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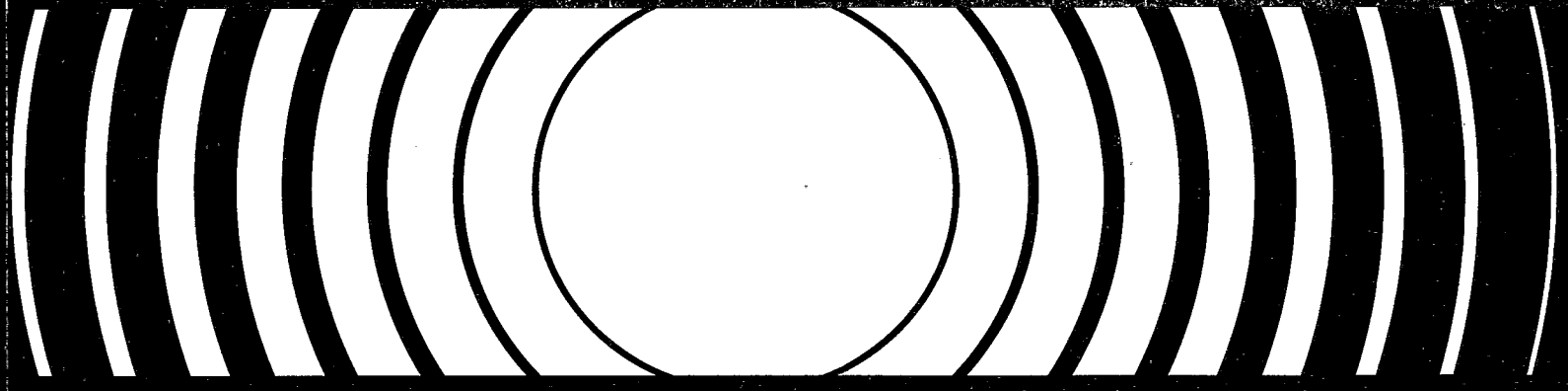
Interim Version



Federal Guidance Report No. 13
Part I – Interim Version

HEALTH RISKS FROM LOW-LEVEL ENVIRONMENTAL EXPOSURE TO RADIONUCLIDES

Radionuclide-Specific Lifetime Radiogenic Cancer Risk Coefficients for the U.S. Population, Based on Age-Dependent Intake, Dosimetry, and Risk Models

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PREFACE

The Federal Radiation Council (FRC) was formed in 1959, through Executive Order 10831. A decade later its functions were transferred to the Administrator of the newly formed Environmental Protection Agency (EPA) as part of Reorganization Plan No. 3 of 1970. Under these authorities it is the responsibility of the Administrator to "advise the President with respect to radiation matters, directly or indirectly affecting health, including guidance for all Federal agencies in the formulation of radiation standards and in the establishment and execution of programs of cooperation with States." The purpose of this guidance is to ensure that the regulation of exposure to ionizing radiation is adequately protective, reflects the best available scientific information, and is carried out in a consistent manner.

Since the mid-1980s EPA has issued a series of Federal guidance documents for the purpose of providing the Federal agencies technical information to assist their implementation of radiation protection programs. The first report in this series, Federal Guidance Report No. 10 (EPA, 1984a), presented derived concentrations of radioactivity in air and water corresponding to the limiting annual doses recommended for workers in 1960. That report was superseded in 1988 by Federal Guidance Report No. 11 (EPA, 1988), which provided dose coefficients for internal exposure of members of the general public and limiting values of radionuclide intake and air concentrations for workers, based on updated biokinetic and dosimetric models. Federal Guidance Report No. 12 (EPA, 1993) tabulated dose coefficients for external exposure to radionuclides in air, water, and soil.

When final, this report is intended to promote consistency in assessments of the risks to health from radiation by Federal agencies and others and to help ensure that such assessments are based on sound scientific information. It is intended as the first of a set of documents, referred to collectively as Federal Guidance Report No.13, that will address risks to health from exposure to specific radionuclides. These documents will make use of state-of-the-art methods and models for estimating the risks to health from internal or external exposure. These methods and models take into account, for the first time in a comprehensive compilation, the age and gender-specific aspects of radiation risk. This interim version of Federal Guidance Report No. 13, Part I, provides tabulations of risk estimates, or "risk coefficients", for cancer attributable to exposure to any of approximately 100 important radionuclides through various environmental media. These risk coefficients apply to populations that approximate the age, gender, and mortality experience characterized by the 1989-91 U.S. decennial life tables. The tabulations in the final version of Part I will extend the methodology of the interim version to the other radionuclides included in Federal Guidance Reports 11 and 12. Subsequent parts of Federal Guidance Report No. 13 may extend the

exposure pathways, and health endpoints addressed. As necessary, these publications will be reissued to update the information provided. EPA has chosen to issue Part I of Federal Guidance Report No. 13 as an interim report at this time in order to provide governmental agencies and other interested parties an opportunity to become familiar with it and its supporting methodology and to provide comments for the Agency's consideration before publishing the final version.

In this report, the risk coefficient for exposure to a given radionuclide through a given environmental medium is expressed as the probability of radiogenic cancer mortality or morbidity per unit activity inhaled or ingested, for internal exposure, or per unit time-integrated activity concentration in air or soil, for external exposure. These risk coefficients may be applied to either chronic or acute exposure to environmental radionuclides. That is, a risk coefficient may be interpreted either as average risk per unit exposure for persons exposed throughout life to a constant activity concentration of a radionuclide in an environmental medium, or as average risk per unit exposure for persons acutely exposed to the radionuclide through the environmental medium, as long as the exposure involved is properly characterized as low acute dose or low dose rate. In this report, "low dose" and "low dose rate" are defined in terms of the range of applicability of the radiogenic risk models applied, rather than as regulatory concepts.

The risk estimates tabulated in this report are intended mainly for prospective assessments of estimated cancer risks from long-term exposure to radionuclides in environmental media. For example, it is anticipated that this document will be used in such activities as preparation of environmental impact statements and development of assessments in support of generic rule making for control of radiation exposure. While it is recognized that these risk coefficients are likely also to be used in retrospective analyses of radiation exposures of populations, it is emphasized that such analyses should be limited to estimation of total or average risks in large populations. The tabulations are not intended for application to specific individuals or to age or gender subgroups, for example, children, and should not be used for that purpose. Also, these risk coefficients are based on radiation risk models developed for application either to low acute doses or low dose rates. Thus, these risk coefficients should not be applied to accident cases involving high doses and dose rates, either in prospective or retrospective analyses. Finally, some risk assessment procedures are established as a matter of policy, and additional steps may be needed before using these risk coefficients. For example, EPA recommends that radiation risk assessments for sites on the National Priorities List under the Comprehensive Environmental Response, Compensation, and Liability Act be performed using the Health Effects Assessment Summary Tables (HEAST), which are periodically updated to reflect new information.

Documents in EPA's Federal Guidance Report series provide reference values for assessing both radiation dose and risk from exposure to radionuclides. Federal Guidance Report Nos. 11 and 12, which address radiation dose, are intended for use in determining conformance with the radiation protection guidance to Federal agencies issued by the President. The present report does not replace either of those documents or affect their use for radiation protection purposes, even though many of the biokinetic and dosimetric models used here are updates of models used in Federal Guidance Report No. 11. The dose coefficients in Federal Guidance Report Nos. 11 and 12 continue to be recommended for determinations of compliance with dose-based regulations and, where applicable, for use in dose assessments. Those reports will be updated in the future as warranted. Federal Guidance Report 13 has a different purpose — it is intended for use in assessing risks from radionuclide exposure, in a variety of applications ranging from analyses of specific sites to the general analyses that support a rule making. Although its use, especially by Federal agencies, is encouraged to promote consistency in risk assessment, such use is, of course, discretionary.

This report would not have been possible without the contributions of the many investigators who produced the building blocks that provided the basis for the results presented here. These include: Jerome S. Puskin and Christopher B. Nelson, who assembled the models for age-dependent, organ-specific cancer risks; Richard W. Leggett, Keith F. Eckerman and many other contributing scientists who developed and compiled the age-specific biokinetic and dosimetric models published by the International Commission on Radiological Protection; Robert Armstrong, who supplied pre-publication values for the 1989-91U.S. decennial life tables; and Keith F. Eckerman and Richard W. Leggett, who provided the basis for calculation of doses from internal and external exposure. Allan C.B. Richardson initiated preparation of this, as well as Reports 10, 11, and 12, and provided guidance on its broad outline. The major effort required to prepare the report itself was carried out by Keith F. Eckerman, Richard W. Leggett, Christopher B. Nelson, Jerome S. Puskin, and Allan C.B. Richardson. Technical review was contributed by William J. Bair, Bernd Kahn, Charles E. Land, John R. Mauro, and Alan Phipps. Preparation of the report was funded by the U.S. Environmental Protection Agency, U.S. Department of Energy (DOE), and U.S. Nuclear Regulatory Commission (NRC). Its technical content has been reviewed by these agencies.

We gratefully acknowledge the work of the authors, the agencies who contributed funding for this work, and the helpful comments by technical reviewers of this interim version of the report. We would appreciate receiving any comments by June 30, 1998, so that they may be taken into

account in the final version, currently planned for publication in the fall of 1998. Comments should be addressed to Allan C. B. Richardson, Associate Director for Radiation Guidance, Radiation Protection Division (6602J), U.S. Environmental Protection Agency, Washington, DC 20460.

Lawrence G. Weinstock, Acting Director
Office of Radiation and Indoor Air

CONTENTS

TABLES

FIGURES

CHAPTER 1. INTRODUCTION

Since the mid-1980s, a series of Federal guidance documents have been issued by the Environmental Protection Agency (EPA) for the purpose of providing Federal agencies with technical information to assist their implementation of radiation protection programs. Previous reports have dealt with numerical factors, called "dose factors" or "dose coefficients", for estimating radiation dose due to exposure to radionuclides. The present report is the first of a set of documents, referred to collectively as Federal Guidance Report No. 13, that will provide numerical factors, called "risk coefficients", for estimating risks to health from exposure to radionuclides. Report No. 13 will apply state-of-the-art methods and models that take into account age and gender dependence of intake, metabolism, dosimetry, radiogenic risk, and competing causes of death in estimating the risks to health from internal or external exposure to radionuclides. This initial volume (Part I) provides tabulations of risk coefficients for internal or external exposure to any of over 100 radionuclides through various environmental media. It is anticipated that Part II will address most remaining radionuclides of environmental significance. Subsequent parts may further expand the exposure pathways and health endpoints considered.

The risk coefficients developed in this report apply to an average member of the public, in the sense that estimates of risk are averaged over the age and gender distributions of a hypothetical closed "stationary" population whose survival functions and cancer mortality rates are based on recent data for the U.S. Specifically, the total mortality rates in this population are defined by the 1989-91 U.S. decennial life table (NCHS, 1997) and cancer mortality rates are defined by U.S. cancer mortality data for the same period (NCHS, 1992, 1993a, 1993b). This hypothetical population is referred to as "stationary" because the gender-specific birth rates and survival functions are assumed to remain invariant over time.

For a given radionuclide and exposure mode, both a "mortality risk coefficient" and a "morbidity risk coefficient" are provided. A mortality risk coefficient is an estimate of the risk to an average member of the U.S. population, *per unit activity inhaled or ingested for internal exposures or per unit time-integrated activity concentration in air or soil for external exposures*, of death from cancer as a result of intake of the radionuclide or external exposure to its emitted radiations. A morbidity risk coefficient is a comparable estimate of the average total risk of experiencing a radiogenic cancer, whether or not the cancer is fatal. The term "risk coefficient" with no modifier should be interpreted throughout this report as "mortality or morbidity risk coefficient".

It is a common practice to estimate the cancer risk from internal or external exposure to a radionuclide as the simple product of a "probability coefficient" and an estimated "effective dose"

to a typical adult (see the Glossary for definitions). For example, a "nominal cancer fatality probability coefficient" of 0.05 Sv^{-1} is given in ICRP Publication 60 (1991) for all cancer types combined. This value is referred to as nominal because of the uncertainties inherent in radiation risk estimates and because it is based on an idealized population receiving a uniform dose over the whole body. It is pointed out by the ICRP (1991) that such a probability coefficient may be a less accurate estimator in situations where the distribution of dose is nonuniform. There are also other situations in which the product of a probability coefficient and the effective dose may not accurately represent the risk implied by current biokinetic, dosimetric, and radiation risk models. For example, such an estimate may understate the implied risk for intakes of radionuclides for which there is an apparently multiplicative effect during childhood of elevated organ doses and elevated risk per unit dose. Such an estimate may overstate the risk implied by current models in the case of intake of a long-lived, tenaciously retained radionuclide, because much of the dose may be received during late adulthood when there is a relatively high likelihood of dying from a competing cause before a radiogenic cancer can be expressed. Finally, the weighting factors commonly used to calculate effective dose do not reflect the most up-to-date knowledge of the distribution of risk among the organs and tissues of the body.

In contrast to risk estimates based on the product of a nominal probability coefficient and effective dose (for intake by the adult), the risk coefficients tabulated in this document take into account the age dependence of the biological behavior and internal dosimetry of ingested or inhaled radionuclides. Also, compared with risk estimates based on effective dose, the risk coefficients in this document characterize more precisely the implications of age and gender dependence in radiogenic risk models, U.S. cancer mortality rates, and competing risks from non-radiogenic causes of death in the U.S. Finally, these risk coefficients take into account the age and gender dependence in the usage of contaminated environmental media, which is generally not considered in risk estimates based the simple product of a nominal probability coefficient and effective dose.

Radionuclides and exposure scenarios addressed

The radionuclides addressed are listed in Table 1.1. With the exceptions noted in the table, risk coefficients are provided for the following modes of exposure to a given radionuclide: inhalation of air, ingestion of food, ingestion of tap water, external exposure from submersion in air, external exposure from the ground surface, and external exposure from soil contaminated to an infinite depth.

Table 1.1. Radionuclides addressed in this report.

H-3
C-14
S-35
Ar-37*, 39*, 41*
Ca-45, 47
Sc-47
Fe-55, 59
Co-57, 58, 60
Ni-59, 63
Zn-65
Se-75, 79
Kr-74*, 76*, 77*, 79*, 81m*, 81*, 83m*, 85m*, 85*, 87*, 88*
Br-74*, 76*, 77*
Rb-87*, 88*
Sr-89, 90
Y-90
Zr-95
Nb-94, 95m, 95
Mo-99
Tc-95m, 95, 99m, 99
Ru-103,106
Rh-103m*, 106*
Ag-108m, 108*, 110m, 110*
Sb-124, 125, 126, 127
Te-125m, 127m, 127, 129m, 129, 131m, 132
I-125, 129, 131, 132, 133, 134, 135
Xe-120*, 121*, 122*, 123*, 125*, 127*, 129m*, 131m*, 133m*, 133*, 135m*, 135*, 138*
Cs-134, 135, 136, 137, 138*
Ba-133, 137m*, 140
La-140
Ce-141, 144
Pr-144m*, 144*
Tl-207*, 208*, 209*
Pb-210, 211*, 212, 214*
Bi-210, 211*, 212, 214*
Po-210, 211*, 212*, 214*, 215*, 216*, 218*
Rn-218*, 219*, 220*, 222*
Fr-223*
Ra-223, 224, 226, 228
Ac-227, 228
Pa-231, 233, 234m*, 234
Th-227, 228, 230, 231, 232, 234
U-232, 233, 234, 235, 236, 238
Np-236a ($T_{1/2}$, 1.15×10^5 y), 236b ($T_{1/2}$, 22.5 h), 237, 239
Pu-236, 238, 239, 240, 241, 242
Am-241, 243
Cm-242, 243, 244

*Risk coefficients are provided only for external exposure scenarios.

For internal exposure, attention has been restricted mainly to radionuclides addressed in the ICRP's series of documents on age-dependent doses to the public from intake of radionuclides (ICRP, 1989, 1993, 1995a, 1995b, 1996). However, risk coefficients for internal exposure are also provided for some additional isotopes of the elements considered in that series, as well as for radionuclides with half-lives of one hour or greater that occur in the decay chains of any of the radionuclides considered in the internal exposure scenarios. For external exposure, risk coefficients are provided for all radionuclides addressed in the internal exposure scenarios and all radionuclides of potential dosimetric importance occurring in the decay chains of those radionuclides (regardless of the radiological half-life), as well as for some important radioisotopes of noble gases and their decay chain members.

For each of the internal exposure modes, the risk coefficient for a radionuclide includes the contribution to dose from production of decay chain members in the body after intake of the parent radionuclide, regardless of the half-lives of the decay chain members. For both internal and external exposure, a risk coefficient for a given radionuclide is based on the assumption that *this is the only radionuclide present in the environmental medium*; that is, doses due to decay chain members produced in the environment prior to intake of, or external exposure to, the radionuclide are not considered. However, a separate risk coefficient is provided for each decay chain member of potential dosimetric significance. This enables the user to assess the risks from ingrowth of radionuclides in the environment.

The risk coefficients tabulated in this report are applicable to either chronic or acute exposure to a radionuclide. That is, a risk coefficient may be interpreted either as the average risk per unit exposure to members of a population exposed throughout life to a constant concentration of a radionuclide through an environmental medium, or as the average risk per unit exposure to members of a population acutely exposed to the radionuclide through the environmental medium. For purposes of computing the risk coefficients, it was assumed that the concentration of the radionuclide in the environmental medium remains constant and that all persons in the population are exposed to that environmental medium throughout their lifetimes.

Applicability to the current U.S. population

The risk coefficients are based on exposure of a hypothetical stationary population with survival functions and cancer mortality rates similar to those of the current U.S. population, but with steady-state gender and age distributions based on these survival functions and fixed gender-specific birth rates. Due to uncertainty in the future composition of the U.S. population, the use of such a

stationary population is appropriate for consideration of long-term, chronic exposures. Because the gender-specific age distributions in the current U.S. population differ considerably from those of the hypothetical stationary population, however, the question arises as to the applicability of these risk coefficients to short-term exposures of the U.S. population that might occur in the near future. This question is addressed in Appendix D, where the tabulated risk coefficients are compared with values calculated for short-term exposure of a hypothetical population with the age and gender distributions of the 1996 U.S. population. As is the case for the hypothetical stationary population, total mortality rates in the hypothetical 1996 population during and after exposure are assumed to be those given in the 1989-91 U.S. decennial life table, and cancer mortality rates are taken to be those given by U.S. cancer mortality data for the same period. The comparison reveals only small differences in risk coefficients for the two populations.

Computation of the risk coefficients for internal exposure

A schematic of the method of computation of a risk coefficient is shown in Fig.1.1 for the case of internal exposure to a radionuclide. The main steps in the computation are shown in the numbered boxes in the figure and are summarized below.

1. Lifetime risk per unit absorbed dose at each age

For each of 14 cancer sites in the body, radiation risk models are used to calculate gender-specific values for the lifetime risk *per unit absorbed dose* received at each age. The age- and gender-specific radiation risk models are described in Chapter 7. These models are taken from a recent EPA report (EPA, 1994) that provides a methodology for calculation of radiogenic cancer risks based on a critical review of data on the Japanese

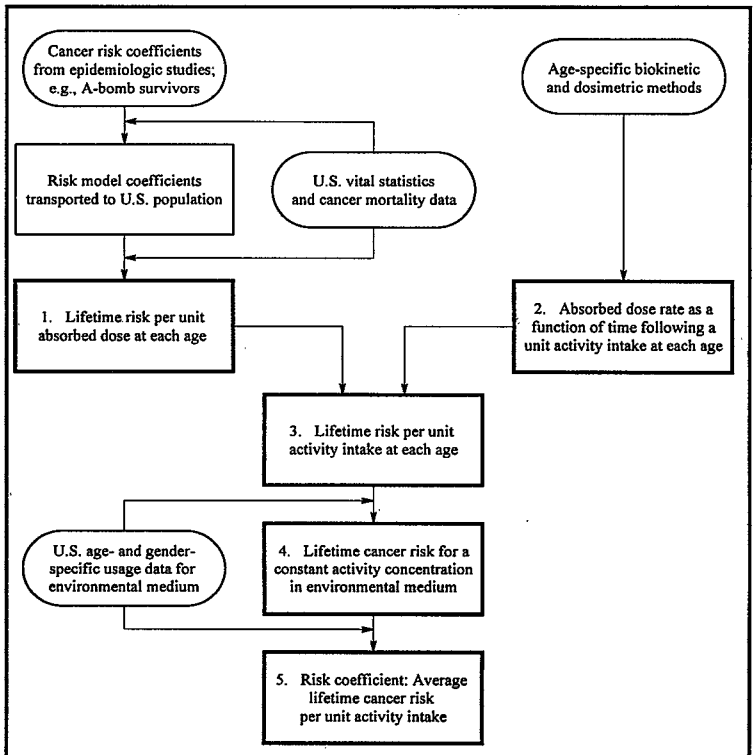

Fig. 1.1. Components of the computation of risk coefficients. (The numbers identify the key steps described in the text.)

atomic bomb survivors and other study groups and methods of transporting radiation risk estimates across populations. Parameter values given in that EPA report have been modified in some cases to reflect updated vital statistics and cancer mortality data for the U.S. and to achieve greater consistency in the assumptions made in this report for different age groups and genders.

