuddihy and Yeh's model, in 2400 min. On this basis, a rounded value of 2 d⁻¹ was chosen, which corresponds to a *t*_{1/2} of 500 min. Given the greater uncertainty about clearance from bb compared to BB, it is proposed that $\Phi_u = 2$.

Slow clearance. The main body of evidence from human studies suggesting a significant slow phase of bronchial clearance comes from the bolus experiments of Stahlhofen et al. (Table 3.6). These indicate that, for small particles ($d_{ae} < 4 \mu\text{m}$), the fraction of the deposited material that is subject to slow clearance, f_s , is about 0.5. For larger particles f_s decreases, being about 0.5 at $d_{ae} = 6 \mu\text{m}$, and <10% for $d_{ae} > 10 \mu\text{m}$. The information currently available suggests that f_s depends on the particle diameter (for the spherical particles tested). For nonspherical particles, it is assumed in the IRTM that the relevant size parameter is the equivalent volume diameter, d_e , i.e., the diameter of a sphere having the same volume as the particle.

From Table 3.6, the largest particles for which A was about 0.5 were 3.8- μm - d_{ae} FAP. Since for FAP, $\rho = 2.2 \text{ g cm}^{-3}$, d_e is 2.5 μm . Therefore, it was assumed for modeling purposes that $f_s = 0.5$ for $d_e \leq 2.5 \mu\text{m}$, and then decreases with increasing size. As a simple decreasing function, the IRTM assumed:

$$f_s = 0.5e^{-k(d_e - 2.5)} \quad \text{for } d_e > 2.5 \mu\text{m}$$

A was found to be about 0.25 for:

- 5.9- μm - d_{ae} Fe₂O₃, for which $\rho = 3.2 \text{ cm}^{-3}$, and therefore $d_e = 3.3 \mu\text{m}$.
- 6.0- μm - d_{ae} FAP, for which $\rho = 2.2 \text{ g cm}^{-3}$, and therefore $d_e = 4.0 \mu\text{m}$.
- 3.7 μm - d_{ae} PSL, for which $\rho = 1.05 \text{ g cm}^{-3}$, and therefore $d_e = 3.6 \mu\text{m}$.

Hence assume $f_s = 0.25$ at $d_e = 3.6 \mu\text{m}$, which gives:

$$f_s = 0.5e^{-0.63(d_e - 2.5)} \quad \text{for } d_e > 2.5 \mu\text{m}$$

For practical application it is more useful to relate f_s to the aerodynamic diameter d_{ae} , i.e., the diameter of the unit density sphere, 1 g cm⁻³, that has the same settling velocity as the particle. To do so, account has to be taken of both particle density, ρ , and shape. The drag on an irregular particle, according to Stokes' law, is given by Hinds (1982):

$$F_D = 3 \pi \mu u d_e \chi$$

where

μ = dynamic viscosity of air

u = particle velocity

χ = the dynamic shape factor, typical values of which lie in the range 1 – 2 (Hinds, 1982).

By equating the terminal settling velocity, u_s , expressed in terms of either the equivalent volume diameter, d_e , or the aerodynamic diameter, d_{ae} , it was shown in Annex D (James et al., 1994) that

$$d_e = d_{ae} \sqrt{\chi/\rho}$$

Hence, in terms of the particle aerodynamic diameter, d_{ae} , the slow-cleared fraction f_s , is given by

$$f_s = 0.5 \text{ for } d_{ae} \leq 2.5 \sqrt{\rho/\chi} \text{ } \mu\text{m}$$

$$f_s = 0.5e^{-0.63(d_{ae}\sqrt{\chi/\rho} - 2.5)} \text{ for } d_{ae} \leq 2.5 \sqrt{\rho/\chi} \text{ } \mu\text{m}$$

In view of the uncertainties associated with the extent of slow clearance, it is assumed in the IRTM that these values of f_s apply to both BB and bb, and that $\Phi_u = 3$. (See ICRP Publication 66 paragraph 181, Chapter 5 (ICRP, 1994a) for aerosol default values used for relating f_s to d_{ae} .)

Stahlhofen (1989) reported the half-time of the slow phase observed in the bolus clearance studies to be 20 ± 10 d. On this basis a rounded value of 0.03 d^{-1} was chosen, which corresponds to a $t_{1/2}$ of 23 d. However, since measurements were only made for a few days after inhalation, this might only be representative of part of the retained material. Generally particle transport rates tend to decrease with time, so an overall rate is more likely to be lower than this value than higher. For the present, however, it is proposed that $\Phi_u = 3$.

Retention in the airway wall. As discussed above, there is evidence for particle retention in the airway wall in the BB and bb regions from studies on several species, including humans. However, when the IRTM was finalized, the phenomenon had only been well quantified for particles deposited in the rat trachea: about 0.7% of the material deposited is retained near the basement membrane of the epithelium, from which it clears to lymph nodes with $t_{1/2}$ of

88 d. These results formed the basis of the IRTM reference values. In the IRTM retention is represented by the sequestration compartments BB_{seq} and bb_{seq} , which receive 0.007 of the deposits in regions BB and bb, respectively. Since these fractions are based only on animal data, and these data were only for the trachea, it was proposed that $\Phi_u = 3$. It is assumed that the sequestered particles are cleared to thoracic lymph nodes (LN_{TH}). The rounded value chosen for the clearance rate from BB_{seq} and bb_{seq} to LN_{TH} is 0.01 d^{-1} ($t_{1/2} = 70$ d). Since this is based on animal data only, it is proposed that $\Phi_u = 3$. However, as noted above, more recent information suggests that the fraction retained is probably much lower than in rats, and similar to that selected in the IRTM for the nasal epithelium. On that basis I have here selected values for both the retained fraction and clearance rate to LN_{TH} to be those selected in the IRTM for the nasal epithelium, i.e., 0.0005 and 0.001 d^{-1} (Table 3.1), to calculate central values, and retained the uncertainty factors of $\Phi_u = 3$ in each case.

Modifying factors

There have been many studies of the effects of factors on LMC, much of the interest resulting from the possible involvement of impaired clearance in the development of lung diseases. Most of the studies fall into one of three categories: effects of lung disease, effects of pharmaceuticals, and effects of air pollutants. There is no information on factors affecting either the magnitude or duration of slow TB clearance, or of particle retention in the airway wall in humans. Several recent reviews have focused on factors affecting LMC (Lippmann and Schlesinger, 1984; Pavia, 1984; Schlesinger, 1985, 1989; Wolff, 1986; Matthys et al., 1987; Camner, 1988; Camner and Mossberg, 1988). Only a brief account is therefore given in Annex E, concentrating on estimating values of Φ_m , the number by which the clearance rate should be multiplied when a specific population group is considered. Transient and permanent factors modifying LMC from the literature are given in Tables E.16 and E.17, respectively. The objective was to obtain values of Φ_m representing the ratio of the clearance rate in the study group to that in the controls. Only cigarette smoking is considered here.

A particular problem that arises in evaluating effects on LMC is that some factors (including smoking) alter the pattern of deposition as well, which in turn affects clearance. Factors resulting in restricted airways may give rise to more central deposition and hence faster overall LMC, even if the clearance itself is actually impaired. This is more likely to arise when small particles ($<5 \mu\text{m}$) are used, which would normally deposit peripherally. It has

given rise to apparently conflicting results, especially where different particle sizes have been used, but this problem has been recognized for some time. Thus the faster LMC observed by Sanchis et al. (1973, 1974) and by Thomson et al. (1973; Thomson and Pavia, 1973, 1974) in smokers and subjects with cystic fibrosis and chronic bronchitis was attributed to more central deposition. Coughing may also compensate for impaired mucociliary clearance in subjects with lung disease. The effects of inhaled irritants such as cigarette smoke and sulfuric acid can be complex. Acute exposures may cause a transient increase or decrease according to the concentration, and may affect clearance in some airways more than others, depending on the size distribution. Chronic exposure can result in permanent impairment of clearance, and also changes to airway dimensions and lung function, which in turn alter the pattern of deposition and subsequent clearance of the test aerosol.

Proposed modifying factors for modeling. Values of Φ_m to be applied to the compartment model for the respiratory tract (Table 3.1) are only proposed in Annex E for factors judged to be important, and only those for which: (1) the effect is large ($\Phi_m > 1.2$ or $\Phi_m < 0.8$); (2) there are reasonably consistent human data; and (3) a reasonable assessment can be made on whether the BB and/or bb are affected. Generally it would be expected that a change in TMTR and bronchial clearance at 1 h or 2 h after inhalation, or in T_{50} , or in inner zone would reflect alteration to clearance from the BB. A change in LMC at three or more hours after inhalation, or in T_{90} , or in peripheral zone would reflect alteration to clearance from the bb. Because of the similarity in clearance mechanism, it is assumed, in the absence of information to the contrary, that a factor that applies to BB applies similarly to bb. It is also assumed that the agents considered do not alter slow clearance or airway wall retention.

Cigarette smoking: Comparing chronic smokers and nonsmokers, Goodman et al. (1978), Chopra et al. (1979), and Toomes et al. (1981) all found a marked reduction in TMTR, but Yeates et al. (1975) did not (Table 3.9). Some studies found little or no difference in LMC (Table 3.10) between smokers and non-smokers (Thomson and Pavia, 1973; Isawa et al. 1984), but others found impaired clearance in smokers (Albert et al., 1969; Lourenço et al., 1971). The faster clearance observed by Sanchis et al. (1974) in smokers was probably due to more central deposition. Camner and Philipson (1972) found LMC to be slower in five out of ten smokers compared with non-smoking monozygotic twin brothers, and similar in the other pairs. Bohning et al. (1975) found similar overall LMC in four such pairs, but noted indications that upper

bronchial clearance was impaired in the smokers. Camner et al. (1973) also found an improvement in clearance by smokers after stopping smoking for 3 months. Lourenço et al. (1971), using a gamma-camera, observed that in non-smokers clearance started immediately and rapidly in the central airways and progressed smoothly, while in smokers it was delayed, especially in the larger airways. Agnew et al. (1986b) found that, while total lung clearance was not significantly different, clearance from the inner zone was slower in smokers. Vastag et al. (1985, 1986) found that LMC (total and inner zone) decreased with cigarette consumption (measured in pack-years) in ex-smokers and current smokers, with or without chronic bronchitis, but some individuals were affected much more than others. It is, therefore, proposed that $\Phi_m = 0.5$ for BB but not bb (Table 3.11).

3.5 The Alveolar-interstitial Region

Retention

Early phase (up to 7 d)

In the ICRP Publication 30 Lung Model (ICRP, 1979) it was assumed that 40% of the initial alveolar deposit (IAD) of a relatively insoluble material is cleared with $t_{1/2}$ of 1 d, and the rest with $t_{1/2}$ of 500 d. (The term pulmonary used in *ICRP Publication 30* is here treated as synonymous with alveolar.) However, more recent human inhalation studies do not support the assumption of a significant rapid phase of particle transport from the AI region. Following deposition of small (d_{ae} 1 – 2 μm) particles, which would be expected to deposit mainly in the AI region, lung retention at 24 h often considerably exceeds 60% of the ILD. Lippmann (1970) noted that if there is a rapid phase of pulmonary clearance, it accounts for much less than 20% IAD. Foord et al. (1977) similarly noted that their results were inconsistent with rapid clearance of 40% IAD. Stahlhofen et al. (1980) administered particles in a manner designed to maximize AI deposition and minimize TB deposition: 1- μm - d_{ae} particles inhaled as a bolus at the beginning of a breath. They found that 1% ILD cleared rapidly (an effect attributed to dissolution), while the rest followed a single exponential with $t_{1/2}$ of 85 d. Bailey et al. (1982) found 8% and 40% ILD, respectively, of 2- μm - d_{ae} and 6- μm - d_{ae} particles cleared within 6 d, but these fractions were less than the corresponding predicted TB deposits, and therefore no rapid phase of pulmonary clearance was observed.

The possibility that a significant fraction of material deposited in TB is not cleared in the rapid phase cannot be excluded. Generally, however, it would be expected that

most of the material cleared in the slow phase would be associated with AI, because of greater initial deposition in AI than in TB, and because much of the material deposited in TB is cleared in the rapid phase. For simplicity, therefore, in the following discussion, material not cleared in the rapid phase is described as being in AI. Similarly, at long times after intake a significant fraction of the material described as being in the lungs may be in lung-associated lymphatic tissue.

Intermediate phase (up to 300 d)

Experimental studies of particle retention in the human lung lasting at least 50 d are summarized in Tables 3.12 and 3.13. Studies appear in Table 3.13 if smokers and nonsmokers were identified, and if there were at least two subjects in each experimental group. Several recent studies extended to 300 d, but when the IRTM was finalized very few measurements have been made beyond 500 d (Bohning et al., 1982; Bailey et al., 1985a; Pearman et al., 1989).

RECENTLY, retention has been followed for about 3 y after inhalation of ^{195}Au -labelled Teflon particles by ten male subjects (Philipson et al., 1992; 1996). Leaching of the label from this material is considered to be even less than that from the materials previously used.

After the initial rapid clearance phase, lung retention of relatively insoluble particles measured over a period of several weeks can be adequately represented by a single exponential function (Table 3.12). The associated half-time can conveniently be used to compare clearance in different groups (Gongora et al., 1983) or to compare clearance of different materials. Generally longer retention half-times have been found for polystyrene and Teflon particles than for iron or manganese oxides, suggesting that dissolution of the oxides is faster (Table 3.12; Stahlhofen et al., 1981).

It is, however, generally found that the retention half-time increases as the duration of measurements increases. When retention is followed for several months, a two-component exponential function is usually required, the components having half-lives of about 30 d (intermediate phase) and several hundred days (slow phase). Similar functions have been fitted to lung retention following accidental intakes (Watts, 1975). The proportion in the intermediate phase varies considerably: typically in experimental studies it is about 20 – 30% (Bohning et al., 1982; Bailey et al., 1985a; Philipson et al., 1985), in some subjects, especially smokers, it is negligible, while for some occupational exposures it is the major component (Ramsden, 1976). Even when subjects have inhaled similar particles under

controlled conditions, there is great variation in both the amplitudes and coefficients of the fitted functions. Some scatter is due to intersubject variation, but it is a feature of multi-exponential curve fitting that, unless marked changes in slope occur, it may well be possible to vary the value of each parameter over a wide range, and by adjusting the values of the other parameters appropriately make little change to the shape of the curve over a fixed interval. Thus while such a function provides a concise and accurate description of retention, the individual parameter values may have little significance, and cannot be used directly to compare the results from different studies. In Table 3.13, therefore, retention as a fraction of the IAD has been evaluated at three specific times from the functions reported.

Retention was very similar in most studies in which nonsmokers inhaled FAP, Teflon, or polystyrene particles, which is consistent with the view that particle transport is independent of the material. Retention of Co_3O_4 was also very similar up to 100 d, but at 300 d was substantially lower, presumably reflecting the progressively increasing dissolution rate of Co_3O_4 (Bailey et al., 1989). Clearance of Fe_3O_4 was faster throughout, probably because of more rapid dissolution in the lungs.

In the recent study with ^{195}Au -labelled Teflon (Philipson et al., 1992; 1996) retention was higher at all times than in any other study. While this is probably partly a reflection of lower leaching, the effect is apparent even at 100 d. There appears to be relatively little associated with the intermediate (30-d) phase, which it not attributed to leaching, but to particle transport. This may be due to the administration conditions or the particular subjects. However, only single exponential functions were fit to each subject's data over each of two periods: 7 – 250 d, and 250 – 900 d. These were used to derive the retention values in Table 3.13, whereas most others were based on two-component exponential functions. This may well have affected the estimates of retention over the first few months. At longer times, the difference would be more due to lower leaching, and it is noteworthy that at 900 d, the end of the experiment, the fractions (of retention at 7 d) were $41 \pm 11\%$, $48 \pm 16\%$ and $71 \pm 5\%$ in the non-smokers, ex-smokers and smokers respectively. These are little, if any lower than found in the earlier studies at 300 d.

Late Phase (>300 d)

Particle retention in the human respiratory tract has been reasonably well characterized in controlled experimental studies up to 300 d after intake, at least in healthy adult males. However, at this time about 50% of the estimated

IAD remains, and when the IRTM was finalized there were insufficient data at later times to determine the subsequent pattern of retention. Measurements of thoracic retention of materials in the human respiratory tract following accidental intakes are therefore reviewed in Annex E. These fall into two main categories: *in vivo* measurements, usually of radioactive materials; and postmortem measurements of the amounts of material in the lungs, which in some cases have been compared with the estimated intakes.

There are several well-known general problems associated with interpreting measurements of thoracic retention of activity in terms of lung retention and clearance. There may be considerable uncertainties about the time course of intake and the physicochemical nature of the material. The fact that an intake has occurred may not be recognized, and days or even years elapse before measurements begin. There may have been other identified or unidentified intakes before measurements started, and also afterwards if the subject is not removed from work with the material. There have, however, also been cases when exposure has occurred as a result of a known incident involving a single, well-defined material. Some extensive sets of measurements have been made during retirement after chronic exposure (e.g., Kalliomäki et al., 1978, 1983; Crawford-Brown and Wilson, 1984), but to interpret them in terms of retention following an acute intake, assumptions must be made about the form of the retention curve.

In general the measurements were made for the purpose of assessing the workers' exposure and may therefore not be as intensive as desirable for modeling purposes. However, in some cases (notably exposures to ^{60}Co) measurements were continued when the intakes were clearly far below levels of concern, because the operational health physicists recognized the value of obtaining human data. There will be errors in the measurement process itself: statistical counting errors where the amount retained is close to the limit of detection; and, for low-energy photon emitters, notably plutonium isotopes, errors due to differences between the true distribution of the radionuclide *in vivo* and that assumed in the calibration, absorption in overlying tissue, etc. (Swinth et al., 1988).

Generally, *in vivo* measurements of activity in the thorax are unable to distinguish between material in the lungs themselves, or in other thoracic organs, although attempts to do so have been made recently (Northcutt et al., 1988; Pomroy and Noel, 1988). In particular, there has been considerable discussion about whether the very long-term thoracic retention of uranium observed by, for example, West et al. (1979) represents material retained in the lungs,

lymph nodes, or skeleton (Scott and West, 1975; Keane and Polednak, 1983; Crawford-Brown and Wilson, 1984). In the recent study with ^{195}Au -labelled Teflon (Philipson et al., 1992; 1996) measurements of activity in the chest were made with two systems: (i) a ring of NaI detectors around the thorax and (ii) a cluster of germanium detectors placed above the thorax. The latter system appeared to show more rapid clearance than the former, which is less sensitive to the position of activity within the thorax. The results suggested translocation of activity within the thorax, and were consistent with most of the activity remaining at the end of the experiment being in the hilar lymph nodes.

In a typical incident, only one or two people are exposed to the extent that requires or enables long-term follow-up measurements to be made. This adds to the difficulty in generalizing the results, since experimental studies have shown considerable intersubject variation in retention, even following closely controlled administrations of the same material (Table 3.13).

While many of the factors above lead to an overestimation of lung retention, all materials dissolve to some extent in the lung, and it is rarely possible to take account of this to estimate lung retention in the absence of dissolution. This is a particular problem following intakes of uranium compounds, because of the extreme range of *in vivo* dissolution rates they exhibit.

Despite these problems, it is necessary to take account of such information because of the lack of human experimental data and the recognized interspecies differences in the rates of particle transport (Figure E.6). Furthermore, a considerable number of such follow-up studies have been made, involving a range of materials.

In vivo measurements of thoracic retention in humans following accidental inhalation.

Information on thoracic retention in humans following accidental inhalation, based on *in vivo* measurements of radionuclides, is summarized in Annex E, Table E.21. Since retention up to 300 d after intake had been characterized in controlled experiments, only studies of accidental intakes in which retention was followed for at least 400 d were included. Nevertheless this includes measurements on nearly 100 persons, and about 40 of these have been followed for more than 10 y. Since a major objective in compiling these data was to obtain guidance on the likely fate of the 50% IAD that remains at 300 d after intake, 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. This also facilitates including information in cases where the first measurement was made some time after intake, and avoids the effects of differences in early clearance due to factors such as aerosol size, breathing patterns, and soluble components.

In Figure E.10 thoracic retention $R(t_f)$ at t_f , the time of the final measurement, as a fraction of $R(300)$, retention at 300 d, is plotted against t_f . Evidence for very long-term retention of a significant fraction (>10%) of the material remaining in the thorax at 300 d has been seen for each of the elements (cobalt, uranium, plutonium, and americium) for which measurements have extended to 10 y after acute intake of the oxide.

It was recognized that this procedure may have resulted in selecting cases showing unusually long retention. In a group exposed under similar conditions, those subjects with relatively slow lung clearance characteristics will remain measurable for longer, and their longer lung retention and hence potential dose will justify more intensive follow-up. Similarly, cases showing unusually long retention might well be considered more worthy of reporting than those showing expected or faster clearance. In addition, since many of the exposures took place in the 1950s and 1960s, it is likely that some of the subjects were cigarette smokers, and consequently may have slower lung clearance than non-smokers (see below).

Further evidence for a long-term component of thoracic retention of uranium comes from measurements on workers whose exposure has stopped through change of work or retirement (e.g., Pomroy and Noel, 1981; Crawford-Brown and Wilson, 1984). However, when the exposure period is long, assumptions must be made to infer the retention pattern following an acute intake, for example, to allow for clearance during the exposure period. Crawford-Brown and Wilson (1984) analyzed thoracic retention of uranium in 22 subjects, who had a mean exposure to uranium compounds of 21 y. Measurements were made from retirement for up to 12.5 y later. A least-squares fit to the pooled data gave a half-time of 2400 d. Assuming constant intake over 21 y, and two-component retention with half-times of 120 d and 2400 d, they estimated that about 5% was associated with the latter.

Comparison of intakes with lung content: (a) in vivo

Kalliomäki et al. (1978, 1983, 1985) used magneto-pneumography (MPG) to measure the lung contents of magnetic dusts in groups of welders with similar exposures. By comparing the lung contents of workers exposed to mild

steel welding fumes for 2 y, 9 y, or 18 y, and assuming a single exponential retention, they obtained a clearance rate constant of 0.1 y^{-1} (retention $t_{1/2}$ of 2500 d). A comparison of working with retired welders gave a clearance rate constant of 0.23 y^{-1} (retention $t_{1/2}$ of 1100 d). For stainless-steel welders they obtained a retention $t_{1/2}$ of 8.5 y (3100 d).

Schieferdecker et al. (1985) compared measured uranium thoracic contents, urinary, and fecal excretion over a 6-y period in 12 workers handling uranium oxides, with amounts predicted using intakes estimated from measured air concentrations and size distributions, and a model based on those used in *ICRP Publication 30*. Agreement was obtained using an AMAD of $8 \mu\text{m}$ and a pulmonary retention half-time of 110 d.

Thind (1987) compared measured uranium thoracic contents and urinary excretion in a group of 29 workers over a 5-y period, with amounts predicted using intakes estimated from measured air concentrations and size distributions, and the *ICRP Publication 30* Model. On this basis the best agreement was obtained using an AMAD of $6 \mu\text{m}$ and a pulmonary retention half-time of 250 d.

Comparison of intakes with lung content: (b) postmortem

Stöber et al. (1967) compared the lung contents postmortem with estimated dust exposures of two groups of coal miners, selected for having little or no pathological changes in their lungs: 49 who died more than a year after their last exposure, and 16 who died in a mining accident. Assuming a single exponential function for alveolar retention, they obtained a clearance half-time of 5 y ($4 \times 10^{-4} \text{ d}^{-1}$).

Fisenne and Welford (1986) found a significant increase with increasing age in the concentration of uranium in the lungs (including lymph nodes) of New York City residents. They observed that the increase was consistent with the measured uranium concentration in New York City air (0.4 ng m^{-3}), an inhalation rate of $20 \text{ m}^3 \text{ d}^{-1}$, and negligible clearance from lymph nodes. Assuming 10% AI deposition, these figures give a rate of deposition in the AI of about 1 ng d^{-1} . The lung content (assuming a lung mass of 1 kg) increased from about $0.25 \mu\text{g}$ uranium at about age 25 y (10^4 d) to about $0.9 \mu\text{g}$ uranium at about age 60 y ($2 \times 10^4 \text{ d}$), i.e., a rate of 0.05 ng d^{-1} . This suggests that about 5% of the AI deposit goes to sites of very long-term retention, since compartments with half-lives less than about 10^3 d would have reached equilibrium by age 25 y.

Sunta et al. (1987) estimated the daily intake of thorium by inhalation in Bombay to be $0.02 \mu\text{g}$, from a measured

concentration of 1 ng m⁻³, and an assumed daily intake of 20 m³ air. Assuming 20% deposition in long-term retention sites and an equilibrium lung content, they obtained a retention half-time of 650 d.

McInroy et al. (1989) found in four whole-body autopsies, where intake was by inhalation about 30 y before death, an average of 45% of the whole-body content in the respiratory tract. They concluded that this indicated respiratory tract retention may be greater than proposed by the *ICRP Publication 30* model.

Particle clearance rate to the GI tract

Experimental studies (up to 300 d)

In three of the studies listed in Table 3.13 (Bailey et al., 1985a; Philipson et al., 1985; Foster et al., 1989) the contribution to lung clearance made by dissolution was estimated from measurements of urinary excretion of the labels. From these it is possible to estimate the particle transport rate: $m(t)$ at time t after inhalation.

For FAP and Teflon, $m(t)$ was estimated as follows:

- a function was fitted to the decay-corrected measurements of lung retention in each subject, $R(t)$;
- the overall rate of clearance from the lung $\lambda(t)$ was calculated as a fraction of the contemporary lung content:

$$\lambda(t) = \frac{-dR(t)/dt}{R(t)}$$

- $m(t)$ was taken to be $\lambda(t) - s(t)$, where $s(t)$ is the estimated rate at which activity is absorbed from the particles into the blood, as a fraction of the contemporary lung content.

In the Co₃O₄ study, fecal excretion was also measured, and therefore $m(t)$ was evaluated from it directly (Bailey et al., 1989):

$$F(t) = g_f m(t) + b_f s(t)$$

where $F(t)$ is the amount excreted in feces per day as a fraction of $R(t)$; b_f and g_f are the fractions of cobalt excreted in feces, respectively, following absorption of cobalt from lung into blood; and after Co₃O₄ particles enter the GI tract.

Average values of $m(t)$ are shown in Figure E.11. For FAP and Teflon it was estimated that for the first 3 months particle transport was the dominant clearance mechanism. Subsequently dissolution made a significant contribution to clearance, and there is considerable uncertainty attached to the estimated value of $s(t)$, and hence that of $m(t)$. For Co₃O₄ it was estimated that absorption into blood was the dominant clearance mechanism after the first few days in all four subjects. Nevertheless the average clearance rates estimated for the three materials are reasonably consistent. The rate decreases with time from about 3 × 10⁻³ d⁻¹ at 25 d after inhalation to 1 × 10⁻³ d⁻¹ at 150 d, and decreases slowly thereafter.

Bailey et al. (1985a) and Philipson et al. (1985) fitted an exponential function (one-, or more often, two-component) to the lung retention measurements on each subject. Bailey (1989), however, considered that using an exponential model might well have influenced the estimated clearance rates, in particular, resulting in relatively constant rates beyond 150 d. He therefore fitted polynomial functions to the original data in the FAP study. Up to 200 d after inhalation the two functions gave very similar clearance rates. For the 1-μm particles at later times, the polynomials gave more variable results than the exponentials but with a similar average value (Figure E.11). For the 4-μm particles at later times, they gave consistently lower values, indicating that $m(t)$ continues to decrease.

Since the estimated value of $m(t)$ for the 4-μm FAP involves a smaller correction for dissolution than that for the 1-μm FAP, and since the results are consistent with those from the studies using Teflon and Co₃O₄ particles (which involved smaller numbers of subjects), it is felt that it provides the best currently available estimate of $m(t)$. Thus by 350 d after inhalation the estimated average value of $m(t)$ has fallen to 5 × 10⁻⁴ d⁻¹ ($t_{1/2}$ about 1400 d). However, it is not clear from these results whether $m(t)$ continues at a rate of about 10⁻³ d⁻¹, or continues to decrease. (See below.)

Late phase (>300 d)

West and Scott (1966, 1969) reported measurements of uranium excretion in five subjects who showed long-term thoracic retention, for times up to about 5 y after removal from uranium work. Urinary and fecal excretion were of similar magnitude in these cases. Since systemic uranium is largely excreted in urine, the fecal excretion rate $F(t)$ would be expected to approximate to the particle transport rate $m(t)$, as for cobalt oxide, discussed above. The values are summarized in Table E.23. Average values at ~1000 d and

1500 d are about $4 \times 10^{-4} \text{ d}^{-1}$, a little lower than estimated from the experimental data at 300 d after intake.

In the recent study with ^{195}Au -labelled Teflon (Philipson et al., 1992; 1996), the average clearance rate from 250 to 900 d (from the half-time of the fitted function) averaged $4.7 \times 10^{-4} \text{ d}^{-1}$, $3.3 \times 10^{-4} \text{ d}^{-1}$ and $3.5 \times 10^{-4} \text{ d}^{-1}$ in non-smokers, ex-smokers and smokers, respectively ($4.1 \times 10^{-4} \text{ d}^{-1}$ in non-smokers and ex-smokers combined).

Animal studies of long-term lung clearance

Several long-term animal studies of lung clearance of particles have indicated that the fractional rate of clearance from the lungs to the GI tract decreases continuously over several years after intake.

Stuart et al. (1970) developed a model to describe the biokinetics of plutonium following inhalation of PuO_2 , based on measurements on dogs up to 8 1/2 y (3100 d) after intake. Pulmonary retention was modeled with 85% IAD clearing with $t_{1/2}$ of 4 y, mainly to lymph nodes, but 9% to the GI tract, i.e., a clearance rate of $4 \times 10^{-5} \text{ d}^{-1}$. The remaining 15% IAD cleared to GI tract with a $t_{1/2}$ of 50 d initially, doubling every year, i.e., a rate of $0.014 \exp(-0.69/365t) \text{ d}^{-1}$.

Métivier et al. (1977) found that, for PuO_2 inhaled by baboons, the lung retention $t_{1/2}$ increased with the duration of measurements up to 1000 d, and concluded that the particle excretion rate decreased continuously.

Snipes et al. (1983) modeled the biokinetics of ^{134}Cs following inhalation of labelled FAP based on measurements on dogs up to 850 d after intake. They estimated $m(t)$ to have the form $0.005 \exp(-0.03t) + 0.0001 \text{ d}^{-1}$, i.e., an initial decrease, to a low but constant level.

Kreyling et al. (1988) studied retention and clearance of ^{57}Co for up to 850 d after inhalation of labelled FAP or Co_3O_4 by dogs. They estimated that for both materials $m(t)$ started at $5 \times 10^{-4} \text{ d}^{-1}$ and decreased exponentially with $t_{1/2}$ of 170 d. These results are notable in that the rate was based on measurements of fecal samples involving a procedure that separated ^{57}Co -FAP from nonparticulate ^{57}Co , and therefore gave a direct measure of $m(t)$.

Conclusions for modeling

Reference values and uncertainties

Most experimental studies (Table 3.13) indicate that about 80% IAD remains at 50 d after intake, 70% at 100 d, and 50% at 300 d. Measurements of activity in the chest up to 5000 d after accidental inhalation (Figure E.9), and of activity in the lungs at autopsy decades after exposure show that some material is retained for a period of the order of 10,000 d.

Similarly, the experimental data indicate that for healthy non-smokers the average clearance rate of particles from the lungs to the GI tract decreases with time from $3 \times 10^{-3} \text{ d}^{-1}$ at 25 d after inhalation to $8 \times 10^{-4} \text{ d}^{-1}$ at 200 d, $5 \times 10^{-4} \text{ d}^{-1}$ at 350 d, and $4 \times 10^{-4} \text{ d}^{-1}$ at 250 – 1500 d. The few data found on fecal excretion of relatively insoluble material at later times indicate a rate of $4 \times 10^{-4} \text{ d}^{-1}$ at 1000 – 1500 d.

These results suggest that AI retention needs to be represented by three compartments, which clear at rates of about 0.02 d^{-1} (i.e., $t_{1/2}$ about 30 d), 0.001 d^{-1} , and 0.0001 d^{-1} (AI_1 , AI_2 , and AI_3 , respectively). The fraction of the AI deposit that goes to AI_1 ($a_1 = \text{DE}[\text{AI}_1]/\text{DE}_{\text{AI}}$) approximates to the fraction of IAD cleared at 100 d, i.e., 0.3. (At this time AI_1 has almost completely cleared, but there is little clearance from either AI_2 or AI_3 .) 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, 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 $a_2 = 0.6$. (The concept of a very long-term retention compartment in lung is not new. Thomas [1968], in modeling transport of relatively insoluble materials from lung to lymph nodes, assumed that 10% of material in the lungs at 10 d was retained with $t_{1/2}$ of 10,000 d).

In considering uncertainty and modifying factors it is unrealistic to treat all the parameters as variables, partly because of lack of information, but also because considerable variation in the retention pattern can be achieved by altering either the amount in each compartment or the clearance rates. Among the considerations in deciding which parameters to make "variable", it was noted that Bohning et al. (1982) had observed that in smokers the "intermediate" phase (AI_1) was often absent, and the slow-phase $t_{1/2}$ increased with cigarette consumption expressed in pack-years. It is therefore proposed that a_1 is treated as one variable; assumed that a_3 remains 0.1; and hence that

$a_2 = 0.9 - a_1$. The clearance rates from Al_2 and Al_3 ($m_{2,4}$ and $m_{3,4}$ in Table 3.1) are also treated as variables.

The value of a_1 is well defined since it approximates the fraction of IAD cleared at 100 d. The experiments with polystyrene, FAP, and Teflon give average retention in nonsmokers at 100 d between 60% and 75% IAD. Varying a_1 by $\pm 20\%$ results in retention at 100 d varying from 64% to 73% IAD (Table 3.14). It is therefore proposed that for $a_1 \Phi_u = 1.2$.

The clearance rate at 200 d to 300 d after inhalation is mainly determined by clearance from Al_2 . From Figure E.8 this is likely to be in the range of $4 \times 10^{-4} \text{ d}^{-1}$ to $1.5 \times 10^{-3} \text{ d}^{-1}$, suggesting that for $m_{2,4} \Phi_u = 2$. The effect of varying $m_{2,4}$ by a factor of two is shown in Table 3.14. The clearance rate at 200 d varies over a range similar to that observed: from $6 \times 10^{-4} \text{ d}^{-1}$ to $1.8 \times 10^{-3} \text{ d}^{-1}$.

From Figure E.10, retention at 5000 d can be up to 60% of that at 300 d, i.e., up to 30% IAD. In view of the great uncertainty in long-term lung retention, it is assumed that for $m_{3,4} \Phi_u = 3$. The effect of varying $m_{3,4}$ by a factor of three is shown in Table 3.14. Retention at 5000 d ranges from 3% to 9% IAD.

Modifying factors: Cigarette smoking.

In all studies included in Table 3.13 that compared smokers with nonsmokers, retention was greater in smokers. The difference was most marked in the MPG studies, suggesting that smoking may impair both particle transport and absorption into blood. Cigarette smoking was found to impair alveolar clearance of $^{239}\text{PuO}_2$ in rats (Filipy et al., 1980, 1981a) provided there was exposure to smoke before and after inhalation of plutonium and also possibly in dogs, but on the basis of small numbers of animals (Filipy et al., 1981b, 1982). Bohning et al. (1982) observed no "intermediate" phase in five out of eight current cigarette smokers, giving an average of 7% for a_1 , compared to 27% in non-smokers and 26% in ex-smokers. Hence Φ_m is taken to be 0.3 for a_1 . They also observed longer slow-phase $t_{1/2}$ in smokers than non-smokers. Clearance rates corresponding to the slow-phase half-times were calculated. The average in smokers was $1.6 \times 10^{-3} \text{ d}^{-1}$, compared to $2.4 \times 10^{-3} \text{ d}^{-1}$ in non-smokers. Hence Φ_m is taken to be 0.7 for $m_{2,4}$. Kathren et al. (1993) found that the concentrations of $^{239+240}\text{Pu}$ and ^{241}Am in lymph nodes, relative to their concentrations in lungs, were greater in non-smokers than in smokers (see Section E.7). This is consistent with long-term lung clearance in smokers also being slower than in

non-smokers, and hence it is proposed that Φ_m is also taken to be 0.7 for $m_{3,4}$.

3.6 The Lymph Nodes

Annex E includes a review of postmortem measurements of the concentrations of materials in lungs and associated lymph nodes (represented by [L] and [LN], respectively). It is restricted to studies on human tissues, on materials which would be expected to be relatively insoluble, and for which measurements on both lung and lymph nodes were reported. Only a summary, and the conclusions are given here.

Results, summarized in Tables E.25 through E.30, are presented where possible on the basis of concentration ratio [LN]/[L], since this facilitates comparisons between results from exposures at different levels, and between observations and model predictions. The presentation of results in the literature is variable: individual values or central values for groups (median, arithmetic, or geometric mean) may be given, expressed as the amounts in lung and lymph nodes, the concentrations, or the concentration ratio. In addition to the general problems relating to the interpretation of measurements following unplanned exposures, particularly uncertainty about the intake, there are specific problems associated with the measurement of lymph node contents. Since it may not be possible to analyze complete organs, concentrations are frequently determined. The concentration of material, even in adjacent nodes in the same individual, is extremely variable. It may be so low that samples from a number of individuals are combined.

The information falls into four main categories: environmental exposure to nuclear weapons fallout, particularly plutonium; and environmental exposure to natural long-lived radionuclides, particularly thorium isotopes; occupational exposure to actinides, especially plutonium; and occupational exposure to mine dusts.

Summary of postmortem measurements of material in lymph nodes

There is now a considerable body of data on retention of inhaled materials in human lymph nodes, which relate to a variety of materials and to both high and low levels of exposure. Material is consistently found in both lungs and lymph nodes, even many years after exposure has ceased. The concentration in lymph nodes is generally higher than in the lungs. The concentration ratio is very variable, but amongst groups with similar exposures median values are usually between 1 and 20. The ratio [LN]/[L] is generally lower for coal miners, but it would not be surprising if their

high dust exposure levels resulted in impairment of clearance from the lungs. Since the mass of the TBLN (typically 0.015 kg) is so much smaller than that of the lungs (1 kg), the concentration ratio would have to exceed 60 for the amount of material in lymph nodes to exceed that in the lungs. While ratios in some individuals have reached such values, they are exceptional, and a ratio of about 10 would seem to be a reasonable representative value.

Particle transport from lymph nodes

The presence of significant amounts of material in the lung-associated lymph nodes of people many years after exposure, and the accumulation of material in lymph nodes observed in animal inhalation studies, indicate that the particle transport rate from lymph nodes to blood must be extremely low. For modeling purposes it is therefore assumed in the IRTM that there is no direct movement of particles from lymph nodes to bloodstream, although, as stated elsewhere, absorption into blood (i.e., dissolution and uptake) is assumed to occur at the same rate as in the respiratory tract itself.

Conclusions for modeling

Reference values and uncertainties

Analyses of tissues taken at autopsies, following exposures many years prior to death, have shown typical concentrations in LN_{TH} about 10 times that in the lungs. However, in the one study that distinguished smokers from non-smokers (Kathren et al., 1993), the ratio was about 10 in smokers, but 20 in non-smokers. The transport rate from AI to LN_{TH} in the compartment model was therefore chosen to give this ratio at 10,000 d after inhalation. For simplicity, it is modeled only by transport from AI₃, and a rate of 0.00002 d⁻¹ is needed to provide the required amount in LN_{TH}. Since it is based on a large amount of human data, it is proposed that $\Phi_u = 2$.

Modifying factors

No modifying factors are recommended. Kathren et al. (1993) did find lower values of [LN]/[L] for both plutonium and americium in smokers than in non-smokers. While this could be due to reduced clearance to lymph nodes, it might result from increased AI retention, which is already taken into account.

3.7 Application to Question 5

5. Assuming completely insoluble particles (1 μm AMAD), retention in each respiratory tract region in mature adults as a % of the total initial deposition in the respiratory tract, as a function of time after deposition?

There are two aspects to this:

- (i) Deposition in each region (TB, AI) as % total initial deposition in the respiratory tract.
- (ii) Retention in each region with respect to time, as % initial deposition in each region. Note that there will be material in TB following deposition in AI, since particles clear from AI through TB to the GI tract, but not vice versa.

Initial deposition in TB and AI

The initial deposits and their uncertainties are based on the results of Questions 3 and 4. However, since the uncertainties on deposition will be combined with uncertainties in retention, I have estimated a narrower range, nominally x₁₅ and x₈₅. The following steps refer to line numbers in Table 3.15.

1. Take x₅, x₅₀ and x₉₅ for ET deposition as % total respiratory tract from Question 3.
2. Calculate x₅, x₅₀ and x₉₅ for Lung deposition as % total respiratory tract from Line 1 (100-ET).
3. Express x₅ and x₉₅ as ratio to x₅₀. Calculate x₁₅ and x₈₅ as ratio to x₅₀ on basis that x₁₅/x₅₀ = $\sqrt{x_5/x_{50}}$ and x₈₅/x₅₀ = $\sqrt{x_{95}/x_{50}}$.
4. Calculate x₁₅, x₅₀ and x₈₅ for Lung deposition as % total respiratory tract from Line 3. (x 43%).
5. Take x₅, x₅₀ and x₉₅ for TB deposition as % total Lung from Question 4.
6. Express x₅ and x₉₅ as ratio to x₅₀. Calculate x₁₅ and x₈₅ as ratio to x₅₀ on basis that x₁₅/x₅₀ = $\sqrt{x_5/x_{50}}$ and x₈₅/x₅₀ = $\sqrt{x_{95}/x_{50}}$.
7. Calculate x₁₅, x₅₀ and x₈₅ for TB deposition as % total Lung from Line 6. (x 29%).
8. Calculate x₁₅ and x₈₅ for TB deposition / total respiratory tract as ratio to x₅₀ from Lines 3 and 6. (r.m.s. of corresponding values).
9. Calculate x₁₅, x₅₀ and x₈₅ for TB deposition as % total respiratory tract from line 8 (x 0.29 \times 43 = 12.5%).
10. Estimate x₁₅, x₅₀ and x₈₅ for AI deposition as % total respiratory tract, by consideration of Line 4 (total lung) and Line 8 (TB / respiratory tract).

Retention of particles deposited in TB and AI

As a starting point I took the parameter values in the IRTM (Table 3.1), which I was largely involved in selecting. Parameters relevant to TB and AI are given in Table 3.16. The only changes to the IRTM values, as discussed above, are the parameters for retention of particles in the TB airway wall. Thus the fraction going to BB_{seq} and bb_{seq} I have set to 0.0005 (as for ET_{seq}), instead of the IRTM value of 0.007. The clearance rates from BB_{seq} and bb_{seq} to LN_{TH} I have set to 0.001 d^{-1} (as for ET_{seq} to LN_{ET}), instead of 0.01 d^{-1} as in the IRTM. (This will reduce the amount transferred to thoracic lymph nodes from BB and bb. To maintain the target concentration ratio of 20, this should be offset by an increase in the transfer from AI to lymph nodes, but the effect on AI retention will be small and I have not attempted to calculate a revised transfer rate $m_{3,10}$.) I took f_s to be 0.5, for particles around $1\text{ }\mu\text{m}$.

To provide lower and upper values (nominal x_{15} and x_{85} values) to use in combination with other retention parameters, and with the depositions above, I have in most cases taken the square root of the "uncertainty factor", and divided and multiplied the reference value by this. I made an exception in the case of slow bronchial clearance. There is still a reasonable chance that it does not occur at all, and therefore the lower estimate on TB retention is to take the slow cleared fraction $f_s = 0$. There is also a possibility that it exists and applies to material cleared through TB from AI, and this is included in taking an upper estimate.

In the first version of the IRTM formulated by the Task Group, slow TB clearance was not included, but had been the subject of debate, and the impact on doses to the TB region of including it was considered (James et al., 1989). Subsequently a revised model was proposed, in which airway retention was included (Bailey et al., 1991). The initial assumption made was that 20% was retained, independent of particle size. This was explicitly stated to be a compromise between two apparently inconsistent observations: about 50% retention from the Frankfurt bolus experiments; no retention when large particles are deposited in the lungs. It was also assumed at that time that material in transit was also subject to retention, and to the same degree. But it was recognized that this assumption was made in the absence of clear evidence one way or the other. After discussion with colleagues, the latter was dropped. The view taken was that strong evidence was needed to introduce a new concept into the model: There was sufficient evidence to include retention of material deposited, but not of material in transit. The sensitivity of

doses to these various assumptions has been investigated (Bailey et al., 1995).

These values were used to calculate retention in the TB and AI regions. LUDEP could not readily be used, since although it will provide the activity remaining in each organ at any chosen time after intake, it only gives the activity in the lungs as a whole. A modified version was kindly prepared by Dr. A. Birchall, NRPB, that displayed the amounts in TB and AI separately and excluding the thoracic lymph nodes. This saved effort in setting up separate calculations. To include the effect of slow clearance in TB on 50% of particles in transit from AI, I reduced the rates of fast clearance in BB and bb to twice the x_{15} slow clearance rate, i.e., twice 0.017 d^{-1} .

Table 3.17 shows the effects of these parameter values. The second and third columns give retention in AI and TB respectively, following 100% deposition in AI. The fourth column gives retention in TB following 100% deposition in TB (of which 54% is in BB and 46% in bb, as given in Table 2.9 for a $1\text{ }\mu\text{m}$ AMAD aerosol). In the final two columns retention in AI and TB are given for the deposition fractions derived in Table 3.15. As also shown in Table 3.17, the modifying factors for cigarette smoking have a marked effect on alveolar retention, especially at later times, when the retention is greater than the estimated 95-percentile for non-smokers. Smoking has relatively little effect on TB retention because its effect on slow TB clearance is not known. The penultimate set of rows of Table 3.17 give the median values for a population with 25% smokers. I did not feel it necessary to include smoking in the estimated 5 and 95% values.

As expected, because of the great uncertainty about the extent of slow TB clearance, the confidence interval on TB retention after 1 hour is very large, and much greater than that of AI retention. The recent 3-y study with ^{195}Au -labelled Teflon (Philipson et al., 1992; 1996) gives greater alveolar retention at 365 and 3650 d (74% and 26% respectively for non-smokers and ex-smokers combined) than even the upper 95-percentile in Table 3.17 (59% and 13% respectively). Had these results been available earlier they might well have influenced the IRTM reference values and uncertainties. However, it should be noted that this study gives higher retention at 30 and 100 d, when leaching should not be a factor; some of the long-term retention may well be lymph nodes rather than lung; and the 3650 d estimate is an extrapolation from 900 d.

3.8 Application to Question 6

6. *By what factors would you expect the median values to be different in 5 year old children?*

In developing the IRTM, consideration was given to possible differences in clearance between children and adults since the model is age-dependent, and was intended for use in assessing doses to the general population. It was concluded, however, in *ICRP Publication 66* Section 7, that "...there are almost no experimental data on clearance in normal children (Section E.4.4.2). Even for laboratory animals there are insufficient data to establish clear quantitative differences between immature and adult mammals, but the limited information suggests that any such differences are not large. Therefore it is not at present possible to define clearance rates specifically for each of the age groups from newborn to adult designated by ICRP. In the absence of such information, the reference values for adults are also applied to children."

ICRP Publication 66 Annex E includes a few relevant references, notably in Section E.4.4.2, which concerns the effects of modifying factors on particle transport by mucociliary clearance in the posterior nasal passage, which is relatively easy to study, compared to the lungs. It is noted there that of particular interest, in view of the objective of providing a model relevant to the general population, is that this is the one aspect of particle clearance from the respiratory tract that has been measured in healthy children. Kärjä et al. (1982) measured mucociliary transport velocities in four children (aged 2, 5, 6, and 15 y) and obtained rates of 6 – 10 mm min⁻¹, well within the range they measured in adults. Passali and Ciampoli (1985) measured transit times in a larger group (33 girls and 21 boys, aged 3 – 12 y). They did not report a direct comparison with adults, but the average transit time measured (10 min) was very similar to those measured in adults (Table E.4).

With respect to lung (TB) mucociliary clearance (LMC), only one study was found that reported LMC in children without lung disease, but there were no equivalent measurements in adults (Hühnerbein et al., 1984). As noted in response to Question 5, because of differences in methodology, it is very difficult to compare measurements of LMC from different studies. Gerrard et al. (1986) found LMC to be significantly faster in female nonsmokers than in males, and suggested that this might well have been due to smaller lung dimensions – particles have shorter distances to travel in smaller lungs. If this explanation for faster LMC in women is correct, then it should also be faster in children.

Animal studies

There have been a number of animal studies of the effect of age on clearance of inhaled particles. These were not reviewed in Annex E, and there has not been time to conduct an exhaustive review for this exercise, but I had a few references to hand. As noted by Hackett et al. (1980) it is important to use monodisperse particles in such studies, since differences in deposition at different sizes will affect the size distribution of particles deposited in each region at different ages.

Raju et al. (1983) and Yeates et al. (1983) measured LMC of monodisperse ^{99m}Tc-labelled iron oxide particles inhaled by infant baboons. The results reported appear to be preliminary, but the time to clear 50% of the "bronchial retention", defined as the initial lung deposit minus that remaining at 24 h, was similar in baboons aged 1 – 4 weeks, 1 – 3 months and 1.5 years.

Davies and Webster (1988) measured LMC of monodisperse ^{99m}Tc-labelled polystyrene particles inhaled by calves at age 2 months and again at age 8 months. The rate constant measured for LMC was higher in the older calves, but not significantly.

Alveolar region

Hackett et al. (1980, 1981) investigated the effects of particle size and animal age on deposition and retention of ⁵¹Cr-labelled monodisperse polymer microspheres inhaled by rats. They found lung retention and clearance to be independent of animal age (juvenile – 13 d old; weanling – 21 d old; adult) over an 8-week observation period.

A number of studies have been conducted at the Inhalation Toxicology Research Institute, Albuquerque, that have investigated the effect of age on the radiation-induced damage following inhalation of relatively insoluble particles. As part of the dosimetry analysis, consideration was given to deposition and retention. Lung retention was found to be similar in immature and adult animals following inhalation of: ²³⁹PuO₂ by dogs (Guilmette et al., 1986, 1987a); ¹⁴⁴Ce-labelled fused aluminosilicate particles (FAP) by dogs (Guilmette et al., 1987); and ¹⁴⁴Ce-oxide by mice (Lundgren et al., 1980a, who refer to the similarities in lung retention being consistent with findings in hamsters and dogs exposed to insoluble forms of ¹⁴⁴Ce).

Mewhinney and Muggenburg (1985) followed lung retention for 2 y after acute inhalation by immature (90-d-old), young adult (12–14 months) and aged (6–10 y) beagle

dogs of polydisperse $^{241}\text{AmO}_2$. There was no significant difference in lung retention between the immature and young adult dogs, but retention in the aged dogs was greater up to about 200 d after exposure.

Collier et al. (1991) studied lung retention following acute inhalation by rats aged 3, 13, 21 and 46 weeks, of monodisperse ^{57}Co -labelled Co_3O_4 , for which the rates of particle transport to GI tract and absorption to blood from the lungs are of similar magnitude and could be determined separately. They concluded that particle transport rates were similar at the different ages, but that absorption to blood was faster in younger animals.

Moskalev et al. (1988) studied lung retention following acute inhalation by rats aged 1, 2, 4 and 16 weeks, of ^7Be -labelled BeF_4 . They reported slower clearance in the

younger animals. However, interpretation is complicated because the aerosol was polydisperse, and the material was moderately soluble: by 4 d the content of the skeleton was similar to that of the lungs.

There is no basis for changing particle clearance rates significantly for children. However, the median values in the answers to Question 5 are for a population containing 25% cigarette smokers with impaired clearance, and this would not be appropriate for children. I have therefore taken the retention pattern for non-smokers. More importantly, the initial deposition pattern is different in children. From Table 2.9, deposits in TB and AI are 17.6% and 37.4% of the total respiratory tract deposit, compared to 12.5% and 30.5% respectively, in adults, mainly because of the lower deposition in ET resulting from more common mouth-breathing.

Table 3.1. Reference Values and Uncertainties of Parameters for the Compartment Model to Represent Time-Dependent Particle Transport from Human Respiratory Tract (ICRP, 1994a; Bailey and Roy, 1994)

A. Clearance rates					
Pathway	From	To	Rate, d^{-1}	Half-time ^(a)	Uncertainty factor, Φ_u
$m_{1,4}$	Al ₁	bb ₁	0.02	35 d	--
$m_{2,4}$	Al ₂	bb ₁	0.001	700 d	2
$m_{2,4}$	Al ₃	bb ₁	0.0001	7000 d	3
$m_{3,10}$	Al ₃	LN _{TH}	0.00002	--	2
$m_{4,7}$	bb ₁	BB ₁	2	8 h	2
$m_{5,7}$	bb ₂	BB ₁	0.03	23 d	3
$m_{6,10}$	bb _{seq}	LN _{TH}	0.01	70 d	3
$m_{7,11}$	BB ₁	ET ₂	10	100 min	1.5
$m_{8,11}$	BB ₂	ET ₂	0.03	23 d	3
$m_{9,10}$	BB _{seq}	LN _{TH}	0.01	70 d	3
$m_{11,15}$	ET ₂	GI tract	100	10 min	3
$m_{12,13}$	ET _{seq}	LN _{ET}	0.001	700 d	3
$m_{14,15}$	ET ₁	Environment	1	17 h	3
B. Partition of deposit in each region between compartments ^(b)					
Region or deposition site	Compartment	Fraction of deposit in region assigned to compartment ^(c)		Uncertainty factor, Φ_u	
ET ₂	ET ₂	0.9995		--	
	ET _{seq}	0.0005		3	
BB	BB ₁	0.993 -- f_s		--	
	BB ₂	f_s		3	
	BB _{seq}	0.007		3	
bb	bb ₁	0.993 -- f_s		--	
	bb ₂	f_s		3	
	bb _{seq}	0.007		3	
Al	Al ₁	0.3		1.2	
	Al ₂	0.6		--	
	Al ₃	0.1		--	

(a) The half-times are approximate since the reference values are specified for the particle transport rates and are rounded in units of d^{-1} . A half-time is not given for the transport rate from Al₃ to LN_{TH}, since this rate was chosen to direct the required amount of material to the lymph nodes. The clearance half-time of compartment Al₃ is determined by the sum of the clearance rates from it.

(b) See paragraph 181, Chapter 5 of ICRP (1994a) for default values used for relating f_s to d_{ae} .

(c) It is assumed that the slow-cleared fraction f_s is size-dependent. For modeling purposes f_s is taken to be: and

$$f_s = 0.5 \text{ for } d_{ae} \leq 2.5\sqrt{\rho/\chi} \text{ } \mu\text{m}$$

$$f_s = 0.5e^{-0.63(d_{ae}\sqrt{\chi/\rho}-2.5)} \text{ for } d_{ae} \leq 2.5\sqrt{\rho/\chi} \text{ } \mu\text{m}$$

Table 3.2. Default absorption rates for Type F, M, and S materials (ICRP, 1994a)^a

Type		F (fast)	M (moderate)	S (slow)
Model parameters (d ⁻¹):	s_p	100	10	01
	s_{pt}	0	90	100
	s_t	--	0.005	0.0001

a No 'bound' state assumed for default Types.

Table 3.3. Tracheal Mucociliary Transport Rates in Healthy Humans (Bailey and Roy, 1994, Table E.8)

Technique	Material	Particle size, μm	Velocity, $\text{mm min}^{-1} \pm \text{SD}$	Reference
Bronchoscope Cine-photography	Teflon	680	2.15 ± 5.5	Santa Cruz et al. (1974)
	Teflon	680	22.9 ± 6.4	Sackner et al. (1975)
	Teflon	1000	11.3 ± 3.2	Wood et al. (1976)
	Teflon	1000	18.5 ± 6.0	Toomes et al. (1981)
	Polyethylene			
Bronchoscope (radiography)	Teflon		9.8 ± 2.4	Goodman et al. (1977)
	Teflon	1000	11.2 ± 3.6	Friedman et al. (1977)
	Teflon	1000	10.1 ± 3.5	Goodman et al. (1978)
	Teflon	1000	11.6 ± 3.6	Mezey et al. (1978)
	Teflon		6.5 ± 2.1	Sackner et al. (1979)
Bronchoscope (radiotracer)	Albumin	3-7	15.4 ± 1.7	Chopra et al. (1979)
Radioaerosol	Albumin	0.5	4.7 ± 3.1	Yeates et al. (1975)
	Albumin	0.5	4.4 ± 1.3	Wong et al. (1977)
	Sulphide	0.02-5	9	Ross et al. (1979)
	Fe_2O_3	-2	4.2 ± 2.5	Yeates et al. (1979)
	Fe_2O_3	-4	5.5 ± 1.0	Foster et al. (1980)
	Fe_2O_3	-2	4.3 ± 1.9	Leikauf et al. (1981)
	Fe_2O_3	-4	5.1 ± 2.9	Yates et al. (1981b)
	Fe_2O_3	-2	6.7 ± 3.0	Gerrity et al. (1983)
	Fe_2O_3	-4	5.7 ± 1.4	Leikauf et al. (1984)
	Albumin	-2	4.8 ± 1.6	Gerrard et al. (1985)
	Albumin	-2	4.3 ± 1.1	Katz et al. (1987)
	Fe_2O_3	-4	4.7 ± 1.3	Zwas et al. (1987)
			4.9 ± 1.3	Mussatto et al. (1988)

Table 3.4. Times for Lung Regions to Clear Tantalum Dust (Gamsu et al., 1973)

Region	50% clearance time		100% clearance time
	Mean	Range	
Trachea and large bronchi(a)	2.5 h	1-4 h	20 h
Trachea and large bronchi	5.5 h	2-9 h	4 d
Small bronchi (1-6 mm dia)	14 h	6-22 h	12 d
Proximal bronchioles	34 h	24-48 h	17 d
Distal bronchioles (last 2-3 generations)	4 d	2-9 d	21 d
Terminal units	Increase up to 2 d; no observable clearance up to 15 months		

(a) Tracheography. All other results for bronchography.

Table 3.5. Calculated Mucus Velocities and Clearance Times (Bailey and Roy, 1994, Table E.10)

Generation ^(a)	Mucus velocities, v_i , mm min ⁻¹			Clearance times, min (Yu et al., 1986)
	Lee et al., 1979	Yu et al., 1986	Cuddihy and Yeh, 1988	
0 (trachea)	5.5	6.5	5.5	22
1	4.1	6.6	3.5	12
2	3.0	2.5	2.0	10
3	2.2	1.2	1.1	8
4	1.4	0.89	0.9	19
5	0.88	0.69	0.9	29
6	0.55	0.54	0.7	35
7	0.34	0.43	0.6	38
8	0.21	0.36	0.4	35
9	0.13	0.29	0.3	28
10	0.074	0.21	0.2	31
11	0.044	0.15	0.1	48
12	0.025	0.11	0.05	65
13	0.015	0.08	0.02	73
14	0.0082	0.05	0.007	104
15 terminal	0.0036	0.04	0.001	115
16 bronchioles		0.03		207

(a) Cuddihy and Yeh (1988) refer to the trachea as generation 1.

Table 3.6. Fraction A of Thoracic Deposit Retained in Slow Phase Following Bolus Inhalation
(Bailey and Roy, 1994, Table E.11)

1. Volumetric flow rate of inspired air $\sim 250 \text{ mL s}^{-1}$ (continued).							
Subject	d_{ae} , μm	$V_B^{(a)}$, mL	$t_b^{(b)}$, s	Position ^(c)	Material	A	Reference
25	1.2	45	29	S	Fe_2O_3	0.66	Stahlhofen et al. (1986b)
6	1.8	50	36	S	Fe_2O_3	0.70	Scheuch (1991)
1	1.4	70	10	S	Fe_2O_3	0.44	Scheuch (1991)
1	1.8	70	24	S	Fe_2O_3	0.60	Stahlhofen et al. (1990)
25	2.9	30	15	L	FAP	0.50	Scheuch (1991)
6	3.0	30	65	S	Fe_2O_3	0.62	Stahlhofen (1989)
4	3.1	35	31	S	Fe_2O_3	0.47	Stahlhofen (1989)
2	3.5	35	9.5	S	Fe_2O_3	0.57	Stahlhofen (1989)
6	2.9	35	52	L	FAP	0.73	Stahlhofen et al. (1987b)
31	2.9	40	15	S	FAP	0.57	Scheuch (1991)
1	3.1	45	5	S	Fe_2O_3	0.41	Stahlhofen (1989)
25	3.4	45	4	S	Fe_2O_3	0.57	Stahlhofen (1989)
25	2.9	45	5	S	Fe_2O_3	0.59	Stahlhofen et al. (1986b)
25	3.4	45	20	S	Fe_2O_3	0.47	Stahlhofen et al. (1986a)
25	2.8	50	10	S	FAP	0.63	Scheuch (1991)
25	3.0	50	40	S	Fe_2O_3	0.62	Stahlhofen (1989)
1	3.4	50	17	S	Fe_2O_3	0.42	Stahlhofen et al. (1986a)
6	3.2	50	13	S	Fe_2O_3	0.57	Stahlhofen (1989)
6	3.4	50	14	S	Fe_2O_3	0.56	Stahlhofen et al. (1986a)
1	3.5	60	7	S	FAP	0.50	Scheuch (1991)
25	3.7	60	20	L	Fe_2O_3	0.51	Scheuch (1991)
25	3.3	64	12.5	S	FAP	0.60	Scheuch (1991)
25	3.1	65	50	L	FAP	0.80	Stahlhofen et al. (1987b)
1	3.2	70	9	S	Fe_2O_3	0.58	Stahlhofen et al. (1986a)
1	3.1	70	9	S	FAP	0.54	Stahlhofen et al. (1987b)
1	2.9	70	9	S	Fe_2O_3	0.45	Stahlhofen et al. (1986b)
4	3.2	70	8.5	S	Fe_2O_3	0.60	Stahlhofen et al. (1986a)
30	3.0	70	6	L	FAP	0.72	Stahlhofen et al. (1987b)
2	3.0	70	49	L	FAP	0.83	Stahlhofen et al. (1987b)
25	3.4	80	1.5	S	Fe_2O_3	0.61	Stahlhofen et al. (1986b)
25	3.4	80	8	S	Fe_2O_3	0.55	Stahlhofen et al. (1986b)
25	3.0	80	8	S	Teflon	0.59	Stahlhofen et al. (1986b)
2	3.0	85	20	S	Fe_2O_3	0.70	Stahlhofen (1989)
25	3.2	120	7	S	Fe_2O_3	0.60	Stahlhofen et al. (1986a)
4	3.0	125	38	L	FAP	0.79	Stahlhofen et al. (1987b)
4	2.9	135	7	S	Fe_2O_3	0.56	Stahlhofen et al. (1986a)
6	3.4	145	16	S	Fe_2O_3	0.57	Stahlhofen et al. (1986a)

**Table 3.6. Fraction A of Thoracic Deposit Retained in Slow Phase Following Bolus Inhalation
(Bailey and Roy, 1994, Table E.11) (Continued)**

1. Volumetric flow rate of inspired air $\sim 250 \text{ mL s}^{-1}$ (continued).							
Subject	$d_{ae}, \mu\text{m}$	$V_F^{(a)} \text{ mL}$	$t_b^{(b)} \text{ s}$	Position ^(c)	Material	A	Reference
1	3.4	200	12	S	Fe_2O_3	0.79	Stahlhofen et al. (1986a)
4	3.4	275	14	S	Fe_2O_3	0.88	Stahlhofen et al. (1986a)
25	3.3	710	6.5	S	Fe_2O_3	0.97	Stahlhofen et al. (1986a)
4	6.0	50	20	S	FAP	0.20	Scheuch (1991)
25	6.0	50	20	L	FAP	0.33	Scheuch (1991)
25	5.6	55	5	S	Fe_2O_3	0.30	Scheuch (1991)
1	5.9	70	8	S	Fe_2O_3	0.25	Stahlhofen et al. (1990)
4	5.6	76	5	S	Fe_2O_3	0.30	Scheuch (1991)
25	3.7	48	10	S	PSL	0.33	Scheuch et al.(d) (1993a)
1	3.7	60	10	S	PSL	0.30	Scheuch, et al.(d) (1993a)
4	3.7	75	10	S	PSL	0.25	Scheuch, et al.(d) (1993a)
4	3.7	76	8	S	PSL	0.23	Scheuch, et al.(d) (1993a)
1	3.7	100	8	S	PSL	0.36	Scheuch, et al.(d) (1993a)
2	3.7	120	8	S	PSL	0.42	Scheuch, et al. (d) (1993a)
2. Volumetric flow rate of inspired air $\sim 700 \text{ mL s}^{-1}$.							
Subject	$d_{ae}, \mu\text{m}$	$V_F^{(a)} \text{ mL}$	$t_b^{(b)} \text{ s}$	Position ^(c)	Material	A	Reference
4	3.8	41	1	S	FAP	0.50	Stahlhofen et al. (1994)
6	3.8	54	1	S	FAP	0.40	Stahlhofen et al. (1994)
30	3.8	60	1	S	FAP	0.65	Stahlhofen et al. (1994)

(a) The bolus front depth, VF, is the volume of air that passes the larynx, from the time that the leading edge of the bolus passes it to the end of inspiration.
 (b) Breath-holding time. Many of these are taken from Stahlhofen (1989).
 (c) S = sitting; L = lying down.
 (d) Further details from G. Scheuch, personal communication.

Table 3.7. Observations of Airway Wall Retention in the Tracheobronchiolar Region (Bailey and Roy, 1994, Table E.12)

Species	Location	Material	Particle Size, μm	Route of Administration	Reference
Mouse	Bronchi	Soot		Inhalation	Duthie (1930)
Baboon	Bronchi	Iron oxide	95% $<5 \mu\text{m}$	Inhalation	Masse et al. (1973b)
Monkey, cat, rat	Bronchi	Tantalum		Inhalation	Masse et al. (1973b)
Guinea pig	Trachea	Horseradish per-oxidase, ferritin		Instillation	Richardson et al. (1976)
Mouse	Trachea, bronchi	Fe_2O_3	0.005	Inhalation	Watson and Brain (1979)
Rat	Trachea	BaSO_4	0.35 (CMD)	Instillation	Stirling and Patrick (1980)
Rat	Trachea	BaSO_4	0.35 (CMD)	Instillation	Gore and Patrick (1982)
Rat	Bronchi, bronchioles	UO_2	0.34 (CMD)	Inhalation	Gore and Patrick (1982)
Human	Tracheal bifurcation	Mineral		Inhalation	Henshaw and Fews (1983)
Rat	Trachea	FAP	5.7 (CMD)	Instillation	Patrick (1983)
Rat	Trachea	U/Pu oxide	0.7 (AMAD)	Inhalation	Briant and Sanders (1987)
Rat	Trachea	Gold Carbon PSL PSL	0.01 0.03 0.27 1.24	Instillation	Takahashi et al. (1989)
Dog	Bronchi, bronchioles	$^{239}\text{PuO}_2$		Inhalation	Filipy et al. (1985, 1986)
Human	Trachea, bronchi	Mineral		Inhalation	Henshaw et al. (1988)
Human (chrysotile miners)	Bronchi	Mineral		Inhalation	Churg and Wright (1988)
Human (lungs with cancer)	Bronchioles	Mineral		Inhalation	Churg and Stevens (1988)
Human (normal, non-smokers)	Bronchi	Mineral		Inhalation	Churg et al. (1990)

**Table 3.8. Long-Term Particle Retention in the Tracheal Wall Following Intratracheal Instillation in rats
(Bailey and Roy, 1994, Table E.13)**

Material	Particle CMD, μm	Time, d	% Retained (mean ± S.E.)	Reference
BaSO ₄	0.35	1	1.1 ± 0.1	Patrick and Stirling (1977)
		7	1.0 ± 0.2	
		30	0.9 ± 0.3	
UO ₂	0.35	1	0.39 ± 0.09	Patrick (1979)
		14	0.69 ± 0.19	
BaSO ₄	1.0	7	0.83 ± 0.55	Patrick (1983)
FAP	1.1	7	0.45 ± 0.12	
FAP	5.7	7	0.75 ± 0.35	
BaSO ₄	0.34	7	0.41 ± 0.13	Takahashi and Patrick (1987a)
		28	0.30 ± 0.09	
		56	0.13 ± 0.05	
		112	0.15 ± 0.10	
		168	0.10 ± 0.03	

Table 3.9. Permanent Factors Modifying Tracheal Mucociliary Transport Rates: Cigarette Smoking

Φ _m ^(a)	Reference
0.3	Goodman et al. (1977)
0.4	Goodman et al. (1978)
0.7	Chopra et al. (1979)
=	Yeates et al. (1975)
0.4	Toomes et al. (1981)

(a) Where a number is given, this is the factor by which the clearance rate should be multiplied. The symbol = indicates no effect.

Table 3.10. Permanent Factors Modifying Lung Mucociliary Clearance: Cigarette Smoking
(Bailey and Roy, 1994, Table E.17C)

Modifying Factor	P-Y ^(a)	Parameter ^(b)	Φ_m ^(c)	Reference
Smokers	16	$1/T_{50}$ $1/T_{90}$	0.5 --	Albert et al. (1969)
Smokers without restrictive disease	40	CT(120, 300)	=	Pavia et al. (1970)
Smokers, asymptomatic	10	CB(120)	0.3	Lourenço et al. (1971)
Smokers		CT(0-360)	=	Thomson and Pavia (1973)
Smokers asymptomatic	>3.7	CT(0-600)	+	Sanchis et al. (1974)
Smokers with chronic obstructive airway disease		CT(0-400)	+	
Smokers		CT(0-360)IZ CT(0-360)PZ	= --	Short et al. (1979)
Ex-smokers with chronic bronchitis	37	CT(60)	0.5	Puchelle et al. (1980)
Smokers, asymptomatic	21	CB(120)	=	Isawa et al. (1984)
Smokers, asymptomatic	17	CB(180)IZ CB(180)PZ	0.6 =	Agnew et al. (1986b)
Smokers, asymptomatic	<7.5	$1/T_{25}$ $1/T_{50}$ $1/T_{75}$	0.6 0.6 0.6	Foster et al. (1985)
Ex-smokers without chronic bronchitis	8	CT(60) CT(60)IZ CT(60)PZ	= = =	Vastag et al. (1985)
Ex-smokers with chronic bronchitis	35	CT(60) CT(60)IZ CT(60)PZ	0.7 0.5 =	Vastag et al. (1985)
Smokers without chronic bronchitis	9	CT(60) CT(60)IZ CT(60)PZ	0.8 0.8 =	Vastag et al. (1986)
Smokers with simple chronic bronchitis	21	CT(60) CT(60)IZ CT(60)PZ	0.5 0.6 0.6	Vastag et al. (1986)
Smokers with obstructive chronic bronchitis	41	CT(60) CT(60)IZ CT(60)PZ	0.4 0.4 0.5	Vastag et al. (1986)
Smokers (smoking-discordant twins)	25 10	CT(60) T_{50}, T_{95}	0.8 =	Camner and Philipson (1972) Bohning et al. (1975)

(a) CT(t) = Fraction of total lung deposit, cleared at time t (min).

CB(t) = Fraction of bronchial mucociliary clearance complete at time t (min), e.g.,

CT(t)CZ, CT(t)IZ, CT(t)MZ, CT(t)PZ As above but for central, inner, intermediate, and peripheral zone, respectively.

CB(t)CZ, CB(t)IZ, CB(t)MZ, CB(t)PZ

T_{avg} is the average retention time of particles cleared by bronchial mucociliary clearance.

$t_{1/2}$ clearance half-time (single exponential fit). T_{50} time for 50% bronchial clearance.

T_{90}, T_{95} time for 90%, 95% bronchial clearance. Changes in these suggest effect on small airways since they clear most slowly and hence determine total clearance time.

(b) Where a number is given, this is the factor by which the parameter is multiplied. The symbol = indicates no effect. The symbols + and - indicate unquantified increased and decreased clearance, respectively; ± indicates no significant change in group mean, but greater variation.

(c) Average cigarette consumption in pack-years (P-Y).

Table 3.11. Proposed Modifying Factors for the Compartment Model to Represent Particle Transport From the Human Respiratory Tract: Cigarette Smoking

Region	Parameter	$\Phi_m^{(a)}$
Bronchial (BB)	$m_{7,11}$ (BB ₁ to ET ₂)	0.5
Bronchiolar (bb)	$m_{4,7}$ (bb ₁ to BB ₁)	1
Alveolar-Interstitial (AI)	a_1	0.3
	$m_{2,4}$ (AI ₂ to bb ₁)	0.7
	$m_{3,4}$ (AI ₃ to bb ₁)	0.7

(a) Amount by which a parameter should be multiplied in order to calculate doses for a specific individual or group for which the modifying factor is recommended.

Table 3.12. Human Lung Retention Studies. (Bailey and Roy, 1994, Table E.19)

Material	Size, ^(a) μm	σ_g	Label	Subject Numbers and Habit ^(b)	Duration of Measurements, d	Retention Half-time, d	Reference
Polystyrene	5	1.1	⁵¹ Cr	7	20 - 160	150 - 300	Booker et al. (1967)
Polystyrene	5	1.1	⁵¹ Cr, ¹⁰³ Pd	3	80	140 - 340	Newton et al. (1978)
Iron oxide	MMD 1.4-2.3	~1.7	⁵⁹ Fe	4	Up to 100	~70	Albert and Arnett (1955); Albert et al. (1967)
Fe ₂ O ₃	3.6	1.2	⁵¹ Cr	1 S	56	70	Albert et al. (1967)
Fe ₂ O ₃	0.8	1.7	⁵¹ Cr	2	~60	62	Morrow et al. (1967a,b)
Fe ₂ O ₃	CMD 0.1	1.2	⁵¹ Cr	2	60	270 ± 20	Waite and Ramsden (1971a)
Fe ₂ O ₃	CMD 0.1 03		²³⁷ Pu	1 1	70	118 160	Waite and Ramsden (1971b)
Fe ₂ O ₃	95% <2.5		⁵⁹ Fe	2	60	100 - 280	LeBouffant et al. (1972); LeBouffant and Henin (1974)
Fe ₂ O ₃	90% <1		⁵⁹ Fe	2 NS 1 S	240	183, 560(c) 117(c)	LeBouffant and Henin (1974)
MnO ₂	MMD 0.9	1.75	⁵⁴ Mn	6	Up to 120	62 - 68	Morrow et al. (1967a,b)

(a) MMD = Mass Median Diameter; CMD = Count Median Diameter
 (b) NS = nonsmokers; S = smokers (where stated)
 (c) Between 10 and 52 d

**Table 3.13. Human Lung Retention Studies; Retention Evaluated at Different Times After Exposure
(based on Bailey and Roy, 1994, Table E.20)**

Material	Diameter, ^(a) μm	σ _g	Label	Habit ^(b)	Retention, ^(c) % Initial alveolar deposit X ± SD (n) or x ₁ , x ₂ (for n < 3)			Reference
					50 d	100 d	300 d	
Polystyrene	0.5		⁵¹ Cr	NS S	86 ± 12 (3) 93 ± 4 (6)			Jammet et al. (1978)
Polystyrene	3.5	~1.6	⁸⁵ Sr	NS S	73 ± 6 (11) 87 ± 11 (8)	61 ± 6 (11) 83 ± 10 (7)	37 ± 5 (6) 64 ± 19 (4)	Bohning et al. (1982)
FAP	1 4	1.1 1.1	⁸⁵ Sr ⁸⁸ Y	NS NS	85 ± 6 (12) 84 ± 6 (12)	75 ± 8 (12) 74 ± 9 (12)	50 ± 10 (12) 58 ± 11 (12)	Bailey et al. (1985)
Teflon H-L ^d L-L	4 4	1.1 1.1	⁵¹ Cr ⁵¹ Cr	NS NS	68 ± 1 (3) 72 ± 6 (3)	58 ± 5 (3) 65 ± 6 (3)	39 ± 18 (3) 53 ± 16 (3)	Philipson et al. (1985)
Fe ₃ O ₄	MMAD 2.8	1.4	MPG ^(e)	NS S	56 ± 6 (9) 76 ± 4 (3)	34 ± 7 (9) 62 ± 5 (3)	11 ± 4 (9) 49 ± 7 (3)	Cohen et al. (1979)
Fe ₃ O ₄	MMAD 1.1	1.4	MPG ^(e)	NS S CF COPD	59 ± 5 (13) 73 ± 7 (12) 69 ± 7 (8) 64 ± 13 (4)	34 ± 6 (13) 52 ± 9 (12) 47 ± 9 (8) 42 ± 16 (4)	16 ± 10 (6) 30 ± 13 (7)	Freedman et al. (1988)
Co ₃ O ₄	MGD 0.8 MGD 1.7 Combined 0.8 & 1.7	1.1 1.1	⁵⁷ Co	NS NS	78, 69 85, 77 77 ± 6 (4)	71, 49 81, 66 67 ± 13 (4)	37, 22 43, 33 34 ± 9 (4)	Foster et al. (1989) Pearman et al. (1989)
Fe ₃ O ₄	<i>d_{ae}</i> 2.8	1.1	MPG ^(e)	NS	76 ± 8 (5)	58 ± 13 (5)	22 ± 17 (5)	Möller (1991) Stahlhofen and Möller (1991)
Teflon	3.6	1.1	¹⁹⁵ Au	NS EX NS + EX S	95 ± 2 (4) 95 ± 2 (3) 95 ± 2 (7) 97 ± 1 (3)	90 ± 4 (4) 90 ± 5 (3) 90 ± 4 (7) 93 ± 1 (3)	76 ± 8 (4) 76 ± 10 (3) 76 ± 9 (7) 83 ± 3 (3)	Philipson et al. (1996)

(a) MGD = Mean geometric diameter; MMAD = Mass median aerodynamic diameter.

(b) NS = nonsmokers; S = smokers; EX = ex-smokers; CF = patients with cystic fibrosis; COPD = patients with chronic obstructive pulmonary disease.

(c) Lung retention, excluding rapid phase.

(d) H-L = "high-leaching"; L-L = "low-leaching" (different heat treatment).

(e) Magnetopneumography, i.e., measurement of the remnant magnetic field produced by aligned particles after a strong magnetic field has been applied to the chest.

Table 3.14. Comparison of Observed and modeled Alveolar-Interstitial Retention and Clearance, and Effects of Varying Parameters Over Ranges of Uncertainty (Bailey and Roy, 1994, Table E.24)

Parameter	Values substituted in model						
	Default	Vary a_1		Vary $m_{2,4}$		Vary $m_{3,4}$	
DE(AI ₁)/DE _{AI} (a_1) ^(a)	0.3	0.24	0.36	0.3	0.3	0.3	0.3
DE(AI ₂)/DE _{AI}	0.6	0.66	0.54	0.6	0.6	0.6	0.6
DE(AI ₃)/DE _{AI}	0.1	0.1	0.1	0.1	0.1	0.1	0.1
$m_{1,4}$ (d ⁻¹)	0.02	0.02	0.02	0.02	0.02	0.02	0.02
$m_{2,4}$ (d ⁻¹)	0.001	0.001	0.001	0.0005	0.002	0.001	0.001
$m_{3,4}$ (d ⁻¹)	0.0001	0.0001	0.0001	0.0001	0.0001	0.000033	0.0003
Retention, % IAD	modeled						
Time, d	Observed	78	82	75	80	75	78
50	80	78	82	75	80	75	78
100	68	68	73	64	71	63	68
300	50	54	59	50	61	43	54
5000	<30	6.5	6.5	6.4	11	6.1	8.9
Clearance Rate, $\times 10^{-4}$ d ⁻¹	modeled						
Time, d	Observed	49	40	58	45	56	49
25	30	49	40	58	45	56	49
200	8	10	9.9	11	6.1	18	10
350	5	8.4	8.5	8.3	4.5	16	8.3
1500	4	6.5	6.7	6.3	4.1	5.9	6.0
(a) The fraction of the AI deposit that goes to AI ₁ .							

Table 3.15. Estimated ranges of initial deposits in TB and AI as % total initial deposit in respiratory tract, 1 μm AMAD, adult

			x_5	x_{50}	x_{95}	x_{15}	x_{50}	x_{85}
1	ET/Total R.T. deposit	(Q3), %	40	57	85			
2	Lung/Total R.T. deposit	(100 - ET), %	15	43	60			
3		Ratio to x_{50}	0.35	1	1.4	0.59	1	1.18
4		% Total R.T.				25	43	51
5	TB/Lung (TB + AI)	%	15	29	60			
6		Ratio to x_{50}	0.52	1	2.07	0.72	1	1.44
7		% Total Lung				15	29	42
8	TB/Total R.T.	Ratio to x_{50} (rms)				0.46	1	1.86
9		% Total R.T.				5.7	12.5	23
10	AI/Total R.T.	% Total R.T.				20	30.5	40

Table 3.16. Values of Parameters for the Particle Transport Model

A. Clearance rates							
Pathway	From	To	Rate, d^{-1}	Uncertainty factor, Φ_U	$\sqrt{\Phi_U}$	x_{15}	x_{85}
$m_{1,4}$	AI ₁	bb ₁	0.02	—	—	—	—
$m_{2,4}$	AI ₂	bb ₁	0.001	2	1.4	0.0007	0.0014
$m_{3,4}$	AI ₃	bb ₁	0.0001	3	1.7	0.00006	0.00017
$m_{3,10}$	AI ₃	LN _{TH}	0.00002	2	1.4	0.000014	0.00003
$m_{4,7}$	bb ₁	BB ₁	2	2	1.4	1.4	3
$m_{5,7}$	bb ₂	BB ₁	0.03	3	1.7	0.017	0.05
$m_{6,10}$	bb _{seq}	LN _{TH}	0.001	3	1.7	0.0006	0.0017
$m_{7,11}$	BB ₁	ET ₂	10	1.5	1.2	8	12
$m_{8,11}$	BB ₂	ET ₂	0.03	3	1.7	0.017	0.05
$m_{9,10}$	BB _{seq}	LN _{TH}	0.001	3	1.7	0.0006	0.0017

B. Partition of deposit in each region between compartments						
Region or deposition site	Compartment	Fraction of deposit in region assigned to compartment	Uncertainty factor, Φ_U	$\sqrt{\Phi_U}$	x_{15}	x_{85}
BB	BB ₁	0.4995	—	—	0.9997	0.4991
	BB ₂	0.5	3	1.7	0	0.5*
	BB _{seq}	0.0005	3	1.7	0.0003	0.0009
bb	bb ₁	0.4995	—	—	0.9997	0.4991
	bb ₂	0.5	3	1.7	0	0.5*
	bb _{seq}	0.0005	3	1.7	0.0003	0.0009
AI	AI ₁	0.3	1.2	1.1	0.27	0.33
	AI ₂	0.6	—	—	0.63	0.57
	AI ₃	0.1	—	—	0.1	0.1

* Also applied to material in transit.

Table 3.17. Lung retention of insoluble particles, 1 μm AMAD

x_{50}	100% AI AI	100% AI TB	100% TB TB	30.5% AI AI	12.5% TB TB
0.0069 d	99.995	0.005	98.07	30.50	12.26
0.0417	99.97	0.027	89.88	30.49	11.24
1	99.34	0.337	50.73	30.30	6.44
30	84.66	0.235	19.63	25.82	2.53
365	51.24	0.026	0.036	15.63	0.012
3650	8.07	0.0013	0.0013	2.46	0.00056
Low (x_5)	100% AI AI	100% AI TB	100% TB TB	20% AI AI	5.7% TB TB
0.0069 d	99.99	0.005	95.64	20.00	5.45
0.0417	99.97	0.031	77.5	19.99	4.42
1	99.26	0.289	3.1	19.85	0.23
30	82.7	0.184	0.029	16.54	0.04
365	43.5	0.021	0.016	8.70	0.0051
3650	5.16	0.0005	0.00006	1.03	0.00010
High (x_{95})	100% AI AI	100% AI TB	100% TB TB	40% AI AI	23% TB TB
0.0069 d	100	0.005	98.45	40.00	22.65
0.0417	99.98	0.024	91.69	39.99	21.10
1	99.42	0.579	54.39	39.77	12.74
30	86.49	11.85	28.99	34.60	11.41
365	58.54	2.16	0.169	23.42	0.90
3650	12.53	0.232	0.01	5.01	0.095
Smokers	100% AI AI	100% AI TB	100% TB TB	30.5% AI AI	12.5% TB TB
0.0069 d	100	0.0016	99.01	30.50	12.38
0.0417	99.99	0.0099	94.41	30.50	11.80
1	99.76	0.138	52.2	30.43	6.57
30	94.23	0.109	19.65	28.74	2.49
365	72.42	0.0313	0.036	22.09	0.014
3650	13.49	0.0034	0.0013	4.11	0.0012
x_{50} 25% smokers	100% AI AI	100% AI TB	100% TB TB	30.5% AI AI	12.5% TB TB
0.0069 d	100.00	0.0042	98.31	30.50	12.29
0.0417	99.98	0.0227	91.01	30.49	11.38
1	99.45	0.2873	51.10	30.33	6.47
30	87.05	0.2035	19.64	26.55	2.52
365	56.54	0.0273	0.036	17.24	0.0128
3650	9.43	0.0018	0.0013	2.87	0.00072

Table 3.17. Lung retention of insoluble particles, 1 μm AMAD (Continued)

Child	100% AI AI	100% AI TB	100% TB TB	37.4% AI AI	17.6% TB TB	Child/ α_{50} (25% smokers)	
						AI	TB
0.0069	99.995	0.005	98.07	37.40	17.26	1.23	1.40
0.0417	99.97	0.027	89.88	37.39	15.83	1.23	1.39
1	99.34	0.337	50.73	37.15	9.05	1.22	1.40
30	84.66	0.235	19.63	31.66	3.54	1.19	1.41
365	51.24	0.026	0.036	19.16	0.016	1.11	1.25
3650	8.07	0.0013	0.0013	3.02	0.00072	1.05	0.99

4. Questions 7-8. Absorption from the Respiratory Tract

Question 7. Absorption (dissolution and transfer) to blood in adults, % of the total initial deposition (1 mm AMAD) in the respiratory tract.

4.1 Treatment in ICRP Publications 66 and 71

As described in the response to Question 5, *ICRP Publication 66* describes the implementation of time-dependent absorption in the IRTM, and Annex E discusses absorption mechanisms, and the model assumptions concerning absorption, notably the assumption that the absorption rate is the same in all regions of the respiratory tract (including lymph nodes), except ET₁, where it is assumed none occurs. It provides default absorption parameters for the three default Types (F, M, S), (Table 3.2) but does not deal with specific materials.

It is intended that in the revision of *ICRP Publication 30* information relating to absorption in the respiratory tract of each element having isotopes of radiological importance will be reviewed. Where possible, absorption rates for important compounds will be recommended, and other compounds will be assigned to the three absorption Types. In *ICRP Publication 68* (ICRP, 1994b), giving inhalation dose coefficients for workers, compounds for which clearance was previously given as Class D, W or Y in *ICRP Publication 30* (ICRP, 1979), were generally assigned to the new Types F, M or S respectively. It should be noted that the classification of compounds into Classes D, W and Y in *ICRP Publication 30* and carried over to *ICRP Publication 68* is based on reviews typically carried out 20-30 years ago. For many elements they go back to the TGLD (1966). In some cases they are based on experimental data, in others chemical analogy.

ICRP Publication 71 (ICRP, 1996), which is currently in press, provides inhalation dose coefficients for members of the public for selected radioisotopes of 31 elements, including the 7 listed here. For each element a review of the literature was conducted to collect information on the biokinetics of compounds of that element, with emphasis on those to which members of the public might be exposed following environmental release. It is thus directly relevant to this question and up-to-date. As convener of the inhalation sub-group of the ICRP Task Group on Internal Dosimetry, I was involved in drafting much of the text of *ICRP Publication 71*, including the sections on several of the elements. I took the lead on drafting the sections on cesium and iodine, and also plutonium (shared with J.C.

Moody and R.A. Guilmette). Scientists in my Department at NRPB took the lead on strontium (M.J. Youngman), ruthenium (S.L. Prosser) and cerium (J.W. Marsh). The lead on the seventh element, tellurium, was taken by Dr. J. Piechowski. (All these sections were subsequently reviewed by other members of the sub-group, and the Task Group.)

In *ICRP Publication 71*, guidance is given for each element on which of the three default absorption Types should be chosen for environmental exposure, according to the information available. For environmental exposure, the physico-chemical form of the radionuclides inhaled is generally less well defined than in the case of workplace exposure, and there have been relatively few studies of respiratory tract clearance of radionuclides administered in forms likely to be representative of environmental exposure. Thus it is not feasible to specify absorption parameters for all the chemical forms likely to be encountered in the environment. Often the radioactive element will be present as a minor constituent of the inhaled particles. Absorption of the radionuclide to body fluids may well then be controlled by dissolution of the particle matrix, rather than by the elemental form of the radionuclide, and so may be different from that generally associated with simple compounds of the element. In particular, this may give rise to Type M or S forms of radionuclides for which most common simple compounds are Type F. Such particle matrices might be soil minerals, particularly clays; condensates or ash formed in fires or high temperature industrial processes; or mixtures of stable and radioactive elements. Following an accidental release from a nuclear reactor, fission and activation products may be present in fragments of irradiated fuel, of which the matrix is mainly uranium oxide (Devell, 1988; Toivonen, et al., 1992). When elements such as cesium, which are generally very readily absorbed from the lungs to body fluids, are incorporated into fused clay particles, which are highly insoluble, only a small fraction may be absorbed rapidly. The rest is retained within the particles and thus absorbed slowly, at a rate of the order of 0.001 d⁻¹ (Snipes et al., 1983). In vitro dissolution studies of coal fly ash have shown that lead, polonium and radium are similarly retained in the particles (Kalkwarf et al., 1984). Fission products could also be present in such insoluble matrices in nuclear weapons fallout (Norman and Winchell, 1970). Conversely, in some circumstances, the absorption rate of a relatively insoluble compound can be faster when it is present as a minor component of a more soluble matrix. In particular, absorption of plutonium is much more rapid from oxides in which it is mixed with metals such as sodium or magnesium, than from pure PuO₂ (Stather et al., 1979; Métivier et al., 1980).

ICRP Publication 71 includes sections on each of the elements covered, giving respiratory tract biokinetic data and inhalation dose coefficients. A brief review is given of available information on the absorption from the respiratory tract to body fluids following deposition of various compounds of the element. Previous ICRP guidance, generally from the report of the Task Group on Lung Dynamics (TGLD, 1966), and *ICRP Publication 30* is outlined. Relevant information for the elements listed in Question 7 is given below, based on an advanced draft of *ICRP Publication 71*.

ICRP Publication 66 does not give criteria for assigning compounds to absorption Types on the basis of experimental results, since it was considered that where such information is available it should be used to determine values of the absorption parameters. Since that approach could not be applied in *ICRP Publication 71*, such criteria were developed. (They are described in detail in Annex D of *ICRP Publication 71*). So that they can be applied uniformly to different mammalian species and different initial patterns of deposition throughout the respiratory tract, as well as to *in vitro* studies, they are defined in terms of the fraction of the deposit absorbed into body fluids in a given time, from a material with a specified constant rate of absorption.

In setting the criterion for Type F, it was considered that there should be no 'significant' component of 'long-term' lung retention: more than about 90% of the deposit in the alveolar region in man would be absorbed into body fluids within a month. Quantitatively, 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), deposited in the respiratory tract under exactly the same conditions.

For Type S, it was considered that in man there should be 'significant' lung retention for 'years' after intake: less than about 10% of the deposit in the alveolar region would be absorbed into body fluids within six months. Quantitatively, 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), deposited in the respiratory tract under exactly the same conditions.

In the reviews of each element the wording "... consistent with assignment to Type" indicates that there was sufficient information to apply the criteria quantitatively. For studies where the results pointed to a particular Type, but it was not possible to apply the criteria, a statement is made to the effect that "... results indicate Type F (M or S) behavior ...".

In general, for important radionuclides, inhalation dose coefficients for Type M materials are intermediate between those for Type F and Type S. Therefore if information is lacking, selection of Type M is least likely to lead to a large overestimate or underestimate of committed effective dose. It is therefore recommended in *ICRP Publication 71* that for most elements, in the absence of more specific information on absorption characteristics, Type M behavior is assumed as a default for environmental exposure of members of the public to radioisotopes in particulate form. Exceptions are made in those cases (such as cesium, iodine and thorium) where there are experimental data that indicate that many of the principal forms of the element likely to be encountered exhibit behavior characteristic of Type F or Type S. A summary table gives the values of fractional GI tract absorption, f_1 , adopted for each absorption Type. Table 4.1 gives the values for adults and 5-y-old children. In most cases (except alkaline earths) values for 5-y-olds are the same as for adults. The values for inhaled compounds may well be different from (normally lower than) those chosen for ingested compounds. In the environment inhaled radionuclides may well be associated with inorganic matrices that are less soluble in body fluids than simple compounds, while ingested radionuclides may well be biologically incorporated, making them more readily absorbed.

4.2 Gases and Vapors

For radionuclides inhaled in particulate form it is assumed that entry and regional deposition in the respiratory tract are governed only by the size distribution of the aerosol particles (and hygroscopic growth). The situation is different for gases and vapors, for which respiratory tract deposition is material-specific. 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 vapor that is deposited in each region thus depends on its solubility and reactivity. Generally, however, the regional deposition of a gas or vapor cannot be predicted on a mechanistic basis, from knowledge of its physical and chemical properties, but has to be obtained from an *in vivo* experimental study.

As a general default approach the *ICRP Publication 66* model assigns gases and vapors to three classes, on the basis of the initial pattern of respiratory tract deposition:

- Class SR-0 Insoluble and non-reactive: negligible deposition in the respiratory tract.
- Class SR-1 Soluble or reactive: deposition may occur throughout the respiratory tract.
- Class SR-2 Highly soluble or reactive: total deposition in the extrathoracic airways (ET₂).

Subsequent retention in the respiratory tract and absorption to body fluids are determined by the chemical properties of the specific gas or vapor.

In *ICRP Publication 71*, guidance is given on the deposition and clearance of potentially important gas and vapor forms of radionuclides. Defaults are recommended for the SR-Class (which determines deposition), and corresponding absorption Type (which determines clearance) to be used for gases and vapors in the absence of further information: more specific guidance is given in summary tables.

Class SR-0. The elements considered do not include any of the noble gases, and no compounds of the elements covered are assigned to SR-0.

Class SR-1. Soluble or reactive gases and vapors may be deposited throughout the respiratory tract. They may irradiate all airways, but generally exposure is dominated by activity absorbed into body fluids. They require individual evaluation, and some guidance is given in *ICRP Publication 66*. It is recommended (*ICRP Publication 66*, Paragraph 221) that in the absence of information 100% deposition is assumed, with the following distribution: 10% ET₁, 20% ET₂, 10% BB, 20% bb and 40% AI. It is recommended in *ICRP Publication 68*, Paragraph A2 that if total deposition is known to be different from 100%, then regional deposits should, by default, be distributed in the same proportions. These recommendations are followed in *ICRP Publication 71*. The key parameter is the total fraction of inhaled material deposited in region ET₂ and the lungs, because it is assumed that there is no absorption to body fluids from ET₁. Regional deposition is of secondary importance, as to some extent is the rate of absorption to body fluids, unless this is low compared to the rate of nuclear transformation.