The cancer sites considered are esophagus, stomach, colon, liver, lung, bone, skin, breast, ovary, bladder, kidney, thyroid, red marrow (leukemia), and residual (all remaining cancer sites combined). An absolute risk model is applied to bone, skin, and thyroid; that is, it is assumed for these sites that the radiogenic cancer risk is independent of the baseline cancer mortality rate, that is, the cancer mortality rate for that site in an unexposed population. For the other cancer sites, a relative risk model is used; that is, it is assumed that the likelihood of a radiogenic cancer is proportional to its baseline cancer mortality rate. The baseline cancer mortality rates are calculated from U.S. cancer mortality data for 1989-91 (NCHS, 1992, 1993a, 1993b).

The computation of gender- and cancer site-specific values for the lifetime risk per unit absorbed dose involves an integration over age, beginning at the age at which the dose is received, of the product of the age-specific risk model coefficient (times the baseline mortality rate of the cancer in the case of a relative risk model) and the survival function. The survival function is used to account for the possibility that the exposed person may die from a competing cause before a radiogenic cancer is expressed. The computation is described in detail in Chapter 7.

The estimates of lifetime risk per unit absorbed dose are independent of the radionuclide and exposure pathway. They are calculated only once and are used as input for the calculation of each risk coefficient.

2. Absorbed dose rates as a function of time post acute intake at each age

Age-specific biokinetic models are used to calculate the time-dependent inventories of activity in various regions of the body *following acute intake of a unit activity of the radionuclide*. For a given radionuclide and intake mode, this calculation is performed for each of six "basic" ages at intake: infancy (100 days); 1, 5, 10, and 15 years; and maturity (usually 20 years, but 25 years in the biokinetic models for some elements). The biokinetic models used in this document are described in Chapter 4. With a few exceptions described in that chapter, the systemic biokinetic models and gastrointestinal uptake fractions are taken from the ICRP's recent series of documents on age-specific doses to members of the public from intake of radionuclides (ICRP, 1989, 1993, 1995a, 1995b, 1996). The respiratory tract model is taken from Publication 66 of the ICRP (1994a),

and the model for transit of material through the gastrointestinal tract is taken from Publication 30 of the ICRP (Part 1, 1979).

Age-specific dosimetric models are used to convert the calculated time-dependent regional activities in the body to absorbed dose rates (per unit intake) to radiosensitive tissues as a function of age at intake and time after intake. Absorbed dose rates for intake ages intermediate to the six basic ages at intake (infancy; 1, 5, 10, and 15 years; and maturity) are determined by interpolation. The dosimetric models used in this document are the models used in the ICRP's series of documents on age-specific doses to members of the public from intake of radionuclides (ICRP, 1989, 1993, 1995a, 1995b, 1996). These models are described in Chapter 5.

3. Lifetime cancer risk per unit intake at each age

For each cancer site, the gender-specific values of lifetime risk per unit absorbed dose received at each age (derived in the first step) are used to convert the calculated absorbed dose rates to lifetime cancer risks, for the case of acute intake of one unit of activity at each age x_i. This calculation involves integration over age of the product of the absorbed dose rate at age x for a unit intake at age x_i, the lifetime risk per unit absorbed dose received at age x, and the value of the survival function at age x divided by the value at age x_i. The survival function is used to account for the probability that a person exposed at age x_i is still alive at age x to receive the absorbed dose. It is assumed that the radiation dose is sufficiently low that the survival function is not significantly affected by the number of radiogenic cancer deaths at any age. The calculation is described in Chapter 7.

4. Lifetime cancer risk for chronic intake

As indicated earlier, the risk coefficients in this document are applicable to either chronic or acute exposures. However, for purposes of computing a risk coefficient, it is assumed that the concentration of the radionuclide in the environmental medium remains constant and that all persons in the population are exposed to that environmental medium throughout their lifetimes.

The usage of environmental media may vary considerably with age and gender, and such variation is taken into account in the calculation of risk coefficients for the internal exposure scenarios. The age- and gender-specific models of usage of environmental media (air, food, or tap water) are described in Chapter 3. It is assumed that daily ingestion of a given radionuclide in food is proportional to age- and gender-specific daily energy intake. For radioisotopes of iodine, alternate

risk coefficients are calculated for food under the assumption that daily ingestion is proportional to age- and gender-specific daily usage of cow's milk. The age- and gender-specific ventilation rates applied here are reference values given by the ICRP, and age- and gender-specific usage rates for tap water, food energy, and cow's milk are average values estimated from recent data for the U.S.

For each cancer site and each gender, the lifetime cancer risk for chronic exposure is obtained by integration over age x of the product of the lifetime cancer risk per unit intake at age x and the expected intake of the environmental medium at age x. The expected intake at a given age is the product of the usage rate of the medium and the value of the survival function at that age.

5. Average lifetime cancer risk per unit activity intake

Because a risk coefficient is an expression of the radiogenic cancer risk *per unit activity intake*, the calculated lifetime cancer risk from chronic intake of the environmental medium must be divided by the expected lifetime intake. The expected lifetime intake is given by the integral over age of the product of the usage rate and the survival function.

Therefore, in the calculation of a gender- and cancer site-specific risk coefficient, usage of the environmental medium appears both in the numerator (see Step 4) and the denominator. This makes the risk coefficient independent of the concentration of the radionuclide in the medium and of the population-averaged usage rate of the medium but does not diminish the importance of the usage rate in the derivation of a risk coefficient. For example, the risk coefficient for a given radionuclide in food may differ considerably from the coefficient for the same radionuclide in tap water because the assumed age-specific patterns of consumption are substantially different for food and tap water.

Except for the calculations of the time-dependent organ activities and absorbed dose rates, each of the steps described above is performed separately for each gender and each cancer site. A total risk coefficient is derived by first adding the risk estimates for the different cancer sites in each gender and then calculating a weighted mean of the coefficients for males and females. The weighted mean of coefficients for males and females involves the presumed gender ratio at birth, the gender-specific risk per unit intake at each age, and the gender-specific survival function at each age.

Computation of the risk coefficients for external exposure

The computation of risk coefficients for external exposure scenarios is similar to that for internal exposure scenarios but involves fewer steps because the absorbed dose rates are taken

directly from Federal Guidance Report No. 12 (EPA, 1993). The methods and models used in that report are summarized in Chapter 6. As in the internal exposure scenarios, it is assumed that the concentration of the radionuclide in the environmental medium remains constant and that all persons in the population are exposed to that environmental medium throughout their lifetimes.

The external dose rates used in the calculation were based on a reference adult male, standing outside with no shielding (EPA, 1993). Although there is expected to be some variation with age in organ dose rates from uniform external exposure (usually less than 30%), comprehensive tabulations of age-specific organ dose rates due to external exposure are not yet available. In the present document, the dose rates calculated for the adult male are applied to all ages and both genders, and no adjustments are made to account for potential reduction in dose rates due to shielding by buildings during time spent indoors.

How to apply a risk coefficient

The risk coefficients in this report may be used to assess *per capita* (population-averaged) risk due to the acute exposure of a population or, equivalently, to assess the risk due to the chronic lifetime exposure of an average individual to a constant environmental concentration. They also may be used to assess the *per capita* lifetime risk in a population from a lifetime exposure to a time varying environmental radionuclide exposure (or intake) rate, using the product of the risk coefficient and the lifetime exposure (or intake) due to that time varying rate.

A risk coefficient, r, is specific to the radionuclide, the environmental medium, and the mode of exposure through that medium. For a given exposure scenario, the computation of lifetime cancer risk, R, associated with intake of, or external exposure to, a given radionuclide involves multiplication of the applicable risk coefficient r by the *per capita* activity intake I or external exposure X. Thus, $R = r \cdot I$ for intake by inhalation or ingestion and $R = r \cdot X$ for external exposure, where X denotes the time-integrated activity concentration of the radionuclide in air, on the ground surface, or within the soil, and I is the activity inhaled or ingested *per capita*.

For external exposure, estimation of the time-integrated activity concentration X requires information on the (constant or time-dependent) concentration of the radionuclide in the medium and the length of the exposure period. For an internal exposure scenario, estimation of the *per capita* activity intake I of the radionuclide requires the same information, plus an estimate of the average usage rate of the medium by members of the population during the exposure period. The user may apply the *per capita* usage rate of air, food, or tap water given in Chapter 3 (see the "combined lifetime average" usage rates in Table 3.1) or, because the risk coefficients are independent of the

usage rate of the medium, may apply an average usage rate better suited to the exposure scenario. For example, if the exposure scenario involves acute inhalation of a radionuclide in a rapidly passing cloud, the average inhalation rate in the exposed population during the exposure period may differ from the 24-h average rate given in Chapter 3. However, the assumptions described in Chapter 3 concerning *relative* age- and gender-specific usage of the environmental media are inherent in the risk coefficients for internal exposure and hence cannot be changed by the user.

Appendix E provides sample calculations that illustrate how the tabulated risk coefficients may be applied to different types of exposure.

Limitations on use of the risk coefficients

Analyses involving the risk coefficients tabulated in this report should be limited to estimation of prospective risks in hypothetical or large existing populations, or retrospective analyses of risks to large actual populations. The tabulations are not intended for application to specific individuals and should not be used for that purpose.

In contrast to situations involving representative population samples, the coefficients tabulated in this report may not be appropriate for assessing the risk to an average individual in an *age-specific* cohort due to chronic exposure to an environmental concentration that varies substantially over the life of the cohort. In such special cases, the time-varying environmental concentration must be incorporated explicitly into the calculations described in Chapter 7. Such applications are beyond the scope of this report.

The risk coefficients are based on radiation risk models developed for application either to low doses, defined as acute absorbed doses less than 0.2 Gy, or to low dose rates, defined as dose rates less than 0.1 mGy min^{-1} (EPA, 1994). Finally, the assumption is made that the absorbed dose is sufficiently low that the survival function is not significantly affected by the number of radiogenic cancer deaths at any age. Thus, these risk coefficients should be applied with care to cases involving large cumulative risks, either in prospective or retrospective analyses.

Uncertainties in the biokinetic, dosimetric, and radiation risk models

The sources and extent of uncertainties in the biokinetic, dosimetric, and radiation risk models used to derive the risk coefficients are discussed in the relevant sections of this report. The discussions of uncertainty are generally qualitative or semi-quantitative in nature and are consistent with recent assessments by experts in the various fields. Because there is not full consensus of

opinion among scientists regarding the reliability of estimates of lifetime cancer risk from low-level exposure to radiation, and because the error in such estimates may vary substantially from one radionuclide to another and one exposure scenario to another, no attempt is made here to characterize the overall uncertainty associated with any given risk coefficient.

Software used to compute the risk coefficients

All computations of dose and risk were performed using the DCAL (DOSE CALCULATION) software (Eckerman et al., to be published). DCAL is a comprehensive biokinetics-dose-risk computational system designed to serve current needs in radiation dosimetry and risk analysis. It performs biokinetic and dosimetric calculations for acute intake of a radionuclide by inhalation, ingestion, or injection into blood at a user-specified age. DCAL couples the generated absorbed dose rates with radiation risk estimators and mortality data to predict organ-specific risk of radiogenic cancer mortality or morbidity from intake of a radionuclide.

DCAL has been extensively tested and has been compared with several widely used solvers for biokinetic models and systems of differential equations. DCAL was used by a task group of the ICRP to derive or check the dose coefficients given in its series of documents on age-specific doses to members of the public from intake of radionuclides (ICRP, 1989, 1993, 1995a, 1995b, 1996).

Organization of the report

Risk coefficients for cancer mortality and morbidity due to exposure to the radionuclides listed in Table 1.1 are tabulated in Chapter 2. To facilitate comparisons as well as conversion to other units, values typically are tabulated to three decimal places. No indication of uncertainty is intended or should be inferred from this practice.

The assumptions and models used to derive the risk coefficients tabulated in Chapter 2 are described in Chapters 3 through 7. The exposure scenarios, including assumptions concerning the vital statistics of the exposed population and the age- and gender-specific usage rates of environmental media by the population, are described in Chapter 3. Biokinetic models, dosimetric models for internal exposure, dosimetric models for external exposure, and radiation risk models are described in Chapters 4, 5, 6, and 7, respectively. The sources and extent of uncertainties in the biokinetic, dosimetric, and radiation risk models are discussed in the chapters in which the respective models are described.

Some additional details concerning the models used in the calculations are given in Appendices A and B. Appendix C provides a detailed illustration of the models and computational steps involved in the derivation of a risk coefficient for ingestion or inhalation of a radionuclide. In Appendix D, the tabulated risk coefficients are compared with values calculated for short-term exposure of a hypothetical population with age and gender distributions based on the 1996 U.S. population. Appendix E provides several sample calculations that illustrate how the tabulated risk coefficients may be applied to different types of exposure. A glossary of terms is provided at the end of the document.

CHAPTER 2. TABULATIONS OF RISK COEFFICIENTS

The risk coefficients tabulated here are based on a hypothetical stationary population with total mortality rates defined by the 1989-91 U.S. decennial life table (NCHS, 1997) and cancer mortality rates defined by U.S. cancer mortality data for the same period (NCHS, 1992, 1993a, 1993b). These coefficients may be interpreted in terms of either acute or chronic exposure to environmental radionuclides. That is, a risk coefficient may be interpreted as the risk per unit exposure of a typical person exposed throughout life to a constant concentration of a radionuclide in an environmental medium, or as the average risk per unit exposure to members of a stationary population that experiences an acute exposure to that radionuclide in that environmental medium. Risk coefficients are tabulated for the following modes of exposure:

1. inhalation of a radionuclide in air (Table 2.1);
2. ingestion of a radionuclide in tap water (Table 2.2);
3. ingestion of a radionuclide in food (Table 2.3a; an alternate set of risk coefficients for radioisotopes of iodine in food is given in Table 2.3b);
4. external exposure to radiation from a radionuclide in air (Table 2.4);
5. external exposure to radiation from a radionuclide on the ground surface (Table 2.4);
6. external exposure to radiation from a radionuclide in soil, assuming contamination to an infinite depth (Table 2.4).

A risk coefficient for a given radionuclide is based on the assumption that this is the only radionuclide present in the environmental medium. In particular, ingrowth of chain members *in the environmental medium* is not considered. For each radionuclide addressed, however, a separate risk coefficient is provided for each subsequent member of the same chain that is of potential dosimetric significance.

Risk coefficients for inhalation

Risk coefficients for inhalation of radionuclides in air are given in Table 2.1. These coefficients are expressed as the risk of cancer mortality or morbidity per unit activity intake (Bq^{-1}). For cases in which one cancer type contributes heavily to the total cancer mortality, Table 2.1 also lists the dominant cancer type and the percentage of the total cancer mortality represented by that

cancer type. If no single cancer type represents more than 40% of the total cancer mortality, then none of the cancer types is considered to be dominant.

The intake rate of a radionuclide in air is assumed to depend on age and gender. The age- and gender-specific inhalation rates used in this report are given in Chapter 3, Table 3.1.

The form of the inhaled material is classified in terms of the rate of absorption from the lungs to blood, using the classification scheme of ICRP Publication 66 (ICRP, 1994a). Type F, Type M, and Type S represent fast, medium, and slow rates, respectively, of absorption of material inhaled in particulate form. Material-specific deposition and absorption models are used for vapors (Type V) and gases (Type G) (ICRP, 1995b). Although the ICRP recommends default absorption types of most of the radionuclides considered in this document, the information underlying the selection of an absorption type is often very limited and in many cases reflects occupational rather than environmental experience. Due to the uncertainties in the form of a radionuclide likely to be inhaled by members of the public, various plausible absorption types have been addressed in the derivation of a risk coefficient for inhalation of a radionuclide. The scheme for selection of plausible absorption types is described in Chapter 3.

It is assumed that airborne radioactivity is in particulate form, except that: tritium is in the form of a vapor (HTO as Type V) or a gas (HT as Type G); carbon is in gaseous form (Type G) as carbon monoxide (CO) or carbon dioxide (CO_2); iodine is in the form of a vapor (Type V), a gas (methyl iodide, CH_3I, as Type G), or a particulate (Type F or Type M); and tellurium is in the form of a vapor (Type V) or a particulate (Type F, Type M, or Type S).

Risk coefficients for inhalation of radionuclides in particulate form are based on an assumed activity median aerodynamic diameter (AMAD) of 1 μm. This particle size is recommended by the ICRP for consideration of environmental exposures in the absence of specific information about the physical characteristics of the aerosol (ICRP, 1994a).

Risk coefficients for ingestion

Ingestion of tap water

Risk coefficients for ingestion of radionuclides in tap water are given in Table 2.2. These risk coefficients are expressed as the risk of cancer mortality or morbidity per unit activity intake (Bq^{-1}). For cases in which one cancer type contributes heavily to the total cancer mortality, Table 2.2 also lists the dominant cancer type and the percentage of the total cancer mortality represented

by that cancer type. If no single cancer type represents more than 40% of the total cancer mortality, then none of the cancer types is considered to be dominant.

The age- and gender-specific usage rates for tap water are given in Chapter 3, Table 3.1. Tap water usage is defined as water drunk directly as a beverage and water added to foods and beverages during preparation. It does not include water that is intrinsic in foods as purchased.

Ingestion of food

Risk coefficients for ingestion of radionuclides in food are given in Table 2.3a. These risk coefficients are expressed as the risk of cancer mortality or morbidity per unit activity intake (Bq^{-1}). For cases in which one cancer type contributes heavily to the total cancer mortality, Table 2.3a also lists the dominant cancer type and the percentage of the total cancer mortality represented by that cancer type. If no single cancer type represents more than 40% of the total cancer mortality, then none of the cancer types is considered to be dominant.

Food usage is defined as the total dietary intake, excluding tap water. The risk coefficients for food in Table 2.3a are based on the assumption that the intake rate of the radionuclide is proportional to food energy usage (kcal d^{-1}). Age- and gender-specific values for daily usage of total food energy are given in Chapter 3, Table 3.1.

The assessment of the intake of a radionuclide in food typically is based on its activity concentration in food (for example, Bq kg^{-1}) and an average usage rate (kg d^{-1}). The relation between food energy usage and food mass usage is discussed in Chapter 3.

Table 2.3b gives a second set of risk coefficients for radioisotopes of iodine in food, based on the assumption that the intake of radioiodine is proportional to intake of cow's milk. Age- and gender-specific values for the assumed daily intake of cow's milk are given in Chapter 3, Table 3.1.

Risk coefficients for external exposure

Risk coefficients are provided in Table 2.4 for each of three external exposure scenarios: external exposure from submersion in contaminated air, external exposure from contamination on the ground surface, and external exposure from soil contaminated to an infinite depth. A risk coefficient for a given radionuclide is expressed as the probability of radiogenic cancer mortality or morbidity per unit time integrated activity concentration in air, on the ground surface, or in soil. The coefficients for submersion in air are given in units of m^3 Bq^{-1} s^{-1}, those for exposure to radiation from the ground surface are given in units of m^2 Bq^{-1} s^{-1}, and those for exposure to radiation from

soil contaminated to an infinite depth are given in units of kg Bq^{-1} s^{-1}. Because the distribution of absorbed dose within the body is fairly uniform for most external exposures, the cancer type with the highest contribution to the total risk is not shown in Table 2.4.

The risk coefficients in Table 2.4 are based on external dose rates tabulated in Federal Guidance Report No. 12 (EPA, 1993). Those dose rates were calculated for a reference adult male, standing outdoors with no shielding. Activity distributions in air, on the ground surface, or in soil were assumed to be of an infinite extent. In this report, no adjustments are made to account for potential differences with age and gender in the external doses received, potential reduction in dose due to shielding by buildings during time spent indoors, or the finite nature of the activity distribution in the environment.

Adjustments for current age and gender distributions in the U.S.

The risk coefficients tabulated in this chapter were developed for a stationary population with gender and age distributions that would eventually occur in a closed population with male-to-female birth ratios indicated by recent U.S. data and with time-invariant survival functions defined by the 1989-91 U.S. decennial life tables. Due to the uncertainty in the future composition of the U.S. population, the use of a stationary population based on recent U.S. vital statistics is judged to be appropriate for consideration of long-term, chronic exposures to the U.S. population. Because the gender-specific age distributions in the current U.S. population differ considerably from those of the hypothetical stationary population, however, the question arises as to the applicability of these risk coefficients to short-term exposures of the U.S. population that might occur in the near future. In Appendix D, risk coefficients for the stationary population are compared with coefficients derived for short-term exposure of a population with gender and age distributions based on the 1996 U.S. population, but with the same survival functions and cancer mortality rates as the stationary population. The comparisons show that the risk coefficients for the stationary population are reasonably good approximations of the corresponding risk coefficients for short-term exposure of the 1996 U.S. population and that, for a given exposure scenario, the ratio of risk coefficients for the two populations varies little from one radionuclide to another. Scaling factors are provided in Appendix D for conversion of risk coefficients for the stationary population to more precise risk coefficients for a hypothetical short-term exposure to the 1996 U.S. population.

Table 2.1. Mortality and morbidity risk coefficients for inhalation.

Explanation of Entries

Risk coefficients for inhalation of radionuclides are expressed as the probability of radiogenic cancer mortality or morbidity per unit intake, where the intake is *averaged over all ages and both genders*. The form of an inhaled radionuclide is classified in terms of the rate of absorption from the lungs to blood, using the classification scheme of ICRP Publication 66 (ICRP, 1994a). Type F, Type M, and Type S represent a fast rate, a medium rate, and a slow rate, respectively, of absorption of material inhaled in particulate form. It is assumed that airborne radioactivity is in particulate form, except that: tritium is in the form of a vapor (HTO as Type V) or a gas (HT as Type G); carbon is in gaseous form (Type G) as carbon monoxide (CO) or carbon dioxide (CO_2); iodine is in the form of a vapor (Type V), a gas (methyl iodide, CH_3I, as Type G), or a particulate (Type F or Type M); and tellurium is in the form of a vapor (Type V) or a particulate (Type F, Type M, or Type S). For all particulate matter, an activity median aerodynamic diameter (AMAD) of 1 μm is assumed. The f_1 values (gastrointestinal absorption fractions) shown are the values applied to the adult and may differ from the values applied to infants and children (see Table 4.1b).

The cancer type that makes the largest contribution to cancer mortality resulting from intake of a radionuclide is given in the column labeled "dominant cancer type", and its percentage contribution to the total cancer mortality is given in the column labeled "% total mortality". For example, the entry for ^{47}Ca in relatively soluble form (Type F) indicates that colon cancer would account for 53.9% of all cancer deaths attributable to this exposure. The entry "none" under "dominant cancer type" means that no single cancer type accounts for more than 40% of the total cancer mortality.

To facilitate application of the risk coefficients, including conversion to other units, the coefficients are tabulated to three decimal places. No indication of uncertainty is intended or should be inferred from this practice.

To express a risk coefficient in conventional units (μCi^{-1}), multiply by 3.7×10^4 Bq μCi^{-1}.

To express a risk coefficient in terms of a constant activity concentration in air (Bq m^{-3}), multiply the coefficient by 2.75×10^4 U_A, where U_A is the lifetime average inhalation rate (for example, 17.8 m^3 d^{-1} in Table 3.1) and 2.75×10^4 d is the average life span. Note that the *relative* age- and gender-specific inhalation rates indicated in Table 3.1 are inherent in the risk coefficient.

Table 2.1. Mortality and morbidity risk coefficients for inhalation.

Nuclide	AMAD (μm)	Type	f_1	Mortality (Bq^{-1})	Morbidity (Bq^{-1})	Dominant cancer type	% total mortality
Hydrogen							
H-3 (HTO)		V	1.0E+00	1.04E-12	1.52E-12	none	–
H-3 (HT)		G	1.0E+00	1.04E-14	1.52E-14	none	–
Carbon							
C-14 (CO)		G	1.0E+00	6.14E-14	9.09E-14	none	–
C-14 (CO_2)		G	1.0E+00	3.68E-13	5.39E-13	none	–
Sulphur							
S-35	1.00	F	8.0E-01	3.93E-12	6.28E-12	colon	43.8
	1.00	M	1.0E-01	1.25E-10	1.36E-10	lung	95.1
	1.00	S	1.0E-02	1.63E-10	1.77E-10	lung	96.0
Calcium							
Ca-45	1.00	F	3.0E-01	2.68E-11	3.23E-11	leukemia	71.5
	1.00	M	1.0E-01	2.35E-10	2.54E-10	lung	93.0
	1.00	S	1.0E-02	3.22E-10	3.47E-10	lung	96.5
Ca-47	1.00	F	3.0E-01	3.44E-11	5.37E-11	colon	53.9
	1.00	M	1.0E-01	1.73E-10	2.13E-10	lung	74.4
	1.00	S	1.0E-02	1.96E-10	2.40E-10	lung	75.7
Scandium							
Sc-47	1.00	M	1.0E-04	6.09E-11	7.51E-11	lung	75.3
	1.00	S	1.0E-04	6.74E-11	8.25E-11	lung	77.0
Iron							
Fe-55	1.00	F	1.0E-01	3.30E-11	4.00E-11	leukemia	53.6
	1.00	M	1.0E-01	1.81E-11	2.16E-11	leukemia	41.7
	1.00	S	1.0E-02	1.59E-11	1.75E-11	lung	88.5
Fe-59	1.00	F	1.0E-01	1.53E-10	2.15E-10	none	–
	1.00	M	1.0E-01	3.08E-10	3.60E-10	lung	76.3
	1.00	S	1.0E-02	3.48E-10	3.97E-10	lung	84.7
Cobalt							
Co-57	1.00	F	1.0E-01	1.25E-11	1.88E-11	none	–
	1.00	M	1.0E-01	4.75E-11	5.65E-11	lung	74.0
	1.00	S	1.0E-02	8.74E-11	1.01E-10	lung	80.0
Co-58	1.00	F	1.0E-01	3.12E-11	4.70E-11	none	–
	1.00	M	1.0E-01	1.34E-10	1.62E-10	lung	70.1
	1.00	S	1.0E-02	1.81E-10	2.15E-10	lung	73.4
Co-60	1.00	F	1.0E-01	3.16E-10	4.62E-10	none	–
	1.00	M	1.0E-01	8.02E-10	9.68E-10	lung	67.8
	1.00	S	1.0E-02	2.32E-09	2.72E-09	lung	73.8
Nickel							
Ni-59	1.00	F	5.0E-02	1.05E-11	1.55E-11	none	–
	1.00	M	5.0E-02	9.73E-12	1.26E-11	lung	56.0
	1.00	S	1.0E-02	3.16E-11	3.43E-11	lung	95.2
Ni-63	1.00	F	5.0E-02	2.52E-11	3.72E-11	none	–
	1.00	M	5.0E-02	3.67E-11	4.43E-11	lung	71.9
	1.00	S	1.0E-02	9.34E-11	1.01E-10	lung	96.1

Table 2.1, continued

Nuclide	AMAD (μm)	Type	f_1	Mortality (Bq^{-1})	Morbidity (Bq^{-1})	Dominant cancer type	% total mortality
Zinc							
Zn-65	1.00	F	5.0E-01	1.41E-10	2.05E-10	none	–
	1.00	M	1.0E-01	1.20E-10	1.57E-10	lung	46.1
	1.00	S	1.0E-02	1.66E-10	2.02E-10	lung	65.0
Selenium							
Se-75	1.00	F	8.0E-01	7.18E-11	1.02E-10	none	–
	1.00	M	1.0E-01	8.90E-11	1.09E-10	lung	62.7
Se-79	1.00	F	8.0E-01	6.30E-11	8.99E-11	none	–
	1.00	M	1.0E-01	2.25E-10	2.50E-10	lung	88.2
Strontium							
Sr-89	1.00	F	3.0E-01	7.60E-11	1.08E-10	colon	42.6
	1.00	M	1.0E-01	5.52E-10	6.32E-10	lung	86.0
	1.00	S	1.0E-02	7.22E-10	8.17E-10	lung	89.5
Sr-90	1.00	F	3.0E-01	1.08E-09	1.17E-09	leukemia	88.6
	1.00	M	1.0E-01	2.65E-09	2.84E-09	lung	80.5
	1.00	S	1.0E-02	1.08E-08	1.15E-08	lung	98.6
Yttrium							
Y-90	1.00	F	1.0E-04	5.77E-11	9.65E-11	colon	79.1
	1.00	M	1.0E-04	1.48E-10	2.13E-10	colon	49.7
	1.00	S	1.0E-04	1.60E-10	2.27E-10	lung	51.1
Zirconium							
Zr-95	1.00	F	2.0E-03	1.33E-10	1.77E-10	none	–
	1.00	M	2.0E-03	3.92E-10	4.47E-10	lung	81.0
	1.00	S	2.0E-03	5.06E-10	5.70E-10	lung	86.2
Niobium							
Nb-94	1.00	F	1.0E-02	3.89E-10	5.42E-10	none	–
	1.00	M	1.0E-02	8.66E-10	1.02E-09	lung	72.0
	1.00	S	1.0E-02	3.20E-09	3.64E-09	lung	80.7
Nb-95m	1.00	F	1.0E-02	1.47E-11	2.31E-11	colon	57.6
	1.00	M	1.0E-02	7.23E-11	8.84E-11	lung	75.9
	1.00	S	1.0E-02	8.13E-11	9.84E-11	lung	78.1
Nb-95	1.00	F	1.0E-02	3.89E-11	5.54E-11	none	–
	1.00	M	1.0E-02	1.26E-10	1.48E-10	lung	78.0
	1.00	S	1.0E-02	1.51E-10	1.74E-10	lung	81.7
Molybdenum							
Mo-99	1.00	F	8.0E-01	1.44E-11	2.15E-11	none	–
	1.00	M	1.0E-01	8.75E-11	1.16E-10	lung	63.0
	1.00	S	1.0E-02	9.80E-11	1.30E-10	lung	63.3
Technetium							
Tc-95m	1.00	F	8.0E-01	1.35E-11	2.16E-11	colon	40.8
	1.00	M	1.0E-01	7.51E-11	9.20E-11	lung	66.9
	1.00	S	1.0E-02	1.03E-10	1.24E-10	lung	69.8
Tc-95	1.00	F	8.0E-01	2.97E-12	5.01E-12	none	–
	1.00	M	1.0E-01	4.66E-12	7.10E-12	colon	46.4
	1.00	S	1.0E-02	4.91E-12	7.43E-12	colon	48.1

Table 2.1, continued

Nuclide	AMAD (μm)	Type	f_1	Mortality (Bq^{-1})	Morbidity (Bq^{-1})	Dominant cancer type	% total mortality
Technetium, continued							
Tc-99m	1.00	F	8.0E-01	3.62E-13	6.90E-13	none	–
	1.00	M	1.0E-01	1.20E-12	1.54E-12	lung	65.1
	1.00	S	1.0E-02	1.29E-12	1.64E-12	lung	66.4
Tc-99	1.00	F	8.0E-01	1.86E-11	3.14E-11	colon	52.0
	1.00	M	1.0E-01	3.49E-10	3.81E-10	lung	94.9
	1.00	S	1.0E-02	9.67E-10	1.03E-09	lung	98.3
Ruthenium							
Ru-103	1.00	F	5.0E-02	3.28E-11	5.12E-11	colon	40.2
	1.00	M	5.0E-02	2.12E-10	2.41E-10	lung	86.4
	1.00	S	1.0E-02	2.59E-10	2.90E-10	lung	88.7
Ru-106	1.00	F	5.0E-02	6.13E-10	9.41E-10	none	–
	1.00	M	5.0E-02	2.42E-09	2.77E-09	lung	85.9
	1.00	S	1.0E-02	5.56E-09	6.02E-09	lung	95.6
Silver							
Ag-108m	1.00	F	5.0E-02	4.09E-10	5.68E-10	none	–
	1.00	M	5.0E-02	5.82E-10	7.21E-10	lung	56.5
	1.00	S	1.0E-02	2.42E-09	2.82E-09	lung	74.3
Ag-110m	1.00	F	5.0E-02	3.90E-10	5.47E-10	none	–
	1.00	M	5.0E-02	6.22E-10	7.65E-10	lung	60.9
	1.00	S	1.0E-02	1.03E-09	1.22E-09	lung	72.0
Antimony							
Sb-124	1.00	F	1.0E-01	8.55E-11	1.30E-10	colon	45.4
	1.00	M	1.0E-02	5.65E-10	6.58E-10	lung	81.1
	1.00	S	1.0E-02	7.54E-10	8.65E-10	lung	84.3
Sb-125	1.00	F	1.0E-01	7.52E-11	1.04E-10	none	–
	1.00	M	1.0E-02	3.99E-10	4.49E-10	lung	84.7
	1.00	S	1.0E-02	9.74E-10	1.08E-09	lung	88.6
Sb-126	1.00	F	1.0E-01	5.90E-11	9.26E-11	colon	50.7
	1.00	M	1.0E-02	2.51E-10	3.10E-10	lung	70.5
	1.00	S	1.0E-02	2.85E-10	3.49E-10	lung	73.1
Sb-127	1.00	F	1.0E-01	3.50E-11	5.83E-11	colon	72.0
	1.00	M	1.0E-02	1.60E-10	2.03E-10	lung	69.8
	1.00	S	1.0E-02	1.77E-10	2.23E-10	lung	72.0
Tellurium							
Te-125m		V	3.0E-01	6.89E-11	1.02E-10	leukemia	50.9
	1.00	F	3.0E-01	2.54E-11	3.87E-11	leukemia	45.5
	1.00	M	1.0E-01	2.88E-10	3.16E-10	lung	93.4
	1.00	S	1.0E-02	3.61E-10	3.92E-10	lung	95.3
Te-127m		V	3.0E-01	2.43E-10	3.28E-10	leukemia	70.1
	1.00	F	3.0E-01	8.65E-11	1.20E-10	leukemia	64.9
	1.00	M	1.0E-01	6.34E-10	6.97E-10	lung	91.7
	1.00	S	1.0E-02	8.60E-10	9.34E-10	lung	95.6

Table 2.1, continued

Nuclide	AMAD (μm)	Type	f_1	Mortality (Bq^{-1})	Morbidity (Bq^{-1})	Dominant cancer type	% total mortality
Tellurium, continued							
Te-127		V	3.0E-01	6.01E-12	9.25E-12	colon	46.0
	1.00	F	3.0E-01	2.99E-12	5.09E-12	colon	73.1
	1.00	M	1.0E-01	1.24E-11	1.65E-11	lung	62.0
	1.00	S	1.0E-02	1.37E-11	1.83E-11	lung	62.0
Te-129m		V	3.0E-01	2.29E-10	3.66E-10	leukemia	48.7
	1.00	F	3.0E-01	9.13E-11	1.50E-10	leukemia	40.3
	1.00	M	1.0E-01	5.83E-10	6.72E-10	lung	86.0
	1.00	S	1.0E-02	7.15E-10	8.11E-10	lung	88.9
Te-129		V	3.0E-01	2.52E-12	3.07E-12	lung	67.5
	1.00	F	3.0E-01	7.77E-13	1.06E-12	none	–
	1.00	M	1.0E-01	2.26E-12	2.69E-12	lung	67.2
	1.00	S	1.0E-02	2.43E-12	2.88E-12	lung	68.3
Te-131m		V	3.0E-01	5.52E-11	2.59E-10	none	–
	1.00	F	3.0E-01	2.52E-11	9.95E-11	colon	49.1
	1.00	M	1.0E-01	7.77E-11	1.14E-10	lung	61.5
	1.00	S	1.0E-02	8.56E-11	1.13E-10	lung	63.1
Te-132		V	3.0E-01	1.40E-10	5.78E-10	none	–
	1.00	F	3.0E-01	6.08E-11	2.19E-10	colon	46.4
	1.00	M	1.0E-01	1.74E-10	2.52E-10	lung	60.4
	1.00	S	1.0E-02	1.91E-10	2.54E-10	lung	62.1
Iodine							
I-125		V	1.0E+00	7.75E-11	7.48E-10	thyroid	96.0
	(CH_3I)	G	1.0E+00	6.03E-11	5.83E-10	thyroid	96.1
	1.00	F	1.0E+00	2.97E-11	2.87E-10	thyroid	95.9
	1.00	M	1.0E-01	2.91E-11	8.71E-11	lung	63.4
I-129		V	1.0E+00	4.42E-10	4.32E-09	thyroid	97.3
	(CH_3I)	G	1.0E+00	3.43E-10	3.36E-09	thyroid	97.8
	1.00	F	1.0E+00	1.68E-10	1.64E-09	thyroid	97.7
	1.00	M	1.0E-01	2.60E-10	7.64E-10	lung	74.9
I-131		V	1.0E+00	1.48E-10	1.36E-09	thyroid	90.8
	(CH_3I)	G	1.0E+00	1.10E-10	1.06E-09	thyroid	95.0
	1.00	F	1.0E+00	5.55E-11	5.27E-10	thyroid	94.0
	1.00	M	1.0E-01	1.29E-10	2.20E-10	lung	76.8
I-132		V	1.0E+00	1.12E-11	3.12E-11	lung	54.8
	(CH_3I)	G	1.0E+00	3.88E-12	2.09E-11	thyroid	44.9
	1.00	F	1.0E+00	2.46E-12	1.01E-11	none	–
	1.00	M	1.0E-01	6.10E-12	8.72E-12	lung	51.0
I-133		V	1.0E+00	5.46E-11	4.38E-10	thyroid	77.2
	(CH_3I)	G	1.0E+00	3.76E-11	3.41E-10	thyroid	88.5
	1.00	F	1.0E+00	1.93E-11	1.69E-10	thyroid	85.2
	1.00	M	1.0E-01	4.02E-11	7.48E-11	lung	46.7
I-134		V	1.0E+00	7.41E-12	1.19E-11	lung	73.7
	(CH_3I)	G	1.0E+00	1.38E-12	5.41E-12	none	–
	1.00	F	1.0E+00	1.15E-12	2.77E-12	none	–
	1.00	M	1.0E-01	2.47E-12	3.12E-12	lung	56.8

Table 2.1, continued

Nuclide	AMAD (μm)	Type	f_1	Mortality (Bq^{-1})	Morbidity (Bq^{-1})	Dominant cancer type	% total mortality
Iodine, continued							
I-135		V	1.0E+00	1.93E-11	9.85E-11	thyroid	43.9
	(CH_3I)	G	1.0E+00	1.01E-11	7.42E-11	thyroid	67.9
	1.00	F	1.0E+00	5.57E-12	3.63E-11	thyroid	59.0
	1.00	M	1.0E-01	1.47E-11	2.38E-11	lung	47.2
Cesium							
Cs-134	1.00	F	1.0E+00	3.05E-10	4.45E-10	none	–
	1.00	M	1.0E-01	7.05E-10	8.36E-10	lung	73.4
Cs-135	1.00	F	1.0E+00	3.40E-11	5.03E-11	none	–
	1.00	M	1.0E-01	2.58E-10	2.82E-10	lung	93.2
Cs-136	1.00	F	1.0E+00	6.39E-11	9.44E-11	none	–
	1.00	M	1.0E-01	2.12E-10	2.54E-10	lung	76.1
Cs-137	1.00	F	1.0E+00	2.19E-10	3.21E-10	none	–
	1.00	M	1.0E-01	7.81E-10	8.91E-10	lung	83.7
Barium							
Ba-133	1.00	F	2.0E-01	1.23E-10	1.69E-10	none	–
	1.00	M	1.0E-01	2.67E-10	3.14E-10	lung	70.7
	1.00	S	1.0E-02	7.74E-10	8.78E-10	lung	81.4
Ba-140	1.00	F	2.0E-01	1.02E-10	1.70E-10	colon	74.8
	1.00	M	1.0E-01	4.61E-10	5.48E-10	lung	79.9
	1.00	S	1.0E-02	5.30E-10	6.20E-10	lung	82.9
Lanthanum							
La-140	1.00	F	5.0E-04	3.67E-11	5.83E-11	colon	60.5
	1.00	M	5.0E-04	8.98E-11	1.29E-10	colon	46.6
	1.00	S	5.0E-04	9.61E-11	1.37E-10	lung	47.8
Cerium							
Ce-141	1.00	F	5.0E-04	4.93E-11	6.41E-11	none	–
	1.00	M	5.0E-04	2.76E-10	3.07E-10	lung	89.8
	1.00	S	5.0E-04	3.30E-10	3.64E-10	lung	92.8
Ce-144	1.00	F	5.0E-04	1.95E-09	2.26E-09	none	–
	1.00	M	5.0E-04	2.65E-09	2.96E-09	lung	73.3
	1.00	S	5.0E-04	4.49E-09	4.87E-09	lung	95.3
Lead							
Pb-210	1.00	F	2.0E-01	1.82E-08	2.47E-08	none	–
	1.00	M	1.0E-01	6.84E-08	7.48E-08	lung	87.9
	1.00	S	1.0E-02	4.06E-07	4.28E-07	lung	99.7
Pb-212	1.00	F	2.0E-01	3.84E-10	5.43E-10	none	–
	1.00	M	1.0E-01	1.48E-08	1.56E-08	lung	99.3
	1.00	S	1.0E-02	1.64E-08	1.73E-08	lung	99.5
Bismuth							
Bi-210	1.00	F	5.0E-02	5.85E-11	9.92E-11	colon	60.3
	1.00	M	5.0E-02	8.10E-09	8.56E-09	lung	99.4
	1.00	S	5.0E-02	1.16E-08	1.23E-08	lung	99.7
Bi-212	1.00	F	5.0E-02	3.75E-10	4.10E-10	lung	92.0
	1.00	M	5.0E-02	1.99E-09	2.10E-09	lung	99.7
	1.00	S	5.0E-02	2.17E-09	2.29E-09	lung	99.9