Class SR-2. In *ICRP Publication 71* compounds are assigned to Class SR-2 on the basis of 100% deposition, and instantaneous absorption to body fluids.

4.3 Strontium

The ICRP Task Group on Lung Dynamics (TGLD, 1966) assigned strontium phosphate to inhalation Class D and the other compounds of strontium listed to Class W. *ICRP Publication 30*, Part 1 (ICRP, 1979) assigned 'soluble' compounds of strontium to Class D, referring to a study by Morrow et al. (1968) which showed that SrCl₂ is rapidly cleared from lungs. Strontium titanate (SrTiO₃) was shown to be much more tenaciously retained (Fish et al., 1967) and was assigned to Class Y.

Absorption Types

Measurements following accidental inhalations by humans of strontium carbonate (SrCO₃) and strontium chloride (SrCl₂) indicate Type F behavior (Rundo and Williams, 1961; Petkau and Pleskach, 1971). Animal experiments have shown that with simple ionic compounds (chloride and sulphate) most of the strontium is rapidly cleared, consistent with assignment to Type F (Bair, 1961; Boecker et al., 1967; Cuddihy and Ozog, 1973; Naményi et al., 1986).

Environmental strontium could well be inhaled in particles in which it is a minor constituent. For example, after an accidental release it could be present in fragments of irradiated fuel. Measurements following the accidental inhalation of a mixture of fresh fission products, indicate Type M behavior of the strontium present (Johnson et al., 1983). Results of an in vitro study on airborne fission products from the Three Mile Island reactor accident are consistent with assignment to Type F (Kanapilly et al., 1980). An in vitro study on aerosols generated during transfer, cutting, storage and shipment of nuclear reactor fuel is consistent with assignment of the strontium present to Type M (Dua et al., 1987).

It has also been demonstrated that when strontium is incorporated into a matrix such as fused aluminosilicate particles (Sr-FAP), only a small fraction may be rapidly absorbed, while the remainder is retained within the particles and absorbed slowly. Estimates of the rate of dissolution of Sr-FAP were in the range 0.0005 – 0.002 d⁻¹ (Snipes et al., 1972; Kanapilly and Goh, 1973; Bailey et al., 1985a; 1985b), and indicate Type S behavior.

4.4 Iodine

The TGLD (1966) assigned halides (including iodides) of elements in Periodic Table Group 1a (alkali metals) and Group 7a (halogens) to inhalation Class D, and most others to Class W. In *ICRP Publication 30*, Part 1 (ICRP, 1979),

all compounds of iodine were assigned to Class D. This classification was supported by the results of experiments in which elemental iodine vapor was inhaled by mice, sheep, rats and dogs, and ^{131}I -labelled silver iodide was inhaled by mice and sheep (Willard and Bair, 1961; Bair et al., 1963). Silver iodide had been studied because it is one of the most insoluble iodine compounds in water, but nevertheless the ^{131}I was rapidly absorbed from the lungs.

Absorption Types

Gases and vapors

Elemental iodine. Detailed studies have been conducted in human volunteers of the deposition and subsequent biokinetics of iodine inhaled as elemental iodine (Black and Hounam, 1968; Morgan et al., 1968). Almost all the inhaled activity was retained. It was inferred that it mainly deposited in the conducting airways, most parts of which cleared with a half-time of the order of 10 minutes. This confirmed the rapid absorption seen previously in animal experiments. The measurements also showed, however, that much of the activity was swallowed and subsequently absorbed from the GI tract. Elemental iodine is therefore assigned to Class SR-1 (100% deposition), with Type F clearance (Table 4.2).

Methyl iodide. Detailed studies have been conducted in human volunteers of the deposition and subsequent biokinetics of iodine inhaled as CH_3I (Morgan et al., 1967a; 1967b; Morgan and Morgan, 1967). The amount retained varied from 50 to 90% (average 70%), increasing with decreasing number of breaths per minute. It was inferred that most of it deposited in the alveoli. Absorption to blood of the deposited activity was very rapid (estimated half-time about 5 seconds). Subsequent biokinetics were very similar to those of injected iodide, suggesting that the CH_3I is rapidly metabolized. Methyl iodide is therefore assigned to Class SR-1 (70% deposition), with Type V clearance.

Particulate aerosols

As with silver iodide (see above), iodine inhaled as sodium iodide is also rapidly absorbed into blood. A half-time of the order of 10 minutes was inferred from studies on monkeys (Thiéblemont et al., 1965; Perraud et al., 1967), and a similar result was obtained in isolated perfused rabbit lung (Dawson et al., 1985). Rapid uptake of iodine, consistent with assignment to Type F, was also observed following inhalation of ^{131}I associated with cesium chloride vector aerosols by rats (Thomas et al., 1970), and dogs (McClellan and Rupprecht, 1968).

4.5 Cesium

The TGLD (1966) assigned all compounds of cesium to inhalation Class D. This was endorsed in *ICRP Publication 30*, Part 1 (ICRP, 1979), which referred to supporting experimental evidence relating to inhalation of chloride, nitrate and sulphate (Lie, 1964; Miller, 1964; Boecker, 1969).

Absorption Types

The results of a human study following accidental inhalation of cesium sulphate (Miller, 1964) indicate Type F behavior. Animal experiments have also shown that simple ionic compounds (chloride and nitrate) of cesium are rapidly and completely absorbed from the respiratory tract, consistent with assignment to Type F (Lie, 1964; Star, 1965; Boecker, 1969; Cuddihy and Ozog, 1973).

Studies of cesium associated with irradiated fuel fragments, including particles released from the Chernobyl accident, indicate that much of the cesium is rapidly absorbed (within days), but a fraction may be retained with the particle matrix and absorbed over a period of months. The results of a human study following accidental inhalation of irradiated uranium indicate Type F behavior of the cesium present (Rundo, 1965). Following administration to rats of a suspension of residues from a reactor fuel cooling pond, the overall behavior of the cesium was consistent with assignment to Type F (Stradling et al., 1989). In two in vitro studies, however, the behavior of the cesium present in irradiated fuel fragments was consistent with assignment to Type M (Dua et al., 1987; Cuddihy et al., 1989).

It has also been demonstrated in animal studies that when cesium is incorporated into FAP only a small fraction is rapidly absorbed from the lungs. The rest is retained within the particles and absorbed slowly, at rates of the order of 0.001 d^{-1} : in some experiments consistent with assignment to Type M and in others to Type S (Boecker et al., 1974; Snipes et al., 1983; Snipes and McClellan, 1986).

4.6 Plutonium

The TGLD (1966) assigned carbides, oxides and hydroxides of plutonium (Pu), to inhalation Class Y, and nitrates to Class W. By default other unspecified compounds were assigned to Class D.

ICRP Publication 19 (ICRP, 1972) provided a comprehensive review of the literature up to 1971 relating to the biokinetics of plutonium, with entry by inhalation

considered in detail. For PuO_2 there was a substantial amount of data from dogs, as well as from rodents. Data were also available for a variety of other compounds, including nitrate, citrate, chloride and ammonium plutonium pentacarbonate, mainly for rats. It was recommended that in view of the ready hydrolysis of most soluble compounds at physiological pH, none should be considered Class D, except those stable enough to remain soluble under physiological conditions, e.g., the DTPA chelate.

ICRP Publication 30, Part 1 (ICRP, 1979), taking *ICRP Publication 19* and later reviews into account, assigned PuO_2 to Class Y, and all other commonly occurring compounds of plutonium to Class W. Chelated forms of plutonium were specifically excluded from the report, because their behavior after uptake to blood is different from that described in the biokinetic model that applies to intakes from all other forms.

ICRP Publication 48 (ICRP, 1986) provided a further review of the literature relating to the biokinetics of plutonium (and related elements). This complemented *ICRP Publication 19* by placing emphasis on the more recent data available, especially that derived from human tissues taken at autopsy. Generally the assignment of the oxide to Class Y and other commonly occurring compounds to Class W was supported, and this was retained in *ICRP Publication 30*, Part 4 (ICRP, 1988). The desirability of using material-specific information was, however, strongly re-emphasized.

Absorption Types

(a) Simple compounds

Of the variety of more soluble compounds, the nitrate and tributyl-phosphate (Pu-TBP) complexes are of particular importance in industrial processes. Results of biokinetic studies of plutonium nitrate following intratracheal instillation into rats, and inhalation by rats, dogs or monkeys are consistent with assignment to Type M (Stather and Howden, 1975; Brooks et al., 1981; Dagle et al., 1983; Stradling et al., 1987; Moody et al., 1994). Measurements following inhalation of Pu-TBP by rats and baboons are also consistent with assignment to Type M (Stradling et al., 1985; Métivier et al., 1989). The importance of the mass of plutonium deposited in the lung has been recognized for both materials, as absorption can be inhibited by relatively high mass loadings, possibly because of colloid formation (Nolibé et al., 1989). Such mass effects are not considered

to be of concern for exposures to environmental levels of plutonium.

The various oxide forms of plutonium have been the most thoroughly studied of actinide aerosols. Generally, two distinct phases of absorption to blood from the respiratory tract are exhibited. A small fraction, typically less than 1%, is absorbed within about a day, with the remainder being cleared from the lung with half-times of the order of years (absorption rate of the order of 10^{-4} d^{-1}) (ICRP, 1972; 1986). Both the fraction rapidly absorbed and the long-term retention half-time can be influenced by the method of formation of the material and its history. Plutonium-239¹ dioxide, formed by complete oxidation of the metal or a salt at about 1000°C (high-fired), for example, has repeatedly demonstrated the very low absorption generally associated with PuO_2 (Type S) (Bair et al., 1980; Morgan et al., 1988a). Material formed at lower temperatures was more readily absorbed (Type M), reflecting incomplete oxidation of the plutonium (Mewhinney et al., 1976).

Studies in several animal species have demonstrated Type S behavior of $^{239}\text{PuO}_2$. Measurements following inhalation of high fired polydisperse $^{239}\text{PuO}_2$ by baboons, rhesus monkeys and dogs indicate Type S behavior (Bair et al., 1980; LaBauve et al., 1980), as do data from dogs that inhaled monodisperse particles (0.72, 1.4, and 2.8 mm AMAD) (Guilmette et al., 1984; 1987b). These data are in general agreement with observed lung retention patterns of $^{239}\text{PuO}_2$ in exposed workers (Ramsden, 1976; 1984; Ramsden et al., 1978; Spitz and Robinson, 1981; Carbaugh et al., 1991; Foster, 1991).

Bioassay data from accidentally exposed workers as well as data from experimental studies have shown a much greater rate of absorption of plutonium to blood following inhalation of $^{238}\text{PuO}_2$ compared with that of $^{239}\text{PuO}_2$. This has been attributed to radiolytic fragmentation of the particles due to the high specific activity of ^{238}Pu (Fleisher and Raabe, 1977; Diel and Mewhinney, 1981). Thus, the lung retention and absorption to blood of ^{238}Pu in dogs inhaling the dioxide form (Mewhinney and Diel, 1983; Park et al., 1986a,b) were consistent with Type M. Similarly, workers inhaling purported oxide or "ceramic" forms of ^{238}Pu showed urinary excretion patterns leading to inferred lung retention patterns also indicative of Type M (Guilmette et al., 1994; Hickman et al., 1995). On the other hand, some cases of exposure to ^{238}Pu oxide have been more consistent with data from workers exposed to $^{239}\text{PuO}_2$, indicating Type S behavior (Fleming and Hall, 1978; Newton et al., 1983).

¹ Where reference is made to ^{239}Pu , this may well include ^{240}Pu .

Plutonium can have different lung clearance characteristics when inhaled as a mixed metal oxide. A higher rate of absorption of plutonium to blood was observed in rats exposed to oxides containing plutonium mixed with sodium, potassium, calcium or magnesium (Stather et al., 1979; Métivier et al., 1980). The extent of increased dissolution/clearance depended on the metal and the relative proportions of plutonium to metal. In general these data support the assignment of Type M.

In contrast, plutonium inhaled as a mixed plutonium-uranium oxide may be absorbed no more readily than from high-fired pure PuO_2 . Results of experiments in which rats, dogs and monkeys inhaled aerosols consisting of mixtures of UO_2 and PuO_2 particles derived from feedstock for a mixed oxide reactor fuel were consistent with assignment of the plutonium present to Type S, even though the PuO_2 had been heat-treated at different temperatures ranging from 750–1750°C (Stanley et al., 1980a; 1980b; 1982). Similarly, inhalation of uranium–plutonium mixed oxides in solid solution by monkeys, dogs and rats (Mewhinney and Eidson, 1983) or by baboons and rats (Lataillade et al., 1995) resulted in lung clearance patterns indistinguishable from those for PuO_2 , therefore also indicating Type S behavior.

(b) Environmental forms

There have been a number of studies of plutonium released into the environment. Plutonium discharged to sea can become attached to sediment, which has potential for resuspension in air. Following intra-tracheal instillation of ^{239}Pu -labelled sediment into rats and hamsters, the behavior of the plutonium was consistent with assignment to Type M (Stather et al., 1978; Morgan et al., 1988b, 1990).

Numerous measurements have been made of the concentration of ^{239}Pu , resulting from the atmospheric testing of nuclear weapons, in tissues (notably lung, liver, skeleton and tracheo-bronchial lymph nodes) taken at autopsy from non-occupationally exposed people. Comparisons with levels predicted from measured air concentrations using the then current ICRP models were broadly consistent with Class Y (Bennett, 1976; McInroy et al., 1981; ICRP, 1986).

Soils and dusts contaminated in the 1960s with plutonium from nuclear weapons tests, and from a weapons accident (Iranzo et al., 1987), have recently been administered by intra-tracheal instillation and/or inhalation into rats (Stradling et al., 1992; 1993). Rates of absorption of plutonium from the dusts varied considerably. Of three

different dust samples contaminated with plutonium and americium obtained from a nuclear weapons testing site in South Australia, behavior of the plutonium in two was consistent with assignment to Type S, and that in the third to Type M (Stradling et al., 1992). It is of interest that in these studies the plutonium remained mainly in insoluble forms even after two or three decades of environmental exposure. Mewhinney et al. (1987) found with in vitro dissolution tests, that alternate wet-dry cycling, simulating that occurring under environmental conditions such as intermittent rainfall in an otherwise arid climate, led to much faster dissolution than during continuous immersion in the same solvents.

4.7 Ruthenium

The TGLD (1966) assigned oxides and hydroxides of ruthenium to inhalation Class Y, halides to Class W and all other compounds to Class D. This was endorsed by ICRP in Publication 30, Part 2 (ICRP, 1980), in which the assignment of ruthenium dioxide to Class Y was supported by reference to experiments with dogs (Stuart, 1970).

Absorption Types

Gases and vapors

Following an accidental human inhalation of ruthenium tetroxide vapor, it was concluded that deposition occurred only in the extrathoracic airways, where the compound was subsequently retained (Webber and Harvey, 1975). Experiments on rats and dogs have confirmed that inhalation of ruthenium tetroxide vapor results in complete deposition in the extrathoracic airways (Runkle and Snipes, 1979; Runkle, et al., 1980; Snipes, 1981). Based on these studies ruthenium tetroxide vapor is assigned to Class SR-1 (100% deposition with default regional distribution), with Type F clearance.

Particulate aerosols

The results of a human investigation following inhalation of an unknown ruthenium compound indicate Type M behavior (Pusch, 1968). Animal investigations using ruthenium citrate and dioxide, indicate that these compounds also exhibit Type M behavior (Bair, et al., 1961; Boecker and Harris, 1969; Snipes, 1979). However, data from other animal experiments indicate Type M behavior for oxalate, with Type S for dioxide (Newton, et al., 1976; Newton and Latven, 1971).

The results of two human studies following accidental inhalation of ruthenium dioxide (Hesp and Coote, 1970; Howells, et al., 1977) indicate Type S behavior. A study on dogs (Stuart, 1970) using ruthenium dioxide also indicates Type S behavior.

Environmental ruthenium could well be inhaled in particles in which it is a minor constituent. For example, following an accidental release it could be present in fragments of irradiated fuel, where the matrix would be predominantly uranium oxide. Results of a study in which irradiated UO₂ powder was administered to rats by intratracheal instillation were consistent with assignment of the ruthenium present to Type M (Lang, et al., 1994). Studies of the in vitro dissolution of particles released from the Chernobyl accident (Cuddihy et al., 1989) were consistent with assignment of the ruthenium present to Type M.

4.8 Cerium

The TGLD (1966) assigned oxides, hydroxides and fluorides of cerium to inhalation Class Y and all other compounds to Class W. This classification was endorsed in *ICRP Publication 30*, Part 1 (ICRP, 1979) which referred to further support from animal experiments. The physical, chemical and biological properties of radiocerium relevant to radiation protection guidelines were reviewed in NCRP Report 60 (NCRP, 1978).

Absorption Types

There have been few reported studies of the biokinetics of cerium involving human inhalation. In one case of an accidental human exposure to a ¹⁴⁴Ce-Pr contaminated atmosphere (Glenn et al., 1979), the results of lung clearance are consistent with assignment to Type M. Results of a retrospective study to evaluate lung retention of cerium particles in subjects (Pairon et al., 1994), indicate Type M or S behavior.

Several animal experiments have been performed with soluble cerium as ¹⁴⁴CeCl₃. Experiments on dogs (Cuddihy et al., 1975) gave results consistent with assignment to Type M. Other experiments on Chinese hamsters (Sturbaum et al., 1970) indicate Type M behavior. Results of a study in which CeCl₃ was administered to rats by intratracheal instillation (Cember and Watson, 1958) were of insufficient duration (2 months) to distinguish between Types M and S. Cember and Stemmer (1964) postulated that the slow clearance of ¹⁴⁴CeCl₃ may be due to cerium being in a bound state, and also showed that protein is capable of binding relatively large quantities of cerium. Kanapilly et

al. (1973), however, remarked that the retention of ¹⁴⁴CeCl₃ in the lung may be attributed to the hydrolysis of ¹⁴⁴Ce. Experiments designed to give a soluble form of ¹⁴⁴CeCl₃, by using CsCl instead of CeCl₃ as the vector aerosol, gave, in this exceptional case, results consistent with assignment to Type F (Boecker and Cuddihy, 1974; Cuddihy et al., 1975).

Results of experiments on rats exposed by inhalation to cerium as ¹⁴⁴CeOH, are consistent with assignment to Type M (Thomas et al., 1972).

Studies have been conducted of the behavior of cerium following inhalation of ¹⁴⁴CeO₂ by mice (Lundgren et al., 1974, 1980a; 1980b), Syrian hamsters (Thomas and McClellan, 1972) and rats (Lundgren et al., 1992; Yan et al., 1988). Some experiments gave results consistent with assignment to Type M, and others to Type S. According to Lundgren et al. (1992), lung clearance decreases as the initial lung deposit increases.

A study of cerium associated with irradiated fuel fragments indicated that ¹⁴⁴Ce is moderately transportable from lung to blood, consistent with assignment to Type M (Stradling et al., 1989). Results of a study in which irradiated UO₂ powder was administered to rats by intratracheal instillation (Lang, et al., 1994) are consistent with assignment of the cerium present to Type M or S but were of insufficient duration (3 months) to distinguish between the two. Studies of the in vitro dissolution of particles released from the Chernobyl accident (Cuddihy et al., 1989) were consistent with assignment of the cerium present to Type M.

It has also been demonstrated in experiments on dogs (Shyr et al., 1991; McClellan et al., 1970) that when cerium is incorporated into fused aluminosilicate particles (Ce-FAP), the cerium is avidly retained in the lung, indicating Type S behavior. McClellan et al. (1970) stated that the retention in the lung of Ce-FAP is primarily influenced by the characteristics of the FAP vector aerosol rather than that of cerium.

4.9 Tellurium

The TGLD (1966) assigned oxides, hydroxides and nitrates of tellurium to inhalation Class W and all other compounds to Class D. In the absence of any relevant experimental data this classification was adopted in *ICRP Publication 30*, Part 1 (ICRP, 1979). Although many studies have been reported on the toxicity of tellurium, and on its biokinetics following ingestion and injection (Kron et al., 1991; ICRP, 1993), no experimental studies of the behavior of tellurium following

deposition in the respiratory tract were identified in the literature, for *ICRP Publication 71*.

Absorption Types

In one study following accidental inhalation of tellurium in the form of hexafluoride gas and possibly also tellurium esters (Blackadder and Manderson, 1975), absorption to blood was described as rapid, but insufficient information was available to estimate the fraction deposited, or the rate of absorption in *ICRP Publication 71*. Tellurium in gas and vapor forms are assigned to Class SR-1 (100% deposition with default regional distribution) with Type F clearance.

4.10 Application to Question 7

There are three aspects to this:

- (i) Deposition in each region (ET₁, ET₂, BB, bb, AI) as % total initial deposition in the respiratory tract. Deposition in ET₁ is particularly important, as it is assumed in the IRTM that there is no absorption from that region nor clearance from it to the GI tract.
- (ii) Absorption from the respiratory tract. Note that this is assumed to compete with particle transport, so that the amount absorbed depends on both processes.
- (iii) Absorption from the GI tract of material cleared to it from the respiratory tract (Table 4.1).

Initial deposition in regions

The initial deposits and their uncertainties are based on the results of Questions 3 and 4. However, since the uncertainties on deposition will be combined with uncertainties in retention, I have estimated a narrower range, nominally x₁₅ and x₈₅. The following steps refer to line numbers in Table 4.3.

1. Take x₁₅, x₅₀ and x₈₅ for Lung deposition as % total respiratory tract from Table 3.15.
2. Calculate x₁₅, x₅₀ and x₈₅ for ET deposition as % total respiratory tract from Line 1 (100-Lung).
3. Express x₁₅ and x₈₅ as ratio to x₅₀.
4. Estimate x₁₅ and x₈₅ for ET₁/ET, given that x₅₀ = 0.4 (Table 2.9).
5. Express x₁₅ and x₈₅ as ratio to x₅₀.
6. Calculate x₁₅ and x₈₅ for ET₁ deposition/total respiratory tract as ratio to x₅₀ from Lines 3 and 5. (r.m.s. of corresponding values).

7. Calculate x₁₅, x₅₀ and x₈₅ for ET₁ deposition as % total respiratory tract from line 6 (x 25%).
8. Estimate x₁₅, x₅₀ and x₈₅ for ET₂ deposition as % total respiratory tract, by consideration of Line 2 (total ET) and Line 7 (ET₁/respiratory tract).
9. State x₁₅, x₅₀ and x₈₅ for TB deposition as % total respiratory tract from Table 3.15.
10. State x₁₅, x₅₀ and x₈₅ for AI deposition as % total respiratory tract from Table 3.15.

From these, regional deposition fractions leading to low absorption (relatively high deposition in ET, and high absorption (relatively high deposition in AI) were estimated (Table 4.4).

For iodine and tellurium, consideration was also given to inhalation and deposition of vapors.

Absorption from the respiratory tract

For each element, estimates were made of low, median and high rates, based largely on the accounts in *ICRP Publication 71*. These were usually default absorption Types, but in some cases intermediate values were chosen.

It was assumed here, as in the IRTM, that the rate of absorption to blood is the same in all parts of the respiratory tract except ET₁, where it is assumed none occurs. It is recognized in *ICRP Publication 66* that this is probably an over-simplification. In particular, it is likely that the absorption rate in the AI region, where the air-blood barrier is thinnest, is higher than in the conducting airways. This is not certain, however, since crossing a single cell membrane could be the rate-determining step. More importantly however, there is no basis for deciding what the relative rates should be in the different regions. The effect of setting different absorption rates in different regions on the amounts absorbed at the specified times was examined (doubling the rate in AI, and reducing that in BB and bb to zero). However, the results were found to lie within the existing 90% confidence intervals.

Similarly, it was assumed here, as in the IRTM, that the rate of absorption to blood is the same in children as in adults, although there is some evidence from animal experiments with BeF₄ and Co₃O₄ that absorption is faster in young animals (Moskalev et al., 1988; Collier et al., 1991). However there is no recognized basis for determining rates at different ages, and it is again likely that the difference will be within the existing confidence interval.

Absorption from the GI tract

For each element, estimates were made of low, median and high f_1 values, based largely on those adopted in *ICRP Publication 71* for the default inhalation Types (Table 4.1). It is known that for Type S compounds the amount eventually reaching blood is not sensitive to the choice of f_1 value, if it less than 0.01. The early absorption could however change.

Calculations

For children only deposition was changed from x_{50} values.

LUDEP was used to evaluate the amount absorbed at each of the set times. This involved setting an acute intake of 100 Bq of a long-lived radionuclide (^{40}K), to avoid radioactive decay, and setting up a biokinetic model so that all the absorbed activity went to one organ with an extremely long retention time. LUDEP outputs the activity in each organ at the specified time after intake. The activity in that organ then gave the amount absorbed.

Strontium (Table 4.5)

Deposition: only particulates considered, take D_{15} , D_{50} , D_{85} and D_c from Table 4.4

Absorption: Many compounds Type F, some Type M. *ICRP Publication 71* takes Type M as default. Take Type M for low absorption, and Type F for high. For x_{50} take intermediate values: $f_r = 0.5$ (0.1 for Type M, 1.0 for Type F), with $s_s = 0.005 \text{ d}^{-1}$, as for Type M.

f_1 : 0.1, 0.3 for Type M, F, respectively (Table 4.1); take 0.2 as intermediate for x_{50} .

Iodine (Table 4.6)

Deposition: Take particulate D_{15} for low absorption; D_{IV} (iodine vapor) for high, and 50% D_{IV} , 50% D_{50} for x_{50} , from Table 4.4.

Absorption: All compounds Type F. *ICRP Publication 71* takes Type F as default. Take Type F for all.

f_1 : Table 4.1 has 1.0 for F. Take 0.9, 0.95 and 1.0 for low, x_{50} , high respectively (Section 5.2).

Cesium (Table 4.7)

Deposition: only particulates considered, take D_{15} , D_{50} , D_{85} and D_c from Table 4.4.

Absorption: Most compounds Type F, some evidence for Type M in fuel. *ICRP Publication 71* takes Type F as default. Take Type M for low absorption, and Type F for high. For x_{50} take intermediate values, but close to Type F: $f_r = 0.9$ (0.1 for Type M, 1.0 for Type F), with $s_s = 0.005 \text{ d}^{-1}$, as for Type M.

f_1 : Table 4.1 has 1.0 for F. Take 0.8, 0.9 and 1.0 for low, x_{50} , high respectively (Section 5.3).

Plutonium (Table 4.8)

Deposition: only particulates considered, take D_{15} , D_{50} , D_{85} and D_c from Table 4.4.

Absorption: Many compounds Type M or Type S. *ICRP Publication 71* takes Type M as default. Take Type S for low absorption, and Type M for high. For x_{50} take intermediate values: $f_r = 0.01$ (0.1 for Type M, 0.001 for Type S), with $s_s = 0.001 \text{ d}^{-1}$ (0.005 for Type M, 0.0001 for Type S).

f_1 : 5×10^{-4} Type M, 10^{-5} for Type S, respectively (Table 4.1); take 10^{-4} as intermediate for x_{50} .

Ruthenium (Table 4.9)

Deposition: RuO_4 vapor unlikely far from source. Only particulates considered, take D_{15} , D_{50} , D_{85} and D_c from Table 4.4.

Absorption: Many compounds Type M, some Type S. Type F unlikely, but possible. *ICRP Publication 71* takes Type M as default. Take Type S for low absorption, and Type M for x_{50} . For high take intermediate between Type M and F values: $f_r = 0.5$ (0.1 for Type M, 1 for Type F), with $s_s = 0.005 \text{ d}^{-1}$, as for Type M (as selected for strontium x_{50}).

f_1 : 0.01, 0.05, 0.05 for Type S, M, F respectively (Table 4.1).

Cerium (Table 4.10)

Deposition: only particulates considered, take D_{15} , D_{50} , D_{85} and D_c from Table 4.4.

Absorption: Many compounds Type M or Type S, with possibility of Type F in some circumstances. *ICRP Publication 71* takes Type M as default. Take Type S for low absorption. For x_{50} , take values intermediate between Type M and S: $f_r = 0.05$ (0.1 for Type M, 0.001 for Type S), with $s_s = 0.005 \text{ d}^{-1}$ (as for Type M); for x_{95} take values intermediate between Type M and F but nearer to Type M: $f_r = 0.2$ (0.1 for Type M, 1.0 for Type F), with $s_s = 0.005 \text{ d}^{-1}$ (as for Type M).

$f_1: 5 \times 10^{-4}$ for all Types (Table 4.1).

Tellurium (Table 4.11)

Deposition: Take particulate D_{15} for low absorption, and D_{50} for x_{50} ; D_V (default vapor for high), and D_C for child, from Table 4.4.

Absorption: Of all 31 elements considered in *ICRP Publication 71* least is known about the behavior of tellurium after inhalation. No experiments were located in the literature, and only one report of measurements following inadvertent intake, and that to non-radioactive vapor forms. Chemical analogy hardly narrows the field. In the Periodic Table it lies between selenium, for which all important compounds are Type F, and polonium, for which studies show compounds with Type F, M, or S behavior. *ICRP Publication 71* takes Type M as default. Take Type S for low absorption, Type M for x_{50} , and Type F for high absorption.

$f_1: 0.01, 0.1, 0.3$ for Type S, M, F, respectively (Table 4.1).

5. Question 9. Absorption of Ingested Activity

Question 9. Absorption to blood as a fraction (f_1) of activity ingested (considering chemical forms most likely to be ingested after an accident)?

The response to this question was prepared by Dr. John D. Harrison of NRPB, who is recognized internationally as having extensive experience of this topic both as experimenter and reviewer.

5.1 Strontium (Sr)

Due to the presence of strontium isotopes in fall-out material and its long-term retention in bone as a calcium analogue, the metabolism of strontium has been the subject of a number of human volunteer studies. Similar absorption values were obtained from studies in which inorganic forms of radiostrontium were administered orally in solution

(Spencer et al., 1960; Suguri et al., 1963; Shimmins et al., 1967) and from experiments where known quantities of radiostrontium incorporated in food were ingested (Fujita et al., 1966; Carr, 1967). In each case, mean values were between 0.1 and 0.3, averaging about 0.2. LeRoy et al. (1966) measured the absorption of strontium from real and simulated fall-out and after administration of ^{85}Sr chloride. Ten volunteers ingested samples of local fallout, largely comprising silicaceous soil constituents (40 – 700 μm particles). The estimated absorption averaged 0.03 with a range of 0 – 0.09. For simulated fallout prepared as glass microspheres (30 – 40 μm), the estimated f_1 value was 0.16 (range 0.06 – 0.25), with a value of 0.17 (0.08 – 0.34) after administration as the chloride.

A number of factors have been found to increase absorption, including fasting and low dietary levels of calcium, magnesium and phosphorus; milk diets and vitamin D may also increase absorption. Spencer et al. (1972) showed that overnight fasting increased absorption from about 0.25 to 0.55. McAughay et al. (1994) also reported an f_1 value of 0.55 (0.38 – 0.72) for four volunteers after an overnight fast compared with 0.11 in a single volunteer ingesting strontium after breakfast. A decrease in calcium content of the diet from 30 – 40 to 0 – 10 $\text{mg d}^{-1} \text{kg}^{-1}$ increased strontium absorption from an average of 0.2, to 0.4 (Shimmins et al., 1967).

Results from animal studies are generally similar (Coughtrey and Thorne, 1983) to those from volunteer studies. Results for the absorption of strontium administered as the titanate (SrTiO_3) to rats show low levels of absorption of about 0.01 (McClellan and Bustad, 1964).

Results obtained by Widdowson et al. (1960) suggest that absorption of strontium in 7-d-old infants fed with cow's milk is greater than 73%. Bedford et al. (1960) reported that absorption in 5- to 15-y-old children was the same as in adults. Taylor et al. (1962a), obtained absorption values of 0.95 ± 0.004 (SE, $n = 31$) for 14 to 18-d-old rats and 0.74 ± 0.024 (SE, $n = 5$) for 22-d-old animals.

In *ICRP Publication 30* (1979), the recommended f_1 values for occupational exposure were 0.01 for SrTiO_3 and 0.3 for all other compounds. An f_1 value of 0.3 is assumed for strontium ingested in food by adults (ICRP, 1989, 1993). An f_1 value of 0.6, twice the adult value, is used for children in the first year of life, consistent with the NEA (1988) approach and available data. For ages 1 – 15 years an intermediate value of 0.4 is assumed.

For intakes after an accident, appropriate central values of f_1 are judged to be 0.2 for adults, 0.3 for children and 0.4 for infants. High confidence intervals on the central values of f_1 are judged to be 0.1 – 0.4 for adults, 0.1 – 0.5 for children and 0.15 – 0.75 for infants.

5.2 Iodine (I)

The absorption of iodide from the gastrointestinal tract of humans is virtually complete with reported values of 0.9 and greater (Riggs, 1952; Willard and Bair, 1961; Wayne et al., 1964; Underwood, 1971). Keating and Albert (1949) reported a rate of absorption of about 5 min^{-1} in fasted individuals, with complete absorption within 2 hours. The rate of absorption of iodide ingested with food was shown to be slower but was nevertheless virtually complete after about 3 hours. Iodide is absorbed in the stomach as well as the small intestine although the latter predominates. For other chemical forms, absorption may not be complete although there is little direct evidence. Studies with dogs have shown that free iodine and iodate are converted to iodide prior to absorption but this takes place quite rapidly (Cohn, 1932). Results obtained for iodine administered to humans as thyroxine suggested absorption of 0.80 – 0.85 (Wayne et al., 1964).

High values of >0.7 – 1 have been reported for absorption of iodine and iodide in goats and a number of studies using cattle, as summarized by Coughtrey et al. (1983).

ICRP (1979, 1989) have adopted an f_1 value of 1 for all ages. For intakes after an accident, the central value is judged to be 0.95 for all ages. A high confidence interval on the central value is judged to be 0.9 – 1 for all ages.

5.3 Cesium (Cs)

Human volunteer studies using ^{137}Cs in soluble inorganic form have shown virtually complete absorption (Rosoff et al., 1963; Rundo et al., 1963; Naversten and Liden, 1964; LeRoy et al., 1966). Thus, for example, Rundo et al. (1963) measured an average fractional absorption of 0.99 for 10 normal subjects following the ingestion of $^{137}\text{CsCl}$.

Studies have shown that ^{137}Cs incorporated into insoluble particles may be less available for absorption. Talbot (1991) measured absorption of ^{137}Cs from irradiated reactor fuel particles (2 – 10 μm) in adult rats to be less than 0.1. Le Roy et al. (1966) reported values of less than 0.1 for the uptake of ^{137}Cs from real and simulated fall-out in a large study on 102 volunteers. In contrast, Fujita et al. (1966) studied the absorption of ^{137}Cs from fall-out in five

volunteers and obtained results consistent with virtually complete absorption.

Measurements of uptake of ^{137}Cs from meat (venison, mutton, caribou) in human volunteers after contamination following the Chernobyl accident have given values in the range 0.6 – 0.99 (Henrichs et al., 1989; Talbot et al., 1993).

ICRP (1979, 1989) have adopted an f_1 value of 1 for all ages. For intakes after an accident, the central value of f_1 is judged to be 0.9 for all ages. High confidence intervals on the central value is judged to be 0.8 – 1 for all ages.

5.4 Plutonium (Pu)

Popplewell et al. (1994) measured the absorption of ^{244}Pu administered in citrate solution with a mid-day meal to three volunteers. The values obtained were in the range of 3×10^{-4} – 9×10^{-4} . Measurements on a further two volunteers are in progress. Hunt et al. (1986, 1990) have carried out two studies of the absorption of plutonium and americium by volunteers eating winkles collected on the Cumbrian coast near to the nuclear-fuel reprocessing plant at Sellafield. The overall f_1 value obtained for plutonium was 2×10^{-4} with a range of 2×10^{-5} – 5×10^{-4} . The absorption of fallout plutonium from reindeer meat was estimated by Mussalo-Rauhamaa et al. (1984). An f_1 of 8×10^{-4} was obtained by comparing the ratio of body content to dietary intake of $^{239/240}\text{Pu}$ in persons who had lived in Lapland or the urban areas of southern Finland. Large uncertainties were associated with this estimate of absorption.

Animal data on the absorption of plutonium in species including rodents, pigs, dogs and primates was extensively reviewed in *ICRP Publication 48* (ICRP, 1986) and by Harrison (1983, 1991). The chemical form ingested is an important factor affecting absorption. The lowest values obtained are for the oxide, ranging from about 2×10^{-4} in the rat (Sullivan, 1980) to about 3×10^{-8} in the pig (Smith, 1970). These large differences are probably a reflection of the solubility of the oxide preparation, which is affected by the temperature of production (Mewhinney et al., 1976), the proportion of small particles present (Stather et al., 1975) and the specific activity of the isotope (Fleischer and Raabe, 1977). Mixed plutonium-sodium oxides contain a higher proportion of very small particles (about 1 nm diameter) than the pure oxides (Stather et al., 1975) and suspensions of ^{238}Pu oxide are more prone than those of ^{239}Pu oxide (6.27×10^8 and $2.25 \times 10^6 \text{ kBq g}^{-1}$, respectively) to radiolytic breakdown to small particles (Fleischer and Raabe, 1977). Comparisons of the behavior of inhaled

plutonium oxide and mixed uranium/plutonium oxides in rats and baboons showed that although solubility in the lung was low in each case, transfer of plutonium to liver and bone was about two to three times greater for the mixed oxide (Lataillade et al., 1995).

The range in values of uptake for plutonium administered to animals as the nitrate, chloride or bicarbonate is not as large as for the oxide. In general, the results are between 10^{-4} and 10^{-5} . Fasting has been shown to increase absorption by up to an order of magnitude. For example, absorption in mice fasted for 8 hours before and 8 hours after the administration of ^{236}Pu bicarbonate was about 10^{-3} compared with 2×10^{-4} in fed animals (Larsen et al., 1981). High values of 10^{-3} to 2×10^{-3} have been reported for uptake of ^{237}Pu nitrate given as a single dose to rats and mice (Sullivan, 1981, Sullivan et al., 1982). These results were taken as evidence of increased absorption at low masses. However, in experiments to determine the effect of chronic ingestion at low concentrations, a value of 3×10^{-5} was obtained for the nitrate in rats (Weeks et al., 1956) and 10^{-5} for the bicarbonate in hamsters (Stather et al., 1981). It would appear that in general ingested mass and valence are not important factors affecting absorption. However, at high masses of Pu(V), absorption may be increased by an order of magnitude as demonstrated by Métivier et al. (1985) in studies using baboons.

The absorption of plutonium administered to animals as organic complexes or incorporated into food materials is generally greater than for inorganic forms (ICRP, 1986). For example, most of the reported values for plutonium citrate are in the range 6×10^{-5} – 6×10^{-4} compared with the range of 10^{-5} – 10^{-4} for the nitrate. An organic form of importance in reprocessing is Pu-tributylphosphate for which Métivier et al. (1983) measured absorption in rats as about 10^{-4} – 2×10^{-4} .

There is strong experimental evidence to conclude that plutonium absorption from the gastrointestinal tract may be increased by at least an order of magnitude in the human neonate, but that any increased absorption would probably decrease rapidly during the first few days or weeks of life (ICRP, 1986). The age by which absorption of plutonium might decrease to adult levels is not known, but animal studies indicate that adult values may be reached by about the time of weaning. An NEA/OECD Expert Group (NEA, 1988) proposed an f_1 of 10^{-2} as an average for the first year of life and 10^{-3} for all succeeding years.

In ICRP Publication 30 (1979), the recommended f_1 values were 10^{-5} for oxides and hydroxides and 10^{-4} for all other

inorganic forms. In ICRP Publication 48 (1986), f_1 values of 10^{-5} for oxides and hydroxides and 10^{-4} for nitrates were recommended. In addition, on the basis of animal data, an f_1 of 0.001 was recommended for all other forms of plutonium and was taken to apply as a general value for all actinides other than uranium. This value was also adopted for adult members of the public in ICRP Publication 56 (1989). However, subsequently in ICRP Publication 67 (1993), the recent human data for neptunium, plutonium, americium and curium were summarized and the conclusion was reached that, together with human data for thorium and available animal data, these provided a sufficient basis for the use of a value of 5×10^{-4} for intakes of plutonium in food by adults. This value is applied, in the absence of specific information, to children from one year of age. For infants in the first year of life, a value of 5×10^{-3} is used.

For intakes after an accident, central values are judged to be 1×10^{-5} for refractory oxide and 5×10^{-4} for relatively soluble forms, for adults and children. Both central values are judged to be a factor of three greater in infants: 3×10^{-5} for refractory oxide and 2×10^{-3} for soluble forms. High confidence intervals for adults and children are judged to be 1×10^{-6} – 5×10^{-5} for refractory oxide and 1×10^{-4} – 1×10^{-3} for soluble forms. For infants, it is possible that absorption is no higher than in adults and children, and therefore the same value is taken for the 5-percentile (x_5). Conversely, it is possible that absorption in infants could be 10 times higher, and therefore x_{95} values for infants are taken to be 10 times those for adults: 5×10^{-4} for refractory oxide and 1×10^{-2} for soluble forms.

6. Questions 10–16. Systemic Distribution and Retention

Questions 10 and 11 relate to strontium, plutonium, cerium, and tellurium. As noted in the Introduction, answers are only given for cerium, and these were largely prepared by my colleague Dr. J.W. Marsh, who reviewed the biokinetics of cerium after inhalation for ICRP Publication 71 (ICRP, 1996). Questions 12 – 14 relate to plutonium and have not been answered. Responses to Question 15, on ruthenium and cesium were largely prepared by my colleague, Ms. S.L. Prosser, who reviewed the biokinetics of ruthenium after inhalation for ICRP Publication 71. Question 16 relates to iodine and has not been answered.

6.1 Strontium, Plutonium, Cerium, Tellurium

Question 10. Considering the total amount reaching blood (as if administered intravenously as a single injection). %

retained in liver and skeleton (bone + bone marrow), as a function of time after entry into blood?

Question 11. Retention in the skeleton, % total retention in liver + skeleton (bone + marrow), as a function of time after entry into blood?

6.1.1 ICRP Publication 30

ICRP Publication 30, Part 1 (ICRP, 1979) states that intravenously injected cerium is mainly deposited in liver, spleen, skeleton, kidney cortex and adrenal glands (Hennacy, 1961; Tombropoulos et al., 1969; McKenney et al., 1961; Plesková et al., 1971; Moskalev, 1959). It assumed that, of cerium entering the “transfer compartment” (systemic uptake), 60% goes to liver, 20% to bone (5% to spleen and 15% to all other tissues). Cerium entering all those tissues was assumed to be retained with a biological half-time of 3500 d, which corresponds to whole body retention of cerium in beagle dogs (Richmond and London, 1966).

6.1.2 ICRP Publication 56

For adults, *ICRP Publication 56* (ICRP, 1989) reports the values chosen in *ICRP Publication 30*. It retains the 3500 d retention half-time, but adopts initial deposition fractions of 50% liver, 30% skeleton and 20% other tissues, citing experiments in dogs (Boecker and Cuddihy, 1974; Guilmette et al., 1987) and rats (Durbin, 1960).

For children, *ICRP Publication 56* notes that experimental results show that skeletal uptake is higher in young animals than adults (Buldakov and Burov, 1967; Guilmette et al., 1987; Kistner et al., 1987), with 70% uptake in newborns (Inaba and Lengemann, 1982). It assumes that skeletal uptake decreases with age and liver uptake correspondingly increases, with the total (80%) being independent of age (Table 6.1). It assumes, without comment, that the 3500 d retention half-time for all tissues, from *ICRP Publication 30*, applies at all ages.

6.1.3 References from ICRP Publications 30 and 56

(i) Moskalev (1959).

Rats were intravenously administered with ^{144}Ce chloride. Contents of organs are given in Table 6.2. The biological half-time in liver was initially 15 d; after 2 weeks it reduced to 7 d; and after a month increased to 30 d.

(ii) Durbin (1960).

Following intramuscular injection of cerium citrate into rats, the distribution of the absorbed activity was given after 4 d as: 51.0% liver and 27.7% skeleton (8% feces, and 6% urine).

(iii) Richmond and London (1966).

Four beagle dogs were injected with cerium chloride and whole body measurements were performed up to 1050 d after injection. A single exponential function was fitted to the data. The fitted biological half-times ranged from 3300 d to 3900 d.

(iv) Tombropoulos et al. (1969).

Not useful as it describes inhalation experiments only. Simulation modeling would be required to interpret.

(v) Plesková et al. (1971).

Mice and rats were intravenously injected with solutions of ^{144}Ce at pH 3 or at pH 8. In mice the amounts in liver were measured at chosen intervals (Table 6.3). In rats the amounts in the liver and the femurs were measured 1 d after injection (Table 6.4).

(vi) Inaba and Lengemann (1972).

One-day-old rats were given ^{141}Ce intraperitoneally. At 24 d after exposure whole body retention was approximately 70% of amount administered.

(vii) Boecker and Cuddihy (1974).

Data on dogs following inhalation of a relatively soluble form ($^{144}\text{CeCl}_3$ in CsCl vector). From the whole body, lung and bioassay data a compartment model was derived for dogs. The rate constants in this model indicate that the initial distribution from blood is: 36% liver, 27% skeleton (23% soft tissues, 12% feces, and 2% urine). However, the uncertainties on these are much greater than they would be for an injection study, because they depend on the fitted rates of absorption from the respiratory and GI tracts to blood. Retention in liver and skeleton were controlled by clearance rates to small intestine and blood respectively of 0.0001 d^{-1} , corresponding to a half-time of 7000 d. The effective retention half-time would be longer because of recycling in the model. However, because of the limited duration of the experiment (512 d), it can only be said to be long compared to the half-life of the radionuclide (284 d).

(viii) Guilmette et al. (1987)

Immature, young adult and aged dogs were exposed by inhalation to a ^{144}Ce -labelled fused aluminosilicate aerosol. Liver and skeleton retention data are given. As this is an inhalation experiment, simulation modeling as carried out by Boecker and Cuddihy (1974) would be needed to estimate transfers from blood to tissues. This was not reported, and would be more difficult since the material is far less soluble. Only comparisons are made between the groups. Compared to the young adults, uptake to liver was less rapid in the immature dogs, although the maximum levels reached were similar. (The maximum level was somewhat lower in the aged dogs). Compared to the young adults, uptake to skeleton was 2.4 times greater for the immature dogs (and approximately 1.6 times lower for the aged dogs).

(ix) Kistner et al. (1987)

^{141}Ce -chloride administered orally to adult and juvenile (aged 1 – 14 d) rats. Results indicated a distribution of: liver 20%; skeleton 60% in the juvenile rats.

(x) Others

The reports by Hennacy (1961) and McKenny (1961) were not readily available. The paper by Buldakov and Burov (1967) was obtained, but is in Russian.

6.1.4 References from ICRP Publication 71

(x) Sturbaum et al. (1970)

Chinese hamsters were injected with ^{144}Ce chloride and with ^{144}Ce citrate. The tissue distributions are given in Table 6.5.

6.1.5 Summary of literature survey on deposition and retention of cerium

Considering the importance of ^{144}Ce as a fission product, surprisingly little useful information was found, and most of the data were from rodents. Inhalation studies have been conducted in dogs, but were not complemented by systemic biokinetics studies. This may well reflect the fact that there is such long-term retention of cerium in tissues such as liver and skeleton that the physical decay dominates overall clearance, and more detailed information was not deemed necessary for dosimetry purposes.

Adults

Table 6.6 gives a summary of the useful results from the literature survey, relating to the amounts initially translocated to liver and skeleton. Most are from rodents, but are quite consistent between the different species, giving a total in liver and skeleton of about 80%, as assumed in *ICRP Publications 30* and 56. Similarly about 30% goes to skeleton, i.e., about 40% of the total to liver + skeleton.

In *ICRP Publications 30* and 56 the biological retention half-times in the skeleton and liver are assumed to be equal to the whole body retention of cerium in beagle dogs reported by Richmond and London (1966). No other retention data were found except in rodents.

Five-year-old children

Kistner et al. (1987) found that total deposition in liver + skeleton was similar in young and adult rats. They also found that in young rats the ratio of deposition in liver to skeleton was 20%:60%, compared with the 60%:20% assumed in *ICRP Publication 30*. Guilmette et al. (1987) found that in immature dogs there was higher skeletal uptake and less rapid liver uptake than in young adult dogs.

The retention half times in liver and bone for 5-y-old children are assumed in *ICRP Publication 56* to be the same as adults. No other retention data were found.

Table 6.7 gives the uncertainty in the expected initial (1 d) distribution in liver and bone. The median values are based on *ICRP Publication 56*. The 5% and 95% quantiles on total deposition in liver + skeleton are based on the information in Table 6.6, and assumed to be the same in children. The 5% and 95% quantiles on deposition in the skeleton of adults are similarly based on the information in Table 6.6. These suggest that x_5 is approximately $0.5x_{50}$, and that x_{95} is approximately $1.5x_{50}$. It was assumed that these ratios also applied to children, and were used to derive x_5 and x_{95} for initial deposition in the skeleton (% injected) from x_{50} . Quantiles for [skeleton / (liver + skeleton)] were derived from those for skeleton and for (liver + skeleton).

6.1.6 Chemical analogy - americium

In view of the limited information available, especially on retention, and the total lack of human data, consideration was given to chemical analogues. *ICRP Publication 48* (ICRP, 1986) Paragraph 6.45, in discussing skeletal retention of actinides, points out that comparison of the long-term retention of a variety of metals in rats suggests

that retention may be more a function of bone turnover rate than of the specific chemical properties of the metal, and a retention half-time of the order of the normal life-span of the animal may be generally descriptive of the process (Taylor, 1983). On this basis the whole-body retention half-time of 3500 d derived from Richmond and London (1966) could be more closely related to beagle dogs than to cerium.

At the video-conference discussion between panel experts in Brussels and Albuquerque on 29 February, 1996, more than one participant mentioned using a model based on americium, rather than the ICRP cerium model.

ICRP Publication 56 adopts an f_1 value of 10^{-3} for cerium, based on analogy with actinides, in preference to the *ICRP Publication 30* value (which was based on cerium compounds), "...Because of the similarity of the physicochemical properties of cerium and some actinides...". Of those actinides for which there is sufficient information to make analogy worthwhile, americium appears to be a suitable choice, at least in terms of the initial deposition in liver and skeleton. In the latest ICRP model for americium (*ICRP Publication 67*), the total deposition in liver and bone surfaces (skeleton) is set to 80%. The relative deposition skeleton:liver is set to 3.5 in adults and 5:3 in 5-y-old children, which are all the same as for cerium in *ICRP Publication 56*.

ICRP Publication 67 gives, in Figures B2 and B3, model predictions of the americium content of skeleton and liver respectively (% americium injected), as a function of time up to 50 y, for infants, 10-y-old children, and adults. The biokinetics in the 5-y-old are likely to be much closer to those of the 10-y-old than to the infant. Indeed, many of the age-specific transfer rates for the americium model (listed in Table B-1) are the same at 5 and 10 y, including those giving the initial distribution to liver and skeleton. We have therefore taken the values for the 10-y-old to be representative of the 5-y-old. (Since the model is a complex recycling model, evaluating retention in liver and skeleton specifically for the 5-y-old would be time-consuming.) Values of the *ICRP Publication 67* model predictions of the americium content of skeleton and liver respectively (% americium injected), at the required times for Questions 10 and 11, taken from Figures B2 and B3, are given in Table 6.8 (adult) and 6.9 (child). These form the basis of our median values. It should be noted that as this is a recycling model, much of the activity that leaves the liver during the first few years transfers to skeleton, with the result that the activity in skeleton reaches a maximum at about 1 y in children and 7 y in adults. From 10 y onwards the effective ("externally-viewed") retention half-time in the skeleton is

about 45 y in children and 60 y in adults. For liver, the "externally-viewed" retention half-time is 2-3 y during the first few years, but at late times, equilibrium is reached at a few per cent of the total systemic activity, with the amount leaving liver per unit time balanced by that depositing from blood through recycling.

ICRP Publication 48 (ICRP, 1986) Section VI.3 (Animal Data on Actinide Distribution and Retention) includes some discussion of uncertainties in americium biokinetics. For liver, Paragraph 6.43 notes that Griffiths et al. (1983), from reviews of animal and human data, estimated a retention half-time of americium in human liver of about 2 y (similar to the *ICRP Publication 67* model), with 95% confidence limits of 1.1 – 8 y. For skeleton, Paragraph 6.45 notes that Griffiths et al. (1983), estimated a retention half-time of americium in human skeleton of about 30 y, and that more recently Durbin and Schmidt (1985) suggested a half-time of 90 y for the major long-term component of americium retention in the human skeleton, based on monkey and human data. These values broadly encompass that predicted by the *ICRP Publication 67* model.

To obtain estimates of the likely range in retention in liver and skeleton for children and adults at the required times, "low" and "high" retention estimates were made. Low and high estimates of initial distribution were taken from Table 6.7.

Adults:	low retention	60% (15% skeleton; 45% liver)
	high retention	90% (45% skeleton; 45% liver)
Children:	low retention	60% (25% skeleton; 35% liver)
	high retention	90% (75% skeleton; 15% liver)

For low retention, half-times in liver and skeleton were taken to be 1.1 and 30 y respectively. For high retention, half-times in liver and skeleton were taken to be 8 and 90 y respectively. Results are given in Tables 6.10 and 6.11 for adults and children, respectively. It should be noted that use of these effectively implies a non-recycling model, with a single compartment for each of liver and skeleton. The results do not encompass those from *ICRP Publication 67* at all times: in particular the very long-term liver retention is not predicted.

6.1.7 Application to questions

Median (x_{50}) values for Questions 10 and 11 are taken from Tables 6.8 and 6.9, i.e., the *ICRP Publication 67* americium model.

For Question 10, total retention in liver + skeleton, x_5 and x_{95} for children were taken from Table 6.11. For adults, x_5 was taken from Table 6.10 at all times and x_{95} was taken from Table 6.10 at times up to 1 y. At later times the "high retention" values in Table 6.10 were very similar to the medians, and therefore higher values were selected (actually similar to those for children).

For Question 10, retention in skeleton / (total retention in liver + skeleton), the relative uncertainties in the initial distribution (from Table 6.7) were followed. These suggest that x_5 is approximately $0.5x_{50}$, and that x_{95} is approximately $1.5x_{50}$. This procedure was followed for values of $x_{50} < 50\%$. For values of $x_{50} > 50\%$ it was assumed that the uncertainty in the complement of the skeletal retention (i.e., liver / skeleton + liver) followed a similar pattern. For example, when x_{50} (skeleton) = 86%, x_{50} (liver) is $100 - 86 = 14\%$. It was assumed that x_5 (liver) is $0.5x_{50}$ (liver) = 7%, and that x_{95} (liver) is $1.5x_{50} = 21\%$. Hence x_5 (skeleton) = $100 - x_{95}$ (liver) = $100 - 21 = 79\%$; and x_{95} (skeleton) = $100 - x_5$ (liver) = $100 - 7\% = 93\%$.

6.2 Ruthenium, Cesium

Question 15. Considering the total amount reaching blood (as if administered intravenously as a single injection), % total retention (whole body or systemic) as a function of time after entry into blood?

Responses to Question 15 were largely prepared by my colleague, Ms. S.L. Prosser. She reviewed the biokinetics of ruthenium after inhalation for *ICRP Publication 71*. She has also reviewed information on cesium whole body retention for other projects. For both elements she reviewed the literature for relevant information, using the latest ICRP reviews (*ICRP Publications 56* and *67*) as starting points.

6.2.1 Ruthenium

The only useful study found was that of Furchner et al. (1971), who followed retention of ^{106}Ru after intraperitoneal injection into mice and rats, and intravenous injection into monkeys and dogs.

Results (parameters of whole-body retention functions) are given in Table 6.12, based on Table 2 of Furchner et al. (1971). The results (probably for monkeys and dogs) are used as the basis for the retention function for man in *ICRP Publication 30*. This is retained in *ICRP Publications 56* and *67*, where it is noted that with the exception of the long-term component, the parameters are similar in the different species. The rate constant of the long-term component

shows considerable variation between species, with no trend with body mass. However, we note that it is inversely related to the duration of the measurements (Table 6.13): as might well arise if the clearance rate was continuing to decrease with time. These functions were used to derive retention at the specified times (Table 6.13). Note that the 5-y value is an extrapolation, since the latest measurements were at 2.7-y, in dogs.

We take the *ICRP Publication 56* values for the median x_{50} . There is quite good agreement between the four species at times up to a year, but wide variation in the 5-y values as a result of extrapolating different functions. As with *ICRP Publication 56*, we assume retention in children is the same for median values, but widen the confidence interval to allow for additional uncertainty.

6.2.2 Cesium

Many useful data are available from studies which examine the retention of cesium in humans. These studies included routine monitoring of workers, whole body measurements of members of the public following exposure to weapons test fallout or Chernobyl ^{137}Cs or, intravenous injection of the radionuclide.

Results of the published studies are given in Table 6.14 for adults and in Table 6.15 for 5-y-old children. The studies varied in that some expressed cesium retention as a single component function whereas others were based on two-component functions. These functions were used to derive retention at the specified times (Tables 6.14 and 6.15). Many of the values at later time periods were extrapolations, since some studies did not proceed beyond a year, or even a month in some cases.

For median values we have taken *ICRP Publication 56* (67) functions:

$$R(t) = a_1 \exp(-k_1 t) + a_2 \exp(-k_2 t)$$

(Biological half-time, d)				
	a_1	a_2	k_1	k_2
Child	45	55	9.1	30
Male	10	90	2	110
Female	10	90	2	63*

* This is not actually recommended in *ICRP Publication 56*. The text refers to two studies in which average values were 61 and 65 d. We have taken 63 d as the arithmetic mean of these. For adult average retention at each time we have taken a geometric mean of the male and female retention.

References

Agnew, J.E., Bateman, J.R.M., Pavia, D. and Clarke, S.W. 1984. A model for assessing bronchial mucus transport. *J. Nucl. Med.* 25, 170-176.

Agnew, J.E., Sutton, P.P., Pavia, D. and Clarke, S.W. 1986a. Radioaerosol assessment of mucociliary clearance: Towards definition of a normal range. *Br. J. Radiol.* 59, 147-151.

Agnew, J.E., Pavia, D. and Clarke, S.W. 1986b. Mucus clearance from peripheral and central airways of asymptomatic cigarette smokers. *Bull. Eur. Physiopathol. Respir.* 22, 263-267.

Albert, R.E. and Arnett, L.C. 1955. Clearance of radioactive dust from the human lung. *AMA Arch. Ind. Health* 12, 99-106.

Albert, R.E., Lippmann, M., Spiegelman, J., Strehlow, C., Briscoe, W., Wolfson, P. and Nelson, N. 1967. The clearance of radioactive particles from the human lung. In: *Inhaled Particles and Vapors II*, Proceedings of an International Symposium Organized by the British Occupational Hygiene Society, Cambridge, 28 September-1 October 1965 (Ed. Davies, C.N.), Pergamon Press, Oxford, pp. 361-378.

Albert, R.E., Lippmann, M. and Briscoe, W. 1969. The characteristics of bronchial clearance in humans and the effects of cigarette smoking. *Arch. Environ. Health* 18, 738-755.

Albert, R.E., Lippmann, M., Peterson, H.T., Berger, J., Sanborn, K. and Bohning, D. 1973. Bronchial deposition and clearance of aerosols. *Arch. Int. Med.* 131, 115-127.

Albert, R.E., Peterson, H.T., Bohning, D.E. and Lippmann, M. 1975. Short-term effects of cigarette smoking on bronchial clearance in humans. *Arch. Environ. Health* 30, 361-367.

Anderson, E., Braune, N., Duletsky, S., Ramig, J. and Warri, T. 1985. Development of Statistical Distributions of Ranges of Standard Factors in Exposure Assessments. EPA 600/8.85/010 Environmental Protection Agency, Washington, D.C.

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

Armbruster, L. and Breuer, H. 1982. Investigations into defining inhalable dust. In: *Inhaled Particles V*, Proceedings of an International Symposium Organized by the British Occupational Hygiene Society, Cardiff, 8-12 September 1980 (Eds. Walton, W.H., Critchlow, A. and Coppock, S.M.), Pergamon Press, Oxford, United Kingdom. *Ann. Occup. Hyg.* 26, 33-45.

Armbruster, L. and Breuer, H. 1984. Sampling efficiency of various probes for sampling inhalable dust. *Ann. Occup. Hyg.* 28, 249-257.

Bailey, M.R. 1989. Application of human volunteer studies in setting exposure limits. *Radiat. Prot. Dosim.* 26, 249-257.

Bailey, M.R. and Roy, M. 1994. Annex E: Clearance of Particles from the Respiratory Tract. In: *Human Respiratory Tract Model for Radiological Protection*. Annals of the ICRP, 24, 301-413.

Bailey, M.R., Fry, F.A. and James, A.C. 1982. The long-term clearance kinetics of insoluble particles from the human lung. *Ann. Occup. Hyg.* 26, 273-290.

Bailey, M.R., Fry, F.A. and James, A.C. 1985a. Long-term retention of particles in the human respiratory tract. *J. Aerosol Sci.* 16, 295-305.

Bailey, M.R., Hodgson, A. and Smith, H. 1985b. Respiratory tract retention of relatively insoluble particles in rodents. *J. Aerosol Sci.* 16, 279-293.

Bailey, M.R., Kreyling, W.G., André, S., Batchelor, A., Collier, C.G., Drosselmeyer, E., Ferron, G.A., Foster, P., Haider, B., Hodgson, A., Masse, R., Métivier, H., Morgan, A., Müller, H.-L., Patrick, G., Pearman, I., Pickering, S., Ramsden, D., Stirling, C. and Talbot, R.J. 1989. An interspecies comparison of the lung clearance of inhaled monodisperse cobalt oxide particles - Part I: Objectives and summary of results. *J. Aerosol Sci.* 20, 169-188.

Bailey, M.R., Birchall, A., Cuddihy, R.G., James, A.C. and Roy, M. 1991. Respiratory tract clearance model for dosimetry and bioassay of inhaled radionuclides. *Radiat. Prot. Dosim.* 53, 153-158.

Bailey, M.R., Dorrian, M.-D. and Birchall, A. 1995. Implications of airway retention for radiation doses from inhaled radionuclides. In: *Proceedings of the Symposium on the Fate of Aerosol Particles in the Airways*, Frankfurt-am-Main, Germany, March 1994. *J. Aerosol Med.* 8, 373.

Bair, W.J. 1961. Deposition, retention, translocation and excretion of radioactive particles. In: *Inhaled Particles and Vapors*, Proceedings of an International Symposium organized by the British Occupational Hygiene Society, Oxford 1960 (Ed. Davies, C.N.). pp. 192-208, Pergamon Press, Oxford.

Bair, W.J., Willard, D.H. and Temple, L.A. 1961. The behavior of inhaled Ru10602 particles. *Health Phys.* 5, 90-98.

Bair, W.J., Snyder, M.D., Walters, R.A. and Keough, R.F. 1963. Effect of I127 on thyroid uptake of inhaled I131. *Health Phys.* 9, 1399-1410.

Bair, W.J., Métivier, H. and Park, J.F. 1980. Comparison of early mortality in baboons and dogs after inhalation of $^{239}\text{PuO}_2$. *Radiat. Res.* 82, 588-610.

Bateman, J.R.M., Clarke, S.W., Pavia, D. and Sheahan, N.F. 1978a. Reduction in clearance of secretions from the human lung during sleep. *J. Physiol.* 284, 55.

Bateman, J.R.M., Pavia, D. and Clarke, S.W. 1978b. The retention of lung secretions during the night in normal subjects. *Clin. Sci. and Mol. Med.* 55, 523-527.

Bateman, J.R.M., Sheahan, N.F., Pavia, D. and Clarke, S.W. 1979. The effect of sleep, circadian rhythm, physical activity and posture on the clearance of secretions from the human lung. *Am. Rev. Respir. Dis.* 120, 200.

Baverstock, K.F. Half-time clearance of isotopes of caesium in man. 1987. In: *Age-related Factors in Radiumuclide Metabolism and Dosimetry*. (Eds. Gerber, G.B., Métivier, H. and Smith, H.), pp. 215-220. Martinus Nijhoff Publishers, Dordrecht.

Becquemin, M.H., Roy, M., Robeau, D., Bonnefous, S., Piechowski, J. and Teillac, A. 1987. Inhaled particle deposition and clearance from the normal respiratory tract. *Respir. Physiol.* 67, 147-158.

Becquemin, M.H., Yu, C.P., Roy, M. and Bouchikhi, A. 1991. Total deposition of inhaled particles related to age: Comparison with age-dependent model calculations. *Radiat. Prot. Dosim.* 38, 23-28.

Bedford J., Harrison G.E., Raymond W.H.A. and Sutton A. 1960. The metabolism of strontium in children, *Br. Med. J.* 1, 589-592.

Bennett, B.G. 1976. Transuranic element pathways to man. In: *Transuranium Nuclides in the Environment* STI/PUB/410, pp. 367-383. International Atomic Energy Agency, Vienna.

Bennett, W.D. 1991. Targeting respiratory drug delivery with aerosol boluses. *J. Aerosol Med.* 4, 69-78.

Bennett, W.D., Chapman, W.F., Lay, J.C. and Gerrity, T.R. 1993. Pulmonary clearance of inhaled particles 24 to 48 hours post deposition: Effects of beta-adrenergic stimulation. *J. Aerosol Med.* 6, 53-62.

Berg, H.F., Christopherson, W.M. and Bryant, J.R. 1954. Time and site study for optimum lymph node concentration of radiogold following intrabronchial injection. *Cancer Res.* 4, 775-779.

Bertholon, J.F. and Roy, M. 1994. Comments on metabolically consistent breathing rates. *Health Phys.* 66, 89.

Black, A. and Hounam, R.F. 1968. Penetration of iodine vapor through the nose and mouth and the clearance and metabolism of the deposited iodine. *Ann. Occup. Hyg.* 11, 209-225.

Blackadder, E.S. and Manderson, W.G. 1975. Occupational absorption of tellurium: a report of two cases. *Brit. J. Ind. Med.* 32, 59-61.

Blanchard, J.D. and Willeke, K. 1984. Total deposition of ultrafine sodium chloride particles in human lungs. *J. Appl. Physiol.* 57, 1850-1856.

Boecker, B.B. 1969. Comparison of ^{137}Cs metabolism in the beagle dog following inhalation and intravenous injection. *Health Phys.* 16, 785-788.

Boecker, B.B. and Cuddihy, R.G. 1974. Toxicity of ^{144}Ce inhaled as $^{144}\text{CeCl}_3$ by the Beagle: Metabolism and dosimetry. *Radiat. Res.* 60, 133-154.

Boecker, B.B. and Harris, A.M. 1969. Tissue distribution, excretion and dosimetry of inhaled ^{106}Ru citrate in the beagle dog. *Fission Product Inhalation Program Annual Report 1968-1969*, LF-41, pp. 111-116. Lovelace Foundation for Medical Education and Research, Albuquerque, New Mexico. Available from National Technical Information Service, Springfield, Virginia.

Boecker, B.B., Chiffelle, T.L., Hobbs, C.H., Jones, R.K., McCellan, R.O., Pickrell, J.A. and Redman, H.C. 1967. Toxicity of inhaled $^{90}\text{SrCl}_2$ in Beagle dogs III. *Fission Product Inhalation Program Annual Report 1968-1969*, LF-41, pp. 1-7. Lovelace Foundation for Medical Education and Research, Albuquerque, New Mexico. Available from National Technical Information Service, Springfield, Virginia.

Boecker, B.B., Cuddihy, R.G., Hahn, F.F. and McClellan, R.O. 1974. A seven year study of the pulmonary retention and clearance of ^{137}Cs inhaled in fused aluminosilicate particles by the beagle dog. *Inhalation Toxicology Research Institute Annual Report 1973-1974*, LF-49, pp. 48-52. Lovelace Biomedical and Research Foundation, Albuquerque, New Mexico. Available from National Technical Information Service, Springfield, Virginia.

Bohning, D.E., Albert, R.E., Lippmann, M. and Foster, W.M. 1975. Tracheobronchial particle deposition and clearance. A study of the effects of cigarette smoking in monozygotic twins. *Arch. Environ. Health* 30, 457-462.

Bohning, D.E., Atkins, H.L. and Cohn, S.H. 1982. Long-term particle clearance in man: Normal and impaired. In: *Inhaled Particles V*, Proceedings of an International Symposium Organized by the British Occupational Hygiene Society, Cardiff, 8-12 September 1980 (Eds. Walton, W.H., Critchlow, A. and Coppock, S.M.), Pergamon Press, Oxford, United Kingdom. *Ann. Occup. Hyg.* 26, 259-271.

Booker, D.V., Chamberlain, A.C., Rundo, J., Muir, D.C.F. and Thomson, M.L. 1967. Elimination of 5m particles from the human lung. *Nature* 215, 30-33.

Bowden, D.H. 1987. Minireview: Macrophages, dust and pulmonary diseases. *Exp. Lung Res.* 12, 89-107.

Brain, J.D. 1985. Macrophages in the Respiratory Tract. Chapter 14. In: *Handbook of Physiology. The Respiratory System. Volume 1. Circulation and Nonrespiratory Functions.* (Eds. Fishman, A.P. and Fisher, A.B.) American Physiological Society, Bethesda, Maryland, pp. 447-471.

Brain, J.D. 1986. Toxicological aspects of alterations of pulmonary macrophage function. *Ann. Rev. Pharmacol. Toxicol.* 26, 547-565.

Brain, J.D. 1988. Lung macrophages: How many kinds are there? What do they do? *Am. Rev. Respir. Dis.* 137, 507-509.

Brain, J.D., Proctor, D.F. and Reid, L.M. (Eds.) (1977). *Respiratory Defense Mechanisms.* Marcel Dekker Inc., New York.

Brain, J.D., Gehr, P. and Kavet, R.I. 1984a. Airway macrophages. The importance of the fixation method. *Am. Rev. Respir. Dis.* 129, 823-826.

Brain, J.D., Bloom, S.B., Valberg, P.A. and Gehr, P. 1984b. Correlation between the behavior of magnetic iron oxide particles in the lungs of rabbits and phagocytosis. *Experim. Lung Res.* 6, 115-131.

Breysse, P.N. and Swift, D.L. 1990. Inhalability of large particles into the human nasal passage: In vivo studies in still air. *Aerosol Sci. Technol.* 13, 459-464.

Briant, J.K. and Sanders, C.L. 1987. Inhalation deposition and retention patterns of a U-Pu chain aggregate aerosol. *Health Phys.* 53, 365-375.

Brooks, A.L., Mewhinney, J.A., Redman, H.C., Guilmette, R.A. and McClellan, R.O. 1981. Distribution and retention of ^{239}Pu , and cytogenetic damage in cynomolgus monkeys after inhalation of $^{239}\text{Pu}(\text{NO}_3)_4$. *Inhalation Toxicology Research Institute Annual Report 1980-1981, LMF-91*, pp. 194-197. Lovelace Biomedical & Environmental Research Foundation, Albuquerque, New Mexico. Available from National Technical Information Service, Springfield, Virginia.

Brown, L. 1983. National radiation survey in the U.K.: Indoor occupancy factors. *Radiat. Prot. Dosim.* 5, 203-208.

Bryant, J.R., Berg, H.F. and Christophersen, W.M. 1953. Localization of radioactivity in the lung and the lymph nodes. *J. Thorac. Surg.* 26, 221-232.

Buldakov, L.A. and Burov, N.I. 1967. Behavior of ^{144}Ce in sheep of different ages. *UDC 578.088.91:577.391*, pp. 127-141.

Camner, P. 1980. Alveolar clearance. *Eur. J. Respir. Dis.* 61 (Suppl. 107), 59-72.

Camner, P. 1981. Clinical aspects on lung clearance. In: *Deposition and Clearance of Aerosols in the Human Respiratory Tract.* Bad Gleichenberg, 1981. (Ed. Hauck, H.) Arbeitsgemeinschaft für Aerosole in der Medizin, pp. 89-105.

Camner, P. 1984. Clearance mechanisms. In: *Lung modeling for Inhalation of Radioactive Materials. Report EUR 9384.* (Eds. Smith, H. and Gerber, G.B.) Commission of the European Communities, Luxembourg, pp. 181-191.

Camner, P. 1988. Minireview: How important is mucociliary clearance? *Exp. Lung Res.* 14, 423-429.

Camner, P. and Lundborg, M. 1977. Alveolar macrophages and teflon particles coated with carbon and metals. In: *Pulmonary Macrophage and Epithelial Cells.* (Eds. Sanders, C.L., Schneider, R.P., Dagle, G.E. and Ragan, H.A.) Available from the National Technical Information Service, Springfield, Virginia, pp. 405-413.

Camner, P. and Mossberg, B. 1988. Mucociliary disorders: A review. *J. Aerosol Med.* 1, 21-28.

Camner, P. and Philipson, K. 1972. Tracheobronchial clearance in smoking discordant twins. *Arch. Environ. Health* 25, 60-63.

Camner, P., Philipson, K. and Arvidsson, T. 1971. Cigarette smoking in man. Short-term effect on mucociliary transport. *Arch. Environ. Health* 23, 421-426.

Camner, P., Philipson, K. and Friberg, L. 1972. Tracheobronchial clearance in twins. *Arch. Environ. Health* 24, 82-87.

Camner, P., Philipson, K. and Arvidsson, T. 1973. Withdrawal of cigarette smoking. A study on tracheobronchial clearance. *Arch. Environ. Health* 26, 90-92.

Camner, P., Hellström, P.-A., Lundborg, M. and Philipson, K. 1977. Lung clearance of 4-mm particles coated with silver, carbon, or beryllium. *Arch. Environ. Health* 32, 58-62.

Carbaugh, E.H., Bihl, D.E. and Sula, M.J. 1991. Long-term follow-up of Han-1, an acute plutonium oxide case. *Radiat. Prot. Dosim.* 38, 99-104.

Carr, T. 1967. An attempt to quantitate the short term movement of strontium in the human adult. In: *Strontium Metabolism*. (Eds. Lenihan, J., Loutit, J. and Martin, J.)

Cember, H. and Stemmer, K. 1964. Lung cancers from radioactive cerium chloride. *Health Phys.* 10, 43-48.

Cember, H. and Watson, J.A. 1958. Lung hazards from inhaled radioactive particulate matter. *Progress Report, U.S.A.E.C. Contract AT(30-1)912.*

Chevalier, C., Bataller, G. and Jeanmaire, L. 1990. The use of post-Chernobyl human data for caesium: Model validation and intake estimation. *Rad. Prot. Dosim.* 32, 1, 113-118.

Chopra, S.K., Taplin, G.V., Elam, D., Carson, S.A. and Golde, D. 1979. Measurement of tracheal mucociliary transport velocity in humans - smokers versus non-smokers (preliminary findings). *Am. Rev. Respir. Dis.* 120, 205.

Churg, A. and Stevens, B. 1988. Association of lung cancer and airway particle concentration. *Environ. Res.* 45, 58-63.

Churg, A. and Wright, J.L. 1988. Mineral particles in airway walls in the lungs of long-term chrysotile miners. In: *Inhaled Particles VI, Proceedings of an International Symposium and Workshop on Lung Dosimetry Organized by the British Occupational Hygiene Society in Co-operation with the Commission of the European Communities, Cambridge, 2-6 September 1985*, (Eds. Dodgson, J., McCallum, R.I., Bailey, M.R. and Fisher, D.R.), Pergamon Press, Oxford, United Kingdom. *Ann. Occup. Hyg.* 32 (Suppl. 1), 173-180.

Churg, A., Wright, J.L. and Stevens, B. 1990. Exogenous mineral particles in the human bronchial mucosa and lung parenchyma. I. Nonsmokers in the general population. *Exp. Lung Res.* 16, 159-175.

Churg, A., Wright, J.L., Stevens, B. and Wiggs, B. 1992. Mineral particles in the human bronchial mucosa and lung parenchyma II. Cigarette smokers without emphysema. *Exp. Lung Res.* 18, 687-714.

Cohen, D., Arai, S.F. and Brain, J.D. 1979. Smoking impairs long-term dust clearance from the lung. *Science* 204, 514-517.

Cohen, B.S., Eisenbud, M. and Harley, N.H. 1980. Measurement of the α -radioactivity on the mucosal surface of the human bronchial tree. *Health Phys.* 39, 619-632.

Cohn, B.N.E. 1932. Absorption of compound solution of iodine from the gastrointestinal tract with special reference to the absorption of free iodine. *Arch. Int. Med.* 49, 950.

Cohn, S.H., Rosoff, B., Gusmano, E.A. and Spencer, H. 1963. Long-term Cs-137 turnover in man as measured by a whole-body counter. *Radiat. Res.* 19, 655-658.

Collier, C., Hodgson, A., Gray, S., Moody, J. and Ball, A. 1991. The lung clearance kinetics of $^{57}\text{Co}_3\text{O}_4$ in rats of various ages. *J. Aerosol Sci.* 22, 537-549.

Cooper, D.M. and Weiler-Ravell, D. 1984. Gas exchange response to exercise in children. *Am. Rev. Respir. Dis.* 129 (Suppl. S47-S48).

Corry, D., Kulkarni, P. and Lipscomb, M.F. 1984. The migration of bronchoalveolar macrophages into hilar lymph nodes. *Am. J. Pathol.* 115, 321-328.

Cotes, J.E. 1979. *Lung Function Assessment and Application in Medicine*. Blackwell Scientific Publications, Oxford.

Coughtrey, P. and Thorne, M. 1983. Radionuclide distribution and transport in terrestrial and aquatic ecosystems, Vol. 1. A.A. Balkema, Rotterdam. pp. 170-178.

Coughtrey, P., Jackson, D. and Thorne, M. 1983. Radionuclide distribution and transport in terrestrial and aquatic ecosystems, Vol. 3. A.A. Balkema, Rotterdam. pp. 341-346.

Crawford-Brown, D.J. and Wilson, J. 1984. Observations on very long-term removal of uranium compounds. *Health Phys.* 47, 443-446.

Cuddihy, R.G. 1976. Respiratory tract retention of inhaled particles in experimental animals. *Inhalation Toxicology*

Research Institute Annual Report 1975-76 LF-56. Lovelace Foundation, Albuquerque, New Mexico, pp. 137-142.

Cuddihy, R.G. 1984. Mathematical models for predicting clearance of inhaled radioactive substances. In: Lung modeling for Inhalation of Radioactive Materials. (Eds. Smith, H. and Gerber, G.), Report EUR 9384, Commission of the European Communities, Luxembourg, pp. 167-179.

Cuddihy, R.G. and Ozog, J.A. 1973. Nasal absorption of CsCl, SrCl₂, BaCl₂, and CeCl₃ in Syrian Hamsters. *Health Phys.* 25, 219-224.

Cuddihy, R.G. and Yeh, H.C. 1988. Respiratory tract clearance of particles and substances dissociated from particles. In: *Inhalation Toxicology: The Design and Interpretation of Inhalation Studies and Their Use in Risk Assessment*. (Ed. Mohr, U.) Springer-Verlag, pp. 169-193.

Cuddihy, R.G., Gomez, S.R. and Pfleger, R.C. 1975. Inhalation exposures of beagle dogs to cerium aerosols: Physical chemical and mathematical analysis. *Health Phys.* 29, 257-265.

Cuddihy, R.G., Boecker, B.B. and Griffith, W.C. 1979. modeling the deposition and clearance of inhaled radionuclides. In: *Biological Implications of Radionuclides Released from Nuclear Industries*, Vol. II. International Atomic Energy Agency, Vienna, Austria, pp. 77-90.

Cuddihy, R.G., Finch, G.L., Newton, G.J., Hahn, F.F., Mewhinney, J.A., Rothenberg, S.J. and Powers, D.A. 1989. Characteristics of radioactive particles released from the Chernobyl nuclear reactor. *Environ. Sci. Technol.* 23, 89-95.

Dagle, G.E., Cannon, W.C., Stevens, D.L. and McShane, J.F. 1983. Comparative distribution of inhaled ²³⁸Pu and ²³⁹Pu nitrates in beagles. *Health Phys.* 44, 275-277.

Davies, C.N. 1980. An algebraical model for the deposition of aerosols in the human respiratory tract during steady breathing - Addendum. *J. Aerosol Sci.* 11, 213-224.

Davies, C.P. and Webster, A.J.F. 1988. Effects of particle size and age on deposition and clearance of monodispersed particles in the calf lung. *J. Aerosol Med.* 3, 195.

Dawson, C.A., Skebba, S.C., Lineham, J.H. and Bronikowski, T.A. 1985. Influence of pulmonary embolism on absorption of inhaled iodide-125. *J. Appl. Physiol.* 58, 1061-1068.

Dejours, P. 1982. *Physiologie de la Respiration*, 3e ed., Flammarion Medecine Sciences, Paris, p. 251.

Dennis, W.L. 1961. Discussion. In: *Inhaled Particles and Vapors*, Proceedings of an International Symposium Organized by the British Occupational Hygiene Society, Oxford, 29 March-1 April, 1960, (Ed. Davies, C.N.), Pergamon Press, New York, pp. 82-91.

Devell, L. 1988. Nuclide composition of Chernobyl hot particles. In: *Hot Particles from the Chernobyl Fallout*. Proceedings of an International Workshop, Theuern, October 1987, (Eds. von Philipsborn, H. and Steinhäuser, F.). pp. 23-34. Bergbau und Industriemuseum Ostbayern, Band 16.

Diel, J.H. and Mewhinney, J.A. 1981. Fragmentation of inhaled ²³⁸PuO₂ particles in lung. *Health Phys.* 44, 135-143.

Dua, S.K., Maniyan, C.G. and Kotrappa, P. 1987. Inhalation exposures during operations in spent fuel bays. *Radiat. Prot. Dosim.* 19, 165-172.

Durbin, P.W. 1960. Metabolic characteristics within a chemical family. *Health Phys.* 2, 225-238.

Durbin, P.W. and Schmidt, C.T. 1985. The US Transuranium Registry report on the ²⁴¹Am content of a whole body. V. Implications for metabolic modeling. *Health Phys.* 2, 225-238.

Duthie, E.S. 1930. Phagocytosis by bronchial epithelium in the lungs of mice. *J. Pathol. Bacteriol.* 33, 547-551.

Egan, M.J. and Nixon, W. 1985. A model of aerosol deposition in the lung for use in inhalation dose assessments. *Radiat. Prot. Dosim.* 11, 5-17.

Egan, M.J. and Nixon, W. 1987. Mathematical modeling of fine particle deposition in the respiratory system. In: *Deposition and Clearance of Aerosols in the Human Respiratory Tract* (Ed. Hoffmann, W.), Facultas Universitätsverlag, Vienna, Austria, pp. 34-40.

Egan, M.J., Nixon, W., Robinson, N.I., James, A.C. and Phalen, R.F. 1989. Inhaled aerosol transport and deposition calculations for the ICRP Task Group. *J. Aerosol Sci.* 20, 1305-1308.

Emmett, P.C. and Aitken, R.J. 1982. Measurements of the total and regional deposition of inhaled particles in the human respiratory tract. *J. Aerosol Sci.* 13, 549-560.

Felicetti, S.A., Wolff, R.K. and Muggenburg, B.A. 1981. Comparison of tracheal mucous transport in rats, guinea pigs, rabbits, and dogs. *J. Appl. Physiol.* 51, 1612-1617.

Ferin, J., Oberdörster, G., Penney, D.P., Soderholm, S.C., Gelein, R. and Piper, H.C. 1990. Increased pulmonary toxicity of ultrafine particles. I. Particle clearance, translocation, morphology. *J. Aerosol Sci.* 21, 381-384.

Ferin, J., Oberdörster, G., Soderholm, S.C. and Gelein, R. 1991. Pulmonary tissue access of ultrafine particles. *J. Aerosol Med.* 4, 57-68.

Ferron, G.A. 1977. The size of soluble aerosol particles as a function of the humidity of the air. Application to the human respiratory tract. *J. Aerosol Sci.* 8, 251-267.

Ferron, G.A., Karg, E. and Peter, J.E. 1993. Estimation of deposition of polydisperse hygroscopic aerosols in the human respiratory tract. *J. Aerosol Sci.* 24, 655-670.

Filipy, R.E., Pappin, J.L., Stevens, D.L. and Irby, S.G. 1980. Effects of Cigarette Smoke Exposure on Pulmonary Clearance of $^{239}\text{PuO}_2$ in Rats. Pacific Northwest Laboratory Annual Report for 1979 to the DOE Assistant Secretary for Environment. PNL-3300 Part 1: Biomedical Sciences, Richland, Washington. Available from National Technical Information Service, Springfield, Virginia, pp. 128-130.

Filipy, R.E., Pappin, J.L., Stevens, D.L. and Bair, W.J. 1981a. The impairment of pulmonary clearance of $^{239}\text{PuO}_2$ in rats by prolonged exposure to cigarette smoke. Pacific Northwest Laboratory Annual Report for 1980 to the DOE Assistant Secretary for Environment. PNL-3700 Part 1: Biomedical Sciences, Richland, Washington. Available from National Technical Information Service, Springfield, Virginia, pp. 110-111.

Filipy, R.E., Pappin, J.L., Stevens, D.L. and Bair, W.J. 1981b. The effect of cigarette smoke exposure on clearance of $^{239}\text{PuO}_2$ from the lungs of beagle dogs. Pacific Northwest Laboratory Annual Report for 1980 to the DOE Assistant Secretary for Environment. PNL-3700 Part 1: Biomedical Sciences, Richland, Washington. Available from National Technical Information Service, Springfield, Virginia, pp. 107-109.

Filipy, R.E., Bair, W.J., Pappin, J.L. and Stevens D.L. 1982. Cigarette smoke and plutonium. Pacific Northwest Laboratory Annual Report for 1981 to the DOE Office of Energy Research. PNL-4100 Part 1: Biomedical Sciences, Richland, Washington. Available from National Technical Information Service, Springfield, Virginia, pp. 95-97.

Filipy, R.E., Bair, W.J. and Buschbom, R.L. 1985. Cigarette smoke and plutonium. Pacific Northwest Laboratory Annual Report for 1984 to the DOE Office of Energy Research. PNL-5500 Part 1: Biomedical Sciences, Richland, Washington. Available from National Technical Information Service, Springfield, Virginia, pp. 31-33.

Filipy, R.E., Bair, W.J. and Buschbom, R.L. 1986. Cigarette smoke and plutonium. Pacific Northwest Laboratory Annual Report for 1985 to the DOE Office of Energy Research. PNL-5750 Part 1: Biomedical Sciences, Richland, Washington. Available from National Technical Information Service, Springfield, Virginia, pp. 27-30.

Fisenne, I.M. and Welford, G.A. 1986. Natural U concentrations in soft tissues and bone of New York City residents. *Health Phys.* 50, 739-746.

Fish, B.R., Bernard, S.R., Royster, G.W. Jr., Farabee, L.B., Brown, P.E. and Patterson, G.R. Jr. 1967. Applied internal dosimetry. In: *Health Physics Division Annual Progress Report Period Ending 31 July 1964*. pp. 222-226. Oak Ridge National Laboratory (ORNL 3697).

Fleisher, R.L. and Raabe, O.G. 1977. Fragmentation of respirable PuO_2 in water - a mode of dissolution. *Health Phys.* 32, 253-262.

Fleming, R.R. and Hall, R.M. 1978. Two ^{238}Pu inhalation incidents. Du Pont Savannah River Plant Report. DPSPU 78-30-4 pp. 1-9.

Foord, N., Black, A. and Walsh M. 1977. Pulmonary Deposition of Inhaled Particles with Diameters in the Range 2.5 to 7.5 mm. In: *Inhaled Particles IV* (in two parts), Part 2, *Proceedings of an International Symposium Organized by the British Occupational Hygiene Society, Edinburgh, 22-26 September 1975*, (Eds. Walton, W.H. and McGovern, B.), Pergamon Press, Oxford, United Kingdom. pp. 137-149.

Foster, W.M. 1988. Is 24 hour lung retention an index of alveolar deposition? *J. Aerosol Med.* 1, 1-9.

Foster, P.P. 1991. Study of a plutonium oxide fuel inhalation case. *Radiat. Prot. Dosim.* 38, 141-146.

Foster, W.M., Langenback, E. and Bergofsky, E.H. 1980. Measurement of tracheal and bronchial mucus velocities in man: Relation to lung clearance. *J. Appl. Physiol: Respir., Environ. Exercise Physiol.* 48, 965-971.

Foster, W.M., Langenback, E.G. and Bergofsky, E.H. 1982. Lung mucociliary function in man: interdependence of bronchial and tracheal mucus transport velocities with lung clearance in bronchial asthma and healthy subjects. In: *Inhaled Particles V, Proceedings of an International Symposium Organized by the British Occupational Hygiene Society, Cardiff, 8-12 September 1980*, (Eds.

Walton, W.H., Critchlow, A. and Coppock, S.M.), Pergamon Press, Oxford, United Kingdom. *Ann. Occup. Hyg.* 26, 227-244.

Foster, W.M., Langenback, E.G. and Bergofsky, E.H. 1985. Disassociation in the mucociliary function of central and peripheral airways of asymptomatic smokers. *Am. Rev. Respir. Dis.* 132, 633-639.

Foster, P.P., Pearman, I. and Ramsden, D. 1989. An interspecies comparison of the lung clearance of inhaled monodisperse cobalt oxide particles - Part II: Lung clearance of inhaled cobalt oxide in man. *J. Aerosol Sci.* 20, 189-204.

Freedman, P.A., Robinson, S.E. and Street, M.R. 1988. Magnetopneumographic study of human alveolar clearance in health and disease. In: *Inhaled Particles VI*, Proceedings of an International Symposium and Workshop on Lung Dosimetry Organized by the British Occupational Hygiene Society in Co-operation with the Commission of the European Communities, Cambridge, 2-6 September 1985, (Eds. Dodgson, J., McCallum, R.I., Bailey, M.R., and Fisher, D.R.), Pergamon Press, Oxford, United Kingdom. *Ann. Occup. Hyg.* 32 (Suppl. 1), 809-820.

Friedman, P.J. and Tisi, G.M. 1972. "Alveolarization" of tantalum powder in experimental bronchography and the clearance of inhaled particles from the lung. *Radiology* 104, 523-535.

Friedman, M., Stott, F.D., Poole, D.O., Dougherty, R., Chapman, G.A., Watson, H. and Sackner, M.A. 1977. A new roentgenographic method for estimating mucous velocity in airways. *Am. Rev. Respir. Dis.* 115, 67-72.

Fry, F.A. and Black, A. 1973. Regional deposition and clearance of particles in the human nose. *J. Aerosol Sci.* 4, 113-124.

Fujita, M., Yabe, A., Akaishi, J. and Ohtani, S. 1966. Relationship between ingestion, excretion and accumulation of fallout caesium-137 in man on a long time scale. *Health Phys.* 12, 1649-1653.

Furchner, J.E., Richmond, C.R., Drake, G.A. 1971. Comparative metabolism of radionuclides in mammals - VII. Retention of ^{106}Ru in the mouse, rat, monkey and dog. *Health Phys.* 21, 355-365.

Gamsu, G., Weintraub, R.M. and Nadel, J.A. 1973. Clearance of tantalum from airways of different caliber in man evaluated by a roentgenographic method. *Am. Rev. Resp. Dis.* 107, 214-224.

Gehr, P., Schürch, S., Berthiaume, Y., Im Hof, V. and Geiser, M. 1990a. Particle retention in airways by surfactant. *J. Aerosol Med.* 3, 27-43.

Gehr, P., Schürch, S., Geiser, M. and Im Hof, V. 1990b. Retention and clearance mechanisms of inhaled particles. *J. Aerosol Sci.* 21 (Suppl. 1), S491-S496.

Geiser, M., Im Hof, V., Gehr, P. and Cruz-Orive, L.M. 1990a. Histological and stereological analysis of particle retention in the conducting airways of hamster lungs. *J. Aerosol Med.* 3, 131-145.

Geiser, M., Cruz-Orive, L.M., Im Hof, V. and Gehr, P. 1990b. Assessment of particle retention and clearance in the intrapulmonary conducting airways of hamster lungs with the fractionator. *J. Microsc.* 160, 75-88.

Gerrard, C.S., Levandowski, R.A., Gerrity, T.R., Yeates, D.B. and Klein, E. 1985. The effects of acute respiratory virus infection upon tracheal mucous transport. *Arch. Environ. Health* 40, 322-325.

Gerrard, C.S., Gerrity, T.R. and Yeates, D.B. 1986. The relationships of aerosol deposition, lung size, and the rate of mucociliary clearance. *Arch. Environ. Health* 41, 11-15.

Gerrity, T.R., Cotromanes, E., Garrard, C.S., Yeates, D.B. and Lourenço, R.V. 1983. The effect of aspirin on lung mucociliary clearance. *N. Engl. J. Med.* 308, 139-141.

Gillett, A.M., Harmsen, A.B. and Stegelmeier, B. 1989. Translocation of particles by macrophages from lung to lymph nodes in rats. *J. Aerosol Med.* 2, 29-39.

Glenn, R.D., Heid, K.R. and Houston, J.R. 1979. Assessments of a cerium-praseodymium-144 inhalation case. *Health Phys.* 36, 117-125.

Godfrey, S., Davies, C.T.M., Wozniak, E. and Barnes, C.A. 1971. Cardio-respiratory response to exercise in normal children. *Clin. Sci.* 40, 419-431.

Gongora, R., Gongora, G., Roy, M., Dautzenberg, B., Becquemin, M.H., Sors, C., Gamain, B., Teillac, A. and Gaillard, J.P. 1983. Particulate lung clearance in sarcoidosis. In: *Sarcoidosis and Other Granulomatosis Disorders: Ninth International Conference, Paris, France, August 31-September 4, 1981*. (Eds. Chrétien, J., Marsac, J. and Saltiel, J.C.). Pergamon Press, Oxford, United Kingdom, pp. 284-287.

Goodman, R.M., Yergin, B.M., Landa, J.F., Golinvaux, M.H. and Sackner, M.A. 1977. Tracheal mucous velocity (TMV) in non-smokers, smokers, and patients with obstructive lung disease. *Fed. Proc.* 36, 607.

Goodman, R.M., Yergin, B.M., Landa, J.F., Golinvaux, M.H. and Sackner, M.A. 1978. Relationship of smoking history and pulmonary function tests to tracheal mucous velocity in nonsmokers, young smokers, ex-smokers, and patients with chronic bronchitis. *Am. Rev. Respir. Dis.* 117, 205-214.

Gore, D.J. 1983. The spatial and temporal distribution of inhaled UO_2 particles in the respiratory tract of the rat. II. The relative concentration of UO_2 between the intrapulmonary airways and the pulmonary tissue. *Radiat. Res.* 93, 276-287.