Table 2.1, continued

Nuclide	AMAD (μm)	Type	f_1	Mortality (Bq^{-1})	Morbidity (Bq^{-1})	Dominant cancer type	% total mortality
Polonium							
Po-210	1.00	F	1.0E-01	1.97E-08	2.69E-08	none	–
	1.00	M	1.0E-01	2.76E-07	2.93E-07	lung	97.8
	1.00	S	1.0E-02	3.71E-07	3.91E-07	lung	99.9
Radium							
Ra-223	1.00	F	2.0E-01	3.91E-09	5.40E-09	none	–
	1.00	M	1.0E-01	6.42E-07	6.76E-07	lung	99.8
	1.00	S	1.0E-02	7.50E-07	7.90E-07	lung	99.9
Ra-224	1.00	F	2.0E-01	2.60E-09	3.61E-09	none	–
	1.00	M	1.0E-01	2.56E-07	2.70E-07	lung	99.7
	1.00	S	1.0E-02	2.90E-07	3.06E-07	lung	99.8
Ra-226	1.00	F	2.0E-01	5.90E-09	8.31E-09	none	–
	1.00	M	1.0E-01	2.93E-07	3.09E-07	lung	99.2
	1.00	S	1.0E-02	7.23E-07	7.61E-07	lung	99.9
Ra-228	1.00	F	2.0E-01	2.34E-08	3.28E-08	none	–
	1.00	M	1.0E-01	1.26E-07	1.40E-07	lung	82.7
	1.00	S	1.0E-02	1.12E-06	1.18E-06	lung	99.4
Actinium							
Ac-227	1.00	F	5.0E-04	3.32E-06	4.17E-06	liver	43.3
	1.00	M	5.0E-04	2.35E-06	2.72E-06	lung	48.5
	1.00	S	5.0E-04	3.88E-06	4.11E-06	lung	96.9
Ac-228	1.00	F	5.0E-04	3.41E-10	4.09E-10	liver	58.5
	1.00	M	5.0E-04	8.56E-10	9.22E-10	lung	85.9
	1.00	S	5.0E-04	1.33E-09	1.41E-09	lung	98.9
Protactinium							
Pa-231	1.00	F	5.0E-04	2.31E-06	3.18E-06	bone	51.8
	1.00	M	5.0E-04	1.19E-06	1.53E-06	none	–
	1.00	S	5.0E-04	1.19E-06	1.29E-06	lung	91.4
Pa-233	1.00	F	5.0E-04	5.57E-11	7.32E-11	leukemia	47.3
	1.00	M	5.0E-04	2.92E-10	3.28E-10	lung	88.3
	1.00	S	5.0E-04	3.45E-10	3.84E-10	lung	91.3
Pa-234	1.00	F	5.0E-04	7.80E-12	1.25E-11	colon	61.7
	1.00	M	5.0E-04	2.80E-11	3.67E-11	lung	61.5
	1.00	S	5.0E-04	3.02E-11	3.94E-11	lung	62.9
Thorium							
Th-227	1.00	M	5.0E-04	7.23E-07	7.62E-07	lung	99.7
	1.00	S	5.0E-04	9.00E-07	9.48E-07	lung	100.0
Th-228	1.00	M	5.0E-04	2.03E-06	2.18E-06	lung	93.5
	1.00	S	5.0E-04	3.40E-06	3.58E-06	lung	99.8
Th-230	1.00	M	5.0E-04	5.28E-07	6.36E-07	lung	54.9
	1.00	S	5.0E-04	7.23E-07	7.70E-07	lung	96.4
Th-231	1.00	M	5.0E-04	2.95E-11	3.78E-11	lung	69.3
	1.00	S	5.0E-04	3.23E-11	4.10E-11	lung	70.9
Th-232	1.00	M	5.0E-04	5.18E-07	6.45E-07	lung	46.8
	1.00	S	5.0E-04	1.10E-06	1.17E-06	lung	97.0

Table 2.1, continued

Nuclide	AMAD (μm)	Type	f_1	Mortality (Bq^{-1})	Morbidity (Bq^{-1})	Dominant cancer type	% total mortality
Thorium, continued							
Th-234	1.00	M	5.0E-04	6.06E-10	7.16E-10	lung	80.0
	1.00	S	5.0E-04	7.11E-10	8.31E-10	lung	84.7
Uranium							
U-232	1.00	F	2.0E-02	7.11E-08	9.96E-08	none	–
	1.00	M	2.0E-02	4.86E-07	5.26E-07	lung	92.3
	1.00	S	2.0E-03	2.37E-06	2.50E-06	lung	99.5
U-233	1.00	F	2.0E-02	1.23E-08	1.74E-08	none	–
	1.00	M	2.0E-02	2.96E-07	3.13E-07	lung	98.6
	1.00	S	2.0E-03	7.27E-07	7.65E-07	lung	99.9
U-234	1.00	F	2.0E-02	1.20E-08	1.70E-08	none	–
	1.00	M	2.0E-02	2.90E-07	3.08E-07	lung	98.6
	1.00	S	2.0E-03	7.14E-07	7.51E-07	lung	99.9
U-235	1.00	F	2.0E-02	1.12E-08	1.59E-08	none	–
	1.00	M	2.0E-02	2.57E-07	2.73E-07	lung	98.5
	1.00	S	2.0E-03	6.42E-07	6.77E-07	lung	99.9
U-236	1.00	F	2.0E-02	1.13E-08	1.61E-08	none	–
	1.00	M	2.0E-02	2.68E-07	2.83E-07	lung	98.6
	1.00	S	2.0E-03	6.63E-07	6.98E-07	lung	99.9
U-238	1.00	F	2.0E-02	1.09E-08	1.54E-08	none	–
	1.00	M	2.0E-02	2.38E-07	2.52E-07	lung	98.4
	1.00	S	2.0E-03	6.07E-07	6.39E-07	lung	99.9
Neptunium							
Np-236a[†]	1.00	F	5.0E-04	4.61E-08	6.33E-08	bone	46.6
	1.00	M	5.0E-04	1.97E-08	2.64E-08	bone	42.5
	1.00	S	5.0E-04	3.06E-08	3.30E-08	lung	92.3
Np-236b[‡]	1.00	F	5.0E-04	7.71E-11	1.04E-10	none	–
	1.00	M	5.0E-04	1.97E-10	2.18E-10	lung	84.6
	1.00	S	5.0E-04	3.28E-10	3.49E-10	lung	97.9
Np-237	1.00	F	5.0E-04	3.48E-07	4.72E-07	bone	44.4
	1.00	M	5.0E-04	4.18E-07	4.79E-07	lung	70.3
	1.00	S	5.0E-04	7.32E-07	7.75E-07	lung	98.1
Np-239	1.00	F	5.0E-04	1.48E-11	2.44E-11	colon	70.5
	1.00	M	5.0E-04	8.75E-11	1.08E-10	lung	75.2
	1.00	S	5.0E-04	9.66E-11	1.18E-10	lung	76.7
Plutonium							
Pu-236	1.00	F	5.0E-04	4.92E-07	5.91E-07	liver	59.5
	1.00	M	5.0E-04	5.60E-07	6.16E-07	lung	69.1
	1.00	S	1.0E-05	7.56E-07	7.99E-07	lung	97.9
Pu-238	1.00	F	5.0E-04	1.19E-06	1.41E-06	liver	62.6
	1.00	M	5.0E-04	8.04E-07	9.07E-07	lung	46.4
	1.00	S	1.0E-05	9.06E-07	9.60E-07	lung	95.0

[†] Np-236 isomer with half-life of 1.15×10^5 y.
[‡] Np-236 isomer with half-life of 22.5 h.

Table 2.1, continued

Nuclide	AMAD (μm)	Type	f_1	Mortality (Bq^{-1})	Morbidity (Bq^{-1})	Dominant cancer type	% total mortality
Plutonium, continued							
Pu-239	1.00	F	5.0E-04	1.26E-06	1.49E-06	liver	62.9
	1.00	M	5.0E-04	7.94E-07	8.99E-07	lung	42.4
	1.00	S	1.0E-05	8.45E-07	8.96E-07	lung	94.2
Pu-240	1.00	F	5.0E-04	1.26E-06	1.50E-06	liver	62.9
	1.00	M	5.0E-04	7.95E-07	9.00E-07	lung	42.4
	1.00	S	1.0E-05	8.47E-07	8.98E-07	lung	94.2
Pu-241	1.00	F	5.0E-04	1.98E-08	2.34E-08	liver	65.2
	1.00	M	5.0E-04	7.67E-09	9.02E-09	liver	64.4
	1.00	S	1.0E-05	3.51E-09	3.82E-09	lung	73.7
Pu-242	1.00	F	5.0E-04	1.19E-06	1.42E-06	liver	62.9
	1.00	M	5.0E-04	7.46E-07	8.46E-07	lung	41.7
	1.00	S	1.0E-05	7.88E-07	8.36E-07	lung	94.1
Americium							
Am-241	1.00	F	5.0E-04	7.98E-07	1.02E-06	none	–
	1.00	M	5.0E-04	6.59E-07	7.60E-07	lung	56.3
	1.00	S	5.0E-04	9.04E-07	9.58E-07	lung	96.5
Am-243	1.00	F	5.0E-04	7.88E-07	1.00E-06	none	–
	1.00	M	5.0E-04	6.33E-07	7.31E-07	lung	55.0
	1.00	S	5.0E-04	8.58E-07	9.11E-07	lung	96.3
Curium							
Cm-242	1.00	F	5.0E-04	5.77E-08	6.80E-08	liver	63.5
	1.00	M	5.0E-04	3.84E-07	4.07E-07	lung	95.9
	1.00	S	5.0E-04	5.15E-07	5.42E-07	lung	99.9
Cm-243	1.00	F	5.0E-04	6.50E-07	8.18E-07	liver	42.6
	1.00	M	5.0E-04	6.43E-07	7.27E-07	lung	63.5
	1.00	S	5.0E-04	9.38E-07	9.93E-07	lung	97.4
Cm-244	1.00	F	5.0E-04	5.68E-07	7.11E-07	liver	44.4
	1.00	M	5.0E-04	6.10E-07	6.84E-07	lung	66.4
	1.00	S	5.0E-04	9.09E-07	9.61E-07	lung	97.8

Table 2.2 Mortality and morbidity risk coefficients for ingestion of tap water.

Explanation of Entries

Risk coefficients for ingestion of radionuclides in tap water are expressed as the probability of radiogenic cancer mortality or morbidity per unit intake, where the intake is *averaged over all ages and both genders*. With two exceptions, the risk coefficient for ingestion of a radionuclide in tap water applies to all forms of the radionuclide. For ^{3}H, separate risk coefficients are given for tritiated water (HTO) and organically bound tritium (OBT), for which different biokinetic models are recommended by the ICRP (1989). Similarly, for ^{35}S, separate risk coefficients are given for inorganic sulfur and organically bound sulfur (I-S and OBS, respectively), for which different biokinetic models also are recommended (ICRP, 1993).

The f_1 values (gastrointestinal absorption fractions) shown are the values applied to the adult and may differ from the values applied to infants and children (see Table 4.1a).

The cancer type that makes the largest contribution to cancer mortality resulting from intake of a radionuclide is given in the column labeled "dominant cancer type", and its percentage contribution to the total cancer mortality is given in the column labeled "% total mortality". For example, the entry for ingestion of ^{47}Ca indicates that colon cancer would account for 81.3% of all cancer deaths attributable to this exposure. The entry "none" under "dominant cancer type" means that no single cancer type accounts for more than 40% of the total cancer mortality.

To facilitate application of the risk coefficients, including conversion to other units, the coefficients are tabulated to three decimal places. No indication of uncertainty is intended or should be inferred from this practice.

To express a risk coefficient in conventional units (μCi^{-1}), multiply by 3.7×10^{4} Bq μCi^{-1}.

To express a risk coefficient in terms of a constant activity concentration in tap water (Bq L^{-1}), multiply the coefficient by 2.75×10^{4} U_W, where U_W is the lifetime average rate of ingestion of tap water (for example, 1.11 L d^{-1} in Table 3.1) and 2.75×10^{4} d is the average life span. Note that the *relative* age- and gender-specific ingestion rates of tap water indicated in Table 3.1 are inherent in the risk coefficient.

Table 2.2. Mortality and morbidity risk coefficients for ingestion of tap water.

Nuclide	f_1	Mortality (Bq^{-1})	Morbidity (Bq^{-1})	Dominant cancer type	% total mortality
Hydrogen					
H-3(HTO)	1.0E+00	9.44E-13	1.37E-12	none	–
H-3(OBT)	1.0E+00	2.09E-12	3.03E-12	none	–
Carbon					
C-14	1.0E+00	2.89E-11	4.20E-11	none	–
Sulfur					
S-35(I-S)	1.0E+00	8.87E-12	1.39E-11	none	–
S-35(OBS)	1.0E+00	4.99E-11	7.36E-11	none	–
Calcium					
Ca-45	3.0E-01	4.74E-11	6.68E-11	leukemia	47.1
Ca-47	3.0E-01	1.19E-10	2.04E-10	colon	81.3
Scandium					
Sc-47	1.0E-04	5.24E-11	9.44E-11	colon	97.2
Iron					
Fe-55	1.0E-01	1.81E-11	2.33E-11	leukemia	46.8
Fe-59	1.0E-01	1.36E-10	2.13E-10	colon	50.0
Cobalt					
Co-57	1.0E-01	1.70E-11	2.81E-11	colon	62.2
Co-58	1.0E-01	4.85E-11	7.97E-11	colon	60.5
Co-60	1.0E-01	2.75E-10	4.25E-10	none	–
Nickel					
Ni-59	5.0E-02	4.44E-12	7.41E-12	colon	66.1
Ni-63	5.0E-02	1.08E-11	1.81E-11	colon	66.6
Zinc					
Zn-65	5.0E-01	2.16E-10	3.15E-10	none	–
Selenium					
Se-75	8.0E-01	1.56E-10	2.20E-10	none	–
Se-79	8.0E-01	1.38E-10	1.97E-10	none	–
Strontium					
Sr-89	3.0E-01	2.10E-10	3.47E-10	colon	75.2
Sr-90	3.0E-01	1.34E-09	1.51E-09	leukemia	82.5
Yttrium					
Y-90	1.0E-04	2.70E-10	4.88E-10	colon	98.3
Zirconium					
Zr-95	1.0E-02	7.09E-11	1.24E-10	colon	85.8
Niobium					
Nb-94	1.0E-02	1.22E-10	2.10E-10	colon	80.6
Nb-95m	1.0E-02	5.49E-11	9.88E-11	colon	97.2
Nb-95	1.0E-02	3.83E-11	6.64E-11	colon	82.7
Molybdenum					
Mo-99	1.0E+00	3.12E-11	4.33E-11	none	–
Technetium					
Tc-95m	5.0E-01	2.96E-11	4.87E-11	colon	56.3
Tc-95	5.0E-01	9.24E-12	1.56E-11	colon	58.6

Table 2.2, continued

Nuclide	f_1	Mortality (Bq^{-1})	Morbidity (Bq^{-1})	Dominant cancer type	% total mortality
Technetium, continued					
Tc-99m	5.0E-01	1.22E-12	2.15E-12	colon	63.5
Tc-99	5.0E-01	4.28E-11	7.44E-11	colon	72.2
Ruthenium					
Ru-103	5.0E-02	5.88E-11	1.04E-10	colon	87.9
Ru-106	5.0E-02	6.45E-10	1.14E-09	colon	87.4
Silver					
Ag-108m	5.0E-02	1.42E-10	2.20E-10	colon	49.8
Ag-110m	5.0E-02	1.68E-10	2.67E-10	colon	56.1
Antimony					
Sb-124	1.0E-01	2.00E-10	3.48E-10	colon	85.6
Sb-125	1.0E-01	7.27E-11	1.18E-10	colon	66.6
Sb-126	1.0E-01	1.72E-10	3.00E-10	colon	83.6
Sb-127	1.0E-01	1.52E-10	2.72E-10	colon	94.4
Tellurium					
Te-125m	3.0E-01	5.42E-11	8.99E-11	colon	64.8
Te-127m	3.0E-01	1.51E-10	2.33E-10	colon	51.8
Te-127	3.0E-01	1.53E-11	2.71E-11	colon	90.9
Te-129m	3.0E-01	2.39E-10	4.14E-10	colon	75.7
Te-129	3.0E-01	3.21E-12	4.62E-12	stomach	52.9
Te-131m	3.0E-01	9.04E-11	2.23E-10	colon	79.5
Te-132	3.0E-01	1.94E-10	4.60E-10	colon	78.3
Iodine					
I-125	1.0E+00	7.14E-11	6.87E-10	thyroid	95.5
I-129	1.0E+00	4.07E-10	3.99E-09	thyroid	97.6
I-131	1.0E+00	1.31E-10	1.23E-09	thyroid	93.2
I-132	1.0E+00	6.87E-12	2.28E-11	none	–
I-133	1.0E+00	4.63E-11	3.90E-10	thyroid	81.3
I-134	1.0E+00	3.68E-12	6.76E-12	stomach	61.9
I-135	1.0E+00	1.39E-11	8.24E-11	thyroid	52.1
Cesium					
Cs-134	1.0E+00	7.91E-10	1.14E-09	none	–
Cs-135	1.0E+00	8.72E-11	1.28E-10	none	–
Cs-136	1.0E+00	1.60E-10	2.34E-10	none	–
Cs-137	1.0E+00	5.66E-10	8.22E-10	none	–
Barium					
Ba-133	2.0E-01	1.27E-10	1.84E-10	none	–
Ba-140	2.0E-01	2.30E-10	4.03E-10	colon	88.2
Lanthanum					
La-140	5.0E-04	1.67E-10	2.96E-10	colon	92.3
Cerium					
Ce-141	5.0E-04	6.93E-11	1.25E-10	colon	97.8
Ce-144	5.0E-04	5.27E-10	9.52E-10	colon	98.3
Lead					
Pb-210	2.0E-01	1.75E-08	2.38E-08	none	–
Pb-212	2.0E-01	4.23E-10	6.76E-10	colon	51.0

Table 2.2, continued

Nuclide	f_1	Mortality (Bq^{-1})	Morbidity (Bq^{-1})	Dominant cancer type	% total mortality
Bismuth					
Bi-210	5.0E-02	1.34E-10	2.41E-10	colon	95.3
Bi-212	5.0E-02	1.35E-11	1.92E-11	stomach	50.8
Polonium					
Po-210	5.0E-01	3.53E-08	4.79E-08	none	–
Radium					
Ra-223	2.0E-01	4.00E-09	6.44E-09	colon	57.7
Ra-224	2.0E-01	2.74E-09	4.50E-09	colon	61.2
Ra-226	2.0E-01	5.32E-09	7.75E-09	none	–
Ra-228	2.0E-01	2.00E-08	2.81E-08	none	–
Actinium					
Ac-227	5.0E-04	4.43E-09	5.43E-09	liver	56.5
Ac-228	5.0E-04	3.13E-11	5.41E-11	colon	85.8
Protactinium					
Pa-231	5.0E-04	4.77E-09	6.74E-09	bone	47.0
Pa-233	5.0E-04	8.34E-11	1.50E-10	colon	96.9
Pa-234	5.0E-04	4.00E-11	6.93E-11	colon	85.6
Thorium					
Th-227	5.0E-04	7.21E-10	1.28E-09	colon	93.2
Th-228	5.0E-04	1.82E-09	2.90E-09	colon	55.9
Th-230	5.0E-04	1.67E-09	2.46E-09	none	–
Th-231	5.0E-04	3.31E-11	5.96E-11	colon	97.2
Th-232	5.0E-04	1.87E-09	2.73E-09	none	–
Th-234	5.0E-04	3.46E-10	6.25E-10	colon	98.6
Uranium					
U-232	2.0E-02	5.52E-09	7.88E-09	none	–
U-233	2.0E-02	1.26E-09	1.94E-09	none	–
U-234	2.0E-02	1.24E-09	1.91E-09	none	–
U-235	2.0E-02	1.21E-09	1.88E-09	none	–
U-236	2.0E-02	1.17E-09	1.81E-09	none	–
U-238	2.0E-02	1.13E-09	1.73E-09	none	–
Neptunium					
Np-236a[†]	5.0E-04	1.78E-10	2.83E-10	colon	51.8
Np-236b[‡]	5.0E-04	1.68E-11	3.01E-11	colon	95.4
Np-237	5.0E-04	1.10E-09	1.67E-09	colon	40.4
Np-239	5.0E-04	7.70E-11	1.39E-10	colon	97.0
Plutonium					
Pu-236	5.0E-04	1.44E-09	2.02E-09	liver	40.2
Pu-238	5.0E-04	2.75E-09	3.55E-09	liver	52.7
Pu-239	5.0E-04	2.85E-09	3.64E-09	liver	53.9
Pu-240	5.0E-04	2.85E-09	3.65E-09	liver	53.8
Pu-241	5.0E-04	3.94E-11	4.77E-11	liver	62.0
Pu-242	5.0E-04	2.71E-09	3.46E-09	liver	53.9
Americium					
Am-241	5.0E-04	2.01E-09	2.81E-09	none	–

[†] Np-236 isomer with half-life of 1.15×10^5 y.
[‡] Np-236 isomer with half-life of 22.5 h.