Gore, D.J. and Patrick, G. 1978. The distribution and clearance of inhaled UO_2 particles on the first bifurcation and trachea of rats. *Phys. Med. Biol.* 23, 730-737.

Gore, D.J. and Patrick, G. 1982. A quantitative study of the penetration of insoluble particles into the tissue of the conducting airways. In: *Inhaled Particles V*, Proceedings of an International Symposium Organized by the British Occupational Hygiene Society, Cardiff, 8-12 September 1980, (Eds. Walton, W.H., Critchlow, A. and Coppock, S.M.), Pergamon Press, Oxford, United Kingdom. *Ann. Occup. Hyg.* 26, 149-161.

Green, G.M., Jakab, G.J., Low, R.B. and Davis, G.S. 1977. Defense mechanisms of the respiratory membrane. *Am. Rev. Respir. Dis.* 115, 479-514.

Griffith, W.C., Mewhinney, J.A., Muggenburg, B.A., Boecker, B.B. and Cuddihy, R.G. 1983. Bioassay model for estimating body burdens of ^{241}Am from excretion analyses. *Health Phys.* 44, Suppl. 1, 545-554.

Guilmette, R.A., Diel, J.H., Muggenburg, B.A., Mewhinney, J.A., Boecker, B.B. and McClellan, R.O. 1984. Biokinetics of inhaled $^{239}PuO_2$ in the Beagle dog: Effect of aerosol particle size. *Int. J. Radiat. Biol.* 45, 563-581.

Guilmette, R.A., Muggenburg, B.A. and Diel, J.H. 1986. Biokinetics of ^{239}Pu in immature dogs that inhaled $^{239}PuO_2$. Inhalation Toxicology Research Institute Annual Report 1985-86, LMF-115. Lovelace Biomedical & Environmental Research Foundation, Albuquerque, New Mexico, Available from National Technical Information Service, Springfield, Virginia, pp. 105-108.

Guilmette, R.A., Boecker, B.B., Muggenburg, B.A., Hahn, F.F. and McClellan, R.O. 1987a. Age-related effects on the disposition and dosimetry of inhaled ^{239}Pu or ^{144}Ce in immature or aged beagle dogs. In: *Age-related Factors in Radiouclide Metabolism and Dosimetry*. (Eds. Gerber, G.B., Métivier, H. and Smith, H.) pp. 109-120. Martinus Nijhoff Publishers, Dordrecht.

Guilmette, R.A., Muggenburg, B.A., Hahn, F.F., Mewhinney, J.A., Seiler, F.A., Boecker, B.B. and McClellan, R.O. 1987b. Dosimetry of ^{239}Pu in dogs that inhaled monodisperse aerosols of $^{239}PuO_2$. *Radiat. Res.* 110, 199-218.

Guilmette, R.A., Griffith, W.C. and Hickman, A.W. 1994. Intake assessment for workers that inhaled ^{238}Pu aerosols. *Radiat. Prot. Dosim.* 53, 127-131.

Hackett, P.L., Sikov, M.R., Skiens, W.E., Cannon, W.C., Hess, J.O. and Hall, D. 1980. Interactions of animal age and particle size with deposition and retention of inhaled ^{51}Cr -labelled microspheres. Pacific Northwest Laboratory Annual Report for 1979 to the DOE Assistant Secretary for Environment. PNL-3300 Part 1: Biomedical Sciences, Richland, Washington. Available from National Technical Information Service, Springfield, Virginia, pp. 186-188.

Hackett, P.L., Sikov, M.R., Skiens, W.E., Cannon, W.C. and Music, R.L. 1981. Interactions of animal age and particle size with deposition and retention of inhaled ^{51}Cr -labelled microspheres. Pacific Northwest Laboratory Annual Report for 1980 to the DOE Assistant Secretary for Environment. PNL-3700 Part 1: Biomedical Sciences, Richland, Washington. Available from National Technical Information Service, Springfield, Virginia, pp. 160-163.

Hahn, F.F., Newton, G.J. and Bryant, P.L. 1977. In vitro phagocytosis of respirable-sized monodisperse particles by alveolar macrophages. In: *Pulmonary Macrophage and Epithelial Cells*. (Eds. Sanders, C.L., Schneider, R.P., Dagle, G.E. and Ragan, H.A.). Available from National Technical Information Service, Springfield, Virginia, pp. 424-435.

Hänel, G. and Heyder, J. 1980. Deposition of hygroscopic atmospheric aerosol particles in the human respiratory tract. *Staub-Reinhalt. Luft* 40, 9-13.

Harmsen, A.G., Muggenburg, B.A., Snipes, M.B. and Bice, D.E. 1985. The role of macrophages in particle translocation from lungs to lymph nodes. *Science* 230, 1277-1280.

Harrison, J.D. 1983. The gastrointestinal absorption of plutonium, americium and curium. *Radiat. Prot. Dosim.* 5, 19-35.

Harrison, J.D. 1991. The gastrointestinal absorption of the actinide elements. *Science Total Environ.* 100, 43-60.

Harrison, J. and McNeill, K.G. 1964. The effect of chlorothiazide on caesium-137 excretion in human

subjects. In: *Assessment of Radioactivity in Man*, Vol. II, pp. 89–96. IAEA, Vienna.

Hennacy, R.A. 1961. Translocation and inhalation of $\text{Ce}^{144}\text{O}_2$. In: *Hanford Biology Research Annual Report for 1960*, pp. 77–80. Washington (HW-69500).

Henrichs, K., Paretzke, H.G., Voight, G. and Berg, D. 1989. Measurement of Cs absorption and retention in man. *Health Phys.* 57, 571–578.

Henshaw, D.L. and Fews, A.P. 1983. Measurements of the Microdistribution of Alpha Active Nuclei in the Human Lung. In: *Current Concepts in Lung Dosimetry*. (Ed. Fisher, D.R.) Technical Information Center, Oak Ridge, Tennessee. Available from National Technical Information Service, Springfield, Virginia, pp. 127–137.

Henshaw, D.L., Fews, A.P., Maharaj, R. and Shepherd, L. 1988. Autopsy studies of the microdistribution of μ -active nuclides in lung tissue. In: *Inhaled Particles VI, Proceedings of an International Symposium and Workshop on Lung Dosimetry Organized by the British Occupational Hygiene Society in Co-operation with the Commission of the European Communities, Cambridge, 2-6 September 1985*, (Eds. Dodgson, J., McCallum, R.I., Bailey, M.R. and Fisher, D.R.), Pergamon Press, Oxford, United Kingdom. *Ann. Occup. Hyg.* 26, 1081–1094.

Hesp, R. 1964. The retention and excretion of caesium-137 by two male subjects. In: *Assessment of Radioactivity in Man*, Vol. II, pp. 61–74. IAEA, Vienna.

Hesp, R. and Coote, J. 1970. Body radioactivity studies on a series of cases in which ruthenium-106 oxide was inhaled. UKAEA PG Report 979 (W) United Kingdom Atomic Energy Authority, UK.

Heyder, J. and Rudolf, G. 1984. Mathematical models of particle deposition in the human respiratory tract. In: *Lung modeling for Inhalation of Radioactive Materials*. (Eds. Smith, H. and Gerber, G.), EUR 9384 EN, Commission of the European Communities, Luxembourg, pp. 17–38.

Heyder, J., Gebhart, J. and Stahlhofen, W. 1980. Inhalation of aerosols: Particle deposition and retention. In: *Generation of Aerosols*. (Ed. Willeke, K.) Ann Arbor Sci., Ann Arbor, Michigan, pp. 65–103.

Hickman, A.W., Griffith, W.C., Roessler, G.S. and Guilmette, R.A. 1995. Application of a canine ^{238}Pu biokinetics/dosimetry model to human bioassay data. *Health Phys.* 68, 359–370.

Hicks, J.F., Pritchard, J.N., Black, A. and Megaw, W.J. 1986. Experimental evaluation of aerosol growth in the human respiratory tract. In: *Aerosols: Formation and Reactivity*. Pergamon Press, Oxford, United Kingdom, pp. 244–247.

Hilding, A.C. 1932. The physiology of drainage of nasal mucus. *I. Arch. Otolaryngol.* 15, 92–100.

Hilding, A.C. 1957a. Ciliary streaming in the bronchial tree and the time element in carcinogenesis. *N. Engl. J. Med.* 256, 634–640.

Hilding, A.C. 1957b. Ciliary streaming in the lower respiratory tract. *Am. J. Physiol.* 191, 404–410.

Hilding, A.C. 1959. Ciliary streaming through the larynx and trachea. *J. Thorac. Cardiovas. Surg.* 37, 108–117.

Hilding, A.C. 1963. Phagocytosis, mucous flow and ciliary action. *Arch. Environ. Health* 6, 61–73.

Hiller, F.C. 1991. Health implications of hygroscopic particle growth in the human respiratory tract. *J. Aerosol Medicine* 4, 1–23.

Hinds, W.C. 1982. *Aerosol Technology: Properties, Behavior, and Measurement of Airborne Particles*. John Wiley & Sons, New York.

Howells, H., Ward, F.A., Coulston, D.J. and Woodhouse, J.A. 1977. In-vivo measurement and dosimetry of ruthenium-106 oxide in the lung. In: *Handling of radiation accidents. Proceedings of a Symposium, Vienna, March 1977*, pp. 83–100. International Atomic Energy Authority, Vienna.

Hühnerbein, J., Otto, J. and Thal, W. 1984. Untersuchungsergebnisse der mukoziliären clearance bei lungengesunden kindern. *Padiatr. Grenzgeb.* 23, 437–443.

Hunt, G.J., Leonard, D.R.P. and Lovett, M.B. 1986. Transfer of environmental plutonium and americium across the human gut. *Sci. Total Environ.* 53, 89–109.

Hunt, G.J., Leonard, D.R.P. and Lovett, M.B. 1990. Transfer of environmental plutonium and americium across the human gut: A second study. *Sci. Total Environ.* 90, 273–282.

ICRP. 1972. *The Metabolism of Compounds of Plutonium and Other Actinides*. ICRP Publication 19. Pergamon Press, Oxford.

ICRP. 1975. *Report of the Task Group on Reference Man*, ICRP Publication 23. Pergamon Press, Oxford.

ICRP. 1979. *Limits on Intakes of Radionuclides for Workers*. ICRP Publication 30, Part 1. *Annals of the ICRP* 2 (3/4). Pergamon Press, Oxford.

ICRP. 1980. Limits for Intakes of Radionuclides by Workers. ICRP Publication 30, Part 2. Annals of the ICRP, 4 (3/4). Pergamon Press, Oxford.

ICRP. 1986. The Metabolism of Plutonium and Related Elements, ICRP Publication 48. Annals of the ICRP, 16 (2/3). Pergamon Press, Oxford.

ICRP. 1987. Lung Cancer Risk from Indoor Exposures to Radon Daughters. ICRP Publication 50. Annals of the ICRP, 17(1). Pergamon Press, Oxford.

ICRP. 1988. Limits for Intakes of Radionuclides by Workers. ICRP Publication 30 Part 4. Annals of the ICRP, 19(4). Pergamon Press, Oxford.

ICRP. 1989. Age-dependent Doses to Members of the Public from Intake of Radionuclides. Part 1. ICRP Publication 56. Annals of the ICRP 20 (2). Pergamon Press, Oxford.

ICRP. 1993. Age-dependent Doses to Members of the Public from Intake of Radionuclides. Part 2, Ingestion Dose Coefficients. ICRP Publication 67. Annals of the ICRP 23, (3/4). Pergamon Press, Oxford.

ICRP. 1994a. Human Respiratory Tract Model for Radiological Protection, ICRP Publication 66. Annals of the ICRP 24 (1-3). Elsevier Science Ltd., Oxford.

ICRP. 1994b. Dose Coefficients for Intakes of Radionuclides by Workers, ICRP Publication 68. Annals of the ICRP 24 (4). Elsevier Science Ltd., Oxford.

ICRP. 1996. Age-dependent Doses to Members of the Public from Intake of Radionuclides: Part 4 Inhalation Dose Coefficients. ICRP Publication 71. Elsevier Science Ltd., Oxford (in press).

Inuma, T., Watari, K., Nagai, T., Iwashima, K. and Yamagata, N. 1967. Comparative studies of Cs-132 and Pb-86 turnover in man using a double-tracer method. *J. Radiat. Res.* 8, 100-115.

Inaba, J. and Lengemann, F.W. 1972. Intestinal uptake and whole-body retention of ¹⁴¹Ce by suckling rats. *Health Phys.* 22, 169-175.

INSEE. 1988. Contours et Caractères. Les jeunes de 15 à 24 ans. Institut National de Statistique et Etudes Economiques, Paris, France.

INSEE. 1989a. Roy, C. La gestion du temps des hommes et des femmes, des actifs et des inactifs. Institut National de Statistique et Etudes Economiques, Economie et Statistiques, n 223, Paris, France.

INSEE. 1989b. Galland, O. La vie quotidienne des jeunes du lycée au mariage. Institut National de Statistique et Etudes Economiques, Economie et Statistiques n 223, Paris, France.

INSEE. 1989c. Garrigues, P. Une France un peu plus sportive qu'il y a 20 ans, grâce aux femmes. Institut National de Statistique et Etudes Economiques, Economie et Statistiques, n 224, Paris, France.

INSEE. 1989d. Arnal, N., Dumontier, F. et Paire, R. Les Téléspectateurs. Institut National de Statistique et Etudes Economiques, Economie et Statistiques n 227, Paris, France.

Irango, E., Salvador, S. and Irango, C.E. 1987. Air concentrations of ²³⁹Pu and ²⁴⁰Pu and potential radiation doses to persons living near Pu-contaminated areas in Palomares, Spain. *Health Phys.* 52, 453-461.

Iravani, J. and van As, A. 1972. Mucus transport in the tracheobronchial tree of normal and bronchitic rats. *J. Pathol.* 106, 81-93.

Isawa, T., Teshima, T., Hirano, T., Ebina, A. and Konno, K. 1984. Mucociliary clearance mechanism in smoking and nonsmoking normal subjects. *J. Nucl. Med.* 25, 352-359.

James, A.C., Birchall, A., Cross, F.T., Cuddihy, R.G. and Johnson, J.R. 1989. The current approach of the ICRP Task Group for modeling doses to respiratory tract tissues. *Health Phys.* 57, (Suppl. 1) 271-282.

James, A.C., Stahlhofen, W., Rudolf, G., Köbrich, R., Briant, J.K., Egan, M., Nixon, W. and Birchall, A. 1994. Annex D. Deposition of Inhaled Particles. In Human Respiratory Tract Model for Radiological Protection, ICRP Publication 66. Annals of the ICRP 24 (1-3). Elsevier Science Ltd., Oxford pp. 231-299.

Jammet, H., Drutel, P., Parrot, R. and Roy, M. 1978. Etude de l'épuration pulmonaire chez l'homme après administration d'aérosols de particules radioactives. *Radioprotection DUNOD*, 13, 143-166.

Jarvis, N.S., Birchall, A., James, A.C., Bailey, M.R. and Dorrian, M.-D. 1993. LUDEP 1.0 Personal computer program for calculating internal doses using the new ICRP respiratory tract model. Chilton, NRPB-SR264.

Johnson, J.R., Dunford, D.W. and Kramer, G.H. 1983. Summary of a strontium-89 contamination case. *Radiat. Prot. Dosim.* 5, 247-249.

Johnson, T., Capel, J., McCoy, M. and Mozier, J.W. 1995. Estimation of ozone exposures experienced by outdoor workers in nine urban areas using a probabilistic version

of NEM. Contract No. 63-D-30094, report JTN 453207-1. US Environmental Protection Agency, Office of Air Quality and Standards, Research Triangle Park, North Carolina 27711, USA.

Jordan, R.D. 1964. Caesium-137 chloride retention following accidental ingestion. In: Assessment of Radioactivity in Man. Vol. II, pp. 103-111. IAEA, Vienna.

Kalkwarf, D.R., Jackson, P.O. and Hardin, J.M. 1984. Lung-clearance classification of radionuclides in coal fly ash. *Health Phys.* 47, 37-45.

Kalliomäki, P.-L., Korhonen, O., Vaaranen, V., Kalliomäki, K. and Koponen, M. 1978. Lung retention and clearance of shipyard arc welders. *Int. Arch. Occup. Environ. Health* 42, 83-90.

Kalliomäki, P.-L., Kalliomäki, K., Rahkonen, E. and Aittomäki, K. 1983. Follow-up study on the lung retention of welding fumes among shipyard welders. *Ann. Occup. Hyg.* 27, 449-452.

Kalliomäki, P.-L., Kalliomäki, K., Rahkonen, E. and Juntilla, M.L. 1985. Magnetopneumography - lung retention and clearance of manual metal arc welding fumes based on experimental and human data. In: Proceedings of the Fifth World Conference on Biomagnetism. (Eds. Weinberg, H., Stroink, G. and Katila, T.) Pergamon Press, New York, pp. 416-421.

Kanapilly, G.M. and Goh, C.H.T. 1973. Some factors affecting the in vitro rates of dissolution of respirable particles of relatively low solubility. *Health Phys.* 25, 225-237.

Kanapilly, G.M., Goh, C.H.T. and Chimenti, R.A. 1973. Measurement of in vitro dissolution of aerosol particles. *Health Phys.* 24, 497-507.

Kanapilly, G.A., Stanley, J.A., Newton, G.J., Wong, B.A. and DeNee, P.B. 1980. Characterization of an aerosol sample from Three Mile Island reactor auxiliary building. Inhalation Toxicology Research Institute Annual Report 1979-1980, LMF-84, pp. 5-9. Lovelace Biomedical and Environmental Research Foundation, Albuquerque, New Mexico. Available from National Technical Information Service, Springfield, Virginia.

Kärjä, J., Nuutinen, J. and Karjalainen, P. 1982. Radioisotopic method for measurement of nasal mucociliary activity. *Arch. Otolaryngol.* 108, 99-101.

Kathren, R.L., Strom, D.J., Sanders, C.L., Filipy, R.E., McInroy, J.F. and Bistline, R.E. 1993. Distribution of plutonium and americium in human lungs and lymph nodes and relationship to smoking status. *Radiat. Prot. Dosim.* 48, 307-315.

Katz, I., Zwas, T., Baum, G.L., Aharonson, E. and Belfer, B. 1987. Ciliary beat frequency and mucociliary clearance: What is the relationship? *Chest* 92, 491-493.

Keane, A.T. and Polednak, A.P. 1983. Retention of uranium in the chest: Implications of findings in vivo and postmortem. *Health Phys.* 44 (Suppl. 1), 391-402.

Keating, F.R. and Albert, A. 1949. The metabolism of iodine in man as disclosed with the use of radioiodine. *Recent Prog. Hormone Research* 4, 429.

Kistner, G., Wiggenhauser, A., Krestel, R., Buheitel, G., Erzberger, A. and Roedler, H.D. 1987. Age-related biokinetics of cerium-141 chloride in rats. In: Age-related Factors in Radionuclide Metabolism and Dosimetry (Eds. Gerber, G.B., Métivier, H. and Smith, H.) pp. 221-228. Martinus Nijhoff Publishers, Dordrecht.

Kreyling, W.G. 1990. Interspecies comparison of lung clearance of "insoluble" particles. *J. Aerosol Med.* 3 (Suppl. 1), S93-S110.

Kreyling, W.G., Schumann, G., Ortmaier, A., Ferron, G.A. and Karg, E. 1988. Particle transport from the lower respiratory tract. *J. Aerosol Med.* 1, 351-369.

Kron, T., Hansen, C. and Werner, E. 1991. Renal excretion of tellurium after peroral administration of tellurium in different forms to healthy human volunteers. *J. Trace. Elem. Electrolytes Health Dis.* 5, 239-244.

LaBauve, R.J., Brooks, A.L., McClellan, R.O. and Mead, D.K. 1980. Cytogenetic and other biological effects of $^{239}\text{PuO}_2$ inhaled by the rhesus monkey. *Radiat. Res.* 82, 310-335.

Lang, S., Kosma, V.M., Kumlin, T., Halinen, A., Salonen, R.O., Servomaa, K., Rytömaa, T. and Ruuskanen, J. 1994. Distribution and short-term effects of intratracheally instilled neutron-irradiated UO_2 particles in the rat. *Env. Res.* 15, 119-131.

Larsen, R.P., Bhattacharyya, M.H., Oldham, R.D., Moretti, E.S. and Spaletto, M.I. 1981. Continued studies of the gastrointestinal absorption of plutonium in rodents. In: Annual Report for Radiological and Environmental Research Division, Argonne National Laboratory, Illinois. ANL-81-85 (Pt. II) pp. 105-116.

Lataillade, G., Verry, M., Rateau, G., Métivier, H. and Masse, R. 1995. Translocation of plutonium from rat and monkey lung after inhalation of industrial plutonium

oxide and mixed uranium and plutonium oxide. *Int. J. Radiat. Biol.* 67, 373-380.

Lauweryns, J.M. and Baert, J.H. 1977. Alveolar clearance and the role of the pulmonary lymphatics. *Am. Rev. Respir. Dis.* 115, 625-683.

Lay, J.C., Berry, C.R., Kim, C.S. and Bennett, W.D. 1995. Bronchial retention of soluble and insoluble materials in dogs. *J. Aerosol Med.* 8, 84.

Layton, D.W. 1993. Metabolically consistent breathing rates for use in dose assessments. *Health Phys.* 64, 23-26.

Layton, D.W. 1994. Response to Bertholon and Roy. *Health Phys.* 66, 89-90.

Le Bouffant, L. 1971. Influence de la nature des poussières et de la charge pulmonaire sur l'épuration. In: *Inhaled Particles III*, Vol. I, Proceedings of an International Symposium Organized by the British Occupational Hygiene Society in London, 14-23 September 1970, (Ed. Walton, W.H.), Unwin Brothers Limited, Old Woking Surrey, England, pp. 227-237.

Le Bouffant, L. and Henin, J.P. 1974. Measure of pulmonary clearance in man with tagged iron oxides. *INSERM*, 29, 193-204.

Le Bouffant, L., Henin, J.P., Martin, J.C. and Daniel, H. 1972. Etude expérimentale de l'épuration pulmonaire. *Lille Med.* 17, 1091-1101.

Leak, L.V. 1977. Pulmonary lymphatics and their role in the removal of interstitial fluids and particulate matter. In: *Respiratory Defense Mechanisms*. (Eds. Brain, J.D., Proctor, D.F. and Reid, L.) Marcel Dekker, New York, pp. 631-685.

Leak, L.V. 1980. Lymphatic removal of fluids and particles in the mammalian lung. *Environ. Health Perspect.* 35, 55-76.

Lebedev, O.V. and Yakovlev, V.A. 1993. The correlation between ^{137}Cs half-time and age, body mass and height in individuals contaminated from the Chernobyl accident. In: *The Chernobyl Papers* (Eds. Merwin, S.E. and Balonov, M.I.), pp. 66-82.

Lee, P.S., Gerrity, T.R., Hass, F.J. and Lourenço, R.V. 1979. A model for tracheobronchial clearance of inhaled particles in man and a comparison with data. *IEEE Trans. Biomed. Eng.* 26, 624-630.

Leggett, R.W. 1986. Predicting the retention of Cs in individuals. *Health Phys.* 50, 747-759.

Leggett, R.W. and Warren, B.P. (Eds). 1987. Age-specific models for evaluating dose and risk from internal exposure to radionuclides: Report of current work of the Metabolism and Dosimetry Research Group. ORNL/TM-10080. Oak Ridge National Laboratory.

Lehnert, B.E. and Sanz-Rodrigues, C. 1988. Expression of Fcg receptor-mediated phagocytosis by airway intraluminal macrophages compared to alveolar macrophages. *Am. Rev. Respir. Dis.*, Suppl. 137, 86.

Lehnert, B.E., Valdez, Y.E. and Stewart, C.C. 1986. Translocation of particles to the tracheobronchial lymph nodes after lung deposition: Kinetics and particle-cell relationships. *Exp. Lung Res.* 10, 245-266.

Leikauf, G., Yeates, D.B., Wales, K.A., Spektor, D., Albert, R.E. and Lippmann, M. 1981. Effects of sulfuric acid aerosol on respiratory mechanics and mucociliary particle clearance in healthy nonsmoking adults. *Am. Ind. Hyg. Assoc. J.* 42, 273-282.

Leikauf, G.D., Spektor, D.M., Albert, R.E. and Lippmann, M. 1984. Dose-dependent effects of submicrometer sulfuric acid aerosol on particle clearance from ciliated human lung airways. *Am. Ind. Hyg. Assoc. J.* 5, 285-292.

Leith, D.E. 1968. Cough. *Phys. Ther.* 48, 439-447.

LeRoy, G., Rust, J. and Hasterlik, J. 1966. The consequences of ingestion by man of real and simulated fall-out. *Health Phys.* 12, 449-473.

Lie, R. 1964. Deposition and retention of ^{137}Cs in the rat following inhalation of the chloride and the nitrate. *Health Phys.* 10, 1071-1076.

Lippmann, M. 1969. Discussion. In: *Inhalation Carcinogenesis*. Proceedings of a Conference, Gatlinburg, Tennessee, October 8-11, 1969. (Eds. Hanna, Jr., M.G., Nettesheim, P. and Gilbert, J.R.). Available from National Technical Information Service, Springfield, Virginia, p. 116.

Lippmann, M. 1970. Discussion. In: *Inhaled Particles III*, Vol. I, Proceedings of an International Symposium Organized by the British Occupational Hygiene Society in London, 14-23 September 1970, (Ed. Walton, W.H.), Unwin Brothers Limited, Old Woking Surrey, England, p. 122.

Lippmann, M. and Albert, R.E. 1969. The effect of particle size on the regional deposition of inhaled aerosols in the human respiratory tract. *Am. Ind. Hyg. Assoc. J.* 30, 257-275.

Lippmann, M. and Schlesinger, R.B. 1984. Interspecies comparisons of particle deposition and mucociliary clearance in tracheobronchial airways. *J. Toxicol. Environ. Health* 13, 441-469.

Lippmann, M., Yeates, D.B. and Albert, R.E. 1980. Deposition, retention, and clearance of inhaled particles. *Br. J. Ind. Med.* 37, 337-362.

Little, J.B., Radford, E.P., Jr., McCombs, H.L. and Hunt, V.R. 1965. Distribution of Polonium 210 in pulmonary tissues of cigarette smokers. *N. Engl. J. Med.* 273, 1343-1351.

Lourenço, R.V., Klimek, M.F. and Borowski, C.J. 1971. Deposition and clearance of 2 m particles in the tracheobronchial tree of normal subjects - smokers and nonsmokers. *J. Clin. Invest.* 50, 1411-1420.

Luchtel, D.L. 1976. Ultrastructural observations on the mucous layer in pulmonary airways. *J. Cell Biol.* 70, 350a.

Luchtel, D.L. 1978. The mucous layer of the trachea and major bronchi of the rat. Scanning Electron Microscopy Vol II. SEM Inc. O'Hare IL, pp. 1089-1098.

Lundborg, M., Lind, B. and Camner, P. 1984. Ability of rabbit alveolar macrophages to dissolve metals. *Exp. Lung Res.* 7, 11-22.

Lundgren, D.L., McClellan, R.O., Thomas, R.L., Hahn, F.F. and Sanchez, A. 1974. Toxicity of inhaled $^{144}\text{CeO}_2$ in mice. *Radiat. Res.* 58, 448-461.

Lundgren, D.L., Hahn, F.F. and McClellan, R.O. 1980a. Influence of age at the time of inhalation exposure to aerosols of $^{144}\text{CeO}_2$ on the ^{144}Ce and retention, dosimetry and toxicity in mice. *Health Phys.* 38, 643-655.

Lundgren, D.L., McClellan, R.O., Hahn, F.F., Newton, G.J. and Diel, J.H. 1980b. Repeated inhalation exposure of mice to $^{144}\text{CeO}_2$. I. Retention and dosimetry. *Radiat. Res.* 82, 106-122.

Lundgren, D.L., Hahn, F.F., Diel, J.H. and Snipes, M.B. 1992. Repeated inhalation exposure of rats to aerosols of $^{144}\text{CeO}_2$. I. Lung, liver and skeletal dosimetry. *Radiat. Res.* 132, 312-324.

Malarbet, J.L., Bertholon, J.F., Becquemin, M.H., Taieb, G., Bouchikhi, A. and Roy, M. 1994. Oral and nasal flowrate partitioning in healthy subjects performing graded exercise. *Rad. Prot. Dosim.* 53, 179-182.

Martonen, T.B., Bell, K.A., Phalen, R.F., Wilson, A.F. and Ho, A. 1982. Growth rate measurements and deposition modelling of hygroscopic aerosols in human tracheobronchial models. In: *Inhaled Particles V, Proceedings of an International Symposium Organised by the British Occupational Hygiene Society, Cardiff, 8-12 September 1980* (Eds. Walton, W.H., Critchlow, A. and Coppock, S.M.), Pergamon Press, Oxford, United Kingdom. *Ann. Occup. Hyg.* 26, 93-108.

Masse, R., Fritsch, P., Ducouso, R., Lafuma, J. and Chrétien, J. 1973. Retention de particules dans les cellules bronchiques, relations possibles avec les carcinogènes inhalés. *C.R. Acad. Sci. Paris* 276, Series D 2923-2925.

Masse, R., Ducouso, R., Nolibé, D., Lafuma, J. and Chrétien, J. 1974. Passage transbronchique des particules métalliques. *Rev. Fr. Mal. Respir.* 2, Suppl. 1, 123-127.

Matthys, H., Köhler, D. and Würtemberger, G. 1987. Deposition of aerosols and bronchial clearance measurements. *Eur. J. Nucl. Med.* 13, S53-S57.

McAughey, J.J., Vernon, L., Haines, J., Sanders, T. and Clark, R. 1994. Fractional gut absorption of strontium, barium and neodymium following administration of stable isotope tracers. In: *Proc. 9th Int. Conf. on Heavy Metals in the Environment*. Toronto, 1993.

McClellan, R.O. and Bustad, L.K. 1964. Gastrointestinal absorption of ^{85}Sr titanate. In: *Hanford Biology Research Annual Report for 1963*. Washington (HW 80500). pp. 100-101.

McClellan, R.O. and Rupprecht, F.C., Eds. 1968. Radioiodine metabolism in the beagle dog - The importance of age and mode of ^{131}I exposure. *Fission Product Inhalation Program Annual Report 1967-1968*, LF-39, pp. 122-127. Lovelace Foundation for Medical Education and Research, Albuquerque, New Mexico. Available from National Technical Information Service, Springfield, Virginia.

McClellan, R.O., Barnes, J.E., Boecker, B.B., Cuddihy, R.G., Hobbs, C.H., Jones, R.K. and Redman, H.C. 1970. Some observations on the toxicity of beta emitting radionuclides inhaled in fused clay particles. *Fission Product Inhalation Program Annual Report, 1969-1970*, LF-43, pp. 197-204. Lovelace Foundation for Medical Education and Research, Albuquerque, New Mexico. Available from National Technical Information Service, Springfield, Virginia.

McInroy, J.F., Boyd, H.A. and Eustler, B.C. 1981. Deposition and retention of plutonium in the United States general population. In: *Actinides in Man and Animals, Proceedings of the Actinide Workshop*, Snowbird, Utah,

October 15-17, 1979. (Ed. Wrenn, M.E.) pp. 161-179. RD Press, Salt Lake City, Utah.

McInroy, J.F., Kathren, R.L. and Swint, M.J. 1989. Distribution of plutonium and americium in whole bodies donated to the United States Transuranium Registry. *Radiat. Prot. Dosim.* 26, 151-158.

McKenney, J.R., McClellan, R.O. and Bustad, L.K. 1961. Preliminary observations on Ce144-Pr144 in sheep. In: *Hanford Biology Research Annual Report for 1960*, p. 60. Washington (HW-69500).

Métivier, H., Masse, R., Nolibé, D. and Lafuma, J. 1977. Effect of time on the determination of the clearance rates of insoluble plutonium 239 oxide. *Health Phys.* 32, 447-449.

Métivier, H., Masse, R., Rateau, G. and Lafuma, J. 1980. Experimental study of respiratory contamination by a mixed oxide aerosol formed from the combustion of a plutonium magnesium alloy. *Health Phys.* 38, 769-776.

Métivier, H., Masse, R. and Lafuma, J. 1983. Metabolism of plutonium introduced as tri-N-butyl phosphate complex in the rat and removal attempts by DTPA. *Health Phys.* 44, 623-634.

Métivier, H., Madic, C., Bourges, J. and Masse, R. 1985. Valency five, similarities between plutonium and neptunium in gastrointestinal uptake. In: *Speciation of Fission and Activation Products in the Environment*. (Eds. Bulman, R.A. and Cooper, J.R.) Elsevier Applied Science Publishers, London. pp. 175-178.

Métivier, H., Piechowski, J., Duserre, C., Rateau, G., Legendre, N., Menoux, B. and Masse, R. 1989. Biological behaviour of plutonium inhaled by baboons as plutonium n-tributylphosphate complex. Comparison with ICRP models. *Radiat. Prot. Dosim.* 26, 287-292.

Mewhinney J.A. and Diel J.H. 1983. Retention of inhaled $^{238}\text{PuO}_2$ in beagles: A mechanistic approach to description. *Health Phys.* 45, 39-60.

Mewhinney, J.A. and Edison, A.F. 1983. Models of retention, distribution and excretion of Pu, Am and U by beagle dogs, cynomolgus monkeys and Fischer-344 rats following inhalation of industrial aerosols. In: *Radiation Dose Estimates and Hazard Evaluations for Inhaled Airborne Radionuclides*, Annual Progress Report July 1981-June 1982, US Nuclear Regulatory Commission document No. NUREG/CR-3313, pp. 21-32. Inhalation Toxicology Research Institute, Lovelace Biomedical & Environmental Research Institute, Albuquerque, New

Mexico. Available from National Technical Information Service, Springfield, Virginia.

Mewhinney, J.A. and Muggenburg, B.A. 1985. Comparison of retention of ^{241}Am in immature, young adult and aged dogs and in monkeys after inhalation of $^{241}\text{AmO}_2$. *Inhalation Toxicology Research Institute Annual Report 1984-1985*, LMF-114, pp. 248-353. Lovelace Biomedical and Environmental Research Institute, Albuquerque, New Mexico. Available from National Technical Information Service, Springfield, Virginia.

Mewhinney, J.A., Muggenburg, B.A., McClellan, R.O. and Miglio, J.J. 1976. The effect of varying physical and chemical characteristics of inhaled plutonium aerosols on metabolism and excretion. In: *Diagnosis and Treatment of Incorporated Radionuclides*, Proceedings of a Seminar held by the International Atomic Energy Agency and the World Health Organization, December 8-12, 1975, Vienna. pp. 87-97. International Atomic Energy Agency, Vienna, Austria.

Mewhinney, J.A., Eidson, A.F. and Wong, V.A. 1987. Effect of wet and dry cycles on dissolution of relatively insoluble particles containing Pu. *Health Phys.* 53, 377-384.

Mezey, R.J., Cohn, M.A., Fernandez, R.J., Januszkiewicz, A.J. and Wanner, A. 1978. Mucociliary transport in allergic patients with antigen-induced bronchospasm. *Am. Rev. Respir. Dis.* 118, 677-684.

Miettinen, J.K., Jokelainen, A., Roine, P., Liden, K. and Naverston, Y. 1963. ^{137}Cs and potassium in people and diet - a study of Finnish Laps. *Annales Academiae Scientiarum Fennicae. Series A. II. Chemica.* Keskushkirjapino, Helsinki.

Miller, C.E. 1964. Retention and distribution of ^{137}Cs after accidental inhalation. *Health Phys.* 10, 1065-1070.

Miller, F.W., Martonen, T.B., Ménache, M.G., Graham, R.C., Spektor, D.M. and Lippmann, M. 1988. Influence of breathing mode and activity level on regional deposition of inhaled particles and implications for regulatory standards. In: *Inhaled Particles VI*, Proceedings of an International Symposium and Workshop on Lung Dosimetry Organised by the British Occupational Hygiene Society in Co-operation with the Commission of the European Communities, Cambridge, 2-6 September 1985, (Eds. Dodgson, J., McCallum, R.I., Bailey, M.R. and Fisher, D.R.), Pergamon Press, Oxford, United Kingdom. *Ann. Occup. Hyg.* 32 (Suppl. 1), 3-10.

Möller, W. 1991. Untersuchung über das Verhalten von magnetischen Teilchen in der Lunge des Menschen mit einem supraleitenden SQUID-Magnetometer (PhD thesis). J.W. Goethe-Universität, Frankfurt am Main, Germany.

Monod, H. and Flandrois, R. 1985. Physiologie du sport, Masson, Ed., Paris.

Moody, J.C., Stradling, G.N. and Britcher, A. 1994. Biokinetics of three plutonium nitrate bearing materials: Implications for human exposure. *Radiat. Prot. Dosim.* 53, 169–172.

Morgan, D.J. and Morgan, A. 1967. Studies on the retention and metabolism of inhaled methyl iodide – I. Retention of inhaled methyl iodide. *Health Phys.* 13, 1055–1065.

Morgan, A., Morgan, D.J., Evans, J.C. and Lister, B.A.J. 1967a. Studies on the retention and metabolism of inhaled methyl iodide - II. Metabolism of methyl iodide. *Health Phys.* 13, 1067–1074.

Morgan, A., Morgan, D.J. and Arkell, G.M. 1967b. A study of the retention and subsequent metabolism of inhaled methyl iodide. In: *Inhaled Particles and Vapours II* (C.N. Davies, Ed.) Pergamon Press, Oxford pp. 309–321.

Morgan, A., Morgan D.J. and Black, A. 1968. A study of the deposition, translocation and excretion of radioiodine inhaled as iodine vapour. *Health Phys.* 15, 313–322.

Morgan, A., Black, A., Knight, D and Moores, S.R. 1988a. The effect of firing temperature on the lung retention and translocation of Pu following the inhalation of $^{238}\text{PuO}_2$ and $^{239}\text{PuO}_2$ by CBA/H mice. *Health Phys.* 54, 301–310.

Morgan, A., Black, A., Holmes, A. and Pratley, F.W. 1988b. Studies of environmental radioactivity in Cumbria. Part 14. Solubility in the rat lung of actinides associated with silt from the Ravenglass estuary and their translocation to liver and bone. AERE-R 12351. DOE Report DOE/RW/88.097.

Morgan, A., Holmes, A. and Pratley, F.W. 1990. Solubility in the rat lung of actinides associated with estuarine silt from West Cumbria. *J. Radiol. Prot.* 10, 135–142.

Morrow, P.E. 1969. Models for the study of particle retention and elimination in the lung. In: *Inhalation Carcinogenesis*. Proceedings of a Conference, Gatlinburg, Tennessee, October 8–11, 1969. (Eds. Hanna, Jr., M.G., Nettlesheim, P. and Gilbert, J.R.). Available from National Technical Information Service, Springfield, Virginia, pp. 103–119.

Morrow, P.E. 1972. Lymphatic drainage of the lung in dust clearance. *Ann. N.Y. Acad. Sci.* 200, 46–65.

Morrow, P.E. 1974. Theoretical and experimental models for dust deposition and retention. *Rev. Environ. Health* 1, 186–213.

Morrow, P.E. 1986. Factors determining hygroscopic aerosol deposition in airways. *Physiol. Review* 66, 330–376.

Morrow, P.E. 1988. Possible mechanisms to explain dust overloading of the lungs. *Fundam. Appl. Toxicol.* 10, 369–384.

Morrow, P.E., Gibb, F.R. and Gazioglu, K.M. 1967a. A study of particulate clearance from the human lungs. *Am. Rev. Respir. Dis.* 96, 1209–1221.

Morrow, P.E., Gibb, F.R. and Gazioglu, K. 1967b. The clearance of dust from the lower respiratory tract of man. An experimental study. In: *Inhaled Particles and Vapours II*, Proceedings of an International Symposium Organised by the British Occupational Hygiene Society, Cambridge, 28 September–1 October 1965, (Ed. Davies, C.N.), Pergamon Press, Oxford, pp. 351–359.

Morrow, P.E., Gibb, F.R., Davies, H. and Fisher, M. 1968. Dust removal from the lung parenchyma: an investigation of clearance stimulants. *Toxicol. and App. Pharmacol.* 12, 372–396.

Mortensen, J., Groth, S., Lange, P. and Rossing, N. 1989. Bronchoscintigraphic visualization of the acute effect of tobacco exposure and terbutaline on mucociliary clearance in smokers. *Eur. Respir. J.* 2, 721–726.

Moschandreas, D.J. 1981. Exposure to pollutants and daily time budgets of people. *Bull. N.Y. Acad. Med.* 57 (10), 845–859.

Moskalev, Yu. I. 1959. Experiments on the distribution of Ce144. *Med. Radiol.* 4, 52. English translation: *JPRS: 2860* (1960).

Moskalev, Yu.I., Bugreshev, P.F. and Zaikina, T.I. 1988. Effect of age on the metabolism of inhaled beryllium fluoride in rats. In: *Inhaled Particles VI*, Proceedings of an International Symposium and Workshop on Lung Dosimetry Organised by the British Occupational Hygiene Society in Co-operation with the Commission of the European Communities, Cambridge, 2–6 September 1985, (Eds. Dodgson, J., McCallum, R.I., Bailey, M.R. and Fisher, D.R.), Pergamon Press, Oxford, United Kingdom. *Ann. Occup. Hyg.* 32 (Suppl. 1), 963–967.

Mussalo-Rauhamaa, H., Jaakola, T., Miettinen, J.K. and Laiho, K. 1984. Plutonium in Finish Lapps - An estimate of the gastrointestinal absorption of plutonium by man based on a comparison of the plutonium content of Lapps and southern Finns. *Health Phys.* 46, 549-559.

Mussatto, D.J., Garrard, C.S. and Lourenço, R.V. 1988. The effect of inhaled histamine on human tracheal mucus velocity and bronchial mucociliary clearance. *Am. Rev. Respir. Dis.* 138, 775-779.

Naményi, J., Gachályi, A. and Varga, L. 1986. Desorption of ^{85}Sr by radioadsorbents from the lungs of rats with bronchial disorders. *Health Phys.* 51, 539-544.

Naversten, Y. and Liden, K. 1964. Half-life studies of radiocaesium in humans. In: *Assessment of Radioactivity in Man*, Vol. II, pp. 79-87 IAEA, Vienna.

NCRP. 1978. Physical, Chemical and Biological Properties of Radiocerium Relevant to Radiation Protection Guidelines. NCRP Report 60, Bethesda, Maryland.

NEA. 1988. Committee on Radiation Protection and Public Health. Gastrointestinal absorption of selected radionuclides - A report by an NEA expert group. NEA/OECD, Paris.

Newton, G.J. and Latven, R.K. 1971. Distribution and excretion in the beagle dog of ^{106}Ru -Rh aerosols subjected to thermal degradation. *Fission Product Inhalation Program Annual Report 1970-1971*, LF-44, pp. 81-85. Lovelace Foundation for Medical Education and Research, Albuquerque, New Mexico. Available from National Technical Information Service, Springfield, Virginia.

Newton, G.J., Snipes, M.B., Boecker, B.B. and Wagner, J.A. 1976. Radiation dose patterns from ^{106}Ru aerosols inhaled by Syrian hamsters II: Retention and tissue distribution. *Inhalation Toxicology Research Institute Annual Report 1975-1976*, LF-56, pp. 79-83. Lovelace Biomedical and Environmental Research Institute, Albuquerque, New Mexico. Available from National Technical Information Service, Springfield, Virginia.

Newton, D., Fry, F.A., Taylor, B.T., Eagle, M.C. and Sharma, R.C. 1978. Interlaboratory comparison of techniques for measuring lung burdens of low-energy photon-emitters. *Health Phys.* 35, 751-771.

Newton, D., Taylor, B.T. and Eakins, J.D. 1983. Differential clearance of plutonium and americium oxides from the human lung. *Health Phys.* 44 (Suppl. 1), 431-439.

Niinimaa, V., Cole, P., Mintz, S. and Shephard, R.J. 1980. The switching point from nasal to oronasal breathing. *Respir. Physiol.* 42, 61-71.

Niinimaa, V., Cole, P., Mintz, S. and Shephard, R.J. 1981. Oronasal distribution of respiratory airflow. *Respir. Physiol.* 43, 69-75.

Nixon, W. and Egan, M.J. 1987. Modelling study of regional deposition of inhaled aerosols with special reference to effects of ventilation asymmetry. *J. Aerosol Sci.* 18, 563-579.

Nolibé, D., Duserre, C., Gil, I., Rateau, G. and Métivier, H. 1989. Biological behaviour of plutonium given as a triaurylamine complex. Comparison with plutonium-tributylphosphate. *Radiat. Prot. Dosim.* 26, 303-306.

Norman, J.H. and Winchell, P. 1970. Physical, chemical and radiological properties of fallout. In: *Survival of Food Crops and Livestock in the Event of Nuclear War*. (Eds. Benson, D. and Sparrow, A.H.) pp. 9-30 USAEC Symposium Series 24.

Northcutt, A.R., Binney, S.E. and Palmer, H.E. 1988. In-vivo counting of ^{241}Am in human lungs and tracheobronchial lymph nodes. *Health Phys.* 54, 73-81.

Oberdörster, G. 1988. Lung clearance of inhaled insoluble and soluble particles. *J. Aerosol Med.* 1, 289-330.

Ogden, T.L. and Birkett, J.L. 1977. The human head as a dust sampler. In: *Inhaled Particles IV, Part 2, Proceedings of an International Symposium Organised by the British Occupational Hygiene Society, Edinburgh, 22-26 September 1975*, (Eds. Walton, W.H. and McGovern, B.), Pergamon Press, Oxford, United Kingdom. pp. 93-105.

Pack, A., Hooper, M.B., Nixon, W. and Taylor, J.C. 1977. A computational model of pulmonary gas transport incorporating effective diffusion. *Respir. Physiol.* 29, 101-124.

Pairon, J.C., Roos, F., Iwatsubo, Y., Janson, X., Billon-Galland, M. A., Bignon, J., Brochard, P. 1994. Lung retention of cerium in humans. *Occupational and Environmental Medicine* 51, 195-199.

Park, J.F., Dagle, G.E., Ragan, H.A., Weller, R.E. and Stevens, D.L. 1986a. Current status of life-span studies with inhaled plutonium in beagles. In: *Life-span Radiation Effects Studies in Animals: What Can They Tell Us? CONF-830951*, pp. 455-470. (Eds. Thompson, R.C. and Mahaffey, J.A.). Available from National Technical Information Service, Springfield, Virginia.

Park, J.F., Apley, G.A., Buschbom, R.L., Dagle, G.E., Fisher, D.R., Gideon, K.M., Gilbert, E.S., Kashmitter, J.D., Powers, G.J., Ragan, H.A., Weller, R.E. and Wierman, E.L. 1986b. Inhaled plutonium oxide in dogs. Pacific Northwest Laboratory Annual Report for 1985 to the DOE Office of Energy Research. PNL-5750, Part 1, Biomedical Sciences. pp. 3-17. Richland, Washington. Available from National Technical Information Service, Springfield, Virginia.

Passali, D. and Ciampoli, M.B. 1985. Normal values of mucociliary transport time in young subjects. *Int. J. Pediatr. Otorhinolaryngol.* 9, 151-156.

Patrick, G. 1979. The retention of uranium dioxide particles in the trachea of the rat. *Int. J. Radiat. Biol.* 35, 571-576.

Patrick, G. 1983. The retention of various types and sizes of particle in the large airways of the rat: Implications for assessing the risk of lung cancer. In: *Current Concepts in Lung Dosimetry*. (Ed. Fisher, D.R.). Available from National Technical Information Service, Springfield, Virginia, pp. 66-72.

Patrick, G. 1989. Requirements for local dosimetry and risk evaluation in inhomogeneously irradiated lung. In: *Low Dose Radiation: Biological Bases of Risk Assessment*. (Eds. Baverstock, K.F. and Stather, J.W.), Taylor & Francis, Bristol, Pennsylvania, pp. 269-277.

Patrick, G. and Stirling, C. 1977. The retention of particles in large airways of the respiratory tract. *Proc. R. Soc. Lond. B.* 198, 455-462.

Patrick, G. and Stirling, C. 1992. Transport of particles of colloidal gold within and from rat lung after local deposition by alveolar microinjection. *Environ. Health Perspect.* 97, 47-51.

Patrick, G. and Stirling, C. 1994. The redistribution of colloidal gold particles in rat lung following local deposition by alveolar microinjection. In: *Inhaled Particles VII*, Proceedings of the Seventh International Symposium on Inhaled Particles, Edinburgh, United Kingdom, September 17-21, 1991, (Eds. Dodgson, J. and McCallum, R.I.), Pergamon Press, Oxford, United Kingdom. *Ann. Occup. Hyg.* 38 (Suppl 1.) 225-234.

Patrick, G., Batchelor, A.L. and Stirling, C. 1996. Long-term retention of inhaled fused aluminosilicate particles in the trachea and first bifurcation of the rat. *J. Aerosol Sci.* 27, 161-172.

Pavia, D. 1984. Lung mucociliary clearance. In: *Aerosols and the Lung: Clinical and Experimental Aspects*. (Eds. Clarke, S.W. and Pavia, D.) Butterworths, Boston, pp. 127-155.

Pavia, D., Short, M.D. and Thomson, M.L. 1970. No demonstrable long term effects of cigarette smoking on the mucociliary mechanism of the human lung. *Nature* 226, 1228-1231.

Pavia, D., Thomson, M.L. and Pocock, S.J. 1971. Evidence for temporary slowing of mucociliary clearance in the lung caused by tobacco smoking. *Nature* 231, 325-326.

Pavia, D., Bateman, J.R.M. and Clarke, S.W. 1980. Deposition and clearance of inhaled particles. *Bull. Eur. Physiopathol. Respir.* 16, 335-366.

Pavia, D., Sutton, P.P., Lopez-Vidriero, M.T., Agnew, J.E. and Clarke, S.W. 1983. Drug effects on mucociliary function. *Eur. J. Respir. Dis.* 64 (Suppl. 128), 304-317.

Pearman, I., Foster, P.P., Ramsden, D. and Bains, M.E.D. 1989. Lung clearance of inhaled cobalt oxide in man. In: *Radiation Protection - Theory and Practice*. Proceedings of the Fourth International Symposium of the SRP. (Ed. Goldfinch, E.P.) IOP Publishing, Bristol and New York, pp. 251-254.

Pendleton, R.C., Mays, C.W., Lloyd, R.D. and Church, B.W. 1965. A trophic level effect on Cs-137 concentration. *Health Phys.* 11, 1503-1510.

Perrault, G., Thiéblemont, P., Pasquier, C. and Marblé, G. 1967. Cinétique du passage du radioiode soluble à travers les épithéliums respiratoires, après inhalation. *Health Phys.* 13, 707-718.

Peterson, B.T. 1989. Pulmonary clearance of aerosolised ^{99m}Tc -DTPA and the lung epithelium. *J. Aerosol Med.* 2, 315-328.

Petkau, A. and Pleskach, S.D. 1971. A case of accidental aspiration of $^{90}\text{SrCl}_2$. *Health Phys.* 22, 87-90.

Philipson, K., Falk, R. and Camner, P. 1985. Long-term lung clearance in humans studied with Teflon particles labeled with chromium-51. *Exp. Lung Res.* 9, 31-42.

Philipson, K., Gustafsson, J., Falk, R. and Camner, P. 1992. Long-term lung clearance in humans studied with ^{195}Au -labelled teflon particles. *J. Aerosol Med.* 5, 288.

Philipson, K., Falk, R., Gustafsson, J. and Camner, P. 1996. Long-term lung clearance of ^{195}Au -labelled teflon particles in humans. *Exp. Lung Res.* 22, 65-83.

Plesková, A., Trnovec, T., Chorvát, D. and Vladár, M. 1971. The effect of colloidal iron hydroxide on radio-cerium

metabolism in rats and mice. *Strahlentherapie* 142, 480–485.

Pomroy, C. and Noel, L. 1981. Retention of uranium thorax burdens in fuel fabricators. *Health Phys.* 41, 393–400.

Pomroy, C. and Noel, L. 1988. The distribution of inhaled uranium dust in the human thorax. In: *Inhaled Particles VI, Proceedings of an International Symposium and Workshop on Lung Dosimetry Organised by the British Occupational Hygiene Society in Co-operation with the Commission of the European Communities, Cambridge, 2-6 September 1985*, (Eds. Dodgson, J., McCallum, R.I., Bailey, M.R. and Fisher, D.R.), Pergamon Press, Oxford, United Kingdom. *Ann. Occup. Hyg.* 32 (Suppl. 1), 877–883.

Popplewell, D.S., Ham, G.J., McCarthy, W. and Lands, C. 1994. Transfer of plutonium across the human gut and its urinary excretion. In: *Proc. Workshop on Intakes of Radionuclides: Detection, Assessment and Limitation of Occupational Exposure*. *Radiat. Prot. Dosim.* 53, 241–244.

Pritchard, J.N., McAughay, J.J., Strong, J.C., Black, A. and Knight, D.A. 1994. The regional deposition of hygroscopic particles. In: *Inhaled Particles VII. Proceedings of an International Symposium Organised by the British Occupational Hygiene Society, Edinburgh, 16–22 September 1991*, (Eds. Dodgson, J. and McCallum, R.I.), Pergamon Press, Oxford, United Kingdom. *Ann. Occup. Hyg.* 38 (Suppl. 1), 107–115.

Puchelle, E., Zahm, J.M., Girard, F., Bertrand, A., Polu, J.M., Aug, F. and Sadoul, P. 1980. Mucociliary transport in vivo and in vitro. *Eur. J. Respir. Dis.* 61, 254–264.

Pusch, W.M. 1968. Determination of effective half-life of ^{103}Ru in man after inhalation. *Health Phys.* 15, 515–517.

Raabe, O.G. 1982. Deposition and clearance of inhaled aerosols. In: *Mechanisms in Respiratory Toxicity*. (Eds. Witschi, H. and Nettesheim, P.), CRC Press, Boca Raton, Florida, pp. 28–76.

Radford, E.P. and Martell, E.A. 1977. Polonium-210: Lead-210 Ratios as an Index of Residence Times of Insoluble Particles from Cigarette Smoke in Bronchial Epithelium. In: *Inhaled Particles IV, Part 2, Proceedings of an International Symposium Organised by the British Occupational Hygiene Society, Edinburgh, 22–26 September 1975* (Eds. Walton, W.H. and McGovern, B.), Pergamon Press, Oxford, United Kingdom, pp. 567–581.

Raju, T.N., Larkridge, C., Musselman, R., Harshbarger, R., Gerrity, T.R., Vidyasager, D., Lourenço, R.V. and Yeates, D.B. 1983) Mucociliary clearance in newborn baboons. *Pediatric Research.* 17, 387A (Abstract).

Ramsden, D. 1976. Assessment of plutonium in lung for both chronic and acute exposure conditions. In: *Diagnosis and Treatment of Incorporated Radionuclides. Proceedings of a Seminar held by the International Atomic Energy Agency and the World Health Organization, December 8–12, 1975, Vienna*, pp. 139–161. International Atomic Energy Agency, Vienna, Austria.

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. and Frazer, D.C. 1978. A case study of multiple low level exposure to plutonium oxide. *Health Phys.* 34, 649–659.

Richardson, J., Bouchard, T. and Ferguson, C.C. 1976. Uptake and transport of exogenous proteins by respiratory epithelium. *Lab. Invest.* 35, 307–314.

Richmond, C.R. and London, J.E. 1966. Long-term in vivo retention of cerium-144 by beagles. *Nature* 211, 1179.

Richmond, C.R., Furchner, J.E. and Langham, W.H. 1962. Long-term retention of radiocaesium in man. *Health Phys.* 8, 201–205.

Richards, D.S. 1952. Quantitative aspects of iodine metabolism. *Pharmacol. Rev.* 4, 284–370.

Robertson, G.B. and Rogers, A.W. 1980. An autoradiographic search for radioactive particles in the lungs of cigarette smokers. *Arch. Environ. Health* 35, 117–122.

Ross, I.T.H., Wallace, J.C. and Waite, D. 1979. A simplified method of monitoring mucociliary transport. *Br. J. Radiology.* 52, 968–971.

Rossoff, B., Cohn, S.H. and Spencer, H. 1963. Caesium-137 metabolism in man. *Radiat. Res.* 19, 643–654.

Roth, C., Scheuch, G. and Stahlhofen, W. 1994. Radioactively labelled ultrafine particles for clearance measurements. In: *Inhaled Particles VII, Proceedings of the Seventh International Symposium on Inhaled Particles, Edinburgh, United Kingdom, September 17–21, 1991*, (Eds. Dodgson, J. and McCallum, R.I.), Pergamon

Press, Oxford, United Kingdom. *Ann. Occup. Hyg.* 38 Suppl. 1., 101–106.

Roy, M. and Courtay, C. 1991. Daily activities and breathing parameters for use in respiratory tract dosimetry. *Radiat. Prot. Doim.* 35, 179–186.

Roy, M., Becquemin, M.H., Dautzenberg, B., Sors, C., Teillac, A. and Gongora, G. 1984. Inhaled particles deposition and clearance in silicosis. In: International Pneumoconiosis Conference, Bochum, September 20–23, 1983. (Ed. Ulmer, W.T.) ILO, Geneva, Switzerland, pp. 425–431.

Roy, M., Becquemin, M.H., Robeau, D., Bonnefous, S., Piechowski, J., Gongora, G. and Teillac, A. 1988. Particle clearance from human lung. In: Inhaled Particles VI, Proceedings of an International Symposium and Workshop on Lung Dosimetry Organised by the British Occupational Hygiene Society in Co-operation with the Commission of the European Communities, Cambridge, 2–6 September 1985, (Eds. Dodgson, J., McCallum, R.I., Bailey, M.R. and Fisher, D.R.), Pergamon Press, Oxford, United Kingdom. *Ann. Occup. Hyg.* 32 (Suppl. 1), pp. 1163–1164.

Roy, M., Becquemin, M.H., Bertholon, J.-F. and Bouchikhi, A. 1994. Annexe B. Respiratory Physiology. In Human Respiratory Tract Model for Radiological Protection, ICRP Publication 66. *Annals of the ICRP* 24 (1–3). Elsevier Science Ltd., Oxford, pp. 167–201.

Roy M. et al. 1995. Final report to CEC of Contract No. FI3P-CT920064a Inhalation and Ingestion of Radionuclides. CEC (in press).

Rudolf, G., Gebhart, J., Heyder, J., Scheuch, G. and Stahlhofen, W. 1983. Modelling the deposition of aerosol particles in the human respiratory tract. *J. Aerosol Sci.* 14, 188–192.

Rundo, J. 1965. A case of accidental inhalation of irradiated uranium. *Brit. J. Radiol.* 38, 39–50.

Rundo, J. and Taylor, B.T. 1964. The assessment of radioactive caesium in man. In: Assessment of Radioactivity in Man, Vol. II, pp. 3–20. IAEA, Vienna.

Rundo, J. and Williams, K. 1961. A case of accidental inhalation of $^{90}\text{SrCO}_3$. *Br. J. Radiol.* 34, 734–740.

Rundo, J., Mason, J., Newton, D. and Taylor, B. 1963. Biological half-life of caesium in man in acute and chronic exposure. *Nature* 200, 188–189.

Runkle, G.E. and Snipes, M.B. 1979. A system for nose-only inhalation exposures of small animals to $^{106}\text{RuO}_4$. *J. Aerosol Sci.* 10, 432–435.

Runkle, G.E., Snipes, M.B., McClellan, R.O. and Cuddihy, R.G. 1980. Metabolism and dosimetry of inhaled $^{106}\text{RuO}_4$ in Fischer-344 rats. *Health Phys.* 39, 543–553.

Sackner, M.A., Landa, J., Hirsch, J. and Zapata, A. 1975. Pulmonary effects of oxygen breathing. *Ann. Int. Med.* 82, 40–43.

Sackner, M.A., Yergin, B.M., Brito, M. and Janusziewicz, A. 1979. Effect of adrenergic agonists on tracheal mucous velocity. *Bull. Eur. Physiopathol. Respir.* 15, 505–511.

Samet, J.M., Lambert, W.E., James, D.S., Mermier, C.M. and Chick, T.W. 1993. Assessment of heart rate as a predictor of ventilation. In: Noninvasive methods for measuring ventilation in mobile subjects, Research Report Number 59. Health Effects Institute, Cambridge, MA, USA, pp. 19–69.

Sanchis, J., Dolovich, M., Chalmers, R. and Newhouse, M. 1972. Quantitation of regional aerosol clearance in the normal human lung. *J. Appl. Physiol.* 33, 757–762.

Sanchis, J., Dolovich, M., Rossman, C., Wilson, W. and Newhouse, M. 1973. Pulmonary mucociliary clearance in cystic fibrosis. *N. Engl. J. Med.* 288, 651–654.

Sanchis, J., Dolovich, M., Rossman, C. and Newhouse, M. 1974. Evaluation of bronchial clearance after inhalation of radioactive material. *INSERM* 29, 79–88.

Santa Cruz, R., Landa, J., Hirsch, J. and Sackner, M.A. 1974. Tracheal mucous velocity in normal man and patients with obstructive lung disease; effects of terbutaline. *Am. Rev. Respir. Dis.* 109, 458–463.

Scherrer, J. 1981. *Précis de physiologie du travail. Notions d'Ergonomie*, Masson, Paris.

Scheuch, G. 1991. Die Dispersion, Deposition und Clearance von Aerosolpartikeln in den menschlichen Atemwegen (Ph.D. thesis). J.W. Goethe-Universität, Frankfurt am Main, Germany.

Scheuch, G. 1995. An approach to deposition and retention measurements in human airways. *J. Aerosol Med.* 8, 78.

Scheuch, G. and Stahlhofen, W. 1988. Particle deposition of inhaled aerosol boluses in the upper human airways. *J. Aerosol Med.* 1, 29–36.

Scheuch, G. and Stahlhofen, W. 1991. Effect of heart rate on aerosol recovery and dispersion in human conducting

airways after periods of breathholding. *Exp. Lung Res.* 17, 763-787.

Scheuch, G. and Stahlhofen, W. 1994. Effect of settling velocity on particle recovery from human conducting airways after breath holding. In: *Inhaled Particles VII*, Proceedings of the Seventh International Symposium on Inhaled Particles, Edinburgh, United Kingdom, September 17-21, 1991, (Eds. Dodgson, J. and McCallum, R.I.), Pergamon Press, Oxford, United Kingdom. *Ann. Occup. Hyg.* 38 (Suppl 1), 159-166.

Scheuch, G., Kreyling, W., Haas, F. and Stahlhofen, W. 1993a. The clearance of polystyrene particles from human intrathoracic airways. *J. Aerosol Med.* 6 (Suppl. 1), 47.

Scheuch, G., Stahlhofen, W., Fang, C. P. and Lippmann, M. 1993b. Aerosol recovery after bolus inhalations into an airway cast. *J. Aerosol Sci.* 24 (Suppl. 1), S355-S356.

Schieferdecker, H., Dilger, H., Doerfel, H., Rudolph, W. and Anton, R. 1985. Inhalation of U aerosols from UO_2 fuel element fabrication. *Health Phys.* 48, 29-48.

Schiller-Scotland, C.F., Hlawa, R., Gebhart, J., Wönnne, R. and Heyder, J. 1992. Total deposition of aerosol particles in the respiratory tract of children during spontaneous and controlled mouth breathing. *J. Aerosol Sci.* 23 (Suppl. 1), S457-S460.

Schiller-Scotland, C.F., Hlawa, R., Gebhart, J., Heyder, J., Roth, C. and Wönnne, R. 1994. Particle deposition in the respiratory tract of children during spontaneous and controlled mouth breathing. In: *Inhaled Particles VII*. Proceedings of an International Symposium Organised by the British Occupational Hygiene Society, Edinburgh, 16-22 September 1991, (Eds. Dodgson, J. and McCallum, R.I.), Pergamon Press, Oxford, United Kingdom. *Ann. Occup. Hyg.* 38 (Suppl. 1), 117-125.

Schlesinger, R.B. 1985. Clearance from the respiratory tract. *Fundam. Appl. Toxicol.* 5, 435-450.

Schlesinger, R.B. 1989. Deposition and clearance of inhaled particles. In: *Concepts in Inhalation Toxicology* (Eds. McClellan, R.O. and Henderson, R.F.), Hemisphere Publishing Corporation, pp. 163-192.

Schürch, S., Gehr, P., Im Hof, V., Geiser, M. and Green, F. 1990. Surfactant displaces particles toward the epithelium in airways and alveoli. *Respir. Physiol.* 80, 17-32.

Schwartz, G. and Dunning, D.E. 1982. Imprecision in estimates of dose from ingested ^{137}Cs due to variability in human biological characteristics. *Health Phys.* 43, 631-645.

Scott, L.M. and West, C.M. 1975. Uranium in vivo spectrometry. In: *Conference on Occupational Health Experience with Uranium, Arlington, Virginia, April 28-30, 1975*. Available from National Technical Information Service, Springfield, Virginia, pp. 264-296.

Shimmins, J., Smith, D., Nordin, B. and Burkinshaw, L. 1967. A comparison between calcium-45 and strontium-85 absorption, excretion and skeletal uptake. In: *Strontium Metabolism*. (Eds. Lenihan, J., Loutit, J. and Martin, J.)

Short, M.D., Dowsett, D.J., Heaf, P.J.D., Pavia, D. and Thomson, M.L. 1979. A comparison between monodisperse $Tc-99m$ -labeled aerosol particles and $Kr-81m$ for the assessment of lung function. *J. Nucl. Med.* 20, 194-200.

Shyr, L.J., Griffith, W.C. and Boecker, B.B. 1991. An optimization strategy for a biokinetic model of inhaled radionuclides. *Fundamental and Applied Toxicology* 16, 423-434.

Smaldone, G.C., Itoh, H., Swift, D.L. and Wasner, H.N. Jr. 1979. Effect of flow-limiting segments and cough on particle deposition and mucociliary clearance in the lung. *Am. Rev. Respir. Dis.* 120, 747-758.

Smaldone, G.C., Perry, R.J., Bennett, W.D., Messina, M.S., Zwang, J. and Ilowite, J. 1988. Interpretation of "24 hour lung retention" in studies of mucociliary clearance. *J. Aerosol Med.* 1, 11-20.

Smith, V.H. 1970. Fate of ingested $^{238}PuO_2$ in miniature swine. In: *Pacific Northwest Laboratory Annual Report for 1969*. Pt. 1. Biomedical Sciences. Richland, Wash. BNWL-1306. pp. 62-63.

Snipes, M.B. 1979. Deposition, retention and dosimetry of inhaled ^{106}Ru attached to inert particles. *Inhalation Toxicology Research Institute Annual Report 1978-1979*, LF-69, pp. 43-48. Lovelace Biomedical and Environmental Research Institute, Albuquerque, New Mexico. Available from National Technical Information Service, Springfield, Virginia.

Snipes, M.B. 1981. Metabolism and dosimetry of ^{106}Ru inhaled as $^{106}RuO_4$ by beagle dogs. *Health Phys.* 41, 303-317.

Snipes, M.B. 1989. Long-term retention and clearance of particles inhaled by mammalian species. *Crit. Rev. Toxicol.* 20, 175-211.

Snipes, M.B. and McClellan, R.O. 1986. Model for deposition and long-term disposition of ^{134}Cs -labeled fused aluminosilicate particles inhaled by guinea pigs.

Inhalation Toxicology Research Institute Annual Report 1985-1986, LMF-115, pp. 91-95. Lovelace Biomedical & Environmental Research Institute, Albuquerque, New Mexico. Available from National Technical Information Service, Springfield, Virginia..

Snipes, M.B., Barnes, J.E., Boecker, B.B., Hahn, F.F., Hobbs, C.H., Mauderly, J.L., McClellan, R.O. and Pickrell, J.A. 1972. Toxicity of inhaled 90Sr fused clay in Beagle dogs III. Fission Product Inhalation Program Annual Report 1971-1972, LF-45, pp. 177-188. Lovelace Foundation for Medical Education and Research, Albuquerque, New Mexico. Available from National Technical Information Service, Springfield, Virginia.

Snipes, M.B., Boecker, B.B. and McClellan, R.O. 1983. Retention of monodisperse or polydisperse aluminosilicate particles inhaled by dogs, rats, and mice. *Toxicol. Appl. Pharmacol.* 69, 345-362.

Snipes, M.B., Griffith, W.C., Nikula, K.J. and Guilmette, R.A. 1993. Clearance of particles deposited in the conducting airways of beagle dogs. *J. Aerosol Med.* 6 Suppl. 49.

Snipes, M.B., Muggenburg, B.A., Griffith, W.C. and Guilmette, R.A. 1995. Clearance patterns for ^{111}In -oxide particles deposited in airways of beagle dogs. *J. Aerosol Med.* 8, 85.

Sorokin, S.P. 1977. Phagocytes in the lungs. In: *Respiratory Defense Mechanisms*. (Eds. Brain, J.D., Proctor, D.F. and Reid, L.M.). Marcel Dekker, Inc., New York, pp. 711-848.

Sorokin, S.P. and Brain, J.D. 1975. Pathways of clearance in mouse lungs exposed to iron oxide aerosols. *Anat. Rec.* 181, 581-625.

Spencer, H., Samachson, J. and Laszlo, D. 1960. Metabolism of strontium-85 and calcium-45 in man. *Metabolism Clinical and Experimental.* 9, 916-925.

Spencer, H., Kramer, L., Norris, C. and Samachson, J. 1972. Certain aspects of radiostrontium metabolism in man. In: *Proceedings of the 2nd International Symposium on Strontium Metabolism*. (Ed. Lenihan, J.M.A.).

Spitz, H.B. and Robinson, R. 1981. Deposition of plutonium in the lungs of a worker following an accidental inhalation exposure. In: *Actinides in Man and Animals, Proceedings of the Actinide Workshop*, Snowbird, Utah, October 15-17, 1979 pp. 115-135 (Ed. Wrenn, M.E.). RD Press, Salt Lake City, Utah.

Spoo, J.W., Snipes, M.B., Griffith, W.C. and Guilmette, R.A. 1991. Clearance of particles deposited in the conducting airways of beagle dogs. *Inhalation Toxicology Research Institute Annual Report 1990-1991*, LMF-134. Lovelace Biomedical and Environmental Research Foundation, Albuquerque, New Mexico, Available from National Technical Information Service, Springfield, Virginia, pp. 59-61.

Stahlhofen, W. 1989. Human lung clearance following bolus inhalation of radioaerosols. In: *Extrapolation of Dosimetric Relationships for Inhaled Particles and Gases*. Academic Press, Washington, D.C., pp. 153-166.

Stahlhofen, W. and Möller, W. 1991. The behaviour of inhaled spherical iron oxide particles in human lungs: Magnetometric studies. In: *Environmental Hygiene III*. (Eds. Seemayer, N.H. and Hadnagy, W.), Springer-Verlag, Berlin, Germany pp. 1-4.

Stahlhofen, W., Gebhart, J. and Heyder, J. 1980. Experimental determination of the regional deposition of aerosol particles in the human respiratory tract. *Am. Ind. Hyg. Assoc. J.* 41, 385-398a.

Stahlhofen, W., Gebhart, J., Heyder, J., Philipson, K. and Camner, P. 1981. Intercomparison of regional deposition of aerosol particles in the human respiratory tract and their long-term elimination. *Exp. Lung. Res.* 2, 131-139.

Stahlhofen, W., Gebhart, J., Heyder, J. and Scheuch, G. 1983. New regional deposition data of the human respiratory tract. *J. Aerosol Sci.* 14, 186-188.

Stahlhofen, W., Gebhart, J., Heyder, J., Scheuch, G. and Jurasko, P. 1984. Particle deposition in extrathoracic airways of health subjects and of patients with early stages of laryngeal carcinoma. *J. Aerosol Sci.* 15, 215-217.

Stahlhofen, W., Gebhart, J., Rudolf, G. and Scheuch, G. 1986a. Measurement of lung clearance with pulses of radioactively-labelled aerosols. *J. Aerosol Sci.* 17, 333-336.

Stahlhofen, W., Gebhart, J., Rudolf, G., Scheuch, G. and Philipson, K. 1986b. Clearance from the human airways of particles of different sizes deposited from inhaled aerosol boli. In: *Aerosols: Formation and Reactivity*. Second International Aerosol Conference, West Berlin, Germany, September 22-26, 1986. Pergamon Journals Ltd., pp. 192-196.

Stahlhofen, W., Gebhart, J., Rudolf, G. and Scheuch, G. 1987a. Retention of Radioactively Labelled Fe_2O_3 -Particles in Human Lungs. In: *Deposition and Clearance of Aerosols in the Human Respiratory Tract*. Second

International Symposium, Salzburg, Austria, September 18–20, 1986. (Ed. Hofmann, W.), Facultas Universitätsverlag Ges.m.b.H. Wien, Austria, pp. 123–128.

Stahlhofen, W., Gebhart, J., Rudolf, G., Scheuch, G. and Bailey, M.R. 1987b. Human lung clearance of inhaled radioactively labelled particles in horizontal and vertical position of the inhaling person. *J. Aerosol Sci.* 18, 741–744.

Stahlhofen, W., Rudolf, G. and James, A.C. 1989. Intercomparison of experimental regional aerosol deposition data. *J. Aerosol Med.* 2, 285–308.

Stahlhofen, W., Koebrich, R., Rudolf, G. and Scheuch, G. 1990. Short-term and long-term clearance of particles from the upper human respiratory tract as function of particle size. *J. Aerosol Sci.* 21 (Suppl. 1), S407–S410.

Stahlhofen, W., Scheuch, G. and Bailey, M.R. 1994. Measurement of the tracheobronchial clearance of particles after aerosol bolus inhalation. In: *Inhaled Particles VII, Proceedings of the Seventh International Symposium on Inhaled Particles*, Edinburgh, United Kingdom, September 17–21, 1991, (Eds. Dodgson, J. and McCallum, R.I.), Pergamon Press, Oxford, United Kingdom. *Ann. Occup. Hyg.* 38 (Suppl. 1), 189–196.

Stahlhofen, W., Scheuch, G. and Bailey, M.R. 1995. Investigations of retention of inhaled particles in the human bronchial tree. In: *Proceedings of the Symposium on the Radiobiology of Inhaled Nuclides*, Richland, WA, USA, November 1993. *Radiat. Prot. Dosim.* 60, 311.

Stanley, J.A., Eidson, A.F., Mewhinney, J.A. and Guilmette, R.A. 1980a. Deposition, retention and dosimetry of inhaled mixed uranium-plutonium oxides (heat-treated at 750°C) in Fischer-344 rats, beagle dogs and cynomolgous monkeys. In: *Radiation Dose Estimates and Hazard Evaluations for Inhaled Airborne Radionuclides, Annual Progress Report July 1978–June 1979, LF-71*, US Nuclear Regulatory Commission Document No. NUREG/CR-1458, pp. 35–46. Inhalation Toxicology Research Institute, Lovelace Biomedical & Environmental Research Institute, Albuquerque, New Mexico. Available from National Technical Information Service, Springfield, Virginia.

Stanley, J.A., Eidson, A.F. and Mewhinney, J.A. 1980b. Deposition, retention and dosimetry of inhaled mixed uranium-plutonium oxides (heat-treated at 1750°C) in Fischer-344 rats, beagle dogs and cynomolgous monkeys. In: *Radiation Dose Estimates and Hazard Evaluations for Inhaled Airborne Radionuclides, Annual Progress Report July 1978–June 1979, LF-71*, US Nuclear Regulatory Commission Document No. NUREG/CR-1458, pp. 47–53. Inhalation Toxicology Research Institute, Lovelace Biomedical & Environmental Research Institute, Albuquerque, New Mexico. Available from National Technical Information Service, Springfield, Virginia.

Stanley, J.A., Eidson, A.F. and Mewhinney, J.A. 1982. Distribution, retention and dosimetry of plutonium and americium in the rat, dog and monkey after inhalation of an industrial-mixed uranium and plutonium oxide aerosol. *Health Phys.* 43, 521–530.

Stara, J.F. 1965. Tissue distribution and excretion of caesium-137 in the guinea pig after administration by three different routes. *Health Phys.* 11, 1195–1202.

Stather, J.W. and Howden, S. 1975. The effect of chemical form on the clearance of plutonium-239 from the respiratory system of the rat. *Health Phys.* 28, 29–39.

Stather, J.W., Howden, S. and Carter, R.F. 1975. A method for investigating the metabolism of the transportable fraction of plutonium aerosols. *Phys. Med. Biol.* 20, 106–124.

Stather, J.W., James, A.C. and Rodwell, P. 1978. Measurement of in vitro and in vivo rates of dissolution of actinides associated with sediment samples. National Radiological Protection Board, Annual Research and Development Report 1977, NRPB/R&D 2, pp. 141–145. National Radiological Protection Board, Chilton, UK.

Stather, J.W., James, A.C., Brightwell, J. and Rodwell, P. 1979. The clearance of Pu and Am from the respiratory system of rodents after the inhalation of oxide aerosols of these actinides either alone or in combination with other metals. In: *Biological Implications of Radionuclides Released from Nuclear Industries, Proceedings of a Symposium*, Vienna, Austria, March 26–30, 1979, Vol. 2, pp. 3–25. International Atomic Energy Agency, Vienna, Austria.

Stather, J.W., Harrison, J.D., David, A.J. and Sumner, S.A. 1981. The gastrointestinal absorption of plutonium in the hamster after ingestion at low concentrations in drinking water. *Health Phys.* 41, 780–783.

Stirling, C. and Patrick, G. 1980. The localisation of particles retained in the trachea of the rat. *J. Pathology.* 131, 309–320.

Stöber, W., Einbrodt, H.J. and Klosterkötter, W. 1967. Quantitative Studies of Dust Retention in Animal and Human Lungs After Chronic Inhalation. In: *Inhaled Particles and Vapours II, Proceedings of an International*

Symposium Organised by the British Occupational Hygiene Society, Cambridge, 28 September-1 October 1965, (Ed. Davies, C.N.), Pergamon Press, Oxford, pp. 409-418.

Stradling, G.N., Stather, J.W., Sumner, S.A., Moody, J.C. and Hodgson, A. 1985. The metabolism and decorporation of plutonium after inhalation of the tributyl phosphate complex by the rat. *Health Phys.* 49, 499-502.

Stradling, G.N., Stather, J.W., Gray, S.A., Moody, J.C., Bailey, M.R., Hodgson, A. and Collier, C.G. 1987. Study on the metabolic behaviour of industrial actinide-bearing aerosols after deposition in the rat lung: an experimental basis for interpreting chest monitoring data and assessing limits on intake for workers. *Human Toxicol.* 6, 365-375.

Stradling, G.N., Stather, J.W., Gray, S.A., Moody, J.C., Ellender, M., and Collier, C.G. 1989. Assessment of intake of an actinide-bearing dust formed from the pond storage of spent magnox fuel. *Radiat. Prot. Dosim.* 26, 201-206.

Stradling, G.N., Stather, J.W., Gray, S.A., Moody, J.C., Ellender, M., Pearce, M.J. and Collier, C.G. 1992. Radiological implications of inhaled ^{239}Pu and ^{241}Am in dusts at the former nuclear test site in Maralinga. *Health Phys.* 63, 641-650.

Stradling, G. N., Gray, S. A., Moody, J. C., Hodgson, A., Ellender, M., Phipps, A., Pearce, M., Wilson, I., Iranzo, C. E., Rivas, P., Espinosa, A., Aragó, A. and Iranzo, E. 1993. Biokinetics of plutonium-239 and americium-241 in the rat after the pulmonary deposition of contaminated dust obtained from soil samples at Palomares: Implications for human exposure. NRPB-M444, National Radiological Protection Board, Chilton, UK.

Stuart, B.O. 1970. Long-term retention and translocation of inhaled ^{106}Ru - $^{106}\text{RhO}_2$ in beagles. Pacific Northwest Laboratory Annual Report for 1968 to the USAEC Division of Biology and Medicine. BNWL-1050 Part 1: Biological Sciences, pp. 3-43. Richland, Washington. Available from National Technical Information Service, Springfield, Virginia.

Stuart, B.O. 1984. Deposition and clearance of inhaled particles. *Environ. Health Perspect.* 55, 369-390.

Stuart, B.O., Dionne, P.J. and Bair, W.J. 1970. A dynamic simulation of the retention and translocation of inhaled plutonium dioxide in beagle dogs. In: Proceedings 11th AEC Air Cleaning Conference, Vol. 2. (Eds. First, M.W. and Morgan, J.M.) Available from National Technical Information Service, Springfield Virginia, pp. 721-737.

Sturbaum, D., Brooks, A.L. and McClellan, R.O. 1970. Tissue distribution and dosimetry of ^{144}Ce in Chinese Hamster. *Radiat. Res.* 44, 459-367.

Sturgess, J.M. 1977a. Structural organization of mucus in the lung. In: *Pulmonary Macrophage and Epithelial Cells*. (Eds. Sanders, C.L., Schneider, R.P., Dagle, G.E. and Ragan, H.A.) Available from National Technical Information Service, Springfield, Virginia, pp. 149-161.

Sturgess, J.M. 1977b. The mucous lining of major bronchi in the rabbit lung. *Am. Rev. Respir. Dis.* 115, 819-827.

Sturgess, J.M. 1985. Mucociliary clearance and mucus secretion in the lung. In *Toxicology of Inhaled Particles*. (Eds. Witschi, H.P. and Brain, J.D.), Springer-Verlag, Berlin, pp. 319-367.

Suguri, S., Ohtani, S., Oshino, M. and Yanagidhita, K. 1963. The behaviour of strontium-85 in a normal man following a single ingestion application of the whole body counter for the retention. *Health Phys.* 9, 529-535.

Sullivan, M.F. 1980. Absorption of actinide elements from the gastrointestinal tract of rats, guinea pigs and dogs. *Health Phys.* 38, 159-171.

Sullivan, M.F. 1981. Influence of plutonium mass on absorption from the gastrointestinal tract of adult and neonatal rodents. In: *Pacific Northwest Laboratory Annual Report for 1980*. Pt. 1. Biomedical Sciences. Richland, Wash. PNL-3700, pp. 185-186.

Sullivan, M.F., Miller, B.M. and Ryan, J.L. 1982. Gut-related radionuclide studies. In: *Pacific Northwest Laboratory Annual Report for 1981*. Pt. 1. Biomedical Sciences. Richland, Wash. PNL-4100, pp. 119-122.

Sunta, C.M., Dang, H.S. and Jaiswal, D.D. 1987. Thorium in man and environment, uptake and clearance. *J. Radioanal. Nucl. Chem.* 115, 149-158.

Svartengren, M. and Camner, P. 1984. Influence of bethanechol on retained particles deposited on the first bifurcation of rabbits. *Bull. Eur. Physiopathol. Respir.* 20, 133-137.

Svartengren, M., Widtskiold-Olsson, K., Philipson, K. and Camner, P. 1981. Retention of particles on the first bifurcation and trachea of rabbits. *Bull. Eur. Physiopathol. Respir.* 17, 87-91.

Svartengren, K., Philipson, K., Svartengren, M., Nerbrink, O. and Camner, P. 1996. Clearance in smaller airways of inhaled 6 um particles in subjects with immotile-cilia syndrome. *Exp. Lung Res.* (in press).

Sweeney, T.L., Blanchard, J.D., Zeltner, T.B., Carter, J.E. and Brain, J.D. 1988. Anatomical distribution of particle deposition in excised dog lungs given aerosol boluses to a shallow depth (abstract). *J. Aerosol Med.* 1, 212.

Swift, D.L. 1985. Effects of inhalation mode on particle deposition in the respiratory tract. Presented at the Second U.S.-Dutch International Symposium on Aerosols, Williamsburg, Virginia, May 19-24, 1985.

Swift, D.L. and Proctor, D.F. 1988. A dosimetric model for particles in the respiratory tract above the trachea. In: *Inhaled Particles VI, Proceedings of an International Symposium and Workshop on Lung Dosimetry Organised by the British Occupational Hygiene Society in Co-operation with the Commission of the European Communities, Cambridge, 2-6 September 1985*, (Eds. Dodgson, J., McCallum, R.I., Bailey, M.R. and Fisher, D.R.), Pergamon Press, Oxford, United Kingdom. *Ann. Occup. Hyg.* 32 (Suppl. 1), 1035-1044.

Swinth, K.L., Traub, R.J., Murphy, B.L. and Palmer, H.E. 1988. Factors affecting the assessment of lung deposits of transuranics. In: *Inhaled Particles VI, Proceedings of an International Symposium and Workshop on Lung Dosimetry Organised by the British Occupational Hygiene Society in Co-operation with the Commission of the European Communities, Cambridge, 2-6 September 1985*, (Eds. Dodgson, J., McCallum, R.I., Bailey, M.R. and Fisher, D.R.), Pergamon Press, Oxford, United Kingdom. *Ann. Occup. Hyg.* 32 (Suppl. 1), 893-899.

Szalai, A. (Ed). 1972. The Multinational Comparative Time Budget Research Project. In: *The Use of Time: Daily Activities of Urban and Suburban Populations in Twelve Countries*. The Hague, Paris, Mouton.

Takahashi, S. and Patrick, G. 1987a. Long-term retention of ^{133}Ba in the rat trachea following local administration as barium sulfate particles. *Radiat. Res.* 110, 321-328.

Takahashi, S. and Patrick, G. 1987b. Patterns of lymphatic drainage to individual thoracic and cervical lymph nodes in the rat. *Lab. Anim.* 21, 31-34.

Takahashi, S., Moriguchi, K., Kubota, Y., Sato, H. and Matsuoka, O. 1989. The deposition pattern of insoluble particles with different sizes in the rat trachea. *Hoken Butsuri* 24, 19-24.

Takahashi, S., Kubota, Y., Sato, H. and Matsuoka, O. 1993. Retention of ^{133}Ba in the trachea of rabbits, dogs and monkeys following local administration of $^{133}\text{BaSO}_4$ particles. *Inhalation Toxicology*. 5, 265-273.

Takenaka, S., Dornhöfer-Takenaka, H. and Muhle, H. 1986. Alveolar distribution of fly ash and of titanium dioxide after long-term inhalation by Wistar rats. *J. Aerosol Sci.* 17, 361-364.

Talbot, R.J. 1991. The gastrointestinal absorption of fission products. In: *Radiological Protection Research 1991 Annual Report, AEA Environment and Energy, AEA Technology*. p. 64.

Talbot, R.J., Newton, D., Warner, A.J., Walters, B. and Sherlock, J.C. 1993. Human uptake of ^{137}Cs in mutton. *Health Phys.* 64, 600-604.

Taussig, L.M., Harris, T.R. and Lebowitz, M.D. 1977. Lung function in infants and young children. *Am. Rev. Respir. Dis.* 116, 233-239.

Taylor, D.M. 1983. The comparative retention of bone-seeking radionuclides in the skeletons of rats. *Health Phys.* 45, 768-772.

Taylor D.M., Bligh P.H. and Duggan, M.H. 1962a. The absorption of calcium, strontium, barium and radium from the gastrointestinal tract of the rat. *Biochem J.* 83, 25-29.

Taylor, M.P., Vennart, J. and Taylor, D.M. 1962b. Retention and excretion of caesium-137 by man. *Phys. Med. Biol.* 7, 157-165.

TGLD. 1966. Deposition and retention models for internal dosimetry of the human respiratory tract. *Health Phys.* 12, 173-207.

Thiéblemont, P., Marblé, G., Perrault, G. and Pasquier, C. 1965. Évaluation de la rétention respiratoire et de l'élimination du radioiode après contamination aérienne du singe. *Int. J. Rad. Biol.* 9, 219-231.

Thind, K.S. 1987. A comparison of ICRP Publication 30 lung model-based predictions with measured bioassay data for airborne natural UO_2 exposure. *Health Phys.* 53, 59-66.

Thomas, R.G. 1968. Transport of relatively insoluble materials from lung to lymph nodes. *Health Phys.* 14, 111-117.

Thomas, R.L. and McClellan, R.O. 1972. Retention and tissue distribution of ^{144}Ce following inhalation of $^{144}\text{CeO}_2$ in Syrian hamsters. *Fission Product Inhalation Program Annual Report, 1971-1972, LF-45*, pp. 74-76. Lovelace Foundation for Medical Education and Research, Albuquerque, New Mexico. Available from National Technical Information Service, Springfield, Virginia.

Thomas, R.L., Scott, J.K. and Chiffelle, T.L. 1970. Metabolism and toxicity of inhaled and injected ^{131}I in the rat. *Amer. Ind. Hyg. Assn. J.* 31, 213-220.

Thomas, R.L., Scott, J.K. and Chiffelle, T.L. 1972. Metabolism and toxicity of inhaled ^{144}Ce in rats. *Radiat. Res.* 49, 589-610.

Thomson, M.L. and Pavia, D. 1973. Long-term tobacco smoking and mucociliary clearance. *Arch. Environ. Health* 26, 86-89.

Thomson, M.L. and Pavia D. 1974. Particle penetration and clearance in the human lung. *Arch. Environ. Health* 29, 214-219.

Thomson, M.L., Pavia, D., Short, M.D. and Norman, A.P. 1973. Lung clearance in two patients with cystic fibrosis. *N. Engl. J. Med.* 289, 749-750.

Toivonen, H., Pöllänen, R., Leppänen, A., Klemola, S. and Lahtinen, J. 1992. Release from the nuclear power plant in Sosnovyy Bor in March 1992. *Radiochimica Acta* 57, 169-172.

Tombropoulos, E.G., Bair, W.J. and Park, J.F. 1969. Removal of inhaled ^{144}Ce - ^{144}Pr oxide by diethylenetriaminepentaacetic acid (DTPA) treatment. I. ^{144}Ce - ^{144}Pr oxide prepared by peroxide oxidation. *Health Phys.* 16, 333-338.

Toomes, H., Vogt-Moykopf, I., Heller, W.D. and Ostertag, H. 1981. Measurement of mucociliary clearance in smokers and nonsmokers using a bronchoscopic video-technical method. *Lung* 159, 27-34.

Tracey, B.L., Kramer, G.H. and Gamarnik, K. 1994. Radiocaesium in children from Belarus. *Health Phys.* 66, 439-443.

Tu, K.W. and Knutson, E.O. 1984. Total deposition of ultrafine hydrophobic and hygroscopic aerosols in the human respiratory system. *Aerosol Sci. Technol.* 3, 453-465.

Uchiyama, M. 1987. Estimation of Cs-137 body burden in Japanese. II. The biological half-life. *J. Radiat. Res.* 19, 246-261.

Underwood, J. 1971. Iodine. In: *Trace Elements in Human and Animal Nutrition*. Academic Press, New York.

UNSCEAR. 1982. *Ionizing Radiations: Sources and Biological Effects*. United Nations Scientific Committee on the Effects of Atomic Radiation. United Nations, Publication N.E.82.IX.8., New York.

UNSCEAR. 1988. *Sources, Effects and Risks of Ionizing Radiation*. United Nations Scientific Committee on the Effects of Atomic Radiation. Report to the General Assembly Publication No. E.88.1X.7. with Annexes, New York.

Van As, A. 1977. Pulmonary airway clearance mechanisms: A reappraisal. *Am. Rev. Resp. Dis.* 115, 721-726.

Van As, A. 1980. Pulmonary airway defense mechanisms: An appreciation of integrated mucociliary activity. *Eur. J. Respir. Dis.* 61 (Suppl. 111), 21-24.

Van As, A. and Webster, I. 1974. The morphology of mucus in mammalian pulmonary airways. *Environ. Res.* 7, 1-12.

Van Dillan, M.A. 1965. On the retention of caesium-137 in people. *Health Phys.* 11, 21-22.

Vastag, E., Matthys, H., Köhler, D., Gronbeck, L. and Daikeler, G. 1985. Mucociliary clearance airways obstruction in smokers, ex-smokers and normal subjects who never smoked. *Eur. J. Respir. Dis.* 68 (Suppl. 139), 93-100.

Vastag, E., Matthys, H., Zsamboki, G., Köhler, D. and Daikeler, G. 1986. Mucociliary clearance in smokers. *Eur. J. Respir. Dis.* 68, 107-113.

Velasquez, D.J. and Morrow, P.E. 1984. Estimation of guinea pig tracheobronchial transport rates using a compartmental model. *Exp. Lung Res.* 7, 163-176.

Vincent, J.H. and Mark, D. 1982. Application of blunt sampler theory to the definition and measurement of inhalable dust. In: *Inhaled Particles V*, Proceedings of an International Symposium Organised by the British Occupational Hygiene Society, Cardiff, 8-12 September 1980, (Eds. Walton, W.H., Critchlow, A. and Coppock, S.M.), Pergamon Press, Oxford, United Kingdom. *Ann. Occup. Hyg.* 26, 3-19.

Vincent, J.H., Mark, D., Miller, B.G., Armbruster, L. and Ogden, T.L. 1990. Aerosol inhalability at higher wind speeds. *J. Aerosol Sci.* 21(4), 577-586.

Waite, D.A. and Ramsden, D. 1971a. The inhalation of insoluble iron oxide particles in the sub-micron range. Part I - Chromium-51 labelled aerosols. AEEW-R740, Atomic Energy Authority, Winfrith, Dorset, United Kingdom.

Waite, D.A. and Ramsden, D. 1971b. The inhalation of insoluble iron oxide particles in the sub-micron range. Part II - Plutonium-237 labelled aerosols. AEEW-R741,

Atomic Energy Authority, Winfrith, Dorset, United Kingdom.

Wanner, A. 1977. Clinical aspects of mucociliary transport. *Am. Rev. Respir. Dis.* 116, 73-125.

Watson, A.Y. and Brain, J.D. 1979. Uptake of iron oxide aerosols by mouse airway epithelium. *Lab. Invest.* 40, 450-459.

Watts, L. 1975. Clearance rates of insoluble plutonium-239 compounds from the lung. *Health Phys.* 29, 53-59.

Wayne, E., Koutras, D. and Alexander, W. 1964. Metabolism of inorganic iodine. In: *Clinical Aspects of Iodine Metabolism*. Blackwell Scientific Publications, Oxford.

Webber, C.E. and Harvey, J.W. 1975. Accidental human inhalation of ruthenium tetroxide. *Health Phys.* 30, 352-355.

Weeks, M.H., Katz, J., Oakley, W.D., Ballou, J.E., George, L.A., Bustad, L.K., Thompson, R.C. and Kornberg, H.A. 1956. Further studies on the gastrointestinal absorption of plutonium. *Radiat. Res.* 4, 339-347.

West, C.M. and Scott, L.M. 1966. A comparison of uranium cases showing long chest burden retentions. *Health Phys.* 12, 1545-1555.

West, C.M. and Scott, L.M. 1969. Uranium cases showing long chest burden retention - an updating. *Health Phys.* 17, 781-791.

West, C.M., Scott, L.M. and Schultz, N.B. 1979. Sixteen years of uranium personnel monitoring experience - in retrospect. *Health Phys.* 36, 665-669.

Widdowson, E.M., Slater, E.J., Harrison, G.E. and Stutton, A. 1960. Absorption, excretion and retention of strontium by breast-fed and bottle-fed babies. *Lancet* 2, 941-944.

Wilkey, D.D., Lee, P.S., Hass, F.J., Gerrity, T.R., Yeates, D.B. and Lourenço, R.V. 1980. Mucociliary clearance of deposited particles from the human lung: Intra- and inter-subject reproducibility, total and regional lung clearance, and model comparisons. *Arch. Environ. Health* 35, 294-303.

Willard, D.H. and Bair, W.J. 1961. Behaviour of ^{131}I following its inhalation as a vapour and as a particle. *Acta Radiol.* 55, 486-496.

Wolff, R.K. 1986. Effects of airborne pollutants on mucociliary clearance. *Environ. Health Perspect.* 66, 223-237.

Wolff, R.K., Tillquist, H., Muggenburg, B.A., Harkema, J.R. and Mauderley, J.L. 1989. Deposition and clearance of radiolabelled particles from small ciliated airways in beagle dogs. *J. Aerosol Med.* 2, 261-270.

Wong, J.W., Keens, T.G., Wannamaker, E.M., Crozier, D.N., Levison, H. and Aspin, N. 1977. Effects of gravity on tracheal mucus transport rates in normal subjects and in patients with cystic fibrosis. *Pediatrics* 60, 146-152.

Wood, R.E., Horowitz, J.G. and Doershuk, C.F. 1976. Bronchofiberscopic determination of tracheal mucus velocity (abstract). *Am. Rev. Respir. Dis., Suppl.* 113, 98.

Würtemberger, G., Montag, A. and Matthys, H. 1987. Effects of therapeutic inhalation on mucociliary and tussive clearance. In: *Deposition and Clearance of Aerosols in the Human Respiratory Tract*. Second International Symposium, Salzburg, Austria, September 18-20, 1986. (Ed. Hofmann, W.), Facultas Universitätsverlag Ges.m.b.H. Wien, Austria, pp. 168-173.

Xu, G.B. and Yu, C.P. 1985. Theoretical lung deposition of hygroscopic NaCl aerosols. *Aerosol Sci. Technol.* 4, 455-461.

Yamagata, N., Iwashima, K., Nagai, T., Watari, K. and Inuma, T.A. 1966. In vivo experiment on the metabolism of caesium in human blood with reference to rubidium and potassium. *J. Radiat. Res.* 7, 29-46.

Yan Shiao-Shan, Shan Yi, Lu Hin-Min, Lan Fu-Shing, Shiao Hin-Juan and Go Shin-G Liang. 1988. Deposition and clearance of $^{141}\text{CeO}_2$ in rats. In: *Inhaled Particles VI*, Proceedings of an International Symposium and Workshop on Lung Dosimetry Organised by the British Occupational Hygiene Society in Co-operation with the Commission of the European Communities, Cambridge, 2-6 September 1985, (Eds. Dodgson, J., McCallum, R.I., Bailey, M.R. and Fisher, D.R.). *Ann. Occup. Hyg.* 32, Suppl. 1, 957-962.

Yeates, D.B., Aspin, N., Levison, H., Jones, M.T. and Bryan, A.C. 1975. Mucociliary tracheal transport rates in man. *J. Appl. Physiol.* 39, 487-495.

Yeates, D.B., Spektor, D. and Pitt, B.R. 1979. Discontinuity of pulmonary mucociliary transport due to metaproterenol sulfate. *Physiologist* 22, (4) 136.

Yeates, D.B., Gerrity, T.R. and Garrard, C.S. 1981a. Particle deposition and clearance in the bronchial tree. *Ann. Biomed. Eng.* 9, 577-592.

Yeates, D.B., Pitt, B.R., Spektor, D.M., Karron, G.A. and Albert, R.E. 1981b. Coordination of mucociliary

transport in human trachea and intrapulmonary airways. *J. Appl. Physiol. Respirat. Environ. Exercise Physiol.* 51, 1057-1064.

Yeates, D.B., Gerrity, T.R. and Garrard, C.S. 1982. Characteristics of tracheobronchial deposition and clearance in man. In: *Inhaled Particles V, Proceedings of an International Symposium Organised by the British Occupational Hygiene Society, Cardiff, 8-12 September 1980*, (Eds. Walton, W.H., Critchlow, A. and Coppock, S.M.), Pergamon Press, Oxford, United Kingdom. *Ann. Occup. Hyg.* 26, 245-257.

Yeates, D.B., Raju, T., Larkridge, C., Musselman, R., Harshbarger, R., Gerrity, T.R. and Lourenço, R.V. 1983. A non-sedated primate model of bronchial mucociliary clearance. *Amer. Rev. Respir. Dis.* 127, 185 (Abstract).

Yu, C.P. 1981. A model of particle clearance in human tracheobronchial tree. In: *Proceedings of 34th ACEMB, Houston, Texas, September 21-23, 1981*, p. 39.

Yu, C.P., Hu, J.P., Yen, B.M., Spektor, D.M. and Lippmann, M. 1986. Models for mucociliary particle clearance in lung airways. In: *Aerosols: Research, Risk Assessment and Control Strategies*. (Eds. Lee, S.D., Schneider, T., Grant, L.D. and Verkerk, P.J.), Lewis, Chelsea, Michigan, pp. 569-578.

Zwas, S.T., Katz, I., Belfer, B., Baum, G.L. and Aharonson, E. 1987. Scintigraphic monitoring of mucociliary tracheo-bronchial clearance of technetium-99m macroaggregated albumin aerosol. *J. Nucl. Med.* 28, 161-167.

Question 1. Average ventilation rates, L min^{-1} , assuming a normal daily (24h) mix of activities (combined male, female average).

	5 year old children			Mature Adults		
	5%	50%	95%	5%	50%	95%
L min^{-1}	5	6.1	9	8	13.9	16.5

Question 2. Total initial deposition in the respiratory tract, % of total amount inhaled, normal daily (24 hr) mix of activities.

	0.1 μm AMAD			1 μm AMAD			10 μm AMAD		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Mature Adults	10	24	60	40	51	80	50	75	90
5 year old children				35	45	80			

Question 3. Initial deposition in the extrathoracic region, % of total deposition in the respiratory tract? (This question also gives the initial deposition in the lung (tracheobronchial (TB) + pulmonary (AI) regions), since lung = total - ET).

	0.1 μm AMAD			1 μm AMAD			10 μm AMAD		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Mature Adults	10	25	50	40	57	85	80	90	95
5 year old children				25	45	75			

Question 4. Initial deposition in the tracheobronchial (TB) region, % of the total deposition in the lung (TB + AI regions).

	0.1 μm AMAD			1 μm AMAD			10 μm AMAD		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Mature Adults	7	23	70	15	29	60	20	63	80
5 year old children				10	32	90			

Question 5. Assuming completely insoluble particles (1 μm AMAD), retention in each respiratory tract region in mature adults as a % of the total initial deposition in the respiratory tract, as a function of time after deposition.

	10 min			1 hr			1 day		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Tracheobronchial airways	5	12	23	4	11	21	0.2	6.5	13
Pulmonary (AI) region	20	30	40	20	30	40	20	30	40
	1 month			1 year			10 years		
Tracheobronchial airways	0.04	2.5	11	0.005	0.01	0.9	0.0001	0.0007	0.1
Pulmonary (AI) region	17	27	35	9	17	23	1.0	2.9	5

Question 6. By what factors would you expect the median values to be different in 5 year old children (1=no difference).

	10 min	1 hr	1 day	1 month	1 year	10 years
Tracheobronchial airways	1.4	1.4	1.4	1.4	1.3	1.0
Pulmonary (AI) region	1.2	1.2	1.2	1.2	1.1	1.1

Question 7. Absorption (dissolution and transfer) to blood in adults, % of the total initial deposition (1 μm AMAD particles) in the respiratory tract.

	1 hr			1 day			1 week		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Sr	2	30	70	6	35	76	7	36	76
I	41	72	80	49	82	90	49	82	90
Cs	8	59	81	25	70	90	27	0	90
Pu	0.04	0.6	7	0.04	0.6	7	0.05	0.8	8
Ru	0.05	5.9	35	0.32	7.9	37	0.35	8.9	38
Ce	0.04	2.9	13.9	0.05	3.2	14.3	0.07	4.1	15.3
Te	0.05	6	79	0.32	9.7	83	0.35	10.9	83
1 month			1 year			10 years			
Sr	9	38	76	18	46	76	19	47	76
I	49	82	90	49	82	90	49	82	90
Cs	31	71	90	42	73	90	42	73	90
Pu	0.1	1.5	12	0.5	6.8	29	1.6	13	32
Ru	0.4	12.1	40	0.9	25	49	2.1	27	51
Ce	0.1	7.2	19	0.5	21	34	1.6	23	36
Te	0.4	14	83	0.9	28	83	2.1	30	83

Question 8. Factors by which the median values would be different in 5 year old children (1=no difference).

5 year old children	1 hr	1 day	1 week	1 month	1 year	10 years
Sr	1.1	1.1	1.1	1.1	1.1	1.1
I	1.1	1.1	1.1	1.1	1.1	1.1
Cs	1.1	1.1	1.1	1.1	1.1	1.1
Pu	1.2	1.2	1.2	1.3	1.2	1.2
Ru	1.2	1.1	1.1	1.2	1.2	1.2
Ce	1.2	1.2	1.2	1.2	1.2	1.3
Te	1.2	1.1	1.1	1.1	1.2	1.2

Question 9. Absorption to blood as a fraction (f_1) of activity ingested.

	3 month old infants			5 year old children			Adults		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Sr	0.15	0.4	0.75	0.1	0.3	0.5	0.1	0.2	0.4
I	0.9	0.95	1	0.9	0.95	1	0.9	0.95	1
Cs	0.8	0.9	1	0.8	0.9	1	0.8	0.9	1
PuO ₂ *	1×10^{-6}	3×10^{-5}	5×10^{-4}	1×10^{-6}	1×10^{-5}	5×10^{-5}	1×10^{-6}	1×10^{-5}	5×10^{-5}
Pu bio†	1×10^{-4}	2×10^{-3}	1×10^{-2}	1×10^{-4}	5×10^{-4}	1×10^{-3}	1×10^{-4}	5×10^{-4}	1×10^{-3}

* Refractory oxide
 † "Biologically incorporated"

Question 10. Considering the total amount reaching blood (as if administered intravenously as a single injection). % retained in liver and skeleton (bone + bone marrow), as a function of time after entry into blood.

	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
Skeleton and liver, 1 day						
Ce	60	80	90	60	80	90
Skeleton and liver, 1 week						
Ce	60	82	90	59	81	90
Skeleton and liver, 1 month						
Ce	58	83	90	58	83	90
Skeleton and liver, 1 year						
Ce	43	81	88	39	81	86
Skeleton and liver, 10 years						
Ce	20	54	76	12	59	75
Skeleton and liver, 50 years						
Ce	8	29	51	5	34	50

Question 11. Retention in the skeleton, % total retention in liver + skeleton (bone + marrow), as a function of time after entry into blood.

	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
Skeleton, 1 day						
Ce	45	63	82	19	38	57
Skeleton, 1 week						
Ce	47	65	83	20	40	60
Skeleton, 1 month						
Ce	50	66	83	21	42	63
Skeleton, 1 year						
Ce	55	70	85	27	51	76
Skeleton, 10 years						
Ce	60	83	92	79	86	93
Skeleton, 50 years						
Ce	89	93	97	91	94	97

Question 12. Retention of plutonium on endosteal bone surfaces (considering a 10 μm depth of bone mineral) as a % of total skeletal retention, as a function of time after entry into blood.

		5 year old children			Adults		
		5%	50%	95%	5%	50%	95%
Endosteal bone surface	1 day						
	1 week						
	1 month						
	1 year						
	10 years						
	50 years						

Question 13. Retention of plutonium on trabecular surfaces, % total endosteal surface retention as a function of time after entry into blood.

		5 year old children			Adults		
		5%	50%	95%	5%	50%	95%
Trabecular bone surface	1 day						
	1 week						
	1 month						
	1 year						
	10 years						
	50 years						

Question 14. Retention of plutonium in red bone marrow as a % total skeletal retention, as a function of time after entry into blood.

		5 year old children			Adults		
		5%	50%	95%	5%	50%	95%
Red bone marrow	1 day						
	1 week						
	1 month						
	1 year						
	10 years						
	50 years						

Question 15. Considering the total amount reaching blood (as if administered intravenously as a single injection), % total retention (whole body or systemic) as a function of time after entry into blood.

	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
1 day						
Ru	50	83	95	60	83	90
Cs	90	95	98	92	96	98
1 week						
Ru	30	65	80	35	65	75
Cs	60	73	85	80	85	90
1 month						
Ru	15	39	70	20	39	60
Cs	20	32	50	60	70	80
1 year						
Ru	0.5	16	60	1	16	50
Cs	1×10^{-3}	1×10^{-2}	5	1	4	10
5 years						
Ru	1×10^{-3}	6	30	1×10^{-2}	6	20
Cs	1×10^{-20}	1×10^{-17}	1	1×10^{-6}	1×10^{-5}	1

Question 16. Considering the total amount of Iodine reaching blood (as if administered intravenously as a single injection), % retained in the thyroid as a function of time after entry into blood.

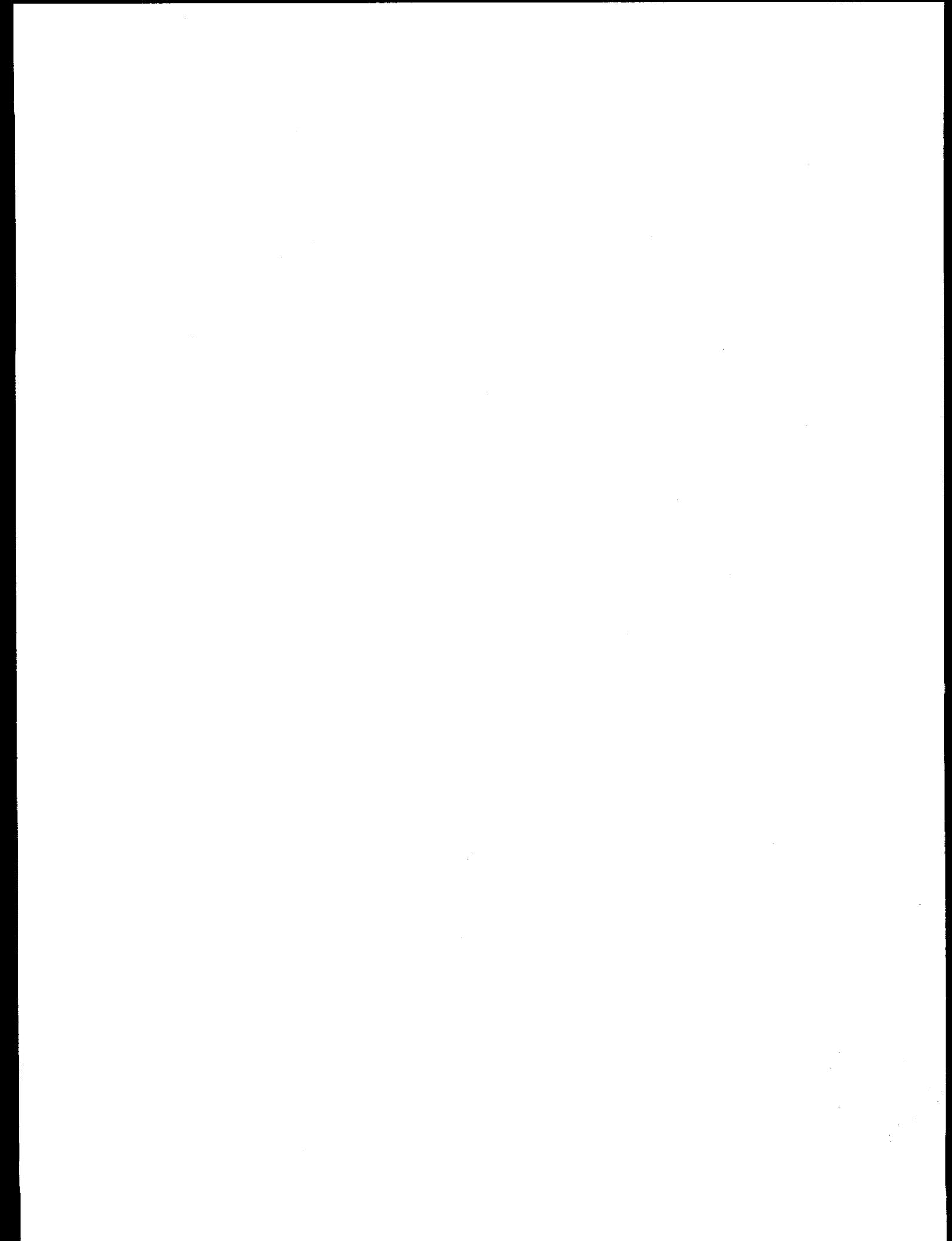
	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
1 day						
1 week						
1 month						
3 months						

Question 17. Intake dose coefficients, absorbed dose to specified organs or tissue per unit activity inhaled or ingested (committed equivalent doses), Gy Bq⁻¹.

	5 year old children			Adult		
	5%	50%	95%	5%	50%	95%
⁹⁰ Sr, inhalation, 1 μm AMAD						
lung						
bone marrow						
bone surface						
⁹⁰ Sr, ingestion						
colon						
bone marrow						
bone surface						
¹³¹ I, inhalation, 1 μm AMAD + vapor (decide proportions)						
thyroid						
¹³¹ I, ingestion						
thyroid						
¹³² Te, inhalation, 1 μm AMAD + vapor (decide proportions)						
lung						
thyroid						
colon						
¹³⁷ Cs, inhalation, 1 μm AMAD						
lung						
colon						
stomach						
bone marrow						
¹³⁷ Cs, ingestion						
lung						
colon						
stomach						
bone marrow						
¹⁴⁴ Ce, inhalation, 1 μm AMAD						
bone surface						
lung						
bone marrow						
liver						
²³⁹ Pu, inhalation, 1 μm AMAD						
bone surface						
bone marrow						
liver						
lung						
²³⁹ Pu, ingestion						
bone surface						
bone marrow						
liver						
colon						

(xi) Joint dosimetry/late effects question: The number of radiation-induced cancer deaths up to 40 years following exposure in a population of a hundred million persons (5×10^7 male, 5×10^7 female) each of whom inhales 10 K Bq of the radionuclides specified.

Nuclide	Physical Form	Chemical Form	Cancer Type	Number of Cancers			
				Quantile	5%	50%	95%
Pu-239	1 μ m AMAD	Oxide	Lung				
			Bone				
			Liver				
			Leukemia				
			All cancers				
Sr-90	1 μ m AMAD	Oxide	Lung				
			Bone				
			Leukemia				
			All cancers				



EXPERT B

General Considerations

Sources of information underlying biokinetic estimates

As a rule, our knowledge or beliefs concerning the biokinetics of a radioelement in humans come mainly from one or more of these four sources of information:

H1: Observations (reported measurements) of the behavior (absorption, distribution, retention, and/or excretion) of the element in human subjects during or after intake of the element.

Surrogates for H1:

H2: Observations of the behavior of one or more chemically similar elements in human subjects.

A1: Observations of the behavior of the element in non-human species.

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

Some quantities such as fractional deposition of an inhaled radionuclide in a segment of the respiratory tract appear to be virtually independent of the element. In such cases, these four sources of information collapse into two categories (in effect): H1 and A1.

The sources H1, H2, A1, and A2 are sometimes supplemented with, or constrained by: quantitative physiological information (e.g., turnover rates of bone mineral or of certain types of cells); considerations of mass balance (sum of organ contents and body losses must equal intake); predictions of theoretical models based on fundamental physical, chemical, and mathematical principles (e.g., deposition model for respiratory tract); experimental data using anatomically realistic physical models (e.g., hollow casts of portions of respiratory tract); and *in vitro* data (e.g., dissolution of compounds in simulated lung fluid). Among these supplemental sources of information, quantitative physiological data and mass balance have particularly wide use. The extent to which physiological data and mass balance can be used to improve or bound biokinetic estimates for an element depends on the conceptual (model) framework used to reduce, collect, organize, and extrapolate data from the various sources. As a rule, a biologically realistic conceptual framework provides a stronger logical basis for introducing quantitative

physiological data than does the conventional, biologically meaningless, curve-fitting approach to biokinetic modeling used, for example, in Publication 30 of the International Commission on Radiological Protection (ICRP, 1979). With a biologically realistic ("physiological systems") framework, mass balance is a built-in constraint, because losses from one part of the system must be registered as gains in another part. Despite the increased accommodation of physiological information and the advantages of built-in mass balance, however, we should not lose sight of the difficulties and uncertainties in representing biological reality in any conceptual framework.

We first examine, in a general setting, some common experimental or observational problems associated with each of the major sources of data (H1, H2, A1, and A2) and additional problems in using observations of type H2, A1, and A2 as surrogates for observations of type H1. We then apply these considerations to the elicitation questions.

Type H1 information

The present project addresses several situations in which there is a relatively large body of H1 data from which to derive biokinetic estimates. For example, H1 data represent the largest and most useful source of information on total and regional deposition in the respiratory tract and removal from these regions. Also, there are abundant high-quality H1 data on the total-body retention of Cs and on uptake and retention of Sr by the skeleton. There are also reasonably good H1 data on uptake and retention of iodine by the thyroid and on the distribution and retention of absorbed Pu. However, Cs, Sr, I, and Pu are not typical radioelements with regard to available biokinetic information; rather, these are some of the most extensively studied radioelements.

Theoretically, a large amount of carefully derived H1 data can provide a high level of confidence in an estimate of typical biokinetics of a radioelement in a human subject or population. In practice, however, H1 data for a given radioelement usually have one or more of the following limitations:

1. Small population size. In many cases, data are available for only one or two subjects.
2. Small samples of tissue. Estimates may be based on small samples of organs and tissues in which the radioelement is potentially heterogeneously distributed. This is a particularly troublesome problem for the skeleton, whose metabolic activity

varies considerably from one bone to another and within different regions of the same bone.

3. Potentially atypical study population. The subjects may be unhealthy or otherwise potentially atypical with regard to the biokinetics of the radioelement.
4. Problems with measurement techniques. The measurement techniques may be inherently inaccurate or difficult to calibrate, or may require considerable subjective interpretation.
5. Uncertain intakes. The pattern or level of intake of the measured radioisotope often may be unknown or poorly characterized. This is particularly true of environmental or occupational exposures but may also occur in relatively controlled studies.
6. Relevance of study conditions. The conditions of the study may be considerably different from real-world conditions of exposure (e.g., determination of lung deposition by controlled breathing through a mouth tube).
7. Inconsistency in reported values.

Example: Biokinetics of uranium in bone

The skeletal biokinetics of U provides a classical example of problems that can plague H1 data. H1 data for U in bone come mainly from an experimental study on several subjects and from autopsy measurements on occupationally and environmentally exposed subjects (Leggett, 1994).

The experimental H1 data for U in bone come from a study in which 11 comatose subjects, all suffering from terminal diseases of the central nervous system, were intravenously injected with U compounds. The subjects died at times ranging from a few days to 1.5 years after injection. The conditions of these subjects are likely to have resulted in atypical uptake and/or release of divalent cations, including the uranyl ion, by bone. Bone samples collected by biopsy or autopsy were small and may not have been representative of the entire skeleton. The mass of the total skeleton or of total bone was not known for any of the subjects. Because of difficulties with the injection apparatus or discrepancies between protocol sheets and analysis of dummy injections, the mass of injected U is in doubt for some subjects. The subjects were administered high masses of U, which may have altered the biokinetics of this element. Finally, as with injection studies in general, there is some question as to

whether the early behavior of the injected U compounds was the same as that of U that is absorbed to blood.

The environmental and occupational H1 data for U in bone also have considerable problems. For example, only a small number of subjects have been examined; the exposure histories of the subjects are poorly known; and there are considerable uncertainties in estimates of total-organ contents of the subjects based on small samples of skeletal tissues. Also, in the case of environmentally exposed subjects, there have been considerable problems over the years with the measurement techniques used to determine the typically low concentrations of environmental U in human tissues and fluids.

Current estimates of the biokinetics of U in the human skeleton, and our confidence in these estimates, are influenced to some extent by all three sources of H1 data (i.e., experimental, occupational, and environmental findings). For many years the prevailing biokinetic models for U were derived as curve fits to the experimental H1 data. The ICRP's new biokinetic model gives more weight to the occupational and environmental H1 data than to the experimental H1 data. The new model is also influenced by information of types H2 and A1 and organizes the information within a biologically meaningful framework that requires the predictions to be consistent with human physiological information. Still, there remains considerable uncertainty in estimates of U in bone as a function of time after introduction of this element to blood.

Type H2 information

Chemical analogy plays an important role in biokinetic modeling of radionuclides in general. With regard to the present elicitation questions, consideration of chemical analogy is most important for consideration of the systemic biokinetics of Ce and of gastrointestinal absorption of Pu.

The use of chemical analogy in biokinetic modeling is based on empirical evidence that chemically similar elements often exhibit close physiological similarities. Some examples of chemically similar element pairs that have proved to be close physiological analogues are Sr-Ca, Ra-Ba, Cm-Am, and K-Rb. In each case, the two elements appear to be closely related in virtually all physiological processes that have been studied.

The chemical analogy is not limited to element pairs that appear to be physiologically similar in all respects. There are groups of element that share similar biokinetics in some but not all respects. For example, lead appears to be a

physiological analogue for calcium in bone but not for calcium in the kidneys.

In most cases, our knowledge concerning the physiological relationships of element pairs comes mainly from comparative studies in laboratory animals. Therefore, the value of H2 data for an element may depend on the availability of A1 and A2 data, i.e., comparative data on the element and its chemical analogue(s) in laboratory animals.

The level of confidence that can be placed in an estimate for radionuclide A based solely on human data for radionuclide B depends on the quality and completeness of the data for B, and the expected strength of the analogy for the given situation. The strength of the chemical analogy depends on the extent to which the physiological relationship of the element pair has been studied, and the physiological closeness of the element pair as revealed by the studies.

H2 data have the same potential limitations as listed earlier for H1 data but also involve additional uncertainties associated with inter-element extrapolation. Compared with application of H1 data, a reduced level of confidence should be placed in estimates based on H2 data because:

1. There are counterexamples to the premise that chemical analogues are also physiological analogues. For example, K and Na show strong chemical similarities but qualitatively different behaviors in the body. Uranium is an actinide element but behaves much differently from the other frequently studied actinides such as Pu, Am, Cm, and Th.
2. Chemically similar elements that behave in a qualitatively similar fashion in the body may exhibit different biokinetics from a quantitative standpoint. For example, Cs follows the movement of potassium in the body but its retention half-time in the body is roughly three times that of potassium in healthy adult humans.
3. As discussed above, the fact that two elements exhibit similar behavior in one organ does not imply that they will have similar behavior throughout the body.

Type A1 or A2 information

Most of our knowledge of the biokinetics of Ce, Ru, and Te (among the radioelements addressed in this project) comes from experimental data on laboratory animals. The

following background information will help to explain the selection of confidence intervals for biokinetic estimates for these elements. More specific rationales are given in a later section.

Data on laboratory animals typically involve some of the same problems as those listed above for data types H1 and H2, e.g., a small number of subjects, small samples from organs with heterogeneously distributed radioactivity, inaccurate measurement techniques, uncertain patterns of intake, and irrelevant experimental situations. Such problems often introduce smaller uncertainties in animal studies than in human studies, mainly because of the greater limitations on radionuclide administration and sample collection in humans. On the other hand, the uncertainties associated with such problems often seem small compared with uncertainties associated with interspecies extrapolation.

Interspecies extrapolation of biokinetic data is based on the concept of a "general biological regularity" (Krasovskii, 1976; Calabrese, 1984) 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, generally are believed to provide better analogies to man than do non-mammalian species with regard to biokinetics of contaminants.

Despite such qualitative similarities among mammalian species, interspecies extrapolation of biokinetic data has proven to be an uncertain, error-prone process. Two species that handle an internally deposited radionuclide in the same qualitative manner may exhibit dissimilar kinetics with regard to that substance. For example, Cs appears to follow essentially the same paths of movement in all mammals but exhibits considerably different whole-body retention times in different mammalian species.

Moreover, there are many counterexamples to the presumed qualitative biological regularity among mammalian species. That is, there are important structural, functional, and biochemical differences among the mammalian species. For example, species differ to some extent with regard to number and functions of organs in the gastrointestinal system, relative length of the digestive tract, types of bacteria in the digestive tract, the microstructure and vasculature of bones, patterns of modeling and remodeling of bone, level of biliary secretion, urine volume and acidity, primary mode of excretion of elements, and amount and types of fat.

Despite such problems, our overall experience has been that biokinetic data derived for laboratory animals have usually provided reasonably good indications of the biokinetics of radioelements in humans, particularly when data were available for several non-human species. The problem here is one of quantifying "usually" and "reasonably good". Both may depend strongly on how the extrapolation is done.

The ideal situation for interspecies transfer of unscaled data is that data are available for a variety of species, and the data suggest that the biokinetic entity is reasonably independent of species. If data exist for several species but vary substantially from one species to another, consideration must be given to the problem of whether certain species may be more "human-like" than others with regard to the biokinetic estimate in question.

No single animal model has been found that consistently predicts human biokinetics of radioelements, even on a compressed or expanded scale. However, our collective experience gives us lower expectations for some species than others with regard to extrapolation of biokinetic data to humans. For example, rats (the most commonly used laboratory model for man) seem to show particularly many qualitative dissimilarities from man. Compared with humans, rats generally have lower protein binding, higher biliary secretion, higher urinary concentrations, and lower urinary excretion of contaminants. Rats are obligatory nose breathers, which results in passage of the entire inhaled dose through the upper respiratory tract, while humans may breathe through the nose or mouth. Rats have numerous flora in the stomach and proximal small intestine, while humans have little or none. The skeletons of rats have somewhat different vasculature, modes of ossification, and patterns of development from those of humans and, unlike the human skeleton, the rat skeleton continues to grow throughout adulthood.

Some of the better models overall appear to be non-human primates (particularly baboons), dogs, and pigs. Still, confidence in extrapolation of biokinetic data from these species to man is limited by their occasional "unhuman-like" biokinetics. For example, compared with humans, dogs have much slower elimination of heavy alkaline earth elements via the GI tract. Dogs also show higher uptake of Am in the thyroid than do other studied species. Non-human primates generally show much faster removal of actinides and lanthanides from the liver than humans. Compared with humans, pigs have very short retention of Cs. Practically all non-human species show much faster removal of Pb from blood than humans.

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. This is because a given species may resemble humans with regard to certain processes and subsystems and not others.

A particularly troublesome aspect of interspecies extrapolation of biokinetic data has been the large differences between species in hepatic retention of elements. For example, different mammalian species show enormously different rates of removal of transuranic elements from the liver, perhaps reflecting differences in the ability to secrete these metals in bile and/or dissimilarities among species in lysosomal composition and function. Rats, tree shrews, macaque monkeys, and baboons show rapid loss of Pu from the liver, with half-times of 4-200 d, while another set of adult animals with an overlapping range of body weights, including hamsters, deermice, grasshopper mice, dogs, pigs, and humans, show tenacious retention of Pu in the liver, with half-times measured in years or decades (Taylor, 1984). Mice and rats have been shown to excrete many essential and non-essential elements in bile better than other commonly studied laboratory animals. For example, the rate of biliary secretion of Cd, Ag, Pb, As, or Mn is many times higher in rats than in either dogs or rabbits (Klaassen and Watkins, 1984).

Because of the apparent quantitative similarities between beagles and man with regard to hepatic handling of many radionuclides including actinides and lanthanides, data on beagles might be given relatively high weight for purposes of modeling the biokinetics of Ce in humans. With regard to Ru and Te, there does not appear to be much logical basis for choosing among the studied animal species.

Many examples have been provided in the literature in support of allometric scaling, i.e., scaling of biokinetics to humans on the basis of body size. However, there are also numerous counterexamples to the "allometric principle". For example, on the basis of comparative results for dogs, monkeys, mice, and rats, allometric scaling of integrated whole-body retention (a fairly gross measure) works fairly well for Zn, Mn, Ag, and Be but fails for Ir, Nb, Co, Sn, Ru, and Se (Furchner et al., 1971). The allometric principle also fails with regard to the biokinetics of the heavy alkaline earth elements, the actinide elements, and the lanthanide elements. Because of the many counterexamples to the allometric principle, generalization cannot confidently be made about rates of unknown metabolic reactions based on body size. For retention at the organ level, allometric

scaling often provides a worse estimate than no scaling at all. Of the radioelements and biokinetic quantities considered in this project, allometric scaling would not appear to add any confidence in any case.

Perhaps the highest potential level of confidence from interspecies extrapolation comes in the general situation in which animal studies reveal the general physiological processes that govern the biokinetics of the element, and there is reasonably good quantitative data on those processes in humans. In this situation, one could extrapolate only general conclusions concerning qualitative physiological processes from animals to humans and could then derive quantitative estimates directly from physiological data for humans. The situation in which this method of extrapolation has been most frequently applied is for bone-seeking radionuclides, whose long-term kinetics generally appear to be controlled largely by processes of bone restructuring. Some use has also been made of such considerations as cell turnover rates, regional blood flow rates, and glomerular filtration.

A physiological systems 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. However, physiological systems modeling in general and this method of extrapolation in particular have obvious limitations. The most important limitations are that either the processes governing the biokinetics of an element or typical rates of those processes in humans, or both, are often not well established.

To summarize, the confidence to be placed in (scaled or unscaled) A1 data depends on:

the strength of the estimates for individual animal species (quality, completeness, and consistency of intra-species observations);

the consistency of (scaled or unscaled) data for different species;

the strength of the logical basis for interspecies extrapolation of data (including selection of preferred species, if applicable).

Selection of Confidence Bands

General comments on questions regarding the respiratory tract

ICRP Publication 66 (1994) provides a detailed review of information on the behavior of inhaled particles in the respiratory tract and on qualitative limitations in those data. The present discussions of data on initial deposition of inhaled material and its subsequent rates and paths of clearance from the respiratory tract are based in large part on discussions given in that document. The reader is referred to ICRP Publication 66 for more detailed discussions of the original data and for citations of the original studies.

The ICRP's respiratory tract model appears to provide reasonable central estimates of deposition and translocation of material in the respiratory tract. Except for some rounding or other slight modifications, the 50% values given as answers to the questions 1-5 and 7 below were derived from that model. However, the confidence bands placed here on the biokinetic parameters are usually different from confidence bands suggested in ICRP Publication 66.

Question 1. Average ventilation rates, $L \text{ min}^{-1}$, assuming a normal daily (24 h) mix of activities (combined male, female average)?

Discussion

There is considerable age- and gender-specific information on breathing rate and tidal volume (volume of inspired air) in individuals at rest or performing selected tasks of varying physical intensity. For any given age, gender, and level of activity, there is reasonable agreement in ventilation rates (tidal volume times breathing frequency) determined in different studies. While ethnic variability exists in lung volumes, flow rates, and tidal volume, minute ventilation at rest and at various levels of exercise does not appear to be very different between the ethnic groups that have been studied (ICRP, 1994).

The average ventilation rate is estimated as a time-weighted average of ventilation rates for rest periods and periods of light and heavy activity. The most troublesome aspect of determining the average ventilation rate for a population is the characterization of a normal daily mix of activities. A normal mix of daily activities may be more difficult to define for young children than for adults. The "time budgets" used in ICRP Publication 66 (1994) were based mainly on surveys conducted in the 1970s and 1980s and

involving thousands of men, women, and children from a few different countries. Hence, these time budgets are not arbitrary, but a nontrivial error might be expected to arise in applications of these budgets to any given population or to the world population.

Estimated average ventilation rates for adult males usually fall in the range $13\text{-}17 \text{ L min}^{-1}$ (UNSCEAR, 1982; ICRP 1975; ICRP, 1987; Roy et al., 1987; Roy and Courtay, 1991; ICRP, 1994). Estimated values for adult females and 5-year-old children are usually about 0.8 and 0.4 times those for adult males, respectively.

Layton (1993) proposed a considerably different approach for estimating average ventilation rates based on the body's oxygen requirements. The reasoning is that total air intake must be metabolically consistent with reasonably well established age- and gender-specific energy expenditure. Layton's method yields substantially lower values for adults than the time-budget approach, but the two methods give reasonably consistent values for children. Specifically, Layton estimated average ventilation rates of about 10 (5.5-13) L min^{-1} for adult males, 7 (5-9) L min^{-1} for adult females, and 5.5 (3.5-7.5) L min^{-1} for 5-year-old children. The values in parentheses indicate differences between relatively inactive and relative active subgroups of the population, rather than uncertainty in the central value. While Layton's method is based on some assumptions that could lead to underestimation of air intake (ICRP, 1994), they still raise doubts about the accuracy of the time-budget method.

Rationale for confidence intervals

The Layton method suggests a slightly lower 50% value than given in ICRP Publication 66. The 5% and 95% confidence levels are an attempt to quantify qualitative conclusions that the average ventilation rate is reasonably well established but not quite as precisely known as commonly supposed. It was considered that there is relatively plentiful, high-quality experimental data on ventilation rates for humans of all ages and both genders, but translation of these data to average ventilation rates relies on incomplete information on time budgets.

Question 2. Total initial deposition in the respiratory tract, % of total amount inhaled, normal daily (24 hr) mix of activities?

Discussion

Total deposition has been studied extensively in human subjects, but most studies have involved adult Caucasian males and particle sizes of 1-10 μm aerodynamic diameter (ICRP, 1994). While the sparsity of age- and gender specific data introduces some uncertainty into estimates for the general population, these uncertainties do not appear to be great for three reasons: (1) age- and gender-specific deposition can be mimicked by relatively sophisticated theoretical deposition models; (2) there are some scattered age- and gender-specific data that allow spot checks of the models; and (3) the adult male respiratory tract is a reasonably good surrogate (model) in most respects for respiratory tracts in women and children. Gaps in human data on deposition of submicron particles have also been filled in to some extent with theoretical models and with experiments involving physical models (e.g., anatomically accurate hollow casts of nasal passages, naso-oropharynx, and larynx).

Because the filtration efficiencies of the nose and mouth are different, a subject's breathing habit (ratio of nose breathing to mouth breathing) has a fairly strong effect on the amount of inhaled material that deposits in the lungs. This factor introduces a non-trivial uncertainty into estimates of average deposition of inhaled particles because there is considerable variability in breathing habits, even among healthy persons of the same age and gender. Estimates of average total deposition of inhaled particles are generally made by combining experimental data for mouth breathing with separately derived information for deposition in the extrathoracic regions. Hence these estimates are sensitive to errors in the characterization of typical breathing habits in the population.

The data on adult males, coupled with physical and theoretical models of deposition in the extrathoracic regions, establish general patterns of deposition as a function of particle size and ventilation rates. While qualitative patterns of deposition seem firmly established, quantitative deposition for a given particle size is less well established. This is particularly true for particles of AMAD in the region of 0.1 μm because of the limited amount of human data and because the deposition curve is steep and hence highly sensitive to small changes in particle size in this region. There is also a high sensitivity to particle size in the region of 1 μm , but to a lesser extent. By contrast, deposition is fairly insensitive to particle size in the range of 10 μm .

Rationale for confidence intervals

It was concluded that total deposition of inhaled particles of diameter 1-10 μm (AMAD) is reasonably well characterized in adult male Caucasian subjects. For the studied particle sizes, uncertainties in estimates of average deposition in the population stem mainly from incomplete knowledge of typical breathing habits and of the effects of age and gender on deposition. Despite such uncertainties, general patterns of deposition as a function of particle size seem reasonably well established. For example, it seems reasonably well established that total deposition will represent on the order of three-fourths of inhaled material of size 10 μm (AMAD) and roughly half of 1- μm material. Deposition is less well established for submicron particles, but extremely low deposition or extremely high deposition of 0.1 μm particles seems unlikely in view of available information.

Question 3. Initial deposition in the extrathoracic region, % of total deposition in the respiratory tract? (This question also gives the initial deposition in the lung (tracheobronchial (TB) + pulmonary (AI) regions), since lung = total - ET)

Discussion

Deposition in the ET region is generally estimated in parts (ICRP, 1994), i.e., as the sum of deposition fractions determined separately for the nose and oropharyngeal-laryngeal regions. Because processes that govern deposition of particles in the extrathoracic region depend strongly on particle size, different measurement techniques have been developed to measure different ranges of particle size. Particles with an aerodynamic diameter larger than about 0.5 μm are deposited primarily by aerodynamic transport processes of inertial motion (impaction) and gravitational settling. For very large particles and fibers, interception with surfaces in the extrathoracic airways also contribute to their deposition. Particles with an equivalent physical diameter less than a few tenths of a micrometer are deposited primarily by the thermodynamic transport process of Brownian diffusion.

Aerodynamic deposition in the nose is much better documented than aerodynamic deposition in other parts of the respiratory tract because this is the most easily observed portion of the tract. However, there are discrepancies in nasal filtration estimates obtained by different techniques. The different measurement techniques include: measurement of the change in particle concentration when an aerosol is drawn in through the nose and out through the

mouth during breath holding; external detection of deposited radioactivity after inhalation of labeled particles; and unfolding of the efficiencies of nasal deposition after measurement of the total respiratory tract deposition under different breathing maneuvers (nose breathing, pure mouth breathing, nose-in mouth-out, and mouth-in nose-out breathing). Measurements of nasal deposition efficiency during normal breathing are broadly consistent from one study to another and indicate that the nasal deposition efficiency increases steadily with the impaction parameter $p = d^2 \times V$, where d is the aerodynamic particle diameter in μm and V is the volumetric flow rate in $\text{cm}^3 \text{s}^{-1}$. The data indicate a deposition efficiency centered near 0.1 for $p < 500 \mu\text{m}^2 \text{cm}^3 \text{s}^{-1}$, 0.2 for $p = 1000$, 0.5 for $p = 10,000$ and > 0.8 for $p > 30,000$. Considerable scatter in the data has been observed at each value of p . Except for high values of the impaction parameter, artificial aspiration of particles through the nose has tended to give lower measured values of nasal deposition than measurements during normal breathing. The extent of scatter in the data was similar for artificial aspiration and normal breathing.

Nasal deposition for submicron-sized particles has not been studied much in human subjects, and the few available results for humans were based on assumptions that later proved to be incorrect (ICRP, 1994). Perhaps the best available information on nasal deposition of small particles comes from measurements of deposition in anatomically accurate hollow casts of nasal passages, naso-oropharynx, and larynx. The casts were made from MRI scans of two adult males (one postmortem) and a 1.5-y-old child. The deposition efficiency plotted against the deposition parameter $D^{0.5}V^{1/8}$ (D = particle diffusion coefficient in $\text{cm}^2 \text{s}^{-1}$ and V = volumetric flow rate through the nose) shows relatively little scatter, and the data for the young child does not differ much from that for the adults. The small amount of variability in measured deposition efficiency in these casts has been interpreted by some authors as indicating that the nasal deposition of submicron-sized particles is well known. However, because of the small number of casts and the fact that deposition in casts is probably an imperfect surrogate for actual nasal deposition, it appears that there remains a fairly sizable uncertainty in fractional deposition of submicron-sized particles.

Most experimental studies of aerodynamic deposition in the oropharyngeal-laryngeal region have been performed with mouth breathing through a tube, since this is a convenient method for aerosol administration. Oral deposition is measured by repeated mouth washings directly after inhalation. The remainder of the extrathoracic deposition (oropharynx and larynx) is measured by external gamma

counting. In some cases, activity in the stomach has also been measured within a short time of deposition and assumed to represent transfer of material from extrathoracic airways to stomach. The results of the studies are weakened by their apparent dependency on the structure and positioning of the mouthpiece and evidence that normal mouth breathing gives higher extrathoracic deposition than breathing through a mouthpiece. Also, determination of typical deposition in the oropharyngeal-laryngeal region is complicated by a large intersubject variability in extrathoracic deposition, most likely due to differences in the morphology and physiology of the region, especially the larynx and vocal cords. Thus, the experimental results are not sufficient to model aerodynamic deposition in the mouth and lower extrathoracic regions with much accuracy. However, there is enough information to conclude that for particles less than about 10 μm aerodynamic diameter, the bulk of the extrathoracic deposition during mouth breathing occurs in the larynx.

Deposition of submicron particles in the oropharyngeal-laryngeal airways has not been studied systematically *in vivo* in human subjects. As is the case for nasal deposition of small particles, reliance must be placed on particle deposition in hollow airway casts. Limited experiments using hollow casts indicated slightly higher deposition in the oropharyngeal-laryngeal airways than in the nasal passageway of the same individual, but there is some question concerning the anatomical realism (positioning of the tongue during open-mouth breathing) in the hollow casts.

Rationale for confidence intervals

It was concluded that reasonably high confidence should be placed in the ICRP Publication 66 estimates of aerodynamic deposition (e.g., 1 or 10 μm particles) in the extrathoracic region, which are based on easily interpreted human data. Less confidence should be placed in estimates of deposition of submicron particles, which has been studied using anatomically accurate hollow casts. For aerodynamic deposition, experimental data are sufficient to characterize nasal deposition with reasonable accuracy but are not sufficient to estimate aerodynamic deposition in the mouth and lower extrathoracic region with much confidence. Despite uncertainties associated with submicron particles and lower extrathoracic deposition of larger particles, it can be discerned from the collective data that deposition in the extrathoracic region increases with particle size and volumetric flow rate. During normal breathing at rest, deposition in the extrathoracic region appears to represent a relatively small portion of total deposition of 0.1- μm

particles but probably much more than half the total deposition of particles of diameter 1 μm or more. Confidence bands for fractional deposition in the ET regions are narrowed considerably by the facts that: (1) there is reasonably good information on the sizes of long-term retention components in the respiratory tract in humans as well as laboratory animals; and (2) it is known that long-term retention occurs almost completely in the lungs and not the ET regions.

Question 4. Initial deposition in the tracheobronchial (TB) region, % of the total deposition in the lung (TB + AI regions)?

Discussion

There have been several studies of regional deposition of material in the lungs of human subjects, but the experimental data are not sufficiently complete or consistent to define the division between the TB and AI regions in the adult male or the effects of different airway size in other subjects. Estimates of regional deposition as a function of age and gender must be based mainly on a theoretical model of the lungs. The theoretical model used in ICRP Publication 66 (1994) evaluates the combined effects of convective and diffusive gas transport, and aerosol loss processes within the airways of the lungs. Predictions of the theoretical model are consistent with measured values of total lung deposition over a wide range of particle sizes.

The published experimental determinations of regional deposition in the respiratory tract are generally interpreted in terms of assumptions and/or models that may not be entirely correct. For example, the data as presented in the literature generally rely on the premise that particles deposited in distinct anatomical regions (e.g., the ciliated tracheobronchiolar airways or the respiratory airways of the alveolar interstitium) are cleared with distinctly different rates. In fact, in experimental studies with human subjects, two distinct phases of particle clearance from the lungs are usually observed: an initial rapid phase succeeded by a slow phase with a fitted half-time of several tens of days. However, results of recent human studies indicate that a substantial fraction of particles deposited in the tracheobronchiolar region may have been cleared at a slow rate that was indistinguishable from clearance from the alveolar-interstitial airways (ICRP, 1994). Thus, the fundamental premise of uniformly rapid ciliary clearance may not be valid.

There are no experimental data on regional lung deposition in children. However, there are some measurements of total

thoracic (lung) deposition in a group of children of average age about 10 y and in another group of average age about 5 y. The data agree reasonably well with the theoretical deposition model, but only a small range of particle sizes was tested. Thus, total lung deposition is less well defined in children than in adults.

Rationale for confidence intervals

Interpretation of the available human data on TB versus AI deposition depend on the assumption that the two regions clear at distinctly different rates. There is now evidence that this assumption is not entirely correct, but it may provide a reasonable first estimate of the actual division. A second and perhaps better estimate can be obtained from a theoretical deposition model that applies to both genders and all ages. The model may provide the best available central estimates of deposition in TB and AI, although the associated uncertainties are non-trivial for adult males and even larger for other subgroups of the population.

Question 5. Assuming completely insoluble particles (1 μm AMAD), retention in each respiratory tract region in mature adults as a % of the total initial deposition in the respiratory tract, as a function of time after deposition?

Particle clearance from the TB region has been studied extensively but is still not well characterized (ICRP, 1994). There is general agreement that mucociliary transport is the principal transport mechanism, that the main flow is towards the pharynx, and that mucous velocities decrease distally. There is much less agreement about the rate at which the decrease occurs or the precise pattern of mucous flow. Reported tracheal mucociliary transport rates (i.e., central values from individual studies) vary from about 4 to about 25 mm min^{-1} . There are many observations that mucociliary clearance associated with deposition in the upper nine or so airway generations is complete in a few hours. For the bronchioles, there are large discrepancies (factor of 40 or more) in reported mucous velocities. There also appears to be a slow phase of clearance from the TB region, but the fraction of the deposited amount associated with this phase as well as the rate of clearance are poorly understood (ICRP, 1994).

There appears to be some retention in the airway walls in portions of the TB region, based on studies of several species, including humans (ICRP, 1994). Retention has been best quantified for the rat trachea, where about 0.7% of the deposit remained and was cleared with an estimated half-time of 88 d. These results form the basis of the reference values describing retention in the TB airway

walls, although we are told that newer studies have yielded somewhat different results.

There have been many studies of the effects of different factors on lung mucociliary clearance. Some permanent factors that can have a significant effect on lung mucociliary clearance are age (older persons may have slower clearance), possibly gender (women could have faster clearance), and cigarette smoking (slower clearance). There is no information on factors affecting either the magnitude or duration of slow TB clearance, or of particle retention in the airways of humans.

Retention of insoluble particles in the alveolar-interstitial region has been reasonably well characterized in controlled experimental studies up to 300 d after intake, at least in healthy adult males. Estimates of subsequent retention must be based on data for accidental intakes by human subjects and/or experimental intakes by laboratory animals. The data for accidental intakes by humans fall into two categories: *in vivo* measurements, usually of radioactive materials, over the thoracic region; and postmortem measurements of the amount of material in the lungs, which in some cases have been compared with the estimated intakes.

The experimental studies involving human subjects indicate that about 80% (range of central estimates, 56-93%) of the initial alveolar deposit remains at 50 d after intake, 70% (34-83%) at 100 d, and 50% (11-64%) at 300 d (ICRP, 1994). This conclusion is based mainly on results of 10 experimental studies on human subjects. In four studies, both smokers and non-smokers were involved and were studied separately. The studies involved five different materials of differing solubilities that would have tended to increase the variability compared to uniformly soluble materials.

Measurements of accidentally exposed persons at times remote from the primary exposure(s) indicate that 10% or more of deposited material may be retained in the lungs at 3000-10,000 d after exposure (ICRP, 1994). The data are generally based on external measurements over the chest and, depending on the energy of the photon emission and knowledge of the biokinetics, may not clearly distinguish between activity in the lungs, lymph nodes, and thoracic bone. These external measurements probably should be regarded as upper-bound estimates on lung retention.

More direct evidence of very long-term retention of material in the lungs comes from postmortem measurements on occupationally exposed persons. In one study, the

distribution of actinides in former workers was estimated on the basis of concentrations in tissues taken at autopsy, roughly three decades after exposure. The measurements revealed that almost half of total-body activity was in lung tissues, indicating a long retention time of a substantial portion of the inhaled material in the lungs.

Evidence of very long-term retention of inhaled material in the lungs also comes from studies on laboratory animals. For example, studies of inhaled PuO_2 in dogs and baboons indicate that the "half-time" of particle clearance increases with time for years after intake and that several percent of the initial alveolar deposit may be retained in the lungs for many years after exposure.

The authors of ICRP Publication 66 (1994) suggest that the mean clearance rate up to 100 d may be known within about 20%, the rate at 200-300 d within a factor of 2, and the rate from Al_3 to bb_1 ($m_{3,4} = 0.0001$ corresponding to a half-time of almost 7000 d) within a factor of 3. Although there is some arbitrariness in the use of three removal half-times to represent removal from this region, these statements appear to provide a reasonable representation of the relative uncertainties in the lung content of highly insoluble particles at short, intermediate, and long times after exposure.

Rationale for confidence intervals

It was concluded that particle clearance from the tracheobronchial (TB) region is not well characterized, although there is convincing evidence that most of the deposit will clear in several hours. Long-term retention of a portion of material deposited in the TB region is suggested by some human data, but it is not clear how this component should be divided between TB and AI. Retention of a small fraction of the deposition in TB airways is indicated by data for different species, but the size and clearance of this fraction are reasonably well determined only for rats and hence do not provide much confidence. It appears to be reasonably well established that most of the long-term retention in the lungs occurs in the AI region, and there is relatively plentiful information on long-term retention of insoluble material in the human lung.

Question 6. By what factors would you expect the median values to be different in 5 year old children (1=no difference)?

Discussion

It has not been determined whether there are significant differences with age in clearance of insoluble material from the lungs. The best available study involved inhalation of Pu or Ce isotopes by immature, young adult, and aged dogs (Guilmette et al., 1987). No age related differences in retention of Pu in the lungs were observed, but Ce was initially cleared at a much faster rate from the deep lungs of immature animals than adults.

Rationale for selections

Available data are for laboratory animals and are equivocal. At this time, there is no compelling reason to believe that there are significant differences with age in lung retention.

Question 7. Absorption (dissolution and transfer) to blood in adults, % of the total initial deposition (1 μm AMAD particles) in the respiratory tract, considering the range of chemical forms which you judge most likely to be inhaled after an accident? (To include absorption to blood from the gastrointestinal tract for material cleared from the respiratory tract via the mucociliary escalator and swallowed). The usual assumption has been that elements will be inhaled in oxide form, apart from iodine in elemental form.

Discussion

Confidence bands are wide for most elements because there is a dearth of information on chemical forms and because fractional absorption to blood often depends strongly on the chemical form inhaled. Also, for most radionuclides, fractional absorption is not particularly well characterized for any given chemical form.

Rationale for confidence intervals

For derivation of a central (50%) estimate in each case, a most likely "absorption type" F, M, or S, or an average of two absorption types, was assigned to each element and the ICRP's respiratory tract model was applied. The absorption types F, M, and S correspond broadly to soluble, moderately soluble, and insoluble materials. Assignment of absorption types was based mainly on D.A. Powers' discussion concerning expected solubility of chemical forms of Sr, I, Cs, Pu, Ru, Ce, and Te that might be found in the environment following a nuclear reactor accident but also reflect information collected in ICRP Publication 30 (1979)

and ICRP Publication 71.¹ Lower (5%) and upper (95%) confidence levels were based on the degree of confidence in the selected absorption type (i.e., expectation that another absorption type may have been more appropriate), plus consideration of uncertainties in the predicted absorption level for a given absorption type.

The following assignments were made for purposes of deriving a 50% value: Sr, average of types F and M; I and Cs, Type F; Pu, Ru, and Ce, Type S; Te, Type M. The following judgments were made with regard to lower (5%) and upper (95%) confidence levels: either Type F or Type M might be more appropriate for Sr; there is little reason to expect a lower absorption level of I or Cs; type M might be more appropriate than Type S for Pu, Ru, and/or Ce; Te is an unknown quantity that could be absorbed as little as Ru or as much as Sr.

Question 8. By what factors would you expect the median values to be different in 5 year old children (1=no difference)?

Discussion and rationale for selections

It is likely that Sr moving from the respiratory tract to the gastrointestinal tract would be absorbed to a greater extent in young children than in adults. Otherwise, there is no compelling reason to modify the values for children.

Ingestion

Factors omitted that might also contribute significantly to uncertainties are doses to sensitive cells from activity in gut contents, particularly for alpha emitters, retention in intestinal tissue, and tissue mass and geometric considerations. Consider a single intake involving ingestion of 1 Bq.

Question 9. Absorption to blood as a fraction (f_1) of activity ingested, (considering chemical forms most likely to be ingested after an accident)?

Discussion

Data on gastrointestinal uptake of Sr, I, Cs, and Pu and chemically related elements by human subjects and laboratory animals have been thoroughly reviewed in ICRP Publications 56 (1989) and 67 (1993).

1. International Commission on Radiological Protection. In press. Age-dependent doses to members of the public from intake of radionuclides, Pergamon Press, Oxford. Part 4: ICRP Publication 71.

Strontium: Gastrointestinal absorption of Sr has been studied in human subjects in a variety of experimental and environmental settings. These data indicate that fractional absorption of Sr in adult humans typically is on the order of 0.15-0.3 but can be higher on occasion. Much indirect information on Sr absorption also exists in the form of animal data and studies of chemically similar elements in humans, but this indirect information does not serve to sharpen the estimates for Sr or increase the level of confidence derived from H1 data.

Iodine: It is well established that iodine ingested in aqueous media or milk is virtually completely absorbed in adult humans and laboratory animals, and similar results have been established in more limited studies involving young children and laboratory animals. Limited data on ingestion of iodine in food also indicate virtually complete absorption in humans.

Cesium: Gastrointestinal absorption of ingested Cs has been virtually complete in most studies involving adult human subjects or laboratory animals. However, values of 0.6-0.99 have been estimated for adult human volunteers consuming ^{137}Cs in contaminated meat. Thus, the possibility of high but not nearly complete GI absorption of Cs cannot be dismissed.

Plutonium: There is some human data on GI uptake of biologically incorporated Pu (in reindeer meat and winkles). These H1 data are supported by H2 data (for Am) and are consistent with data on ingestion of soluble but non-biological forms of Pu, Cm, Np, Th by humans. Estimates for GI uptake of PuO_2 must be derived from A1 data.

For many elements, particularly non-essential elements, the f_1 value for adults is applied in ICRP Publication 56 to children of age 1-15 y. This is generally done when there is evidence that the adult level of absorption is approached early in life, and when the f_1 value for adults is sufficiently cautious to account for the fact that absorption may remain slightly elevated for a prolonged period during childhood. Thus, it seems reasonable to apply the approach described above to non-essential as well as to essential elements.

Rationale for confidence intervals

It was concluded that, for adult humans, typical GI uptake factors are well established for both iodine and cesium and are near 1 in each case. It would be very surprising if average absorption of Sr were less than 0.15 or higher than 0.35. Coupled data on ^{90}Sr in food and in human bone

indicate that GI uptake of Sr is likely to be higher in young children than adults. A fairly large body of recently developed H1 and H2 data for biologically incorporated Pu or Pu in citrate solution indicates a typical absorption fraction around 0.0002. A1 data on Pu indicate that absorption of this element is at least an order of magnitude lower when ingested as PuO_2 than when ingested as citrate or nitrate; this fact is used to derive a central estimate for PuO_2 , but a wide confidence band is used to reflect the uncertainty in the transport of the data to humans. Confidence intervals for uptake of Sr and Pu in children were obtained by modifying confidence intervals derived for adults, based on evidence that: (1) fractional absorption of either essential or non-essential elements is at least as great in children as in adults and often greater; and (2) with the exception of infants, fractional absorption is rarely more than twofold greater in children than adults. Given a high-confidence interval [B,A] for fractional absorption of an element in adults, a reasonable high-confidence interval for fractional absorption in children, excluding infants, is [B,2A] or [B,1] if $2A > 1$. Depending on the element, a higher upper bound is probably appropriate for infants.

Strontium, Plutonium, Cerium, Tellurium

Questions 10-11. Considering the total amount reaching blood (as if administered intravenously as a single injection). % retained in liver and skeleton (bone + bone marrow), as a function of time after entry into blood? Retention in the skeleton, % total retention in liver + skeleton (bone + marrow), as a function of time after entry into blood?

Strontium

Discussion

Among the bone-seeking elements, skeletal behavior is probably best understood for Sr and its physiological analogue, calcium (Leggett et al., 1982; Leggett, 1992; ICRP, 1987; ICRP, 1989). For Sr, there is an abundance of experimental H1 data, and an enormous amount of data from the 1950s and 1960s on ^{90}Sr (from fallout) in human bones that can be related to an equally large pool of information on ^{90}Sr in food. The experimental data are mainly for healthy or terminally ill adult males, but some data are available for women and children. The human data, particularly the fallout data, have been used over the years to construct several biokinetic models for Sr (some of which are age-specific) with various types of conceptual frameworks. The various models are in good agreement with regard to long-term retention of Sr in bone. Less

agreement is found with regard to short-term retention in bone, and some of the models do not address retention of Sr in soft tissues.

There are sufficient H1 data on Sr that conventional curve-fitting approaches to modeling also provide reasonably high confidence because the regions of observation cover a non-trivial portion of the landscape. The use of a physiological systems framework provides some additional confidence in extrapolating to situations outside the regions of observation. Considerations of mass balance serve to increase our confidence in biokinetic estimates for Sr to some extent. For example, these considerations allow us to consider potential time-dependent feeds from bone to soft tissues, urine, or feces, thus increasing confidence in estimates for the time-dependent content of Sr in soft tissues and excreta.

Experimental H1 and A1 data indicate that the time-dependent concentration of Sr in liver is similar to that of other soft tissues. Thus, the liver is expected to contain at most a few percent of total-body Sr soon after injection and at most a few tenths or hundredths of a percent at times remote from injection.

Rationale for confidence intervals for Sr

The central (50%) estimates given here were based on a recently developed age-specific physiological systems model for Sr and the other alkaline earth elements (Leggett, 1992; ICRP, 1993). This model was based on experimental and environmental H1 data as its primary source of information, but supporting information came from the large body of H2, A1, and A2 data on Sr. For example, analysis of A1 and A2 data indicate that early uptake by bone is quantitatively similar for all alkaline earth elements. This strengthens confidence in estimates of Sr in bone at early times, where whole-body retention data are good but the division between bone and soft tissues is less well established. Also, the plentiful age-specific A1 data on Sr help strengthen confidence in judgments, usually based on not readily interpretable fallout data, concerning relative patterns of buildup and decline of Sr in bone at different stages of bone development.

With regard to the biokinetics of the absorbed amount, Sr is one of the better understood elements. Whole body retention as a function of time is particularly well characterized for adult males. This translates into a good characterization of intermediate- and long-term retention in bone, known to be the main long-term store of Sr in the body. In general, retention in bone is best understood for

adult males and for periods of several weeks to a few years after introduction of Sr to blood. However, bone retention is reasonably well characterized for other times and for other healthy subgroups of the population. One qualitative factor that helps improve estimates for children at early and intermediate times, but not late times, is the large amount of information indicating that bone-seeking radionuclides in general are taken up to a much greater extent by immature bone than by mature bone. H1 and A1 data provide convincing evidence that the ratio liver/(skeleton + liver) is very small at all times after injection of Sr.

Plutonium

Discussion

Discussions of available data on the biokinetics of Pu and methods for converting these data to predictive models for humans are given in the following review articles and reports: Durbin, 1972; Leggett, 1985; Leggett and Eckerman, 1987; ICRP, 1989; ICRP, 1993. New H1 data are available for healthy subjects injected with ^{237}Pu (Talbot et al., 1993; N.D. Priest, personal communication).

Observations of the fate of Pu in human bone are similar in nature to those for U (discussed earlier in general considerations). For various reasons, however, the Pu data provide a more narrow band of possible values of the time-dependent content in bone. For example, there was an early experimental study of the fate of injected Pu in humans (Langham et al., 1950; Durbin, 1972; Leggett, 1985). Some of the subjects were terminally ill, and Pu was measured in bone samples taken at autopsy. However, the subjects in the Pu experiment were not as ill as those of the U study and did not all suffer from a common disease. Occupational data for Pu also provide better definition of retention in bone than do the occupational data for U. One obvious reason is that the Pu data are more abundant than the U data. A less obvious reason is that considerations of mass balance provide much tighter constraints for Pu in bone than for U in bone. From excretion data, we know that most of the absorbed Pu remains in the body for decades. Excretion data on U tell us that little U remains in the body at times remote from injection but does not pinpoint total-body retention in a relative sense. For these reasons, autopsy measurements of systemic activity provide a reasonably good estimate of the amount of Pu absorbed to blood but generally provide little information on the amount of U absorbed to blood. Therefore, autopsy measurements of Pu in bone can be interpreted in terms of the absorbed amount, while measurements of U in bone generally cannot.

The long-term distribution of Pu in adult humans (i.e., several years after exposure) is better characterized than the distribution at times of a few days, weeks, or months. There are two major studies of short-term distribution of Pu in several adult subjects, including some female subjects. One of these, mentioned above, is for unhealthy human subjects. This study has obvious disadvantages but has the advantage that postmortem samples of bone and soft-tissue samples were collected. The other experimental study has the advantage that the subjects are healthy but the disadvantage that the distribution between bone and liver can be estimated only by external measurements and the liver is the only organ that can be isolated to any extent. While the investigators of the second study have gone to great effort to insure accuracy in liver measurements (e.g., calibration using a human phantom), substantial uncertainties remain in the results. For the first several months after injection of Pu, the study on unhealthy subjects indicates a central ratio skeleton/(liver + skeleton) of about 0.6, while the second study suggests a central value of about 0.3.

There is virtually no H1 data on Pu in children, but H2, A1, and A2 data provide strong support for the assumption that the ratio skeleton/(skeleton + liver) is much greater in children than adults. A1 data indicate that Pu may be lost slightly faster from immature animals than from mature animals.

Rationale for confidence intervals for Pu

It was concluded that there is strong evidence that Pu is lost very slowly from the body, with most absorbed Pu still retained after several years. There is also strong evidence that most of the body's Pu is sequestered in skeleton and liver. The long-term division between these two organs is reasonably well established. The short-term division is less well established, but there is convincing evidence that each of these organs contains a substantial portion of total-body Pu at all times after injection. These facts translate into reasonably good knowledge of the liver and skeleton content at all times.

Cerium

Information on the biokinetics of Ce comes from studies of Ce isotopes in laboratory animals (i.e., A1 data), supplemented with data on the behavior of chemically related elements in humans and laboratory animals. The animal species that have been studied include dogs, mice, rats, guinea pigs, hamsters, rabbits, cats, and miniature swine.

H1 data on Ce are limited to a study of one subject who was accidentally exposed while grinding and polishing uranium metal, and the data are difficult to interpret. These data indicate that Ce was tenaciously retained in the lungs and may have translocated slightly to the liver and skeleton. There is also some information on retention of environmental Ce, presumably from weapons tests, in the lungs and lymph nodes of a few subjects. These data indicate only that a portion of the environmental Ce was tenaciously retained in the lungs.

A1 data for Ce indicate that the systemic biokinetics of Ce fits into the same broad pattern that has been observed for lanthanides, rare earths, and actinides (excluding U), i.e., relatively slow excretion, with most of the systemic deposit divided between liver and skeleton. Two different patterns of removal from the liver are observed in different species: fast removal from livers of rodents and some larger animals, and slow removal from livers of dogs, swine, and hamsters. Comparative human and animal data on related elements (e.g., Pu, Am, Np, Cm, Th, Y) indicate that humans should fit into the same pattern as dogs, swine, and hamsters.

Rationale for confidence intervals for Ce

It was concluded that, although we do not have useful H1 data on Ce, the available H2, A1, and A2 data establish a likely qualitative pattern for the distribution and retention of Ce in humans. That is, absorbed Ce is likely to follow the same general pattern as Pu (for example). Cerium is likely to be lost from the body at a very slow rate, and most of the body's Ce is likely to be divided between liver and skeleton, with each containing a substantial portion of the total. Much H2, A1, and A2 data provide support for the standard assumption that the ratio skeleton/(skeleton + liver) is considerably greater in children than adults.

Tellurium

Human data related to the systemic behavior of absorbed Te consist mainly of autopsy measurements on environmentally exposed humans. These autopsy data indicate that bone contains almost all of the body's Te, but the data were derived several years ago and there is some question about the accuracy of the analytical technique. Moreover, interpretation of the data in absolute terms depends on uncertain information on the level of Te in the environment and, specifically, in food.

Thus, as is the case for Ce, information on the uptake and retention of Te comes mainly from studies on laboratory animals. However, confidence bands about estimates of

fractional retention in bone must be considerably larger for Te than for Ce because of incompleteness of the Te data for any of the more human-like species, and because of inconsistencies in the Te data from one species to another. Data for rats, guinea pigs, sheep, and swine (Wright and Bell, 1966; Hollins, 1969; ICRP, 1979; ICRP, 1993) yield highly variable estimates of the bone content during the first few days after exposure, with most values falling in the range 5-35% of the injected or absorbed amount. The animal data indicate that the liver may contain more Te than the skeleton in the first several days after injection, but this pattern may be reversed at later times because of relative rapid loss from the liver and slow loss from the skeleton.

There does not appear to be any particularly useful H2 data on Te in bone. Comparative studies of Te and the chemical analogue, Se, reveal that these two elements have much different biokinetics, despite some qualitative physiological similarities.

Rationale for confidence intervals for Te

A new Te model was constructed with parameter values based on A1 data (mainly for swine) but set for broad consistency with the environmental data for humans. This model was used to generate 50% values. Wide confidence bands were selected in view of the inconsistencies in the animal data on bone uptake of Te and the lack of a strong logical basis for extrapolating from animals to humans in this case.

Questions 12-14. Retention of plutonium on endosteal bone surfaces (considering a 10 μm depth of bone mineral) as a % of total skeletal retention, as a function of time after entry into blood? Retention of plutonium on trabecular surfaces, % total endosteal surface retention as a function of time after entry into blood? Retention of plutonium in red bone marrow as a % total skeletal retention, as a function of time after entry into blood?

Discussion

The qualitative behavior of Pu in the skeleton is reasonably well understood, and this behavior is reflected in the ICRP's current biokinetic model for Pu (ICRP, 1993). The rates of some of these processes, such as burial of Pu in bone volume, can be predicted on the basis of reasonably well known human bone turnover rates. Also, there is some quantitative A1 and A2 data on the time-dependent Pu content of different portions of the skeleton to help guide the selection of parameter values.

Much radiographic data for laboratory animals and humans indicate that actinides and lanthanides are deposited in similar sites on bone surfaces, although the densities of the deposits vary from site to site (Leggett, 1982, 1985; ICRP 1989, 1993). These include surfaces of trabeculae, the periosteum and endosteum, the articular cartilage, and surfaces near blood vessels in the compact bone of the diaphysis. These deposits change very slowly, apparently under the influence of bone remodeling processes. Activity is gradually removed from bone surfaces by bone restructuring processes. A substantial portion of this may be returned to blood, perhaps after temporary residence in macrophages in the bone cavities, but a portion is buried in bone volume, some within a short time after deposition. It is not known how much, if any, of the activity resorbed from bone is redeposited locally on bone surfaces without first reentering the circulation. Local redeposition of Pu could be a particularly important process in poorly vascularized areas of cortical bone. Activity in bone volume, particularly in the relatively rapidly remodeling trabecular bone, is gradually resorbed by bone turnover processes and recycled to blood, bone marrow, and/or bone surfaces. There are measurements in humans, baboons, and dogs of the fraction of total skeletal activity that resides in red bone marrow (e.g., Priest et al., 1992).

Rationale for confidence intervals (Questions 12-14)

The ICRP's biokinetic model for Pu used in ICRP Publication 67 (1993) is a physiological systems model that includes all of the processes of bone restructuring and associated burial and uncovering of Pu described above. The 50% values are mainly rounded predictions of that model, but in some cases the predictions have been shifted slightly in the direction of H1 data that have been developed since the completion of the model (about 1991).

The 5% and 95% confidence levels for retention on endosteal surfaces (Question 12), trabecular surfaces (Question 13), and red marrow (Question 14) were based in part on sensitivity analysis in which the dominant parameters were varied within seemingly reasonable bounds. These bounds were based on considerations of the quality and completeness of available H1, H2, A1, and A2 data, as described earlier. However, confidence bands suggested by these sensitivity analyses were generally expanded in consideration of the potential inadequacy of the model structure in representing actual movement of Pu in the skeleton.

Ruthenium, Cesium

Question 15. Considering the total amount reaching blood (as if administered intravenously as a single injection), % total retention (whole body or systemic) as a function of time after entry into blood?

Ruthenium

Discussion

Our knowledge of the biological behavior of Ru comes mainly from A1 data. The biokinetics of Ru has been studied in mice, rats, guinea pigs, rabbits, cats, dogs, and monkeys. The biokinetic model for Ru used in ICRP Publications 30 and 56 was based largely on a study of the comparative metabolism of Ru in mice, rats, monkeys, and dogs (Furchner et al., 1971). Long-term retention, representing roughly 20% of the injected amount, varied between 200 and 1600 d in the four species and showed no trend with body mass. The length of the observation period was different for each species, however, and data for individual species indicated that conclusions concerning cumulative activity of long-lived radioisotopes in the body may depend strongly on the length of the observation period.

The ICRP's biokinetic model for Ru is inconsistent with results of other animal studies. Data of Runkle et al. (1980) for rats indicates that more than 99% of absorbed ^{106}Ru would be excreted over a period of weeks. Retention times intermediate to those determined by Furchner et al. and Runkle et al. are indicated by data for guinea pigs that received ^{106}Ru by subcutaneous injection (Burykina, 1962).

On the other hand, some support is given to the findings of Furchner et al. by the limited available H1 data, specifically, whole-body retention data for a healthy human subject who ingested different chemical forms of ^{103}Ru or ^{106}Ru on different occasions (Yamagata et al., 1969, 1971). Studies involving ingestion of ^{103}Ru ($T_{1/2} = 39.3$ d) indicated a biological half-time of absorbed Ru of about 30 d, but it was not possible to investigate long-term retention in this experiment. A later experiment involving ingestion of ^{106}Ru ($T_{1/2} = 368.2$ d) indicated that the best estimate of a single biological half-time of absorbed Ru continued to increase with the period of observation (about 32 d during the time from 10 to 40 d after ingestion and 81, 122, 158, and 385 d at times of 40-80, 80-150, 150-350, and 350-660 d, respectively, following administration).

Rationale for confidence intervals for Ru

Available models for Ru, including a preferred model constructed for this project, give a wide range of retention values at any given time after injection. The range of values generated by the various models was used as a confidence band, except that the confidence band was expanded for times of 1 year or later to reflect the lack of a strong logical basis for extrapolating beyond observation periods.

Cesium

Discussion

With regard to total-body retention, Cs is perhaps the most thoroughly studied and best understood radioelement. Detailed studies of the biological behavior of Cs began in the 1880s, when it was discovered that Cs is a physiological analogue of potassium and competes with this essential element for both active and passive membrane transport, although Cs is generally transported less readily than K by these processes. Studies of relative selectivity among K, Cs, and other alkali metals by cell membranes have revealed much about the structure and functions of ionic channels and carriers.

In the 1950s a new importance was attached to investigation of the biological behavior of Cs when increasing quantities of fission-produced ^{137}Cs were identified in humans. Subsequently, a large literature arose concerning the distribution and residence times of radiocesium in the body and means of enhancing its excretion. The physiological behavior of Cs has also been of much interest in the past two decades because of potential medical uses of Cs as an imaging agent, a tumor marker, an antitumor agent, and an antidepressant, and several possible industrial uses of Cs have led to the investigation of the potentially toxic properties of large doses of natural Cs.

For these reasons, there is an enormous literature on the biokinetics of Cs in the human body. Unusually plentiful age- and gender-specific data on total-body retention of Cs are available. Two different measurement techniques, external photon measurements and determination of activity in excreta, have given similar results concerning retention times in the body. It appears from these data that retention of Cs can be described adequately by the sum of two exponential terms, with the longer-term component representing most of the initial deposition (roughly 90% in the adult). Therefore, in contrast to the situation for most radioelements, retention of Cs can be discussed in terms of a

half-time, meaning the half-time of the long-term component.

Rationale for confidence intervals for Cs

There have been sufficiently many high-quality studies of Cs retention in human subjects, including women and children, that one can derive reasonable bounds on those estimates by analyzing the scatter in the data.

Iodine

Question 16. Considering the total amount of iodine reaching blood (as if administered intravenously as a single injection), % retained in the thyroid as a function of time after entry into blood?

Discussion

There is a large body of age-specific H1 data on iodine uptake and retention in the thyroid. Most measurements have involved the relatively short-lived ^{131}I , and the data have been interpreted in terms of a perhaps oversimplified assumption that iodine has a specific half-time in the thyroid. Longer-lived isotopes of iodine have been used in a few studies, and results give broad support to results obtained with ^{131}I .

For ^{131}I , the critical factor for dose estimates is not how long iodine is retained in the thyroid but how much deposits there after absorption to blood. This depends strongly on whether the thyroid is "iodine replete" or "iodine deficient." The level of iodine in food and resulting thyroid iodine repletion or deficiency varies greatly from one population to another. An incomplete characterization of this variability presents the greatest uncertainty in estimating typical kinetics of iodine in the thyroid.

Rationale for confidence intervals

These intervals were based on an attempt to balance two facts: (1) iodine metabolism has been reasonably well characterized in some populations, and (2) iodine metabolism depends on the iodine intake level, which varies greatly from one population to another.

Dose coefficients

Inhalation and ingestion dose coefficients represent ACA code inputs. Uncertainties will include dosimetric modeling considerations as well as the parameters considered above.

Question 17. Intake dose coefficients, absorbed dose to specified organs or tissue per unit activity inhaled or ingested (committed equivalent doses), Gy Bq⁻¹?

Discussion and rationale

Central estimates and confidence bands for dose coefficients were generated using biokinetic models. Only models with biologically meaningful frameworks were considered. That is, it was required that parameters were in some sense measurable in the real world. When best current models did not meet these criteria (Ce, Te, Ru), new models were developed.

The ideal analysis in this situation is to check different plausible models that are consistent with current information, given the uncertainties in that information. This includes different model structures as well as reasonable ranges of parameter values. In reality, it is not practical to perform sensitivity analyses on uncertain factors using more than one or two model structures.

For the inhalation cases, the treatment of chemical form was essentially the same as for Question 7. In general, it is the inadequate information concerning chemical form that leads to the wide confidence bands assigned to most inhalation cases.

⁹⁰Sr, inhalation, 1 μ m AMAD

Uncertainties stem mainly from the solubility of inhaled Sr and the extent of absorption to blood. By comparison, the systemic behavior of absorbed Sr is well known for all age groups.

⁹⁰Sr, ingestion

Uncertainties stem from the GI uptake value and the systemic behavior of absorbed Sr. Both are reasonably well known. They are less well known for children than for adults.

¹³¹I, inhalation, 1 μ m AMAD + vapor (decide proportions)

An assumption of 50% vapor was made. The ranges of values were generated using the following values. For the adult, the median half-time in the thyroid is 80 d with a range of 40-150 d, and uptake by the thyroid is 20% with a range of 8-40%. For the child, the same central value and range were used for uptake by the thyroid, but the median half-time in the thyroid was assumed to be 25 d with a range

of 10-50 d. In each case, there is some uncertainty in the total deposition in the respiratory tract.

¹³¹I, ingestion

The uncertainties are essentially the same as for the inhalation case (minus the deposition problem), because absorption to blood should be nearly complete in each case.

¹³²Te, inhalation, 1 μ m AMAD + vapor (decide proportions)

Assume no vapor. There are substantial uncertainties in the behavior of Te in the respiratory tract, the behavior of absorbed Te, and the relative behaviors of ¹³²Te and its daughter ¹³²I. However, dosimetric errors are limited by the short half-time of ¹³²Te and the fact that penetrating radiations are emitted.

¹³⁷Cs, inhalation, 1 μ m AMAD

The dominant uncertainties are the total and regional deposition in the respiratory tract, retention time of absorbed ¹³⁷Cs, and the extent of relocation of the ingrowing daughter, ^{137m}Ba (which has been shown to relocate to a non-trivial extent, despite its short half-life). None of these will have large effects on estimates of dose, except perhaps for the lungs. Other small uncertainties include dosimetric errors and the not entirely correct assumption that absorbed Cs is uniformly distributed. Absorption from the lungs should be nearly complete.

¹³⁷Cs, ingestion

The dominant uncertainty is the retention time of absorbed ¹³⁷Cs. Lesser uncertainties include the extent of relocation of the ingrowing daughter, ^{137m}Ba, and the assumption that absorbed Cs is uniformly distributed. None of these uncertainties is large.

¹⁴⁴Ce, inhalation, 1 μ m AMAD

The greatest uncertainties stem from the solubility of Ce in the lungs and extent of absorption to blood. Additional uncertainties are associated with the systemic distribution of absorbed material, the extent of burial of Ce in bone volume, and the residence time of absorbed Ce in the body. However, the uncertainties in dose estimates are considerably less than the uncertainties in biokinetics might indicate because of the relatively short radiological half-life of ¹⁴⁴Ce and the fact that emitted radiations are penetrating

(which tends to reduce uncertainties associated with distribution in the body).

^{239}Pu , inhalation, 1 μm AMAD

The greatest uncertainties stem from the solubility of Pu in the lungs and extent of absorption to blood. Smaller uncertainties are associated with the systemic distribution of absorbed material and the extent of burial of Pu in bone volume.

^{239}Pu , ingestion

The greatest uncertainty is the GI uptake value. Other nontrivial uncertainties are the systemic distribution of absorbed material and the extent of burial in bone volume.

References

Burykina, L.N. 1962. The metabolism of radioactive ruthenium in the organism of experimental animals. In: The toxicology of radioactive substances, Vol. 1 (A.A. Letavet and E.B. Kurlyandskaya, eds.) Oxford: Pergamon Press. 60-76.

Calabrese, E.J. 1984. Suitability of animal models for predictive toxicology: theoretical and practical considerations. *Drug Metab. Rev.* 15:505-523.

Durbin, P.W. 1972. Plutonium in man: a new look at the old data, in *Radiobiology of Plutonium*, ed. B.J. Stover and W.S.S. Jee, J.W. Press, Salt Lake City, 469-530.

Furchner, J.E., Richmond, C.R., Drake, G.A. 1971. Comparative metabolism of radionuclides in mammals. VII. Retention of ^{106}Ru in the mouse, rat, monkey and dog. *Health Phys.* 21:355-365.

Guilmette, R.A., Boecker, B.B., Muggenburg, B.A., Hahn, F.F., McClellan, R.O. 1987. Age-related effects on the disposition and dosimetry of inhaled ^{239}Pu or ^{144}Ce in immature or aged beagle dogs. In: *Developments in Nuclear Medicine, Age-related factors in radionuclide metabolism and dosimetry*. Proceedings of a workshop held in Angers, France, Nov. 26-28, 1986; Dordrecht: Martinus Nijhoff Publishers. 109-120.

Hollins, J.G. 1969. The metabolism of tellurium in rats. *Health Phys.* 17:497-505.

International Commission on Radiological Protection. 1975. Report of the Task Group on Reference Man. ICRP Publication 23. Oxford: Pergamon Press.

International Commission on Radiological Protection. 1979. Limits for intakes of radionuclides by workers. ICRP Publication 30, Part 1. Pergamon Press, Oxford.

International Commission on Radiological Protection. 1987. Lung cancer risk from indoor exposures to radon daughters. ICRP Publication 50. Oxford: Pergamon Press.

International Commission on Radiological Protection. 1989. Age-dependent doses to members of the public from intake of radionuclides, Pergamon Press, Oxford. Part 1: ICRP Publication 56.

International Commission on Radiological Protection. 1993. Age-dependent doses to members of the public from intake of radionuclides, Pergamon Press, Oxford. Part 2: ICRP Publication 67.

International Commission on Radiological Protection. 1994. Human respiratory tract model for radiological protection. ICRP Publication 66. Oxford: Pergamon Press.

Klaassen, C.D., Watkins, J.B. 1984. Mechanisms of bile formation, hepatic uptake, and biliary excretion. *Pharmacol. Rev.* 36:1-67.

Krasovskii, G.N. 1976. Extrapolation of experimental data from animals to man. *Environ. Health Perspectives* 13:51-58.

Langham, W.H., Bassett, S.H., Harris, P.S., Carter, R.E. 1950. Distribution and excretion of plutonium administered to man, Los Alamos Scientific Laboratory, LA-1151.

Layton, D.W. 1993. Metabolically consistent breathing rates for use in dose assessments. *Health Phys.* 64:23-36.

Leggett, R.W. 1985. A model of the retention, translocation, and excretion of systemic plutonium, *Health Phys.* 49:1115-1137.

Leggett, R.W. 1992. A generic age-specific biokinetic model for calcium-like elements, *Radiation Protection Dosimetry* 41:183-198.

Leggett, R.W. 1994. Basis for the ICRP's age-specific biokinetic model for uranium. *Health Physics* 67:589-610.

Leggett, R.W., Eckerman, K.F. 1987. A method for estimating the systemic burden of Pu from urinalyses. *Health Phys.* 52:337-346.

Leggett, R.W., Eckerman, K.F., Williams, L.R. 1982. Strontium-90 in bone: A case study in age-dependent dosimetric modeling. *Health Phys.* 43:307-322.

Priest, N.D., Haines, J.W., Humphreys, J.A.M., Métivier, 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.* 156:33-53.

Roy, M., Becquemin, M.H., Bonnefous, S., Bouchikhi, A., Patty, E., Lafuma, A., Perrier, J.C. 1987. Clinical study of inhaled aerosols total deposition in healthy adults and children. In: *Developments in Nuclear Medicine, Age-related factors in radionuclide metabolism and dosimetry. Proceedings of a workshop held in Angers, France, Nov. 26-28, 1986*; Dordrecht: Martinus Nijhoff Publishers; 79-86.

Roy, M., Courtay, C. 1991. Daily activities and breathing parameters for use in respiratory tract dosimetry. *Radiat. Prot. Dosim.* 35:179-186.

Runkle, G.E., Snipes, M.B., McClellan, R.O., Cuddihy, R.G. 1980. Metabolism and dosimetry of inhaled $^{106}\text{RuO}_4$ in Fischer-344 rats. *Health Phys.* 39:543-553.

Talbot, R.J., Newton, D., Warner, A.J. 1993. Metabolism of injected plutonium in two healthy men. *Health Phys.* 65:41-46.

Taylor, D.M. 1984. The retention of plutonium and americium in liver: An interspecies comparison. In: *Radiation - Risk - Protection, Compacts Volume I*, ed. by A. Kaul; R. Neider; J. Pensko; F.-E. Stieve; H. Brunner. 6th International Congress organized by the Fachverband für Strahlenschutz e.v. Berlin (West) May 7-12, 1984; IRPA:431-434.

UNSCEAR. 1982. *Ionizing radiations: Sources and biological effects*. United Nations Scientific Committee on the Effects of Atomic Radiation. New York.

Wright, P.L., Bell, M.C. 1966. Comparative metabolism of selenium and tellurium in sheep and swine. *Am. J. Physiol.* 211:6-10.

Yamagata, N., Iwashima, K., Iinuma, T.A., Ishihara, T., Watari, K. 1971. Long-term retention of radioruthenium in man. *Health Phys.* 21:63.

Yamagata, N., Iwashima, K., Iinuma, T.A., Watari, K., Nagai, T. 1969. Uptake and retention experiments of radioruthenium in man - I. *Health Phys.* 16:159-166.

Question 1. Average ventilation rates, L min^{-1} , assuming a normal daily (24h) mix of activities (combined male, female average).

	5 year old children			Mature Adults		
	5%	50%	95%	5%	50%	95%
L min^{-1}	4	6	9	9	12	18

Question 2. Total initial deposition in the respiratory tract, % of total amount inhaled, normal daily (24 hr) mix of activities.

	0.1 μm AMAD			1 μm AMAD			10 μm AMAD		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Mature Adults	20	40	75	25	50	70	55	75	95
5 year old children				15	50	80			

Question 3. Initial deposition in the extrathoracic region, % of total deposition in the respiratory tract? (This question also gives the initial deposition in the lung (tracheobronchial (TB) + pulmonary (AI) regions), since lung = total - ET).

	0.1 μm AMAD			1 μm AMAD			10 μm AMAD		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Mature Adults	2	10	25	40	70	85	50	75	95
5 year old children				30	70	90			

Question 4. Initial deposition in the tracheobronchial (TB) region, % of the total deposition in the lung (TB + AI regions).

	0.1 μm AMAD			1 μm AMAD			10 μm AMAD		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Mature Adults	5	15	50	10	25	60	25	50	95
5 year old children				5	25	60			

Question 5. Assuming completely insoluble particles (1 μm AMAD), retention in each respiratory tract region in mature adults as a % of the total initial deposition in the respiratory tract, as a function of time after deposition.

	10 min			1 hr			1 day		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Tracheobronchial airways	2	7	30	1	6	25	0.5	4	20
Pulmonary (AI) region	10	23	60	10	23	60	10	23	55
	1 month			1 year			10 years		
Tracheobronchial airways	0.01	2	10	10^{-5}	0.1	2	10^{-6}	0.0001	0.1
Pulmonary (AI) region	10	20	50	5	12	30	0.5	2	15

Question 6. By what factors would you expect the median values to be different in 5 year old children (1=no difference).

	10 min	1 hr	1 day	1 month	1 year	10 years
Tracheobronchial airways	1	1	1	1	1	1
Pulmonary (AI) region	1	1	1	1	1	1

Question 7. Absorption (dissolution and transfer) to blood in adults, % of the total initial deposition (1 μm AMAD particles) in the respiratory tract.

	1 hr			1 day			1 week		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Sr	1	4	15	3	30	50	3	35	60
I	10	60	90	30	70	90	40	70	90
Cs	10	60	90	30	70	90	40	70	90
Pu	0.01	0.04	5	0.01	0.06	5	0.01	0.06	5
Ru	0.02	0.08	5	0.1	0.4	10	0.1	0.5	10
Ce	0.01	0.04	5	0.01	0.06	5	0.01	0.06	5
Te	0.02	5	15	0.1	13	50	0.1	14	60
	1 month			1 year			10 years		
Sr	10	35	70	10	35	70	10	35	70
I	40	70	90	40	70	90	40	70	90
Cs	40	70	90	40	70	90	40	70	90
Pu	0.02	0.1	10	0.1	0.6	15	0.3	2	15
Ru	0.1	0.6	15	0.3	1.5	25	0.5	3	25
Ce	0.02	0.1	10	0.1	0.6	15	0.3	2	15
Te	0.1	17	70	0.3	27	70	0.5	30	70

Question 8. Factors by which the median values would be different in 5 year old children (1=no difference).

5 year old children	1 hr	1 day	1 week	1 month	1 year	10 years
Sr	1.1	1.2	1.2	1.2	1.2	1.2
I	1	1	1	1	1	1
Cs	1	1	1	1	1	1
Pu	1	1	1	1	1	1
Ru	1	1	1	1	1	1
Ce	1	1	1	1	1	1
Te	1	1	1	1	1	1

Question 9. Absorption to blood as a fraction (f_1) of activity ingested.

	3 month old infants			5 year old children			Adults		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Sr	.25	.60	.90	.15	.30	.70	.15	.25	.35
I	.90	.99	1.0	.90	.99	1.0	.90	.99	1.0
Cs	.80	.95	1.0	.80	.95	1.0	.80	.95	1.0
PuO_2^*	1×10^{-6}	2×10^{-4}	1×10^{-3}	1×10^{-6}	4×10^{-5}	1×10^{-3}	1×10^{-6}	2×10^{-5}	5×10^{-4}
Pu biol†	5×10^{-5}	2×10^{-3}	1×10^{-2}	4×10^{-5}	4×10^{-4}	4×10^{-3}	5×10^{-5}	2×10^{-4}	2×10^{-3}

* Refractory oxide
† "Biologically incorporated"

Question 10. Considering the total amount reaching blood (as if administered intravenously as a single injection). % retained in liver and skeleton (bone + bone marrow), as a function of time after entry into blood.

	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
Skeleton and liver, 1 day						
Sr	30	45	95	20	33	65
Pu	20	35	95	20	35	80
Ce	20	70	95	20	70	95
Te	1	10	80	1	10	80
Skeleton and liver, 1 week						
Sr	24	32	80	14	22	40
Pu	60	75	95	60	75	90
Ce	40	80	95	40	80	95
Te	1	15	80	1	15	80
Skeleton and liver, 1 month						
Sr	20	29	60	13	20	35
Pu	60	80	95	70	80	90
Ce	50	82	95	60	81	95
Te	1	10	80	1	10	80
Sb						
Skeleton and liver, 1 year						
Sr	10	17	40	9	13	20
Pu	40	77	90	65	80	90
Ce	30	80	90	50	80	90
Skeleton and liver, 10 years						
Sr	0.5	2	6	4	7	12
Pu	20	59	59	40	73	85
Ce	10	51	51	20	61	85
Skeleton and liver, 50 years						
Sr	0.1	0.4	2	0.5	1.5	4
Pu	10	37	70	20	49	70
Ce	1	20	70	2	28	70

Question 11. Retention in the skeleton, % total retention in liver + skeleton (bone + marrow), as a function of time after entry into blood.

	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
Skeleton, 1 day						
Sr	90	97	99	90	94	99
Pu	50	75	95	25	50	65
Ce	30	63	95	20	38	80
Te	5	30	80	5	30	80
Skeleton, 1 week						
Sr	90	97	99	90	95	99
Pu	50	75	95	25	50	65
Ce	10	63	95	20	38	70
Te	10	50	90	10	50	90
Skeleton, 1 month						
Sr	95	99	99.9	95	99	99.9
Pu	50	75	95	25	50	65
Ce	10	62	95	20	38	70
Te	30	90	99	30	90	99
Skeleton, 1 year						
Sr	99	99.9	99.98	99	99.9	99.98
Pu	50	77	95	25	50	65
Ce	10	58	95	20	40	70
Skeleton, 10 years						
Sr	99.5	99.9	99.98	99.5	99.9	99.98
Pu	20	44	80	35	60	75
Ce	10	44	95	20	51	90
Skeleton, 50 years						
Sr	99.5	99.9	99.98	99.5	99.9	99.98
Pu	35	64	85	40	70	85
Ce	10	71	95	10	74	95

Question 12. Retention of plutonium on endosteal bone surfaces (considering a 10 μm depth of bone mineral) as a % of total skeletal retention, as a function of time after entry into blood.

		5 year old children			Adults		
		5%	50%	95%	5%	50%	95%
Endosteal bone surface	1 day	90	99	99.9	95	99	99.9
	1 week	90	98	99	95	99	99.8
	1 month	70	92	95	95	98.5	99
	1 year	25	52	75	70	91	95
	10 years	5	43	70	20	78	90
	50 years	5	65	80	20	67	80

Question 13. Retention of plutonium on trabecular surfaces, % total endosteal surface retention as a function of time after entry into blood.

		5 year old children			Adults		
		5%	50%	95%	5%	50%	95%
Trabecular bone surface	1 day	20	50	70	40	60	80
	1 week	20	50	70	40	60	80
	1 month	20	50	70	40	60	80
	1 year	5	46	70	30	55	75
	10 years	2	37	60	15	28	50
	50 years	2	20	40	10	17	40

Question 14. Retention of plutonium in red bone marrow as a % total skeletal retention, as a function of time after entry into blood.

		5 year old children			Adults		
		5%	50%	95%	5%	50%	95%
Red bone marrow	1 day	0.05	0.2	5	0.05	0.2	5
	1 week	0.05	0.5	5	0.05	0.2	5
	1 month	0.2	2	10	0.1	0.7	5
	1 year	1	9	20	0.3	3	10
	10 years	0.7	5	15	0.2	2	10
	50 years	0.3	1	5	0.1	1	5

Question 15. Considering the total amount reaching blood (as if administered intravenously as a single injection), % total retention (whole body or systemic) as a function of time after entry into blood.