Table 2.2, continued

Nuclide	f_1	Mortality (Bq^{-1})	Morbidity (Bq^{-1})	Dominant cancer type	% total mortality
Americium, continued					
Am-243	5.0E-04	2.00E-09	2.79E-09	none	–
Curium					
Cm-242	5.0E-04	6.15E-10	1.04E-09	colon	80.3
Cm-243	5.0E-04	1.81E-09	2.56E-09	none	–
Cm-244	5.0E-04	1.59E-09	2.26E-09	none	–

Table 2.3a. Mortality and morbidity risk coefficients for ingestion of food.

Explanation of Entries

The intake rate of a radionuclide in food (total diet, excluding tap water) is assumed to be proportional to the energy intake rate. Risk coefficients for ingestion of radionuclides in food are expressed as the probability of radiogenic cancer mortality or morbidity per unit intake, where the intake is *averaged over all ages and both genders*. With two exceptions, the risk coefficient for ingestion of a radionuclide in food applies to all forms of the radionuclide. For ^{3}H, separate risk coefficients are given for tritiated water (HTO) and organically bound tritium (OBT), because different biokinetic models are used for the two forms. Similarly, for ^{35}S, separate risk coefficients are given for inorganic sulfur (I-S) and organically bound sulfur (OBS) because different biokinetic models are applied to the two forms.

The f_1 values (gastrointestinal absorption fractions) shown are the values applied to the adult and may differ from the values applied to infants and children (see Table 4.1a).

The cancer type that makes the largest contribution to cancer mortality resulting from intake of a radionuclide is given in the column labeled "dominant cancer type", and its percentage contribution to the total cancer mortality is given in the column labeled "% total mortality". For example, the entry for ^{47}Ca indicates that colon cancer would account for 83.5% of all cancer deaths attributable to this exposure. The entry "none" under "dominant cancer type" means that no single cancer type accounts for more than 40% of the total cancer mortality.

To facilitate application of the risk coefficients, including conversion to other units, the coefficients are tabulated to three decimal places. No indication of uncertainty is intended or should be inferred from this practice.

To express a risk coefficient in conventional units (μCi^{-1}), multiply by 3.7×10^{4} Bq μCi^{-1}.

To express a risk coefficient in terms of a constant activity concentration in food (Bq kg^{-1}), multiply by 2.75×10^{4} U_F, where U_F is the lifetime average intake rate of food in terms of mass (for example, 1.2 kg d^{-1}, suggested in Chapter 3), and 2.75×10^{4} d is the average life span. To express a risk coefficient in terms of activity per unit energy (Bq $kcal^{-1}$), multiply by 2.75×10^{4} U_E, where U_E is the lifetime average intake rate of food energy (for example, 2048 kcal d^{-1} in Table 3.1). Note that the *relative* age- and gender-specific food intake rates indicated in Table 3.1 are inherent in the risk coefficient.

Table 2.3a. Mortality and morbidity risk coefficients for ingestion of food.

Nuclide	f_1	Mortality (Bq^{-1})	Morbidity (Bq^{-1})	Dominant cancer type	% total mortality
Hydrogen					
H-3(HTO)	1.0E+00	1.20E-12	1.76E-12	none	–
H-3(OBT)	1.0E+00	2.66E-12	3.89E-12	none	–
Carbon					
C-14	1.0E+00	3.68E-11	5.40E-11	none	–
Sulfur					
S-35(I-S)	1.0E+00	1.21E-11	1.90E-11	none	–
S-35(OBS)	1.0E+00	6.72E-11	1.00E-10	none	–
Calcium					
Ca-45	3.0E-01	6.27E-11	9.10E-11	colon	51.4
Ca-47	3.0E-01	1.69E-10	2.92E-10	colon	83.5
Scandium					
Sc-47	1.0E-04	7.67E-11	1.38E-10	colon	97.4
Iron					
Fe-55	1.0E-01	2.39E-11	3.14E-11	leukemia	43.6
Fe-59	1.0E-01	1.91E-10	3.01E-10	colon	52.3
Cobalt					
Co-57	1.0E-01	2.43E-11	4.03E-11	colon	63.6
Co-58	1.0E-01	6.82E-11	1.13E-10	colon	62.3
Co-60	1.0E-01	3.88E-10	6.03E-10	none	–
Nickel					
Ni-59	5.0E-02	6.26E-12	1.05E-11	colon	68.8
Ni-63	5.0E-02	1.53E-11	2.57E-11	colon	69.3
Zinc					
Zn-65	5.0E-01	2.82E-10	4.15E-10	none	–
Selenium					
Se-75	8.0E-01	2.04E-10	2.91E-10	none	–
Se-79	8.0E-01	1.82E-10	2.62E-10	none	–
Strontium					
Sr-89	3.0E-01	2.97E-10	4.96E-10	colon	78.2
Sr-90	3.0E-01	1.62E-09	1.86E-09	leukemia	79.5
Yttrium					
Y-90	1.0E-04	3.96E-10	7.16E-10	colon	98.4
Zirconium					
Zr-95	1.0E-02	1.01E-10	1.78E-10	colon	87.7
Niobium					
Nb-94	1.0E-02	1.73E-10	3.01E-10	colon	82.8
Nb-95m	1.0E-02	8.03E-11	1.45E-10	colon	97.5
Nb-95	1.0E-02	5.43E-11	9.47E-11	colon	84.7
Molybdenum					
Mo-99	1.0E+00	4.06E-11	5.71E-11	none	–
Technetium					
Tc-95m	5.0E-01	4.09E-11	6.79E-11	colon	59.1
Tc-95	5.0E-01	1.28E-11	2.17E-11	colon	61.4

Table 2.3a, continued

Nuclide	f_1	Mortality (Bq^{-1})	Morbidity (Bq^{-1})	Dominant cancer type	% total mortality
Technetium, continued					
Tc-99m	5.0E-01	1.73E-12	3.07E-12	colon	65.9
Tc-99	5.0E-01	6.17E-11	1.08E-10	colon	73.9
Ruthenium					
Ru-103	5.0E-02	8.48E-11	1.50E-10	colon	89.3
Ru-106	5.0E-02	9.35E-10	1.65E-09	colon	88.5
Silver					
Ag-108m	5.0E-02	1.92E-10	3.03E-10	colon	53.2
Ag-110m	5.0E-02	2.30E-10	3.71E-10	colon	59.4
Antimony					
Sb-124	1.0E-01	2.86E-10	5.01E-10	colon	87.4
Sb-125	1.0E-01	1.01E-10	1.66E-10	colon	70.2
Sb-126	1.0E-01	2.46E-10	4.29E-10	colon	85.6
Sb-127	1.0E-01	2.22E-10	3.97E-10	colon	95.1
Tellurium					
Te-125m	3.0E-01	7.51E-11	1.27E-10	colon	69.0
Te-127m	3.0E-01	2.03E-10	3.23E-10	colon	56.8
Te-127	3.0E-01	2.25E-11	3.97E-11	colon	91.6
Te-129m	3.0E-01	3.39E-10	5.95E-10	colon	78.9
Te-129	3.0E-01	4.55E-12	6.61E-12	stomach	51.3
Te-131m	3.0E-01	1.30E-10	3.21E-10	colon	81.1
Te-132	3.0E-01	2.78E-10	6.60E-10	colon	80.5
Iodine					
I-125	1.0E+00	9.64E-11	9.28E-10	thyroid	95.6
I-129	1.0E+00	5.31E-10	5.21E-09	thyroid	97.6
I-131	1.0E+00	1.85E-10	1.75E-09	thyroid	93.7
I-132	1.0E+00	9.22E-12	3.17E-11	none	–
I-133	1.0E+00	6.51E-11	5.58E-10	thyroid	83.1
I-134	1.0E+00	4.97E-12	9.28E-12	stomach	62.6
I-135	1.0E+00	1.90E-11	1.17E-10	thyroid	54.8
Cesium					
Cs-134	1.0E+00	9.57E-10	1.39E-09	none	–
Cs-135	1.0E+00	1.07E-10	1.59E-10	none	–
Cs-136	1.0E+00	2.05E-10	3.04E-10	none	–
Cs-137	1.0E+00	6.88E-10	1.01E-09	none	–
Barium					
Ba-133	2.0E-01	1.73E-10	2.55E-10	none	–
Ba-140	2.0E-01	3.34E-10	5.86E-10	colon	89.4
Lanthanum					
La-140	5.0E-04	2.41E-10	4.30E-10	colon	93.2
Cerium					
Ce-141	5.0E-04	1.02E-10	1.83E-10	colon	98.0
Ce-144	5.0E-04	7.73E-10	1.40E-09	colon	98.5
Lead					
Pb-210	2.0E-01	2.31E-08	3.18E-08	none	–
Pb-212	2.0E-01	5.95E-10	9.59E-10	colon	53.0

Table 2.3a, continued

Nuclide	f_1	Mortality (Bq^{-1})	Morbidity (Bq^{-1})	Dominant cancer type	% total mortality
Bismuth					
Bi-210	5.0E-02	1.95E-10	3.52E-10	colon	95.9
Bi-212	5.0E-02	1.88E-11	2.71E-11	stomach	49.8
Polonium					
Po-210	5.0E-01	4.44E-08	6.09E-08	none	–
Radium					
Ra-223	2.0E-01	5.63E-09	9.15E-09	colon	60.4
Ra-224	2.0E-01	3.88E-09	6.42E-09	colon	63.7
Ra-226	2.0E-01	7.15E-09	1.05E-08	none	–
Ra-228	2.0E-01	2.74E-08	3.86E-08	none	–
Actinium					
Ac-227	5.0E-04	5.34E-09	6.63E-09	liver	53.9
Ac-228	5.0E-04	4.52E-11	7.85E-11	colon	87.1
Protactinium					
Pa-231	5.0E-04	6.15E-09	8.73E-09	bone	44.9
Pa-233	5.0E-04	1.22E-10	2.20E-10	colon	97.2
Pa-234	5.0E-04	5.77E-11	1.00E-10	colon	86.8
Thorium					
Th-227	5.0E-04	1.05E-09	1.87E-09	colon	94.0
Th-228	5.0E-04	2.46E-09	3.99E-09	colon	60.4
Th-230	5.0E-04	2.16E-09	3.22E-09	none	–
Th-231	5.0E-04	4.86E-11	8.75E-11	colon	97.4
Th-232	5.0E-04	2.45E-09	3.60E-09	none	–
Th-234	5.0E-04	5.07E-10	9.18E-10	colon	98.7
Uranium					
U-232	2.0E-02	7.22E-09	1.04E-08	none	–
U-233	2.0E-02	1.69E-09	2.62E-09	colon	40.4
U-234	2.0E-02	1.66E-09	2.58E-09	colon	40.8
U-235	2.0E-02	1.62E-09	2.55E-09	colon	43.4
U-236	2.0E-02	1.57E-09	2.44E-09	colon	40.8
U-238	2.0E-02	1.51E-09	2.34E-09	colon	40.9
Neptunium					
Np-236a[†]	5.0E-04	2.42E-10	3.90E-10	colon	55.8
Np-236b[‡]	5.0E-04	2.46E-11	4.41E-11	colon	95.8
Np-237	5.0E-04	1.44E-09	2.24E-09	colon	45.1
Np-239	5.0E-04	1.13E-10	2.03E-10	colon	97.3
Plutonium					
Pu-236	5.0E-04	1.87E-09	2.68E-09	none	–
Pu-238	5.0E-04	3.50E-09	4.58E-09	liver	50.5
Pu-239	5.0E-04	3.63E-09	4.70E-09	liver	52.0
Pu-240	5.0E-04	3.63E-09	4.71E-09	liver	51.9
Pu-241	5.0E-04	5.07E-11	6.17E-11	liver	61.4
Pu-242	5.0E-04	3.45E-09	4.47E-09	liver	51.9
Americium					
Am-241	5.0E-04	2.56E-09	3.63E-09	none	–

[†] Np-236 isomer with half-life of 1.15×10^5 y.
[‡] Np-236 isomer with half-life of 22.5 h.

Table 2.3a, continued

Nuclide	f_1	Mortality (Bq^{-1})	Morbidity (Bq^{-1})	Dominant cancer type	% total mortality
Americium, continued					
Am-243	5.0E-04	2.54E-09	3.61E-09	none	–
Curium					
Cm-242	5.0E-04	8.65E-10	1.48E-09	colon	83.8
Cm-243	5.0E-04	2.30E-09	3.33E-09	none	–
Cm-244	5.0E-04	2.02E-09	2.93E-09	none	–

Table 2.3b. Mortality and morbidity risk coefficients for ingestion of iodine in food, based on usage of cow's milk.

Explanation of Entries

This table provides additional risk coefficients for intake of radioisotopes of iodine in diet. In this tabulation, the rate of intake of a radioisotope of iodine is assumed to be proportional to the ingestion rate of cow's milk.

Risk coefficients for ingestion of radioisotopes of iodine in cow's milk are expressed as the probability of radiogenic cancer mortality or morbidity per unit intake, where the intake is *averaged over all ages and both genders*. The cancer type that makes the largest contribution to the total cancer mortality rate is given in the column labeled "dominant cancer type", and its percentage contribution to the total radiogenic cancer mortality is given in the column labeled "% total mortality".

To facilitate application of the risk coefficients, including conversion to other units, the coefficients are tabulated to three decimal places. No indication of uncertainty is intended or should be inferred from this practice.

To express a risk coefficient in conventional units (μCi^{-1}), multiply by 3.7×10^4 Bq μCi^{-1}.

To express a risk coefficient in terms of a constant activity concentration in milk (Bq L^{-1}), multiply the coefficient by 2.75×10^4 U_M, where U_M is the lifetime average rate of ingestion of milk (for example, 0.243 L d^{-1} in Table 3.1) and 2.75×10^4 d is the average life span. Note that the *relative* age- and gender-specific energy intake rates specified in Table 3.1 are inherent in the risk coefficient.

Table 2.3b. Mortality and morbidity risk coefficients for ingestion of iodine in food, based on usage of cow's milk.

Isotope	f_1	Mortality (Bq^{-1})	Morbidity (Bq^{-1})	Dominant cancer type	% total mortality
I-125	1.0E+00	1.76E-10	1.70E-09	thyroid	95.8
I-129	1.0E+00	8.86E-10	8.69E-09	thyroid	97.7
I-131	1.0E+00	3.78E-10	3.61E-09	thyroid	94.6
I-132	1.0E+00	1.65E-11	6.33E-11	none	–
I-133	1.0E+00	1.34E-10	1.19E-09	thyroid	86.5
I-134	1.0E+00	8.64E-12	1.74E-11	stomach	61.8
I-135	1.0E+00	3.63E-11	2.43E-10	thyroid	61.3

Table 2.4. Mortality and morbidity risk coefficients for external exposure from environmental media.

Explanation of Entries

Risk coefficients are provided for each of three external exposure scenarios: submersion in contaminated air, exposure from contamination on the ground surface, and exposure from soil contaminated to an infinite depth. It is assumed that the contaminated ground surface is an infinite plane and the contaminated air or soil occupies an infinite half-space. Risk coefficients are expressed as the probability of radiogenic cancer mortality or morbidity per unit time-integrated activity concentration in air, on the ground surface, or in soil. These risk coefficients are based on the dosimetric data of Federal Guidance Report No. 12 (EPA, 1993).

Because the distribution of absorbed dose within the body is fairly uniform for most external exposures, the cancer type with the highest contribution to the total risk is not shown in this table.

To facilitate application of the risk coefficients, including conversion to other units, the coefficients are tabulated to three decimal places. No indication of uncertainty is intended or should be inferred from this practice.

To express a risk coefficient in terms of a constant activity concentration of the radionuclide in the environmental medium, multiply the coefficient by 2.37×10^{9} s.

To express a risk coefficient in conventional units of activity, multiply the coefficient by 3.7×10^{4} Bq μCi^{-1}.

To express a risk coefficient in time units of year (y), multiply the coefficient by 3.16×10^{7} s y^{-1}.

To express a risk coefficient for submersion in volume units of cm^3, multiply the coefficient by 1×10^{6} cm^3 m^{-3}.

To express a risk coefficient for ground plane in area units of cm^2, multiply the coefficient by 1×10^{4} cm^2 m^{-2}.

To express a risk coefficient for soil in mass units of g, multiply the coefficient by 1×10^{3} g kg^{-1}.

Table 2.4. Mortality and morbidity risk coefficients for external exposure from environmental media.

Nuclide	Mortality			Morbidity		
	Submersion m^3/Bq-s	Ground Plane m^2/Bq-s	Soil kg/Bq-s	Submersion m^3/Bq-s	Ground Plane m^2/Bq-s	Soil kg/Bq-s
Hydrogen						
H-3	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Carbon						
C-14	3.23E-20	5.30E-22	4.46E-21	3.66E-20	8.24E-22	6.71E-21
Sulfur						
S-35	3.79E-20	5.60E-22	5.00E-21	4.27E-20	8.68E-22	7.51E-21
Argon						
Ar-37	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Ar-39	1.46E-18	3.67E-20	3.46E-19	1.66E-18	4.39E-20	5.09E-19
Ar-41	3.38E-15	6.54E-17	3.73E-15	4.96E-15	9.60E-17	5.47E-15
Calcium						
Ca-45	1.79E-19	1.69E-21	2.28E-20	1.97E-19	2.59E-21	3.39E-20
Ca-47	2.78E-15	5.41E-17	3.06E-15	4.09E-15	7.95E-17	4.49E-15
Scandium						
Sc-47	2.46E-16	5.39E-18	2.11E-16	3.63E-16	7.92E-18	3.10E-16
Iron						
Fe-55	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Fe-59	3.09E-15	6.05E-17	3.40E-15	4.54E-15	8.90E-17	4.99E-15
Cobalt						
Co-57	2.63E-16	5.86E-18	2.07E-16	3.89E-16	8.63E-18	3.04E-16
Co-58	2.43E-15	5.07E-17	2.62E-15	3.58E-15	7.46E-17	3.84E-15
Co-60	6.55E-15	1.27E-16	7.23E-15	9.63E-15	1.87E-16	1.06E-14
Nickel						
Ni-59	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Ni-63	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Zinc						
Zn-65	1.50E-15	2.97E-17	1.64E-15	2.20E-15	4.37E-17	2.41E-15
Selenium						
Se-75	9.02E-16	1.97E-17	8.41E-16	1.33E-15	2.89E-17	1.24E-15
Se-79	4.80E-20	6.94E-22	6.25E-21	5.39E-20	1.08E-21	9.40E-21
Krypton						
Kr-74	2.81E-15	6.16E-17	2.86E-15	4.13E-15	9.03E-17	4.20E-15
Kr-76	1.01E-15	2.20E-17	1.01E-15	1.49E-15	3.24E-17	1.48E-15
Kr-77	2.43E-15	5.34E-17	2.46E-15	3.58E-15	7.83E-17	3.61E-15
Kr-79	6.09E-16	1.31E-17	6.29E-16	8.97E-16	1.92E-17	9.24E-16
Kr-81m	2.97E-16	6.45E-18	2.68E-16	4.38E-16	9.49E-18	3.94E-16
Kr-81	1.32E-17	3.06E-19	1.27E-17	1.94E-17	4.54E-19	1.87E-17
Kr-83m	4.44E-20	1.07E-20	6.99E-21	7.61E-20	1.76E-20	1.15E-20
Kr-85m	3.61E-16	8.00E-18	3.18E-16	5.33E-16	1.17E-17	4.68E-16
Kr-85	7.23E-18	2.15E-19	6.15E-18	1.00E-17	2.79E-19	9.02E-18
Kr-87	2.15E-15	4.06E-17	2.34E-15	3.16E-15	5.92E-17	3.43E-15
Kr-88	5.37E-15	9.45E-17	5.94E-15	7.89E-15	1.39E-16	8.72E-15