	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
1 day						
Ru	25	85	95	25	85	95
Cs	90	97	99	90	98	99
1 week						
Ru	15	66	85	15	66	85
Cs	75	87	95	80	87	95
1 month						
Ru	1	39	70	1	39	70
Cs	20	32	60	50	75	85
1 year						
Ru	0.5	16	50	0.5	16	50
Cs	0.001	0.02	2	2	10	15
5 years						
Ru	0.01	6	20	0.01	6	20
Cs	10^{-5}	0.001	2	10^{-5}	0.001	1

Question 16. Considering the total amount of Iodine reaching blood (as if administered intravenously as a single injection), % retained in the thyroid as a function of time after entry into blood.

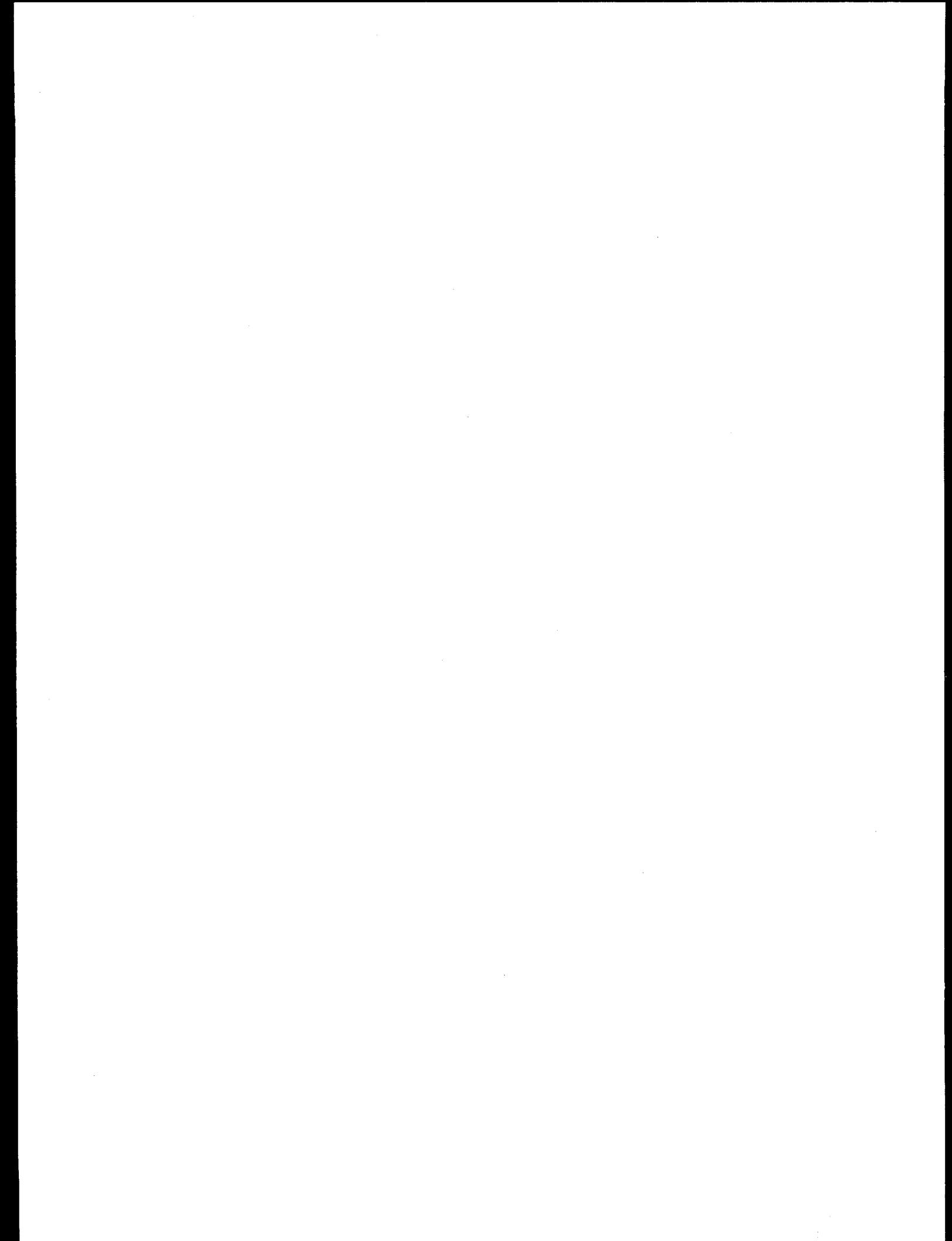
	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
1 day	15	27	60	15	28	40
1 week	10	22	50	15	28	40
1 month	3	8	40	10	24	35
3 months	0.1	0.6	30	5	16	30

Question 17. Intake dose coefficients, absorbed dose to specified organs or tissue per unit activity inhaled or ingested (committed equivalent doses), Gy Bq⁻¹.

	5 year old children			Adult		
	5%	50%	95%	5%	50%	95%
⁹⁰Sr, inhalation, 1 μm AMAD						
lung	2×10^{-9}	3×10^{-8}	3×10^{-7}	1×10^{-10}	1×10^{-9}	1×10^{-8}
bone marrow	1×10^{-8}	1×10^{-7}	1×10^{-6}	1×10^{-8}	1×10^{-7}	5×10^{-7}
bone surface	3×10^{-8}	3×10^{-7}	3×10^{-6}	5×10^{-8}	3×10^{-7}	1×10^{-6}
⁹⁰Sr, ingestion						
colon	5×10^{-9}	5×10^{-8}	5×10^{-7}	2×10^{-9}	1×10^{-8}	1×10^{-7}
bone marrow	5×10^{-8}	3×10^{-7}	2×10^{-6}	5×10^{-8}	1.5×10^{-7}	5×10^{-7}
bone surface	1×10^{-7}	5×10^{-7}	3×10^{-6}	1×10^{-7}	3×10^{-7}	1×10^{-6}
¹³¹I, inhalation, 1 μm AMAD + vapor (decide proportions)						
thyroid	2.5×10^{-7}	1.1×10^{-6}	3.5×10^{-6}	6×10^{-8}	2.3×10^{-7}	6×10^{-7}
¹³¹I, ingestion						
thyroid	4×10^{-7}	1.4×10^{-6}	3×10^{-6}	1×10^{-7}	2.9×10^{-7}	6×10^{-7}
¹³²Te, inhalation, 1 μm AMAD + vapor (decide proportions)						
lung	1×10^{-9}	2×10^{-8}	4×10^{-8}	5×10^{-10}	1×10^{-8}	2×10^{-8}
thyroid	2×10^{-8}	6×10^{-8}	2×10^{-7}	4×10^{-8}	9×10^{-8}	2×10^{-7}
colon	1×10^{-8}	2×10^{-8}	4×10^{-8}	2×10^{-9}	4×10^{-9}	8×10^{-9}
¹³⁷Cs, inhalation, 1 μm AMAD						
lung	1×10^{-9}	3×10^{-9}	9×10^{-9}	1.5×10^{-9}	4.3×10^{-9}	1.2×10^{-8}
colon	2×10^{-9}	7×10^{-9}	2×10^{-8}	2×10^{-9}	5.6×10^{-9}	1.5×10^{-8}
stomach	1×10^{-9}	3×10^{-9}	9×10^{-9}	1.5×10^{-9}	4.5×10^{-9}	1.2×10^{-8}
bone marrow	1×10^{-9}	3×10^{-9}	9×10^{-9}	1.5×10^{-9}	4.5×10^{-9}	1.2×10^{-8}
¹³⁷Cs, ingestion						
lung	3×10^{-9}	8×10^{-8}	2×10^{-9}	0.7×10^{-8}	1.3×10^{-8}	2.5×10^{-8}
colon	8×10^{-9}	2×10^{-8}	5×10^{-8}	0.9×10^{-8}	1.7×10^{-8}	3.3×10^{-8}
stomach	3×10^{-9}	9×10^{-8}	2×10^{-9}	0.7×10^{-8}	1.3×10^{-8}	2.5×10^{-8}
bone marrow	3×10^{-9}	8×10^{-8}	2×10^{-9}	0.7×10^{-8}	1.3×10^{-8}	2.5×10^{-8}
¹⁴⁴Ce, inhalation, 1 μm AMAD						
bone surface	1×10^{-7}	4×10^{-7}	1×10^{-6}	5×10^{-8}	2×10^{-7}	5×10^{-7}
lung	4×10^{-10}	1.5×10^{-9}	6×10^{-9}	1×10^{-9}	3.5×10^{-9}	1×10^{-8}
bone marrow	3×10^{-10}	1.3×10^{-9}	5×10^{-9}	8×10^{-10}	2×10^{-9}	6×10^{-9}
liver	5×10^{-10}	2×10^{-9}	8×10^{-9}	3×10^{-10}	1×10^{-9}	3×10^{-9}
²³⁹Pu, inhalation, 1 μm AMAD						
bone surface	1×10^{-6}	2×10^{-5}	4×10^{-4}	3×10^{-6}	3×10^{-5}	3×10^{-4}
bone marrow	1×10^{-7}	2×10^{-6}	4×10^{-5}	1×10^{-7}	1×10^{-6}	1×10^{-5}
liver	7×10^{-7}	7×10^{-6}	1×10^{-4}	1×10^{-6}	5×10^{-6}	3×10^{-5}
lung	2×10^{-7}	5×10^{-6}	2×10^{-5}	3×10^{-7}	3×10^{-6}	3×10^{-5}
²³⁹Pu, ingestion						
bone surface	1×10^{-8}	3×10^{-7}	6×10^{-6}	2×10^{-8}	2×10^{-7}	3×10^{-6}
bone marrow	7×10^{-10}	2×10^{-8}	5×10^{-7}	1×10^{-9}	8×10^{-9}	2×10^{-7}
liver	3×10^{-9}	1×10^{-7}	1.5×10^{-6}	5×10^{-9}	4×10^{-8}	5×10^{-7}
colon	1×10^{-9}	8×10^{-9}	2×10^{-7}	3×10^{-10}	2×10^{-9}	3×10^{-8}

(xi) Joint dosimetry/late effects question: The number of radiation-induced cancer deaths up to 40 years following exposure in a population of a hundred million persons (5×10^7 male, 5×10^7 female) each of whom inhales 10 K Bq of the radionuclides specified.

Nuclide	Physical Form	Chemical Form	Cancer Type	Number of Cancers		
				Quantile	5%	50%
Pu-239	1 μ m AMAD	Oxide	Lung			
			Bone			
			Liver			
			Leukemia			
			All cancers			
Sr-90	1 μ m AMAD	Oxide	Lung			
			Bone			
			Leukemia			
			All cancers			



EXPERT C

The International Commission on Radiological Protection (ICRP) has used in Publication 30 (ICRP, 1979) dosimetric and biokinetic data to establish secondary limits (Annual Limits on Intake, [ALI]) to control intakes of radionuclides by workers. After the Chernobyl reactor accident, the necessity for new dose coefficients for members of the public initiated significant revision of dosimetric models and selected biokinetic parameters.

During the same period two meetings were held in France, in Angers, November 26-28, 1986 (Gerber et al., 1987) and Versailles, May 30 - June 2, 1988 (Gerber et al., 1989). The meetings, sponsored by the French Institute for Protection and Nuclear Safety (IPSN) and the British National Radiological Protection Board (NRPB), were supported by the European Communities (Radioprotection Program) and USDOE. The meetings established the concepts of dose calculations with age-dependent parameters for public and site-specific parameters for workers (specific Dose per Unit Intake [DPUI]). This was the beginning of the ICRP "age-dependent" series (publications 56, 67, 69, and 71) resulting in recommended ALIs (ICRP, 1990; ICRP, 1994b; ICRP, 1995; ICRP, 1996). In the Bath meeting (September 13-17, 1993), the reliability of biokinetic and dosimetric models and parameters used to assess individual doses for risk assessment purposes was discussed for the first time in an international forum by the leader of an NCRP expert group (Bouville et al., 1994).

In the 1990's NCRP and ICRP developed Task Groups for this purpose. However, the progress of evaluation is very slow due to the difficulty of the approaches, the lack of expert panels for probabilistic assessment, and sometimes either the paucity or excess of information.

This probabilistic accident consequence uncertainty analysis is an important step in this new direction and should be encouraged. However, with this exercise arise the difficulties of making such estimations.

One of the first difficulties is the time required to prepare and present a lot of data with complete rationale and references. The second difficulty is the "solitaire game" aspect, which has advantages and disadvantages. Advantages have been presented in the first meeting (held in Annapolis, MD) as school cases. A disadvantage could be in the selection of experts. If they are in the same field of interest with the same background, collective error is highly possible. For example, how to determine correctly the

source term without any discussion with safety experts, chemists, and physicists? The collective expertise takes advantage of the diversity of experts' backgrounds. Increased radioprotection quality is an obvious benefit of the interaction of different professions.

One of the main uncertainties of this exercise is in the definition of solubility of released material in case of a severe PWR accident. In the first place, the evaluation of source term is difficult because it depends on how the accident occurred. In the second place, the knowledge of chemical forms needs further certification from experiments such as the international project called Phebus-FP (Tattegrain,¹ Livolant et al., 1995) or in the HEVA/VERCORS tests (Lewis et al., 1995). Indeed, it is difficult to learn something from a former accident like Three Mile Island, or from Chernobyl which was not representative of a PWR accident.

The word solubility does not mean the same thing to all experts, particularly chemists and biologists. The chemist speaks of rapid solubility, the biologist does not have the same scale of time, especially when discussing "committed dose." The example of plutonium dioxide illustrates the difficulties of estimation of solubility which depends on real stoichiometry rather than temperature formation. In their 1980s paper W.J. Bair and H. Métivier describe similar biological behavior of oxides prepared at 560° and 1100°C (Bair et al., 1980). However, in another experiment, J. Mewhinney clearly demonstrated a difference of clearance between oxides prepared at these temperatures (Mewhinney et al., 1976). The two first oxides were prepared by chemists as pure dioxide, while the other oxides were representative of fire with incomplete burning. In spite of a quite good agreement with the Powers approach (1996), I think that a source of uncertainties remains in the dioxide formation process. Moreover, it is true that the hydrate forms of plutonium appear as colloidal particles suspended in water, but it has been demonstrated that these reactions could be kinetically limited when the concentration is low, as happens in biological conditions (Métivier, 1984a).

It has been shown that the chemical behavior of plutonium at low-level concentrations is different from the chemical behavior described for classical chemistry in laboratories and factories. The behavior of a monomeric species of an element in aqueous solution depends to a large extent on the conditions which favor hydrolysis. This first step of hydrolysis for plutonium at low pH is followed by

1. Tattegrain, A. 1994. *The PHEBUS Severe Accident Experiment*. Nuclear Engineering. To be published.

hydrolytic reactions which are generally complex because of the role of radiocolloids at low concentration of plutonium and that of polymers at higher concentration.

Figure 1 shows, using the ^{238}Pu isotope at very low concentration, that the dismutation reaction and colloid formation could be biokinetically slowed, if the plutonium is in a biological fluid rich in citrate ions (10^{-6} M in blood) in competition with complexation by cationic complexes. These conditions occur in biological conditions in case of an accident with contamination of the public. (Métivier et al., 1972a, 1972b). This hypothesis has been confirmed in an experiment comparing the behavior of plutonium after inhalation and intratracheal instillation for 239 and 238 isotopes (Stradling et al., 1984).

More recently, extreme confusion surrounded the interpretation of solubilities of cesium from Chernobyl fallout in different countries. The cesium dispersed from the "first" accident at Chernobyl (explosion) was cesium probably included in the fuel fragments and could be more insoluble than cesium dispersed during the "second" accident (fire), ten days later (Borzilov et al., 1993). Moreover, the part of the reactor dispersed in these two phases was not the same and can show isotopic differences.

The complication of a "two-phase" accident, plus dispersion in aerosols produced by different ways (explosion and fire) under different climatic conditions had clearly led to some misinterpretation and brought up some doubts about premature conclusions given after the accident (NEA, 1996). This explains my scepticism about conclusions drawn from the Chernobyl accident regarding information about chemical form, as noted by Powers (1996).

The expert elicitation exercise arrives at a good time because ICRP and NCRP have revised the former lung model. The rationales of answers for inhalation in this report are based mainly on ICRP Publication 66 (ICRP, 1994a) which the author fully endorses. Moreover, it is a future task of the ICRP Dosimetry Task Group to evaluate reliability of parameters. We encourage the group to work hard and complete the exercise as soon as possible.

The revision of systemic models appears in the "age-dependent" series of ICRP Publications 56, 67, and 69 (ICRP, 1990; ICRP, 1994b; ICRP, 1995). One of them, the actinide bone model, drew some criticism. It will be interesting to use this elicitation session to strengthen the argument for a new consensus model.

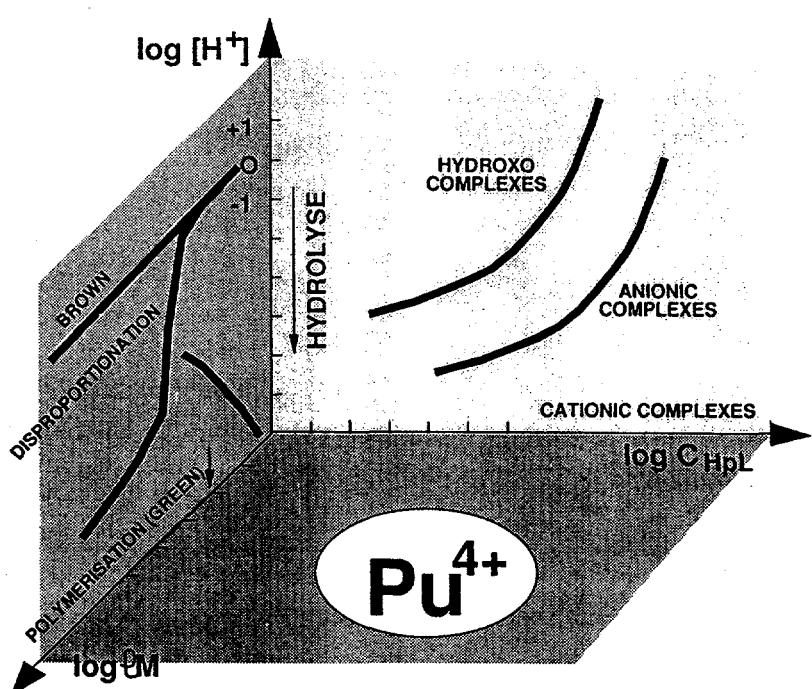


Figure 1. Biokinetic showing dismutation reaction and colloid formation in low-concentration ^{238}Pu isotope.

Lastly, the GI tract model will be revised in the next years. However, GI transfer to blood is currently well documented, and the Task Group has recently given a draft report for reliability of f_I data. This draft report is used in this exercise.

It is hoped that this elicitation exercise will be an impelling force to accelerate uncertainties analyses and will be strongly helpful for research decision-makers.

After the general introduction which sets the basis of analysis, this report is presented question by question. The references used in this document are listed at the end of the report.

Elicitors

For each question, a median estimate is required for an average individual, representative of the group under consideration. The uncertainty associated with the median estimate should be given as 5th and 95th percentiles.

Inhalation

These questions cover important contributions to uncertainty in calculating doses from inhaled radionuclides, considering the behavior of materials in the respiratory tract. Factors omitted that might also contribute significantly to uncertainties are the location of sensitive cells in different regions, the relative radiosensitivity of the different regions, and tissue mass and geometric consideration. Intake can be considered to be due to exposure to air concentrations of radionuclides of say, 1 Bq m⁻³, for short duration of say, one minute.

Question 1. Rationale

Children:

ICRP 71 (1996) gives in Tables 4 and 5 ventilation parameters and daily time budget for reference subjects for all age groups. From the data given in the tables we can obtain for 5-year-old children, on the basis of 12 h of sleep, 9 h sitting, and 3 h light exercise, a ventilation rate of 5.2 L min⁻¹. A slightly different basis of time budget (Table B16A, ICRP, 1994a), leads to a value of 6.1 L min⁻¹. These differences are obtained only by repartition of time budget without any physiological consideration. Physiological considerations are more difficult to evaluate but seem to balance any variation and not to enhance the range of uncertainties (Roy et al., 1991a, 1991b). The central value

of 6.0 L min⁻¹ with a variation of 10% to 15% is adopted here.

Adults:

The same exercise with Tables B16B and B17 of ICRP 66 (ICRP, 1994a) and Tables 4 and 5 of ICRP 71 (ICRP, 1996), leads to values from 14.1 to 14.8 L min⁻¹. However, the time budget and ventilation rate variations are larger in adults because we have several categories which greatly differ: male and female, sedentary and heavy workers, old people, etc. The central value of ICRP 66 is adopted here with a variation ranging from 25% to 30%.

Lung diseases considerations are not taken into consideration here, because it seems to me that they are already included in ICRP Publication 71.

Question 2. Rationale

Total initial deposition is strongly dependent on ventilation rates. In ICRP 66 (ICRP, 1994a) data for deposition are given in Tables F1, 2, 3, 4 and 5. A reference subject for members of the public has been calculated by the ICRP 71 Task Group (1996). The combination of different parameters given in tables leads to central values adopted here. James et al. in Annex D (James et al., 1994) of ICRP 66 reported many experiments performed in humans. It seems from many data shown in this report that dispersion increases with aerosol size and that a 15% for 0.1 μ m particles to 35% for 10 μ m particles could be adopted.

Becquemin et al. (1991) and Schiller-Scotland et al. (1992) have measured the total respiratory tract deposition in children. These references are the basis of this estimation with a dispersion of 20% around the central value.

Hygroscopic growth of particles is not taken into consideration in this rationale, because aspersion is a common technique for reducing the source term inside the reactor building. We can assume that all the reactions with water occurred before release of particles; however, this point is questionable.

Question 3 and Question 4. Rationale

The bases of the answers for Questions 3 and 4 are also given by ICRP 66, Tables F1, F2, F3, F4 and F5 (ICRP, 1994a), which give fractional deposition in regions of the respiratory tract in different situations. The variations representing male and female, and light exercise, sitting, and sleep are performed on a time budget hypothesis of 6 h,

8 h, and 10 h respectively for adults and 3 h, 9 h and 12 h for 5-year-old children. The results are the central values. The dispersion is based here on the extreme modifications of time budget and dispersion described in reported experiments (James et al., 1994), without addition of the two uncertainties.

One of the most surprising results in implementing the new lung model is the fraction of 10 μm particles which reaches the alveolar-interstitial region.

Question 5. Rationale

The central values are calculated with the LUDEP logiciel (Birchall et al., 1991) applying the ICRP respiratory tract model with only mechanical clearance since the hypothesis is completely insoluble particles. The initial deposit is that in ICRP 71 (6.7% in tracheobronchial airways and 23.6% for pulmonary region) for a reference member of the public.

$$\text{TB} = (\text{BB}+\text{bb})100/48.58 = 6.67\% \text{ of total initial lung burden}$$

$$\text{AI} = 11.48 \times 100/48.58 = 23.63\% \text{ of total initial lung burden}$$

48.58% being the total initial lung burden given by ICRP 71 instead of 51.2% for workers (Birchall et al., 1991).

The three half-times used in this calculation are 30 d, 700 d, and 7000 d for the three fractions assumed in the model for 30, 60, and 10% respectively. The 10-min values are higher than the initial deposit because of clearance from farther regions.

Total uncertainty is the sum of uncertainty of deposit estimated for Questions 3 and 4 for James et al. (1994) and uncertainty on the clearance of the fraction cleared with the two long half-times, 700 and 7000 days (Bair et al., 1974; Métivier et al., 1977; Kreyling et al., 1991; Bailey et al., 1994). Indeed we can estimate that the fractions and half-times chosen by ICRP are gross-estimations, well representative of the central value but with large individual variations. Moreover, some modifying factors can change half-time clearance for insoluble particles, for example smoking habits (Filipy et al., 1980a) or lung diseases (Roy et al., 1984). The percentage of uncertainty estimated to be about 20-25% at 1 month reaches 40% and 50% at 1 year and 10 years after inhalation respectively.

Question 6. Rationale

There may be differences between children and adults concerning mechanical clearance of insoluble particles. However, there are almost no experimental data on clearance in normal children and insufficient data in animals to establish clear quantitative differences (Bailey et al., 1994). The two experiments quoted in ICRP 66 gave results for mucociliary transport and transit time within the range measured in adults (Kärjä et al., 1982; Passali et al., 1985). Moreover, the reported experimental results are not the most important for dose calculations.

Finally, ICRP, in its Publication 66 (ICRP, 1994a) accepted in absence of further information, that clearance of children and adults is the same. It is certainly a source of uncertainty, and it is difficult to verify at this time whether the factor given as an answer for Question 6 is lower or higher than a factor of 1.

Question 7. Rationale

The first difficulty with the question was to decide if we treated the element or the radionuclide, i.e., with radioactive decay. In the second case (radionuclide), the second part of the table (1 month, 1 year, and 10 years) was not relevant. As the word "element" was used and no figures were provided for any radionuclide, we have retained the cumulative dissolution of the stable element.

The second point is the choice of solubility type for each element which will lead us to select some f_I factors.

Strontium:

Powers (1996) recommends use of a water-insoluble form. Once more this classification is too imprecise regarding the chemistry of strontium and its behavior at very low concentration. If strontium is incorporated in a matrix, its behavior will be that of the matrix (Snipes et al., 1972) and in some cases will be a very insoluble compound (Type S, $f_I = 0.01$). Johnson et al. (1983) described an accidental release of fragments of irradiated fuel and gave results indicating a Type M behavior of the strontium present. Lastly, Kanapilly et al. (1980) reported an experiment on airborne fission products from the Three Mile Island reactor accident consistent with Type F ($f_I = 0.3$ for adults and 0.4 for 5 year-old children).

On these bases, we take as central value the behavior of a Type M compound with an f_I of 0.1, and for the 5th and

95th percentiles, the same type M but with $f_I = 0.05$ and 0.2 respectively. It is clear that the range given in this exercise is probably largely overestimated and that the reality for 10 years could be 10% to 40%.

Iodine:

The hypothesis used here is an elemental iodine or particulate iodine, Type F, with a value of 1 for the f_I coefficient. As the dose coefficients for such different types as F to S do not differ by more than a factor of 5, the uncertainty given here is about 15% with exception for the first hour where we think the uncertainty is highest.

Cesium:

The hypothesis used for this exercise is that 80% of cesium is released as soluble particles and 20% as insoluble form. The 95th percentile is obtained for a release as 100% of soluble form (Powers, 1996). The 5th percentile is obtained for a 50-50% combination of soluble and insoluble forms.

Plutonium:

The hypothesis used in this exercise is that plutonium dioxide is released as an insoluble form, Type S. In this Type S we have calculated the central value with an $f_I = 10^{-4}$. We have also calculated values for f_I values ranging from 1×10^{-6} to 1×10^{-4} to take into account different gut absorption rate linked to relative solubility of "insoluble" forms (Bair et al. 1980; ICRP, 1986); results do not differ significantly. The 95th percentile is obtained with 50% of the plutonium transformed during the two days before release.

The most significant choice for uncertainty evaluation is not in the value of f_I for the insoluble fraction but between the Type F or M. The 95th percentile is obtained on the basis of M classification (Mewhinney et al., 1976). However, all the reported data which tend to assess plutonium oxide as Type M are the result of observation with $^{238}\text{PuO}_2$ oxide. It is clearly demonstrated that ^{238}Pu oxide is more soluble than ^{239}Pu . This is due to the differences of specific radioactivities of the two isotopes which leads $^{238}\text{PuO}_2$ particles to a faster fragmentation of initial particles and consequently to a higher solubility (Bair et al., 1980; Park et al., 1991; Guilmette et al., 1994).

Ruthenium:

The central value is estimated for a Type S ruthenium, with a f_I of 0.01. It is consistent with Powers (1996)

recommendations but different from ICRP 71 (1996) recommendations which adopted a Type M as a default value, with a f_I of 0.05 for public. The 5th percentile is based on more insoluble compounds (Type S with $f_I = 0.001$) but this change is not significant in terms of dosimetry. The 95th percentile is based on *in vitro* dissolution of particles released from the Chernobyl accident (Cuddihy et al., 1989).

Anyway, ruthenium remains an enigmatic element that is difficult to study by representative experimentation. For example, an experiment built to simulate source-term for contamination of lysimeters did not release any significant amounts of ruthenium for speciation studies. (Madoz-Escande and Jouler)*. This could be due to the particularly high melting point and boiling point temperatures of this element, 2310°C and 3900°C respectively. If ruthenium is released alone and not included in fragments of fuel, this means that the temperature of the accident is so high that there is production of elemental form inside the building, then condensation on nuclei, and that in this case particles are composed of nanometric aggregates and become particularly soluble. This phenomenon has been observed in another experimental program HEVA (Levèque et al., 1991).

Cerium:

The model used is the same as for plutonium. However, central value is based on a Type M compound (ICRP, 1996) but with an f_I value of 1×10^{-4} rather than 5×10^{-4} which is judged much too soluble for such a situation. The 5th percentile is based on Type S becoming more soluble with time. For 95%, a slightly more soluble compound (Type M, $f_I = 1 \times 10^{-3}$) is adopted for the calculation (Cuddihy et al., 1989; Stradling et al., 1989).

Tellurium:

The hypothesis used here is a Type F compound with an $f_I = 0.01$ (ICRP, 1996). The 5th and 95th percentiles are based on a change of $f_I = 0.1$ for Type F and a Type M with an $f_I = 0.1$. It is not really significant. As with ruthenium, tellurium is an enigmatic element in terms of speciation of particles released; we need more information on chemical form of particles. However, the choice adopted here strongly differs from Powers (1996) recommendations.

* Madoz-Escande, C., Jouler, H. 1996. "Caractérisation des contaminations des expérimentations effectuées par le four POLYR." *Radio-protection*. To be published.

Question 8. Rationale

As with the answer to Question 6, we consider here that there are no differences between 5-year-old children and adults in terms of clearance (ICRP, 1994a). Moreover, for the selected elements and hypothesis (Powers, 1996), there are no differences between the f_I values for 5-year-old children and adults (ICRP, 1996); we consider here that the median value would not change between 5-year-old children and adults.

This assumption should be slightly wrong for soluble strontium, since in this case the f_I coefficient differs for 5-year-old children (0.4) and adults (0.3).

Once again, the main uncertainty for this question is the knowledge of chemical form of dispersed material.

Ingestion

Factors omitted that might also contribute significantly to uncertainties are doses to sensitive cells from activity in gut contents, particularly for alpha emitters, retention in intestinal tissue, and tissue mass and geometric considerations. Consider a single intake involving ingestion of 1 Bq.

Question 9. Rationale

The values given in the table are the results of the Task Group of ICRP in charge of uncertainties estimation of f_I values. As chairman of this sub-group, I have no other solution than to endorse the work of this group. The results presented here are the results of a panel discussion, and some experts might give different data. However, I think that currently it is probably the best estimate.

Strontium:

Gastrointestinal absorption has been mainly studied in humans; animal data are also available. Data from chemically similar elements (alkaline earth) are well documented.

Fujita (1965) measured absorption in four volunteers given low concentration of ^{90}Sr in milk and obtained a range from 0.28 to 0.56 (mean value 0.38). Taking into account the Ca dietary intake, he estimated the uncertainties linked to this, suggesting a larger range of 0.09 to 0.63. In a previous experiment where carrier-free ^{85}Sr was given to volunteers as chloride (Fujita et al., 1963), the f_I was estimated to be 0.12 or 0.14 depending on time after ingestion. Using

urinary excretion analysis, Suguri et al. (1963) determined the value of f_I to be 0.13 in one normal subject after ingestion of $^{85}\text{SrCl}_2$. Reviewing existing data on ^{90}Sr and after developing a mathematical model, Snyder et al. (1964) derived a range of f_I values from 0.17 to 0.5 in five individuals. In a similar experiment where $^{85}\text{SrCl}_2$ was given orally to twelve volunteers, Le Roy et al. (1966) obtained a mean value for f_I of 0.17 with a range of 0.08 to 0.34. In a more extensive study, in which nine subjects received $^{85}\text{SrCl}_2$ orally, and eight by intravenous injection, comparison of the results gave a mean f_I value of 0.17 ranging from 0.06 to 0.31 (Likhtarev et al., 1975). These values were quite similar to those obtained for 45 adult rats by Taylor et al. (1962) who observed a mean f_I value of 0.25 with a standard deviation of 0.01.

Results obtained by Widdowson et al. (1960) suggest that absorption of strontium in 7 day old infants fed with cow's milk is greater than 73%. Bedford et al. (1960) reported that absorption in 5- to 15-year-old children was the same as in adults.

Taylor et al. (1962), obtained absorption values of 0.95 ± 0.004 (SE, n=31) for 14 to 18 day old rats and 0.74 ± 0.024 (SE, n=5) for 22 day old animals.

Central values are those of ICRP 56 and 67, (ICRP, 1990, 1994b). High confidence intervals on the central values are judged to be 0.1 to 0.4. for adults, 0.1 to 0.5 for 5-year-old children, and 0.15 to 0.75 for infants.

Iodine:

The absorption of dietary iodine in the iodine form in the gastrointestinal tract of humans is rapid and virtually complete, as demonstrated in a number of human volunteer studies reviewed by Underwood (1971). An f_I value of 1 is applied to all intakes of iodine at all ages (3,7) (ICRP, 1990). A high confidence interval on the central value of f_I is chosen to be 0.9 - 1.

Cesium:

Reliable data are available for cesium absorption in humans, and these show that absorption is virtually complete following administration of Cs in soluble inorganic form (Rossof et al., 1963; Le Roy et al., 1966; Rundo et al., 1963). However, values of 0.6-0.99 have been estimated for volunteers consuming ^{137}Cs in contaminated meat (Henrichs et al., 1989; Talbot et al., 1993). Thus, an absorption fraction as low as 0.8 or 0.9 should not be excluded. An f_I value of 1 is applied as the central value to

all intakes of cesium at all ages (ICRP, 1990). High confidence intervals on the central values of f_I are judged to be 0.8 - 1 for all classes of age.

Plutonium biologically incorporated:

Three studies of absorption in humans have been reported. The first by Mussalo-Rauhamaa et al. (1984) used autopsy analyses from tissues from residents of Lapland and of southern Finland to derive an indirect value for f_I in two subjects which suggested values between 8×10^{-4} and 1×10^{-4} . In view of the indirect nature of this study the inherent variability of these values must be assumed to be probably rather larger than the two latter studies which involved direct measurements.

Hunt et al. (1986, 1990) calculated f_I values from 16 data sets derived from studies in eight volunteers, two women and six men aged 31 to 46 years, who ingested winkles containing $^{239/240}\text{Pu}$ and ^{241}Am . The reported values ranged from 4×10^{-4} to 5×10^{-4} , with a median of 1×10^{-4} and a mean of 2×10^{-4} (± 1 SD), suggesting 90% confidence limits of about 8×10^{-5} to 4×10^{-4} .

Most recently Popplewell et al. (1994) reported measurements of the absorption of ^{244}Pu citrate in five volunteers and more recently data for two additional volunteers have become available.¹

There is strong experimental evidence to conclude that Pu absorption from the gastrointestinal tract may be increased by at least an order of magnitude in the human neonate, but that any increased absorption would probably decrease rapidly during the first few days or weeks of life (ICRP, 1986). The age by which absorption of Pu might decrease to adult levels is not known, but animal studies indicate that adult values may be reached by about the time of weaning. An NEA/OECD Expert Group (NEA, 1988) proposed an f_I of 10^{-2} as an average for the first year of life and 10^{-3} for all following years.

On these bases, central values are those of ICRP 56, and a range of f_I is chosen to be 1×10^{-4} to 1×10^{-3} for adults and 5-year-old children and 1×10^{-4} to 1×10^{-2} for infants. This latter value differs from the task group opinion which suggests 5×10^{-3} .

Plutonium as refractory oxide:

ICRP Publication 48 (ICRP, 1986) provided a further review of the literature relating to the gastrointestinal absorption of refractory oxide. The main confusion found in this document is the classification of oxide as a unique type. Indeed, the document sometimes mixed PuO_2 data corresponding to pure oxide with data corresponding to oxide produced during accidental situations (Stather et al., 1979; Métivier et al., 1980). In these latter cases, the solubility is enhanced, but does not really correspond to "Refractory oxide." This explains why central values and 5th and 95th percentiles given here are lower than those given by ICRP 48 and 71 (ICRP, 1986; ICRP, 1996).

Systemic distribution and retention

These questions cover important contributions to uncertainty in calculating doses from radionuclides reaching blood. Factors omitted that might also contribute significantly to uncertainties are the location of sensitive cells in bone and absorbed fractions for alpha- and beta-emitting bone-seekers, and tissue mass and geometric considerations. The behavior of the elements is considered, taking no account of radioactive half-lives of isotopes.

Strontium, Plutonium, Cerium, Tellurium

Questions 10 and 11. Rationale

Strontium:

Most of the strontium is deposited in skeleton from which it is slowly released. The model used here is the ICRP 67 model (ICRP, 1994b) which is derived from the Leggett's recycling model (Leggett, 1992).

Plutonium:

The main uncertainty is the choice of systemic model. In LUDEP for example, calculations are performed with the ICRP 30 model (ICRP, 1979). Since its publication ICRP 30 has revised its actinides model in its publication 56 (ICRP, 1990) then introduced new modifications in its publication 67 (ICRP, 1994b), adding new soft tissues compartments, one liver compartment, one kidney compartment, gonads, and modifying some transfers in bone. This new model is a recycling model and leads to halftime for plutonium longer than ICRP 30 and significantly longer than ICRP 56.

1. Popplewell, D.S., Ham, G.J., McCarthy, W., Lands, C. 1995. Private communication.

These new modifications elicited some comments (Priest, 1990) and introduced some skepticism in regard to comparisons with human data.

The ICRP model based on Leggett's model (Leggett and Eckerman, 1984; Leggett, 1985) is assumed to be in good agreement with human data reported by Kathren et al. (1988) and McInroy et al. (1989) for liver. Toohey (1994) compared the ICRP 48 model and data reported by Kathren and McInroy in 1992 and found a good similarity not consistent with the previous assumptions. However, a description of experience obtained during 25 years by the United States Transuranium and Uranium Registries clearly showed a good similarity between ICRP 48 model and USTUR observations (Kathren, 1994).

For example, in ICRP 30 retention of plutonium in the skeleton was concluded to be $T_{1/2} = 100$ years. On the basis of new human data, ICRP 48 recommended a retention time of 50 years. The new recycling model leads to values ranging from 100 to 140 years which seems not to be in agreement with more recent human data (Kathren, 1994) and observations in dogs and baboons (Métivier et al., 1989; Park et al., 1991).

These controversial models raise the problem of uncertainty or credibility of the new ICRP model. Central values obtained by approximation or graphic estimation of ICRP 67 are not too far from Bennet's estimates (1974).

Calculations with the LUDEP logiciel using the ICRP 30 model do not really differ.

Table 1.

ICRP Report No.	30	56	67 Graphic estimate	67
1 day	84	80		80
1 week	90	80		80
1 month	89	80		80
1 year	88	77		79
10 years	79.8	58.5	75	72
50 years	50.7	25	50-53	49

For children, there are no data for ICRP 30.

Table 2.

ICRP Report No.	56	67 Graphic estimate	67
1 day	80		80
1 week	79.2		80
1 month	77		80
1 year	52		74
10 years	10	63	47
50 years	0.6	45-48	12

Therefore, we have to consider here that the type of model is the main source of uncertainty.

Another source of uncertainty is the chemical form of injected plutonium. Results should be very different if plutonium were injected as nitrate, but as an estimation of uncertainty of plutonium systemically incorporated we have to consider either the distribution of citrate form or transferring complex. The two complexes (thermodynamically strong) distribute plutonium preferentially in bone.

Cerium:

There is no new systemic model for rare earth, so the model of ICRP 30 (ICRP, 1979) is still valid or still used. Data are not very numerous, and dispersion is difficult to establish. Data obtained in dogs (Boecker and Cuddihy, 1974) showed a range of a factor of two in individual distribution. On this scarce basis, the central values are those obtained in ICRP 30 or 56 for adult and ICRP 56 for 5-year-old children. The distribution between liver and skeleton is assumed to be constant with time in the absence of more information.

Distribution between liver and skeleton varies in the rare-earth family (Durbin, 1960). This has been attributed (Masse et al., 1973) to the stability constant of the complex in blood. Variation is important in the series but should remain constant for an element with time. However, if a recycling model were introduced, this parameter should be taken in account for each element of the series.

Uncertainties are estimated to be quite similar to those for plutonium.

Tellurium:

There are limited published data on the distribution and retention of tellurium in humans and animal. As urinary to

fecal excretion is assumed by ICRP 67 (ICRP, 1994b) to be 4:1, it is assumed that the liver retention not described here exists for initial times but disappears quickly because of the large difference between the two half-times retained by ICRP 67. It was the rationale used for this exercise.

Adult values are taken for children because information appears to be unavailable.

Uncertainties are assumed to be at least 20% around central values.

Antimony:

There are even fewer published data on antimony than on tellurium. Uncertainty determination here is quite an impossible exercise. Values given are ICRP 69 values (ICRP, 1995). Moreover, antimony distribution is species-dependent.

Adult values are taken for children because information appears to be unavailable.

Uncertainties are assumed to be at least 30% around central values.

Questions 12 and 13. Rationale

There are some controversies around the recycling model and I think that some discussions about this model are again needed. However some values are given for adults on the basis of the more extensive studies in humans and baboons (Priest et al., 1992). Wronski et al. (1980) have shown that the dynamic microdistribution of ^{239}Pu within the skeleton of beagles is intimately correlated with skeletal remodeling which could explain a decrease from 1 day to 1 year by a factor of 2 to 4. If we use recycling models of ICRP Publication 67 (1994b) these values, obtained by a new code (Malarbet, 1997), slowly differs for endosteal surfaces in adult then plutonium is brought again to the surface by recycling. For 5 year old infants, the values greatly differ due to bone remodelling. However, these values do not seem consistent with ICRP Publication 48 (1986).

Question 14. Rationale

The values given are the retention in red bone marrow as a percent of total skeletal retention: total skeleton = bone + bone marrow.

After the ICRP 30 model, ICRP has developed an age-dependent systemic model (ICRP, 1990) modified a few

years later (ICRP, 1994b). This model strongly differs from ICRP Publication 48.

There are several differences between Publication 56 and Publication 67, particularly at bone level. There is no longer transfer from cortical marrow to cortical surface and trabecular marrow to trabecular surface. As results do not strongly differ from ICRP Publication 30 (Priest et al., 1990; Priest et al., 1992), the old model and Priest's data are used as central values for bone marrow estimates.

These values seem in good agreement with Smith's observations in adults (Smith et al., 1984). However, the values could differ if they are calculated as trabecular bone marrow/bone or bone marrow to bone. This is not very important for adults, in amount, as 50 years after entry in blood the values range from 1.1 to 2, but this more important, as dosimetric end-point, for infants 9 to 18, even if the ratio remains the same.

Ruthenium, Cesium

Question 15. Rationale

Ruthenium:

Estimations of central values are based on ICRP Publications 56 and 67 (ICRP, 1990; ICRP, 1994b). The only difference between the two publications is the introduction of urinary/fecal excretion for bladder wall dose calculation. Moreover the parameters now recommended are the same as ICRP 30 (1979) for adults and all ages, except for 3-month infants for whom the f_1 value is twice that of the other groups.

Ruthenium is assumed to be uniformly distributed with a range of a factor of three in four different animal species (Furchner et al., 1971). This distribution is largely independent of the form of initial intake (Thompson et al., 1958). Uncertainty lies in a slow turnover compartment for which Furchner et al. (1971) reported that for 15-20% of activity entering the blood, biological half-time varied between 200 and 1670 days. There are also differences for shorter compartments, but incidence on dose calculation is smaller.

Calculations are made with ICRP-recommended parameters for central values, and 5th and 95th percentiles are obtained with variations of half-time for the long-term compartment. This modification is not significant for early times but becomes significant after 1 month. In regard to the lack of

human data, the uncertainties should be higher than those assumed in this report.

There are no data on age-related changes in the retention of ruthenium. The central value is assumed to be the same as for adults, with the uncertainties for 5th and 95th percentiles increased.

Cesium:

In contrast to ruthenium, there are many reliable cesium data about humans.

Central values used here are based on ICRP Publications 30, 56, and 67 (ICRP, 1979; ICRP, 1990; ICRP, 1994b) where two compartments are described. The first one, 10%, has a biological half-time of two days, and the second one, 90%, has an average biological half-time of 110 days. It is this second compartment which is significant for dose calculations.

Retention half-times for the long-term compartment in females are reported to be less than in males: 96 days for males (range 47-152 days) (ICRP, 1979), 61 - 65 days for females (range 30-141 days) (Clemente et al., 1971; Miltenberg et al., 1981; Schwartz et al., 1982; Henrichs et al., 1989).

The uncertainties for 5th and 95th percentiles determination are based on a variation of biological half-time from 40 to 140 days.

We also have reliable data for children. The rate of loss of cesium from the body is increased, compared to adults, with the exception of neonates (ICRP, 1990). Age-dependent variations in the retention of cesium in the body have been related to changes in body mass and body potassium content (Leggett, 1986).

The central values are obtained with two compartments which represent 45% and 55% of cesium distribution with biological half-times of 9.1 and 30 days respectively. As the half-times are not so different from the adults, the uncertainties will depend on the estimation for each compartment. Because the retention is mass-dependent, we have adopted for the 5th and 95th percentiles some extreme situations (i.e., 1-year-old children and 10-year-old children).

Iodine

Question 16. Rationale

ICRP has introduced in ICRP Publication 56 (ICRP, 1990) an age-dependent model for iodine which is not really different from ICRP 30 (ICRP, 1979) for adults. For central estimation, uptake by thyroid is assumed to be 30% with two compartments, the first one at 28.7% with a biological half-time of 110 days and the second one at 1.3% with a biological half-time of 11.5 days. The half-time in blood compartment, 0.25 days, is constant.

A second estimation is done with only one compartment with an half-time of 80 days as recommended by ICRP 56.

For children, the ICRP Publication 56 (ICRP, 1990) is used as a reference for central values. For determination of the 5th and 95th percentiles we have changed the half-times, 17-23 days as lower values and 30-50 days as higher values.

The new model is a recycling model which is important for long lived isotopes. In the case of an accident, the long-lived isotopes do not play a significant role in dose calculation. The significant parameters are the uptake by blood from gut absorption and uptake by the thyroid gland from the blood compartment during the first 50 days following accident. The main uncertainty lies in uptake to blood, not considered here, which depends on the health status of the public.

Whether this uptake plays a significant role in the half-time is the main question for dosimetric purposes. Ermans (1991) gave similar retention curves for an uptake of 25 $\mu\text{g}/\text{day}$, 100 $\mu\text{g}/\text{day}$, and 500 $\mu\text{g}/\text{day}$ and clearly assumed that clearance of iodine from the thyroid gland is slightly dependent on an overload of the gland with thyroid.

This assumption confirms to us that the main uncertainty in dose estimation lies in biological half-time.

Dose coefficients

Inhalation and ingestion dose coefficients represent ACA code inputs. Uncertainties will include dosimetric modeling considerations as well as the parameters considered above.

Question 17. Rationale

Strontium:

As explained in rationale for Question 7, the central values are based on strontium Type M compounds with an f_I of 0.1.

For ingestion, the 95th/5th percentiles ratios are taken as 3.6 for adults and 4 for children as the results of dose calculations with parameters discussed in rationale for Question 9.

As interim values, the same ratios are used for 95th/5th percentile ratios for inhalation.

Tellurium and Iodine: decide proportions of particulate and vapors:

In case of severe accident, the nature of tellurium or iodine released by the reactor will depend on the scenario:

1. The first one is a LOCA, with rupture of the fuel claddings and aspersion inside the reactor building. Release of radionuclides occurs after filtration during the first 48 hours after the beginning of the accident. With this scenario we estimate that tellurium is always released as particulate form, and for iodine, 74% as particulates, 24% in organic form, and 2% in molecular form (Collinet and Rousseau, 1990).
2. The second scenario is a core fusion without aspersion into the building, with release through filters during the first 48 hours after the beginning of the accident. In this case tellurium is again released as particulate form, and for iodine, 90% as particulates, 6% in organic form, and 4% in molecular form.¹

These two sets of data are integrated data during the first 48 hours; it is a rough estimate which could be discussed.

1. Manesse, D. 1996. Personal communication.

These ratios could change significantly depending on the kinetics of release. In any case, the physicochemical form of iodine mainly influences ground deposition because in the models, depositions of organic or elemental forms are considered as negligible compared to particulate forms.

Iodine:

Inhalation:

Adult: Central values given by ICRP 71 (ICRP, 1996) are 1.5×10^{-7} Sv/Bq for the particulate form, 3.1×10^{-7} Sv/Bq for the methyl iodine form, and 3.9×10^{-7} Sv/Bq for the elemental form. Scenario 1 gives a dose for thyroid of 1.9×10^{-7} Sv/Bq; the second scenario gives 1.7×10^{-7} Sv/Bq.

Children: Central values given by ICRP 71 (ICRP, 1996) are 7.3×10^{-7} Sv/Bq for the particulate form, 1.5×10^{-6} Sv/Bq for the methyl iodine form and 1.9×10^{-6} Sv/Bq for the elemental form. Scenario 1 gives a dose for thyroid of 9×10^{-7} Sv/Bq, and the second scenario gives 8×10^{-7} Sv/Bq.

The main uncertainty lies in the chemical form and the kinetics of release. It is certain that the more iodine is released as particulate form, the less the dose to the thyroid. But as the particulate form content decreases with time, the relative proportion of elemental or organic forms will increase. This conclusion is of significant importance for the management of a severe accident. Indeed, the decision maker has to know that if release is postponed, the particulate form content will decrease, but the consequences in terms of dose calculation could be affected if the content of elemental and organic forms is not similarly decreased.

Table 3.

	Percentage of release	Thyroid dose Sv/Bq	Contribution to total dose		
			Type F	Type M	Type S
Methyl iodine	24	3.1×10^{-7}	0.74×10^{-7}	0.74×10^{-7}	0.74×10^{-7}
Elemental	2	3.9×10^{-7}	0.078×10^{-7}	0.078×10^{-7}	0.078×10^{-7}
Type F particles	74	1.5×10^{-7}	1.1×10^{-7}		
Type M particles	74	0.22×10^{-7}		0.16×10^{-7}	
Type S particles	74	0.011×10^{-7}			0.008×10^{-7}
Total			1.9×10^{-7}	1×10^{-7}	0.8×10^{-7}

The first estimate is given with Type F soluble particles.

What will be the difference if particulate iodine is released as AgI insoluble form from a reactor containing Ag-In-Cd control rods? Table 3 gives total dose for Scenario 1 (24%, 2%, 74%) with the particulate form having fast (F), moderate (M) and slow (S) solubility.

We can conclude, however, from Power's solubility hypothesis for particles (Powers, 1996), the total dose to the thyroid for adults does not differ by more than a factor of two if we retain any scenario of a mixed release of gas and aerosol. The main uncertainty lies with the chemical form of release, elemental or particulate, which could lead to a more than two orders-of-magnitude difference if the two extreme situations, gas or insolubles particles, are considered.

Consequently, we can assume that uncertainty due to dose calculation is estimated to be around 20 - 40%, and depends only on the source term. The critical effort needed in safety research is a better knowledge of the source term both in quantity and in quality.

For ingestion, the scenario is quite different because we consider deposits of elemental or methyl forms negligible compared to particulate form. The central value is organically incorporated or a deposited form with an f_I of 1.

Uncertainty is estimated to be around 10%.

Tellurium:

Inhalation: the dose to lungs from ^{132}Te differs by a factor of three depending on whether gas and vapor forms or Type F particulate forms are assumed, as explained in the rationale of Question 7. The doses are 6.6×10^{-10} Sv/Bq and 2×10^{-10} Sv/Bq, respectively, for adults and 1.8×10^{-9} Sv/Bq and 6.1×10^{-10} Sv/Bq for children. The ratio is the same for thyroid and colon. However, the scenario used assumes that all the tellurium is released as particulate form.

If we compare Type M or Type S, the dose to lungs is almost the same, 1×10^{-8} Sv/Bq or 1.1×10^{-8} Sv/Bq, respectively, but 100 times higher than for Type F.

The main uncertainty is the solubility of particles, and it is in this area that future research efforts should be made to provide better information to decision makers.

Another uncertainty lies in the complexity of biological behavior of different chemical forms of tellurium. We do not really know the chemical form released: tellurites, tellurides, tellurates. It is clear that this knowledge is important. Elemental tellurium is insoluble, while tellurites and tellurates are soluble.

We consider here that the tellurium is released in soluble form, in disagreement with Powers' recommendations (1996).

Cesium:

Dose calculations and percentiles are given on the basis of the rationale of Question 7 for inhalation and the rationale of Question 9 for ingestion.

Cerium:

Dose calculations and percentiles are given on the basis of the rationale of Question 7 for inhalation.

Plutonium:

Dose calculations and percentiles are given on the basis of the rationale for Questions 7 and 9. The main uncertainty for inhalation is once more dependent on time of release after the accident, because during this period of time some reactions should strongly change the solubility of particles. For ingestion, the uncertainty is probably less if plutonium is transformed and ingested as biologically incorporated, but greater if ingestion is of particles deposited on vegetables not washed well before ingestion. Indeed, we know now that the washing of vegetables recommended after Chernobyl is not really efficient (NEA, 1996).

Question (xi) Joint dosimetry/late question: Rationale

This question is a joint question for the Internal Dosimetry and Late (Stochastic) Health Effects panels and we have to thank the elicitors for organizing a bridge between the two groups. However, the rationale for this question will perhaps show that in some cases, uncertainties, so important to internal dosimetry, could be irrelevant to the real problem which is the evaluation of uncertainties of cancer deaths in the two probabilistic accident consequence assessment codes, MACCS and COSYMA (EC/NEA, 1994).

I wish to introduce a discussion about the objectives of cancer-risks estimates because two differing concepts could be developed.

Management of workers in a practice as defined by Publication 60 of the ICRP (ICRP, 1991):

In this case the decision maker has to manage a complex situation with potential or real irradiation of workers. He has to use an easy system that is useful in complex situations and provides occupational medicine specialists, health physicists, managers, and social partners with a common tool for risk management and discussions. This system exists; it is the ICRP system. With the new recommendations (ICRP, 1991), the concept of dose is clarified; a dose equivalent system permits the use of a unique concept for the different ionizing radiations α , β , γ , and neutrons, despite many differences in the fundamental interactions; and the effective dose takes into account the new data on relative risks for different organs. The ICRP system, based on caution, uses only one unit (Sievert) for risk estimates, which permits use of statistics and implementation of the ALARA principle.

The general feeling of the health physicists was that, with implementation of this system, a substantial and progressive decrease of doses has been shown during the last decades for workers and members of the public, and consequently a substantial decrease of risk. It is the main argument for implementing such a system.

Is this system universal? It does not seem so to me.

When the expert has to manage an accident situation, the general philosophy should not be the same as when assessing accident consequences.

After an accident, ICRP's concepts are clear. Intervention does not need any regulation, and the decision makers assess constraints or objectives but do not have to establish limits for their actions.

Why do we use a different concept for accidental consequence assessment?

As the panel is for internal dosimetry, the elicitors set the question for two interesting nuclides ^{239}Pu and ^{90}Sr . Do we have sufficient information in these two cases to permit a different approach for consequence assessment?

239-Plutonium dioxide:

This oxide is insoluble, and the dose estimates made by using default values of ICRP Publication 71 (1996), or any other calculations like the one used in the answers to Question 17 lead to the conclusions in Table 4.

Table 4.

	Dose (Sv)
Lung	9×10^{-1}
Bone	1.8
Liver	4×10^{-1}
Bone marrow	9×10^{-2}
Effective dose	1.6×10^{-1}

We can use cancer risk factors for the answer to the question.

The first set of values could be the ICRP 60 (ICRP, 1991) cancer risk factors; the second set of values could be the GSF cancer risk factors given in COSYMA code; and the third set of values could be the cancer risk factors given by the BEIR IV report, which are obtained from animal experiments in different species for plutonium and human observations (radium and thorium). All these values are questionable.

- The lung cancer risk factors plotted in Table 5 are obtained from animal experiments for insoluble alpha-emitters already described in ICRP Publication 31 (1980).
- The bone cancer risk factors are obtained from experiments where plutonium is intravenously injected and range from 0.4×10^{-3} to $5.4 \times 10^{-3}/\text{Sv}$ (95% confidence interval). These values are five to ten times higher than the corresponding estimates of the effects of two isotopes of radium.

Table 5.

	ICRP 60	COSYMA	BEIR IV
Lung	$8.5 \times 10^{-3}/\text{Sv}$	$9 \times 10^{-3}/\text{Sv}$	$3.6 \times 10^{-4}/\text{Sv}$
Bone	$5 \times 10^{-4}/\text{Sv}$	$1.33 \times 10^{-4}/\text{Sv}$	$0.85 \times 10^{-4}/\text{Sv}$
Liver	$1.5 \times 10^{-3}/\text{Sv}$	$4.67 \times 10^{-3}/\text{Sv}$	$2.6 \times 10^{-4}/\text{Sv}$
Bone marrow	$5 \times 10^{-3}/\text{Sv}$	$5.16 \times 10^{-3}/\text{Sv}$	$1 \times 10^{-4}/\text{Sv}$
All cancers	$5 \times 10^{-2}/\text{Sv}$		

- The liver cancer risk factors are obtained from experiments where transuranic elements were intravenously injected. This value is three to three and a half times higher than the corresponding estimates for internally deposited thorotrust in humans. The higher estimate could be used here because there are

significant differences between thorotrast aggregates deposit and plutonium deposit which is transported to liver by transferring or citrate complexes. In this second situation we can expect a more diffuse location of alpha emitter and consequently a higher cancer risk.

- Risk estimates from transuranic elements for leukemia are applied to cancers known to originate from other sources of irradiation such as external gamma and X radiation.

All these risk factors are controversial. Calculations that may be appropriate for radiation protection purposes (e.g., those by the ICRP) may be entirely misleading for projecting risks of cancer mortality from plutonium exposures.

Leukemia:

Leukemia has only rarely been observed in extensive studies on primates, dogs, and several species of rodent. Leukemia has never been identified as a probable cause of death in animal experiments with transuranic elements (Bair et al., 1980; Bair et al., 1989; Métivier et al., 1989; Park et al., 1991) or observed following human exposures (Wilkinson et al., 1987; Wiggs et al., 1994).

Leukemia has been observed in a particular strain of mouse injected with small amounts of plutonium nitrate (Humphreys et al., 1987). On the basis of these experiments on one strain of mouse, there is no strong evidence that plutonium causes leukemia at low doses, but a possible causal relationship cannot be excluded entirely (Clarke et al., 1995).

The central value is equal to zero. The 95% percentile could be derived from ICRP publication, i.e., 73,500. It seems to me that a value one thousand time less, 70, is more rational and probably less pessimistic.

Lung cancers:

The dose-effect relationship for lung cancers following inhalation of plutonium dioxide is not a linear curve because there are results that indicate the presence of a "practical" threshold dose of about 1 Gy for lung tumor formation, below which radiation-induced lung cancer would be extremely unlikely. The dose-response relationship appears to fit to a quadratic function and a maximum lung tumor incidence at about 8 Gy (Sanders et al., 1988; Sanders and McDonald, 1992; Sanders and Lundgren, 1995). The Sanders' observations are confirmed by two experiments in beagle dogs which had inhaled $^{238}\text{PuO}_2$ and $^{239}\text{PuO}_2$ (Park

et al., 1991). The dogs data fitted well to a pure quadratic model with different coefficients for $^{238}\text{PuO}_2$ and $^{239}\text{PuO}_2$ (Raabe et al., 1994). Moreover, another rat strain, F344, appears to be more sensitive than the Wistar strain to formation of lung tumors, and the same dose-effect relationship was recently observed (Sanders et al., 1995). Linear models were strongly rejected, and a linear-quadratic function did not improve the fit over a pure quadratic one (Raabe, 1994). The extrapolated lifetime risk at 1 rad was estimated to be 7 per 10^6 for $^{239}\text{PuO}_2$. For comparison, the lifetime risk at 1 rad for uranium miners in BEIR IV was 700 per 10^6 .

Experiments with baboons have shown a cancer deficit when compared to canine experiments and, moreover, with the ICRP estimates, which leads us to think that primates could be less sensitive than dogs and rats (Bair et al., 1980; Métivier et al., 1989).

This practical threshold could be explained by the effects of plutonium irradiation on immune modulation (Park et al., 1972; Nolibé et al., 1981; Nolibé et al., 1983).

These observations in animals are consistent with the follow-up of the Manhattan project workers and more significantly by follow-up on the Los Alamos workers (Voelz and Lawrence, 1991; Wiggs et al., 1994).

The ICRP risks factors on the other hand are based mainly on Hiroshima-Nagasaki survivors and external irradiation.

The numbers of cancers obtained from these different risk-factors differ: 740,000 fatal cancers from ICRP estimates, 30,600 fatal cancers from BEIR IV estimates. The threshold obtained in rat and dog experiments, corresponding to inhalation of 100,000-200,000 Bq by a man, explains why the value of zero is given as 5% percentile for lung cancers.

Populations are not homogeneous, and some members smoke. The combined effect of an α -emitter and cigarette smoke has been investigated in both experimental animals and epidemiological studies. In the same context, Métivier et al. (1984b) described a joint exposure of $^{239}\text{PuO}_2$ and B(a)P, a major component of tobacco smoke; a multiplicative relative risk model was found. The relative risks described increased with inhaled plutonium dioxide amount by rats, from a value of 8.4 for the lowest exposure (220 Bq) to 18.8 for the highest (6300 Bq) for total tumor incidence, but from a factor of 26 to one of 47 for the highest exposure but B(a)P remaining at the same dose. The lower exposure corresponds in humans to inhalation of

about 100 kBq of plutonium and heavy smoking habits (two packs/day).

On the basis of these observations, the most pessimistic observation could lead to about one thousand cancers from the combined effects of tobacco and plutonium for a population of smokers. The central value would be less.

These estimates could change with the size of particles, because it has been demonstrated that the smaller the size of particles, the more lung is irradiated, and the higher the risk of cancer.

These estimates could also change with the amount of inhaled material, because the dose-effect relationship is threshold-linear quadratic. However, the probability for the public to breath an amount of plutonium higher than 200 Bq is very low and quite inconsistent with a calculation for 1.108 persons.

Bone cancers:

The same rationale could be developed for bone cancers. If we assume that the risk factor is ten times higher than for radium (5 to 10 for BEIR IV report, 16+5 for Lloyd et al., 1994), we do not exceed the threshold dose of one tenth of the threshold observed with ^{226}Ra , i.e., about 8 Gy. This threshold cannot be dismissed today (Rowland, 1994). Clarke et al. (1995) assumed that bone tumors have not been seen after deposition of high-fired plutonium-239 oxides in the respiratory tract, presumably reflecting the fact that only small amounts of plutonium reach the bone and that most of the plutonium is buried in the bone matrix before it decays. In contrast, bone tumors have frequently been seen when soluble forms of $^{238}\text{PuO}_2$ and $^{239}\text{PuO}_2$ and plutonium-238 compounds including the oxide have been inhaled (Métivier et al., 1988). However, in such cases plutonium is released as very small size particles (40 nm), and solubility is consequently increased. The hypothesis retained here by elicitors is an insoluble plutonium dioxide.

The estimate from ICRP Publication 60 should be about 92,000 bone cancers; the estimate from the BEIR IV report should be about 15,300 bone cancers; and a 95th percentile value of a value one hundred less could be proposed.

Liver cancers:

Primary liver tumors involving functional liver cells have rarely been seen in dogs and rodents after the inhalation or

ingestion of plutonium compounds (Clarke et al., 1995). This is confirmed by Weller et al. (1995) which concluded that in an experiment where beagles inhaled $^{238}\text{PuO}_2$, more soluble than $^{239}\text{PuO}_2$, liver cancer was an uncommon cause of death, and the majority of liver tumors, which were incidental findings at necropsy, occurred in dogs that survived beyond the median life expectancy of the controls'.

The estimate from ICRP Publication 60 should be about 51,000 liver cancers; the estimate from the BEIR IV report should be about 10,000 bone cancers. A 95th percentile value one hundred fewer could be proposed.

Table 6.

Particle size (μm)	Particles number	Particle activity (mBq)	Number of irradiated cells	Irradiated lung fraction
0.1	5.4×10^7	0,01	3×10^7	30
0.3	2.0×10^6	0,4	1.3×10^6	1
0.7	1.8×10^5	3	1.2×10^5	0.1
1.0	5.4×10^4	11	3.6×10^4	0.03

All cancers:

On the basis of experiments already quoted above, it is difficult to give a clear answer for the number of other cancers. Sanders and McDonald (1992) described nonpulmonary tumors in control and exposed group to $^{239}\text{PuO}_2$ aerosol. Reduced incidence of nonpulmonary tumors was observed at the highest dose levels, associated with decreased survival from induced lung tumors and radiation pneumonitis/fibrosis. This level is higher than the level of the elicitor's question.

In conclusion, the risk assessment of an accident with release of plutonium dioxide could not be performed with the classical estimation recommended by ICRP for radiation protection purposes. It seems important to use the information specific to each case. For example, a nuclear weapon accident will be different from a dispersion during PuO_2 transportation and different from a nuclear reactor accident. The late effects induced by inhalation of plutonium oxide would be dependent on chemical form of plutonium released, size of particles, and inhaled quantity. However, we have observed in several accidents (Windscale, Rocky Flats, and Chernobyl) that plutonium is an element which is never broadly released.

Strontium oxide:

This oxide is insoluble (Powers, 1996). The dose estimates using default values of ICRP Publication 71 (1996) or any other calculations, like those used in the answers to Question 17 lead to Table 7 data.

Table 7.

	Dose (Sv)
Lung	1.3×10^{-2}
Bone	1.9×10^{-4}
Bone Marrow	8.5×10^{-5}
Effective dose	1.6×10^{-3}

The rationale developed here could be the same as for plutonium, although I have not enough time to collect all the information required for such an estimation.

If we adopt the ICRP cancers risk factors, there will be 9350 lung cancers, 28 bone cancers, 55 leukemia, and about 7000 cancers of all types. On the basis of a lifetime beagle study based at the University of California-Davis, Raabe and Parks (1993) assumed that bone cancer and leukemia demonstrated marked threshold-like responses with no evidence of radiation induced cancer death in any of the lowest dose levels. These levels are higher than the doses reported in the above table.

In an study of population of the Techa River, an absolute excess risk of $0.48 - 1.1$ per 10^4 persons Gy has been obtained for leukemia (Kossenko and Degteva, 1994). This estimate is three to five times lower than the corresponding estimate obtained from the follow up on the atomic bomb survivors.

This could explain why for leukemia the central value is assumed to be 10. The ICRP estimates are adopted here for the 95th percentile. However, it is necessary to have stronger dosimetry (Degteva et al., 1994) before using these data which are not in great contradiction with ICRP recommendations.

For lung tumors, with regard to the lack of information, we shall take into account the results of the Techa River population study and the conclusion of the authors who assumed that lung cancer risk factors are not statistically confirmed but are not substantially different from the corresponding values attributed to the atomic bomb survivors. However, the standard errors are considerable (Kossenko and Degteva, 1994).

Seed variables (unpublished data).

Study 1

A study is in progress in which ^{244}Pu citrate has been administered to three healthy adult volunteers as a single intravenous injection in citrate solution. Predict 5%, 50%, and 95% values for 24 hour urinary excretion as % total administered for the following times after administration: 45 d, 68 d, 75 d, 325 d, 378 d, 382 d, and 1155 d.

Table 8.

24 hours urinary excretion as % total administered after	5%	50%	95%
45 days	7×10^{-3}	9.6×10^{-3}	1.3×10^{-2}
68 days	4.5×10^{-3}	7.3×10^{-3}	1.1×10^{-2}
75 days	4×10^{-3}	6.8×10^{-3}	1×10^{-2}
325 days	1.5×10^{-3}	2.4×10^{-3}	3.5×10^{-3}
378 days	1.3×10^{-3}	2.2×10^{-3}	3.2×10^{-3}
382 days	1.3×10^{-3}	2.2×10^{-3}	3.2×10^{-3}
1155 days	7×10^{-4}	1.1×10^{-3}	1.6×10^{-3}

Study 2

A study is in progress in which terbium oxide ($^{160}\text{Tb}_4\text{O}_7$) has been inhaled by four healthy human volunteers, all non-smokers with normal respiratory and metabolic functions. Monodisperse particles (1.2 mm) were generated from a water suspension and inhaled over a period of a few minutes. Predict 5%, 50% and 95% values for lung retention and 24 hour urinary excretion as a % total inhaled for the following times after administration: Lung: 2 d, 9 d, 16 d, 26 d, 90 d, and 180 d; Urine: 2 d, 8 d, 15 d 30 d, 90 d, and 180 d.

Table 9.

lung retention after as % of total inhaled	5%	50%	95%
2 days	7	10	15
9 days	6	8.1	12
16 days	5	7	10
26 days	4	5.6	7.5
90 days	1	1.7	2.5
180 days	0.1	0.33	0.8

Table 10.

urinary excretion as a % total inhaled after	5%	50%	95%
2 days	0.1	0.19	0.29
8 days	0.02	0.036	0.054
15 days	0.015	0.024	0.036
30 days	0.09	0.014	0.021
90 days	0.003	0.0045	0.0068
180 days	0.0007	0.0011	0.0017

Study 3

A study has been undertaken in which two adult female baboons in late pregnancy were given a single intravenous injection of a mixture of plutonium-239, americium-241, neptunium-237, and polonium-210 in citrate solution. The animals were killed after seven days. Predict 5%, 50%, and 95% values for retention of the four nuclides in maternal liver, femora, spleen, and kidneys as a percentage of total administered activities.

Table 11.

retention as a % of total administered activities	5%	50%	95%
in maternal liver for:			
Pu-239	25	30	40
Am-241	22	28	38
Np-237	2	4	8
Po-210	1.5	3	6
in femora for:			
Pu-239	1.5	3	7
Am-241	2	4	8
Np-237	1.5	3	6
Po-210	0.03	0.06	0.12
in spleen for:			
Pu-239	0.2	0.4	0.8
Am-241	0.1	0.2	0.4
Np-237	0.02	0.04	0.1
Po-210	0.2	0.4	0.8
in kidneys for:			
Pu-239	0.2	0.4	0.8
Am-241	0.1	0.2	0.4
Np-237	0.02	0.04	0.1
Po-210	0.7	1.4	2

The question is not well enough defined. The valiancy of each element is deduced, not given. We have no idea of

concentration of citrate ion, and for ^{237}Np a mass effect could be observed.

Study 4

A. A study has been undertaken in which five adult rats (male, 3 months old) were given a single intravenous injection of polonium-210 in citrate solution. Predict 5%, 50%, and 95% values for whole body retention of ^{210}Po as a % total administered for the following times after administration: 1 d, 7 d, 50 d, and 200 d.

Table 12.

whole body retention of ^{210}Po as a % of total administered after	5%	50%	95%
1 day	90	95	98
7 days	65	70	75
50 days	43	48	52
200 days	2	6	9

B. A study has been undertaken in which five adult rats (female, 3 months old) have been fed crabmeat containing natural levels of polonium-210. The crabmeat was eaten over a three week period; regular laboratory food and water were available to the animals at all times. The animals were killed on the Monday following their last Friday eating crabmeat, and tissue retention of ^{210}Po was measured. A parallel study will be undertaken to measure tissue retention in a similar group of rats given a series of intravenous injections of ^{210}Po citrate solution over the same period. In the absence of these data, results from 4A have been used in the estimation of the gastrointestinal absorption of ^{210}Po in animals fed crabmeat. Predict 5%, 50%, and 95% values for the absorbed fraction (f_1) for ingested ^{210}Po obtained in this way.

Table 13.

absorbed fraction of ^{210}Po in animals fed crabmeat	5%	50%	95%
fraction (f_1)	0.07	0.13	0.2

Study 5

A study is in progress in which a dust from a nuclear power plant was administered by intratracheal instillation to adult rats (female, 5 months old). The dust, of unknown chemical form, contained ^{60}Co and ^{137}Cs . The respirable fraction

(<5 mm AMAD) was instilled (about 3 mg in 0.15 ml); information on particle size distribution is not currently available. Predict 5%, 50% and 95% values for lung retention and total retention in other tissues as a percentage of the initial lung deposit for the following times after administration: 1 d, 7 d, 28 d, 168 d. As a clue, estimated f_1 values for this material are 0.77 for ^{137}Cs and 0.08 for ^{60}Co .

Table 14.

lung retention as a % of the initial lung deposit after	5%	50%	95%
1 day			
7 days			
28 days			
168 days			

Table 15.

total retention in other tissue as a % of the initial lung deposit after	5%	50%	95%
1 day			
7 days			
28 days			
168 days			

References:

Bailey, M.R., Kreyling, W.G., André, S., Batchelor, A., Collier, C.G., Drosselmeyer, E., Ferron, G.A., Foster, P., Haider, B., Hodgson, A., Masse, R., Métivier, H., Morgan, A., Muller, H.L., Patrick, G., Pearman, I., Pickering, S., Ramsden, D.S. 1989. "An interspecies comparison of the lung clearance of inhaled monodisperse cobalt oxide particles - Part 1: Objectives and summary of results." *J. Aerosol Sci.* 20, 169-188.

Bailey, M.R., Roy, M. 1994. *Annex E. Clearance of Particles from Respiratory Tract*. In: *Human Respiratory Tract Model for Radiological Protection*, ICRP Publication 66, Elsevier Science Ltd., Oxford, pp. 301-413.

Bair, W.J., Richmond, C.R., Wacholz, B.W. 1974. *A radiobiological assessment of the spatial distribution of radiation dose from inhaled plutonium*. USEC WASH-132.

Bair, W.J., Métivier, H., Park, J.F. 1980. "Comparison of Early Mortality in Baboons and Dogs After Inhalation of 239 Plutonium Dioxide," *Radiat. Res.* 82, 588-610.

Bair, W.J., Park, J., Dagle, G.E., James, A.C. 1989. "Overview of biological consequences of exposure to plutonium and higher actinides." *Radiat. Prot. Dosim.* 26, 125-139.

Becquemin, M.H., Yu, C.P., Roy, M., Bouchiki, A. 1991. "Total deposition of inhaled particles related to age: Comparison with age-dependent model calculations." *Radiat. Prot. Dosim.* 38, 32-28.

Bedford, J., Harrison, G.E., Raymond, W.H.A., Sutton, A. 1960. "The metabolism of strontium in children," *Br. Med J.* 1, 589-592.

BEIR. 1988. *Health risks of radon and other internally deposited alpha-emitters*. Committee on the Biological Effects of Ionizing radiations. National Research Council. National Academy Press, Washington, D.C.

Bennett, B.G. 1974. "Transfer of plutonium from the environment to man." In: *Health and Safety Laboratory Fallout Program Quarterly Summary Report*, HASL 278, New York.

Birchall, A., Bailey, M.R., James, A.C. 1991. "LUDEP: A Lung Dose Evaluation Program." *Radiat. Prot. Dosim.* 38, 167-174.

Boecker, B.B., Cuddihy, R.G. 1974. "Toxicity of ^{144}Ce inhaled as $^{144}\text{CeCl}_3$ by the beagle: metabolism and dosimetry." *Radiat. Res.* 60, 133-154.

Bouville, A., Eckerman, K., Griffith, W., Hoffman, O., Leggett, R., Stubbs, J. 1994. "Evaluating the reliability of biokinetic and dosimetric models and parameters used to assess individual doses for risk-assessment purposes." *Radiat. Prot. Dosim.* 53, 211-215.

Borzilov, V.A., Klepikova, N.V. 1993. "Effect of meteorological conditions and release composition on radionuclide deposition after the Chernobyl accident." In: *The Chernobyl papers, Vol. I, Doses to the Soviet Population and Early Health Effects Studies*, Merwin, S.E., Balonov, M.I. Ed., pp. 47-68, Research Enterprises Publishing Segment, Richland, WA.

Clarke, R.H., Nenot, J.C., Voeltz, G., Dunster, J., Smith, H. 1995. "The environmental safety and health implications of plutonium." In: *Protection and Management of Plutonium*. American Nuclear Society Special Panel Report, G.T. Seaborg, Honorary Chair, pp. 59-74. American Nuclear Society, La Grange Park, IL.

Clemente, G.F., Mariani, A., Santaroni, G.P. 1971. "Sex differences in Cs metabolism." *Health Phys.* 21, 709-711.

Collinet, J., Rousseau, L. 1990. *Propositions de règles utilisables pour les études de sûreté radiologique relatives à l'ensemble des conditions de fonctionnement des réacteurs à eau sous pression.* Rapport Technique IPSN/SASC 90.77.

Cuddihy, R.G., Finch, G.L., Newton, G.J., Hahn, F.F., Mewhinney, J.A., Rothenberg, S.J., Powers, D.A. 1989. "Characteristics of Radioactive Particles Released from the Chernobyl Nuclear Reactor." *Environ. Sci. Technol.* 23, 89-95.

Degteva, M.O., Kozheurov, V.P., Vorobiova, M.I. 1994. "General approach to dose reconstruction in the population exposed as a results of the release of radioactive wastes into the Techa river." *The Science of the Total Environment* 142, 49-61.

Durbin, P.W. 1960. "Metabolic characteristics within a chemical family." *Health Phys.* 2, 225-238.

European Commission - Nuclear Energy Agency. 1994. *Probabilistic accident consequence assessment codes. Second international comparison.* Joint report OECD/NEA and EC, EUR 15109 EN, European Commission, Luxembourg.

Ermans, A. 1991. "Blocage de l'activité thyroïdienne par l'iode stable." In: *Irradiation par l'iode radioactif*, EDF Comité de Radioprotection No. 7, pp. 12-14, EDF, Paris.

Filipy, R.E., Pappin, J.L., Stevens, D.L., Irby, S.G. 1980a. *Effects of cigarette smoke exposure on pulmonary clearance of $^{239}\text{PuO}_2$ in rats.* Pacific Northwest Laboratory Annual report PNL-3300, Pt. 1, pp. 128-130.

Filipy, R.E., Borst, F.J., Cross, F.T., Park, J.F., Moss, O.R., Roswell, R.L., Stevens, D.L., Bair, W.J., Faust, L.G., Hoenes, G.R., Thompson, R.C., Watson, C., Feldon, D. 1980b. *A mathematical model for predicting the probability of acute mortality in a human population exposed to accidentally released airborne radionuclides.* NUREG/CR 1261 - PNL-3257.

Fujita, M., Yabe, A., Ueno, K., Oshino, M., Okuyama, N. 1963. "The behavior of strontium 85 in a normal man following a single ingestion-absorption and excretion." *Health Phys.* 9, 407-415.

Fujita, M. 1965. "Absorption of strontium-90 in Man." *Health Phys.* 11, 47-60.

Furchner, J.E., Richmond, C.R., Drake, G.A. 1971. "Comparative metabolism of radionuclide in mammals, VII." Retention of 106-Ru in the mouse, rat, monkey and dog. *Health Phys.* 21, 355-365.

Gerber, G.B., Métivier, H., Smith, H. 1987. *Age-related Factors in Radionuclide Metabolism and Dosimetry.* Marinus Nijhoff Publishers for the Commission of the European Communities, Dordrecht.

Gerber, G.B., Métivier, H., Stather, J. 1989. "Biological Assessment of Occupational Exposure to Actinides," *Radiat. Prot. Dosim.* 26, N°1-4.

Guilmette, R.A., Griffith, W.C., Hickman, A.W. 1994. "Intake Assessment for Workers who have Inhaled ^{238}Pu Aerosols." *Radiat. Prot. Dosim.* 53, 127-131.

Henrichs, K., Paretzke, H.G., Voigt, G., Berg, D. 1989. "Measurements of cesium absorption and retention in man." *Health Phys.* 57, 571-578.

Humphreys, E.R., Loutit, J.F., Stones, V.A. 1987. "The induction by ^{239}Pu of myeloid leukemia and osteosarcoma in female CBA mice." *Int. J. Radiat. Biol.* 51, 331-339.

Hunt, G.J., Leonard, D.R.P., Lovett, M.B. 1986. "Transfer of environmental plutonium and americium across human gut." *Sci. Total Environ.* 53, 89-109.

Hunt, G.J., Leonard, D.R.P., Lovett, M.B. 1990. "Transfer of environmental plutonium and americium across the human gut: A second study." *Sci. Total Environ.* 90, 273-282.

ICRP. 1979. *Limits for Intakes of Radionuclides by Workers*, ICRP Publication 30, Pt. 1, Pergamon Press, Oxford.

ICRP. 1980. *Biological Effects of Inhaled Radionuclides*, ICRP Publication 31, Pergamon Press, Oxford.

ICRP. 1986. *The Metabolism of Plutonium and Related Elements*, ICRP Publication 48, Pergamon Press, Oxford.

ICRP. 1990. *Age-Dependent Doses to Members of the Public from Intakes of Radionuclides: Part 1.* ICRP Publication 56, Pergamon Press, Oxford.

ICRP. 1991. *1990 Recommendations of the International Commission on Radiological Protection*, ICRP Publication 60. Pergamon Press, Oxford.

ICRP. 1994a. *Human Respiratory Tract Model for Radiological Protection*, ICRP Publication 66, Elsevier Science Ltd., Oxford.

ICRP. 1994b. *Age-Dependent Doses to Members of the Public from Intakes of Radionuclides: Part 2 Ingestion Dose Coefficients*, ICRP Publication 67, Elsevier Science Ltd., Oxford.

ICRP. 1995. *Age-Dependent Doses to Members of the Public from Intakes of Radionuclides: Part 3 Ingestion Dose Coefficients*, ICRP Publication 69, Elsevier Science Ltd., Oxford.

ICRP. 1996. *Age-Dependent Doses to Members of the Public from Intakes of Radionuclides: Part 4 Inhalation Dose Coefficients*, ICRP Publication 71, Elsevier Science Ltd., Oxford.

James, A.C., Stahlofen, W., Rudolf, G., Köbrich, R., Briant, J.K., Egan, M.J., Nixon, W., Birchall, A. 1994. *Annex D. Deposition of Inhaled Particles*. In *Human Respiratory Tract Model for Radiological Protection*, ICRP Publication 66, Elsevier Science Ltd., Oxford, pp. 231-299.

Johnson, J.R., Dunford, D.W., Kramer, G.H. 1983. "Summary of a strontium-89 contamination case." *Radiat. Prot. Dosim.* 5, 247-249.

Kanapilly, G.A., Stanley, J.A., Newton, G.J., Wong, B.A., DeNee, P.B. 1980. *Characterization of an aerosol from the Three Mile Island reactor auxiliary building*. Inhalation Toxicology Research Institute Annual report 1979-1980, LMF-84, pp. 5-9. Springfield VA.

Kärjä, J., Nuutinen, J., Karjalainen, P. 1982. Radioisotopic method for measurement of nasal mucociliary activity. *Azch. Otolaryngol.* 108, 99-101.

Kathren, R.L., McInroy, J.F., Reichert, M.M., Swint, M.J. 1988. "Partitioning of Pu-238, Pu-239 and Am-241 in skeleton and liver of US. Transuranium Registry autopsy cases." *Health Phys.* 54, 181-188.

Kathren, R.L., McInroy, J.F. 1992. "Implications of postmortem human tissue analysis on biokinetic models or actinides." *J. Radioanal. Nucl. Chem.* 156, 413-424.

Kathren, R.L. 1994. "Toward improved biokinetic models for actinides: The United States transuranium and Uranium registries, a twenty-five year report." *Radiat. Prot. Dosim.* 53, 219-227.

Kossenko, M.M., Degteva, M.O. 1994. "Cancer mortality and radiation risk evaluation for the Techa river population." *The Science of Total Environment* 142, 73-89.

Kreling, W.G., André, S., Collier, C.G., Ferron, G.A., Métivier, H., Schumann, G. 1989. "Interspecies Comparison of Lung Clearance after Inhalation of Monodisperse, Solide Cobalt Oxide Aerosol Particles." *J. Aerosol Sci.* 20, 1317-1320.

Leggett, R.W., Eckerman, K.F. 1984. "A model for the age-dependent skeletal retention of plutonium." In: *Radiation-Risk-Protection* (A. Kaul, R. Neider, J. Pensko, F-E. Stieve, H. Brunner Eds.) Vol. 1, pp. 454-457, Fachverband für Strahlenschutz e. V. Berlin.

Leggett, R.W. 1985. "A model of the retention, translocation and excretion of synthetic plutonium." *Health Phys.* 49, 1115-1137.

Leggett, R.W. 1986. Predicting the retention of cesium in individuals. *Health Phys.* 50, 747-759.

Leggett, R.W. 1992. "A generic age specific biokinetic model for calcium-like elements." *Radiat. Prot. Dosim.* 41, 183-198.

LeRoy, G.V., Rust, J.H., Hasterlik, R.J. 1966. "The consequences of ingestion by man of real and simulated fallout." *Health Phys.* 12, 449-473.

Léveque, J.P., Lhiaubet, G., Boulaud, D. 1991. *Program experimental HEVA*, rapport final DTP/SECC 69/91-DPEI/SEAC 91/08.

Lewis, B.J., Andre, B., Morel, B., Dehaut, P., Maro, D., Purdy, P.L., Cox, D.S., Iglesias, F.C., Osborne, M.F., Lorenz, R.A. 1995. "Modeling the release behavior of cesium during severe fuel degradation." *J. Nucl. Mater.* 227, 83-109.

Likhtarev, I.A., Dobroskok, L.A., Ilyin, A., Krasnoschekova, G.P., Likhtareva, T.M., Sminov, B.I., Sobolev, E.P., Shamov, V.P., Shapiro, E.L. 1975. "A study of certain characteristics of strontium metabolism in a homogeneous group of human subjects." *Health Phys.* 28, 49-60.

Livolant, M., Schwarz, M., von der Hardt, P. 1995. "The Phebus FP program." In: *Symposium on EU Research on Severe Accidents* (FISA 95) Luxembourg, Nov. 1995, pp. 27-50.

Lloyd, R.D., Miller, C.C., Taylor, G.N., Bruenger, F.W., Jee, W.S.S., Angus, W. 1994. "Relative effectiveness of ^{239}Pu and some other internal emitters for bone cancer induction in beagles." *Health Phys.* 67, 346-353.

McInroy, J.F., Kathren, R.L., Swint, M.J. 1989. "Distribution of plutonium and americium in whole bodies donated to the United States Transuranium Registry." *Radiat. Prot. Dosim.* 26, 151-158.

Masse, R., Métivier, H., Guillaumont, R. 1973. "Fixation osseuse des terres rares et des éléments transuraniens." In: *Physico-chimie et cristallographie des apatites d'intérêt*

biologique Colloques internationaux du CNRS, 30, 441-450.

Métivier, H., Guillaumont, R. 1972a. "Hydrolysis of tetravalent plutonium." *Radiochem. Radioanal. Lett.* 10, 27-35.

Métivier, H., Guillaumont, R. 1972b. "Citric complexes of tetravalent plutonium." *Radiochem. Radioanal. Lett.* 10, 239-250.

Métivier, H., Masse, R., Nolibé, D., Lafuma, J. 1977. "Effect of Time on the Determination of the Clearance Rates of Insoluble Plutonium 239 Oxide." *Health Phys.* 32, 447-449.

Métivier, H., Masse, R., Rateau, G., Lafuma, J. 1980. "Experimental study of respiratory contamination by a mixed oxide aerosol formed from the combustion of a plutonium magnesium alloy." *Health Phys.* 38, 769-776.

Métivier, H. 1984a. "Physico-chimie du Plutonium." *Radioprotection* 19, 115-128.

Métivier, H., Wahrendorf, J., Masse, R. 1984b. "Multiplicative effect of inhaled plutonium oxide and benzo (a) pyrene on lung carcinogenesis in rats." *Br. J. Cancer*, 50, 215-221.

Métivier, H., Warhendorf, J., Nolibé, D., André, S., Masse, R. 1988. "Inhalation carcinogenesis of fine particles of plutonium oxide formed from the combustion of a plutonium-magnesium alloy. Comparison to pure micrometric PuO₂." *Ann. Occup. Hyg.* 32, 1141-1148.

Métivier, H., Masse, R., Rateau, G., Nolibé, D., Lafuma, J. 1989. "New data on the toxicity and translocation of inhaled ²³⁹PuO₂ in baboons." *Radiat. Prot. Dosim.* 26, 167-172.

Mewhinney, J.A., Muggenburg, B.A., McClellan, R.O., Miglio, J.J. 1976. "The Effect of Varying Physical and Chemical Characteristics of Inhaled Plutonium Aerosols on Metabolism and Excretion." In: *Diagnosis and Treatment of Incorporated Radionuclides*. IAEA-Sr-6/29, pp. 87-97, IAEA, Vienna.

Miltenberger, R.P., Lessard, E.T., Grenhouse, N.A. 1981. "60-Cobalt and 137-Cesium long term biological removal rate constants for the Marshallese population." *Health Phys.* 40, 615-623.

Mussalo-Rauhamaa, H., Jaakola, T., Miettinen, J.K., Laiho, K. 1984. "Plutonium in Finnish Lapps- An estimate of the gastrointestinal absorption of plutonium by man based on a comparison of the plutonium content of Lapps and Southern Finns." *Health Phys.* 46, 549-559.

NEA/OECD. 1988. *Committee on Radiation Protection and Public health. Report of an Expert Group on Gut transfers factors.* NEA/OECD Report, Paris.

NEA/OECD. 1996. *Chernobyl, ten years on. Radiological and health impact.* NEA/OECD Report, Paris.

Nolibé, D., Masse, R., Lafuma, J. 1981. "The effect of neonatal thymectomy on lung cancers induced in rats by plutonium dioxide." *Radiat. Res.* 87, 90-99.

Nolibé, D., Berel, E., Masse, R., Lafuma, J. 1983. "An argument for the concepts of the dose to the lung: radiosensitivity of intracapillary natural killer cells." In: *Eight Symposium on Microdosimetry*, J. Booz, H.G. Ebert, Eds., EUR 8395 EN pp. 767-775.

Park, J.F., Bair, W.J., Busch, R.H. 1972. "Progress in beagle dogs studies with transuranium elements at Battelle Northwest." *Health Phys.* 22, 803-810.

Park, J.F., Lund, J.E., Ragan, H.A., Hackett, P.L., Frazier, M.E. 1976. "Bone tumors induced inhalation of ²³⁸PuO₂ in dogs." *Cancer Research* 54, 17-35.

Park, J.F., Buschbom, R.L., Dagle, G.E., Gideon, K.M., Gilbert, E.S., Powers, G.J., Ragan, H.A., Romsos, C.O., Watson, C.R., Weller, R.E., Wierman, E.L. 1991. "Inhaled Plutonium Oxide in Dogs." In: *Pacific Northwest Laboratory Annual Report for 1990, Part I*, PNL-7600, pp. 13-23. Battelle, Richland, WA.

Passàli, D. and Ciampoli, M.B. 1985. Normal values of mucociliary transport time in young subjects. *Int. J. Pediatr. Otorhinolaryngol.* 9, 151-156.

Popplewell, D.S., Ham, G.J., McCarthy, W., Lands, C. 1994. "Transfer of plutonium across the human gut and its urinary excretion." *Radiat. Prot. Dosim.* 53, 241-244.

Powers, D.A. 1996. Solubilities and Chemical Forms of Radionuclides, Sandia communication. This elicitation documents.

Priest, N.D., Dickens, C., Briden, P.E. 1990. "A comparison of the predictions of the ICRP 56 dosimetric model for plutonium with those calculated using other age-related models and using the ICRP 48 model." *J. Radiol. Prot.* 10, 291-297.

Priest, N.D., Haines, J.W., Humphreys, J.A.M., Métivier, 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.*, Articles, 156, 33-53.

Raabe, O.G., Parks, N.J. 1993. "Skeletal uptake and lifetime retention of ⁹⁰Sr and ²²⁶Ra in beagles." *Radiat. Res.* 133, 204-218.

Raabe, O.G. 1994. "Cancer and injury risks from internally deposited radionuclides." In: *Actualités sur le Césium*. EFF. Paris, *Comité de Radioprotection* 8, 39-48.

Rossof, B., Cohn, S.H., Spencer, H. 1963. "Cesium-137 metabolism in man." *Radiat. Res.* 19, 643-654.

Rowland, R.E. 1994. *Radium in humans. A review of U.S. studies*. Argonne National Laboratory report ANL/ER-3, UC-408, NTIS, Springfield, VA.

Roy, M., Becquemin, M.H., Dautzenberg, B., Sors, C., Teillac, A., Gongora, G. 1984. Inhaled Particles Deposition and Clearance in Silicosis; In "International Pneumoconiosis Conference." Ulmer, W.T., Ed., ILO, Geneva, pp. 425-431.

Roy, M., Courtay, C. 1991a. "Daily activities and breathing parameters for use in respiratory tract dosimetry." *Radiat. Prot. Dosim.* 35, 179-186.

Roy, M., Becquemin, M.H., Bouchiki, A. 1991b. "Ventilations rates and lung volumes for lung modeling purposes in ethnic groups." *Radiat. Prot. Dosim.* 38, 49-55.

Rundo, J., Mason, J.I., Newton, D., Taylor, B.T. 1963. "Biological half-life of cesium in man in acute and chronic exposure." *Nature* 200, 188-189.

Sanders, C.L., McDonald, K.E., Lauhala, K.E. 1988. "Promotion of pulmonary carcinogenesis by plutonium particle aggregation following inhalation of ²³⁹PuO₂." *Radiat. Res.* 116, 393-405.

Sanders, C.L., McDonald, K.E. 1992. "Malignancy of proliferative pulmonary lesions in the Syrian hamsters following inhalation of ²³⁹PuO₂." *J. Environ. Pathol. Toxicol. Oncol.* 11(3), 151-156.

Sanders, C.L., Lauhala, K.E., McDonald, K.E. 1993. "Lifespan studies in rats exposed to ²³⁹PuO₂. III Survival and lung tumors." *Int. J. Radiat. Biol.* 64, 417-430.

Sanders, C.L., Lundgren, D.L. 1995. "Pulmonary carcinogenesis in the F344 and Wistar rat after inhalation of plutonium dioxide." *Radiat. Res.* 144, 206-214.

Schiller-Scotland, C.F., Hlawa, R., Gebhart, J., Heyder, J., Roth, C., Wönnne, R. 1992. "Total deposition of aerosol particles in the respiratory tract of children during spontaneous and controlled mouth breathing." *Ann. Occup. Hyg.* 32 (Suppl. 1) 41-49.

Schwarz, G., Dunning, D.E. 1982. "Imprecision in estimates of dose from ingested ¹³⁷Cs due to variability in human biological characteristics." *Health Phys.* 43, 631-645.

Smith, J.M., Miller, S.C., Jee, W.S.S. 1984. "The relationship of bone marrow type and microvasculature to the microdistribution and local dosimetry in the adult skeleton." *Radiat. Res.* 99, 324-335.