Table 2.4, continued

Nuclide	Mortality			Morbidity		
	Submersion m^3/Bq-s	Ground Plane m^2/Bq-s	Soil kg/Bq-s	Submersion m^3/Bq-s	Ground Plane m^2/Bq-s	Soil kg/Bq-s
Bromine						
Br-74	1.25E-14	2.19E-16	1.36E-14	1.84E-14	3.21E-16	1.99E-14
Br-76	6.95E-15	1.32E-16	7.54E-15	1.02E-14	1.93E-16	1.11E-14
Br-77	7.60E-16	1.63E-17	7.81E-16	1.12E-15	2.41E-17	1.15E-15
Rubidium						
Rb-87	3.87E-19	3.36E-21	5.25E-20	4.25E-19	5.11E-21	7.80E-20
Rb-88	1.77E-15	3.37E-17	1.96E-15	2.60E-15	4.88E-17	2.88E-15
Strontium						
Sr-89	7.30E-18	7.72E-19	4.37E-18	9.04E-18	8.25E-19	6.16E-18
Sr-90	1.24E-18	2.60E-20	2.80E-19	1.40E-18	3.20E-20	4.13E-19
Yttrium						
Y-90	1.53E-17	1.31E-18	1.16E-17	1.96E-17	1.43E-18	1.64E-17
Zirconium						
Zr-95	1.84E-15	3.85E-17	1.98E-15	2.71E-15	5.68E-17	2.91E-15
Niobium						
Nb-94	3.94E-15	8.18E-17	4.25E-15	5.79E-15	1.21E-16	6.24E-15
Nb-95m	1.44E-16	3.17E-18	1.35E-16	2.12E-16	4.68E-18	1.99E-16
Nb-95	1.91E-15	3.99E-17	2.06E-15	2.81E-15	5.88E-17	3.02E-15
Molybdenum						
Mo-99	3.71E-16	8.16E-18	3.87E-16	5.45E-16	1.18E-17	5.69E-16
Technetium						
Tc-95m	1.63E-15	3.45E-17	1.71E-15	2.40E-15	5.08E-17	2.51E-15
Tc-95	1.96E-15	4.10E-17	2.12E-15	2.89E-15	6.04E-17	3.11E-15
Tc-99m	2.79E-16	6.15E-18	2.29E-16	4.12E-16	9.06E-18	3.37E-16
Tc-99	3.38E-19	2.98E-21	4.69E-20	3.72E-19	4.53E-21	6.97E-20
Ruthenium						
Ru-103	1.14E-15	2.45E-17	1.19E-15	1.67E-15	3.61E-17	1.75E-15
Ru-106	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Rh-103m	2.17E-19	2.85E-20	4.75E-20	3.78E-19	4.79E-20	7.97E-20
Rh-106	5.36E-16	1.26E-17	5.64E-16	7.85E-16	1.80E-17	8.27E-16
Silver						
Ag-108m	3.96E-15	8.44E-17	4.19E-15	5.82E-15	1.24E-16	6.15E-15
Ag-108	5.06E-17	1.74E-18	5.01E-17	7.27E-17	2.23E-18	7.33E-17
Ag-110m	6.97E-15	1.42E-16	7.57E-15	1.03E-14	2.09E-16	1.11E-14
Ag-110	9.81E-17	3.27E-18	9.97E-17	1.41E-16	4.22E-18	1.45E-16
Antimony						
Sb-124	4.74E-15	9.22E-17	5.18E-15	6.97E-15	1.35E-16	7.61E-15
Sb-125	1.02E-15	2.22E-17	1.06E-15	1.50E-15	3.27E-17	1.55E-15
Sb-126	7.00E-15	1.48E-16	7.47E-15	1.03E-14	2.18E-16	1.10E-14
Sb-127	1.69E-15	3.61E-17	1.79E-15	2.49E-15	5.31E-17	2.63E-15
Tellurium						
Te-125m	1.32E-17	1.04E-18	3.77E-18	2.14E-17	1.65E-18	5.95E-18
Te-127m	4.49E-18	3.30E-19	1.51E-18	7.18E-18	5.21E-19	2.34E-18

Table 2.4, continued

Nuclide	Mortality			Morbidity		
	Submersion m^3/Bq-s	Ground Plane m^2/Bq-s	Soil kg/Bq-s	Submersion m^3/Bq-s	Ground Plane m^2/Bq-s	Soil kg/Bq-s
Tellurium, continued						
Te-127	1.32E-17	3.22E-19	1.22E-17	1.89E-17	4.50E-19	1.80E-17
Te-129m	7.83E-17	2.03E-18	8.05E-17	1.15E-16	2.91E-18	1.18E-16
Te-129	1.41E-16	3.63E-18	1.43E-16	2.06E-16	5.10E-18	2.10E-16
Te-131m	3.59E-15	7.31E-17	3.86E-15	5.28E-15	1.08E-16	5.66E-15
Te-131	1.03E-15	2.24E-17	1.04E-15	1.51E-15	3.26E-17	1.53E-15
Te-132	4.97E-16	1.13E-17	4.57E-16	7.35E-16	1.68E-17	6.71E-16
Iodine						
I-125	1.48E-17	1.22E-18	3.89E-18	2.41E-17	1.94E-18	6.20E-18
I-129	1.17E-17	8.05E-19	3.34E-18	1.85E-17	1.26E-18	5.22E-18
I-131	9.14E-16	1.98E-17	9.28E-16	1.35E-15	2.92E-17	1.36E-15
I-132	5.73E-15	1.19E-16	6.18E-15	8.43E-15	1.75E-16	9.08E-15
I-133	1.50E-15	3.21E-17	1.58E-15	2.20E-15	4.71E-17	2.33E-15
I-134	6.68E-15	1.36E-16	7.25E-15	9.83E-15	2.00E-16	1.06E-14
I-135	4.15E-15	7.96E-17	4.57E-15	6.10E-15	1.17E-16	6.71E-15
Xenon						
Xe-120	9.69E-16	2.15E-17	9.91E-16	1.43E-15	3.17E-17	1.46E-15
Xe-121	4.73E-15	9.04E-17	5.09E-15	6.95E-15	1.33E-16	7.48E-15
Xe-122	1.16E-16	3.03E-18	1.07E-16	1.72E-16	4.54E-18	1.57E-16
Xe-123	1.53E-15	3.20E-17	1.59E-15	2.26E-15	4.71E-17	2.33E-15
Xe-125	5.79E-16	1.31E-17	5.46E-16	8.56E-16	1.94E-17	8.03E-16
Xe-127	6.01E-16	1.37E-17	5.54E-16	8.88E-16	2.02E-17	8.15E-16
Xe-129m	4.06E-17	1.82E-18	2.44E-17	6.19E-17	2.80E-18	3.64E-17
Xe-131m	1.47E-17	6.88E-19	8.11E-18	2.24E-17	1.06E-18	1.21E-17
Xe-133m	6.30E-17	1.74E-18	5.38E-17	9.34E-17	2.61E-18	7.92E-17
Xe-133	6.59E-17	1.96E-18	3.83E-17	9.86E-17	2.93E-18	5.67E-17
Xe-135m	1.03E-15	2.24E-17	1.09E-15	1.52E-15	3.30E-17	1.59E-15
Xe-135	5.87E-16	1.29E-17	5.66E-16	8.65E-16	1.89E-17	8.31E-16
Xe-138	3.01E-15	5.61E-17	3.28E-15	4.42E-15	8.22E-17	4.81E-15
Cesium						
Cs-134	3.86E-15	8.11E-17	4.14E-15	5.68E-15	1.19E-16	6.08E-15
Cs-135	1.12E-19	1.18E-21	1.35E-20	1.23E-19	1.81E-21	2.02E-20
Cs-136	5.44E-15	1.11E-16	5.86E-15	8.01E-15	1.64E-16	8.60E-15
Cs-137	1.20E-18	3.96E-20	3.14E-19	1.37E-18	4.57E-20	4.56E-19
Cs-138	6.31E-15	1.19E-16	6.93E-15	9.27E-15	1.75E-16	1.02E-14
Cerium						
Ce-141	1.62E-16	3.69E-18	1.32E-16	2.39E-16	5.44E-18	1.94E-16
Ce-144	3.90E-17	9.61E-19	2.92E-17	5.78E-17	1.42E-18	4.30E-17
Praseodymium						
Pr-144m	1.01E-17	4.75E-19	4.99E-18	1.56E-17	7.23E-19	7.48E-18
Pr-144	1.09E-16	3.27E-18	1.14E-16	1.56E-16	4.22E-18	1.66E-16
Barium						
Ba-133	8.70E-16	1.99E-17	8.37E-16	1.28E-15	2.95E-17	1.23E-15
Ba-137m	1.47E-15	3.12E-17	1.57E-15	2.16E-15	4.60E-17	2.30E-15

Table 2.4, continued

Nuclide	Mortality			Morbidity		
	Submersion m^3/Bq-s	Ground Plane m^2/Bq-s	Soil kg/Bq-s	Submersion m^3/Bq-s	Ground Plane m^2/Bq-s	Soil kg/Bq-s
Barium, continued						
Ba-140	4.32E-16	9.57E-18	4.44E-16	6.36E-16	1.40E-17	6.52E-16
Lanthanum						
La-140	6.10E-15	1.17E-16	6.70E-15	8.96E-15	1.71E-16	9.83E-15
Thallium						
Tl-207	1.11E-17	7.10E-19	8.95E-18	1.49E-17	8.01E-19	1.30E-17
Tl-208	9.33E-15	1.62E-16	1.03E-14	1.37E-14	2.37E-16	1.51E-14
Tl-209	5.30E-15	1.03E-16	5.74E-15	7.79E-15	1.50E-16	8.42E-15
Lead						
Pb-210	2.11E-18	9.43E-20	8.06E-19	3.22E-18	1.43E-19	1.21E-18
Pb-211	1.29E-16	3.15E-18	1.34E-16	1.89E-16	4.42E-18	1.96E-16
Pb-212	3.31E-16	7.35E-18	2.97E-16	4.89E-16	1.08E-17	4.36E-16
Pb-214	5.85E-16	1.28E-17	5.72E-16	8.62E-16	1.89E-17	8.41E-16
Bismuth						
Bi-210	3.79E-18	3.89E-19	1.66E-18	4.52E-18	4.13E-19	2.36E-18
Bi-211	1.10E-16	2.41E-18	1.10E-16	1.62E-16	3.54E-18	1.61E-16
Bi-212	4.78E-16	1.01E-17	5.18E-16	7.02E-16	1.46E-17	7.60E-16
Bi-214	3.98E-15	7.65E-17	4.37E-15	5.85E-15	1.12E-16	6.41E-15
Polonium						
Po-210	2.13E-20	4.43E-22	2.30E-20	3.13E-20	6.52E-22	3.38E-20
Po-211	1.95E-17	4.06E-19	2.09E-17	2.86E-17	5.98E-19	3.07E-17
Po-212	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Po-214	2.09E-19	4.34E-21	2.26E-19	3.07E-19	6.39E-21	3.31E-19
Po-215	4.24E-19	9.21E-21	4.36E-19	6.24E-19	1.36E-20	6.41E-19
Po-216	4.24E-20	8.82E-22	4.59E-20	6.24E-20	1.30E-21	6.74E-20
Po-218	2.30E-20	4.74E-22	2.48E-20	3.38E-20	6.99E-22	3.65E-20
Radon						
Rn-218	1.86E-18	3.96E-20	1.97E-18	2.73E-18	5.83E-20	2.90E-18
Rn-219	1.33E-16	2.89E-18	1.31E-16	1.96E-16	4.25E-18	1.93E-16
Rn-220	9.40E-19	2.02E-20	9.91E-19	1.38E-18	2.97E-20	1.46E-18
Rn-222	9.67E-19	2.09E-20	1.01E-18	1.42E-18	3.08E-20	1.49E-18
Francium						
Fr-223	1.06E-16	2.94E-18	8.16E-17	1.57E-16	4.23E-18	1.20E-16
Radium						
Ra-223	2.91E-16	6.55E-18	2.53E-16	4.30E-16	9.64E-18	3.72E-16
Ra-224	2.30E-17	5.00E-19	2.17E-17	3.40E-17	7.35E-19	3.19E-17
Ra-226	1.51E-17	3.32E-19	1.33E-17	2.23E-17	4.89E-19	1.96E-17
Ra-228	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00
Actinium						
Ac-227	2.67E-19	6.91E-21	2.02E-19	3.96E-19	1.04E-20	2.98E-19
Ac-228	2.45E-15	4.99E-17	2.64E-15	3.61E-15	7.33E-17	3.88E-15
Protactinium						
Pa-231	8.41E-17	1.96E-18	8.09E-17	1.24E-16	2.92E-18	1.19E-16
Pa-233	4.58E-16	1.01E-17	4.32E-16	6.75E-16	1.49E-17	6.36E-16

Table 2.4, continued

Nuclide	Mortality			Morbidity		
	Submersion m^3/Bq-s	Ground Plane m^2/Bq-s	Soil kg/Bq-s	Submersion m^3/Bq-s	Ground Plane m^2/Bq-s	Soil kg/Bq-s
Protactinium, continued						
Pa-234m	4.17E-17	1.73E-18	4.04E-17	5.88E-17	2.11E-18	5.88E-17
Pa-234	4.77E-15	9.81E-17	5.08E-15	7.02E-15	1.44E-16	7.46E-15
Thorium						
Th-227	2.37E-16	5.30E-18	2.20E-16	3.50E-16	7.81E-18	3.24E-16
Th-228	4.24E-18	1.07E-19	3.25E-18	6.29E-18	1.60E-19	4.79E-18
Th-230	7.46E-19	2.69E-20	4.74E-19	1.12E-18	4.17E-20	7.01E-19
Th-231	2.25E-17	7.05E-19	1.42E-17	3.36E-17	1.08E-18	2.10E-17
Th-232	3.51E-19	1.73E-20	1.97E-19	5.35E-19	2.74E-20	2.93E-19
Th-234	1.50E-17	3.86E-19	9.52E-18	2.23E-17	5.74E-19	1.40E-17
Uranium						
U-232	5.66E-19	2.97E-20	3.45E-19	8.67E-19	4.78E-20	5.12E-19
U-233	7.24E-19	2.51E-20	5.70E-19	1.09E-18	3.91E-20	8.41E-19
U-234	2.79E-19	2.01E-20	1.44E-19	4.37E-19	3.29E-20	2.16E-19
U-235	3.45E-16	7.60E-18	3.02E-16	5.09E-16	1.12E-17	4.44E-16
U-236	1.66E-19	1.65E-20	7.03E-20	2.67E-19	2.73E-20	1.07E-19
U-238	9.95E-20	1.34E-20	2.70E-20	1.66E-19	2.25E-20	4.27E-20
Neptunium						
Np-236a[†]	2.48E-16	5.81E-18	1.89E-16	3.67E-16	8.62E-18	2.78E-16
Np-236b[‡]	9.99E-17	2.31E-18	7.81E-17	1.48E-16	3.41E-18	1.15E-16
Np-237	4.56E-17	1.24E-18	3.11E-17	6.79E-17	1.86E-18	4.59E-17
Np-239	3.67E-16	8.24E-18	3.15E-16	5.42E-16	1.22E-17	4.63E-16
Plutonium						
Pu-236	1.87E-19	2.33E-20	6.56E-20	3.13E-19	3.92E-20	1.02E-19
Pu-238	1.34E-19	1.95E-20	3.88E-20	2.28E-19	3.30E-20	6.18E-20
Pu-239	1.65E-19	9.99E-21	1.15E-19	2.56E-19	1.63E-20	1.71E-19
Pu-240	1.31E-19	1.88E-20	3.76E-20	2.24E-19	3.17E-20	5.98E-20
Pu-241	3.29E-21	8.44E-23	2.39E-21	4.89E-21	1.27E-22	3.52E-21
Pu-242	1.12E-19	1.57E-20	3.38E-20	1.91E-19	2.64E-20	5.35E-20
Americium						
Am-241	3.33E-17	1.11E-18	1.59E-17	5.00E-17	1.68E-18	2.36E-17
Am-243	9.45E-17	2.51E-18	5.49E-17	1.41E-16	3.71E-18	8.11E-17
Curium						
Cm-242	1.50E-19	2.20E-20	4.10E-20	2.59E-19	3.71E-20	6.62E-20
Cm-243	2.81E-16	6.31E-18	2.44E-16	4.16E-16	9.31E-18	3.59E-16
Cm-244	1.22E-19	2.00E-20	2.46E-20	2.15E-19	3.39E-20	4.15E-20

[†] Np-236 isomer with half-life of 1.15×10^5 y.

[‡] Np-236 isomer with half-life of 22.5 h.

CHAPTER 3. EXPOSURE SCENARIOS

The risk coefficients developed in this report are gender-averaged values based on biokinetic, dosimetric, and radiation risk models that represent typical or "reference" male and female members of the U.S. population, from infancy through old age. Although the coefficients may be interpreted in terms of either acute or chronic exposure, computations are based on the assumption that these persons are exposed throughout life, beginning at birth, to a constant concentration of a radionuclide in a given environmental medium. *In utero* exposures are not considered in this document.

Characteristics of the exposed population

The physical characteristics of the reference male and reference female at different ages are described in reports by Cristy and Eckerman (1987, 1993). The vital statistics for these reference persons are based on the 1989-91 U.S. decennial life table (NCHS, 1997) and U.S. cancer mortality data for the same period (NCHS, 1992, 1993a, 1993b). That is, it is assumed that the exposed male and female are subject to the risk of dying from a competing cause (any cause other than a cancer produced by the radiation exposure hypothesized here) indicated by the 1989-91 U.S. decennial life table and are subject to the risk of experiencing or dying from cancer at a specific site indicated by U.S. cancer mortality data for the same period. Gender-specific survival functions (fractions of live-born individuals surviving to different ages) for the stationary population are shown in Fig. 3.1. Methods of extending or smoothing the U.S. vital statistics for use in this report are described in Appendix A.

Growth of decay chain members

For each of the internal exposure scenarios, the risk coefficient for a radionuclide includes the contribution to dose from production of decay chain members in the body after intake of the parent radionuclide. However, for either an internal or external exposure scenario, the risk coefficient for a given radionuclide is based on the assumption that this is the only radionuclide present in the environmental medium. Growth of chain members in the environment is not considered because this would require the assumption of a temporal pattern of contamination and environmental behavior of decay chain members and thus would limit the applicability of the risk coefficients. For each of the radionuclides addressed in this document, however, a separate risk

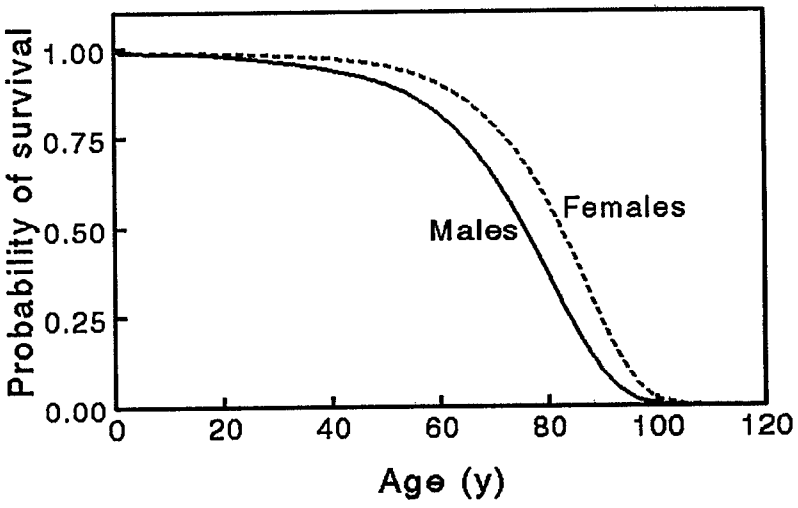

Fig. 3.1. Gender-specific survival functions for the stationary population.

coefficient is provided for any subsequent chain member that is of potential dosimetric significance. This enables the user to assess the risks from ingrowth of radionuclides in the environment.

Inhalation of radionuclides

Risk coefficients (Bq^{-1}) for inhalation of radionuclides in air are expressed as risk of cancer mortality or morbidity per unit activity intake. The age- and gender-specific inhalation rates used in this report (Table 3.1, Fig. 3.2) are taken from ICRP Publication 66 (1994a). These inhalation rates are based on breathing rates measured during periods of rest, light activity, or heavy activity. The average 24-h ventilation rate is estimated as a time-weighted average of ventilation rates for rest periods and periods of light and heavy activity.

Recently, Layton (1993) proposed a different approach for the estimation of average inhalation rates at different ages. Estimates are based on typical oxygen consumption associated with energy expenditure and are derived using the equation $V_E = E \times H \times VQ$, where V_E is the

ventilation rate (L min^{-1}), E is the average rate of energy expenditure (kilojoules min^{-1}), H is the volume of oxygen (at standard temperature and pressure) consumed in the production of 1 kilojoule

Table 3.1. Age- and gender-specific usage rates of environmental media, for selected ages.[a]

	Air[b] (m^3 d^{-1})		Tap water[c] (L d^{-1})		Food energy[d] (kcal d^{-1})		Cow's milk[e] (L d^{-1})	
Age (y)	M	F	M	F	M	F	M	F
0	2.9	2.9	0.191	0.188	478	470	0.339	0.350
1	5.2	5.2	0.223	0.216	791	752	0.349	0.358
5	8.8	8.8	0.542	0.499	1566	1431	0.413	0.409
10	15.3	15.3	0.725	0.649	1919	1684	0.486	0.428
15	20.1	15.7	0.900	0.712	2425	1828	0.519	0.356
20	22.2	17.7	1.137	0.754	2952	1927	0.414	0.249
50	22.2	17.7	1.643	1.119	2570	1758	0.192	0.139
75	22.2	17.7	1.564	1.179	1990	1508	0.192	0.139
Lifetime average	19.2	16.5	1.29	0.93	2418	1695	0.282	0.207
Combined lifetime average[f]	17.8		1.11		2048[g]		0.243	

[a]All values are based on estimated averages for the U.S. population for the indicated age. Ages refer to birthdays; e.g., a given rate at age 5 y indicates the rate on the fifth birthday. Data reported for age intervals were converted to point estimates by preserving the total intake in each interval using a cubic spline fitting method (Fritsch and Carlson, 1980). Fitted curves were smoothed using a 3-point moving average. The listed usage rates are the values used in the calculation and are generally more precise than the data would support.