Snipes, M.B., Barnes, J.E., Boecker, B.B., Hahn, F.F., Hobbs, C.H., Mauderly, J.L., McClellan, R.O., Pickrell, J.A. 1972. *Toxicity of inhaled ⁹⁰Sr fused clay in Beagle dogs III*. Fission Product Inhalation Program Annual Report 197-1972, LF-45, pp. 177-188. Springfield, VA.

Snyder, W.S., Cook, M.J., Ford, M.R. 1964. "Estimates of (MPC)w for occupational exposure to Sr90, Sr89 and Sr 85." *Health Physics* 10, 171-182.

Stather, J.W., James, A.C., Brightwell, J., Rodwell, P. 1979. "The clearance of Pu and Am from the respiratory system of rodents after the inhalation of oxide aerosols of these actinides either alone or in combination with other metals." In: *Biological Implications of Radionuclides Released from Nuclear Industries*. Vol. 2, IAEA - SM - 237/5, IAEA, Vienna; 3-25.

Stather, J.W., Karaoglu, A. 1994. "Intakes of Radionuclides." *Radiat. Prot. Dosim.* 53, N°1-4.

Stradling, G.N., Stather, J.W., Sumner, S.A., Strong, J.C., Lennox, A.M., Ham, S.E. 1984. "Decorporation of inhaled Plutonium nitrate from hamsters using Zn-DTPA." *Health Phys.* 46, 919-924.

Stradling, G.N., Stather, J.W., Gray, S.A., Moody, J.C., Ellender, M., Collier, C.G. 1989. "Assessment of intake of an accident-bearing dust formed from pond storage of spent Magnox Fuel." *Radiat. Prot. Dosim.* 26, 201-206.

Suguri, S., Ohtani, S., Oshino, M., Yanagishita, K. 1963. "The behavior of strontium 85 in a normal man following a single ingestion - application of the whole body counter for the retention." *Health Physics* 9, 529-535.

Talbot, R.J., Newton, D., Warner, A.J., Walters, B., Sherlock, J.C. 1993. "Human uptake of ¹³⁷Cs in mutton." *Health Phys.* 64, 600-604.

Taylor, D.M., Bligh, P.H., Duggan, M.H. 1962. "The absorption of calcium, strontium, barium and radium from the gastrointestinal tract of the rat." *Biochem J.* 83, 25-29.

Thompson, R.C., Weeks, M.H., Hollis, L., Ballou, J.F., Oakley, W.D. 1958. "Comparative metabolism of

radioruthenium in the rat." *Am J. Roentgen* 79, 1026-1044.

Toohey, R.E. 1994. "Biokinetics of bone-seeking radionuclides." In: *Internal Radiation Dosimetry*, O.G. Raabe Ed., pp. 197-216, Medical Physics Publishing, Madison, WI.

Underwood, J. 1971. "Iodine." In: *Trace Elements in Human and Animal Nutrition*. Academic Press, New York.

Voelz, G.L., Lawrence, J.N.P. 1991. "A 41-y medical follow-up of Manhattan project plutonium workers." *Health Phys.* 61, 181-190.

Weller, R.E., Buschbom, R.L., Dagle, G.E., Ragan, H.A., Park, J.F. 1995. "Hepatic effects of inhaled plutonium dioxide in beagles." *Radiat. Res.* 144, 73-81.

Widdowson, E.M., Slater, E.J., Harrison, G.E., Stutton, A. 1960. "Absorption, excretion and retention of strontium by breast-fed and bottle-fed babies." *Lancet* 2, 941-944.

Wiggs, L.D., Johnson, E.R., Cox-De Vore, C.A., Voelz, G.L. 1994. "Mortality through 1990 among white male workers at the Los Alamos National Laboratory: considering exposures to plutonium and external radiation." *Health Phys.* 67, 577-588.

Wilkinson, G.S., Tietjen, G.L., Wiggs, L.D., Galke, W.A., Acquavella, J.F., Reyes, M., Voelz, G.L., Waxweiler, R.J. 1987. "Mortality among plutonium and other radiation workers at a plutonium weapons facility." *Am. J. Epid.* 125, 231-250.

Wronski, T.J., Smith, J.M., Jee, W.S.S. 1980. "The microdistribution and retention of injected ^{239}Pu on trabecular bone surfaces of the beagle: Implications for the induction of osteosarcoma." *Radiat. Res.* 83, 74-89.

Question 1. Average ventilation rates, L min^{-1} , assuming a normal daily (24 h) mix of activities (combined male, female average).

	5 year old children			Mature Adults		
	5%	50%	95%	5%	50%	95%
L min^{-1}	5.2	6.0	6.6	11.4	14.2	18.5

Question 2. Total initial deposition in the respiratory tract, % of total amount inhaled, normal daily (24 hr) mix of activities.

	0.1 μm AMAD			1 μm AMAD			10 μm AMAD		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Mature Adults	28	32	37	40	49	59	53	72	94
5 year old children				43	53	63			

Question 3. Initial deposition in the extrathoracic region, % of total deposition in the respiratory tract? (This question also gives the initial deposition in the lung (tracheobronchial (TB) + pulmonary (AI) regions), since lung = total - ET).

	0.1 μm AMAD			1 μm AMAD			10 μm AMAD		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Mature Adults	10	20	35	51	60	69	80	93.5	97
5 year old children				60	71	82			

Question 4. Initial deposition in the tracheobronchial (TB) region, % of the total deposition in the lung (TB + AI regions).

	0.1 μm AMAD			1 μm AMAD			10 μm AMAD		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Mature Adults	21	25	29	18	22	26	35	41	47
5 year old children				19	22	26			

Question 5. Assuming completely insoluble particles (1 μm AMAD), retention in each respiratory tract region in mature adults as a % of the total initial deposition in the respiratory tract, as a function of time after deposition.

	10 min			1 hr			1 day		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Tracheobronchial airways	6.5	8.7	10.8	6.0	7.9	9.8	3.6	4.8	5.9
Pulmonary (AI) region	15.1	21.6	27	16.3	21.7	27.0	16.6	22.1	27.7
	1 month			1 year			10 years		
Tracheobronchial airways	1.5	1.9	2.4	0.009	0.012	0.015	1×10^{-9}	1×10^{-8}	1×10^{-7}
Pulmonary (AI) region	14.5	19.4	24.2	7.0	12	17.0	1	2	4

Question 6. By what factors would you expect the median values to be different in 5 year old children (1=no difference).

	10 min	1 hr	1 day	1 month	1 year	10 years
Tracheobronchial airways	1	1	1	1	1	1
Pulmonary (AI) region	1	1	1	1	1	1

Question 7. Absorption (dissolution and transfer) to blood in adults, % of the total initial deposition (1 μm AMAD particles) in the respiratory tract.

	1 hr			1 day			1 week		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Sr	0.1	0.45	1	6.5	8.2	11.7	7.8	8.9	14
I	3	5	7	53	62	71	59	69	79
Cs	2	3.5	4.4	23.4	38.6	48.3	25.6	41.4	51.7
Pu	2×10^{-3}	4.4×10^{-3}	0.2	3×10^{-3}	5.2×10^{-2}	2.2	5.5×10^{-2}	7.2×10^{-2}	2.7
Ru	3.8×10^{-3}	5.1×10^{-3}	0.4	0.08	0.4	6.2	0.11	0.5	7.8
Ce	4.4×10^{-3}	0.44	1.2	5.0×10^{-2}	5.7	6	4.5	5.7	7
Te	2	4	5	23	47	55	10	50	60
	1 month			1 year			10 years		
Sr	10.2	12.4	16.9	19.9	22.9	280	22	24	30
I	59	69	79	59	69	79	59	69	79
Cs	27	41.3	51.6	33	43.0	53	36	47	60
Pu	0.85	0.13	3.8	0.55	0.61	9	1.5	2	9.5
Ru	0.17	0.6	10.2	0.66	1.2	20.4	2.1	2.7	22
Ce	6	8	11	12	18	23	12	29	25
Te	15	50	60	23	50	60	25	50	65

Question 8. Factors by which the median values would be different in 5 year old children (1=no difference).

5 year old children	1 hr	1 day	1 week	1 month	1 year	10 years
Sr	1	1	1	1	1	1
I	1	1	1	1	1	1
Cs	1	1	1	1	1	1
Pu	1	1	1	1	1	1
Ru	1	1	1	1	1	1
Ce	1	1	1	1	1	1
Te	1	1	1	1	1	1

Question 9. Absorption to blood as a fraction (f_1) of activity ingested.

	3 month old infants			5 year old children			Adults		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Sr	0.15	0.6	0.75	0.1	0.4	0.5	0.1	0.3	0.4
I	0.9	0.95	1	0.9	0.95	1	0.9	0.95	1
Cs	0.8	0.9	1	0.8	0.9	1	0.8	0.9	1
PuO ₂ *	5×10^{-5}	1×10^{-4}	2×10^{-4}	1×10^{-6}	1×10^{-5}	5×10^{-5}	1×10^{-6}	1×10^{-5}	5×10^{-5}
Pu biol†	1×10^{-4}	5×10^{-3}	1×10^{-2}	1×10^{-4}	5×10^{-4}	1×10^{-3}	1×10^{-4}	5×10^{-4}	1×10^{-3}

* Refractory oxide
 † "Biologically incorporated"

Question 10. Considering the total amount reaching blood (as if administered intravenously as a single injection). % retained in liver and skeleton (bone + bone marrow), as a function of time after entry into blood.

	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
Skeleton and liver, 1 day						
Sr	55	65	70	22	30	40
Pu	65	80	95	60	80	95
Ce	65	80	92	65	80	92
Te	12	20	25	12	20	25
Sb	18	34	50	24	34	44
Skeleton and liver, 1 week						
Sr	55	65	70	20	28	35
Pu	65	80	95	60	80	95
Ce	65	80	92	65	80	92
Te	19	24	29	20	24	28
Sb	10	19	29	14	19	25
Skeleton and liver, 1 month						
Sr	55	65	70	15	23	32
Pu	65	80	90	60	80	95
Ce	65	80	92	65	80	92
Te	20	26	32	21	26	30
Sb	3	6	9	4	6	8
Skeleton and liver, 1 year						
Sr	40	45	50	10	18	26
Pu	35	75	85	60	75	90
Ce	65	74	84	65	74	84
Skeleton and liver, 10 years						
Sr	1	8	15	1	5	10
Pu	15	55	65	55	72	85
Ce	33	39	45	33	39	45
Skeleton and liver, 50 years						
Sr	0.1	1	2	0.1	1	2.5
Pu	12	35	45	25	49	55
Ce	1	2.1	2.5	1	2	5

Question 11. Retention in the skeleton, % total retention in liver + skeleton (bone + marrow), as a function of time after entry into blood.

	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
Skeleton, 1 day						
Sr	90	94	99	90	94	99
Pu	65	75	85	55	62.5	70
Ce	43.5	50	56.5	26	30	34
Te	56	70	84	24	30	36
Sb	63	90	98	63	90	98
Skeleton, 1 week						
Sr	90	95	99	90	95	99
Pu	65	75	85	55	62.5	70
Ce	53.5	50	56.5	26	30	34
Te	68	85	95	12	15	18
Sb	60	90	98	63	90	98
Skeleton, 1 month						
Sr	95	98	99.5	95	98	99.5
Pu	65	75	85	55	64	72
Ce	43.5	50	56	26	30	34
Te	80	95	98	--	5	7
Sb	60	90	98	63	90	98
Skeleton, 1 year						
Sr	95	99	99.9	95	99	99.9
Pu	62	72	82	55	66	75
Ce	43.5	50	56	26	30	34
Skeleton, 10 years						
Sr	95	99	99.9	95	99	99.9
Pu	50	60	70	65	75	85
Ce	42	50	58	26	30	34
Skeleton, 50 years						
Sr	95	99	99.9	95	99	99.9
Pu	14	20	28	80	95	99
Ce	30	50	70	25	30	35.4

Question 12. Retention of plutonium on endosteal bone surfaces (considering a 10 μm depth of bone mineral) as a % of total skeletal retention, as a function of time after entry into blood.

		5 year old children			Adults		
		5%	50%	95%	5%	50%	95%
Endosteal bone surface	1 day	95	99.8	100	95	99.5	100
	1 week	92	98	99.9	92	99	99.9
	1 month	85	92	98	90	98.7	99.8
	1 year	40	50	65	80	91	95
	10 years	30	38	50	65	77	85
	50 years	30	40	52	52	67	78

Question 13. Retention of plutonium on trabecular surfaces, % total endosteal surface retention as a function of time after entry into blood.

		5 year old children			Adults		
		5%	50%	95%	5%	50%	95%
Trabecular bone surface	1 day	40	50	63	50	60	75
	1 week	40	50	63	49	60	76
	1 month	40	50	65	48	60	77
	1 year	36	46	60	42	55	72
	10 years	35	45	60	22	28	36
	50 years	36	46	60	12	17	25

Question 14. Retention of plutonium in red bone marrow as a % total skeletal retention, as a function of time after entry into blood.

		5 year old children			Adults		
		5%	50%	95%	5%	50%	95%
Red bone marrow	1 day	0.03	0.05	0.1	0.005	0.02	0.04
	1 week	0.3	0.5	1	0.08	0.15	0.3
	1 month	1.3	2.2	4.1	0.4	0.75	1.3
	1 year	6	9.5	18	1.7	3.4	5
	10 years	5.5	9	18	1.1	2.2	4.4
	50 years	5.5	9	18	0.6	1.1	2

Question 15. Considering the total amount reaching blood (as if administered intravenously as a single injection), % total retention (whole body or systemic) as a function of time after entry into blood.

	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
1 day						
Ru	74	84	92	80	84	88
Cs	93.5	97	99.5	95	97.6	99
1 week						
Ru	55	66	76	60	66	70
Cs	71	74.3	77	82	87.3	88
1 month						
Ru	31	39	47	34	39	45
Cs	21	32.4	45	54	74.7	76.5
1 year						
Ru	3	16	20	7	16	18
Cs	1×10^{-4}	1.2×10^{-2}	5	0.2	9	13.5
5 years						
Ru	0.04	5.7	13	0.04	5.7	9.4
Cs				3×10^{-12}	9×10^{-4}	1×10^{-2}

Question 16. Considering the total amount of Iodine reaching blood (as if administered intravenously as a single injection), % retained in the thyroid as a function of time after entry into blood.

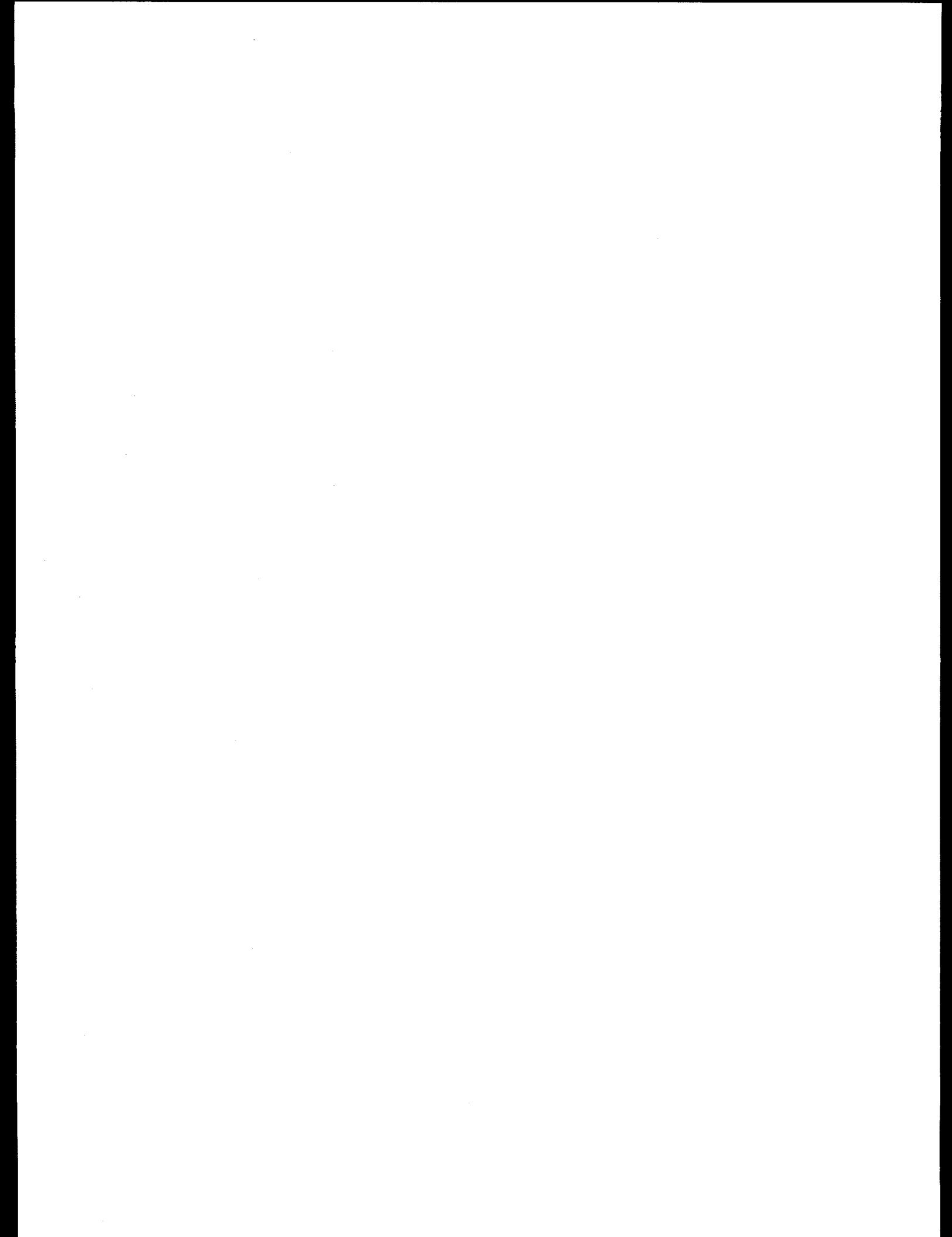
	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
1 day						
	17	27.7	44	9.9	30.5	60
1 week						
	15	25.7	41	9.4	30.5	56
1 month						
	7.5	15.1	23	7.7	24.3	46
3 months						
	1.3	3.8	5.5	4.6	14.2	27

Question 17. Intake dose coefficients, absorbed dose to specified organs or tissue per unit activity inhaled or ingested (committed equivalent doses), Gy Bq⁻¹.

5 year old children			Adult			
	5%	50%	95%	5%	50%	95%
⁹⁰Sr, inhalation, 1 μm AMAD						
lung	2.1×10^{-7}	4.2×10^{-7}	8.4×10^{-7}	6.3×10^{-8}	1.9×10^{-7}	5.7×10^{-7}
bone marrow	3.9×10^{-8}	7.7×10^{-8}	1.5×10^{-7}	2.3×10^{-8}	6.9×10^{-8}	2.1×10^{-7}
bone surface	9×10^{-8}	1.8×10^{-7}	3.6×10^{-7}	5×10^{-8}	1.5×10^{-7}	4.5×10^{-7}
⁹⁰Sr, ingestion						
colon	3.6×10^{-8}	7.2×10^{-8}	1.4×10^{-7}	1.8×10^{-8}	5.2×10^{-8}	9.4×10^{-8}
bone marrow	1.9×10^{-8}	3.7×10^{-8}	7.4×10^{-8}	3.2×10^{-8}	5.9×10^{-8}	1.2×10^{-7}
bone surface	2.5×10^{-8}	4.9×10^{-8}	9.8×10^{-8}	7×10^{-8}	1.3×10^{-7}	3×10^{-7}
¹³¹I, inhalation, 1 μm AMAD + vapor (decide proportions)						
thyroid	7×10^{-7}	9×10^{-7}	1.2×10^{-6}	1.2×10^{-7}	1.8×10^{-7}	2.4×10^{-7}
¹³¹I, ingestion						
thyroid	1.9×10^{-6}	2.1×10^{-6}	2.3×10^{-6}	3.9×10^{-7}	4.3×10^{-7}	4.7×10^{-7}
¹³²Te, inhalation, 1 μm AMAD + vapor (decide proportions)						
lung	4×10^{-10}	6×10^{-10}	2×10^{-9}	1×10^{-10}	2×10^{-10}	2×10^{-9}
thyroid	3×10^{-8}	1.4×10^{-7}	2.8×10^{-7}	4.3×10^{-9}	2.5×10^{-8}	3.5×10^{-10}
colon	4×10^{-9}	7×10^{-9}	1.3×10^{-8}	1×10^{-9}	1.7×10^{-9}	3.3×10^{-9}
¹³⁷Cs, inhalation, 1 μm AMAD						
lung	3×10^{-9}	2.6×10^{-8}	1×10^{-7}	4.3×10^{-9}	1.5×10^{-8}	6.3×10^{-8}
colon	5×10^{-9}	6.2×10^{-9}	1×10^{-8}	3×10^{-9}	4.9×10^{-9}	6×10^{-9}
stomach	2.4×10^{-9}	3×10^{-9}	3.5×10^{-9}	2.5×10^{-9}	4.2×10^{-9}	5×10^{-9}
bone marrow	2×10^{-9}	2.8×10^{-9}	3.3×10^{-9}	2.5×10^{-9}	4.2×10^{-9}	5×10^{-9}
¹³⁷Cs, ingestion						
lung	0.9×10^{-9}	1.2×10^{-9}	3×10^{-9}	0.9×10^{-8}	1.3×10^{-8}	3×10^{-8}
colon	0.9×10^{-8}	1.4×10^{-8}	3×10^{-8}	0.9×10^{-8}	1.3×10^{-8}	3×10^{-8}
stomach	1×10^{-9}	1.8×10^{-9}	3.2×10^{-9}	0.9×10^{-8}	1.4×10^{-8}	3×10^{-8}
bone marrow	0.9×10^{-9}	1.5×10^{-9}	3×10^{-9}	0.9×10^{-8}	1.3×10^{-8}	3×10^{-8}
¹⁴⁴Ce, inhalation, 1 μm AMAD						
bone surface	1.8×10^{-7}	3.9×10^{-7}	8.5×10^{-7}	1×10^{-7}	1.9×10^{-7}	4.2×10^{-7}
lung	1.4×10^{-8}	2.1×10^{-7}	4×10^{-7}	5×10^{-9}	3×10^{-8}	5×10^{-8}
bone marrow	1.2×10^{-8}	1.6×10^{-7}	3.2×10^{-7}	3×10^{-9}	1.8×10^{-8}	3×10^{-8}
liver	1.8×10^{-8}	2.2×10^{-7}	5×10^{-7}	1.2×10^{-8}	1.7×10^{-7}	3×10^{-7}
²³⁹Pu, inhalation, 1 μm AMAD						
bone surface	2.5×10^{-6}	7.5×10^{-6}	3.5×10^{-5}	5×10^{-6}	1×10^{-5}	4×10^{-5}
bone marrow	1.5×10^{-7}	5×10^{-7}	2.5×10^{-6}	2.5×10^{-7}	5×10^{-7}	1.5×10^{-6}
liver	1×10^{-6}	2.5×10^{-6}	1×10^{-5}	1.5×10^{-6}	2×10^{-6}	8×10^{-6}
lung	5×10^{-6}	8.5×10^{-6}	1.5×10^{-5}	3×10^{-6}	4.5×10^{-6}	1×10^{-5}
²³⁹Pu, ingestion						
bone surface	5×10^{-8}	3.5×10^{-7}	5×10^{-6}	5×10^{-8}	4.5×10^{-7}	9×10^{-6}
bone marrow	5×10^{-8}	3×10^{-8}	5×10^{-7}	7.5×10^{-9}	3.5×10^{-8}	7×10^{-7}
liver	1.5×10^{-8}	1.3×10^{-7}	2×10^{-6}	1.5×10^{-8}	7.5×10^{-8}	1.5×10^{-6}
colon	5×10^{-9}	1×10^{-8}	1.5×10^{-8}	2.2×10^{-9}	2.5×10^{-9}	3×10^{-9}

(xi) Joint dosimetry/late effects question: The number of radiation-induced cancer deaths up to 40 years following exposure in a population of a hundred million persons (5×10^7 male, 5×10^7 female) each of whom inhales 10 K Bq of the radionuclides specified.

Nuclide	Physical Form	Chemical Form	Cancer Type	Number of Cancers		
				5%	50%	95%
Pu-239	1 μm AMAD	Oxide	Lung	1	5	1000
			Bone	1	4	153
			Liver	1	3	100
			Leukemia	1	2	70
			All cancers	10	100	1300
Sr-90	1 μm AMAD	Oxide	Lung	10	400	4000
			Bone	1	2	20
			Leukemia	1	10	50
			All cancers	100	550	6000



EXPERT D

Seed Variables

Study 1 – Prediction of urinary excretion of plutonium in human subjects

Justification

Three published data sets were identified upon which predictions of excretion and uncertainties could be based. The early human ^{239}Pu intravenous injection study of Langham et al. (1980) yielded data for four patients who were considered by Durbin (1972) to have normal renal and hematological function. Popplewell et al. (1994) injected three normal volunteers with ^{244}Pu and Talbot et al. (1993) injected two male volunteers with ^{237}Pu . The data from these three studies suggest that the urinary excretion over days 0-20 post injection did not differ significantly between the studies and that the median daily urinary excretion at day 20 post injection was estimated for 8 subjects to be 0.021% with 5% [A] and 95% [B] confidence values of 0.017 and 0.028, respectively. This indicates that the ratio of B/A was ~3, suggesting that the 95% confidence limits lie at a factor of ~1.5 on either side of the median (50%) value. This type of uncertainty expression, which has been called the “factor of ‘x’ approach” (R.W. Leggett — *Private communication* 1995), has been widely used in the uncertainty considerations in this document.

Popplewell et al. (1994) state that from 30 days after injection the urinary excretion data fit closest to the Jones function (Jones, 1985); therefore this function has been used to calculate the 24 hour urinary excretion at the various times, setting the median value at 20 days to 0.021%. The Jones function represents a fairly robust model; changes of $\pm 20\%$ in the rate constants resulting in changes of the calculated values by <10%; thus the inter-subject variation appears to be predominant and the overall uncertainty could well be represented by multiplying or dividing the median values by a scaling factor of 1.5 up to 100 days and 3 thereafter. The predicted values are listed in Table 1.

Table 1. Urinary excretion of plutonium in humans
[%/day]

24-hour urinary excretion as % total administered after:	5%	50%	95%
45 days	0.0043	0.013	0.020
68 days	0.0033	0.011	0.016
75 days	0.0033	0.010	0.015
325 days	0.0015	0.0047	0.014
378 days	0.0014	0.0041	0.012
382 days	0.0013	0.0040	0.012
1155 days	0.0004	0.0013	0.040

Study 2 – Lung retention and urinary excretion of inhaled $1.2\text{ }\mu\text{m}$ AMAD particles of terbium oxide in humans.

Justification

Hodgson et al. (1994) have published a preliminary report on the clearance of intratracheally instilled particles of terbium oxide [Tb_4O_7] of $1.2\text{ }\mu\text{m}$ AMAD in rats. These data suggest that the particles fall into the type M clearance category of the new ICRP Respiratory Tract Model (ICRP66, 1994a), but with about 20% clearing rapidly to the gastrointestinal tract. The fractional absorption from the gastrointestinal tract (f_i) is assumed to be 1E-4. As the studies of Hodgson et al. form part of a European study of the clearance of terbium oxide particles in various species, it has been assumed that the particles used in the human study under discussion have the same properties.

Hodgson et al. (1994) calculated transfer rates for absorption from rat lung to blood of 9.3×10^{-3} (± 2.1 SD) for the period 0 - 7 days; 8.5×10^{-3} (± 7.4 SD) for 0 - 28 days and 7.9×10^{-3} (± 7.1 SD) for 7 - 84 days. These data indicate considerable uncertainty in the dissolution rates, and it is unusual for them not to decrease markedly after about one month. For calculation of lung retention the following transfer rates have been assumed: 9×10^{-3} for the period 0-30 days and 4×10^{-3} from 30 to 180 days; 10% of the inhaled activity has been assumed to clear from the lung within 1 day and the rest has been assumed to clear with the

ICRP66 (1994a) default values, i.e., 30% of the total inhaled activity clearing with a half-time of 30 days and 60% with a half-time of 700 days.

Studies of the clearance of Co_3O_4 particles of $0.8 + 1.7 \mu\text{m}$ AMAD in 4 human subjects indicated coefficients of variation ranging from about 8% at 50 days and 25% at 300 days post inhalation (Pearman et al., 1989).

Table 2. Lung retention of inhaled $1.2 \mu\text{m}$ terbium oxide particles in humans

Lung retention after:	5%	50%	95%
2 days	60	87	99
9 days	52	78	99
16 days	46	70	99
26 days	40	60	90
90 days	20	40	60
180 days	12	25	50

Table 3. 24-hour urinary excretion of terbium after inhalation of terbium oxide (% total inhaled dose/day)

24-hour urinary excretion of terbium as % total inhaled dose/day after:	5%	50%	95%
2 days	8×10^{-3}	1.6×10^{-2}	3.2×10^{-2}
8 days	4.3×10^{-4}	8.6×10^{-4}	1.7×10^{-3}
15 days	1.7×10^{-4}	3.4×10^{-4}	7×10^{-4}
30 days	9×10^{-5}	1.9×10^{-4}	4×10^{-4}
90 days	2×10^{-5}	4×10^{-5}	8×10^{-5}
180 days	2×10^{-6}	5×10^{-6}	1×10^{-5}

For the prediction of the uncertainties following inhalation of dust from the Maralinga test site, Stradling et al. (1992) assumed that the lower bound of lung retention could be represented by assuming that the transfer rates were twice those assumed for calculation of the median and that the long-term retention half-time had decreased to 500 days; for the upper bound the dissolution rate was assumed to be only one half of the median value and the half-time was 1000 days. Applying a similar assumption to the terbium oxide situation suggests scaling factors ranging from 1.1 at 2 days

to 1.5 at 180 days. Subjective judgement suggests that these may be too low; a value of 1.5 has been used up to 30 days and 2 thereafter. Urinary excretion was assumed to be 1.5 times that observed for plutonium in humans by Langham et al. (1980); the uncertainty is assumed to be quite large and scaling factors of 2 up to 30 days, and 3 thereafter, were used to estimate the upper and lower limits. The predicted lung retention and the urinary excretion are shown in Tables 2 and 3, respectively.

Study 3 – Actinide and polonium retention in pregnant baboons

Justification

In predicting maternal organ retention, it is necessary to consider how much of the injected radioactivity is likely to have been diverted to the feto-placental unit. Some information on actinide and polonium transfer to the fetus and placenta of animals is available (Stather et al., 1987; Sikov, 1987; Levack et al., 1994), and Stather¹ indicates that at 7 days after injection into a 5-month pregnant baboon, the fetus contained about 0.3% of the injected ^{210}Po , compared with 0.4% and 4% of the simultaneously injected ^{241}Am and ^{239}Pu , respectively; placental retention was 1%, 2% and 10%, respectively. Thus in predicting organ burdens it has been assumed that 2% of the injected ^{210}Po , 3% of ^{241}Am , and 15% of the ^{239}Pu were diverted to the feto-placental unit; ^{237}Np was assumed to behave similarly to ^{241}Am .

No data on the retention of ^{210}Po in the baboon was found and it has been assumed that the retention of this nuclide will be similar to that in rats at 7 days. Some data have been reported for the retention of ^{241}Am in baboons (Cohen et al., 1974; Guilmette et al., 1980) and this has been taken into account in the assessments made here, as have tissue distribution studies of ^{238}Pu in Macaque monkeys (Durbin et al., 1985).

The predicted retention of the nuclides in liver, spleen, kidneys, and femora are listed in Table 4. Because of the large uncertainties in extrapolating from other species, a scaling factor of 1.5 has been applied for the actinides in liver, and 2 for ^{210}Po in this tissue; for all other tissues a scaling factor of 3 has been applied.

1. Stather, J.W. 1995. Private communication.

Table 4. Retention of actinides and polonium in pregnant baboons

Retention as % of total administered activities:		5%	50%	95%
in maternal liver for:	Pu-239	35	52	78
	Am-241	15	23	34
	Np-237	6	10	15
	Po-210	5	10	20
in femora for:	Pu-239	0.87	2.6	7.5
	Am-241	1.1	3.3	9.9
	Np-237	1.6	4.8	14.4
	Po-210	0.27	0.8	2.4
in spleen for:	Pu-239	0.05	0.14	0.42
	Am-241	0.05	0.14	0.42
	Np-237	0.03	0.10	0.30
	Po-210	1.6	5	15
in kidneys for:	Pu-239	0.14	0.44	1.4
	Am-241	0.18	0.54	1.6
	Np-237	0.16	0.48	1.5
	Po-210	1.6	5	15

Study 4

A. Polonium retention in rats

Justification

Published data on the retention of polonium in rats has been summarized in ICRP Publications 30 (1979) and 67 (1993) and by Stara et al. (1971); these publications suggest a half-time of ~30 days for the whole body retention. Examination of published data for the short term retention of polonium in rat tissues (Volf, 1973; Haines et al., 1994; Rencova et al., 1994) suggest that there is some quite rapid loss of polonium, principally from the liver, and also perhaps some longer term retention due to recycling of activity in the reticuloendothelial system. The data do not permit mathematically meaningful relationships for either the early or the late clearance to be calculated, thus for the purposes of these predictions the following retention function has been assumed.

$$TB(\%) = 5e^{-6.9t} + 90e^{-0.02(\pm 0.003)t} + 5e^{-0.004(\pm 0.001)t}$$

The predicted retention is listed in Table 5. To address the uncertainties a scaling factor of 1.1 has been used up to 7 days and 1.5 thereafter.

B. Absorption of polonium from crabmeat by rats

Justification

Haines et al. (1994) reported an f_I value of 0.13 (95% confidence limits 0.008-0.018) for ^{210}Po "biologically incorporated" into rat liver. Hunt and Allington (1993) studied crabmeat containing ^{210}Po biologically incorporated from the natural environment into humans and calculated an f_I value of ~0.8. The chemical speciation patterns of ^{210}Po in rat liver and in crabmeat are not understood, but they could be different, since fish is reported to contain more cystine than liver (Documenta Geigy, 1977) and Po binds strongly to cystine and other S-containing ligands. Human studies suggest that the f_I for ^{210}Po absorption from reindeer meat lies in the range 0.3-0.5, rather higher than that for ^{210}Po in rat liver (Harrison, 1995). Thus the f_I for ^{210}Po absorption from crabmeat in rats may well be lower than that observed in humans. A median value of 0.6 has been assumed for the predictions in Table 6 with the 5% and 95% values being presumed to be 0.15 and 1.5 times the median value.

Table 5. Total body retention of polonium in rats

Whole body retention of ^{210}Po as % of total administered after	5%	50%	95%
1 day	85	94	99
7 days	75	83	91
50 days	25	37	55
200 days	3	4	6

Table 7. The retention of dust from a nuclear power plant in the lungs of rats

lung retention as % of the initial lung deposit after	5%	50%	95%
1 day	5	15	45
7 days	5	14	42
28 days	4	12	36
168 days	2	6	18

Table 6. Fractional absorption of ^{210}Po from crabmeat

absorbed fraction of ^{210}Po in animals fed crabmeat	5%	50%	95%
Fraction	0.1	0.6	0.9

Study 5 – Dust from a nuclear power plant administered intratracheally to rats

Justification

This exercise, in this expert's view, requires too many assumptions concerning the material inhaled to permit any really meaningful assignment of errors. The material was assumed to have a particle diameter of 3 μm AMAD and to contain equal activities of ^{60}Co and ^{137}Cs . The estimated f_f values suggest that the material might be regarded as 80% soluble and 20% rather less soluble, or 80% ICRP Type F and 20% Type M. The initial lung deposit is assumed to be made up of the tracheobronchial and alveolar-interstitial regions and for instilled particles the distribution is assumed to be 20% TB and 80% AI (Birchall et al., 1995). Retention in rat tissues has been calculated using data for Cs from Stara et al. (1971) and for Sr excretion from Taylor (1959). For the retention in lung, because the composition and characteristics of the inhaled particles have been assigned randomly, a scaling factor of 3 has been applied at all times. A similar level of uncertainty in the retention and the transfer to blood is reflected in the scaling factors for tissue retention which were set at 1.3 for 1 and 7 days, 2 at 28 days and 3 at 168 days. The results are listed in Tables 7 and 8.

Table 8. The total retention of dust from a nuclear power plant in tissues other than lung in rats.

total retention in other tissues as % of the initial lung deposit after	5%	50%	95%
1 day	60	78	99
7 days	40	52	68
28 days	11	22	44
168 days	1	4	12

Elicitation Questions

Inhalation – Introductory Comment

In the preparation of the assessments presented in the following pages, I have relied extensively on the data and the discussions contained in ICRP Publications 56, 66, 67, 68, 69 and 71, not only because I have been heavily involved in the preparation of several of these documents, but also because I believe that they represent the best contemporary state of the art reviews of the biokinetic behavior of the radiologically most important elements, and of the anatomy and physiology of the human respiratory tract.

Question 1 – Average ventilation rates:

For this exercise an adult population of 50% females + 50% males was selected having the patterns of activities shown in Table 9.

Using this activity pattern, the median ventilation rate averaged over the whole day was calculated. On the basis of the information summarized in ICRP66 (1994a), ventilation rates do not vary widely from individual to individual or from ethnic population to ethnic population. However, changes in the activity pattern, especially in relation to the amount of heavy exercise can cause changes in average ventilation rate of ~40+%

Table 9.

	Females		Males	
	Time hours	VR* $\text{m}^3 \text{h}^{-1}$	Time hours	VR* $\text{m}^3 \text{h}^{-1}$
Sleeping	8	0.32	8	0.45
Occupational				
Sitting	4	0.39	4	0.54
Light exercise	4	1.25	4	1.50
Leisure				
Sitting	4	0.39	4	0.54
Light exercise	3	1.25	3	1.5
Heavy exercise	1	2.70	1	3.0

* Ventilation rates from ICRP66

For the purpose of this assessment a scaling factor of 1.5 has been assumed for setting the 5% and 95% levels. For the 5-year old child, a standard pattern of 12 h sleep [VR $0.24 \text{ m}^3 \text{h}^{-1}$] plus 4 h sitting [VR $0.32 \text{ m}^3 \text{h}^{-1}$] plus 8 h light exercise [VR $0.57 \text{ m}^3 \text{h}^{-1}$] has been assumed. For the child of this age variations within the daily activity pattern produce relatively small changes (~10%) in the ventilation, thus the same scaling factor of 1.5 has been adopted for setting the 5% and 95% levels.

Questions 2-4

For these questions the adult and child population has been assumed to be the same as that in Question 1. The particles have been assumed to have thermodynamic and aerodynamic properties such that the $\text{AMTD} = 0.6 \times \text{AMAD } \mu\text{m}$ so that deposition parameters could be calculated using the data presented in ICRP66 (1994a), Tables F1 to F5.

For particle deposition the uncertainties may arise from many factors, inter-individual variations in anatomy and physiology, in the efficiency of the mucociliary escalator, the physico-chemical behavior of the particles in the respiratory tract and factors, such as coughing or nose blowing. These factors are difficult to quantify on the basis of the available information; ICRP66 (1994a) suggests scaling factors of ~1.5 for the tracheobronchial and alveolar-interstitial regions; although these factors appear to be reasonable estimates, a single, and larger, scaling factor of 2 has been used here for the estimation of the upper and lower values.

Question 5

In order to estimate these parameters, the basic models of ICRP66 (1994a) for the retention of particles in the AI and TB regions in the absence of absorption have been used, namely that the following retention equations have been applied:

$$\text{AI Rai} = 0.3e^{-0.03t} + 0.6e^{-0.001t} + 0.1e^{-0.0001t}$$

$$\text{TB Rtb} = 0.43e^{-2t} + 0.5e^{-0.03t} + 0.007e^{-0.01t}$$

The uncertainties were estimated assuming scaling factors of 2 for the period up to 1 month, 3 at 1 year and 5 at 10 years.

Question 6

According to the information summarized in ICRP66 (1994a) there are no large age-related variations in particle clearance or absorption rates from the lungs, thus major differences between the adult and the 5 year old would not be expected in relation to the parameters considered, at least at the earlier times after intake. The data in the response to Question 6 provide a rather subjective assessment of the possible differences between adults and 5 year olds. The figure of 1 should be taken to mean similar, i.e., close to [$\pm 10\%$], but not necessarily identical with the adult values.

Question 7

There are a number of factors contributing to the uncertainties underlying the absorption of inhaled materials to blood. These include the absorption of material cleared from the respiratory tract to the intestine (f_1), the chemical reactivity of the inhaled material and especially the solubility of particles in the lung. Solubility may change with time due to chemical changes and/or as the particles decrease in size on dissolution. These uncertainties are

superimposed on those concerning particle deposition and retention in the respiratory tract. Measurements of particle dissolution rates have generally been obtained from animal or *in vitro* studies and the extrapolation of the data to humans adds to the overall uncertainty. The detailed assessment of the combined influence of the various uncertainties in this field requires quite complicated and iterative mathematical analysis using sophisticated computer codes. I do not have access to such codes and I have attempted to predict 50% values using simple mathematical models, and relying heavily on information from ICRP66 (1994a) and ICRP71 (1996). I have then sought to attempt a realistic assessment of the uncertainties in the form of scaling factors which indicate the uncertainties about these tentative median values.

The elements were assumed to have been inhaled in the forms shown in Table 10. The predicted values are listed in the response to Question 7.

For Sr, the absorption to blood from material cleared to the gastrointestinal tract is quite large, ~11 - 16% of the total deposition in the respiratory tract. For this assessment, the inhaled Sr has been assumed to have the characteristics of a Type M material. It is considered that the use of a scaling factor of 2 for time periods up to 1 month, and 1.5 at 1 and 10 years, probably adequately represents the overall uncertainty for absorption of Sr to blood.

Most chemical forms of I are likely to be very soluble and the uncertainties about dissolution rates are probably much smaller than for less soluble elements. An overall scaling factor of 1.2 has been applied to time intervals up to 1 month and 2 at later times.

Cs is also a fairly soluble material for which a considerable body of human data is available. An overall scaling factor of 1.2 has been applied, except that since by definition transfers to blood in excess of 100% are not possible, the maximum for the 95% limit has been set at 99-100%.

Pu has been considered to be in a form having broadly similar characteristics to the Maralinga dusts studied by Stradling et al. (1992) and the same assumptions have been made in setting the upper and lower levels. Basically the levels have been set assuming that absorption rates either increase or decrease by a factor of 2 and the longer term component of clearance increases by a factor of 1.4; in general this leads to a scaling factor of 2 for most time intervals, except for the very early time intervals. For Pu, the contribution from absorption from the material cleared to the gastrointestinal tract is negligibly small.

For Ru the ICRP66 (1994a) default Type M has been assumed. The data for Ru, especially human data, are rather scanty, scaling factors of between 2 for early times and up to 5 for later times have been applied.

Ce has been assumed to show similar characteristics to Pu, which is justified by the chemical similarities between the actinides and lanthanides. Similar scaling factors have been used for Pu and Ce.

Te has been assumed rather arbitrarily to have been inhaled as a mixture of vapor and a fairly soluble particulate. Data on the biokinetics of Te are rather sparse and are mostly derived from animal studies. Because of this a scaling factor of 1.6 has been generally applied.

Question 8 – Differences in median absorption/dissolution rates in the respiratory tract between a 5 year old child and an average adult.

The differences have to be addressed for each element. For all elements, there is a basic assumption that absorption is independent of age and this should be seen as one element of uncertainty. For the more soluble elements such as I and Cs, the likely uncertainties with respect to solubility may be small and could be covered by assuming that child and adult values will lie within $\pm 10\%$ of each other. For elements such as Sr and Te, which may be relatively soluble *in vivo*, the deposition in adults and 5 year olds might also be quite similar, lying broadly within $\pm 10\%$, at least up to 1 month, and within a factor of 2 thereafter. For the poorly soluble elements such as Pu, Ce, and Ru, and for which predictions about chemical behavior and speciation *in vivo* are difficult to make with reasonable confidence, it would appear prudent to use an uncertainty factor of 1.5 for up to 1 month and 3 thereafter.

Question 9 – Ingestion

For this exercise, Sr has been assumed to be ingested from a mixed diet, which I believe is the most likely form of ingestion after the initial cloud has passed following a reactor accident. Iodine is assumed to have been ingested as an iodide, which could well have been CsI and Cs has been assumed to have been ingested in biologically incorporated form in meat. Biologically incorporated Pu is assumed to have been in a similar form to that which occurs in shellfish or reindeer meat, which may well be a hydrated oxide or phosphate formed *in vivo* following uptake of soluble Pu. The predicted fractional absorption (f_I) values are shown in the table in the response to Question 9. In assessing uncertainties, the chemical properties of the ingested

Table 10. Assumed properties of inhaled elements

Element	Physico-chemical form	Solubility	ICRP Type
Sr	particulate mixed composition	moderate, good in gut	M
I	iodide [e.g., CsI] 50% + vapor 50%	very good	F + SR-1
Cs	particulate mixed composition	moderate, good in gut	F/M
Pu	oxide containing particles	poorly soluble	S
Ru	particulate mixed composition	poor to moderate	M/S
Ce	oxide containing particles	poorly soluble	S
Te	particulate mixed composition 50% + vapor 50%	variable	M + F/ SR-1

element and the chemical environment at the site of absorption, i.e., in the duodenum and jejunum, are important determinants of the fractional absorption. For elements such as iron, plutonium, and the lanthanides, which cannot exist as free ions at neutral pH, the interactions of the metals with water and complexing ligands in the small intestine are very important, as well as the chemical environment which changes according to the time since the last meal and with the types of food ingested. This means that there may be considerable variations between separate intakes of the same element in any single individual, as well as inter-individual variations. Thus the uncertainties on the fractional absorption will often be quite large.

Sr absorption

For adults, the 5%, 50%, and 95% values shown were derived from two small sets of human data (Carr et al., 1967; Shimmins et al., 1967). The number of subjects involved was small, five and nine, respectively, but the range of values appears to span the range which might be expected to be observed in a much larger population. For children, the assessments follow the general policy of ICRP56 (1989) but the values are based much more on subjective judgement than for the adults. A scaling factor of 1.5 has been applied to adults and 2 for children.

I absorption

The values were derived principally from the summarized data presented in ICRP56 (1989). A scaling factor of 1.2 has been applied for all ages for assessing the upper and lower percentiles

Cs absorption

The assumption made here was that the major ingestion of Cs following a reactor accident is likely to be from the diet. The adult data have been derived by analysis of two studies of Cs absorption from meat, 10 subjects eating venison (Henrichs et al., 1989) and eight persons (Talbot et al., 1993) eating sheepmeat, both contaminated following the Chernobyl accident. The same pattern of variation has been assumed to occur in children. For Cs, the measured values fall within a tight range; nevertheless a scaling factor of only 1.2 has been applied at all ages.

PuO₂

The 50% value adopted for the f_1 for PuO₂ is the ICRP default value, which was derived from a few experimental animal data. The 5% and 95% values are somewhat arbitrarily assumed to be ten times less and ten times more than the median value. This range is in my view realistic, since there is evidence, mainly from rodents, to suggest that the absorption of PuO₂ may depend on particle size, with smaller, or polydisperse, particles being absorbed to near the same extent as soluble Pu.

Biologically incorporated Pu

The selected values for adults are based principally upon the human data of Hunt et al. (1990) for the absorption of Pu from shellfish. The upper and lower bounds have been selected to be one-fifth and 10 times the median value derived from the Hunt study. This quite large variation is considered to be realistic for the situation following an accidental release into the environment; it is also consistent with the data for human subjects who ingested ²⁴⁴Pu as a citrate complex.

Questions 10 and 11

Retention of Strontium, Plutonium, Cerium and Tellurium:

The assessments for all four elements have been made using the information summarized in ICRP56 (1989) and ICRP67 (1993), with reference back to the original data so far as this was easily accessible. The assessments for each organ/tissue are listed in the responses to Questions 10 and 11.

Sr

The assessments are based on the ICRP67 (1993) Alkaline Earth model, which is based on reasonably good human data and shows relatively little variation. A scaling factor of 1.2 has been assigned for time intervals up to 1 month and 1.5 at 1 year; thereafter a factor of 2 has been used to reflect the uncertainties in the long-term retention of the alkaline earth metals (Rowland,1993).

Pu

The values were derived using the ICRP67 (1993) model. This model is based on a combination of animal and human data but the uncertainties are considered to be larger than those for the Sr model. For 5 year old children, this model predicts a virtually constant retention of Pu in the skeleton between 10 and 50 years. For the first year, a scaling factor of 1.5 was adopted with 3 for 10 years and 50 years.

Ce

ICRP56 (1989) model is not considered to be realistic because the retention times have been assumed to be equal for liver and bone and to have the same numerical value as that observed in beagles. The behavior of Ce is considered to be more like that of Am and the ICRP67 (1993) model for Am has been applied. The uncertainty has been assessed using the same scaling factors as those used for Pu.

Te

Biokinetic data on Te is sparse and the ICRP67 (1993) model probably represents the best assessment of the available data, which is derived very largely from experimental animals. The uncertainties are considered to be very large and scaling factors of 0.2 and 2 have been used to derive the 5% and 95% values respectively. The ICRP67 (1993) model has been used here but as this model gives no data for uptake in liver, the liver has been assumed to receive 2.5% of the element entering the systemic pool.

For the skeleton, the same models and uncertainties have been used, except that a 95% value in excess of 100% is considered to be impossible and the maximum upper value has been set at 99%. There is some ambiguity about what is actually required for Question 11. It has been assumed that the data required are the amounts retained in the skeleton expressed as a percent of the total remaining in liver plus skeleton at the desired time 't' after entry to blood.

Questions 12, 13, 14

These questions have been addressed on the basis of the ICRP56 (1989) and ICRP67 (1993) Biokinetic Models for Pu and using the default transfer rates constants as the median (50%) values. I do not have the computer codes for this recycling model and it has been necessary to use some simplifications to enable the computations to be made by hand. In this model considerable assumptions have been made about the magnitudes of the deposition fractions and transfer rates. Thus the uncertainties in modeling deposition and retention on bone surfaces are likely to be quite large.

The model assumes 100% of the total skeletal activity deposits on bone surfaces, but it seems unlikely that there is no uncertainty in this value; therefore for the purpose of this exercise, surface deposition has been assumed to be 96% with 5% and 95% limits of 93% and 99% respectively. For calculation of the median values the ICRP transfer constants for cortical surface→marrow; trabecular surface→marrow; cortical surface→bone volume; trabecular surface→bone volume; and liver→plasma have been assumed. Deposition in bone has been assumed to be 0.5 of the activity entering the plasma in adults, with 60% depositing on trabecular surfaces and 40% on cortical surfaces. For five year old children deposition is assumed to be 0.6 with equal deposition on trabecular and cortical surfaces.

For the estimation of the 5% values, the transfer rates are assumed to have been double the 50% rates, and for the 95%, one half those rates in both adults and children. In addition, for the 5% values the deposition distribution has been assumed to be 70% trabecular:30% cortical for adults and 60%:40% for 5 year olds. For the 95% values, the trabecular:cortical surface distribution was assumed to be 40%:60% for adults and for 5 year olds.

Question 14

This question was addressed in a similar manner to Questions 12 and 13. Only trabecular bone surfaces were assumed to deliver Pu to marrow and the transfer rates were

the ICRP56 (1989), ICRP 67 (1993) default rate for the 50% value. However, because of the very small values and the uncertainties inherent in this part of the model, the 5% and 95% values were assumed to be one fifth and 5 times the 50% value.

Question 15 – Systemic retention of ruthenium and cesium

The assessments for Cs are based on a quite large body of human data, whereas no human data are available for Ru and the calculations must be based entirely on extrapolation of data from experimental animals. Thus rather more confidence can be placed in the assessments for Cs than in those for Ru.

To derive the values tabulated for Cs, the information summarized by Leggett (1986) and contained in two later papers, Henrichs et al. (1989) and Talbot et al. (1993), have been analyzed to provide assessments of the inter-individual variations in a group of 44 adult males and 12 adult females. The data presented in the response to Question 15 are for an average population of 50% males and 50% females and are derived assuming a two compartment exponential model: This model assumes a compartment containing ~4 to 16% of the total Cs entering the blood and clearing with half-times in the range 0.5 to ~5 days; the second compartment clears much more slowly with half-times ranging from 45 to 210 days. The clearance in females is more rapid than that in males, median values for the long term clearance being 69 days and 90 days, respectively. The effect of the shorter retention time of Cs in females has little effect on the total body retention up to one month but at one year the retention in females is about one half of that in males. The 95% values listed for Cs in the response to Question 15 reflect the longer retention times in the males, while the 5% values reflect the more rapid turnover in the female.

The retention functions used to derive the 50%, 5%, and 95% values were as follows:

$$R50\% = [0.5(0.12e^{-0.57t} + 0.88e^{-0.0077t}) + 0.5(0.06e^{-1.26t} + 0.94e^{-0.010t})]$$

$$R5\% = [0.04e^{-6.93t} + 0.95e^{-0.015t}]$$

$$R95\% = [0.14e^{-1.39t} + 0.86e^{-0.0045t}]$$

Because of the scarcity of human data for Ru, the ICRP54 (1988) model has been used for both adults and 5 year old children. In view of the absence of adequate human or even animal data, a subjective assessment has been made that the

5% and 95% values could be assessed by dividing or multiplying the 50% value by a scaling factor; the scaling factors used were 1.2 at 1 and 7 days. Because of the relatively rapid rate of loss, the uncertainties are likely to increase with time; therefore a scaling factor of 2 was used at 30 days and 4.0 at 1 year and 8 at 5 years.

For 5 year old children, the direct data on Cs retention are few. For the purposes of calculation, the ICRP56 (1989) default half-times and compartment sizes have been used. For the 5% and 95% values, the half times were multiplied or divided by 1.5, respectively, and the relative sizes of the two compartments were changed. The retention functions used were as follows:

$$R50\% = 0.45e^{-0.076t} + 0.55e^{-0.023t}$$

$$R5\% = 0.55e^{-0.114t} + 0.45e^{-0.035t}$$

$$R95\% = 0.35e^{-0.051t} + 0.65e^{-0.0153t}$$

Question 16 – Retention of I in the thyroid

This assessment has been based entirely on the human data summarized in ICRP56 (1989), and the references cited therein. The overall retention of iodine in the thyroid gland is controlled by the rate of removal of organic iodide from the gland into the blood and by the recycling of I released from breakdown of the organic iodides in the tissues. Strictly this retention is described by a two component exponential expression, but because of the short physical half-time of ^{131}I , the most suitable radionuclide for *in vivo* studies in humans, it has not been possible to resolve the components of this exponential expression and the available data are reported in terms of an “apparent” half-time. A median “apparent” half-time of 72 days appears to be consistent with the data for adults, but half-times ranging from 21 to 372 days have been reported. For this assessment, the median half-time has been set at 72 days for adults and the ICRP56 (1989) value of 30 days has been adopted for 5 year old children. The second major factor in estimating thyroid retention is the initial uptake from the blood into the gland. Again the reported range of values is large, from 8 to 46% in adults and from 6 to 97% in children. For this assessment, the median value has been assumed to be 30% for adults and 40% for 5 year olds. For the assessment of the 5% and 95% values, half-times of 36 and 144 days and uptakes of 60% and 15% respectively were assumed. For 5 year old children, the corresponding half-times and uptakes were 15 and 60 days and 20% and 80% respectively. The values are listed in the response to Question 16.

Question 17 – Dose Coefficients

The 50% dose coefficients are taken from the appropriate ICRP publications as these are believed to be the best available values.

Uncertainties on dose calculations

The uncertainties on the dose coefficients calculated for any given radionuclide enfold all the uncertainties associated with the biokinetic and dosimetric models assumed, that is uncertainties in the uptake and excretion parameters at different times after exposure, and uncertainties in physical parameters such as the conversion of cumulative organ activities into absorbed doses. Because in some cases the available data does not allow accurate prediction of the short term uptake and excretion kinetics, the uncertainty on dose calculations for a short lived radionuclide may be larger than that for a long-lived radionuclide of the same element whose retention in the body is prolonged into a time span for which more accurate information about the retention of the element is available. Thus uncertainties may be radionuclide-specific rather than element-specific. The most attractive model for assessing the uncertainties on overall dose is the “factor-of-x” which has been used throughout this document.

For ⁹⁰Sr, the biokinetic models are based on relatively large amounts of information derived directly from studies in humans, thus reasonably high confidence can be placed on the models and the doses derived using them. Thus a scaling factor of 1.5 has been applied to estimate the 5% and 95% limits for all tissues following either ingestion or inhalation. Similarly, the biokinetic models for ¹³⁷Cs are based predominantly on human data and the same scaling factor has been assumed to apply.

For ¹³¹I, although there are also good human data on which to base the models, the variability in the biokinetics imposed by varying levels of iodine in the diet and variations of thyroid function, and hence uptake of ¹³¹I into the thyroid gland, may be quite large. Thus a scaling factor of 2 has been used in setting the upper and lower levels.

For ²³⁹Pu, the biokinetic models are based on a mixture of a few human data with a much larger body of information from experimental studies in animals. This fact, plus the uncertainties about the chemical behavior of Pu in the human body and in the environment make it probable that the uncertainties surrounding the doses calculated using the best available biokinetic models are quite large and a factor of 3 appears to be appropriate for predicting the upper and

lower limits. The same criteria have also been applied to ¹⁴⁴Ce.

For ¹³²Te, the models are based largely on few animal data and the uncertainties are likely to be large. A factor of 5 has been used to set the limits.

Question xi – Joint dosimetry question

After consultation with my partner we have decided that, in view of our associations with a national and an international advisory body, it would not be appropriate for us to prepare any written statement about the limits of the uncertainties of the risks of cancer induction.

References:

Birchall, A., Bailey, M.R. and Jarvis, N.S. 1995. “Application of the new ICRP respiratory tract model to inhaled plutonium nitrate using experimental biokinetic data.” In: *Proceedings of the International Conference on Radiation Dose Management in the Nuclear Industry, Windermere, UK 9-11 October, 1995* pp. 216-223, British Nuclear Engineering Society.

Carr, T.E.F. 1967. “An Attempt to Quantitate the Short-term Movement of Strontium in the Human Adult.” In: *Strontium Metabolism* (Eds. J.M.A. Lenihan, J.F. Loutit, J.J. Martin) Academic Press, London, pp. 139-148.

Cohen, N., Guilmette, R.A. and Wrenn, M.E. 1974. “Chelation of ²⁴¹Am from the Liver and Skeleton of the Adult Baboon.” *Radiation Research* 58, 439-447.

Durbin, P.W. 1972. “Plutonium in man: A new look at the old data.” In: *Radiobiology of Plutonium* (B.J. Stover and W.S.S. Jee, Editors) J.W. Press, Salt Lake City, pp. 469-530.

Durbin, P.W., Jeung, N. and Schmidt, C.T. 1985. ²³⁸Pu(IV) in Monkeys, NUREG/CR-4355:LBL-20022 Vol. 1, US Nuclear Regulatory Commission, Washington, DC.

Guilmette, R.A., Cohen, N. and Wrenn, M.E. 1980. “Distribution and Retention of ²⁴¹Am in the Baboon.” *Radiation Research* 81, 100-119.

Haines, J.W., Naylor, G.P.L., Pottinger, H. and Harrison, J.D. 1994. “Gastrointestinal absorption and retention of polonium in adult and newborn rats and guinea pigs.” *Int. J. Radiat. Biol.* 64(1) 127-132.

Harrison, J.D. 1995. “Ingested Radionuclides.” In: *Radiation and Gut* (Eds. C.S. Pott and J.H. Hendry) Elsevier Science, Amsterdam, pp. 253-289.

Henrichs, K., Paretzke, H.G., Voight, G. and Berg, D. 1989. "Measurements of Cs absorption and retention in man." *Health Phys.* 57, 571-578.

Hodgson, A., Stradling, G.N., Foster, P.P., Kreyling, W.G. and Pearce, M.J. 1994. "The Biokinetics of $^{160}\text{Terbium Oxide}$: Studies to Determine its Suitability for an Interspecies Comparison of Lung Clearance Kinetics." In: *Portsmouth 1994: Proceedings of the 17th IRPA Regional Congress* (Editors W. Nimmo-Scott and D.J. Golding) Nuclear Technology Publishing, Ashford UK, pp. 193-196.

Hunt, G.J., Leonard, D.R.P. and Lovett, M.B. 1990. "Transfer of environmental plutonium and americium across the human gut. A second study." *Sci. Total Environ.* 90, 273-282.

Hunt, G.J. and Allington, D.J. 1993. "Absorption of environmental ^{210}Po by the human gut." *J. Radiol.Prot.* 13, 119-126.

ICRP. 1979. ICRP Publication 30. *Limits for Intakes of Radionuclides by Workers*. Annals of the ICRP, 2(3/4).

ICRP. 1988. ICRP Publication 54. *Individual Monitoring for Intakes of Radionuclides by Workers: Design and Interpretation*. Annals of the ICRP, 19(1-3).

ICRP. 1989. ICRP Publication 56. *Age-Dependent Doses to Members of the Public from Intakes of Radionuclides: Part 1*. Annals of the ICRP, 20(2).

ICRP. 1993. "ICRP Publication 67. Age-Dependent Doses to Members of the Public from Intakes of Radionuclides: Part 2." Annals of the ICRP, 23(3/4).

ICRP. 1994a. ICRP Publication 66. *Human Respiratory Tract Model for Radiological Protection*. Annals of the ICRP, 24(1-3).

ICRP. 1994b. ICRP Publication 68. *Dose Coefficients for Intakes of Radionuclides by Workers*. Annals of the ICRP, 24(4).

ICRP. 1995. ICRP Publication 69. *Age-Dependent Doses to Members of the Public from Intakes of Radionuclides: Part 3*. Annals of the ICRP, 25(1).

ICRP. 1996. ICRP Publication 71. *Age-Dependent Doses to Members of the Public from Intakes of Radionuclides: Part 4*. Annals of the ICRP in the press.

Jones, S.R. 1985. "Derivation and validation of a urinary excretion function for plutonium applicable over tens of years post uptake." *Radiation Protection Dosimetry* 11, 19-27.

Langham, W.H., Bassett, S.H., Harris, P.S. and Carter, R.E. 1980. "Distribution and excretion of plutonium administered intravenously to man." *Health Physics* 38, 1031-1060.

Leggett, R.W. 1986. "Predicting the retention of Cs in individuals." *Health Phys.* 50, 747-759.

Levack, V.M., Pottinger, H., Ham, G.J., Harrison, J.D. and F. Paquet. 1994. "The Fetal Transfer of Ruthenium, Cerium, Plutonium and Americium." In: *Portsmouth 1994: Proceedings of the 17th IRPA Regional Congress* (Editors W. Nimmo-Scott and D.J. Golding) Nuclear Technology Publishing, Ashford UK, pp. 161-164.

Popplewell, D.S., Ham, G.J., McCarthy, W. and Lands, C. 1994. "Transfer of plutonium across the human gut and its urinary excretion." *Radiation Protection Dosimetry* 53, 241-244.

Pearman, I., Foster, P.P., Ramsden, D. and Bains, M.E.D. 1989. "Lung Clearance of Inhaled Cobalt Oxide in Man." In: *Radiation Protection — and Practice. Proceedings of the Fourth International Symposium of the SRP*, (Ed. Goldfinch, E.P.) IOP Publishing, Bristol, pp. 252-254.

Rencova, J., Volf, V., Jones, M.M. and Singh, P.K. 1994. "Decorporation of polonium from rats by new chelating agents." *Radiat. Prot. Dosim.* 53(1-4), 311-313.

Rowland, R.E. 1993. "Low-level radium retention by the human body: a modification of the ICRP 20 retention equation." *Health Phys.* 65, 507-513.

Shimmins, J., Smith, D.A., Nordin, B.E.C. and Burkinshaw, L. 1967. "A comparison between Calcium-45 and Strontium-85 Absorption, Excretion and Skeletal Uptake." In: *Strontium Metabolism* (Eds. J.M.A. Lenihan, J.F. Loutit, J.J. Martin) Academic Press, London, pp. 149-159.

Sikov, M.R. 1987. "Placental Transfer of the Actinides and related Heavy Elements." In: *Age-related Factors in Radionuclide Metabolism and Dosimetry* (Eds. G.B. Gerber, H. Métivier and H. Smith) Martinus Nijhoff, Dordrecht, pp. 303-314.

Stara, J.F., Nelson, N.S., Della Rosa, R.J. and Bustad, L.K. 1971. "Comparative Metabolism of Radionuclides in Mammals: A Review." *Health Phys.* 20, 113-137.

Stather, J.W., Adams, N., Gray, S.A. and Rees, M. 1987. "Comparative Studies on the Transfer of Radionuclides to the Fetus in the Rat — Implications for Human Dosimetry." In: *Age-related Factors in Radionuclide Metabolism and Dosimetry* (Eds. G.B. Gerber, H. Métivier and H. Smith) Martinus Nijhoff, Dordrecht, pp. 371-380.

Stradling, G.N., Stather, J.W., Gray, S.A., Moody, J.C., Ellender, M., Pearce, M.J. and Collier, C.G. 1992. "Radiological implications of inhaled ^{239}Pu and ^{241}Am in dusts at the former nuclear test site in Maralinga." *Health Physics* 63, 641-650.

Talbot, R.J., Newton, D., Warner, A.J., Walters, B. and Sherlock, J.C. 1993. "Human uptake of ^{137}Cs in mutton." *Health Phys.* 64, 571-578.

Talbot, R.J., Newton, D. and Warner, A.J. 1993. "Metabolism of injected plutonium in two healthy men." *Health Physics* 65, 41-46.

Taylor, D.M. 1959. *The Metabolism of some Trace Elements in Animals and Man*. Ph.D. Thesis, University of London.

Volf, V. 1973. "Dekorporierung von Radionukliden: Untersuchungen an Polonium." *Strahlentherapie* 145, 101-115.

Wissenschaftliche Tabellen Geigy, CIBA-GEIGY, Basel, 1977 p. 257.

Question 1. Average ventilation rates, L min^{-1} , assuming a normal daily (24h) mix of activities (combined male, female average).

	5 year old children			Mature Adults		
	5%	50%	95%	5%	50%	95%
L min^{-1}	4.1	6.1	9.1	8.9	13.4	20.1

Question 2. Total initial deposition in the respiratory tract, % of total amount inhaled, normal daily (24 hr) mix of activities.

	0.1 μm AMAD			1 μm AMAD			10 μm AMAD		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Mature Adults	22	44	88	18	36	72	38	77	99
5 year old children				22	44	88			

Question 3. Initial deposition in the extrathoracic region, % of total deposition in the respiratory tract? (This question also gives the initial deposition in the lung (tracheobronchial (TB) + pulmonary (AI) regions), since lung = total - ET).

	0.1 μm AMAD			1 μm AMAD			10 μm AMAD		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Mature Adults	3.1	6.1	12.2	8.6	17.3	34.6	32	64	99
5 year old children				10.8	21.7	43.4			

Question 4. Initial deposition in the tracheobronchial (TB) region, % of the total deposition in the lung (TB + AI regions).

	0.1 μm AMAD			1 μm AMAD			10 μm AMAD		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Mature Adults	14	28	56	16	33	66	22	44	88
5 year old children				23	46	92			

Question 5. Assuming completely insoluble particles (1 μm AMAD), retention in each respiratory tract region in mature adults as a % of the total initial deposition in the respiratory tract, as a function of time after deposition.

	1 hr			1 day			1 week			
	5%	50%	95%	5%	50%	95%	5%	50%	95%	
Tracheobronchial airways	3	6	12	2	4	8	1.2	2.5	5	
Pulmonary (AI) region	6	12	24	6	12	24	6	11	23	
			1 month			1 year			10 years	
Tracheobronchial airways	0.6	1.3	2.6	0.07	0.2	0.6	0.02	0.1	0.2	
Pulmonary (AI) region	5	10	20	2	6	18	0.1	1	5	

Question 6. By what factors would you expect the median values to be different in 5 year old children (1=no difference).

	1hr	1 day	1 week	1 month	1 year	10 years
Tracheobronchial airways	1	1	1	1	1.5	2
Pulmonary (AI) region	1	1	1	1	1.5	2

Question 7. Absorption (dissolution and transfer) to blood in adults, % of the total initial deposition (1 μm AMAD particles) in the respiratory tract.

	1 hr			1 day			1 week		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Sr	7	14	28	8	15	30	9	17	34
I	80	96	99	82	99	99	82	99	99
Cs	34	41	49	38	46	55	48	63	82
Pu	0	0.02	0.1	0.05	0.3	0.9	1	2.4	5
Ru	0.5	2.5	12	0.6	3	15	1	5	20
Ce	0	0.02	0.1	0.05	0.3	0.9	1	2.4	5
Te	27	54	99	27	55	99	28	56	99
	1 month			1 year			10 years		
Sr	11	22	44	27	40	60	51	66	86
I	82	99	99	90	99	99	90	99	99
Cs	70	88	99	89	95	100	89	95	100
Pu	2	5	11	6	12	25	9	15	28
Ru	2	11	50	6	30	60	10	50	75
Ce	2	5	11	6	12	25	9	15	28
Te	29	58	99	34	67	99	40	80	99

Question 8. Factors by which the median values would be different in 5 year old children (1=no difference).

5 year old children	1 hr	1 day	1 week	1 month	1 year	10 years
Sr	1	1	1	1	2	2
I	1	1	1	1	2	2
Cs	1	1	1	1	2	2
Pu	1.5	1.5	1.5	1.5	3	3
Ru	1.5	1.5	1.5	1.5	3	3
Ce	1.5	1.5	1.5	1.5	3	3
Te	1	1	1	1	2	2

Question 9. Absorption to blood as a fraction (f_1) of activity ingested.

	3 month old infants			5 year old children			Adults		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Sr	0.2	0.6	0.99	0.15	0.3	0.60	0.09	0.14	0.23
I	0.80	0.95	0.99	0.80	0.95	0.99	0.80	0.95	0.99
Cs	0.7	0.9	0.99	0.7	0.9	0.99	0.70	0.85	0.99
PuO ₂ *	1×10^{-5}	1×10^{-4}	1×10^{-3}	1×10^{-6}	1×10^{-5}	1×10^{-4}	1×10^{-6}	1×10^{-5}	1×10^{-4}
Pu biol†	2×10^{-4}	1×10^{-3}	1×10^{-2}	2×10^{-5}	1×10^{-5}	1×10^{-4}	2×10^{-5}	1×10^{-4}	1×10^{-3}

* Refractory oxide
† "Biologically incorporated"

Question 10. Considering the total amount reaching blood (as if administered intravenously as a single injection). % retained in liver and skeleton (bone + bone marrow), as a function of time after entry into blood.

	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
Skeleton and liver, 1 day						
Sr	57	68	82	29	35	42
Pu	53	80	99	53	80	99
Ce	53	80	99	53	80	99
Te	5	27.2	54	5	27	54
Skeleton and liver, 1 week						
Sr	47	56	67	20	24	29
Pu	51	77	99	53	80	99
Ce	53	79	99	53	80	99
Te	5	26.7	53	5	26.7	53
Skeleton and liver, 1 month						
Sr	43	52	62	19	23	28
Pu	51	77	99	53	80	99
Ce	50	75	99	53	80	99
Te	5	26	51	5	26	51
Skeleton and liver, 1 year						
Sr	21	32	48	8	13	20
Pu	25	75	99	26	79	99
Ce	25	75	99	26	79	99
Skeleton and liver, 10 years						
Sr	4	8	16	3	6	12
Pu	19	57	99	24	73	99
Ce	15	45	99	20	61	99
Skeleton and liver, 50 years						
Sr	2	4	8	1	3	9
Pu	10	31	93	16	50	99
Ce	7	22	66	11	33	99

Question 11. Retention in the skeleton, % total retention in liver + skeleton (bone + marrow), as a function of time after entry into blood.

	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
Skeleton, 1 day						
Sr	81	87	99	81	91	99
Pu	58	87	99	54	81	99
Ce	50	75	99	27	50	75
Te	46	92	99	46	92	99
Skeleton, 1 week						
Sr	77	92	99	56	92	99
Pu	56	84	99	54	81	99
Ce	50	75	99	27	50	75
Te	46	92	99	46	92	99
Skeleton, 1 month						
Sr	67	98	99	80	96	99
Pu	55	83	99	53	79	99
Ce	50	75	99	27	50	75
Te	46	92	99	46	92	99
Skeleton, 1 year						
Sr	67	100	100	33	100	47
Pu	53	80	99	24	58	90
Ce	53	80	99	13	53	40
Skeleton, 10 years						
Sr	50	99	99	50	98	99
Pu	16	47	99	18	55	99
Ce	28	85	99	30	85	99
Skeleton, 50 years						
Sr	50	100	100	7	100	11
Pu	23	68	99	22	68	66
Ce	31	95	99	30	94	99

Question 12. Retention of plutonium on endosteal bone surfaces (considering a 10 μm depth of bone mineral) as a % of total skeletal retention, as a function of time after entry into blood.

		5 year old children			Adults		
		5%	50%	95%	5%	50%	95%
Endosteal bone surface	1 day	92.3	95.6	99.8	92.9	95.9	99.9
	1 week	90	93.7	98.9	92.2	95.8	99.8
	1 month	84	92	98	89.9	94.1	99.4
	1 year	13	51	84	69	87	99
	10 years	12	38	90	14	38	68
	50 years	6	19	60	3	10	30

Question 13. Retention of plutonium on trabecular surfaces, % total endosteal surface retention as a function of time after entry into blood.

		5 year old children			Adults		
		5%	50%	95%	5%	50%	95%
Trabecular bone surface	1 day	33	50	67	40	60	70
	1 week	33	50	59	40	60	70
	1 month	33	50	59	40	59	69
	1 year	31	45	58	37	54	60
	10 years	15	40	60	4	13	39
	50 years	15	40	60	2	6	18

Question 14. Retention of plutonium in red bone marrow as a % total skeletal retention, as a function of time after entry into blood.

		5 year old children			Adults		
		5%	50%	95%	5%	50%	95%
Red bone marrow	1 day	0.006	0.03	0.15	0.006	0.03	0.15
	1 week	0.04	0.2	1	0.04	0.2	1
	1 month	0.14	0.7	3.5	0.14	0.7	3.5
	1 year	0.12	0.6	3.0	0.12	0.6	3.0
	10 years		<0.01			<0.01	
	50 years		<0.01			<0.01	

Question 15. Considering the total amount reaching blood (as if administered intravenously as a single injection), % total retention (whole body or systemic) as a function of time after entry into blood.

	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
1 day						
Ru	69	83	99	69	83	99
Cs	93	95	97	87	94	98
1 week						
Ru	54	65	78	54	65	78
Cs	60	73	83	79	84	88
1 month						
Ru	18	39	78	18	39	78
Cs	18	32	47	56	70	75
1 year						
Ru	4	16	64	4	16	64
Cs	0.0003	0.01	0.4	0.4	4	17
5 years						
Ru	0.7	6	48	0.7	6	48
Cs	1E-28	3E-19	2E-13	1E-10	7E-5	2E-2

Question 16. Considering the total amount of Iodine reaching blood (as if administered intravenously as a single injection), % retained in the thyroid as a function of time after entry into blood.

	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
1 day						
	19	39	79	15	30	48
1 week	15	34	74	13	28	45
1 month	5	20	56	8	22	35
3 months	0.3	5	28	3	13	39

Question 17. Intake dose coefficients, absorbed dose to specified organs or tissue per unit activity inhaled or ingested (committed equivalent doses), Gy Bq⁻¹.

	5 year old children			Adult		
	5%	50%	95%	5%	50%	95%
⁹⁰Sr, inhalation, 1 μm AMAD						
lung	2.8E-07	4.2E-07	6.3E-07	1.4E-07	2.1E-07	3.1E-07
bone marrow	1.3E-07	1.9E-07	2.8E-07	1.1E-07	1.6E-07	2.8E-07
bone surface	3.0E-07	4.5E-07	6.7E-07	2.5E-07	3.7E-07	5.5E-07
⁹⁰Sr, ingestion						
colon	3.3E-08	5E-08	7.5E-08	1E-08	1.5E-08	2.2E-08
bone marrow	1.1E-08	1.7E-08	2.5E-08	3.2E-09	4.8E-09	7.2E-08
bone surface	2.1E-08	3.1E-08	4.6E-08	4E-09	6.0E-09	9.0E-09
¹³¹I, inhalation, 1 μm AMAD + vapor (proportions given in original)						
thyroid	4.8E-07	9.6E-07	1.9E-06	1.0E-07	2.0E-07	4.0E-07
¹³¹I, ingestion						
thyroid	1.0E-06	2.1E-06	4.2E-06	2.1E-07	4.3E-07	8.6E-07
¹³²Te, inhalation, 1 μm AMAD + vapor (proportions given in original)						
lung	1.7E-10	8.5E-10	4.2E-09	6E-11	2.9E-10	1.4E-09
thyroid	4.1E-08	1.9E-07	9.5E-07	7E-09	3.5E-08	1.8E-07
colon	1.5E-09	7.4E-09	3.7E-08	4E-10	2.0E-09	1E-08
¹³⁷Cs, inhalation, 1 μm AMAD						
lung	0.8E-08	1.2E-07	1.8E-07	5E-08	6.3E-08	9.4E-08
colon	6.5E-09	9.8E-09	1.5E-08	3.1E-09	3.9E-09	5.8E-09
stomach	1.8E-09	2.7E-09	4.0E-09	1.5E-09	2.2E-09	3.3E-09
bone marrow	1.3E-09	2.0E-09	3.0E-09	1.5E-09	2.2E-09	3.3E-09
¹³⁷Cs, ingestion						
lung	2.8E-09	8.1E-09	1.3E-08	8.7E-09	1.2E-08	1.9E-08
colon	4.7E-09	1.3E-08	2.1E-08	1.1E-08	1.5E-08	2.4E-08
stomach	3.2E-09	9.1E-09	1.4E-08	8.7E-09	1.2E-08	1.9E-08
bone marrow	2.8E-09	8.0E-09	1.2E-08	8.7E-09	1.2E-08	1.9E-08
¹⁴⁴Ce, inhalation, 1 μm AMAD						
bone surface	1.3E-07	3.9E-07	1.2E-06	6.3E-08	1.9E-07	5.7E-07
lung	5.3E-08	1.6E-07	4.8E-07	9.3E-09	2.8E-08	8.4E-08
bone marrow	1.2E-07	2.1E-07	6.3E-07	1.6E-08	4.9E-08	1.5E-07
liver	7.3E-08	2.2E-07	6.6E-07	4.6E-09	1.4E-07	4.2E-07
²³⁹Pu, inhalation, 1 μm AMAD						
bone surface	2.5E-06	7.5E-06	2.3E-05	3.0E-06	9.0E-06	2.7E-05
bone marrow	1.7E-07	5.0E-07	1.5E-06	1.5E-07	4.6E-07	1.4E-06
liver	7.7E-07	2.3E-06	6.9E-06	6.6E-07	2.0E-06	6.0E-06
lung	2.8E-07	8.5E-07	2.5E-06	1.4E-06	4.3E-06	1.3E-05
²³⁹Pu, ingestion						
bone surface	1.2E-07	3.6E-07	1.1E-06	2.6E-08	8E-08	2.4E-07
bone marrow	5.3E-10	1.6E-09	5.8E-09	1.3E-09	4.0E-09	2.0E-08
liver	4.3E-08	1.3E-07	3.9E-07	3.4E-09	1.7E-08	5E-08
colon	2.6E-08	8.0E-08	2.4E-07	8.3E-10	2.5E-09	7.5E-09

(xi) Joint dosimetry/late effects question: The number of radiation-induced cancer deaths up to 40 years following exposure in a population of a hundred million persons (5×10^7 male, 5×10^7 female) each of whom inhales 10 K Bq of the radionuclides specified.

Nuclide	Physical Form	Chemical Form	Cancer Type	Number of Cancers		
				5%	50%	95%
Pu-239	1 μm AMAD	Oxide	Lung			
			Bone			
			Liver			
			Leukemia			
			All cancers			
Sr-90	1 μm AMAD	Oxide	Lung			
			Bone			
			Leukemia			
			All cancers			

EXPERT E

Ingestion of Strontium, Caesium and Plutonium

Strontium

Much data exists on the bioavailability of strontium. A series of human volunteer experiments were conducted by AEA Technology at Harwell. These measured strontium uptake, using dual stable-isotope methods, for subjects of a variety of ages, including new-born infants. The results revealed considerable inter-subject variability, although uptake in infants was generally higher. Uptake was found to depend upon the presence or absence of food in the stomach, on dietary make-up and on the mass of strontium administered. The uptake fractions given in the table section are based upon the AEA Technology data.

Caesium

Human volunteer studies have consistently shown that caesium is highly bioavailable following its entry into the gut. Experiments using caesium-contaminated sheep meat which was fed to adults showed a range of bioavailabilities with it approaching unity in some individuals. Given this very high level of bioavailability it could hardly be expected to be significantly greater in children. As most caesium salts are soluble then it may be expected that their intake will result in levels of uptake approaching those following the ingestion of biologically incorporated metal. Nevertheless, the possibility exists that some caesium, bound within other matrices as nuclear fuel fleas, will be less bioavailable. This possibility is reflected in the 5% confidence limits of the mean given in the table.

Plutonium

Compared with caesium plutonium is not bioavailable. Limited human information is available for some forms of plutonium, but no human data is available for children.

Animal data is available for different ages and species. All indicate that young infants demonstrate enhanced plutonium uptake, but that this enhanced uptake probably ceases at weaning. The bioavailability of plutonium oxide will depend upon the production temperature of the oxide being greater for high fired oxides. It will also depend upon particle size, being greater where surface: volume ratios are higher. These uncertainties are reflected in the absorbed fractions given in the table section.

Biokinetic Models for Strontium, Plutonium, Cerium and Tellurium

Answers to those questions relating to the systemic distribution and retention of the elements strontium, plutonium, cerium and tellurium are given in the table section. In each case answers to elicitation questions 10 to 15 have been attempted by the application of element-specific biokinetic models. In most cases these models represent adaptations of existing published models; however, in the case of tellurium another model was employed that was based on a re-evaluation of published metabolic data for its nuclides.

Strontium

The biokinetic model for strontium employed to predict the residual fractions of this element remaining in soft tissues and skeleton following an intake at time zero was that given in "Appendix A: Age-Specific Biokinetic Models for the Alkaline Earth Elements and Lead," of ICRP *Publication 67*. This model was based on human data including that generated by human volunteer studies conducted by the MRC and UKAEA at Harwell. Also, in the case of strontium considerable information has been published on its behavior in children. This is unusual and results from the availability of data on stable strontium which has for many years been used as a calcium analogue in metabolic studies. Other information has been gained from the study of fallout strontium in Western populations resulting from the testing of nuclear weapons.

The fraction present in liver was assessed on the basis of the partial mass of the liver c.f., other soft tissues - their being no evidence to suggest that the liver is a special site of strontium deposition.

The fractions of strontium present in the skeletal departments were based on the following assumptions:

- the initial site of strontium deposition is bone surfaces;
- that ion exchange results in the loss of strontium from most surfaces;
- that strontium gradually becomes relocated in the bone mineral by heterionic exchange and by its incorporation in newly formed bone mineral;
- that strontium will, with time become preferentially distributed in cortical bone due to its slower turnover rate (3-10 times slower);
- that no strontium is deposited in macrophages within the red bone marrow.

The base-line predictions for strontium are given in Table 1. Note: Table 1 *cs* denotes the surfaces of cortical bone. This classification includes all internal structural surfaces and the internal endosteal surface. For the assessment *cs* is taken to be equivalent to endosteal surface (*es*). The errors arising from this assumption are considered to be minor compared with other uncertainties in the model; this is especially so since little is known about the relative radiosensitivity of different bone surfaces.

Question 10: Fraction of the blood intake retained by the skeleton plus liver

The data given in the table section for the 50% probability of the mean values have been extracted directly from Table 1. It can be seen that the values given are little different for the adult and child, aged five years, for times 10 years post-intake and longer. This reflects an assumed similar bone concentration in both cases at the time of skeletal maturation. The assumption of intake at other ages during childhood would have resulted in different values for the fraction remaining at 50 years post-intake.

The 95 and 5% confidence limits of the mean have been derived by simple multiplication by factors of 1.5X and 0.5X, respectively, for times up to 1 year post-intake and by larger factors at later times. These are based upon observed spread in the biological data, the earlier variations being mostly due to differences in ion exchange rates and kidney function. The later times incorporate an additional factor to take account of inter-person variations in the rate of bone turnover.

Question 11: Fraction of strontium in the skeleton plus liver. Present in the skeleton

The values given in the table section reflect the lack of evidence suggesting that the liver is an important site of strontium deposition. At all times after intake the overwhelming fraction of strontium will be present in the skeleton. The values given assume an intake of soluble strontium by the bloodstream. If particulate strontium were to gain entry to blood by translocation from lungs or by the uptake of the particles via the skin/gut then the liver fraction would be higher. However, there is little evidence to suggest that this is an important route of intake.

Table 1. Showing the Assumed Distribution of Strontium at Different Times Post - Intake by an Adult and Child - Aged 5 years.

Adult	1d	1w	1m	1y	10y	50y
total	43	32	27	15	8	4
soft tiss	13	11	7	.2	.01	0
skeleton	30	21	20	15	8	4
bs	25	10	6	1	.5	.2
<i>ts</i>	13	5	2	.1	.05	.02
<i>cs</i>	12	5	4	.9	.45	.18
bv	5	11	14	14	7.5	3.8
<i>tv</i>	3	6	5	1	.5	.2
<i>cv</i>	2	5	9	13	7	3.6
rbm	0	0	0	0	0	0
liver	0.3	0.3	.02	0	0	0
Child	1d	1w	1m	1y	10y	50y
total	80	72	63	36	9	4
soft tiss	15	12	8	1	.02	0
skeleton	65	60	55	35	9	4
bs	54	29	17	2	.5	.2
<i>ts</i>	28	15	6	.2	.05	.02
<i>cs</i>	26	14	11	1.8	.45	.18
bv	11	31	39	33	8.5	3.8
<i>tv</i>	7	17	14	2	.5	.2
<i>cv</i>	4	14	25	31	8	3.6
rbm	0	0	0	0	0	0
liver	.4	.4	.05	.005	0	0

Question 12: The fraction of skeletal deposit present on endosteal (cortical) bone surfaces

The 50% probability fractions given below are those presented in Table 1. It can be seen that the assumed fraction present on the cortical bone surfaces drops relatively rapidly following strontium intake - this decrease resulting from the transfer of strontium from bone surface to the bone volume. The 95 and 5% confidence limits have been estimated to allow for inter-subject variability and uncertainties in the importance of cortical bone remodelling as a vector returning strontium from the bone volume to bone surfaces as a consequence of bone turnover. These uncertainties will be greater for children than for adults.

Question 13: The fraction of skeletal strontium present on trabecular bone surfaces

As for question 12 the 50% probability of the mean values have been reproduced directly from Table 1. It can be seen that compared with the fraction present on cortical bone surfaces the fraction of strontium present on trabecular bone surfaces decreases much more rapidly with increasing time after intake. This rapid decrease has been suggested to account for the more rapid loss of strontium from trabecular bone than from cortical bone due to its much higher rate of bone turnover. This is consistent with autoradiographic results which for a variety of alkaline earth elements show that at late times after their injection trabecular bone contains much less radionuclide than cortical bone. Also, due to its higher turnover rate at any time a higher fraction of cortical bone strontium than trabecular bone strontium may be expected to be present on their respective bone surfaces.

The 95 and 5% confidence limits have been derived by judgment; there are no autoradiographic data available to make a more systematic assessment. However, it would be difficult to imagine mean values falling outside the range of values presented in the tables overleaf.

Question 14: The fraction of the skeletal strontium deposit present in the red bone marrow

There is no evidence to suggest that either strontium or any other alkaline earth element deposits in the red bone marrow. All irradiation of this organ will be by radiostrontium present either on bone surfaces or within the bone volume adjacent to the red bone marrow. It follows that a zero fraction has been suggested for all times post-intake.

Plutonium

There is considerable information available on the distribution of plutonium in the internal organs of man. In addition, several human volunteer studies have been undertaken, most recently at Harwell using the isotope ^{237}Pu . These data have been used to derive the distribution data given in Table 2. If ICRP models had been used to derive the data then different, but similar values would have been produced. The most significant differences arise from the higher than ICRP fractions of plutonium originally deposited in the liver and the relatively long time taken for liver and bone deposits to accumulate following plutonium

Table 2. Showing the Assumed Distribution of Plutonium at Different Times Post-Intake by an Adult and Child - Aged 5 years.

Adult	1d	1w	1m	1y	10y	50y
total	98	95	93	90	84	62
soft tiss	42	16	6	7	7	6
skeleton	21	21	22	23	37	39
bs	21	21	21	18	19	20
ts	14	14	14	11	11	12
cs	7	7	7	6	8	8
bv	0	0	1	5	17	18
tv	0	0	0.5	3	10	4
cv	0	0	0.5	2	7	14
rbm	0	0	0.4	0.9	1.1	1.1
liver	35	58	65	60	40	20
Child	1d	1w	1m	1y	10y	50y
total	98	95	85	80	61	46
soft tiss	39	23	10	8	6	5
skeleton	49	52	55	50	34	25
bs	50	52	37	23	16	13
ts	30	31	21	13	7	8
cs	20	21	16	10	9	5
bv	0	0	17	24	18	12
tv	0	0	12	10	5	2
cv	0	0	5	14	13	10
rbm	0	0	1.5	3.0	2.0	0.8
liver	10	20	20	22	21	17

injection. The model used to calculate the values in Table 2 was a modified Harwell model. Unlike the ICRP model the latest version of the Harwell model is a probabilistic model where transfer coefficients are also given specific probability distributions. Using this model the variability in human postmortem data can be reproduced. For example, the model predictions of the considerable variations in measured bone: liver plutonium ratios are reproduced. It follows that in most cases the 95 and 5% confidence limits have been derived by modeling rather than by judgment and assessment. The only exceptions apply to the derivation of some bone fractions.

Question 10: The fraction of plutonium intake present in the skeleton plus liver

The human volunteer studies at Harwell and postmortem observations consistently show that at times longer than 1 day after intake most plutonium in the body is present either in the skeleton or the liver. Consequently, for early times after intake the mean fractions given in the table section range from a low 46% to a high of 90%. At later times the fraction decreases due to excretion losses of plutonium. The data presented are based on data available for men; it is possible that in women plutonium excretion is more rapid and that normal retained fractions approach the five percentile values of the mean. Nevertheless, the confidence limits given are regarded as being sufficiently wide to account for both males and females.

Little information is available on the fraction of plutonium present in the liver and skeleton of children, but again all the available human and animal data point to high fractions. Nevertheless, compared with adults, the fraction at 10 years and 50 years post-intake will be lower due to high plutonium excretion during childhood.

Question 11: The fraction of skeleton plus liver plutonium present in skeleton

Contrary to predictions made using ICRP *Publication 67* age-specific biokinetic model for plutonium all of ten adult human volunteers at Harwell demonstrated much higher levels of plutonium uptake by the liver than by the skeleton at early times after injection. Liver uptake levels at 10 days post-injection ranged from 43–70% of the injected dose in females and 55 to 85% in males. Moreover, maximum liver concentrations did not occur until about 10 days after injection. These levels are reflected in the predictions given. It also follows that since most late autopsy data show more plutonium in the skeleton than liver, but with considerable variation, the rate of plutonium loss from the liver must be significantly higher than that from the skeleton.

For children, the fraction of plutonium taken up by the skeleton will be higher. This is a consistent observation in all animal studies and is probably related to the more extensive marrow sinusoidal system present in young animals which gives bone surfaces better access to circulating plutonium. A higher skeletal uptake fraction has been assumed for the five-year-old child.

For both adults and child the 95 and 5% confidence limits on the mean have been set sufficiently wide to account for the wide individual variations in organ content noted at

autopsy. These range in extreme cases from 90% skeleton: 10% liver to 10% skeleton: 90% liver. It should be pointed out however, that some of this variation may be due to the presence of translocated oxide particles from lung to liver in some cases where exposure has been to highly insoluble plutonium oxide and where little soluble radionuclide transfer to extra-pulmonary tissues has occurred.

Question 12: The fraction of skeletal plutonium present on cortical (endosteal) bone surfaces

Compared with many other metals plutonium seems to find diffusing through the wall of blood vessels more difficult; consequently those bone surfaces showing the highest plutonium uptake are those close to leaky blood sinusoids rather than blood vessels. Blood sinusoids are not commonly associated with cortical bone surfaces in adults; consequently these surfaces do not accumulate the highest levels of plutonium. In children blood sinusoids, associated with red bone marrow are much more extensive and are more accessible to plutonium. For the present exercise it assumed that despite accounting for 50% of total bone surface area the surfaces of cortical bone in the adult accumulate only 33% of the plutonium. This fraction is consistent with the levels of plutonium accumulated by adult baboon bone surfaces.

The 95 and 5% confidence limits in the mean given in the table section reflect both uncertainties in the concentration of plutonium by cortical bone surfaces and uncertainties in the fraction cortical: trabecular bone surface area. Note that because of low bone turnover rates most cortical bone plutonium is assumed to be in the cortical bone volume at 50 years post-intake.

Question 13: Fraction of skeletal plutonium deposit present on trabecular bone surfaces

Contrary to the situation for cortical bone, the surfaces of trabecular bone are commonly associated with red bone marrow and close to blood sinusoids. It follows that trabecular bone surfaces are best placed to concentrate plutonium. For the purposes of the present exercise it is assumed that two-thirds of the plutonium that deposits in the adult skeleton is deposited on trabecular bone surfaces. In the 5-year-old child this fraction is smaller.

For both trabecular and cortical bone types the plutonium distribution given in Table 2 for children has been derived from direct measurements made in juvenile baboon bone, corrected for the different volume/area fractions of the

components. For adults the fractions are consistent with the limited human autoradiographic data.

Note that whereas for cortical bone at late times after intake most plutonium was present in the bone volume, for trabecular (in adults) most is present on bone surfaces. This is because of the high turnover of trabecular bone which redistributes volume deposits onto bone surfaces.

Question 14: The fraction of skeletal plutonium present in the red bone marrow

Soluble plutonium does not deposit in the red bone marrow hence at early times after intake no plutonium is present in this tissue. Later, some of the plutonium released by bone turnover is retained for a relatively short time within bone marrow macrophages. At low-radiation doses these deposits are rapidly lost and the radionuclide re-cycled. At high-radiation doses evidence exists to suggest that marrow macrophage deposits survive longer. Also, there is evidence to suggest that in females, that commonly have high levels of iron turnover, plutonium is lost more rapidly from macrophages.

In both adults and children the amount of plutonium present in the red bone macrophages has been modeled to correspond with levels found in autoradiographs of adult human bone and adult and juvenile baboon bone. In general an equilibrium between macrophage and bone plutonium will become established; the position of equilibrium depends on the relative rates of bone turnover and the rates of plutonium loss from macrophages. It should be noted that in children a higher than adult fraction of the total bone marrow is of the red type. This observation adds an additional layer of complexity to models.

In the table section the 50% probability of the mean levels has been set as listed in Table 2, and the confidence limits given reflect possible variations due to variations in the local rate of bone turnover and in the rate of plutonium loss from macrophages. They are also set sufficiently wide to encompass most accident situations where high skeletal radiation doses might be expected.

Cerium

Compared with plutonium and strontium very little is known about the specific behavior of cerium in man and animals. However, cerium is a trivalent lanthanide element, and these have been shown to behave similarly to the trivalent actinide elements. Indeed animal studies suggest that the behavior of the lanthanide promethium and the

actinide americium, both of which have the same ionic radius, is close to identical. Studies have also shown that cerium, having a larger ionic radius than promethium will be more likely to deposit in the liver. It follows that for the present purposes the americium model as given in "Appendix B: Age-Specific Biokinetic Models for Plutonium, Americium and Neptunium" (ICRP Publication 67) has been employed for cerium with appropriate modifications within the range of confidence limits to include a higher level of liver uptake. Nevertheless, in man most americium is assumed to be concentrated initially by the liver; therefore, substantial modifications were considered unnecessary.

Question 10: Fraction of the blood intake retained by the skeleton plus liver

The 50% probability of the mean fraction of cerium retained by the liver plus skeleton for different times after intake are given in the table section. Compared with plutonium, the later fractions are very low. This is because cerium, like americium, is assumed to be lost from the liver with a short retention half-time. However, because information on cerium is less readily available than for the actinide elements quite wide 95 and 5% confidence limits are given.

Question 11: Fraction of cerium in the skeleton plus liver in the skeleton

The 50% probability of the mean values for the fraction of hepatic plus skeletal cerium present in the skeleton, as presented in the table section, have been calculated directly from the tissue content fractions given in Table 3. The 95 and 5% confidence limits have been set wide so as to include all likely liver:skeleton cerium ratios ranging from 10 to 60% skeletal for adults and 30 to 88% skeletal for 5-year-old children. Note that at late times after intake skeletal deposits of cerium dominate; this is because of the assumed relatively short half-time for cerium in the liver.

Note that unlike for plutonium no delay in the uptake of cerium by the liver is assumed. This may be an oversight, but is only likely to be dosimetrically important for radioisotopes with a very short half-life. In practise these are not the most likely to be dosimetrically significant following reactor accidents.

Question 12: Fraction skeletal deposit on cortical (endosteal) bone surfaces

The fractions presented in the tables below assume that cerium, like promethium and americium diffuses relatively

Table 3. Showing the Assumed Distribution of Cerium at Different Times Post-Intake by an Adult and Child - Aged 5 years.

Adult	1d	1w	1m	1y	10y	50y
total	95	93	90	87	63	44
soft tiss	30	13	10	7	5	5
skeleton	25	30	30	40	50	35
bs	25	30	29	31	25	10
ts	13	13	14	15	10	5
cs	12	12	15	16	15	5
bv	0	0	1	9	25	25
tv	0	0	0.5	4	3	5
cv	0	0	0.5	5	22	20
rbm	0	0	0.3	0.5	0.6	0.4
liver	40	50	50	40	8	4
Child	1d	1w	1m	1y	10y	50y
total	98	95	90	81	51	26
soft tiss	39	23	10	8	6	4
skeleton	49	52	60	55	35	20
bs	50	52	38	29	16	10
ts	25	26	18	14	8	5
cs	25	26	20	15	8	5
bv	0	0	21	24	18	10
tv	0	0	12	6	5	2
cv	0	0	9	18	13	8
rbm	0	0	1.0	2.0	10	0.4
liver	10	20	20	18	10	2

easily through blood vessel walls and is deposited evenly on all types of bone surface, where it will remain until lost due to burial or removal during bone resorption.

Given the lack of data on cerium metabolism the 95 and 5% confidence limits on the mean fraction have been set much wider than for plutonium.

Question 13: Fraction skeletal cerium on trabecular bone surfaces

The fractions given for trabecular bone surfaces are the same as those given for cortical bone surfaces. This reflects the lack of lanthanide discrimination between cortical and trabecular bone surfaces and uncertainties in the data.

Question 14: Fraction skeletal deposit in red bone marrow

Like plutonium and americium, the lanthanide elements are not initially deposited in the red bone marrow, following their entry into the bloodstream, but are accumulated gradually as the result of bone resorption. It follows that the fractions given in Table 3 and in the table section have been calculated in the same way as for plutonium. However, little is known about the retention half-time of cerium in marrow macrophages; consequently, the 95 and 5% confidence limits on the mean skeletal fraction of cerium in the red bone marrow have been set rather wide ranging from a low of zero for early times to a high value of 8% for the 5-year-old child at one year post-intake, considering its lack of specificity for bone surfaces in red bone marrow areas.

Autoradiographs prepared from adult human bone contaminated with americium suggest that americium, if not cerium, accumulates in red bone marrow to a similar extent as plutonium. Also, as for plutonium the peak red bone marrow levels for children will be higher than for adults because of their higher rates of bone resorption and because of their more extensive red bone marrow, which in children extends into those bones which contain only fatty yellow marrow in adults.

Tellurium

Recourse to the literature has revealed little about the distribution and metabolism of tellurium. Analysis of the stable isotopes present in autopsy samples has suggested that about 90% (450 mg) of the body's tellurium is present in the skeleton with about 50% of the remainder in the liver. The remaining 25 mg seemed to be distributed within other soft tissues to a greater or lesser extent. Animals studies with radioisotopes of tellurium suggest a rather different distribution with only a little tellurium present in the skeleton and liver at early times after injection, but with only the skeleton retaining its deposits. ICRP for dosimetry purposes assumes that 50% of tellurium is immediately excreted and of the remainder half is deposited in the skeleton and the remainder in soft tissues with no specific concentration in the liver. Biological half-times of 10,000 and 20 are allocated to the tellurium in the skeletal and soft-tissue compartments respectively. No information is available on any age-related differences in metabolism.

Owing to the paucity of data for this element it was considered inappropriate to allocate different models for adults and children as any errors introduced by this omission would be small compared with uncertainties in the

model. Also, a model intermediate between that suggested by the postmortem data and animal data has been adopted. A retention time of 10 years has been assumed for tellurium in the skeleton and 30 days for that in soft tissues. However, the long half-time of retention for the skeleton is somewhat academic given the short half-lives of tellurium radioisotopes.

One further complication is that the position of skeletal tellurium is unknown and given the chemical similarities between this element and polonium (also in group VIb of the periodic table) which is known to deposit mostly within the bone marrow, a conservative approach would suggest that the fraction of skeletal tellurium present in the red bone marrow could be anything between 0 and 1, although a low figure is considered most likely. A conservative approach has also been made with regard to the position of tellurium in bone. For the purposes of this exercise it is assumed to be retained upon bone surfaces, perhaps the most reasonable assumption given the short life of tellurium radioisotopes.

The assumed distribution pattern is given in Table 4.

Table 4. Showing the Assumed Distribution of Tellurium at Different Times Post-Intake for all Ages.

Adult and Child	1d	1w	1m	1y	10y	50y
total	.49	.42	.30	.11	.09	.06
soft tiss	.21	.18	.10	0	0	0
skeleton	.15	.15	.14	.11	.09	.06
bs	.15	.15	.14	.11	.09	.06
ts	.075	.075	.07	.065	.045	.03
cs	.075	.075	.07	.065	.045	.03
bv	0	0	0	0	0	0
tv	0	0	0	0	0	0
cv	0	0	0	0	0	0
rbm	.01	.01	.01	.01	.01	.01
liver	.13	.10	.06	0	0	0

Question 10: The fraction of tellurium intake present in the skeleton plus liver

The 95 and 5% confidence limits are set wide so as to cover most contingencies, reflecting the great uncertainty in the distribution of the element.

Question 11: Fraction of tellurium in the skeleton plus liver that is present in the skeleton

At long times after intake tellurium is assumed to be present only in the skeleton.

Question 12: The fraction of skeletal deposit present on cortical (endosteal) bone surfaces

Consistent with the area fraction of this type of bone surface, 50% of tellurium is predicted to be present on cortical bone surfaces. Confidence limits are set sufficiently wide to accommodate most other possibilities.

Question 13: Fraction skeletal tellurium deposit present on trabecular bone surfaces

Fractions and confidence limits as for cortical bone surfaces above.

Question 14: Fraction of the skeletal tellurium deposit present in the red bone marrow

It is considered most likely that little tellurium will be present in the red bone marrow. However, as mentioned above, the possibility that this element behaves like polonium cannot be excluded. Therefore, the confidence limits have been set sufficiently wide to accommodate the possibility that all skeletal tellurium is present in the red bone marrow.

Uncertainties In Radiation Dose Arising from the Inhalation/ingestion Of Radionuclides.

Strontium-90 (Ingestion)

Base line doses for the colon, red bone marrow and bone surfaces were derived from ICRP Publication 56. These values were adjusted to account for uncertainties identified in the fractional gut uptake of strontium and the biokinetic model. All doses are given as Gy/Bq.

Cerium-144 (Inhalation)

For inhalation it was assumed that 30% of the inhaled deposit is translocated to blood as a single bolus at time zero. It follows that the confidence limits given account only for uncertainties in the metabolic model for cerium and that uncertainties in the amount of cerium transferred to the lung to blood should be added. The metabolic model assumed for the dosimetry is that given earlier in this

document and is based upon the metabolic model for americium. All doses given as Gy/Bq.

Plutonium-239 (Inhalation and Ingestion)

In the case of ingestion, for both adults and the five-year-old child, the greatest uncertainties in dose to internal organs are due to uncertainties in gut uptake factors and, in the gut itself to the uncertain fraction of alpha-particle energy deposited in sensitive cells within the gut wall. For example, for the 5% confidence limit of the mean dose to the colon wall, for ingested plutonium, a reduction by a factor of 100 over the 50% value is indicated. This is to account for the possibility that the dose to these cells may be very substantially overestimated due to the failure of current dosimetry systems to account for attenuation of alpha-particles in the mucus layer which coats the internal surfaces of the colon. In contrast, the 95% confidence limit of this mean is only a little increased to allow for errors in the assumed transit time of plutonium in the lower large intestine. For the internal organs the uncertainty in the gut uptake factor, as described in relation to a previous question, is considered to be so great, compared with that arising from uncertainties in the biokinetic models for absorbed

plutonium, that the latter have been ignored, except in the case of the red bone marrow (see below). Doses are given as 50-year committed doses (Gy Bq⁻¹).

In the case of plutonium inhalation, as for cerium and strontium, the calculations have been made assuming a fixed, 30% fraction of the inhaled radioactivity enters the bloodstream instantaneously. It follows, that all the uncertainties indicated in the table section arise from uncertainties in the biokinetic model for plutonium within the body. These uncertainties have been assessed using a sensitivity analysis of the Harwell model predictions for plutonium (see appended details of this analysis). It should be noted, that for the 5% confidence limit of the mean radiation dose received by the red bone marrow a special reduction has been made (also in the case of ingestion) to allow for the possibility that the only important radiation dose is that which arises from plutonium in the marrow, rather than from bone surface plutonium. The means and confidence limits suggested are given in the table section. Further information can be obtained from the appendix on the dose range for individual organs, following the entry of a bolus of plutonium into the blood supply.

Question 1. Average ventilation rates, L min⁻¹, assuming a normal daily (24h) mix of activities (combined male, female average).

	5 year old children			Mature Adults		
	5%	50%	95%	5%	50%	95%
L min ⁻¹	NR	NR	NR	NR	NR	NR

Question 2. Total initial deposition in the respiratory tract, % of total amount inhaled, normal daily (24 hr) mix of activities.

	0.1 μm AMAD			1 μm AMAD			10 μm AMAD		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Mature Adults	NR	NR	NR	NR	NR	NR	NR	NR	NR
5 year old children				NR	NR	NR			

Question 3. Initial deposition in the extrathoracic region, % of total deposition in the respiratory tract? (This question also gives the initial deposition in the lung (tracheobronchial (TB) + pulmonary (AI) regions), since lung = total - ET).

	0.1 μm AMAD			1 μm AMAD			10 μm AMAD		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Mature Adults	NR	NR	NR	NR	NR	NR	NR	NR	NR
5 year old children				NR	NR	NR			

Question 4. Initial deposition in the tracheobronchial (TB) region, % of the total deposition in the lung (TB + AI regions).

	0.1 μm AMAD			1 μm AMAD			10 μm AMAD		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Mature Adults	NR	NR	NR	NR	NR	NR	NR	NR	NR
5 year old children				NR	NR	NR			

Question 5. Assuming completely insoluble particles (1 μm AMAD), retention in each respiratory tract region in mature adults as a % of the total initial deposition in the respiratory tract, as a function of time after deposition.

	10 min			1 hr			1 day		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Tracheobronchial airways	NR	NR	NR	NR	NR	NR	NR	NR	NR
Pulmonary (AI) region	NR	NR	NR	NR	NR	NR	NR	NR	NR
	1 month			1 year			10 years		
Tracheobronchial airways	NR	NR	NR	NR	NR	NR	NR	NR	NR
Pulmonary (AI) region	NR	NR	NR	NR	NR	NR	NR	NR	NR

Question 6. By what factors would you expect the median values to be different in 5 year old children (1=no difference).

	10 min	1 hr	1 day	1 month	1 year	10 years
Tracheobronchial airways	NR	NR	NR	NR	NR	NR
Pulmonary (AI) region	NR	NR	NR	NR	NR	NR

Question 7. Absorption (dissolution and transfer) to blood in adults, % of the total initial deposition (1 μm AMAD particles) in the respiratory tract.

	1 hr			1 day			1 week		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Sr	NR	NR	NR	NR	NR	NR	NR	NR	NR
I	NR	NR	NR	NR	NR	NR	NR	NR	NR
Cs	NR	NR	NR	NR	NR	NR	NR	NR	NR
Pu	NR	NR	NR	NR	NR	NR	NR	NR	NR
Ru	NR	NR	NR	NR	NR	NR	NR	NR	NR
Ce	NR	NR	NR	NR	NR	NR	NR	NR	NR
Te	NR	NR	NR	NR	NR	NR	NR	NR	NR
	1 month			1 year			10 years		
Sr	NR	NR	NR	NR	NR	NR	NR	NR	NR
I	NR	NR	NR	NR	NR	NR	NR	NR	NR
Cs	NR	NR	NR	NR	NR	NR	NR	NR	NR
Pu	NR	NR	NR	NR	NR	NR	NR	NR	NR
Ru	NR	NR	NR	NR	NR	NR	NR	NR	NR
Ce	NR	NR	NR	NR	NR	NR	NR	NR	NR
Te	NR	NR	NR	NR	NR	NR	NR	NR	NR

Question 8. Factors by which the median values would be different in 5 year old children (1=no difference).

5 year old children	1 hr	1 day	1 week	1 month	1 year	10 years
Sr	NR	NR	NR	NR	NR	NR
I	NR	NR	NR	NR	NR	NR
Cs	NR	NR	NR	NR	NR	NR
Pu	NR	NR	NR	NR	NR	NR
Ru	NR	NR	NR	NR	NR	NR
Ce	NR	NR	NR	NR	NR	NR
Te	NR	NR	NR	NR	NR	NR

Question 9. Absorption to blood as a fraction (f_1) of activity ingested.

	3 month old infants			5 year old children			Adults		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Sr	.10	.70	.90	.10	.50	.90	.10	.35	.80
I	NR								
Cs	.40	.80	1.0	.40	.80	1.0	.40	.80	1.0
PuO_2^*	1.0×10^{-3}	1.0×10^{-4}	1.0×10^{-6}	1.0×10^{-4}	1.0×10^{-5}	1.0×10^{-6}	1.0×10^{-4}	1.0×10^{-5}	1.0×10^{-6}
Pu biol†	1.0×10^{-2}	1.0×10^{-3}	1.0×10^{-4}	1.0×10^{-3}	1.0×10^{-4}	1.0×10^{-5}	1.0×10^{-3}	1.0×10^{-4}	1.0×10^{-5}

* Refractory oxide
† "Biologically incorporated"

Question 10. Considering the total amount reaching blood (as if administered intravenously as a single injection). % retained in liver and skeleton (bone + bone marrow), as a function of time after entry into blood.

	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
Skeleton and liver, 1 day						
Sr	.33	.65	.98	.15	.30	.45
Pu	.46	.59	.80	.46	.56	.71
Ce	.40	.59	.88	.30	.65	.84
Te	.07	.28	.75	.07	.28	.75
Skeleton and liver, 1 week						
Sr	.30	.60	.90	.11	.21	.32
Pu	.61	.72	.83	.66	.79	.84
Ce	.40	.72	.90	.35	.80	.89
Te	.06	.25	.60	.06	.25	.60
Skeleton and liver, 1 month						
Sr	.27	.55	.83	.10	.20	.30
Pu	.68	.70	.84	.79	.87	.90
Ce	.50	.80	.88	.46	.80	.87
Te	.05	.20	.50	.05	.20	.50
Sb	NR	NR	NR	NR	NR	NR
Skeleton and liver, 1 year						
Sr	.18	.35	.53	.07	.15	.23
Pu	.61	.64	.78	.75	.83	.87
Ce	.40	.73	.85	.52	.80	.90
Te	.03	.11	.22	.03	.11	.22
Skeleton and liver, 10 years						
Sr	.03	.09	.16	.02	.08	.14
Pu	.45	.55	.69	.68	.77	.81
Ce	.15	.45	.76	.40	.58	.85
Te	.02	.09	.18	.02	.09	.18
Skeleton and liver, 50 years						
Sr	.01	.04	.12	.01	.04	.12
Pu	.29	.42	.58	.44	.56	.66
Ce	.05	.22	.66	.20	.39	.76
Te	.01	.06	.12	.01	.06	.12

Question 11. Retention in the skeleton, % total retention in liver + skeleton (bone + marrow), as a function of time after entry into blood.

	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
Skeleton, 1 day						
Sr	.90	.90	1.0	.90	.99	1.0
Pu	.41	.83	.88	.24	.38	.59
Ce	.30	.83	.88	.10	.38	.59
Te	.27	.54	1.0	.27	.54	1.0
Skeleton, 1 week						
Sr	.95	.99	1.0	.94	.99	1.0
Pu	.37	.72	.84	.19	.27	.54
Ce	.30	.80	.85	.10	.38	.60
Te	.30	.60	1.0	.30	.60	1.0
Skeleton, 1 month						
Sr	.95	.99	1.0	.95	.99	1.0
Pu	.37	.71	.84	.18	.25	.53
Ce	.30	.75	.80	.15	.38	.59
Te	.50	.70	1.0	.50	.70	1.0
Skeleton, 1 year						
Sr	.98	1.0	1.0	.98	1.0	1.0
Pu	.39	.78	.86	.19	.28	.54
Ce	.33	.75	.85	.19	.50	.66
Te	.90	1.0	1.0	.90	1.0	1.0
Skeleton, 10 years						
Sr	.99	1.0	1.0	.98	1.0	1.0
Pu	.34	.62	.80	.29	.48	.64
Ce	.40	.77	.90	.29	.86	.90
Te	.90	1.0	1.0	.90	1.0	1.0
Skeleton, 50 years						
Sr	.99	1.0	1.0	.99	1.0	1.0
Pu	.33	.60	.80	.37	.64	.72
Ce	.50	.91	.98	.37	.90	.95
Te	.90	1.0	1.0	.90	1.0	1.0

Question 12. Retention of plutonium on endosteal bone surfaces (considering a 10 μm depth of bone mineral) as a % of total skeletal retention, as a function of time after entry into blood.

		5 year old children			Adults		
		5%	50%	95%	5%	50%	95%
Endosteal bone surface	1 day	.62	.95	.999	.56	.98	.999
	1 week	.62	.95	.999	.56	.98	.999
	1 month	.43	.67	.93	.53	.96	.99
	1 year	.29	.46	.63	.46	.54	.99
	10 years	.34	.47	.60	.31	.52	.71
	50 years	.31	.52	.73	.33	.55	.77

Question 13. Retention of plutonium on trabecular surfaces, % total endosteal surface retention as a function of time after entry into blood.

		5 year old children			Adults		
		5%	50%	95%	5%	50%	95%
Trabecular bone surface	1 day	.41	.61	.79	.45	.67	.89
	1 week	.40	.60	.78	.45	.67	.89
	1 month	.25	.38	.49	.43	.64	.85
	1 year	.17	.26	.34	.32	.48	.64
	10 years	.14	.21	.27	.20	.30	.40
	50 years	.21	.32	.42	.22	.33	.44

Question 14. Retention of strontium in red bone marrow as a % total skeletal retention, as a function of time after entry into blood.

		5 year old children			Adults		
		5%	50%	95%	5%	50%	95%
Red bone marrow	1 day	0	0	0	0	0	0
	1 week	0	0	0	0	0	0
	1 month	0	0	0	0	0	0
	1 year	0	0	0	0	0	0
	10 years	0	0	0	0	0	0
	50 years	0	0	0	0	0	0

Question 14. Retention of plutonium in red bone marrow as a % total skeletal retention, as a function of time after entry into blood.

		5 year old children			Adults		
		5%	50%	95%	5%	50%	95%
Red bone marrow	1 day	0	0	.005	0	0	.005
	1 week	0	0	.005	0	0	.005
	1 month	.02	.03	.06	.01	.02	.04
	1 year	.03	.06	.12	.02	.04	.08
	10 years	.03	.06	.12	.02	.03	.06
	50 years	.02	.03	.06	.02	.03	.06

Question 14. Retention of cerium in red bone marrow as a % total skeletal retention, as a function of time after entry into blood.

		5 year old children			Adults		
		5%	50%	95%	5%	50%	95%
Red bone marrow	1 day	0	0	.005	0	0	.005
	1 week	0	0	.005	0	0	.005
	1 month	.01	.02	.04	.005	.01	.05
	1 year	.02	.04	.08	.005	.01	.05
	10 years	.02	.03	.06	.005	.01	.05
	50 years	.01	.02	.04	.005	.01	.05

Question 14. Retention of tellurium in red bone marrow as a % total skeletal retention, as a function of time after entry into blood.

		5 year old children			Adults		
		5%	50%	95%	5%	50%	95%
Red bone marrow	1 day	0	.01	1.0	0	.01	1.0
	1 week	0	.01	1.0	0	.01	1.0
	1 month	0	.01	1.0	0	.01	1.0
	1 year	0	.01	1.0	0	.01	1.0
	10 years	0	.01	1.0	0	.01	1.0
	50 years	0	.01	1.0	0	.01	1.0

Question 15. Considering the total amount reaching blood (as if administered intravenously as a single injection), % total retention (whole body or systemic) as a function of time after entry into blood.

	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
1 day						
Ru	NR	NR	NR	NR	NR	NR
Cs	NR	NR	NR	NR	NR	NR
1 week						
Ru	NR	NR	NR	NR	NR	NR
Cs	NR	NR	NR	NR	NR	NR
1 month						
Ru	NR	NR	NR	NR	NR	NR
Cs	NR	NR	NR	NR	NR	NR
1 year						
Ru	NR	NR	NR	NR	NR	NR
Cs	NR	NR	NR	NR	NR	NR
5 years						
Ru	NR	NR	NR	NR	NR	NR
Cs	NR	NR	NR	NR	NR	NR

Question 16. Considering the total amount of Iodine reaching blood (as if administered intravenously as a single injection), % retained in the thyroid as a function of time after entry into blood.

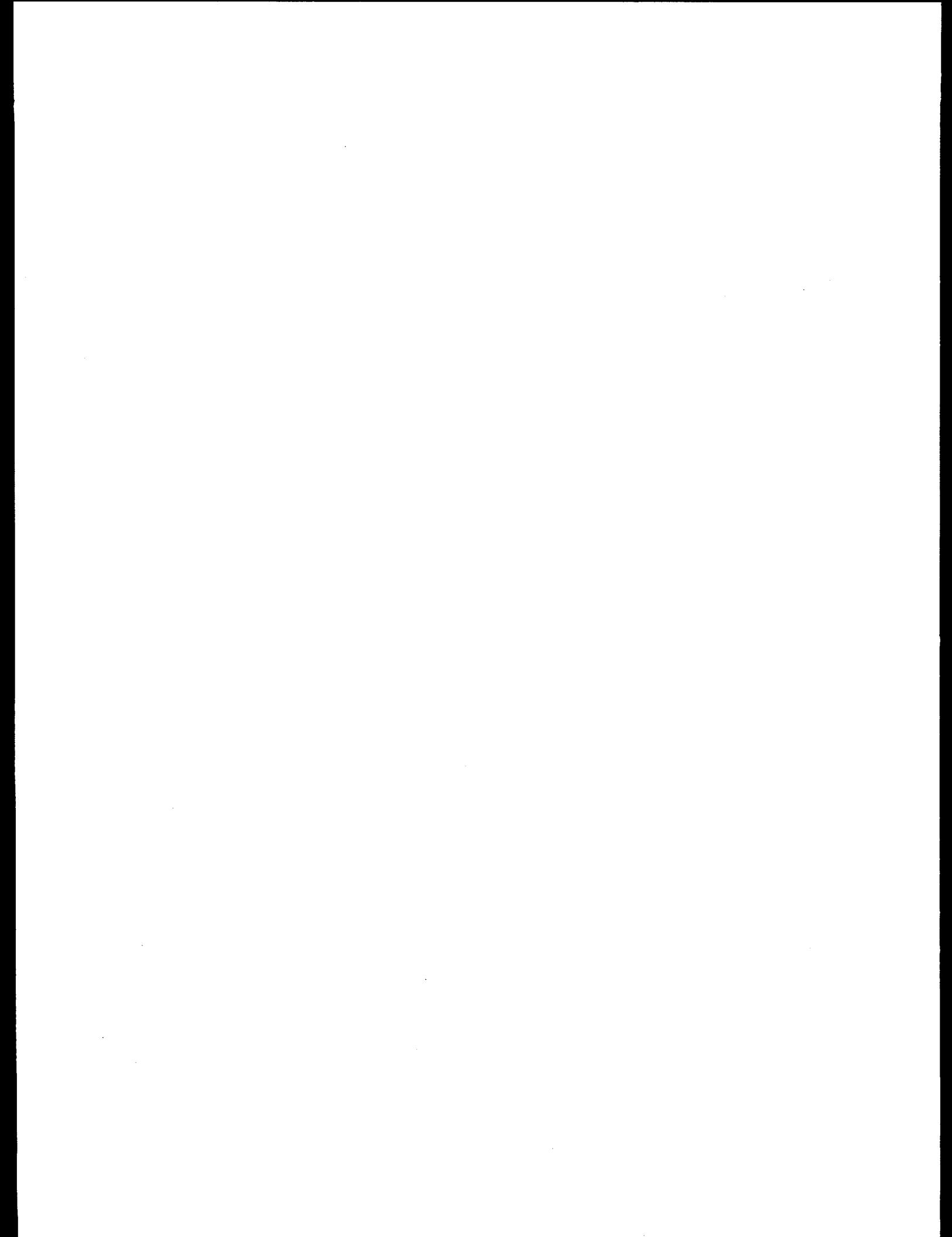
	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
1 day						
	NR	NR	NR	NR	NR	NR
1 week						
	NR	NR	NR	NR	NR	NR
1 month						
	NR	NR	NR	NR	NR	NR
3 months						
	NR	NR	NR	NR	NR	NR

Question 17. Intake dose coefficients, absorbed dose to specified organs or tissue per unit activity inhaled or ingested (committed equivalent doses), Gy Bq⁻¹.

5 year old children			Adult			
	5%	50%	95%	5%	50%	95%
⁹⁰Sr, inhalation, 1 μm AMAD						
lung	NR	NR	NR	NR	NR	NR
bone marrow	NR	NR	NR	NR	NR	NR
bone surface	NR	NR	NR	NR	NR	NR
⁹⁰Sr, ingestion						
colon	1.0×10^{-8}	5.1×10^{-8}	9.1×10^{-8}	5.4×10^{-9}	1.8×10^{-8}	2.4×10^{-8}
bone marrow	1.1×10^{-8}	2.8×10^{-7}	1.3×10^{-6}	1.2×10^{-8}	3.0×10^{-7}	1.4×10^{-6}
bone surface	2.5×10^{-8}	6.4×10^{-7}	3.0×10^{-6}	2.5×10^{-8}	6.3×10^{-7}	3.0×10^{-6}
¹³¹I, inhalation, 1 μm AMAD + vapor (decide proportions)						
thyroid	NR	NR	NR	NR	NR	NR
¹³¹I, ingestion						
thyroid	NR	NR	NR	NR	NR	NR
¹³²Te, inhalation, 1 μm AMAD + vapor (decide proportions)						
lung	NR	NR	NR	NR	NR	NR
thyroid	NR	NR	NR	NR	NR	NR
colon	NR	NR	NR	NR	NR	NR
¹³⁷Cs, inhalation, 1 μm AMAD						
lung	NR	NR	NR	NR	NR	NR
colon	NR	NR	NR	NR	NR	NR
stomach	NR	NR	NR	NR	NR	NR
bone marrow	NR	NR	NR	NR	NR	NR
¹³⁷Cs, ingestion						
lung	NR	NR	NR	NR	NR	NR
colon	NR	NR	NR	NR	NR	NR
stomach	NR	NR	NR	NR	NR	NR
bone marrow	NR	NR	NR	NR	NR	NR
¹⁴⁴Ce, inhalation, 1 μm AMAD						
bone surface	1.0×10^{-6}	2.0×10^{-6}	4.0×10^{-6}	2.2×10^{-6}	4.4×10^{-6}	8.8×10^{-6}
lung	NR	NR	NR	NR	NR	NR
bone marrow	8.0×10^{-7}	1.6×10^{-6}	3.2×10^{-6}	1.3×10^{-6}	2.6×10^{-6}	5.2×10^{-6}
liver	3.1×10^{-7}	7.3×10^{-7}	1.4×10^{-6}	3.1×10^{-7}	6.3×10^{-7}	1.2×10^{-6}
²³⁹Pu, inhalation, 1 μm AMAD						
bone surface	2.0×10^{-4}	9.0×10^{-4}	1.4×10^{-3}	1.4×10^{-4}	2.7×10^{-4}	4.1×10^{-4}
bone marrow	1.6×10^{-5}	6.5×10^{-5}	2.6×10^{-4}	1.4×10^{-6}	1.4×10^{-5}	5.6×10^{-5}
liver	1.5×10^{-4}	2.9×10^{-4}	5.9×10^{-4}	3.0×10^{-5}	6.0×10^{-5}	1.2×10^{-4}
colon	NR	NR	NR	NR	NR	NR
²³⁹Pu, ingestion						
bone surface	6.9×10^{-11}	6.9×10^{-9}	6.9×10^{-7}	9.0×10^{-11}	9.0×10^{-9}	9.0×10^{-7}
bone marrow	5.0×10^{-12}	5.0×10^{-10}	5.0×10^{-8}	1.0×10^{-12}	4.5×10^{-10}	4.5×10^{-8}
liver	2.2×10^{-11}	2.2×10^{-9}	2.2×10^{-7}	2.0×10^{-11}	2.0×10^{-9}	2.0×10^{-7}
colon	8.8×10^{-11}	8.8×10^{-9}	1.8×10^{-8}	3.3×10^{-11}	3.3×10^{-9}	6.6×10^{-9}

(xi) Joint dosimetry/late effects question: The number of radiation-induced cancer deaths up to 40 years following exposure in a population of a hundred million persons (5×10^7 male, 5×10^7 female) each of whom inhales 10 K Bq of the radionuclides specified.

Nuclide	Physical Form	Chemical Form	Cancer Type	Number of Cancers		
				5%	50%	95%
Pu-239	1 μm AMAD	Oxide	Lung	NR	NR	NR
			Bone	NR	NR	NR
			Liver	NR	NR	NR
			Leukemia	NR	NR	NR
			All cancers	NR	NR	NR
Sr-90	1 μm AMAD	Oxide	Lung	NR	NR	NR
			Bone	NR	NR	NR
			Leukemia	NR	NR	NR
			All cancers	NR	NR	NR



EXPERT F

Rationale for Elicitation Questions

General Remarks

The general approach to answer these questions is as follows

- (i) to look for direct human data
- (ii) to look for direct animal data
- (iii) to look for human data for similar situations (for example chemically similar elements)
- (iv) to look for animal data for similar situations
- (v) to use results of model calculations.

Based on these observations central values (50% percentiles) have been chosen. In the case of (i), if many direct human data are available, the lower (5%) and the upper (95%) percentiles can be estimated from the data available. If data are sparse, the percentiles must be estimated—possibly by using data of the type (ii)–(v). For the cases (ii) to (iv) the approach is similar but with broader ranges of uncertainty - increasing with increasing numbers - due to uncertainties in extrapolating from animals to humans or to the specific given situation. In the case of (v) the range of uncertainty is dependent on the data on which the model is based.

In many cases, due to lack of data the percentiles and central values can only be rough estimates. In these cases the percentiles and central values chosen here are often rounded values (in many cases rounded to one significant figure) to avoid the impression these values would be known to a high precision.

The questions are answered here for the “international reference man” as the average of both sexes, not considering regional differences. If the sex-specific differences, however, are very large a broader range of uncertainty has been chosen. Also if regional differences may be very obvious (for example iodine deficiency influencing the iodine biokinetics) a broader range of uncertainty has been chosen. Generally members of the public are considered here, not workers. Adults are considered here to be “mature adults” who may be older than 25 years in some cases (for example for the biokinetics of strontium and plutonium).

In most cases ICRP recently reviewed human and animal data which can be used to determine the biokinetic behavior

(ICRP, 1989; 1993; 1994; 1995a; 1995c). Often results of this review are quoted here without giving explicitly the references of all the informations contained. For details the respective ICRP publications have to be consulted.

In some cases also extreme values such as 100% retention have been estimated as percentiles. If for example such a value has been chosen as 95% percentile it is assumed that at least 5% of experiments will result in this extreme value in this situation.

Inhalation

1. Average ventilation rates, $L \text{ min}^{-1}$, assuming a normal daily (24 h) mix of activities (combined male, female average)?

For ventilation rates several data for humans of different sexes, ages and races are available; they are reviewed in ICRP Publication 66 (ICRP, 1994). Average values of male and female adults given in ICRP Publication 66 are taken here as central values. Available data show no large differences between various ethnic groups because differences in tidal volume and respiratory frequency are smoothing the resulting differences in ventilation rates; therefore altogether a rather narrow range of uncertainty is assumed here. Outdoor workers have not been considered because they are only a rather small fraction of the population. The range of uncertainty is expected to be larger in the direction of higher values because there may be a trend to have a mix of activities more on the active side. Also older assumptions (ICRP, 1975; UNSCEAR, 1982; ICRP, 1987a) are within the given range of uncertainty. For children less data are available and additionally there is a trend to larger uncertainties due to different stages of growth; therefore broader ranges of uncertainties are given here.

For deposition (questions 2–4) there are some experimental data available but only for the adult Caucasian male and for a limited range of particle sizes (1–10 μm). The many gaps of knowledge have been filled by a deposition model (ICRP, 1994) giving regional deposition values for members of different ages engaged in various activities. This model has been used here as a basis for the assessment of central values and percentiles for deposition. For this informations given in Annex F of ICRP Publication 66 (ICRP, 1994) and results obtained by the LUDEP code (NRPB) have been used. Hygroscopic growth has not been considered here.

2. Total initial deposition in the respiratory tract, % of total amount inhaled, normal daily (24 hr) mix of activities?

Deposition values for different activities given in Annex F of ICRP Publication 66 (ICRP, 1994) together with the daily mix of activities from ICRP Publication 71 (ICRP, 1995c)—which is very similar to that of Publication 66—have been taken as central values for the AMADs of 1 and 10 μm . For the AMAD of 0.1 μm deposition values due to results obtained by the LUDEP code have been chosen. For the shape, particle density and aerosol-size dispersion standard ICRP assumptions have been taken for choosing the central values. Due to these data there are no large differences between sexes but in some cases for different activities. Therefore for an AMAD of 1 μm a larger range of uncertainties has been chosen compared to an AMAD of 10 μm . The range of uncertainty is rather narrow for the 10 μm AMAD because this value is partly based on human experiments and seems to be rather independent of changes of other parameters. For the AMAD of 0.1 μm a larger range of uncertainty has been chosen because the deposition fraction is more strongly dependent on aerosol properties and because the model is much less based on experimental data in this case. For the child a broader range of uncertainty has been chosen because the model is extrapolating from adult data to children parameters.

3. Initial deposition in the extrathoracic (ET) region, % of total deposition in the respiratory tract? (This question also gives the initial deposition in the lung (tracheobronchial (TB) + pulmonary (AI) regions), since lung = total - ET)

For an AMAD of 1 μm this ratio is much higher for light exercise; therefore this parameter depends on the mix of activities very much and a higher uncertainty range than for a 10 μm AMAD—for which almost all deposition occurs in the ET region—has been given here. This range is broader for children because for this age group less information is available, similar to the 0.1 μm AMAD for which also a broader range of uncertainty has been assumed.

4. Initial deposition in the tracheobronchial (TB) region, % of the total deposition in the lung (TB + AI regions)?

Due to the deposition model of ICRP Publication 66 (ICRP, 1994) the differences due to different activity mixes may be largest for the 10 μm AMAD and lowest for the 1 μm AMAD. On the other hand less information is available for the 0.1 μm AMAD and for children resulting in broader uncertainty ranges for these cases.

5. Assuming completely insoluble particles (1 μm AMAD), retention in each respiratory tract region in mature adults as a % of the total initial deposition in the respiratory tract, as a function of time after deposition?

The answer to this question depends on deposition parameters as treated in questions 3 and 4, as well as mechanical transport rates.

Retention in tracheobronchial airways and in pulmonary region has been calculated with the DOSAGE code (developed by BfS) with the parameters given in ICRP Publication 66 (ICRP, 1994) assuming that clearance is due only to mechanical clearance and that no absorption occurs. It is assumed that no radioactive decay is occurring.

The retention values after 10 minutes are governed by deposition values. Here it is assumed that these values are identical to the respective deposition values and the central values and the ranges of uncertainty have been estimated with the results of questions 3 and 4, taking into account, however, that for example the 5% percentile for the lung deposition must be larger than the result obtained by the sum of the 5% percentiles of the pulmonary and tracheobronchial deposition. On the other hand, the 5% percentiles for pulmonary and tracheobronchial deposition must be larger than those calculated by the respective percentiles of questions 3 and 4.

For pulmonary retention due to recent human studies reported in ICRP Publication 66 (ICRP, 1994) it is assumed that no significant part of activity is removed fast. Therefore it is assumed that during the first day no activity is removed from the pulmonary region. Many human studies show that 50 days after deposition more than 60%, in most cases more than 75%, of the initial deposit is still retained. These studies show that 300 days after deposition more than 11–64% of the deposited material is retained. These results have been used for the assessment of the 1 month and 1 year values. For later times there are no results of human experiments but only information from assessments of accidents which, however, is not easy to interpret. Therefore for 10 years after deposition a rather broad range of uncertainty is assumed here.

As in the discussion of the pulmonary region, the tracheobronchial airways are considered here more generally as the tracheobronchial region, including lymph nodes which are important at times of one or more years after deposition. The amount of activity transferred to lymph nodes and the assumption that insoluble material

remains in the lymph nodes is considered to be rather uncertain resulting in broad ranges of uncertainty at long times after deposition.

6. By what factors would you expect the median values to be different in 5 year old children (1=no difference)?

The activity deposited in these regions for the 5-year-old child is about 0.8 of that for the adult. No age-dependence of clearance seems to be known; for this reason it is assumed here that clearance is independent of age. Therefore a fraction of 0.8 is assumed for all times after deposition.

7. Absorption (dissolution and transfer) to blood in adults, % of the total initial deposition (1 μm AMAD particles) in the respiratory tract, considering the range of chemical forms which you judge most likely to be inhaled after an accident? (To include absorption to blood from the gastrointestinal tract for material cleared from the respiratory tract via the mucociliary escalator and swallowed). The usual assumption has been that elements will be inhaled in oxide form, apart from I in elemental form.

For these calculations again no physical decay has been considered. Calculations have been performed by the DOSAGE code (BfS) using respiratory tract data given in ICRP Publication 71 (ICRP, 1995c).

Due to the results of question 3, the 5% percentile of activity deposited in the extrathoracic region is 50%. About 40% of the activity deposited in the extrathoracic region is assumed to be deposited in the anterior nasal passage where no absorption to blood and no transfer to the gastrointestinal tract is assumed. Therefore not more than 80% as a maximum of the activity deposited in the respiratory tract is expected to be ready for absorption.

To consider the fraction of the activity cleared to the gastrointestinal tract which is absorbed to blood the results of question 9—as far as applicable—are used here. For more unsoluble material, however, the general approach of ICRP Publication 71 (ICRP, 1995c) that Type M (S) material has no f_1 -value larger than 0.1 (0.01) has been considered.

For strontium a study with accidental inhalation of a mixture of fresh fission products showed Type M behavior, few *in vitro* studies with similar materials showed a behavior between F and M. Here a behavior similar to M with a tendency to F has been assumed. The differences

between Type F and Type M behavior are especially at short times after inhalation very large leading to a wide range of uncertainty.

Iodine studies show very fast and complete absorption. For elemental iodine almost complete uptake is assumed with a fraction of 10% in the ET₁ compartment (the anterior nasal passage) which is not absorbed to blood and not transferred to the gastrointestinal tract; therefore a broader range of uncertainty has been chosen due to lack of knowledge of the fractions of elemental iodine and iodine aerosols.

The absorption behavior of caesium is assumed to be similar to that of iodine in particulate form. However, a fraction of caesium may be retained longer in the lung. This is endorsed by some *in vitro* studies indicating a Type M behavior. Therefore a Type F behavior with a slight tendency to Type M is assumed here. The 5% percentile reflects the possibility of a Type M behavior.

Plutonium in the oxide form tends to have a Type S behavior. For ²³⁸Pu due to radiolytic fragmentation, however, a larger fraction may be absorbed. Mainly for this reason the range of uncertainty also covers rather high values. If only ²³⁹Pu would be considered, the 95% percentile would be considerably lower.

Human and dog studies with ruthenium dioxide indicate a Type S behavior. *In vitro* studies of dissolution of particles released from the Chernobyl accident, however, were consistent with a Type M behavior of ruthenium. Here a Type M behavior of ruthenium with a tendency to Type S has been assumed.

A few human studies as well as several animal studies indicate a Type M behavior of cerium with the tendency to Type S. Such behavior has also been assumed here.

There seems to be only one human study on the tellurium absorption (following accidental inhalation in the form of hexafluoride gas) from the respiratory tract indicating a fast absorption. In the lack of specific information a behavior similar to Type M (with a tendency to faster absorption) is assumed here with a large range of uncertainty.

8. By what factors would you expect the median values to be different in 5 year old children (1=no difference)?

Because no changes of removal rates with age are known it is assumed that the removal from the respiratory tract due to mechanical clearance and absorption is independent of age. For the fractions of activity absorbed from the

gastrointestinal tract only for strontium slight age-dependent changes are known which do not influence the fraction of activity deposited in the respiratory tract absorbed to blood very much. Therefore differences in the absorbed fraction with age are due mainly to differences in deposition sites with age. The values given here are rounded values calculated by the DOSAGE code (BfS).

Ingestion

9. Absorption to blood as a fraction (f_1) of activity ingested, (considering chemical forms most likely to be ingested after an accident)?

As f_1 here it is considered the "apparent" fraction of gastrointestinal absorption obtained by external view. This may be different from the "true" fraction due to possible rapid secretion from systemic circulation into the intestine and subsequent re-absorption of parts of activity. The "apparent" fraction is considered here because most experiments determine the "apparent" fraction and because the re-absorption generally is not considered in the biokinetic models of ICRP.

Generally normal situations of health and nutritional status are assumed in the assessment here.

For strontium the review in ICRP Publication 56 (ICRP, 1989) shows several human studies giving results in the range of 0.15–0.45 for adults under normal circumstances and higher values for special circumstances as milk diets, overnight fasting etc. This range with a central value of 0.3 has been chosen here. For infants in the first year of life the NEA/OECD (NEA/OECD, 1988) approach assumes the double adult value. This approach is consistent with an about 35 year old human study and is adopted here. For children and adolescents animal experiments indicate a higher absorption than for adults. One human study (1960) could not endorse this. Therefore higher values with the same 5% percentile compared to adults have been chosen here.

For iodine many data are available all showing an almost complete absorption. For all ages a range of 0.8–0.99 with a central value of 0.9 has been chosen here.

Also for caesium many studies show an almost complete absorption. For this element, however, there are studies (Henrichs et al., 1989) indicating a little bit lower values. Therefore, a 5% quantile of 0.7 has been chosen for the adult and the 5-year-old child.

For biologically incorporated plutonium there are few human studies indicating values between 10^{-4} and 10^{-3} . A comparison of animal data for biologically incorporated plutonium and plutonium oxide leads to the assumption of an f_1 value of 10^{-5} for PuO_2 . There is no indication of different values for 5-year-old children but a higher value could be possible. For 3-month-old infants 10-fold values are assumed here due to experimental results obtained by administration of plutonium oxide to neonatal animals and due to the general NEA/OECD approach (NEA/OECD, 1988).

Systemic distribution and retention

The answers to these questions are based mainly on calculations with the DOSAGE code (BfS) with the models of ICRP (ICRP, 1993; 1995a).

Strontium, Plutonium, Cerium, Tellurium

10. Considering the total amount reaching blood (as if administered intravenously as a single injection). % retained in liver and skeleton (bone + bone marrow), as a function of time after entry into blood?

For strontium only distribution and retention in the skeleton is considered here. The contribution of liver is considered to be negligible within the uncertainty range of distribution and retention of strontium in the skeleton. As central values model predictions of ICRP Publication 67 (ICRP, 1993) have been taken here. It is assumed that deposition in skeleton is similar for all alkaline earth elements. For calcium there have been autoradiographic measurements of activity on bone surfaces in adult humans to whom ^{45}Ca has been injected shortly before death. These studies show considerable variability around the time of 1 day after injection and even more about 1 week after injection. This does not necessarily prove a larger variability in the whole skeleton but a larger variability for the time of 1 week is assumed here. For 1 month and especially for 1 year a lower uncertainty is assumed here because there are some human strontium retention data very close to model results. For later times after injection no information seems to be available and larger ranges of uncertainty are assumed here. For 5-year-old children a higher bone uptake is assumed but, due to beagle results and due to a higher bone turnover rate, also a higher rate of loss of activity from skeleton.

During the last years most studies suggest a total amount of 80% of systemic activity to be distributed to liver and skeleton for actinides. Therefore, for plutonium this value has been assumed here but it has been considered a delayed

uptake from circulation. The retention in liver and skeleton is assumed to be in the range of half-times given in ICRP Publications 30 (ICRP, 1979) (50–100 years) and 48 (ICRP, 1986) (20–50 years) which is consistent with the systemic model of ICRP Publication 67 (ICRP, 1993). Similarly to strontium a higher rate of loss of activity from the skeleton is assumed for 5-year-old children.

There are chemical similarities of cerium and the actinides. Also cerium deposits mainly in skeleton and liver with about 80% of systemic activity. Beagle experiments, however, show an elimination from the body a little bit faster than for plutonium. Because there is not much information on long-term retention available for cerium a broad uncertainty interval is assumed.

Limited animal data show that shortly after administration activity of tellurium is concentrated mainly in the kidneys and the thyroid. In later periods activity concentration in skeleton has been observed to be highest. ICRP Publications 30 (ICRP, 1979) and 67 (ICRP, 1993) assume that 25% of systemic tellurium is deposited in skeleton. It is assumed here that this fraction is hardly higher than 50% because of a large fraction of early excretion observed in animal experiments. Retention time in skeleton is considered to be high compared to the physical half-life of important tellurium isotopes. For children no data seem to be available and a larger uncertainty range is assumed here.

11. Retention in the skeleton, % total retention in liver + skeleton (bone + marrow), as a function of time after entry into blood?

For strontium no data seem to be available indicating an enhanced liver uptake; therefore, liver is not considered to be a source organ in the systemic model of ICRP Publication 67. Here a slightly higher liver concentration than in other soft tissue has been assumed (double of the average concentration) because data for radium, another alkaline earth element, show a higher liver uptake; furthermore the contribution of blood to liver activity has been considered here using the fractional blood content in liver given in ICRP Publication 53 (ICRP, 1987b).

The same method as for strontium also has been applied here to tellurium for which no specific liver uptake is known. But due to lack of data there are larger ranges of uncertainty for tellurium.

In ICRP Publication 30 (ICRP, 1979) a fraction of 50% of skeleton and liver uptake for plutonium has been assumed to be distributed in skeleton but later results indicate a fraction

of 5/8 for adults, based on a reanalysis of the Langham data. This value is rounded here to 65% but the old value of 50% is still in the range of uncertainty. This fraction is expected to increase slowly with time because of the longer retention in skeleton compared to liver. However, the model of ICRP Publication 67 (ICRP, 1993) gives a slower fraction 10 years after injection. This is because the model assumes a later decrease of liver activity because the former assumptions underestimated autopsy results obtained from occupationally exposed humans. For 5-year-old children as well as for older children a fraction of 3/4 of activity deposited in skeleton compared to that deposited in skeleton and liver is assumed on the basis of animal results. In this case this fraction is assumed to diminish with time due to the larger rate of bone turnover. Because of lack of information available especially for long times after injection the range of uncertainty is rather broad for children.

For cerium ICRP Publication 30 (ICRP, 1979) and 67 (ICRP, 1993) gave values of 25% and 37.5%, respectively, for adults for all times after intake. An investigation of the chemical relationships of lanthanides resulted in a value of 44% (EURATOM, 1995). A central value of 40% has been assumed therefore for adults allowing an increase with time because retention in skeleton may be longer than in liver. Animal experiments indicate that there is an enhanced skeleton uptake for children. Therefore a value of 67.5% is assumed by ICRP (ICRP, 1989). It may be possible that activity in skeleton is decreasing faster due to faster bone turnover in children as it is probably for plutonium. Therefore the range of uncertainty is increasing here very much at long times after intake.

12. Retention of plutonium on endosteal bone surfaces (considering a 10 μm depth of bone mineral) as a % of total skeletal retention, as a function of time after entry into blood?

Plutonium is known as a "surface-seeker" which prefers to stay at bone surfaces. Therefore the simplifying assumption of ICRP Publication 30 (ICRP, 1979) was that all skeletal activity of plutonium would reside on bone surfaces. Nevertheless, it is expected that with time some amount of plutonium will be incorporated within the bone volume and may be translocated to bone marrow. Very soon after injection it is expected that (nearly) all of plutonium in skeleton will still be at the bone surfaces. Later on it is expected that up to 1/3 will be incorporated into the bone volume based on physiological bone formation rates. This ratio will be higher for 5-year-old children because of faster

bone restructuring processes in childhood and during adolescence.

13. *Retention of plutonium on trabecular surfaces, % total endosteal surface retention as a function of time after entry into blood?*

Trabecular surfaces are considered here as bone surfaces of trabecular bone in the definition by ICRP, i.e., bones with a surface to volume ratio greater than 60 cm^{-1} .

Trabecular surfaces are about 50% of bone surfaces (ICRP, 1995b). According to beagle data plutonium prefers to be deposited at sites with active marrow which are preferably at trabecular compared to cortical bones. Therefore a higher fraction for trabecular bone surfaces than 50% is assumed here for adults. Removal from bone surfaces is assumed to be due to bone formation and resorption. These processes are considered to be faster in trabecular bone than in cortical bone leading to smaller fractions on trabecular bone surfaces compared to the overall bone surfaces activity.

For non-adults the distribution on bone surfaces may be more uniform leading to an estimated fraction of 50% for the trabecular surfaces. The retention half-times for cortical and trabecular bone surfaces are more similar for 5-year-old children than for adults, therefore it is assumed that this ratio will not change very much with time, but it is expected that the uncertainty will increase.

14. *Retention of plutonium in red bone marrow as a % total skeletal retention, as a function of time after entry into blood?*

For this assessment not only activity transferred to red bone marrow as a source organ from the bone compartments due to bone restructuring processes is considered—as it is described by the biokinetic model of ICRP Publication 67 (ICRP, 1993)—but also a fraction (due to ICRP Publication 53 (ICRP, 1987b) 3.65% for the adult and 2.75% for the 5-year-old child) of blood which is contained in red bone marrow which is important—and leads to rather high values—for times shortly after intake. This approach, however, is not very consistent within this report because for the estimation of activity in the skeleton the blood has not been considered here. But for the whole skeleton the contribution of blood is not very important.

For younger ages there is a faster loss of activity from bone surfaces and therefore a more rapid uptake to bone marrow.

Further on there is a larger fraction of activity transferred from bone surfaces to red bone marrow leading to a larger fraction of activity retained within the marrow.

Ruthenium, Caesium

15. *Considering the total amount reaching blood (as if administered intravenously as a single injection), % total retention (whole body or systemic) as a function of time after entry into blood?*

For ruthenium there are animal retention data from studies with rats, mice, monkeys, and dogs. The interspecies intercomparison showed good agreement; only the long half-time varied with species. Because of lack of human data a broad range of uncertainty is assumed here. For children no data seem to be available. As central values therefore the adult values are assumed here but with a tendency of the range of uncertainty to lower values.

For caesium, however, there are lots of human data available for all age groups. The adult data show, however, remarkable differences between males and females in some studies. Intermediate values have been assumed here as central values with a tendency to higher values for the range of uncertainty. The values given for 5 years after injection are of a more theoretical nature: They are expected to be so low that they would not be detectable in normal circumstances.

Iodine

16. *Considering the total amount of I reaching blood (as if administered intravenously as a single injection), % retained in the thyroid as a function of time after entry into blood?*

Also for iodine many human data are available. The iodine uptake by the thyroid (and subsequently the thyroid retention values) are very strongly dependent on the stable iodine content of the diet which varies regionally very much. For the derivation of central values the “international reference man” considered by ICRP Publications 30 (ICRP, 1979) and 67 (ICRP, 1993) is considered here with an average iodine uptake by the thyroid of 30%. This is not suitable for special populations, mainly for those with iodine deficiency such as in Germany. This variation is partly considered here by broader ranges of uncertainty which are considered to be even larger for children.

Dose coefficients

17. Intake dose coefficients, absorbed dose to specified organs or tissue per unit activity inhaled or ingested (committed doses), Gy Bq⁻¹?

In contrast to the items asked by the other questions dose coefficients are no observable quantities but are computed with results from observations discussed above. The answers to these questions should be results of probabilistic calculations using the results of the questions before. It may be that those calculations may show that the range of uncertainty is overestimated here in some cases. In general it must be kept in mind that the range of uncertainty for the effective dose would be smaller because in many cases a higher value for one organ implies a lower value for another organ.

Generally dose coefficients given in ICRP Publications 67 (ICRP, 1993) for ingestion and 71 (ICRP, 1995c) for inhalation are taken here to be reference values which sometimes, however, are modified due to deviant central parameters assumed in this report. The percentiles derived in the questions above are also taken to estimate the percentiles for the dose coefficients. However, it has been taken into account that there is less probability that all parameters are extreme values. On the other hand additional uncertainties of dose coefficients due to the uncertainties of further parameters not asked above such as dosimetric data (S-values) have been considered here.

As in ICRP Publication 67 (ICRP, 1993) the colon dose is considered here to be the mass weighted dose to upper and lower large intestine. Because absorbed doses are asked here and no equivalent doses the average lung dose has been considered here as lung dose and not a dose weighted by apportionment factors due to the radiosensitivity of several lung tissues as proposed by ICRP Publication 66 (1994) and given by ICRP Publication 71 (1995c).

For inhalation of strontium intermediate values between Type F and M have been assumed as central values. The range of uncertainties for the lungs is determined by the uncertainties of lung deposition and retention as well as (to a lower extent) S-values. For bone surface and bone marrow the fraction of lung activity absorbed to blood and bone uptake and retention values determine the range of uncertainty. The central values and the upper percentiles for the red bone marrow dose are considered to be a little bit higher because of the possibility that there may be some activity uptake to bone marrow which is not considered by the model.

For ingestion instead of the fraction absorbed to blood from the lungs the gastrointestinal absorption fraction f_1 is considered. The colon dose is mainly dependent on $(1-f_1)$ but also on a larger uncertainty concerning transit times.

For iodine not the whole range of variability in the thyroid retention has been taken into account because of the correlation of the concurrent parameters thyroid uptake and thyroid mass resulting in similar doses for different uptake values. For inhalation because of lack of knowledge equal parts of iodine in particulate and elemental form have been assumed for the derivation of central values. This lack of knowledge leads to a higher range of uncertainty.

For the inhalation of tellurium because of lack of knowledge equal parts of tellurium in particulate and vapor form have been assumed for the derivation of central values. The largest uncertainty for the thyroid dose is due to the uncertainty of the fraction absorbed from the lung into the systemic circulation. The uncertainty for the colon dose is larger to lower values due to uncertainties of Type F/M/S absorption. A possible Type S absorption has no such large influence on the colon dose because of the relative short half-life of ^{132}Te and its decay product ^{132}I .

For caesium a probabilistic calculation of ingestion dose coefficients* showed an uncertainty range from the 2.5% percentile to the 97.5% percentile of a factor of a little bit more than a factor of 2. These calculations, however, were based on calculations for male adults whereas here the range of uncertainty has been chosen to be larger because of the additional uncertainty of lower retention half-times for females who also have been considered here for the derivation of central adult values. Altogether the range of uncertainty is not very large because of a correlation of the concurrent parameters biological half-time and total body mass.

The largest uncertainties for cerium dose coefficients are due to the uncertainty of lung retention and the uncertainty of the fraction absorbed from the lung to the systemic circulation, respectively, which is important especially for the 5% percentile. For red bone marrow a larger 95% percentile has been chosen because of a possible transfer of cerium to red bone marrow which has not been considered by the model of ICRP (ICRP, 1993).

* D. Gilby. 1995. Probabilistische Berechnung von Ingestionsdosiskoeffizienten; Quantifizierung der Zuverlässigkeit. Zwischenbericht 1994. Personal communication, interim report of a research project.

For plutonium the dose coefficients given in ICRP Publications 67 and 71 have been divided by 20 because the dose to source organs as considered here is mainly due to α -radiation with a radiation weighting factor of 20. For inhalation a Type S behavior has been assumed. The largest uncertainties for ingestion are generally due to the f_1 value which however hardly influences the colon dose. For red bone marrow the uncertainties are considered to be a little bit higher because of the uncertainty of the fraction transferred to red bone marrow. For the colon dose the uncertainties are mainly due to different transit times which may be lower for younger ages. For inhalation the uncertain fraction absorbed from the lung to systemic circulation influences much the uncertainty of dose coefficients. For ingestion biologically incorporated plutonium has been considered here.

Seed variables

To answer the seed variables the same methods as described above have been used. To give uncertainty ranges for experimental results of excretion measurements it has been considered that individual excretion measurement results can vary very much from one day to the next leading to high uncertainties of single experiments. Furthermore for terbium, for example, almost no knowledge of biokinetic and excretion data was available for me. Also the influence of pregnancy on the biokinetics of baboons was unknown to me. In Study 5 "other tissues" is considered to be total body without respiratory tract and without contents of urinary bladder and GI tract. The fractions of caesium and cobalt contained in the dust were unknown which also leads to a higher uncertainty.

References

EURATOM. 1995. EURATOM Nuclear fission safety programme 1990–94; Radiation protection research action 1992–94; Progress report 1992–93. Report EUR 16003, p. 242.

Henrichs, K., Paretzke, H.G., Voigt, G. and Berg, D. 1989. Measurement of Cs absorption and retention in man. *Health Phys.* 57 571–578.

ICRP. 1975. *Report of the Task Group on Reference Man.* ICRP Publication 23. Oxford: Pergamon Press.

ICRP. 1979. *Limits for Intakes of Radionuclides by Workers.* ICRP Publication 30, Part 1. Oxford: Pergamon Press.

ICRP. 1986. *The Metabolism of Plutonium and Related Elements.* ICRP Publication 48. Oxford: Pergamon Press.

ICRP. 1987a. *Lung Cancer Risk from Indoor Exposures to Radon Daughters.* ICRP Publication 50. Oxford: Pergamon Press.

ICRP. 1987b. *Radiation Dose to Patients from Radiopharmaceuticals.* ICRP Publication 53. Oxford: Pergamon Press.

ICRP. 1989. *Age-dependent Doses to Members of the Public from Intake of Radionuclides: Part 1.* ICRP Publication 56. Oxford: Pergamon Press.

ICRP. 1993. *Age-dependent Doses to Members of the Public from Intake of Radionuclides: Part 2.* ICRP Publication 67. Oxford: Elsevier Science Ltd.

ICRP. 1994. *Human Respiratory Tract Model for Radiological Protection.* ICRP Publication 66. Oxford: Elsevier Science Ltd.

ICRP. 1995a. *Age-dependent Doses to Members of the Public from Intake of Radionuclides: Part 3.* ICRP Publication 69. Oxford: Elsevier Science Ltd.

ICRP. 1995b. *Basic Anatomical and Physiological Data for use in Radiological Protection: The Skeleton.* ICRP Publication 70. Oxford: Elsevier Science Ltd.

ICRP. 1995c. *Age-dependent Doses to Members of the Public from Intake of Radionuclides: Part 4 Inhalation Dose Coefficients.* ICRP Publication 71. Oxford: Elsevier Science Ltd.

NEA/OECD. 1988. Committee on Radiation Protection and Public Health. Report of an Expert Group on Gut Transfer Factors. NEA/OECD Report, Paris.

UNSCEAR. 1982. *Ionizing Radiations: Sources and Biological Effects.* United Nations Scientific Committee on the Effects of Atomic Radiation, Publication N.E.82.IX.8., New York.

Question 1. Average ventilation rates, $L \text{ min}^{-1}$, assuming a normal daily (24h) mix of activities (combined male, female average).

	5 year old children			Mature Adults		
	5%	50%	95%	5%	50%	95%
$L \text{ min}^{-1}$	4	6	10	12	14	18

Question 2. Total initial deposition in the respiratory tract, % of total amount inhaled, normal daily (24 hr) mix of activities.

	0.1 μm AMAD			1 μm AMAD			10 μm AMAD		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Mature Adults	25	45	65	35	45	55	70	75	80
5 year old children				30	50	70			

Question 3. Initial deposition in the extrathoracic region, % of total deposition in the respiratory tract? (This question also gives the initial deposition in the lung (tracheobronchial (TB) + pulmonary (AI) regions), since lung = total - ET).

	0.1 μm AMAD			1 μm AMAD			10 μm AMAD		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Mature Adults	3	15	40	50	70	90	80	93	98
5 year old children				45	75	95			

Question 4. Initial deposition in the tracheobronchial (TB) region, % of the total deposition in the lung (TB + AI regions).

	0.1 μm AMAD			1 μm AMAD			10 μm AMAD		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Mature Adults	10	25	45	10	20	35	20	45	70
5 year old children				8	20	45			

Question 5. Assuming completely insoluble particles (1 μm AMAD), retention in each respiratory tract region in mature adults as a % of the total initial deposition in the respiratory tract, as a function of time after deposition.

	10 min			1 hr			1 day		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Tracheobronchial airways	1.5	6.6	15	1	6.2	15	0.3	3.6	12
Pulmonary (AI) region	7.5	24	40	7	24	40	7	24	40
	1 month			1 year			10 years		
Tracheobronchial airways	0.1	1.4	6	0.001	0.1	1	0.0001	0.1	1
Pulmonary (AI) region	5	20	40	1	12	25	0.1	2	15

Question 6. By what factors would you expect the median values to be different in 5 year old children (1=no difference).

	10 min	1 hr	1 day	1 month	1 year	10 years
Tracheobronchial airways	0.8	0.8	0.8	0.8	0.8	0.8
Pulmonary (AI) region	0.8	0.8	0.8	0.8	0.8	0.8

Question 7. Absorption (dissolution and transfer) to blood in adults, % of the total initial deposition (1 μm AMAD particles) in the respiratory tract.

	1 hr			1 day			1 week		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Sr	3	10	50	7	20	60	8	20	60
I	50	70	90	65	80	90	65	80	90
Cs	20	50	70	30	60	80	30	60	80
Pu	0.01	0.05	2	0.02	0.07	2	0.02	0.09	3
Ru	0.06	2	8	0.2	3	10	0.2	3	12
Ce	0.05	1	6	0.05	1	7	0.1	2	8
Te	0.1	5	50	0.5	10	60	0.7	10	60
1 month			1 year			10 years			
Sr	10	25	60	15	35	60	20	40	60
I	65	80	90	65	80	90	65	80	90
Cs	30	60	80	30	65	80	30	70	80
Pu	0.06	0.2	5	0.2	0.6	12	0.05	2	15
Ru	0.5	5	15	1	10	25	2	15	30
Ce	0.2	3	10	0.5	8	20	1	12	25
Te	1	15	60	2	25	60	3	30	60

Question 8. Factors by which the median values would be different in 5 year old children (1=no difference).

5 year old children	1 hr	1 day	1 week	1 month	1 year	10 years
Sr	0.9	0.95	0.95	0.95	0.9	0.9
I	0.98	0.98	0.98	0.98	0.98	0.98
Cs	0.95	0.95	0.95	0.95	0.95	0.95
Pu	0.9	0.9	0.9	0.9	0.8	0.8
Ru	0.9	0.95	0.9	0.9	0.85	0.85
Ce	0.9	0.9	0.9	0.85	0.85	0.85
Te	0.9	0.95	0.95	0.95	0.9	0.9

Question 9. Absorption to blood as a fraction (f_1) of activity ingested.

	3 month old infants			5 year old children			Adults		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Sr	0.3	0.6	0.9	0.15	0.4	0.6	0.15	0.3	0.45
I	0.8	0.9	0.99	0.8	0.9	0.99	0.8	0.9	0.99
Cs	0.8	0.9	0.99	0.7	0.9	0.99	0.7	0.9	0.99
PuO_2^*	5×10^{-6}	10^{-4}	10^{-3}	10^{-6}	10^{-5}	5×10^{-4}	10^{-6}	10^{-5}	10^{-4}
Pu biol†	0.0005	0.005	0.01	0.0001	0.0005	0.005	0.0001	0.0005	0.001

* Refractory oxide
 † "Biologically incorporated"

Question 10. Considering the total amount reaching blood (as if administered intravenously as a single injection). % retained in liver and skeleton (bone + bone marrow), as a function of time after entry into blood.

	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
Skeleton and liver, 1 day						
Sr	20	45	80	15	30	60
Pu	20	35	90	20	35	90
Ce	20	45	90	20	45	90
Te	3	15	65	7	15	50
Skeleton and liver, 1 week						
Sr	15	30	80	10	20	60
Pu	50	75	90	60	75	90
Ce	40	80	90	50	80	90
Te	5	25	65	10	25	50
Skeleton and liver, 1 month						
Sr	10	30	60	8	20	40
Pu	50	80	90	60	80	90
Ce	40	80	90	50	80	90
Te	2	25	65	5	25	50
Skeleton and liver, 1 year						
Sr	5	16	30	5	13	25
Pu	40	75	90	50	80	90
Ce	30	75	90	50	75	90
Skeleton and liver, 10 years						
Sr	0.1	2	15	2	6.5	15
Pu	20	55	80	35	70	85
Ce	10	40	80	20	40	80
Skeleton and liver, 50 years						
Sr	0.01	0.3	8	0.3	1.6	8
Pu	5	20	50	20	50	70
Ce	0.1	2	40	0.5	2	60

Question 11. Retention in the skeleton, % total retention in liver + skeleton (bone + marrow), as a function of time after entry into blood.

5 year old children			Adults			
	5%	50%	95%	5%	50%	95%
Skeleton, 1 day						
Sr	85	95	99.5	90	98	99.8
Pu	50	75	90	50	65	75
Ce	30	65	90	20	40	60
Te	70	85	98	70	85	98
Skeleton, 1 week						
Sr	80	96	99.5	85	97	99.8
Pu	50	75	90	50	65	75
Ce	30	65	90	20	40	60
Te	85	97	99.8	75	97	99.9
Skeleton, 1 month						
Sr	80	99	100	85	99	99.9
Pu	50	75	90	50	65	75
Ce	30	65	90	20	40	60
Te	85	98	99.8	75	98	99.9
Skeleton, 1 year						
Sr	85	99.9	99.99	90	99.9	99.9
Pu	40	70	85	50	65	80
Ce	30	65	90	20	40	65
Skeleton, 10 years						
Sr	92	99.99	100	95	99.9	100
Pu	10	45	85	40	60	85
Ce	15	65	95	20	40	75
Skeleton, 50 years						
Sr	95	99.99	100	97	99.99	100
Pu	20	65	95	50	70	95
Ce	7	65	95	20	40	90

Question 12. Retention of plutonium on endosteal bone surfaces (considering a 10 μm depth of bone mineral) as a % of total skeletal retention, as a function of time after entry into blood.

		5 year old children			Adults		
		5%	50%	95%	5%	50%	95%
Endosteal bone surface	1 day	95	99	100	95	99.9	100
	1 week	85	98	100	90	99	100
	1 month	80	92	99	90	99	100
	1 year	30	50	85	75	90	98
	10 years	20	45	80	60	77	90
	50 years	10	65	90	45	67	85

Question 13. Retention of plutonium on trabecular surfaces, % total endosteal surface retention as a function of time after entry into blood.

		5 year old children			Adults		
		5%	50%	95%	5%	50%	95%
Trabecular bone surface	1 day	40	50	80	45	60	80
	1 week	40	50	80	45	60	80
	1 month	35	50	80	40	60	80
	1 year	30	45	80	30	55	75
	10 years	10	35	80	10	25	60
	50 years	5	20	70	5	15	50

Question 14. Retention of plutonium in red bone marrow as a % total skeletal retention, as a function of time after entry into blood.

		5 year old children			Adults		
		5%	50%	95%	5%	50%	95%
Red bone marrow	1 day	0.5	5	15	1	7	15
	1 week	0.01	0.6	3	0.05	0.4	2
	1 month	0.1	2	10	0.2	0.7	3
	1 year	0.5	9	25	0.5	3	6
	10 years	0.1	5	20	0.1	2	6
	50 years	0.01	1.3	10	0.01	1	5

Question 15. Considering the total amount reaching blood (as if administered intravenously as a single injection), % total retention (whole body or systemic) as a function of time after entry into blood.

	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
1 day						
Ru	60	87	99	65	87	98
Cs	90	98	99.5	90	98	99.5
1 week						
Ru	30	67	90	40	67	90
Cs	60	75	90	70	85	95
1 month						
Ru	10	40	70	20	40	70
Cs	15	30	60	60	75	95
1 year						
Ru	1	15	50	5	15	50
Cs	0.001	0.2	3	1	5	20
5 years						
Ru	0.01	6	25	1	6	25
Cs	10^{-3}	10^{-5}	0.01	10^{-4}	0.007	0.1

Question 16. Considering the total amount of Iodine reaching blood (as if administered intravenously as a single injection), % retained in the thyroid as a function of time after entry into blood.

	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
1 day						
	15	28	60	20	28	50
1 week						
	12	25	55	18	28	50
1 month						
	7	15	35	15	24	45
3 months						
	1	4	10	5	16	30

Question 17. Intake dose coefficients, absorbed dose to specified organs or tissue per unit activity inhaled or ingested (committed equivalent doses), Gy Bq⁻¹.

5 year old children			Adult		
5%	50%	95%	5%	50%	95%
⁹⁰Sr, inhalation, 1 μm AMAD					
lung	1×10^{-9}	2×10^{-7}	5×10^{-7}	1×10^{-9}	1×10^{-7}
bone marrow	2×10^{-8}	2×10^{-7}	5×10^{-7}	3×10^{-8}	2×10^{-7}
bone surface	5×10^{-8}	3×10^{-7}	9×10^{-7}	6×10^{-8}	2.5×10^{-7}
⁹⁰Sr, ingestion					
colon	7×10^{-9}	4.5×10^{-8}	2×10^{-7}	4×10^{-9}	1.3×10^{-8}
bone marrow	6×10^{-8}	5×10^{-7}	1×10^{-6}	5×10^{-8}	3×10^{-7}
bone surface	6×10^{-8}	6.4×10^{-7}	2×10^{-6}	5×10^{-8}	4.1×10^{-7}
¹³¹I, inhalation, 1 μm AMAD + vapor (decide proportions)					
thyroid	8×10^{-7}	1.3×10^{-6}	2×10^{-6}	2×10^{-7}	3×10^{-7}
¹³¹I, ingestion					
thyroid	1.2×10^{-6}	2×10^{-6}	3×10^{-6}	3×10^{-7}	4×10^{-7}
¹³²Te, inhalation, 1 μm AMAD + vapor (decide proportions)					
lung	3×10^{-10}	6×10^{-9}	1×10^{-8}	1.5×10^{-10}	3×10^{-9}
thyroid	6×10^{-9}	2×10^{-7}	2×10^{-6}	1.5×10^{-9}	4×10^{-8}
colon	1.5×10^{-9}	1×10^{-8}	4×10^{-8}	7×10^{-10}	3×10^{-9}
¹³⁷Cs, inhalation, 1 μm AMAD					
lung	1.5×10^{-9}	3×10^{-9}	5×10^{-9}	2×10^{-9}	4×10^{-9}
colon	2×10^{-9}	5.4×10^{-9}	9×10^{-9}	2×10^{-9}	4.5×10^{-9}
stomach	1.5×10^{-9}	3.1×10^{-9}	5×10^{-9}	2×10^{-9}	4×10^{-9}
bone marrow	1.5×10^{-9}	2.9×10^{-9}	5×10^{-9}	2×10^{-9}	4×10^{-9}
¹³⁷Cs, ingestion					
lung	5×10^{-9}	8.4×10^{-9}	1.5×10^{-8}	8×10^{-9}	1.2×10^{-8}
colon	8×10^{-9}	1.6×10^{-8}	3×10^{-8}	8×10^{-9}	1.4×10^{-8}
stomach	5×10^{-9}	9.5×10^{-9}	1.5×10^{-8}	8×10^{-9}	1.2×10^{-8}
bone marrow	5×10^{-9}	8.3×10^{-9}	1.5×10^{-8}	8×10^{-9}	1.2×10^{-8}
¹⁴⁴Ce, inhalation, 1 μm AMAD					
bone surface	2×10^{-7}	6×10^{-7}	1.5×10^{-7}	1.2×10^{-6}	3×10^{-7}
lung	3×10^{-9}	1×10^{-7}	5×10^{-7}	9×10^{-10}	2×10^{-8}
bone marrow	2×10^{-9}	8×10^{-8}	4×10^{-7}	4×10^{-10}	1×10^{-8}
liver	3×10^{-9}	1×10^{-7}	6×10^{-7}	3×10^{-9}	6×10^{-8}
²³⁹Pu, inhalation, 1 μm AMAD					
bone surface	1.5×10^{-6}	8×10^{-6}	5×10^{-5}	4×10^{-6}	9×10^{-6}
bone marrow	6×10^{-8}	5×10^{-7}	3×10^{-6}	8×10^{-8}	4.5×10^{-7}
liver	4×10^{-7}	2×10^{-6}	1×10^{-5}	5×10^{-7}	2×10^{-6}
lung	7×10^{-6}	2×10^{-5}	5×10^{-5}	3×10^{-6}	9×10^{-6}
²³⁹Pu, ingestion					
bone surface	5×10^{-8}	3.6×10^{-7}	5×10^{-6}	6×10^{-8}	4.2×10^{-7}
bone marrow	2×10^{-9}	3×10^{-8}	4×10^{-7}	2×10^{-9}	2×10^{-8}
liver	2×10^{-8}	1.3×10^{-7}	1.5×10^{-6}	1×10^{-8}	8.5×10^{-8}
colon	1.5×10^{-9}	7.8×10^{-9}	2.5×10^{-8}	7×10^{-10}	2.4×10^{-9}

(xi) Joint dosimetry/late effects question: The number of radiation-induced cancer deaths up to 40 years following exposure in a population of a hundred million persons (5×10^7 male, 5×10^7 female) each of whom inhales 10 K Bq of the radionuclides specified.

Nuclide	Physical Form	Chemical Form	Cancer Type	Number of Cancers		
				Quantile	5%	50%
Pu-239	1 μm AMAD	Oxide	Lung			
			Bone			
			Liver			
			Leukemia			
			All cancers			
Sr-90	1 μm AMAD	Oxide	Lung			
			Bone			
			Leukemia			
			All cancers			

EXPERT G

Question 1. Average ventilation rates, $L \text{ min}^{-1}$, assuming a normal daily (24h) mix of activities (combined male, female average).

	5 year old children			Mature Adults		
	5%	50%	95%	5%	50%	95%
$L \text{ min}^{-1}$	5.0	7.5	9.0	8	12	14

Question 2. Total initial deposition in the respiratory tract, % of total amount inhaled, normal daily (24 hr) mix of activities.

	0.1 μm AMAD			1 μm AMAD			10 μm AMAD		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Mature Adults	24	46	52	26	46	62	67	76	88
5 year old children				28	55	74			

Question 3. Initial deposition in the extrathoracic region, % of total deposition in the respiratory tract? (This question also gives the initial deposition in the lung (tracheobronchial (TB) + pulmonary (AI) regions), since lung = total - ET).

	0.1 μm AMAD			1 μm AMAD			10 μm AMAD		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Mature Adults	10	14	15	24	64	73	64	92	94
5 year old children				39	79	87			

Question 4. Initial deposition in the tracheobronchial (TB) region, % of the total deposition in the lung (TB + AI regions).

	0.1 μm AMAD			1 μm AMAD			10 μm AMAD		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Mature Adults	20	24	27	17	19	22	34	38	42
5 year old children				21	23	27			

Question 5. Assuming completely insoluble particles (1 μm AMAD), retention in each respiratory tract region in mature adults as a % of the total initial deposition in the respiratory tract, as a function of time after deposition.

	10 min			1 hr			1 day		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Tracheobronchial airways	6.7	6.8	6.9	5.9	6.5	6.8	1.2	3.9	6.3
Pulmonary (AI) region	30	30	30	30	30	30	29	29	29
	1 month			1 year			10 years		
Tracheobronchial airways	0.12	1.5	4.5	4×10^{-3}	9×10^{-3}	0.21	2×10^{-4}	4×10^{-4}	8×10^{-4}
Pulmonary (AI) region	24	25	26	10	15	19	0.78	2.4	5.8

Question 6. By what factors would you expect the median values to be different in 5 year old children (1=no difference).

3-mo-old	10 min	1 hr	1 day	1 month	1 year	10 years
Tracheobronchial airways	0.75	0.74	0.74	0.74	0.52	0.48
Pulmonary (AI) region	0.48	0.48	0.48	0.48	0.48	0.49

5-year-old	10 min	1 hr	1 day	1 month	1 year	10 years
Tracheobronchial airways	0.71	0.70	0.70	0.70	0.58	0.56
Pulmonary (AI) region	0.56	0.56	0.56	0.56	0.56	0.56

Question 7. Absorption (dissolution and transfer) to blood in adults, % of the total initial deposition (1 μm AMAD particles) in the respiratory tract.

	1 hr			1 day			1 week		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Sr	0.6	5.6	16	1.7	9.2	22	1.9	10	27
I	41	56	64	61	70	72	65	70	72
Cs	31	46	64	45	60	72	46	60	72
Pu	0.05	0.5	5.4	0.08	0.6	5.6	0.10	0.7	6.4
Ru	0.6	5.5	17	1.4	7.4	19	1.6	8.3	20
Ce	0.5	5.4	11	0.6	5.6	12	0.7	6.4	15
Te	0.07	5.6	54	0.4	9.2	59	0.5	10	59
	1 month			1 year			10 years		
Sr	2.5	13	36	6.6	26	42	12	28	42
I	65	70	72	65	70	72	65	70	72
Cs	48	62	72	56	66	72	57	66	72
Pu	0.17	1.2	9.2	0.78	5.0	22	2.5	11	23
Ru	2.1	11	22	6.1	24	32	12	26	33
Ce	1.2	9.2	24	5.0	22	32	11	23	32
Te	0.6	13	59	1.3	26	59	3.2	28	59

Question 8. Factors by which the median values would be different in 5 year old children (1=no difference).

5 year old children	1 hr	1 day	1 week	1 month	1 year	10 years
Sr	0.87	0.82	0.96	0.84	0.70	0.70
I	0.87	0.86	0.91	0.91	0.91	0.91
Cs	0.87	0.85	0.94	0.92	0.90	0.90
Pu	0.87	0.81	0.81	0.69	0.59	0.58
Ru	0.87	0.82	0.90	0.78	0.67	0.66
Ce	0.87	0.81	0.80	0.71	0.62	0.62
Te	0.87	0.83	0.97	0.84	0.70	0.70

Question 9. Absorption to blood as a fraction (f_1) of activity ingested.

	3 month old infants			5 year old children			Adults		
	5%	50%	95%	5%	50%	95%	5%	50%	95%
Sr	0.2	0.4	0.7	0.15	0.27	0.5	0.1	0.2	0.4
I	0.98	1.0	1.0	0.95	1.0	1.0	0.9	1.0	1.0
Cs	0.8	0.95	1.0	0.7	0.9	1.0	0.7	0.9	1.0
PuO_2^*	1×10^{-5}	1×10^{-4}	1×10^{-3}	1×10^{-6}	1×10^{-5}	1×10^{-4}	1×10^{-6}	1×10^{-5}	1×10^{-4}
Pu biol†	3×10^{-4}	3×10^{-3}	1×10^{-2}	1×10^{-4}	5×10^{-4}	2×10^{-3}	1×10^{-4}	5×10^{-4}	2×10^{-3}

* Refractory oxide

† "Biologically incorporated"

Question 10. Considering the total amount reaching blood (as if administered intravenously as a single injection). % retained in liver and skeleton (bone + bone marrow), as a function of time after entry into blood.

	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
Skeleton and liver, 1 day						
⁸⁹ Sr	.35	.46	.60	.25	.33	.45
⁹⁰ Sr	.35	.47	.60	.25	.33	.45
²³⁹ Pu	.20.	.38	.75	.20	.38	.75
¹⁴¹ Ce	.50	.72	.90	.60	.72	.85
¹⁴⁴ Ce	.50	.73	.90	.60	.73	.85
¹³² Te	.05	.15	.60	.07	.15	.45
Skeleton and liver, 1 week						
⁸⁹ Sr	.24	.31	.41	.15	.20	.26
⁹⁰ Sr	.26	.34	.45	.17	.22	.29
²³⁹ Pu	.50	.76	.90	.50	.76	.90
¹⁴¹ Ce	.53	.67	.80	.54	.67	.77
¹⁴⁴ Ce	.61	.77	.93	.66	.77	.88
¹³² Te	.09	.27	.95	.13	.27	.80
Skeleton and liver, 1 month						
⁸⁹ Sr	.14	.20	.28	.09	.13	.17
⁹⁰ Sr	.20	.30	.40	.13	.20	.26
²³⁹ Pu	.60	.80	.95	.60	.80	.95
¹⁴¹ Ce	.32	.41	.50	.33	.41	.47
¹⁴⁴ Ce	.61	.78	.93	.62	.78	.89
¹³² Te	.06	.27	.95	.13	.27	.80
Skeleton and liver, 1 year						
⁸⁹ Sr	0.08	0.12	0.2	0.06	.088	.15
⁹⁰ Sr	.10	.17	.25	.09	.13	.20
²³⁹ Pu	.60	.77	.95	.65	.80	.95
¹⁴¹ Ce	.40	.60	.80	.50	.60	.70
¹⁴⁴ Ce	.49	.73	.97	.58	.73	.85
Skeleton and liver, 10 years						
⁸⁹ Sr	0	0	0	0	0	0
⁹⁰ Sr	.0013	.0034	.013	.025	.064	.13
²³⁹ Pu	.40	.54	.75	.50	.73	.85
¹⁴¹ Ce	0	0	0	0	0	0
¹⁴⁴ Ce	.04	.37	.85	.07	.37	.75
Skeleton and liver, 50 years						
⁸⁹ Sr	0	0	0	0	0	0
⁹⁰ Sr	.00000003	.00000025	.000002	.0033	.016	.066
²³⁹ Pu	.20	.36	.60	.30	.49	.75
¹⁴¹ Ce	0	0	0	0	0	0
¹⁴⁴ Ce	0	0	0	0	0	0

Question 11. Retention in the skeleton, % total retention in liver + skeleton (bone + marrow), as a function of time after entry into blood.

	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
Skeleton, 1 day						
Sr	95	97	99	93	95	99
Pu	40	72	95	30	61	90
Ce	30	63	75	20	37	50
Te	90	96	100	80	96	100
Skeleton, 1 week						
Sr	95	97	99	93	95	97
Pu	50	75	90	45	63	80
Ce	30	63	75	20	37	50
Te	90	97	100	80	97	100
Skeleton, 1 month						
Sr	98	99	100	98	99	100
Pu	50	75	90	45	63	80
Ce	30	63	75	20	37	50
Te	90	98	100	80	98	100
Skeleton, 1 year						
Sr	99	100	100	99	100	100
Pu	50	70	90	45	62	80
Ce	30	63	75	20	37	50
Skeleton, 10 years						
Sr	99	100	100	99	100	100
Pu	20	32	70	45	59	80
Ce	20	63	75	25	37	50
Skeleton, 50 years						
Sr	--	--	--	99	100	100
Pu	35	63	90	45	68	90
Ce	--	--	--	--	--	--

Question 12. Retention of plutonium on endosteal bone surfaces (considering a 10 μm depth of bone mineral) as a % of total skeletal retention, as a function of time after entry into blood.

		5 year old children			Adults		
		5%	50%	95%	5%	50%	95%
Endosteal bone surface	1 day	.88	.94	.96	.88	.93	.98
	1 week	.86	.98	.99	.89	.9999	.9999
	1 month	.68	.92	.95	.87	.99	.9999
	1 year	.14	.50	.65	.66	.91	.96
	10 years	.12	.38	.50	.43	.77	.86
	50 years	.25	.68	.76	.23	.67	.77

Question 13. Retention of plutonium on trabecular surfaces, % total endosteal surface retention as a function of time after entry into blood.

		5 year old children			Adults		
		5%	50%	95%	5%	50%	95%
Trabecular bone surface	1 day	.35	.50	.65	.50	.60	.70
	1 week	.35	.50	.65	.50	.60	.70
	1 month	.32	.50	.66	.49	.60	.70
	1 year	.16	.46	.77	.42	.55	.68
	10 years	.18	.46	.84	.12	.27	.55
	50 years	.09	.19	.45	.08	.17	.41

Question 14. Retention of plutonium in red bone marrow as a % total skeletal retention, as a function of time after entry into blood.

		5 year old children			Adults		
		5%	50%	95%	5%	50%	95%
Red bone marrow	1 day	.00019	.0006	.0014	.00004	.00016	.0004
	1 week	.0018	.0058	.014	.0004	.0015	.004
	1 month	.008	.028	.068	.002	.0074	.02
	1 year	.025	.12	.40	.008	.034	.092
	10 years	.015	.09	.39	.006	.022	.067
	50 years	.004	.013	.039	.004	.011	.033

Question 15. Considering the total amount reaching blood (as if administered intravenously as a single injection), % total retention (whole body or systemic) as a function of time after entry into blood.

	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
1 day						
Ru	.73	.84	.94	.76	.84	.92
Cs	.93	.97	.99	.95	.98	.99
1 week						
Ru	.44	.66	.83	.51	.66	.79
Cs	.51	.74	.88	.80	.87	.93
1 month						
Ru	.22	.39	.62	.28	.39	.55
Cs	.11	.32	.59	.64	.74	.84
1 year						
Ru	.09	.16	.22	.12	.16	.19
Cs	2.000E-0008	1.200E-0004	1.000E-0002	.028	.09	.20
5 years						
Ru	.0044	.056	.14	.016	.056	.11
Cs	1.000E-0012	2.000E-0012	5.000E-0010	3.000E-0008	9.000E-0006	5.000E-0004

Question 16. Considering the total amount of Iodine reaching blood (as if administered intravenously as a single injection), % retained in the thyroid as a function of time after entry into blood.

	5 year old children			Adults		
	5%	50%	95%	5%	50%	95%
1 day	.14	.27	.43	.15	.28	.43
1 week	.12	.26	.42	.15	.29	.44
1 month	.48	.15	.29	.11	.23	.39
3 months	.47	.35	.13	.59	.15	.28

Question 17. Intake dose coefficients, absorbed dose to specified organs or tissue per unit activity inhaled or ingested (committed equivalent doses), Gy Bq⁻¹.

5 year old children			Adult			
	5%	50%	95%	5%	50%	95%
⁹⁰Sr, inhalation, 1 μm AMAD						
lung	5.9×10^{-8}	4.6×10^{-7}	3.6×10^{-6}	3.9×10^{-8}	2.3×10^{-7}	1.4×10^{-6}
bone marrow	1.2×10^{-8}	8.4×10^{-8}	5.8×10^{-7}	1.7×10^{-8}	7.6×10^{-8}	3.5×10^{-7}
bone surface	6.7×10^{-8}	3.4×10^{-7}	1.7×10^{-6}	5.0×10^{-8}	1.7×10^{-7}	5.7×10^{-7}
⁹⁰Sr, ingestion						
colon	2.7×10^{-10}	1.9×10^{-9}	1.3×10^{-8}	9.3×10^{-11}	4.4×10^{-10}	2.1×10^{-9}
bone marrow	2.8×10^{-8}	1.8×10^{-7}	1.2×10^{-6}	2.8×10^{-8}	1.2×10^{-7}	5.2×10^{-7}
bone surface	9.3×10^{-8}	4.3×10^{-7}	2.0×10^{-6}	8.8×10^{-8}	2.7×10^{-7}	8.3×10^{-7}
¹³¹I, inhalation, 1 μm AMAD + vapor (decide proportions)						
thyroid	4.2×10^{-7}	1.2×10^{-6}	3.5×10^{-6}	1.1×10^{-7}	2.4×10^{-7}	5.3×10^{-7}
¹³¹I, inhalation, vapor						
thyroid	8.9×10^{-7}	2.1×10^{-6}	4.9×10^{-6}	2.4×10^{-7}	4.3×10^{-7}	7.8×10^{-7}
¹³¹I, ingestion						
thyroid	1.5×10^{-6}	3.6×10^{-6}	8.5×10^{-6}	4.1×10^{-7}	7.4×10^{-7}	1.3×10^{-6}
¹³²Te, inhalation, 1 μm AMAD						
lung	2.2×10^{-9}	1.9×10^{-8}	1.7×10^{-7}	1.1×10^{-10}	9.8×10^{-9}	5.0×10^{-8}
thyroid	4.3×10^{-11}	4.9×10^{-10}	5.6×10^{-9}	1.5×10^{-11}	8.7×10^{-11}	5.0×10^{-10}
colon	7.4×10^{-10}	4.3×10^{-9}	2.5×10^{-8}	3.8×10^{-10}	1.1×10^{-9}	3.2×10^{-9}
¹³²Te, inhalation, vapor						
lung	1.7×10^{-10}	9.9×10^{-10}	5.9×10^{-9}	1.1×10^{-10}	3.3×10^{-10}	9.8×10^{-10}
thyroid	4.8×10^{-10}	3.0×10^{-9}	1.9×10^{-8}	1.9×10^{-10}	6.0×10^{-10}	1.9×10^{-9}
colon	9.4×10^{-10}	4.3×10^{-9}	2.0×10^{-8}	4.8×10^{-10}	1.1×10^{-9}	2.5×10^{-9}
¹³⁷Cs, inhalation, 1 μm AMAD						
lung	3.7×10^{-9}	2.0×10^{-8}	1.1×10^{-7}	5.5×10^{-9}	2.0×10^{-8}	7.2×10^{-8}
colon	2.1×10^{-9}	6.4×10^{-9}	1.9×10^{-8}	2.3×10^{-9}	4.6×10^{-9}	9.2×10^{-9}
stomach	1.3×10^{-9}	4.0×10^{-9}	1.2×10^{-8}	2.2×10^{-9}	4.5×10^{-9}	9.0×10^{-9}
bone marrow	1.1×10^{-9}	3.4×10^{-9}	1.0×10^{-8}	2.3×10^{-9}	4.7×10^{-9}	9.4×10^{-9}
¹³⁷Cs, ingestion						
lung	2.7×10^{-9}	7.2×10^{-9}	1.9×10^{-8}	6.1×10^{-9}	1.1×10^{-8}	2.0×10^{-8}
colon	5.7×10^{-9}	1.5×10^{-8}	3.9×10^{-8}	8.0×10^{-9}	1.4×10^{-8}	2.5×10^{-8}
stomach	3.5×10^{-9}	9.5×10^{-9}	2.6×10^{-8}	7.2×10^{-9}	1.3×10^{-8}	2.3×10^{-8}
bone marrow	3.1×10^{-9}	8.3×10^{-9}	2.2×10^{-8}	7.2×10^{-9}	1.3×10^{-8}	2.3×10^{-8}
¹⁴⁴Ce, inhalation, 1 μm AMAD						
lung	7.8×10^{-8}	4.6×10^{-7}	2.7×10^{-6}	7.4×10^{-8}	2.2×10^{-7}	6.5×10^{-7}
bone marrow	2.9×10^{-8}	2.0×10^{-7}	1.4×10^{-6}	9.4×10^{-9}	3.2×10^{-8}	1.1×10^{-7}
liver	4.1×10^{-8}	2.7×10^{-7}	1.8×10^{-6}	3.7×10^{-8}	1.2×10^{-7}	3.9×10^{-7}

	5 year old children			Adult		
	5%	50%	95%	5%	50%	95%
^{239}Pu, inhalation, 1 μm AMAD						
bone surface	8.0×10^{-5}	6.3×10^{-4}	4.9×10^{-3}	1.5×10^{-4}	7.9×10^{-4}	4.1×10^{-3}
bone marrow	5.5×10^{-6}	4.6×10^{-5}	3.9×10^{-4}	6.8×10^{-6}	3.8×10^{-5}	2.1×10^{-4}
liver	3.2×10^{-5}	1.9×10^{-4}	1.1×10^{-3}	4.0×10^{-5}	1.6×10^{-4}	6.3×10^{-4}
lung	1.9×10^{-5}	1.3×10^{-4}	8.7×10^{-4}	1.4×10^{-5}	6.2×10^{-5}	2.8×10^{-4}
^{239}Pu, ingestion						
bone surface	5×10^{-7}	7.4×10^{-6}	4.2×10^{-5}	8×10^{-7}	8.2×10^{-6}	3.1×10^{-5}
bone marrow	4×10^{-8}	5.9×10^{-7}	3.9×10^{-6}	4×10^{-8}	3.9×10^{-7}	1.7×10^{-6}
liver	2×10^{-7}	2.6×10^{-6}	1.1×10^{-5}	2×10^{-7}	1.7×10^{-6}	4.7×10^{-6}
colon	3×10^{-9}	1.6×10^{-7}	6.9×10^{-7}	1×10^{-9}	4.7×10^{-8}	1.4×10^{-7}

EXPERT H

1. Considerations:

Daily distribution of time, hours

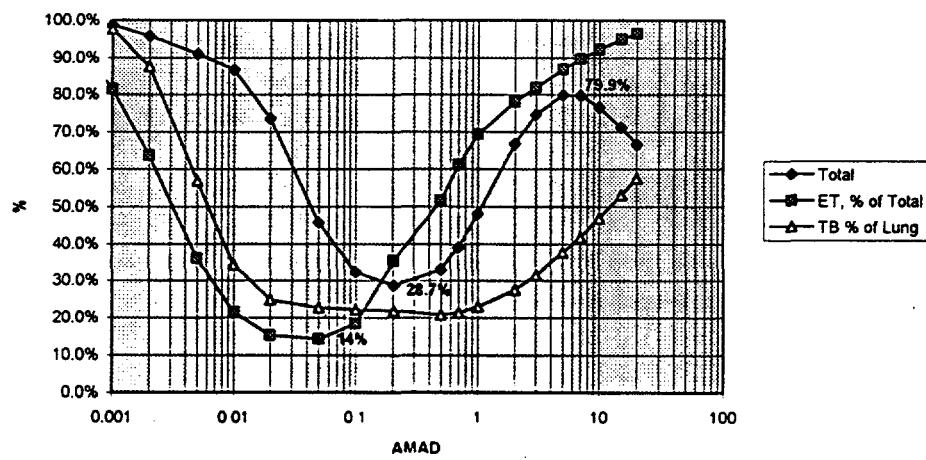
	5 year old			Adults		
	Min	50%	Max	Min	50%	Max
Sleep	13	11	8	9	8	7
Sitting	9	5	6	11	7	4
L. exercise	2	8	10	4	8.5	5
H. exercise				0	0.5	8

Ventilation rates, m³h⁻¹

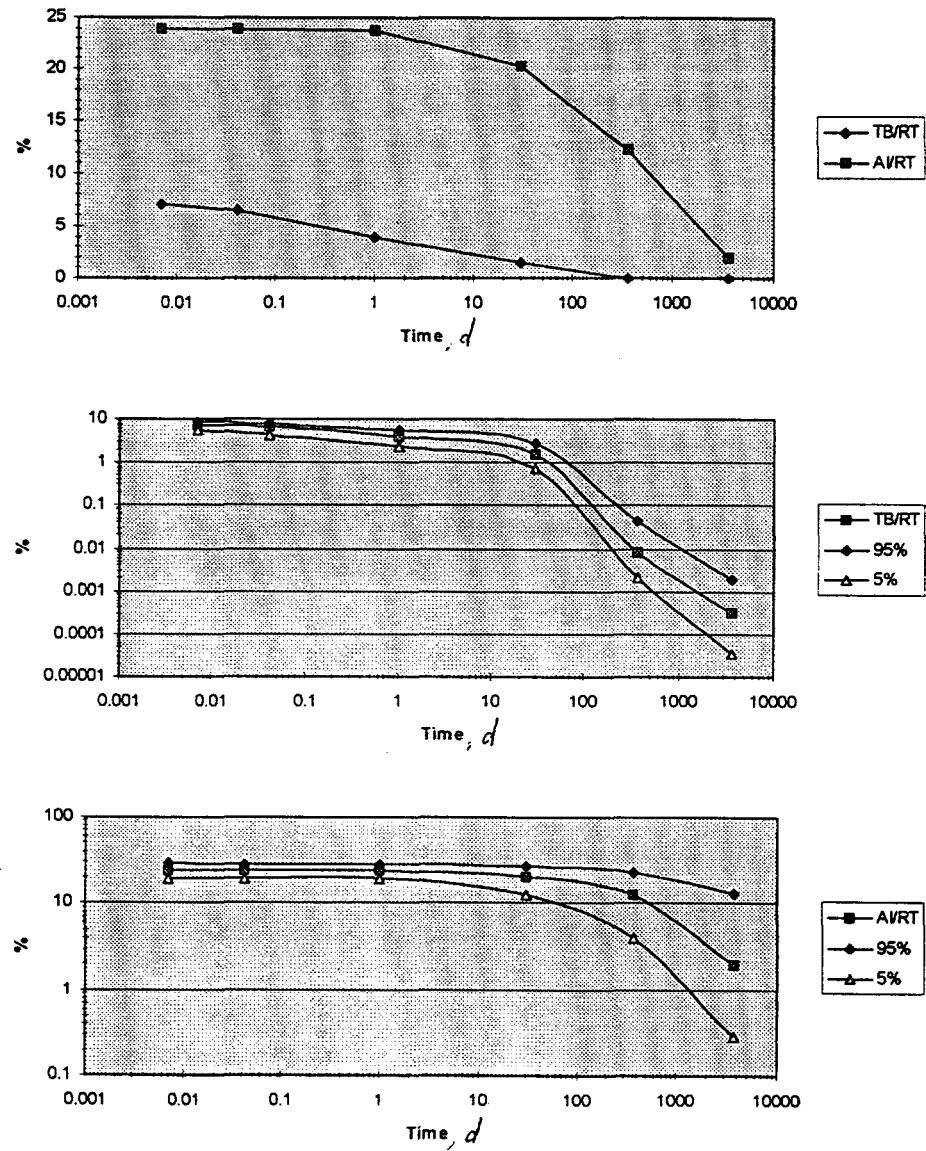
	5 year old			Adults		
	-20%	50%	20%	-20%	50%	20%
Sleep	0.19	0.24	0.29	0.31	0.385	0.46
Sitting	0.26	0.32	0.38	0.37	0.465	0.56
L. exercise	0.46	0.57	0.68	1.10	1.375	1.65
H. exercise				2.28	2.85	3.42

2. 3. and 4. Considerations:

- 50% - Ventilation rates as defined in 1.
- 95% - 9 g/cm³, depositions increased by 20%
- Different pattern of aerosol dispersion: monodisperse and log-normal with σ_g as defined in ICRP-66.
- Quality analysis of curves:



5, 6. Retention in TB and AI region as % of total deposition in RT.