[b]From Tables B.16A and B.16B of ICRP Publication 66, 1994a.

[c]Based on survey data of the U.S. Department of Agriculture (Ershow and Cantor, 1989). Includes drinking water, water added to beverages, and water added to foods during preparation, but not water intrinsic in food as purchased.

[d]Based on data from the Third National Health and Nutrition Examination Survey (McDowell et al., 1994).

[e]Used in one of two scenarios for ingestion of radioisotopes of iodine in diet. The other scenario assumes that iodine intake is proportional to food energy usage. Milk usage is based on data from EPA report 520/1-84-021 (1984b).

[f]Based on the male-to-female ratio at birth, the gender-specific survival function, and the gender-specific usage function.

[g]For a typical U.S. diet, equivalent to a lifetime average intake of about 1.2 kg food d^{-1} (see text).

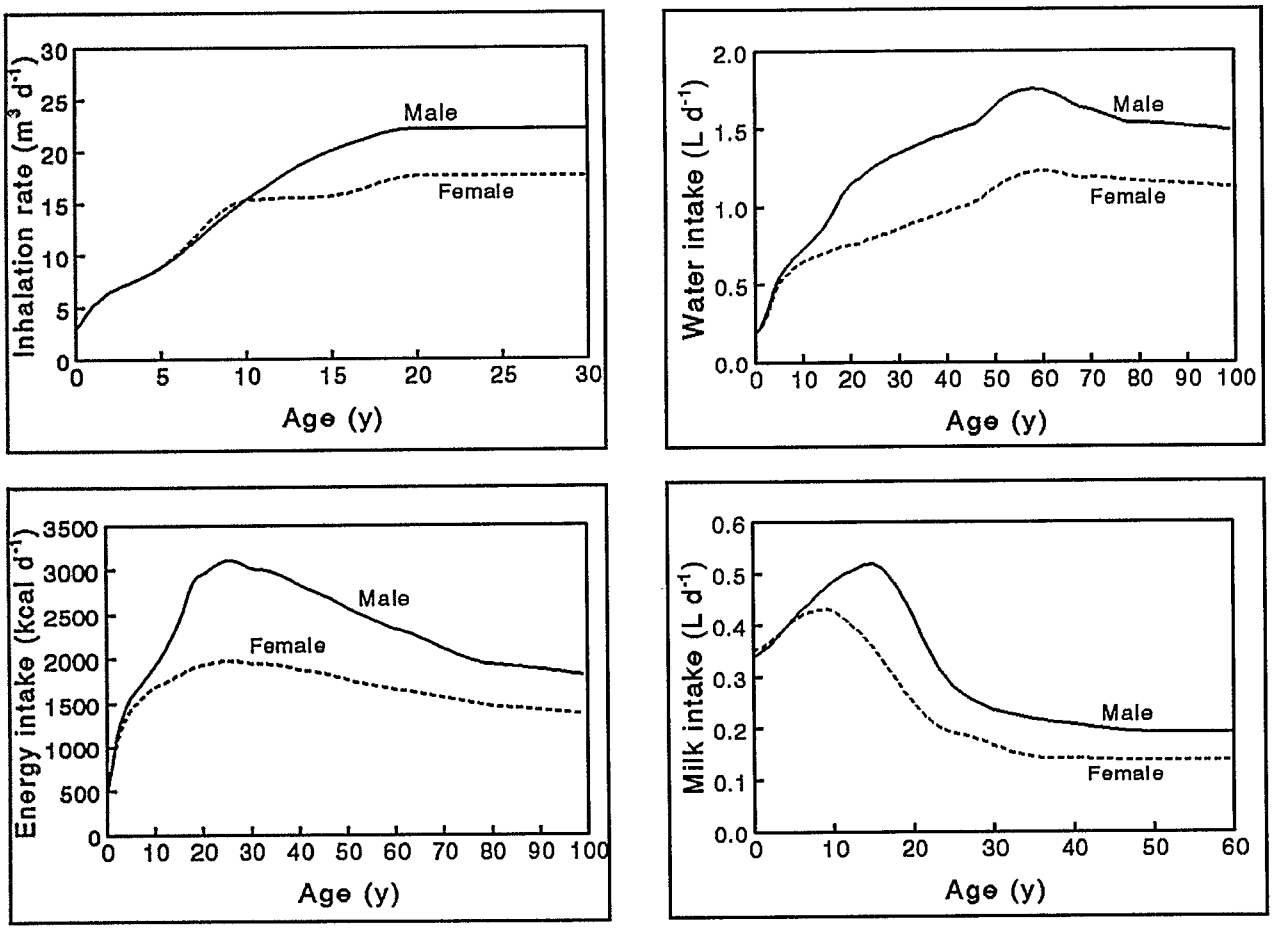

Fig. 3.2. Age- and gender-specific usage rates used to derive risk coefficients for inhalation, ingestion of tap water, ingestion of food (energy intake), and ingestion of milk.

of energy, and VQ is the ventilatory equivalent (ratio of ventilation rate to oxygen uptake rate). The value H has been determined within narrow bounds, and the average daily energy expenditure E at a given age in the U.S. population can be estimated reasonably well on the basis of data from food consumption surveys when biases in the data are taken into account. The major uncertainty in this method lies in the ventilatory equivalent, VQ. Layton concluded that VQ is nearly independent of the ventilation rate and proposed the value $VQ = 27$ for all ages and activity levels and for both genders. This value is based on data for adult humans (almost all male subjects, a large portion of which were highly trained athletes) and data from two studies on newborns. Little information is available for adult females, but results of a study on children of age 7-17 y (Zapletal et al., 1987) give a mean value for VQ of about 36 and suggest a slight increase with age, from about 35 at age

7 y to about 37 at age 17 y. Because reliable age- and gender-dependent central values for VQ have not been established, the ICRP's recommended age- and gender-specific inhalation rates, rather than rates derived from Layton's method, are applied in the present study.[1]

Risk coefficients for inhalation are based on an activity median aerodynamic diameter (AMAD) of 1 μm. This particle size is recommended by the ICRP for consideration of environmental exposures in the absence of specific information about the physical characteristics of the aerosol (ICRP, 1994a).

The rate of clearance of a radionuclide from the respiratory tract and the extent of absorption of the radionuclide to blood depend on the rate of dissolution of the inhaled particulate. For application of the ICRP's respiratory tract model (ICRP, 1994a) to radionuclides inhaled in particulate form, a given compound of a radioelement usually is assigned to one of three default absorption types: Type F, indicating fast dissolution and a high level of absorption to blood; Type M, indicating an intermediate rate of dissolution and an intermediate level of absorption to blood; and Type S, indicating slow dissolution and a low level of absorption to blood. For application of the model to radionuclides inhaled as a gas (Type G) or vapor (Type V), material-specific parameter values are applied (ICRP, 1995b).

For each of the elements addressed in the ICRP's series on doses to the public from intake of radionuclides, a recommendation is made by the ICRP concerning a default absorption type to be used in the absence of specific information (ICRP Publication 71, 1995b). For other elements, recommendations in ICRP Publication 30 (1979, 1980, 1981, 1988) concerning clearance classes are generally applied, with clearance classes D, W, and Y assumed to correspond to absorption Types F, M, and S, respectively. For some radionuclides, different default clearance classes are listed in ICRP Publication 30 for different chemical forms.

The data underlying the ICRP's selections of default absorption types are often very limited and in many cases reflect occupational rather than environmental experience. Due to the uncertainty in the form of a radionuclide likely to be inhaled by members of the public, a range of plausible absorption types is addressed in this document. For a given radionuclide, the different absorption types considered generally include the default absorption type(s) recommended by the ICRP, plus the "adjacent" absorption type(s). If the default absorption type is Type S, then calculations are made for the "adjacent" absorption type, Type M, as well as for Type S. If the default is Type F,

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[1]The problem also arises that fractional deposition in different regions of the respiratory tract depends on the tidal volume and respiratory frequency associated with the various daily activities (ICRP, 1994a). Layton's method does not address these individual components of the inhalation rate, and it is not evident how these two parameters should be adjusted for application of Layton's estimates of daily air intake.

then calculations are made for Type M as well as for Type F. If the default is Type M, or if the ICRP does not specify a single default absorption type, then calculations are made for all three absorption types. This scheme eliminates some presumably unlikely cases such as highly insoluble forms of cesium or iodine, or highly soluble forms of thorium. Because Type M is the default absorption type in most cases, all three absorption types are usually considered.

Except for tritium and radioisotopes of carbon, iodine, and tellurium, radionuclides are assumed to be inhaled only in particulate form. It is assumed that tritium is in the form of a vapor (HTO as Type V) or a gas (HT as Type G); carbon is in gaseous form (Type G) as carbon monoxide (CO) or carbon dioxide (CO_2); iodine is in the form of a vapor (Type V), a gas (methyl iodide, CH_3I, as Type G), or a particulate (Type F or Type M); and tellurium is in the form of a vapor (Type V) or a particulate (Type F, Type M, or Type S).

Intake of radionuclides in food

Risk coefficients (Bq^{-1}) for ingestion of radionuclides in food are expressed as risk of cancer mortality or morbidity per unit activity intake. The intake rate of a radionuclide in food is assumed to be proportional to food energy usage (kcal per day). Age- and gender-specific values for food energy usage (Table 3.1) are based on data from the Third National Health and Nutrition Examination Survey (NHANES III), Phase 1, 1989-91 (McDowell et al., 1994).

Food usage is often expressed in terms of mass rather than energy. Based on a 1994-95 food-intake survey by the U.S. Department of Agriculture, the lifetime average intake rate of food is approximately 1.2 kg per day (Wilson *et al.*, 1997). This value and the lifetime average energy intake of 2048 kcal per day given in Table 3.1 imply an average energy density for the U.S. diet of about 1700 kcal per kg food.

For radioiodine, a second set of risk coefficients is derived under the assumption that the intake rate is proportional to usage of cow's milk, typically the dominant source of radioiodine in diet (UNSCEAR, 1982). Age- and gender-specific values for average daily usage of cow's milk (Table 3.1) are based on data tabulated by the EPA (EPA, 1984b).

For ^{3}H in diet, separate risk coefficients are given for tritiated water and organically bound tritium, for which different biokinetic models are recommended by the ICRP (ICRP, 1989). Also, for ^{35}S in diet, separate risk coefficients are given for inorganic and organic sulfur, for which different biokinetic models are recommended (ICRP, 1993).

Intake of radionuclides in tap water

Risk coefficients (Bq^{-1}) for ingestion of radionuclides in tap water are expressed as risk of cancer mortality or morbidity per unit activity intake. Age-specific usage rates for tap water (Table 3.1) are based on results of the 1977-1978 Nationwide Food Consumption Survey of the U.S. Department of Agriculture as analyzed by Ershow and Cantor (1989). The data for usage of tap water in Table 3.1 include drinking water, water added to beverages, and water added to foods during preparation but do not include usage of water intrinsic in food as purchased. The reported data for tap water usage (Ershow and Cantor, 1989) were not divided by gender. Gender-specific values were derived by assuming (before the intake rate curves were smoothed) that the male-to-female intake rate ratio at a given age is the same as that observed for food energy intake (McDowell et al., 1994).

As is the case for intake in food, separate risk coefficients for tap water usage are given for tritiated water and organically bound tritium and for inorganic and organic ^{35}S.

External exposure to radionuclides in air

Risk coefficients ($m^3\ Bq^{-1}\ s^{-1}$) for submersion are expressed as risk of cancer mortality or morbidity per unit integrated exposure to a radionuclide in air. The external dose rates used in the calculations (EPA, 1993) were calculated for a reference adult male, standing outdoors with no shielding. No adjustments are made in this exposure scenario to account for potential differences with age and gender in the external doses received or for potential reduction in dose due to shielding by buildings during time spent indoors.

External exposure to radionuclides in soil

Risk coefficients are tabulated for two different scenarios for exposure to contaminated soil: (1) external exposure to radiations from the ground surface, and (2) external exposure to radiations from soil contaminated to an infinite depth. In both cases the contamination is assumed to be of infinite lateral extent. The risk coefficients are expressed as risk of cancer mortality or morbidity per unit integrated exposure to a radionuclide. The units are $m^2\ Bq^{-1}\ s^{-1}$ for contaminated ground surface and $kg\ Bq^{-1}\ s^{-1}$ for soil contaminated to an infinite depth.

The tabulations of dose coefficients in Federal Guidance Report No. 12 (EPA, 1993) for cases of external exposure to radiations from contaminated soil were calculated for a reference adult

male standing on the contaminated soil. No adjustments are made in this exposure scenario to account for potential differences with age and gender in the external doses received or for potential reduction in dose due to shielding by buildings during time spent indoors.

Recommendations concerning cleanup of contaminated soil are sometimes based on the radionuclide concentration in soil to a depth of 15 cm (NRC, 1977). As indicated by the tabulations of dose coefficients in Federal Guidance Report No. 12, dose rates from soil contaminated to a depth of 15 cm generally differ by only 0-20% from dose rates from soil contaminated to an infinite depth (that is, to several meters below the surface) due to shielding provided by the top 15 cm of soil against radiations emitted at lower depths (EPA, 1993). Because risk coefficients for external exposure to soil contaminated to 15 cm would differ only slightly from those for contamination to an infinite depth, it would not be useful to provide tabulations of risk coefficients for both situations.

CHAPTER 4. BIOKINETIC MODELS FOR RADIONUCLIDES

In the dose-computation scheme of the ICRP, information on the behavior of radionuclides in the body is condensed into three main types of biokinetic models: a respiratory tract model, a gastrointestinal tract model, and element-specific systemic models. The generic respiratory tract model is used to describe the deposition and retention of inhaled material in the respiratory tract and its subsequent clearance to blood or to the gastrointestinal tract. The generic gastrointestinal tract model is used to describe the movement of swallowed or endogenously secreted material through the stomach and intestines, and, together with element-specific gastrointestinal absorption fractions (f_1 values), to describe the rate and extent of absorption of radionuclides from the small intestine to blood. Element-specific systemic biokinetic models are used to describe the time-dependent distribution and excretion of radionuclides after their absorption into blood.

The respiratory tract

The ICRP recently introduced a new respiratory tract model that involves considerably greater detail and physiological realism than previous models of the respiratory system (ICRP, 1994a). The model structure is shown in Fig. 4.1. The model divides the respiratory system into extrathoracic (*ET*) and thoracic regions. The airways of the *ET* region are further divided into two categories: the anterior nasal passages, in which deposits are removed by extrinsic means such as nose blowing, and the posterior nasal passages including the nasopharynx, oropharynx, and the larynx, from which deposits are swallowed. The airways of the thorax include the bronchi (compartments labeled BB_i), bronchioles (compartments labeled bb_i), and alveolar region (compartments labeled AI_i). Material deposited in the thoracic airways may be cleared into blood by absorption, to the GI tract by mechanical processes (that is, transported upward and swallowed), and to the regional lymph nodes via lymphatic channels.

The number of compartments in each region was chosen to allow duplication of the different kinetic phases observed in humans or laboratory animals. In Fig. 4.1, particle transport rates shown beside the arrows are reference values in units of d^{-1}. For example, particle transport from bb_1 to BB_1 is assumed to occur at a fractional rate of 2 d^{-1}, and particle transport from ET_2 to the gastrointestinal tract is assumed to occur at a fractional rate of 100 d^{-1}.

For an inhaled compound, the mechanical clearances of particles indicated in Fig. 4.1 are in addition to dissolution rates and absorption to blood, which depend on the element and the chemical

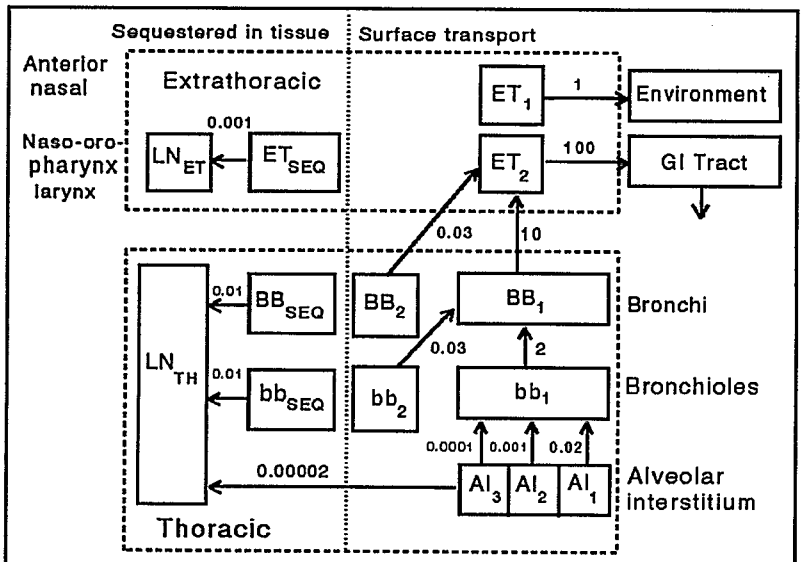

Fig. 4.1. Structure of the ICRP's respiratory tract model (ICRP, 1994a). Except for ET_1 to environment, each of the indicated mechanical clearances of particles is in addition to dissolution and absorption to blood (see text). Abbreviations: AI = alveolar interstitium, BB = bronchi, bb = bronchioles, ET = extrathoracic, LN = lymph nodes, SEQ = sequestered, TH = thoracic.

and physical form in which it is inhaled. Although the model permits consideration of compound-specific dissolution rates, a particulate is generally assigned to one of three default absorption types: Type F (fast dissolution and a high level of absorption to blood), Type M (an intermediate rate of dissolution and an intermediate level of absorption to blood), and Type S (slow dissolution and a low level of absorption to blood). The fractional rate of absorption (d^{-1}) assigned to the default types are

$$\textit{Type F}: \; 100 \;,$$

$$\textit{Type M}: \; 10.0 \; e^{-100\,t} + 5.0\times10^{-3} \; e^{-0.005\,t} \;,$$

$$\textit{Type S}: \; 0.1 \; e^{-100\,t} + 1.0\times10^{-4} \; e^{-0.0001\,t} \;,$$

where t is time (days) since deposition. Ideally, the user selects Type F, Type M, or Type S on the basis of experimental data on compounds expected to be encountered in practice.

The gastrointestinal tract

The model of the gastrointestinal (GI) tract applied in this report has been used by the ICRP for many years (ICRP, 1979, 1995a, 1995b). The model, shown in Fig. 4.2, divides the GI tract into four segments or compartments: *stomach* (*St*), *small intestine* (*SI*), *upper large intestine* (*ULI*), and *lower large intestine* (*LLI*), and depicts first-order transfer of material from one segment to the next. Material is assumed to transfer from *St* to *SI* at the fractional rate of 24 d^{-1}, from *SI* to *ULI* at 6 d^{-1}, from *ULI* to *LLI* at 1.8 d^{-1}, and from *LLI* to feces at 1 d^{-1}.

Absorption of ingested material to blood generally is assumed to occur only in *SI*. Absorption to blood is described in terms of a fraction f_1. In the absence of radioactive decay, the fraction f_1 of ingested material moves from *SI* to *BLOOD* and the fraction $1\text{-}f_1$ moves from *SI* to *ULI* and eventually is excreted in feces. The transfer coefficient from *SI* to *BLOOD* is $6f_1 / (1\text{-}f_1)$ d^{-1}.

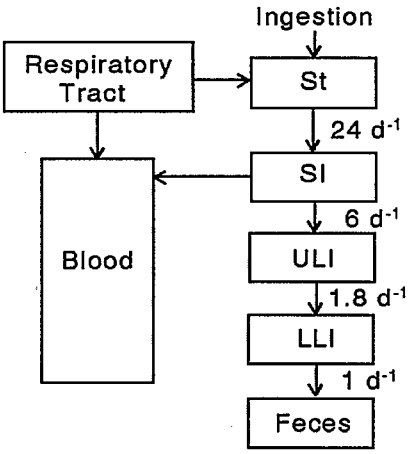

Fig. 4.2. Model of transit of material through the gastrointestinal tract (ICRP, 1979).