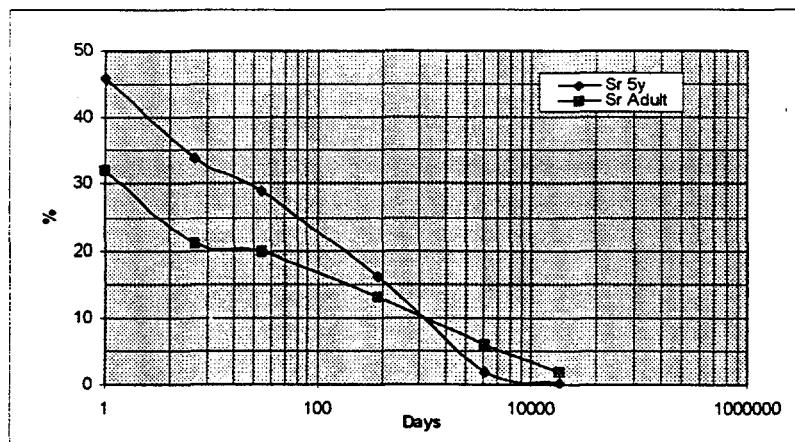
7.

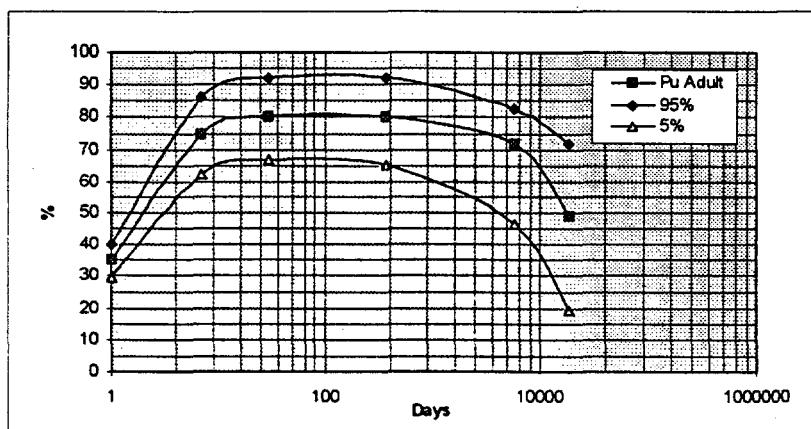
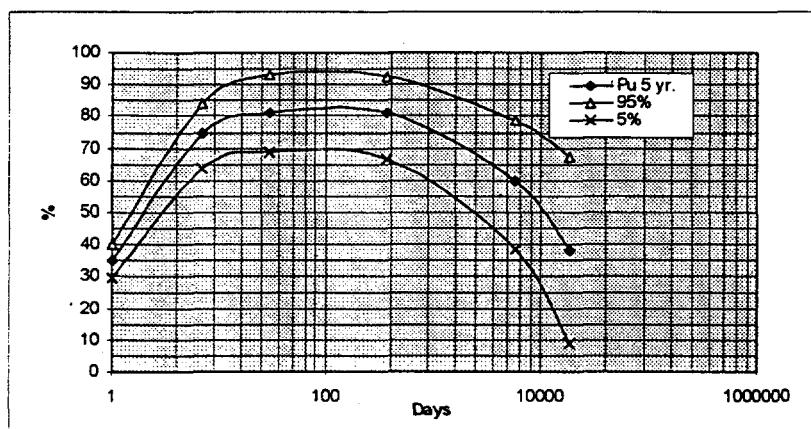
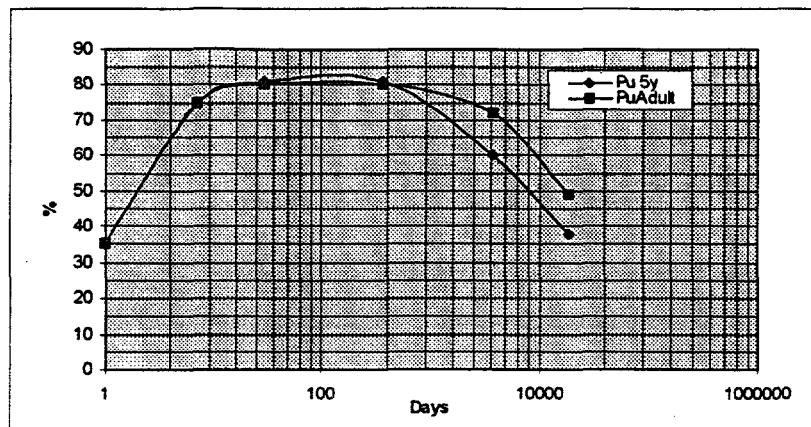
	5%	50%	95%
Sr	M, 0.1	F, 0.3	F, 0.4
I		F, 0.99	
Cs		F, 0.99	
Pu	½ on 50%	S, 1E-5	M, 5E-4
Ru	S, 5E-3	S, 1E-2	M, 5E-2
Ce	S, 1E-4	S, 5E-4	M, 1E-3
Te	M, 0.05	M, 0.1	F, 0.3

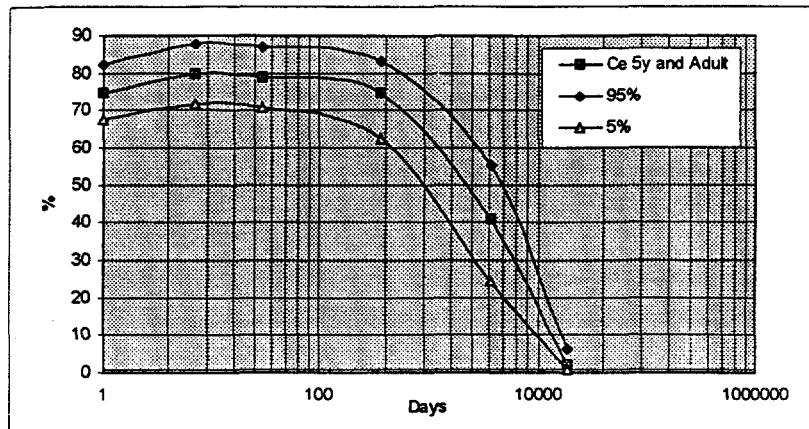
8. In contrast to ICRP-66 we assumed that there are age-dependence in translocation to blood. Short-term processes in 5 year old children are faster than in Adult. The long-term translocation is determine mainly by chemical forms of aerosol and independent on age.

9. Self-explanatory.

10.

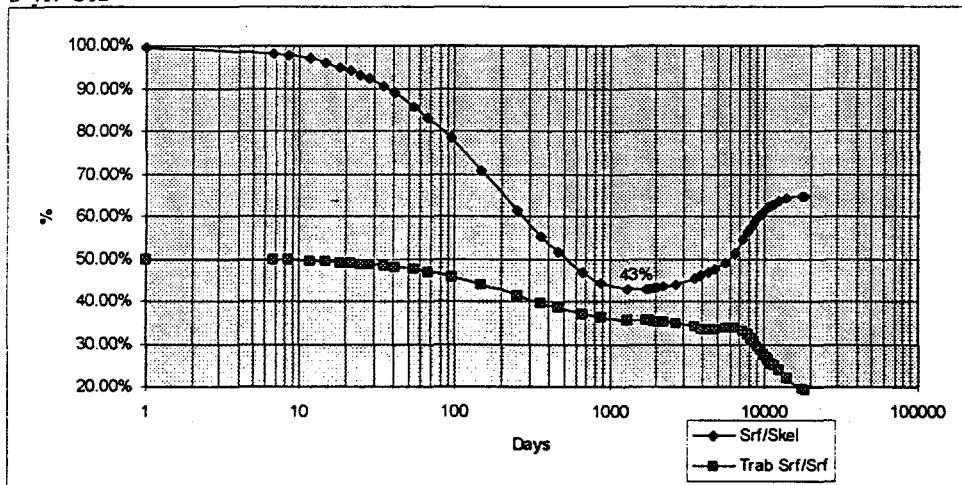




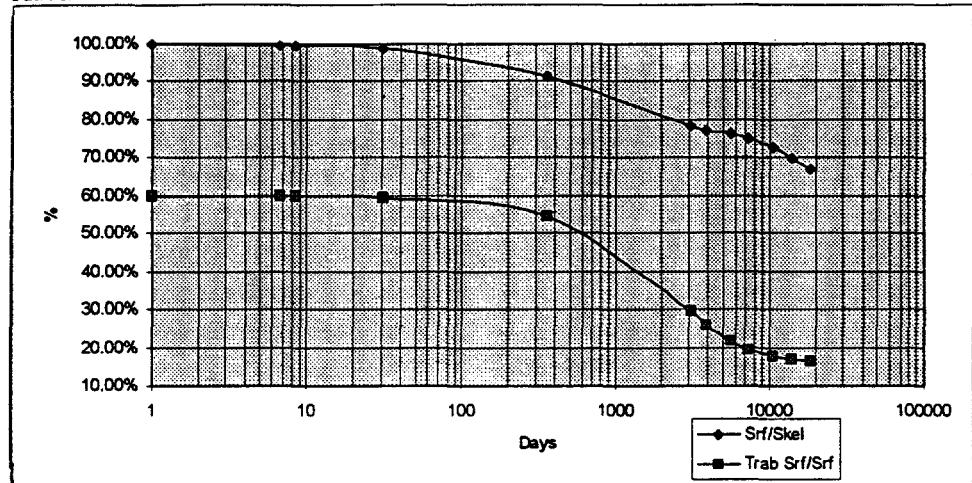


12,13.

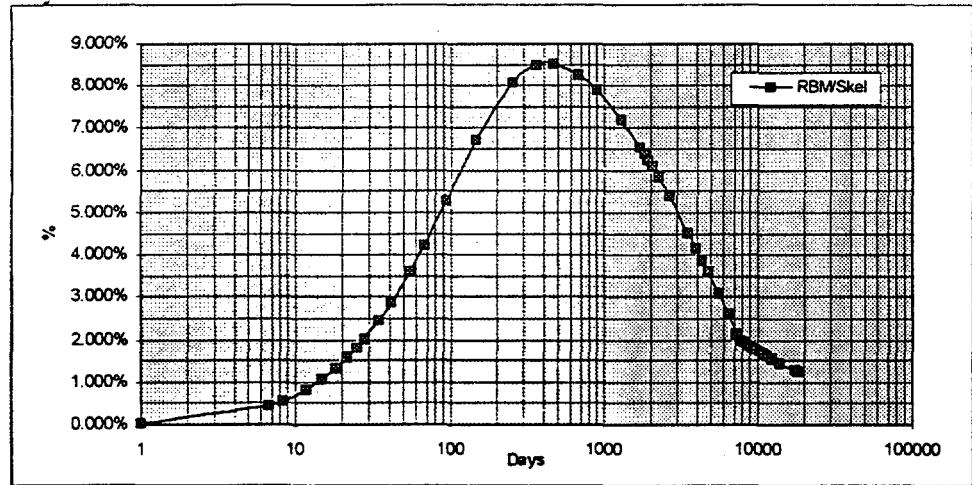
5 yr. Old



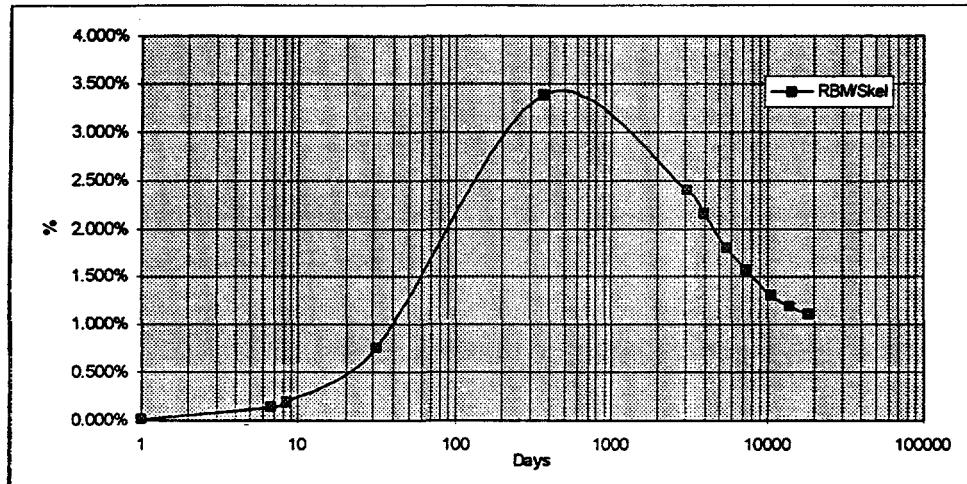
Adult



14.
5 yr.



Adult



15.

Cs:

5 yr. : $A_1 = 0.3-0.5$; $T_1 = 5 - 15$; $A_2 = 0.5-0.7$; $T_2 = 20 - 60$;
Adult : $A_1 = 0.05 - 0.2$; $T_1 = 1 - 3$; $A_2 = 0.8 - 0.95$; $T_2 = 60 - 160$.

17.

Sr-90: F, M, S; 5 yr.: $f_1 = 0.1-0.7$; Adult $f_1 = 0.05-0.4$.

I-131: F, 90% vapour.

Te-132: Adult and Child: 10% vapour. M, 0.1; F, 0.3; F, 0.4.

Cs-137: Adult and Child: F and M; f_1 as in table 9.

Ce-144: Adult and Child: S, $1E-4$; M, $5E-4$; M, $1E-3$.

Pu-239: Inhalation, Adult and Child: Y, $1E-5$; Y, $5E-4$; M, $1E-3$.

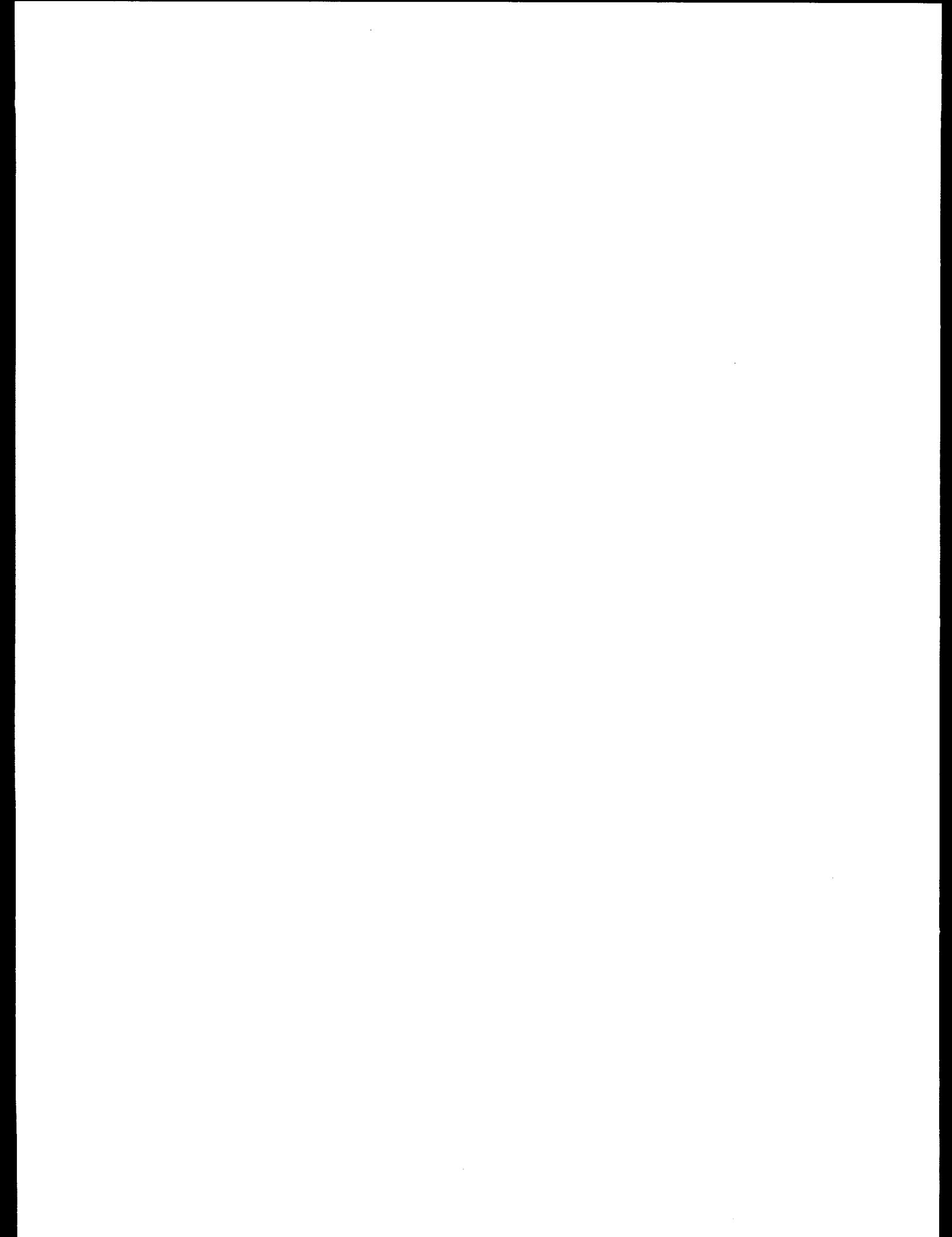
Pu-239: Ingestion, $1E-5$; $5E-4$; $1E-3$.

18.

- Dose variability lead to uncertainty factor no more than about 2, due to chemical form is defined (oxide).
- The age structure of population (see Annex), UNSCER risk coefficients and age-specific spontaneous mortality rates are used.
- The number of total cancer cases calculated as integral by age-dependent dose and risk factors.
- The spontaneous mortality rate is taken into account as age-dependent risk modification factor.

APPENDIX D

Short Biographies of the Internal Dosimetry Experts



Short Biographies of the Internal Dosimetry Experts

Michael R. Bailey, UK

Dr. Bailey graduated in Physics at the University of Cambridge in 1971. His first post was in the Health Physics Research Section at the Berkeley Nuclear Laboratories of the then Central Electricity Generating Board. Since 1976 he has worked for the National Radiological Protection Board and received his Ph.D. for work on clearance of inhaled particles in 1983. He has been Head of the Dose Assessments Department since 1990. He was a member of the ICRP Task Group which developed the new respiratory tract model (*ICRP Publication 66*). He is currently a member of the ICRP Task Group on Internal Dosimetry and coordinator of the EC Fourth Framework Programme Multi-national Project on "Inhalation of Radionuclides."

Dr. Bailey referred several questions to colleagues at NRPB and is grateful to them for their contributions:

Dr. J.D. Harrison
Ms. K.A. Jones
Dr. J.W. Marsh
Ms. S.L. Prosser

Keith F. Eckerman,* USA

Dr. Eckerman received his Ph.D. in Environmental Health Engineering from Northwestern University. His career has included various aspects of health physics while at Argonne National Laboratory, at the US Nuclear Regulatory Commission, and now at Oak Ridge National Laboratory. His work has focused on mathematical modeling of the behavior of radionuclides in the environment and within man, including the deposition of ionizing energy within the tissues of the body. Dr. Eckerman is a member of the US National Council on Radiation Protection and Measurements and a member of Committee 2 of the International Commission on Radiological Protection. He chairs the ICRP Task Groups on Dose Calculation and on Revision of Reference Man. He is the leader of the Dosimetry Research Group at Oak Ridge National Laboratory.

*. Eckerman and Leggett worked jointly.

Anthony C. James, USA

Dr. James graduated in Physics at London University in 1964 where he also earned his Ph.D. in Radiation Biology in 1969. He joined the National Radiological Protection Board, UK at its inception in 1971, where he specialized in research on the practical monitoring, experimental metabolism, and theoretical internal dosimetry aspects of protection against α -emitting transuranium elements and radon progeny. He continued his research career at Battelle Pacific Northwest Laboratories, Richland, WA (as a Staff Scientist 1988-91, and a Chief Scientist 1991-94). He now practices as an independent private consultant on Internal Dosimetry. Dr. James was a member of the US National Research Council's Scientific Panel which reported on "Comparative Dosimetry of Radon in Mines and Homes," and a member of the ICRP task groups that prepared the reports "Protection Against ^{222}Rn at Home and at Work" and "Human Respiratory Tract Model for Radiological Protection," respectively. He is presently a corresponding member of the ICRP's Task Group on Dose Calculations and is also a member of the US Interagency Nuclear Safety Review Panel/Biomedical and Environmental Effects Subpanel (INSRP/BEES), which advises the President on possible radiological consequences of spaceflight missions involving reactors or radionuclide heat sources.

Rich Leggett,* USA

Rich Leggett is a Research Staff Member of the Health Sciences Research Division of Oak Ridge National Laboratory (ORNL). After receiving his Ph.D. in mathematics from the University of Kentucky in 1972, he taught mathematics at the Ruhr University in Bochum, Germany and later at the University of Tennessee. He has been at ORNL since 1976. His main research interest is in mathematical modeling of physiological systems, with primary applications to biokinetics of radionuclides and radiopharmaceuticals. He is a member of the National Council on Radiological Protection's Committee on Dosimetry and Metabolism of Radionuclides and of four task groups of the International Commission on Radiological Protection (ICRP). He is the author of ICRP Publication 71, "Basic Anatomical and Physiological Data for Use in Radiological Protection: The Skeleton" and the developer of several of the ICRP's age-specific biokinetic models for radionuclides. In 1995 his paper "An age-specific kinetic model of Pb metabolism in humans" was

selected as Oak Ridge National Laboratory's Publication of the Year.

Ilya A. Likhtarev, Ukraine

Prof. Likhtarev, presently General Director of the Ukraine Radiation Protection Institute and Head of the Department of Dosimetry and Radiation Hygiene of the Ukraine Scientific Centre for Radiation Medicine, has over 30 years' experience in the field of external and internal dosimetric processes related to human beings exposed to nuclear radiation. He was head of the Dosimetric Physics Laboratory in the Leningrad Institute of Radiation Hygiene. He is member of the International Committee for Radiation Protection (ICRP) and is also professor of Radiation Physics. He is author of over 100 peer-reviewed publications.

Henry J. Métivier, France

Dr. Métivier graduated in 1962 in Paris and received his Ph.D. in Nuclear Chemistry in 1973. He is now Head of Research at the CEA/Nuclear Protection and Safety Institute in France. Since 1963 he has been working at the CEA/ IPSN in the fields of toxicology, radiation biology, cancer research and nuclear protection and safety. He was decorated "Chevalier dans l'ordre des Palmes Académiques" in 1992. He has been functioning as an expert for several other EC projects. He is a member of ICRP Committee 2.

Dietmar Noßke, Germany

Dr. Noßke earned his Ph.D. in Mathematics in 1984 at the Ludwig-Maximilians Universität in München. Currently he is head of the nuclear medicine section of the Institute of Radiation Hygiene, Federal Office for Radiation Protection. His current area of specialization is internal dosimetry. He has been working on the implementation of biokinetic and dosimetric models in computer codes to calculate dose coefficients and annual limits on intake. He assesses doses after incorporation of radionuclides in accidental situations, estimating doses by application of radiopharmaceuticals and developing biokinetic and dosimetric models, e.g., for the GI tract.

Nicholas D. Priest, UK

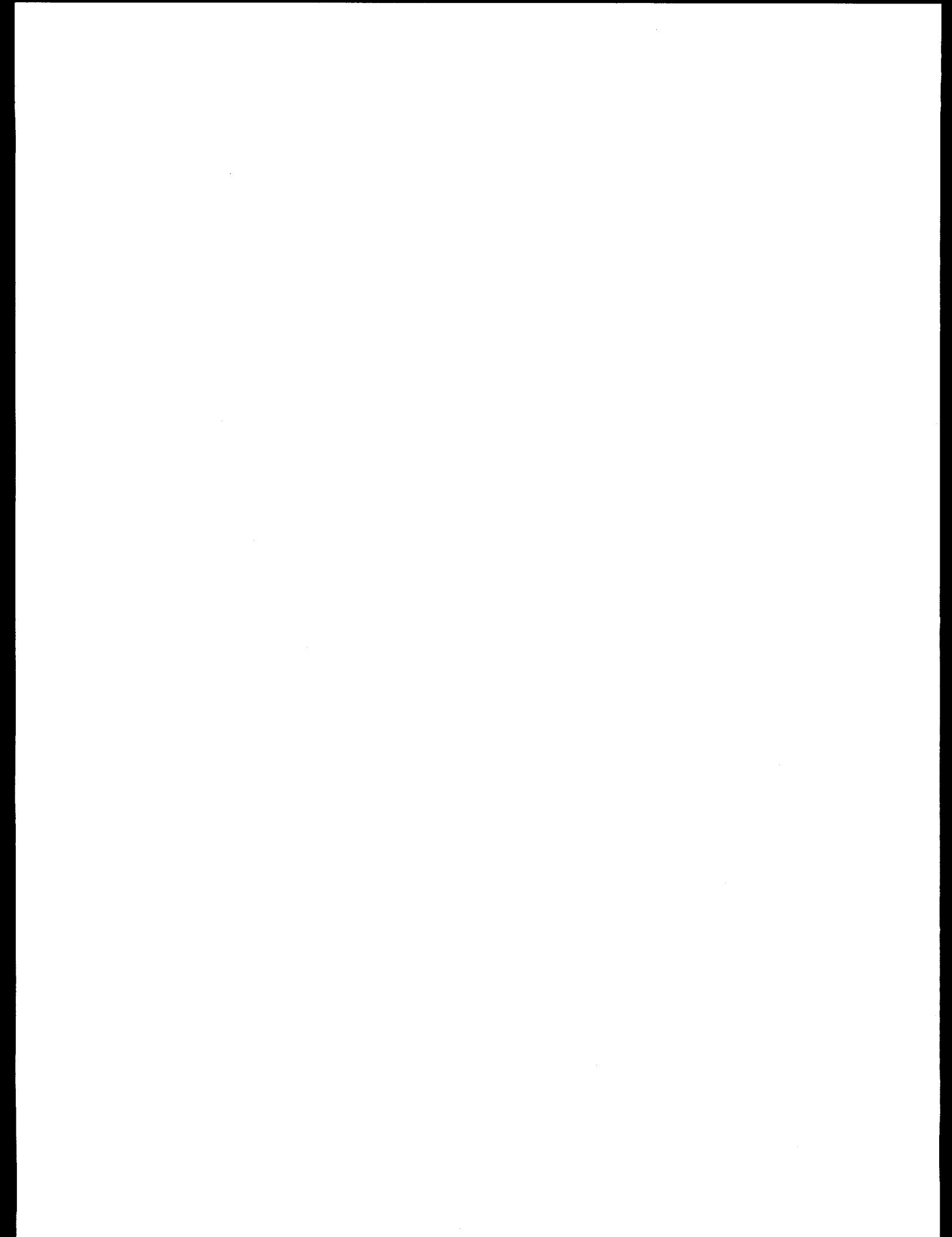
Nick Priest is Professor of Environmental Toxicology and Head of the Urban Pollution Research Centre at Middlesex University. He is a Chartered Biologist who received his Ph.D. from the University of London in 1974. Prior to joining Middlesex University he was employed, for 13 years, by the National Radiological Protection Board and subsequently by the United Kingdom Atomic Energy Authority, where he was Head of the Biomedical Research Department and then Business Development Manager-Health Care. Professor Priest's area of expertise is toxicology and radiobiology specializing in the bioavailability, biokinetics and toxicity of stable and radioactive metals in animals and man. Other interests include the health effects of urban and industrial pollution.

David M. Taylor, UK

Prof. Taylor is a Chartered Chemist and Fellow of the Royal Society of Chemistry as well as of the Royal College of Pathologists. He earned his Ph.D. in Biochemistry at the University of London in 1959, his DSc at the University of Liverpool in 1972. He is Emeritus Professor of Radiotoxicology at the University of Heidelberg and an Honorary Professor at the Chemistry Department of the University of Wales, Cardiff. His research interests are currently radiation dosimetry of internally deposited radionuclides, chemical mechanisms concerned in the biokinetics and distribution of metals in biological systems and in the induction of toxic effects, metal speciation in biological and environmental systems, and the role of metal complexes in normal and pathological metabolic processes. Prof. Taylor is editor of *Nuclear Medicine and Biology* and editor-in-chief of *Applied Radiation and Isotopes*.

APPENDIX E

Aggregated Results of Expert Responses



**Table 1. Aggregated results of Internal Dosimetry Panel expert responses
for Probabilistic Accident Consequence Uncertainty Analysis**

	Quantiles		
	5%	50%	95%
Question 1 average ventilation rates, $L \text{ min}^{-1}$, assuming normal daily (24 hr) mix of activities (combined male, female average) adult	8.30E+00	1.33E+01	2.35E+01
Question 1 average ventilation rates, $L \text{ min}^{-1}$, assuming normal daily (24 hr) mix of activities (combined male, female average) 5 year old children	4.12E+00	6.19E+00	9.25E+00
Question 2 total initial deposition in respiratory tract (mature adults) , as % of total amount inhaled, normal daily (24 hr) mix of activities, 0.1 μm AMAD	1.25E-01	3.61E-01	7.54E-01
Question 2 total initial deposition in respiratory tract (mature adults) , as % of total amount inhaled, normal daily (24 hr) mix of activities, 1 μm AMAD	2.17E-01	4.73E-01	7.33E-01
Question 2 total initial deposition in respiratory tract (mature adults) , as % of total amount inhaled, normal daily (24 hr) mix of activities, 10 μm AMAD	4.71E-01	7.52E-01	9.49E-01
Question 2 total initial deposition in respiratory tract (5 year old children) , as % of total amount inhaled, normal daily (24 hr) mix of activities, 1 μm AMAD	2.11E-01	5.02E-01	8.01E-01
Question 3 initial deposition in extrathoracic region (mature adults) , as % of total deposition in the respiratory tract, 0.1 μm AMAD {Question also gives initial deposition in lung [tracheobronchial (TB) + pulmonary (AI) regions], since lung = total - ET.}	2.10E-02	1.42E-01	3.98E-01
Question 3 initial deposition in extrathoracic region (mature adults) , as % of total deposition in the respiratory tract, 1 μm AMAD {Question also gives initial deposition in lung [tracheobronchial (TB) + pulmonary (AI) regions], since lung = total - ET.}	1.26E-01	6.07E-01	8.52E-01
Question 3 initial deposition in extrathoracic region (mature adults) , as % of total deposition in the respiratory tract, 10 μm AMAD {Question also gives initial deposition in lung [tracheobronchial (TB) + pulmonary (AI) regions], since lung = total - ET.}	4.40E-01	8.83E-01	9.78E-01
Question 3 initial deposition in extrathoracic region (5 year olds) , as % of total deposition in the respiratory tract, 1 μm AMAD {Question also gives initial deposition in lung [tracheobronchial (TB) + pulmonary (AI) regions], since lung = total - ET.}	1.54E-01	6.41E-01	9.00E-01
Question 4 initial deposition in tracheobronchial (TB) region (mature adults) , as % of total deposition in the lung (TB + AI regions), 0.1 μm AMAD	7.47E-02	2.41E-01	5.71E-01
Question 4 initial deposition in tracheobronchial (TB) region (mature adults) , as % of total deposition in the lung (TB + AI regions), 1 μm AMAD	1.14E-01	2.25E-01	5.86E-01
Question 4 initial deposition in tracheobronchial (TB) region (mature adults) , as % of total deposition in the lung (TB + AI regions), 10 μm AMAD	2.27E-01	4.27E-01	8.48E-01
Question 4 initial deposition in tracheobronchial (TB) region (5 year old children) , as % of total deposition in the lung (TB + AI regions), 1 μm AMAD	8.77E-02	2.42E-01	8.15E-01
Question 5 assuming completely insoluble particles (1 μm AMAD), retention in each respiratory tract region in mature adults as % of total initial deposition in respiratory tract, 10 min after deposition: tracheobronchial airways	2.46E-02	7.21E-02	2.30E-01
Question 5 assuming completely insoluble particles (1 μm AMAD), retention in each respiratory tract region in mature adults as % of total initial deposition in respiratory tract, 1 hr after deposition: tracheobronchial airways	1.03E-02	6.58E-02	1.90E-01
Question 5 assuming completely insoluble particles (1 μm AMAD), retention in each respiratory tract region in mature adults as % of total initial deposition in respiratory tract, 1 day after deposition: tracheobronchial airways	2.30E-03	3.96E-02	1.29E-01
Question 5 assuming completely insoluble particles (1 μm AMAD), retention in each respiratory tract region in mature adults as % of total initial deposition in respiratory tract, 1 month after deposition: tracheobronchial airways	1.51E-04	1.66E-02	8.00E-02

Table 1. Aggregated results of Internal Dosimetry Panel expert responses for Probabilistic Accident Consequence Uncertainty Analysis (Continued)

Question 5 assuming completely insoluble particles (1 μm AMAD), retention in each respiratory tract region in mature adults as % of total initial deposition in respiratory tract, <i>1 year after deposition: tracheobronchial airways</i>	4.29E-07	1.50E-04	9.27E-03
Question 5 assuming completely insoluble particles (1 μm AMAD), retention in each respiratory tract region in mature adults as % of total initial deposition in respiratory tract, <i>10 years after deposition: tracheobronchial airways</i>	2.68E-11	5.14E-06	3.59E-03
Question 5 assuming completely insoluble particles (1 μm AMAD), retention in each respiratory tract region in mature adults as % of total initial deposition in respiratory tract, <i>10 min after deposition: pulmonary (AI) region</i>	7.74E-02	2.43E-01	4.83E-01
Question 5 assuming completely insoluble particles (1 μm AMAD), retention in each respiratory tract region in mature adults as % of total initial deposition in respiratory tract, <i>1 hr after deposition: pulmonary (AI) region</i>	7.65E-02	2.42E-01	4.83E-01
Question 5 assuming completely insoluble particles (1 μm AMAD), retention in each respiratory tract region in mature adults as % of total initial deposition in respiratory tract, <i>1 day after deposition: pulmonary (AI) region</i>	7.49E-02	2.43E-01	4.53E-01
Question 5 assuming completely insoluble particles (1 μm AMAD), retention in each respiratory tract region in mature adults as % of total initial deposition in respiratory tract, <i>1 month after deposition: pulmonary (AI) region</i>	6.22E-02	2.11E-01	4.12E-01
Question 5 assuming completely insoluble particles (1 μm AMAD), retention in each respiratory tract region in mature adults as % of total initial deposition in respiratory tract, <i>1 year after deposition: pulmonary (AI) region</i>	1.10E-02	1.25E-01	2.48E-01
Question 5 assuming completely insoluble particles (1 μm AMAD), retention in each respiratory tract region in mature adults as % of total initial deposition in respiratory tract, <i>10 years after deposition: pulmonary (AI) region</i>	1.05E-03	2.02E-02	1.16E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Sr (1 hr)</i>	1.12E-03	8.48E-02	5.18E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Sr (1 day)</i>	3.62E-02	1.83E-01	6.32E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Sr (1 week)</i>	3.98E-02	2.01E-01	6.42E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Sr (1 month)</i>	5.65E-02	2.45E-01	6.73E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Sr (1 year)</i>	1.08E-01	3.41E-01	6.86E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Sr (10 years)</i>	1.37E-01	3.85E-01	7.89E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>I (1 hr)</i>	3.09E-02	6.34E-01	9.83E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>I (1 day)</i>	4.24E-01	7.44E-01	9.96E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>I (1 week)</i>	4.87E-01	7.61E-01	9.96E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>I (1 month)</i>	4.87E-01	7.61E-01	9.96E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>I (1 year)</i>	4.88E-01	7.62E-01	9.96E-01

**Table 1. Aggregated results of Internal Dosimetry Panel expert responses
for Probabilistic Accident Consequence Uncertainty Analysis (Continued)**

Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>I (10 years)</i>	4.88E-01	7.62E-01	9.96E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Cs (1 hr)</i>	2.06E-02	4.10E-01	8.01E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Cs (1 day)</i>	2.65E-01	5.36E-01	8.66E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Cs (1 week)</i>	2.92E-01	6.07E-01	8.70E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Cs (1 month)</i>	3.07E-01	6.51E-01	9.50E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Cs (1 year)</i>	3.48E-01	6.74E-01	9.78E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Cs (10 years)</i>	3.62E-01	6.79E-01	9.77E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Pu (1 hr)</i>	2.87E-05	1.24E-03	5.37E-02
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Pu (1 day)</i>	1.44E-04	4.15E-03	5.49E-02
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Pu (1 week)</i>	2.40E-04	9.27E-03	6.34E-02
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Pu (1 month)</i>	4.65E-04	1.51E-02	1.04E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Pu (1 year)</i>	2.26E-03	4.69E-02	2.41E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Pu (10 years)</i>	5.26E-03	7.38E-02	2.72E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Ru (1 hr)</i>	4.57E-05	1.91E-02	2.47E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Ru (1 day)</i>	1.42E-03	3.29E-02	2.69E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Ru (1 week)</i>	1.06E-03	2.43E-02	2.37E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Ru (1 month)</i>	1.09E-03	3.59E-02	3.47E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Ru (1 year)</i>	3.20E-03	8.20E-02	4.77E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Ru (10 years)</i>	5.43E-03	1.25E-01	6.21E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Ce (1 hr)</i>	1.32E-05	3.44E-03	9.79E-02
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Ce (1 day)</i>	1.10E-04	6.91E-03	1.06E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Ce (1 week)</i>	1.17E-04	2.25E-02	1.24E-01

**Table 1. Aggregated results of Internal Dosimetry Panel expert responses
for Probabilistic Accident Consequence Uncertainty Analysis (Continued)**

Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Ce (1 month)</i>	2.28E-04	3.87E-02	1.81E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Ce (1 year)</i>	1.13E-03	9.99E-02	3.04E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Ce (10 years)</i>	3.33E-03	1.33E-01	3.16E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Te (1 hr)</i>	3.03E-04	5.08E-02	7.87E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Te (1 day)</i>	1.41E-03	2.07E-01	8.21E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Te (1 week)</i>	1.42E-03	1.90E-01	8.29E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Te (1 month)</i>	1.47E-03	2.32E-01	8.39E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Te (1 hr)</i>	3.99E-03	3.33E-01	8.64E-01
Question 7 <i>absorption (dissolution and transfer) to blood in adults</i> , as % of total initial deposition (1 μm AMAD particles) in respiratory tract: <i>Te (10 years)</i>	6.43E-03	3.55E-01	9.07E-01
Question 9 <i>absorption to blood</i> as a fraction (f_1) of activity ingested: <i>Sr, 3 month old infants</i>	1.47E-01	5.48E-01	9.05E-01
Question 9 <i>absorption to blood</i> as a fraction (f_1) of activity ingested: <i>Sr, 5 year old children</i>	5.26E-02	3.27E-01	7.42E-01
Question 9 <i>absorption to blood</i> as a fraction (f_1) of activity ingested: <i>Sr, adults</i>	8.76E-02	2.42E-01	6.27E-01
Question 9 <i>absorption to blood</i> as a fraction (f_1) of activity ingested: <i>I, 3 month old infants</i>	8.23E-01	9.66E-01	1.00E+00
Question 9 <i>absorption to blood</i> as a fraction (f_1) of activity ingested: <i>I, 5 year old children</i>	8.23E-01	9.63E-01	1.00E+00
Question 9 <i>absorption to blood</i> as a fraction (f_1) of activity ingested: <i>I, adults</i>	8.22E-01	9.61E-01	1.00E+00
Question 9 <i>absorption to blood</i> as a fraction (f_1) of activity ingested: <i>Cs, 3 month old infants</i>	5.55E-01	9.07E-01	9.99E-01
Question 9 <i>absorption to blood</i> as a fraction (f_1) of activity ingested: <i>Cs, 5 year old children</i>	5.48E-01	9.00E-01	9.99E-01
Question 9 <i>absorption to blood</i> as a fraction (f_1) of activity ingested: <i>Cs, adults</i>	5.43E-01	8.93E-01	9.99E-01
Question 9 <i>absorption to blood</i> as a fraction (f_1) of activity ingested: <i>PuO₂ (refractory oxide), 3 month old infants</i>	2.20E-07	8.56E-05	8.95E-04
Question 9 <i>absorption to blood</i> as a fraction (f_1) of activity ingested: <i>PuO₂ (refractory oxide), 5 year old children</i>	1.70E-07	1.22E-05	4.33E-04
Question 9 <i>absorption to blood</i> as a fraction (f_1) of activity ingested: <i>PuO₂ (refractory oxide), adults</i>	1.70E-07	1.15E-05	2.81E-04
Question 9 <i>absorption to blood</i> as a fraction (f_1) of activity ingested: <i>Pu (biologically incorporated), 3 month old infants</i>	5.27E-05	1.46E-03	9.82E-03

**Table 1. Aggregated results of Internal Dosimetry Panel expert responses
for Probabilistic Accident Consequence Uncertainty Analysis (Continued)**

Question 9 absorption to blood as a fraction (f_1) of activity ingested: Pu (biologically incorporated), 5 year old children	1.16E-05	3.09E-04	2.82E-03
Question 9 absorption to blood as a fraction (f_1) of activity ingested: Pu (biologically incorporated), adults	1.16E-05	3.27E-04	1.49E-03
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), % retained in liver and skeleton (bone + bone marrow) of 5 year old children, 1 day after entry into blood: Sr	2.71E-01	5.58E-01	9.07E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), % retained in liver and skeleton (bone + bone marrow) of 5 year old children, 1 week after entry into blood: Sr	1.98E-01	4.47E-01	8.16E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), % retained in liver and skeleton (bone + bone marrow) of 5 year old children, 1 month after entry into blood: Sr	1.60E-01	3.94E-01	7.39E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), % retained in liver and skeleton (bone + bone marrow) of 5 year old children, 1 year after entry into blood: Sr	8.23E-02	2.31E-01	4.94E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), % retained in liver and skeleton (bone + bone marrow) of 5 year old children, 10 years after entry into blood: Sr	1.18E-03	3.20E-02	1.47E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), % retained in liver and skeleton (bone + bone marrow) of 5 year old children, 50 years after entry into blood: Sr	5.55E-08	6.46E-03	8.05E-02
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), % retained in liver and skeleton (bone + bone marrow) of adults, 1 day after entry into blood: Sr	1.70E-01	3.24E-01	5.76E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), % retained in liver and skeleton (bone + bone marrow) of adults, 1 week after entry into blood: Sr	1.17E-01	2.29E-01	4.76E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), % retained in liver and skeleton (bone + bone marrow) of adults, 1 month after entry into blood: Sr	1.04E-01	2.11E-01	3.51E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), % retained in liver and skeleton (bone + bone marrow) of adults, 1 year after entry into blood: Sr	6.75E-02	1.38E-01	2.43E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), % retained in liver and skeleton (bone + bone marrow) of adults, 10 years after entry into blood: Sr	1.81E-02	6.45E-02	1.37E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), % retained in liver and skeleton (bone + bone marrow) of adults, 50 years after entry into blood: Sr	1.11E-03	1.85E-02	8.88E-02
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), % retained in liver and skeleton (bone + bone marrow) of 5 year old children, 1 day after entry into blood: Pu	2.16E-01	5.54E-01	9.47E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), % retained in liver and skeleton (bone + bone marrow) of 5 year old children, 1 week after entry into blood: Pu	5.25E-01	7.54E-01	9.49E-01

**Table 1. Aggregated results of Internal Dosimetry Panel expert responses
for Probabilistic Accident Consequence Uncertainty Analysis (Continued)**

Question 10 considering total amount reaching blood (as if administered intravenously as single injection), <i>% retained in liver and skeleton (bone + bone marrow) of 5 year old children, 1 month after entry into blood: Pu</i>	5.44E-01	7.81E-01	9.51E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), <i>% retained in liver and skeleton (bone + bone marrow) of 5 year old children, 1 year after entry into blood: Pu</i>	3.59E-01	7.44E-01	9.47E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), <i>% retained in liver and skeleton (bone + bone marrow) of 5 year old children, 10 years after entry into blood: Pu</i>	2.05E-01	5.60E-01	8.84E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), <i>% retained in liver and skeleton (bone + bone marrow) of 5 year old children, 50 years after entry into blood: Pu</i>	5.16E-02	3.47E-01	7.21E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), <i>% retained in liver and skeleton (bone + bone marrow) of adults, 1 day after entry into blood: Pu</i>	2.16E-01	5.38E-01	9.41E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), <i>% retained in liver and skeleton (bone + bone marrow) of adults, 1 week after entry into blood: Pu</i>	5.45E-01	7.68E-01	9.48E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), <i>% retained in liver and skeleton (bone + bone marrow) of adults, 1 month after entry into blood: Pu</i>	5.88E-01	8.15E-01	9.55E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), <i>% retained in liver and skeleton (bone + bone marrow) of adults, 1 year after entry into blood: Pu</i>	4.22E-01	8.00E-01	9.53E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), <i>% retained in liver and skeleton (bone + bone marrow) of adults, 10 years after entry into blood: Pu</i>	3.53E-01	7.31E-01	9.25E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), <i>% retained in liver and skeleton (bone + bone marrow) of adults, 50 years after entry into blood: Pu</i>	2.00E-01	5.05E-01	8.52E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), <i>% retained in liver and skeleton (bone + bone marrow) of 5 year old children, 1 day after entry into blood: Ce</i>	2.71E-01	7.33E-01	9.49E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), <i>% retained in liver and skeleton (bone + bone marrow) of 5 year old children, 1 week after entry into blood: Ce</i>	4.42E-01	7.87E-01	9.50E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), <i>% retained in liver and skeleton (bone + bone marrow) of 5 year old children, 1 month after entry into blood: Ce</i>	4.92E-01	7.93E-01	9.47E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), <i>% retained in liver and skeleton (bone + bone marrow) of 5 year old children, 1 year after entry into blood: Ce</i>	3.30E-01	7.48E-01	9.48E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), <i>% retained in liver and skeleton (bone + bone marrow) of 5 year old children, 10 years after entry into blood: Ce</i>	1.08E-01	4.21E-01	8.61E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), <i>% retained in liver and skeleton (bone + bone marrow) of 5 year old children, 50 years after entry into blood: Ce</i>	1.25E-03	9.00E-02	5.96E-01

**Table 1. Aggregated results of Internal Dosimetry Panel expert responses
for Probabilistic Accident Consequence Uncertainty Analysis (Continued)**

Question 10 considering total amount reaching blood (as if administered intravenously as single injection), <i>% retained in liver and skeleton (bone + bone marrow) of adults, 1 day after entry into blood: Ce</i>	2.68E-01	7.34E-01	9.47E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), <i>% retained in liver and skeleton (bone + bone marrow) of adults, 1 week after entry into blood: Ce</i>	4.44E-01	7.95E-01	9.49E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), <i>% retained in liver and skeleton (bone + bone marrow) of adults, 1 month after entry into blood: Ce</i>	5.23E-01	7.97E-01	9.48E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), <i>% retained in liver and skeleton (bone + bone marrow) of adults, 1 year after entry into blood: Ce</i>	3.99E-01	7.56E-01	9.37E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), <i>% retained in liver and skeleton (bone + bone marrow) of adults, 10 years after entry into blood: Ce</i>	1.41E-01	4.57E-01	8.80E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), <i>% retained in liver and skeleton (bone + bone marrow) of adults, 50 years after entry into blood: Ce</i>	5.32E-03	1.32E-01	7.41E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), <i>% retained in liver and skeleton (bone + bone marrow) of 5 year old children, 1 day after entry into blood: Te</i>	1.10E-02	1.67E-01	6.51E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), <i>% retained in liver and skeleton (bone + bone marrow) of 5 year old children, 1 week after entry into blood: Te</i>	4.68E-02	2.51E-01	7.80E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), <i>% retained in liver and skeleton (bone + bone marrow) of 5 year old children, 1 month after entry into blood: Te</i>	1.11E-02	2.41E-01	7.14E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), <i>% retained in liver and skeleton (bone + bone marrow) of adults, 1 day after entry into blood: Te</i>	3.90E-02	1.77E-01	6.81E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), <i>% retained in liver and skeleton (bone + bone marrow) of adults, 1 week after entry into blood: Te</i>	5.16E-02	2.51E-01	7.07E-01
Question 10 considering total amount reaching blood (as if administered intravenously as single injection), <i>% retained in liver and skeleton (bone + bone marrow) of adults, 1 month after entry into blood: Te</i>	3.95E-02	2.50E-01	7.01E-01
Question 11 retention in skeleton of 5 year old children, as % total retention in liver + skeleton (bone + marrow) 1 day after entry into blood: Sr	8.34E-01	9.61E-01	9.98E-01
Question 11 retention in skeleton of 5 year old children, as % total retention in liver + skeleton (bone + marrow) 1 week after entry into blood: Sr	8.12E-01	9.66E-01	9.98E-01
Question 11 retention in skeleton of 5 year old children, as % total retention in liver + skeleton (bone + marrow) 1 month after entry into blood: Sr	8.42E-01	9.85E-01	1.00E+00
Question 11 retention in skeleton of 5 year old children, as % total retention in liver + skeleton (bone + marrow) 1 year after entry into blood: Sr	7.84E-01	9.97E-01	1.00E+00
Question 11 retention in skeleton of 5 year old children, as % total retention in liver + skeleton (bone + marrow) 10 years time after entry into blood: Sr	6.82E-01	9.97E-01	1.00E+00
Question 11 retention in skeleton of 5 year old children, as % total retention in liver + skeleton (bone + marrow) 50 years after entry into blood: Sr	6.63E-01	9.97E-01	1.00E+00

**Table 1. Aggregated results of Internal Dosimetry Panel expert responses
for Probabilistic Accident Consequence Uncertainty Analysis (Continued)**

Question 11 retention in skeleton of <i>adults, as % total retention in liver + skeleton (bone + marrow) 1 day after entry into blood: Sr</i>	8.46E-01	9.56E-01	9.98E-01
Question 11 retention in skeleton of <i>adults, as % total retention in liver + skeleton (bone + marrow) 1 week after entry into blood: Sr</i>	8.22E-01	9.58E-01	9.98E-01
Question 11 retention in skeleton of <i>adults, as % total retention in liver + skeleton (bone + marrow) 1 month after entry into blood: Sr</i>	8.51E-01	9.84E-01	1.00E+00
Question 11 retention in skeleton of <i>adults, as % total retention in liver + skeleton (bone + marrow) 1 year after entry into blood: Sr</i>	7.70E-01	9.94E-01	1.00E+00
Question 11 retention in skeleton of <i>adults, as % total retention in liver + skeleton (bone + marrow) 10 years after entry into blood: Sr</i>	6.79E-01	9.95E-01	1.00E+00
Question 11 retention in skeleton of <i>adults, as % total retention in liver + skeleton (bone + marrow) 50 years after entry into blood: Sr</i>	6.39E-01	9.96E-01	1.00E+00
Question 11 retention in skeleton of <i>5 year old children, as % total retention in liver + skeleton (bone + marrow) 1 day after entry into blood: Pu</i>	4.32E-01	7.61E-01	9.61E-01
Question 11 retention in skeleton of <i>5 year old children, as % total retention in liver + skeleton (bone + marrow) 1 week after entry into blood: Pu</i>	4.29E-01	7.51E-01	9.52E-01
Question 11 retention in skeleton of <i>5 year old children, as % total retention in liver + skeleton (bone + marrow) 1 month after entry into blood: Pu</i>	4.28E-01	7.49E-01	9.51E-01
Question 11 retention in skeleton of <i>5 year old children, as % total retention in liver + skeleton (bone + marrow) 1 year after entry into blood: Pu</i>	4.23E-01	7.29E-01	9.46E-01
Question 11 retention in skeleton of <i>5 year old children, as % total retention in liver + skeleton (bone + marrow) 10 years after entry into blood: Pu</i>	1.70E-01	5.19E-01	8.83E-01
Question 11 retention in skeleton of <i>5 year old children, as % total retention in liver + skeleton (bone + marrow) 50 years after entry into blood: Pu</i>	1.62E-01	5.97E-01	9.30E-01
Question 11 retention in skeleton of <i>adults, as % total retention in liver + skeleton (bone + marrow) 1 day after entry into blood: Pu</i>	2.69E-01	5.97E-01	9.26E-01
Question 11 retention in skeleton of <i>adults, as % total retention in liver + skeleton (bone + marrow) 1 week after entry into blood: Pu</i>	2.21E-01	6.01E-01	9.22E-01
Question 11 retention in skeleton of <i>adults, as % total retention in liver + skeleton (bone + marrow) 1 month after entry into blood: Pu</i>	2.08E-01	6.02E-01	9.17E-01
Question 11 retention in skeleton of <i>adults, as % total retention in liver + skeleton (bone + marrow) 1 year after entry into blood: Pu</i>	2.24E-01	5.80E-01	8.11E-01
Question 11 retention in skeleton of <i>adults, as % total retention in liver + skeleton (bone + marrow) 10 years after entry into blood: Pu</i>	2.70E-01	6.02E-01	8.90E-01
Question 11 retention in skeleton of <i>adults, as % total retention in liver + skeleton (bone + marrow) 50 years after entry into blood: Pu</i>	2.88E-01	6.69E-01	9.75E-01
Question 11 retention in skeleton of <i>5 year old children, as % total retention in liver + skeleton (bone + marrow) 1 day after entry into blood: Ce</i>	3.25E-01	6.23E-01	9.29E-01
Question 11 retention in skeleton of <i>5 year old children, as % total retention in liver + skeleton (bone + marrow) 1 week after entry into blood: Ce</i>	2.49E-01	6.25E-01	9.30E-01
Question 11 retention in skeleton of <i>5 year old children, as % total retention in liver + skeleton (bone + marrow) 1 month after entry into blood: Ce</i>	2.48E-01	6.25E-01	9.29E-01
Question 11 retention in skeleton of <i>5 year old children, as % total retention in liver + skeleton (bone + marrow) 1 year after entry into blood: Ce</i>	2.44E-01	6.19E-01	9.17E-01

Table 1. Aggregated results of Internal Dosimetry Panel expert responses for Probabilistic Accident Consequence Uncertainty Analysis (Continued)

Question 11 retention in skeleton of <i>5 year old children, as % total retention in liver + skeleton (bone + marrow) 10 years after entry into blood: Ce</i>	1.86E-01	6.35E-01	9.51E-01
Question 11 retention in skeleton of <i>5 year old children, as % total retention in liver + skeleton (bone + marrow) 50 years after entry into blood: Ce</i>	7.58E-02	6.43E-01	9.69E-01
Question 11 retention in skeleton of <i>adults, as % total retention in liver + skeleton (bone + marrow) 1 day after entry into blood: Ce</i>	1.66E-01	3.69E-01	7.12E-01
Question 11 retention in skeleton of <i>adults, as % total retention in liver + skeleton (bone + marrow) 1 week after entry into blood: Ce</i>	1.67E-01	3.72E-01	6.81E-01
Question 11 retention in skeleton of <i>adults, as % total retention in liver + skeleton (bone + marrow) 1 month after entry into blood: Ce</i>	1.93E-01	3.74E-01	6.81E-01
Question 11 retention in skeleton of <i>adults, as % total retention in liver + skeleton (bone + marrow) 1 year after entry into blood: Ce</i>	2.10E-01	3.96E-01	7.13E-01
Question 11 retention in skeleton of <i>adults, as % total retention in liver + skeleton (bone + marrow) 10 years after entry into blood: Ce</i>	2.19E-01	4.34E-01	9.32E-01
Question 11 retention in skeleton of <i>adults, as % total retention in liver + skeleton (bone + marrow) 50 years after entry into blood: Ce</i>	1.39E-01	5.13E-01	9.75E-01
Question 11 retention in skeleton of <i>5 year old children, as % total retention in liver + skeleton (bone + marrow) 1 day after entry into blood: Te</i>	1.65E-01	8.18E-01	9.94E-01
Question 11 retention in skeleton of <i>5 year old children, as % total retention in liver + skeleton (bone + marrow) 1 week after entry into blood: Te</i>	2.53E-01	9.10E-01	9.97E-01
Question 11 retention in skeleton of <i>5 year old children, as % total retention in liver + skeleton (bone + marrow) 1 month after entry into blood: Te</i>	4.67E-01	9.46E-01	9.99E-01
Question 11 retention in skeleton of <i>adults, as % total retention in liver + skeleton (bone + marrow) 1 day after entry into blood: Te</i>	1.57E-01	7.99E-01	9.93E-01
Question 11 retention in skeleton of <i>adults, as % total retention in liver + skeleton (bone + marrow) 1 week after entry into blood: Te</i>	1.31E-01	8.41E-01	9.97E-01
Question 11 retention in skeleton of <i>adults, as % total retention in liver + skeleton (bone + marrow) 1 month after entry into blood: Te</i>	1.03E-06	8.98E-01	9.99E-01
Question 12 retention of <i>plutonium on endosteal bone surfaces of 5 year old children</i> (considering a 10 μm depth of bone mineral) as a % of total skeletal retention, <i>1 day after entry into blood</i>	7.40E-01	9.75E-01	1.00E+00
Question 12 retention of <i>plutonium on endosteal bone surfaces of 5 year old children</i> (considering a 10 μm depth of bone mineral) as a % of total skeletal retention, <i>1 week after entry into blood</i>	7.34E-01	9.73E-01	9.99E-01
Question 12 retention of <i>plutonium on endosteal bone surfaces of 5 year old children</i> (considering a 10 μm depth of bone mineral) as a % of total skeletal retention, <i>1 month after entry into blood</i>	5.29E-01	9.10E-01	9.80E-01
Question 12 retention of <i>plutonium on endosteal bone surfaces of 5 year old children</i> (considering a 10 μm depth of bone mineral) as a % of total skeletal retention, <i>1 year after entry into blood</i>	1.58E-01	5.16E-01	7.86E-01
Question 12 retention of <i>plutonium on endosteal bone surfaces of 5 year old children</i> (considering a 10 μm depth of bone mineral) as a % of total skeletal retention, <i>10 years after entry into blood</i>	5.37E-02	4.25E-01	7.60E-01
Question 12 retention of <i>plutonium on endosteal bone surfaces of 5 year old children</i> (considering a 10 μm depth of bone mineral) as a % of total skeletal retention, <i>50 years after entry into blood</i>	5.42E-02	4.96E-01	8.19E-01

**Table 1. Aggregated results of Internal Dosimetry Panel expert responses
for Probabilistic Accident Consequence Uncertainty Analysis (Continued)**

Question 12 retention of plutonium on endosteal bone surfaces of adults (considering a 10 μm depth of bone mineral) as a % of total skeletal retention, 1 day after entry into blood	7.11E-01	9.83E-01	1.00E+00
Question 12 retention of plutonium on endosteal bone surfaces of adults (considering a 10 μm depth of bone mineral) as a % of total skeletal retention, 1 week after entry into blood	6.62E-01	9.88E-01	1.00E+00
Question 12 retention of plutonium on endosteal bone surfaces of adults (considering a 10 μm depth of bone mineral) as a % of total skeletal retention, 1 month after entry into blood	6.83E-01	9.80E-01	1.00E+00
Question 12 retention of plutonium on endosteal bone surfaces of adults (considering a 10 μm depth of bone mineral) as a % of total skeletal retention, 1 year after entry into blood	4.94E-01	8.91E-01	9.78E-01
Question 12 retention of plutonium on endosteal bone surfaces of adults (considering a 10 μm depth of bone mineral) as a % of total skeletal retention, 10 years after entry into blood	1.84E-01	7.06E-01	8.84E-01
Question 12 retention of plutonium on endosteal bone surfaces of adults (considering a 10 μm depth of bone mineral) as a % of total skeletal retention, 50 years after entry into blood	3.21E-02	6.17E-01	8.07E-01
Question 13 retention of plutonium on trabecular surfaces of 5 year old children , as % total endosteal surface retention, 1 day after entry into blood	2.81E-01	5.13E-01	7.61E-01
Question 13 retention of plutonium on trabecular surfaces of 5 year old children , as % total endosteal surface retention, 1 week after entry into blood	2.81E-01	5.10E-01	7.55E-01
Question 13 retention of plutonium on trabecular surfaces of 5 year old children , as % total endosteal surface retention, 1 month after entry into blood	2.46E-01	4.74E-01	7.10E-01
Question 13 retention of plutonium on trabecular surfaces of 5 year old children , as % total endosteal surface retention, 1 year after entry into blood	5.42E-02	4.09E-01	7.24E-01
Question 13 retention of plutonium on trabecular surfaces of 5 year old children , as % total endosteal surface retention, 10 years after entry into blood	2.30E-02	3.56E-01	7.34E-01
Question 13 retention of plutonium on trabecular surfaces of 5 year old children , as % total endosteal surface retention, 50 years after entry into blood	2.20E-02	2.69E-01	5.88E-01
Question 13 retention of plutonium on trabecular surfaces of adults , as % total endosteal surface retention, 1 day after entry into blood	5.12E-02	6.05E-01	8.29E-01
Question 13 retention of plutonium on trabecular surfaces of adults , as % total endosteal surface retention, 1 week after entry into blood	4.23E-01	6.06E-01	8.30E-01
Question 13 retention of plutonium on trabecular surfaces of adults , as % total endosteal surface retention, 1 month after entry into blood	4.11E-01	6.01E-01	8.00E-01
Question 13 retention of plutonium on trabecular surfaces of adults , as % total endosteal surface retention, 1 year after entry into blood	3.21E-01	5.37E-01	7.23E-01
Question 13 retention of plutonium on trabecular surfaces of adults , as % total endosteal surface retention, 10 years after entry into blood	4.15E-02	2.63E-01	5.16E-01
Question 13 retention of plutonium on trabecular surfaces of adults , as % total endosteal surface retention, 50 years after entry into blood	2.08E-02	1.69E-01	4.24E-01
Question 14 retention of plutonium in red bone marrow of 5 year old children , as % total skeletal retention, 1 day after entry into blood	6.61E-05	9.07E-04	8.46E-02
Question 14 retention of plutonium in red bone marrow of 5 year old children , as % total skeletal retention, 1 week after entry into blood	1.28E-04	3.97E-03	2.73E-02

**Table 1. Aggregated results of Internal Dosimetry Panel expert responses
for Probabilistic Accident Consequence Uncertainty Analysis (Continued)**

Question 14 retention of <i>plutonium in red bone marrow</i> of <i>5 year old children</i> , as % total skeletal retention, <i>1 month after entry into blood</i>	1.14E-03	2.21E-02	7.92E-02
Question 14 retention of <i>plutonium in red bone marrow</i> of <i>5 year old children</i> , as % total skeletal retention, <i>1 year after entry into blood</i>	1.34E-03	7.11E-02	2.70E-01
Question 14 retention of <i>plutonium in red bone marrow</i> of <i>5 year old children</i> , as % total skeletal retention, <i>10 years after entry into blood</i>	1.25E-03	6.41E-02	2.60E-01
Question 14 retention of <i>plutonium in red bone marrow</i> of <i>5 year old children</i> , as % total skeletal retention, <i>50 years after entry into blood</i>	1.54E-04	2.23E-02	1.38E-01
Question 14 retention of <i>plutonium in red bone marrow</i> of <i>adults</i> , as % total skeletal retention, <i>1 day after entry into blood</i>	4.34E-05	4.12E-04	9.79E-02
Question 14 retention of <i>plutonium in red bone marrow</i> of <i>adults</i> , as % total skeletal retention, <i>1 week after entry into blood</i>	3.09E-04	1.91E-03	1.94E-02
Question 14 retention of <i>plutonium in red bone marrow</i> of <i>adults</i> , as % total skeletal retention, <i>1 month after entry into blood</i>	1.05E-03	8.82E-03	3.70E-02
Question 14 retention of <i>plutonium in red bone marrow</i> of <i>adults</i> , as % total skeletal retention, <i>1 year after entry into blood</i>	1.30E-03	2.89E-02	8.78E-02
Question 14 retention of <i>plutonium in red bone marrow</i> of <i>adults</i> , as % total skeletal retention, <i>10 years after entry into blood</i>	1.13E-03	2.40E-02	7.45E-02
Question 14 retention of <i>plutonium in red bone marrow</i> of <i>adults</i> , as % total skeletal retention, <i>50 years after entry into blood</i>	1.42E-04	1.32E-02	5.07E-02
Question 15 considering total amount reaching blood of <i>5 year old children</i> (as if administered intravenously as single injection), as % total retention (whole body or systemic), <i>1 day after entry into blood: Ru</i>	4.16E-01	8.11E-01	9.76E-01
Question 15 considering total amount reaching blood of <i>5 year old children</i> (as if administered intravenously as single injection), as % total retention (whole body or systemic), <i>1 week after entry into blood: Ru</i>	2.72E-01	6.34E-01	8.52E-01
Question 15 considering total amount reaching blood of <i>5 year old children</i> (as if administered intravenously as single injection), as % total retention (whole body or systemic), <i>1 month after entry into blood: Ru</i>	1.03E-01	3.78E-01	7.06E-01
Question 15 considering total amount reaching blood of <i>5 year old children</i> (as if administered intravenously as single injection), as % total retention (whole body or systemic), <i>1 year after entry into blood: Ru</i>	5.34E-03	1.48E-01	5.17E-01
Question 15 considering total amount reaching blood of <i>5 year old children</i> (as if administered intravenously as single injection), as % total retention (whole body or systemic), <i>5 years after entry into blood: Ru</i>	2.47E-05	5.51E-02	2.93E-01
Question 15 considering total amount reaching blood of <i>adults</i> (as if administered intravenously as single injection), as % total retention (whole body or systemic), <i>1 day after entry into blood: Ru</i>	4.38E-01	8.30E-01	9.70E-01
Question 15 considering total amount reaching blood of <i>adults</i> (as if administered intravenously as single injection), as % total retention (whole body or systemic), <i>1 week after entry into blood: Ru</i>	2.96E-01	6.57E-01	8.50E-01
Question 15 considering total amount reaching blood of <i>adults</i> (as if administered intravenously as single injection), as % total retention (whole body or systemic), <i>1 month after entry into blood: Ru</i>	1.23E-01	3.91E-01	7.02E-01
Question 15 considering total amount reaching blood of <i>adults</i> (as if administered intravenously as single injection), as % total retention (whole body or systemic), <i>1 year after entry into blood: Ru</i>	5.94E-03	1.59E-01	4.93E-01

**Table 1. Aggregated results of Internal Dosimetry Panel expert responses
for Probabilistic Accident Consequence Uncertainty Analysis (Continued)**

Question 15 considering total amount reaching blood of adults (as if administered intravenously as single injection), as % total retention (whole body or systemic), 5 years after entry into blood: Ru	1.30E-04	5.73E-02	2.78E-01
Question 15 considering total amount reaching blood of 5 year old children (as if administered intravenously as single injection), as % total retention (whole body or systemic), 1 day after entry into blood: Cs	7.78E-01	9.56E-01	9.94E-01
Question 15 considering total amount reaching blood of 5 year old children (as if administered intravenously as single injection), as % total retention (whole body or systemic), 1 week after entry into blood: Cs	5.71E-01	7.51E-01	9.23E-01
Question 15 considering total amount reaching blood of 5 year old children (as if administered intravenously as single injection), as % total retention (whole body or systemic), 1 month after entry into blood: Cs	1.46E-01	3.21E-01	5.83E-01
Question 15 considering total amount reaching blood of 5 year old children (as if administered intravenously as single injection), as % total retention (whole body or systemic), 1 year after entry into blood: Cs	7.89E-08	1.84E-04	2.62E-02
Question 15 considering total amount reaching blood of 5 year old children (as if administered intravenously as single injection), as % total retention (whole body or systemic), 5 years after entry into blood: Cs	1.11E-12	2.94E-09	2.41E-03
Question 15 considering total amount reaching blood of adults (as if administered intravenously as single injection), as % total retention (whole body or systemic), 1 day after entry into blood: Cs	8.70E-01	9.62E-01	9.92E-01
Question 15 considering total amount reaching blood of adults (as if administered intravenously as single injection), as % total retention (whole body or systemic), 1 week after entry into blood: Cs	7.45E-01	8.59E-01	9.43E-01
Question 15 considering total amount reaching blood of adults (as if administered intravenously as single injection), as % total retention (whole body or systemic), 1 month after entry into blood: Cs	5.45E-01	7.24E-01	8.93E-01
Question 15 considering total amount reaching blood of adults (as if administered intravenously as single injection), as % total retention (whole body or systemic), 1 year after entry into blood: Cs	2.38E-03	6.48E-02	2.64E-01
Question 15 considering total amount reaching blood of adults (as if administered intravenously as single injection), as % total retention (whole body or systemic), 5 years after entry into blood: Cs	1.21E-10	1.08E-05	6.30E-03
Question 16 considering total amount of Iodine reaching blood of 5 year old children (as if administered intravenously as single injection), % retained in thyroid 1 day after entry into blood	1.25E-01	2.96E-01	7.00E-01
Question 16 considering total amount of Iodine reaching blood of 5 year old children (as if administered intravenously as single injection), % retained in thyroid 1 week after entry into blood	1.08E-01	2.68E-01	6.67E-01
Question 16 considering total amount of Iodine reaching blood of 5 year old children (as if administered intravenously as single injection), % retained in thyroid 1 month after entry into blood	3.87E-02	1.54E-01	4.86E-01
Question 16 considering total amount of Iodine reaching blood of 5 year old children (as if administered intravenously as single injection), % retained in thyroid 3 months after entry into blood	1.08E-03	3.46E-02	2.69E-01
Question 16 considering total amount of Iodine reaching blood of adults (as if administered intravenously as single injection), % retained in thyroid 1 day after entry into blood	1.16E-01	2.87E-01	5.63E-01

**Table 1. Aggregated results of Internal Dosimetry Panel expert responses
for Probabilistic Accident Consequence Uncertainty Analysis (Continued)**

Question 16 considering total amount of <i>Iodine reaching blood of adults</i> (as if administered intravenously as single injection), % retained in thyroid <i>1 week after entry into blood</i>	1.12E-01	2.86E-01	5.48E-01
Question 16 considering total amount of <i>Iodine reaching blood of adults</i> (as if administered intravenously as single injection), % retained in thyroid <i>1 month after entry into blood</i>	8.54E-02	2.36E-01	4.57E-01
Question 16 considering total amount of <i>Iodine reaching blood of adults</i> (as if administered intravenously as single injection), % retained in thyroid <i>3 months after entry into blood</i>	4.15E-02	1.54E-01	3.66E-01
Question 17 Intake dose coefficients: absorbed ⁹⁰ Sr dose to lung per unit activity inhaled (committed equivalent doses), Gy Bq ⁻¹ : <i>5 year old children</i>	1.63E-09	2.93E-07	1.85E-06
Question 17 Intake dose coefficients: absorbed ⁹⁰ Sr dose to bone marrow per unit activity inhaled (committed equivalent doses), Gy Bq ⁻¹ : <i>5 year old children</i>	5.75E-09	1.11E-07	5.98E-07
Question 17 Intake dose coefficients: absorbed ⁹⁰ Sr dose to bone surface per unit activity inhaled (committed equivalent doses), Gy Bq ⁻¹ : <i>5 year old children</i>	1.36E-08	2.80E-07	1.69E-06
Question 17 Intake dose coefficients: absorbed ⁹⁰ Sr dose to colon per unit activity ingested (committed equivalent doses), Gy Bq ⁻¹ : <i>5 year old children</i>	3.99E-10	4.51E-08	2.90E-07
Question 17 Intake dose coefficients: absorbed ⁹⁰ Sr dose to bone marrow per unit activity ingested (committed equivalent doses), Gy Bq ⁻¹ : <i>5 year old children</i>	1.12E-08	1.19E-07	1.23E-06
Question 17 Intake dose coefficients: absorbed ⁹⁰ Sr dose to bone surface per unit activity ingested (committed equivalent doses), Gy Bq ⁻¹ : <i>5 year old children</i>	2.14E-08	2.28E-07	2.21E-06
Question 17 Intake dose coefficients: absorbed ¹³¹ I dose to thyroid per unit activity inhaled (committed equivalent doses), Gy Bq ⁻¹ : <i>5 year old children</i>	3.78E-07	1.14E-06	4.81E-06
Question 17 Intake dose coefficients: absorbed ¹³¹ I dose to thyroid per unit activity ingested (committed equivalent doses), Gy Bq ⁻¹ : <i>5 year old children</i>	5.90E-07	2.12E-06	6.99E-06
Question 17 Intake dose coefficients: absorbed ¹³² Te dose to lung per unit activity inhalation (committed equivalent doses), Gy Bq ⁻¹ : <i>5 year old children</i>	1.90E-10	2.98E-09	7.84E-08
Question 17 Intake dose coefficients: absorbed ¹³² Te dose to thyroid per unit activity inhalation (committed equivalent doses), Gy Bq ⁻¹ : <i>5 year old children</i>	7.91E-11	8.58E-08	1.00E-06
Question 17 Intake dose coefficients: absorbed ¹³² Te dose to colon per unit activity inhalation (committed equivalent doses), Gy Bq ⁻¹ : <i>5 year old children</i>	8.37E-10	9.05E-09	3.63E-08
Question 17 Intake dose coefficients: absorbed ¹³⁷ Cs dose to lung per unit activity inhalation (committed equivalent doses), Gy Bq ⁻¹ : <i>5 year old children</i>	1.03E-09	7.65E-09	1.52E-07
Question 17 Intake dose coefficients: absorbed ¹³⁷ Cs dose to colon per unit activity inhalation (committed equivalent doses), Gy Bq ⁻¹ : <i>5 year old children</i>	7.15E-10	6.63E-09	1.69E-08
Question 17 Intake dose coefficients: absorbed ¹³⁷ Cs dose to stomach per unit activity inhalation (committed equivalent doses), Gy Bq ⁻¹ : <i>5 year old children</i>	5.59E-10	3.02E-09	8.87E-09
Question 17 Intake dose coefficients: absorbed ¹³⁷ Cs dose to bone marrow per unit activity inhalation (committed equivalent doses), Gy Bq ⁻¹ : <i>5 year old children</i>	8.90E-10	2.75E-09	8.56E-09
Question 17 Intake dose coefficients: absorbed ¹³⁷ Cs dose to lung per unit activity ingested (committed equivalent doses), Gy Bq ⁻¹ : <i>5 year old children</i>	9.19E-10	6.98E-09	1.82E-08
Question 17 Intake dose coefficients: absorbed ¹³⁷ Cs dose to colon per unit activity ingested (committed equivalent doses), Gy Bq ⁻¹ : <i>5 year old children</i>	5.16E-09	1.53E-08	4.19E-08

Table 1. Aggregated results of Internal Dosimetry Panel expert responses for Probabilistic Accident Consequence Uncertainty Analysis (Continued)

Question 17 Intake dose coefficients: absorbed ^{137}Cs dose to stomach per unit activity ingested (committed equivalent doses), Gy Bq $^{-1}$: 5 year old children	1.04E-09	7.97E-09	2.05E-08
Question 17 Intake dose coefficients: absorbed ^{137}Cs dose to bone marrow per unit activity ingested (committed equivalent doses), Gy Bq $^{-1}$: 5 year old children	9.32E-10	7.04E-09	1.91E-08
Question 17 Intake dose coefficients: absorbed ^{144}Ce dose to bone surface per unit activity inhaled (committed equivalent doses), Gy Bq $^{-1}$: 5 year old children	5.10E-10	2.45E-07	2.94E-06
Question 17 Intake dose coefficients: absorbed ^{144}Ce dose to lung per unit activity inhaled (committed equivalent doses), Gy Bq $^{-1}$: 5 year old children	4.63E-09	3.00E-07	1.70E-06
Question 17 Intake dose coefficients: absorbed ^{144}Ce dose to bone marrow per unit activity inhaled (committed equivalent doses), Gy Bq $^{-1}$: 5 year old children	3.99E-10	1.38E-07	2.24E-06
Question 17 Intake dose coefficients: absorbed ^{144}Ce dose to liver per unit activity inhaled (committed equivalent doses), Gy Bq $^{-1}$: 5 year old children	6.40E-10	1.87E-07	1.24E-06
Question 17 Intake dose coefficients: absorbed ^{239}Pu dose to bone surface per unit activity inhaled (committed equivalent doses), Gy Bq $^{-1}$: 5 year old children	1.24E-06	2.67E-05	1.99E-03
Question 17 Intake dose coefficients: absorbed ^{239}Pu dose to bone marrow per unit activity inhaled (committed equivalent doses), Gy Bq $^{-1}$: 5 year old children	7.24E-08	1.93E-06	2.09E-04
Question 17 Intake dose coefficients: absorbed ^{239}Pu dose to liver per unit activity inhaled (committed equivalent doses), Gy Bq $^{-1}$: 5 year old children	4.54E-07	7.45E-06	5.65E-04
Question 17 Intake dose coefficients: absorbed ^{239}Pu dose to lung per unit activity inhaled (committed equivalent doses), Gy Bq $^{-1}$: 5 year old children	2.69E-07	1.28E-05	3.98E-04
Question 17 Intake dose coefficients: absorbed ^{239}Pu dose to bone surface per unit activity ingestion (committed equivalent doses), Gy Bq $^{-1}$: 5 year old children	1.60E-09	5.51E-07	2.04E-05
Question 17 Intake dose coefficients: absorbed ^{239}Pu dose to bone marrow per unit activity ingestion (committed equivalent doses), Gy Bq $^{-1}$: 5 year old children	5.15E-11	2.67E-08	1.76E-06
Question 17 Intake dose coefficients: absorbed ^{239}Pu dose to liver per unit activity ingestion (committed equivalent doses), Gy Bq $^{-1}$: 5 year old children	9.36E-10	1.93E-07	6.77E-06
Question 17 Intake dose coefficients: absorbed ^{239}Pu dose to colon per unit activity ingestion (committed equivalent doses), Gy Bq $^{-1}$: 5 year old children	1.21E-09	2.75E-08	4.09E-07
Question 17 Intake dose coefficients: absorbed ^{90}Sr dose to lung per unit activity inhaled (committed equivalent doses), Gy Bq $^{-1}$: adults	1.54E-10	1.44E-07	8.20E-07
Question 17 Intake dose coefficients: absorbed ^{90}Sr dose to bone marrow per unit activity inhaled (committed equivalent doses), Gy Bq $^{-1}$: adults	1.09E-08	1.10E-07	3.87E-07
Question 17 Intake dose coefficients: absorbed ^{90}Sr dose to bone surface per unit activity inhaled (committed equivalent doses), Gy Bq $^{-1}$: adults	2.71E-08	2.32E-07	7.48E-07
Question 17 Intake dose coefficients: absorbed ^{90}Sr dose to colon per unit activity ingested (committed equivalent doses), Gy Bq $^{-1}$: adults	1.33E-10	1.42E-08	8.04E-08
Question 17 Intake dose coefficients: absorbed ^{90}Sr dose to bone marrow per unit activity ingested (committed equivalent doses), Gy Bq $^{-1}$: adults	3.35E-09	1.01E-07	8.10E-07
Question 17 Intake dose coefficients: absorbed ^{90}Sr dose to bone surface per unit activity ingested (committed equivalent doses), Gy Bq $^{-1}$: adults	4.22E-09	2.08E-07	1.64E-06
Question 17 Intake dose coefficients: absorbed ^{131}I dose to thyroid per unit activity inhaled (committed equivalent doses), Gy Bq $^{-1}$: adults	6.19E-08	2.30E-07	8.60E-07
Question 17 Intake dose coefficients: absorbed ^{131}I dose to thyroid per unit activity ingested (committed equivalent doses), Gy Bq $^{-1}$: adults	1.31E-07	4.37E-07	1.12E-06

**Table 1. Aggregated results of Internal Dosimetry Panel expert responses
for Probabilistic Accident Consequence Uncertainty Analysis (Continued)**

Question 17 Intake dose coefficients: absorbed ^{132}Te dose to lung per unit activity inhaled (committed equivalent doses), Gy Bq $^{-1}$: <i>adults</i>	6.60E-11	8.88E-10	2.77E-08
Question 17 Intake dose coefficients: absorbed ^{132}Te dose to thyroid per unit activity inhaled (committed equivalent doses), Gy Bq $^{-1}$: <i>adults</i>	2.43E-11	2.69E-08	1.92E-07
Question 17 Intake dose coefficients: absorbed ^{132}Te dose to colon per unit activity inhaled (committed equivalent doses), Gy Bq $^{-1}$: <i>adults</i>	4.00E-10	2.17E-09	8.32E-09
Question 17 Intake dose coefficients: absorbed ^{137}Cs dose to lung per unit activity inhaled (committed equivalent doses), Gy Bq $^{-1}$: <i>adults</i>	1.55E-09	9.10E-09	8.11E-08
Question 17 Intake dose coefficients: absorbed ^{137}Cs dose to colon per unit activity inhaled (committed equivalent doses), Gy Bq $^{-1}$: <i>adults</i>	7.00E-10	4.60E-09	1.29E-07
Question 17 Intake dose coefficients: absorbed ^{137}Cs dose to stomach per unit activity inhaled (committed equivalent doses), Gy Bq $^{-1}$: <i>adults</i>	1.27E-09	3.86E-09	9.88E-09
Question 17 Intake dose coefficients: absorbed ^{137}Cs dose to bone marrow per unit activity inhaled (committed equivalent doses), Gy Bq $^{-1}$: <i>adults</i>	1.28E-09	3.89E-09	9.92E-09
Question 17 Intake dose coefficients: absorbed ^{137}Cs dose to lung per unit activity ingested (committed equivalent doses), Gy Bq $^{-1}$: <i>adults</i>	6.53E-09	1.26E-08	2.63E-08
Question 17 Intake dose coefficients: absorbed ^{137}Cs dose to colon per unit activity ingested (committed equivalent doses), Gy Bq $^{-1}$: <i>adults</i>	7.36E-09	1.50E-08	3.22E-08
Question 17 Intake dose coefficients: absorbed ^{137}Cs dose to stomach per unit activity ingested (committed equivalent doses), Gy Bq $^{-1}$: <i>adults</i>	6.90E-09	1.30E-08	2.70E-08
Question 17 Intake dose coefficients: absorbed ^{137}Cs dose to bone marrow per unit activity ingested (committed equivalent doses), Gy Bq $^{-1}$: <i>adults</i>	6.90E-09	1.28E-08	2.64E-08
Question 17 Intake dose coefficients: absorbed ^{144}Ce dose to bone surface per unit activity inhaled (committed equivalent doses), Gy Bq $^{-1}$: <i>adults</i>	9.90E-10	1.15E-07	6.26E-06
Question 17 Intake dose coefficients: absorbed ^{144}Ce dose to lung per unit activity inhaled (committed equivalent doses), Gy Bq $^{-1}$: <i>adults</i>	1.38E-09	9.82E-08	5.22E-07
Question 17 Intake dose coefficients: absorbed ^{144}Ce dose to bone marrow per unit activity inhaled (committed equivalent doses), Gy Bq $^{-1}$: <i>adults</i>	4.03E-10	2.10E-08	3.47E-06
Question 17 Intake dose coefficients: absorbed ^{144}Ce dose to liver per unit activity inhaled (committed equivalent doses), Gy Bq $^{-1}$: <i>adults</i>	3.74E-10	1.06E-07	8.75E-07
Question 17 Intake dose coefficients: absorbed ^{239}Pu dose to bone surface per unit activity inhaled (committed equivalent doses), Gy Bq $^{-1}$: <i>adults</i>	3.12E-06	3.04E-05	2.29E-03
Question 17 Intake dose coefficients: absorbed ^{239}Pu dose to bone marrow per unit activity inhaled (committed equivalent doses), Gy Bq $^{-1}$: <i>adults</i>	9.10E-08	1.36E-06	1.17E-04
Question 17 Intake dose coefficients: absorbed ^{239}Pu dose to liver per unit activity inhaled (committed equivalent doses), Gy Bq $^{-1}$: <i>adults</i>	5.49E-07	6.16E-06	4.04E-04
Question 17 Intake dose coefficients: absorbed ^{239}Pu dose to lung per unit activity inhaled (committed equivalent doses), Gy Bq $^{-1}$: <i>adults</i>	3.62E-07	8.99E-06	1.54E-04
Question 17 Intake dose coefficients: absorbed ^{239}Pu dose to bone surface per unit activity ingested (committed equivalent doses), Gy Bq $^{-1}$: <i>adults</i>	8.75E-10	3.46E-07	1.76E-05
Question 17 Intake dose coefficients: absorbed ^{239}Pu dose to bone marrow per unit activity ingested (committed equivalent doses), Gy Bq $^{-1}$: <i>adults</i>	2.20E-10	2.73E-08	9.48E-07
Question 17 Intake dose coefficients: absorbed ^{239}Pu dose to liver per unit activity ingested (committed equivalent doses), Gy Bq $^{-1}$: <i>adults</i>	2.07E-10	6.58E-08	3.78E-06

**Table 1. Aggregated results of Internal Dosimetry Panel expert responses
for Probabilistic Accident Consequence Uncertainty Analysis (Continued)**

Question 17 Intake dose coefficients: absorbed ^{239}Pu dose to colon per unit activity ingested (committed equivalent doses), Gy Bq $^{-1}$: <i>adults</i>	3.57E-10	3.31E-09	9.01E-08
Question xi number of <i>radiation-induced cancer deaths up to 40 years following exposure in population of hundred million persons</i> (5×10^7 male, 5×10^7 female) <i>each of whom inhales 10 K Bq of ^{239}Pu</i>	4.59E-01	6.36E-01	8.00E-01

<p>NRC FORM 335 (2-89) NRCM 1102, 3201, 3202</p> <p style="text-align: center;">BIBLIOGRAPHIC DATA SHEET (See instructions on the reverse)</p>		<p style="text-align: center;">U.S. NUCLEAR REGULATORY COMMISSION</p>				
<p>2. TITLE AND SUBTITLE</p> <p>Probabilistic Accident Consequence Uncertainty Analysis</p> <p>Uncertainty Assessment for Internal Dosimetry</p> <p>Appendices</p>		<p>1. REPORT NUMBER (Assigned by NRC, Add Vol., Supp., Rev., and Addendum Numbers, if any.)</p> <p style="text-align: center;">NUREG/CR-6571 EUR 16773 SAND98-0119 Vol. 2</p>				
<p>3. DATE REPORT PUBLISHED</p> <table border="1" style="width: 100%;"> <tr> <td style="width: 50%;">MONTH</td> <td style="width: 50%;">YEAR</td> </tr> <tr> <td style="text-align: center;">April</td> <td style="text-align: center;">1998</td> </tr> </table>		MONTH	YEAR	April	1998	<p>4. FIN OR GRANT NUMBER W6352</p>
MONTH	YEAR					
April	1998					
<p>5. AUTHOR(S)</p> <p>L.H.J. Goossens (TUD), J.D. Harrison (NRPB), B.C.P. Kraan (TUD), R.M. Cooke (TUD), F.T. Harper (SNL), S.C. Hora (UHH)</p>		<p>6. TYPE OF REPORT Technical</p>				
<p>7. PERIOD COVERED (Inclusive Dates)</p>						
<p>8. PERFORMING ORGANIZATION - NAME AND ADDRESS (If NRC, provide Division, Office or Region, U.S. Nuclear Regulatory Commission, and mailing address; if contractor, provide name and mailing address.)</p> <table style="width: 100%; border-collapse: collapse;"> <tr> <td style="width: 50%; vertical-align: top;"> <p>Sandia National Laboratories Albuquerque, NM 87185-0736</p> </td> <td style="width: 50%; vertical-align: top;"> <p>Commission of European Communities DG XII and XI 200, rue de la Loi B-1049 Brussels, Belgium</p> </td> </tr> </table>			<p>Sandia National Laboratories Albuquerque, NM 87185-0736</p>	<p>Commission of European Communities DG XII and XI 200, rue de la Loi B-1049 Brussels, Belgium</p>		
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<p>9. SPONSORING ORGANIZATION - NAME AND ADDRESS (If NRC, type "Same as above"; if contractor, provide NRC Division, Office or Region, U.S. Nuclear Regulatory Commission, and mailing address.)</p> <p>Division of Systems Technology Office of Nuclear Regulatory Research U.S. Nuclear Regulatory Commission Washington, DC 20555-0001</p>						
<p>10. SUPPLEMENTARY NOTES J. Randall, NRC Project Manager</p>						
<p>11. ABSTRACT (200 words or less)</p> <p>The development of two new probabilistic accident consequence codes, MACCS and COSYMA, was completed in 1990. These codes estimate the consequence from the accidental releases of radiological material from hypothesized accidents at nuclear installations. In 1991, the U.S. Nuclear Regulatory Commission and the Commission of the European Communities began cosponsoring a joint uncertainty analysis of the two codes. The ultimate objective of this joint effort was to systematically develop credible and traceable uncertainty distributions for the respective code input variables. A formal expert judgment elicitation and evaluation process was identified as the best technology available for developing a library of uncertainty distributions for these consequence parameters. This report focuses on the results of the study to develop distribution for variables related to the MACCS and COSYMA internal dosimetry models.</p>						
<p>12. KEY WORDS/DESCRIPTORS (List words or phrases that will assist researchers in locating the report.)</p> <p>uncertainty analysis, internal dosimetry, radiological health effects, accident consequence analysis, nuclear accident analysis, probabilistic analysis, expert elicitation, MACCS, COSYMA, consequence uncertainty analysis</p>		<p>13. AVAILABILITY STATEMENT unlimited</p>				
<p>14. SECURITY CLASSIFICATION (This Page)</p>		<p>unclassified</p>				
<p>15. NUMBER OF PAGES</p>		<p>unclassified</p>				
<p>16. PRICE</p>		<p>unclassified</p>				