Most of the f_1 values used in this report are taken from the ICRP's recent series of documents on intakes of radionuclides by members of the public (ICRP, 1989, 1993, 1995a, 1995b, 1996). In those documents, different f_1 values are applied in some cases to ingested forms of a radionuclide and inhaled forms that are subsequently cleared from the respiratory tract to the stomach.

Six of the elements considered in the internal exposure scenarios in this report were not addressed in the ICRP documents on intakes of radionuclides by members of the public (Sc, Y, La, Bi, Ac, Pa). In lieu of recommendations concerning environmental intakes of these elements, f_1 values for the adult are taken from the ICRP's most recently published document on occupational exposures (ICRP, 1994b), and f_1 values for infants and children are based on a default approach applied in ICRP Publication 69 (ICRP, 1995a) for intakes of radionuclides by members of the public. For present purposes, that default approach consists of the following rules: the f_1 value for adults

is applied to ages ≥1 y; if f_1 for adults is ≤0.002, then f_1 for infants is 10 times the value for adults; and if f_1 for adults is in the range 0.01-0.5, then f_1 for infants is 2 times the value for adults.

The f_1 values used here for ingestion and inhalation are listed in Tables 4.1a and 4.1b, respectively. For ages intermediate to those indicated in Table 4.1a and in the footnotes to Table 4.1b, (that is, from infant to 1 y and from 15 y to mature adulthood), the transfer coefficient from *SI* to *BLOOD* (which is derived from f_1 as described earlier) is interpolated linearly with age. The f_1 values as well as other biokinetic parameter values for "infant" apply to ages 0-100 days. Biokinetic parameter values are assumed to vary with age up to age 20 y for some elements (e.g., Fe, Cs, I) and up to age 25 y for others (e.g., Ca, Ra, Pu) and to be constant thereafter.

Systemic biokinetic models

The sources of the systemic biokinetic models used in this report are given in Table 4.2. Most of the models are taken from the ICRP's recent series of documents on age-dependent dosimetry for internal emitters (ICRP, 1989, 1993, 1995a, 1995b, 1996). However, six of the elements considered here (Sc, Y, La, Bi, Ac, and Pa) were not addressed in that series. The elements Sc, Y, La, and Bi are assigned the systemic biokinetic models recommended in ICRP Publication 30 (1979, 1980, 1981, 1988), which addresses occupational exposure to radionuclides. For consistency with other actinide elements considered in this document, the ICRP's generic model structure for bone-surface-seeking elements (ICRP, 1993) is applied to Ac and Pa. Parameter values for Am are assigned to Ac and parameter values for Th are assigned to Pa, due mainly to similarities in the biokinetics of these element pairs in laboratory animals (Hamilton, 1948; Durbin, 1960; Taylor, 1970; Ralston, et al., 1985). External measurements as well as bioassay measurements on workers accidentally exposed to isotopes of Ac and Pa also provide some support for the models selected here for these two elements (Newton, 1966; Newton and Brown, 1974).

With regard to model structure, the systemic biokinetic models used in this report may be divided into two main classes, referred to here as "retention models" and "physiologically based models".

A retention model is not intended as a biologically realistic depiction of actual paths of movement of a radionuclide in the body; rather, it is a mathematically convenient representation of the estimated inventories of the radionuclide in its major repositories as a function of time after its initial entry into blood. The initial distribution of activity leaving blood is represented by compartment-specific deposition fractions, and subsequent time-dependent inventories in the compartments are described in terms of compartment-specific biological removal half-times.

Material leaving a tissue compartment is assumed either to move directly to excretion or to move to excretion via an excretion pathway such as the contents of the urinary bladder or the gastrointestinal tract.

Table 4.1a. Gastrointestinal absorption fractions (f_1 values) for ingestion of radionuclides.[a,b]

Element	Age (y)			Reference	Element	Age (y)			Reference
	Infant	1-15 y	Adult			Infant	1-15 y	Adult	
H	1.0	1.0	1.0	ICRP, 1989	Te	0.6	0.3	0.3	ICRP, 1993
C	1.0	1.0	1.0	ICRP, 1989	I	1.0	1.0	1.0	ICRP, 1989
S	1.0	1.0	1.0	ICRP, 1993	Cs	1.0	1.0	1.0	ICRP, 1989
Ca	0.6	0.4	0.3	ICRP, 1995b	Ba	0.6	0.3	0.2	ICRP, 1993
Sc	0.001	0.0001	0.0001	c	La	0.005	0.0005	0.0005	c
Fe	0.6	0.3	0.2	ICRP, 1995a	Ce	0.005	0.0005	0.0005	ICRP, 1993
Co	0.6	0.3	0.1	ICRP, 1993	Pb	0.6	0.4	0.2	ICRP, 1993
Ni	0.1	0.05	0.05	ICRP , 1993	Bi	0.1	0.05	0.05	c
Zn	1.0	0.5	0.5	ICRP, 1993	Po	1.0	0.5	0.5	ICRP, 1993
Se	1.0	0.8	0.8	ICRP, 1995a	Ra	0.6	0.3	0.2	ICRP, 1993
Sr	0.6	0.4	0.3	ICRP, 1993	Ac	0.005	0.0005	0.0005	c
Y	0.001	0.0001	0.0001	c	Th	0.005	0.0005	0.0005	ICRP, 1995a
Zr	0.02	0.01	0.01	ICRP, 1989	Pa	0.005	0.0005	0.0005	c
Nb	0.02	0.01	0.01	ICRP, 1989	U	0.04	0.02	0.02	ICRP, 1995a
Mo	1.0	1.0	1.0	ICRP, 1993	Np	0.005	0.0005	0.0005	ICRP, 1993
Tc	1.0	0.5	0.5	ICRP, 1993	Pu	0.005	0.0005	0.0005	ICRP, 1993
Ru	0.1	0.05	0.05	ICRP, 1993	Am	0.005	0.0005	0.0005	ICRP, 1993
Ag	0.1	0.05	0.05	ICRP, 1993	Cm	0.005	0.0005	0.0005	ICRP, 1995b
Sb	0.2	0.1	0.1	ICRP, 1995a					

[a]This document follows the recommendations in the ICRP Publication 56 series (ICRP, 1989, 1993, 1995a, 1995b, 1996) on exposures of the public. That series does not recommend separate f_1 values for food and water. While there is some experimental evidence of differential absorption of certain radionuclides from food and water, the data are not definitive.

[b]Values for ages between infancy (100 d) and 1 y and between 15 y and adulthood are derived by interpolation with age.

[c]Value for adult taken from ICRP Publication 68 (1994b). Values for infants and children based on default approach of ICRP (1995a), described in the text.

Table 4.1b. Gastrointestinal absorption fractions (f_1 values) for inhalation of radionuclides.[a,b]

Element	Absorption Type			Reference	Element	Absorption Type			Reference
	F	M	S			F	M	S	
H	1.0	1.0	1.0	ICRP, 1995b	Te	0.3	0.1	0.01	ICRP, 1995b
C	1.0	1.0	1.0	ICRP, 1995b	I	1.0	0.1	c	ICRP, 1995b
S	0.8	0.1	0.01	ICRP, 1995b	Cs	1.0	0.1	c	ICRP, 1995b
Ca	0.3	0.1	0.01	ICRP, 1995b	Ba	0.2	0.1	0.01	ICRP, 1995b
Sc	c	0.0001	0.0001	ICRP, 1994b	La	0.001	0.001	0.001	ICRP, 1994b
Fe	0.1	0.1	0.01	ICRP,1995b	Ce	0.0005	0.0005	0.0005	ICRP, 1995b
Co	0.1	0.1	0.01	ICRP, 1995b	Pb	0.2	0.1	0.01	ICRP, 1995b
Ni	0.05	0.05	0.01	ICRP, 1995b	Bi	0.05	0.05	0.05	ICRP, 1994b
Zn	0.5	0.1	0.01	ICRP, 1995b	Po	0.1	0.1	0.01	ICRP, 1995b
Se	0.8	0.1	c	ICRP, 1995b	Ra	0.2	0.1	0.01	ICRP, 1995b
Sr	0.3	0.1	0.01	ICRP,1995b	Ac	0.001	0.001	0.001	ICRP, 1994b
Y	0.0001	0.0001	0.0001	ICRP, 1994b	Th	0.0005	0.0005	0.0005	ICRP,1995b
Zr	0.002	0.002	0.002	ICRP, 1995b	Pa	0.001	0.001	0.001	ICRP, 1994b
Nb	0.01	0.01	0.01	ICRP, 1995b	U	0.02	0.02	0.002	ICRP,1995b
Mo	0.8	0.1	0.01	ICRP, 1995b	Np	0.0005	0.0005	0.0005	ICRP,1995b
Tc	0.8	0.1	0.01	ICRP, 1995b	Pu	0.0005	0.0005	0.0005	ICRP,1995b
Ru	0.05	0.05	0.01	ICRP, 1995b	Am	0.0005	0.0005	0.0005	ICRP, 1995b
Ag	0.05	0.05	0.01	ICRP,1995b	Cm	0.0005	0.0005	0.0005	ICRP, 1995b
Sb	0.1	0.01	0.01	ICRP,1995b					

[a]This document follows the recommendations in ICRP Publication 71 (ICRP, 1995b) on exposures of the public. That report recommends f_1 values for material cleared from the respiratory system to the stomach that differ in some cases from values recommended for ingested food and water.

[b]The tabulated f_1 values are for adults. Modification of these values for application to infants is explained in the text. The value for the adult is applied at ages 1 y and older with the exceptions that for Type F forms of Ca, Fe, Co, Sr, Ba, Pb and Ra, the values applied to ages 1-15 y correspond to the data of Table 4.1a.

[c]Not applicable because this absorption type is not considered for this element.

Table 4.2. Systemic biokinetic models used in this report.

Element	ICRP Publication	Element	ICRP Publication
H	56 (1989)	Te	67 (1993)
C	56 (1989)	I	56 (1989)
S	67 (1993)	Cs	56 (1989)
Ca	71 (1995b)	Ba	67 (1993)
Sc	30 (Part 3, 1981)	La	30 (Part 3, 1981)
Fe	69 (1995a)	Ce	67 (1993)
Co	67 (1993)	Pb	67 (1993)
Ni	67 (1993)	Bi	30 (Part 2, 1980)
Zn	67 (1993)	Po	67 (1993)
Se	69 (1995a)	Ra	67 (1993)
Sr	67 (1993)	Ac	a
Y	30 (Part 2, 1980)	Th	69 (1995a)
Zr	67 (1993)	Pa	b
Nb	56 (1989)	U	69 (1995a)
Mo	67 (1993)	Np	67 (1993)
Tc	67 (1993)	Pu	67 (1993)
Ru	56 (1989)	Am	67 (1993)
Ag	67 (1993)	Cm	71 (1995b)
Sb	69 (1995a)		

[a]Assigned the biokinetic model for Am given in ICRP Publication 67 (1993).

[b]Assigned the biokinetic model for Th given in ICRP Publication 69 (1995a).

An example of the type of retention models used by the ICRP is the model for zirconium originally described in ICRP Publication 30 (1979) and updated in ICRP Publications 56 (1989) and 67 (1993). The structure of this model is shown in Fig. 4.3. Parameter values were based largely on observations of the behavior of zirconium in rats and mice. For all age groups, 50% of zirconium leaving blood is assumed to deposit on bone surfaces and the remainder is assumed to be uniformly

distributed in the rest of the body, referred to as *Other*. For the adult, zirconium is assumed to be removed to excretion with a biological half-time of 10,000 days. In the absence of age-specific data on zirconium in humans, the removal half-time from bone in children is assumed to be proportional to the bone turnover rate, which is considerably greater in children than in adults; for example, a removal half-time from bone to excretion pathways of 1000 days is applied to the 10-year-old child. For all age groups, zirconium is assumed to be removed from *Other* to excretion pathways with a biological half-time of 7 days. Of zirconium going to excreta, five-sixths is assigned tk the urinary bladder contents and one-sixth is assigned to the contents of the upper large intestine. Generic models are used to describe removal from the contents of the urinary bladder and the gastrointestinal tract to excretion (ICRP, 1993).

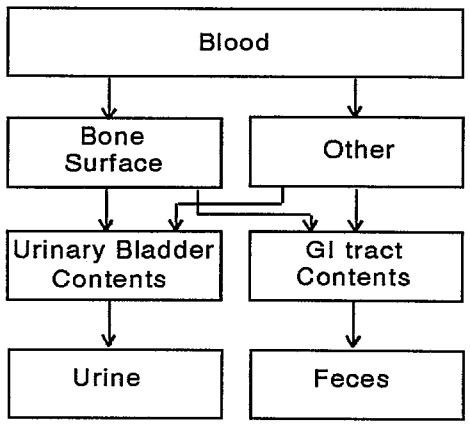

Fig. 4.3. Structure of the ICRP's biokinetic model for zirconium (ICRP, 1993).

In the ICRP's documents on age-dependent dosimetry (ICRP, 1989, 1993, 1995a, 1995b, 1996), physiologically based models were used for radioisotopes of calcium, iron, strontium, iodine, barium, lead, radium, thorium, uranium, neptunium, plutonium, americium, and curium. The model frameworks applied to these elements depict loss of material by specific excretion pathways, feedback of material from organs to blood plasma, and certain physiological processes that are known to influence the distribution and translocation of the elements in the body. Clearly, the degree of biological realism incorporated into each of the models is limited by practical considerations regarding the amount and quality of information available to determine actual paths of movement and parameter values for specific elements.

The model for iodine (Fig. 4.4) is essentially the same as that used in ICRP Publication 30 (1979), except that parameter values were extended to pre-adult ages. The

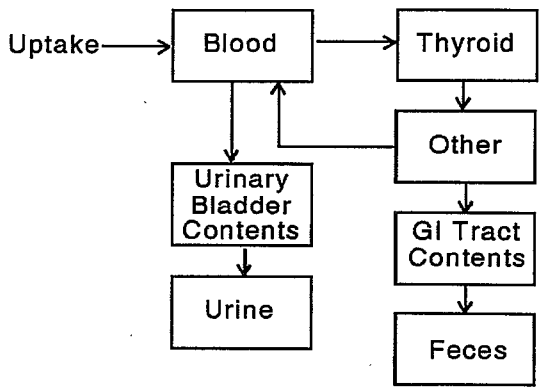

Fig. 4.4. Structure of the ICRP's biokinetic model for iodine (ICRP, 1989).

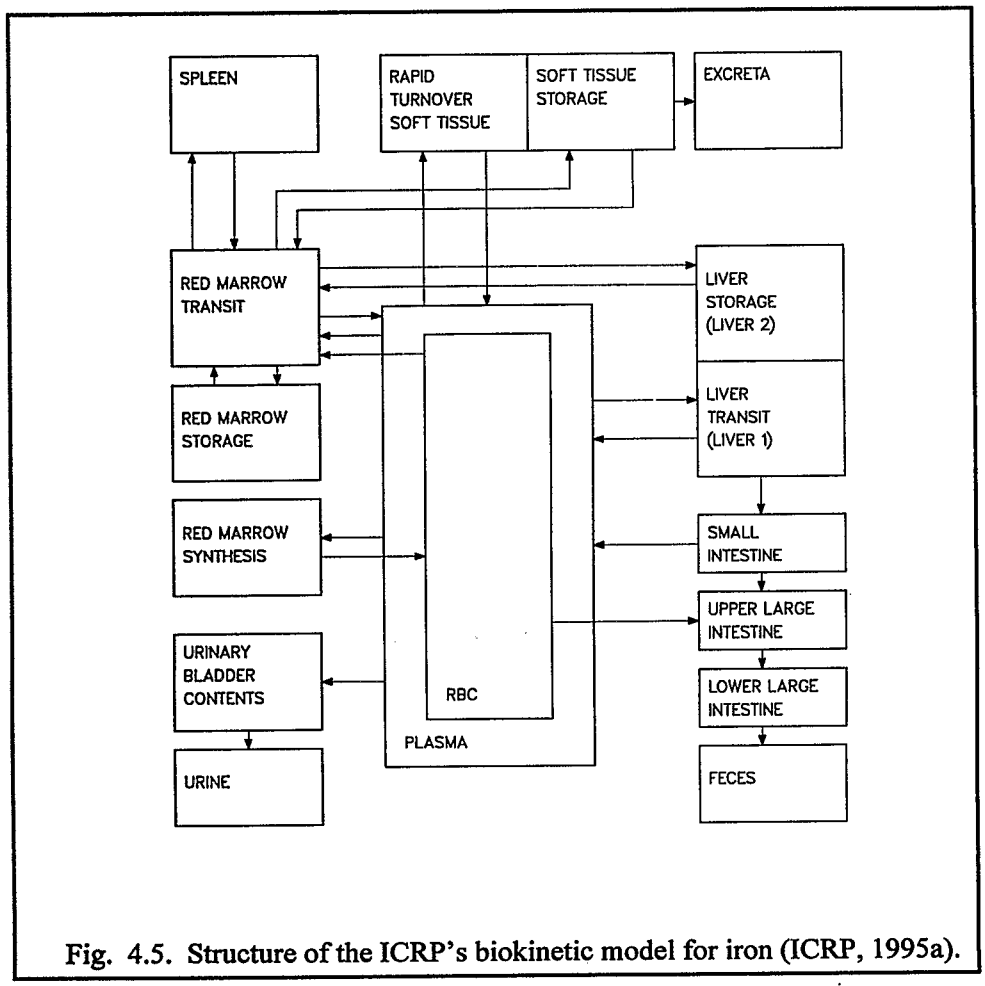

Fig. 4.5. Structure of the ICRP's biokinetic model for iron (ICRP, 1995a).

model structure is relatively simple compared with the other physiologically based models used in the ICRP Publication 56 series. According to this model, iodine entering blood is taken up by the thyroid or excreted in urine. It leaves the thyroid in organic form and is metabolized by the tissues in the rest of the body. A portion of iodine leaving these tissues is excreted in feces and the remainder is returned to blood in inorganic form and behaves the same as the original input to blood.

The model structure for iron is shown in Fig. 4.5. The model describes three main aspects of iron metabolism: (1) the hemoglobin cycle, including uptake of transferrin-bound iron by the erythroid marrow for incorporation into hemoglobin, subsequent appearance of iron in red blood cells, uptake of old and damaged red blood cells by the reticuloendothelial system, and eventual return of iron to plasma; (2) removal of transferrin-bound iron from plasma to the extravascular spaces and return to plasma via the lymphatic system; and (3) uptake and retention of iron by the

parenchymal tissues. The soft tissues include a pool of extravascular iron that exchanges rapidly with plasma iron. Storage iron is divided among liver, spleen, red marrow, and other soft tissues. Destruction of red blood cells is viewed as occurring in the red marrow. The liver is viewed as consisting of two pools: a transit pool representing parenchymal tissues that exchange iron with plasma, and a storage pool associated with the reticuloendothelial system. Excretion of iron is depicted as occurring through exfoliation of skin, losses of plasma iron in urine, and leakage of red blood cells into the intestines and subsequent removal in feces.

The ICRP's physiologically based models for bone-seeking elements were developed within one of two generic model frameworks (Leggett 1992a, 1992b; ICRP, 1993), one designed for application to a class of "calcium-like" or bone-volume-seeking elements such as strontium, radium, and lead (Fig. 4.6), and the other designed for application to a class of "plutonium-like" or bone-surface-seeking elements such as americium, neptunium, and thorium (see Appendix C). In contrast to the treatment of bone-seeking radionuclides in ICRP Publication 30 (1979), the new bone models account for the facts that bone-surface seekers are buried to a large extent in bone volume, bone-volume seekers may have a significant residence time on bone surfaces, and elements from both groups may be recycled to tissues to a significant extent after removal from their initial repositories to blood plasma. The physiologically based systemic biokinetic model for thorium, which is typical of bone-surface seekers, is described in detail in Appendix C.

Treatment of decay chain members formed in the body

Systemic biokinetic models for decay chain members formed *in vivo* are taken from the ICRP's series on age-dependent dosimetry (ICRP, 1989, 1993, 1995a, 1995b, 1996) or, for elements not addressed in that document, from ICRP Publication 30 (1979, 1980, 1981, 1988). In most cases, decay chain members produced *in vivo* are assigned the systemic biokinetic model of the parent (that is, the radionuclide taken into the body). However, the following exceptions are made:

1. Iodine as a daughter of tellurium is assumed to be translocated at a fractional rate of 1000 d^{-1} to the transfer compartment in inorganic form and then to follow the same kinetics as iodine introduced into blood as a parent radionuclide.
2. Xenon produced *in vivo* by decay of iodine is assumed to escape from the body without decay. This assumption is carried over from ICRP Publication 30 (Part 1, 1979).
3. If the parent is an isotope of lead, radium, uranium, or thorium, then a radionuclide other than a noble gas formed in soft tissues or on bone surfaces is assigned the characteristic biokinetics of that radionuclide. That is, a radionuclide born either in soft tissues or on bone surfaces is

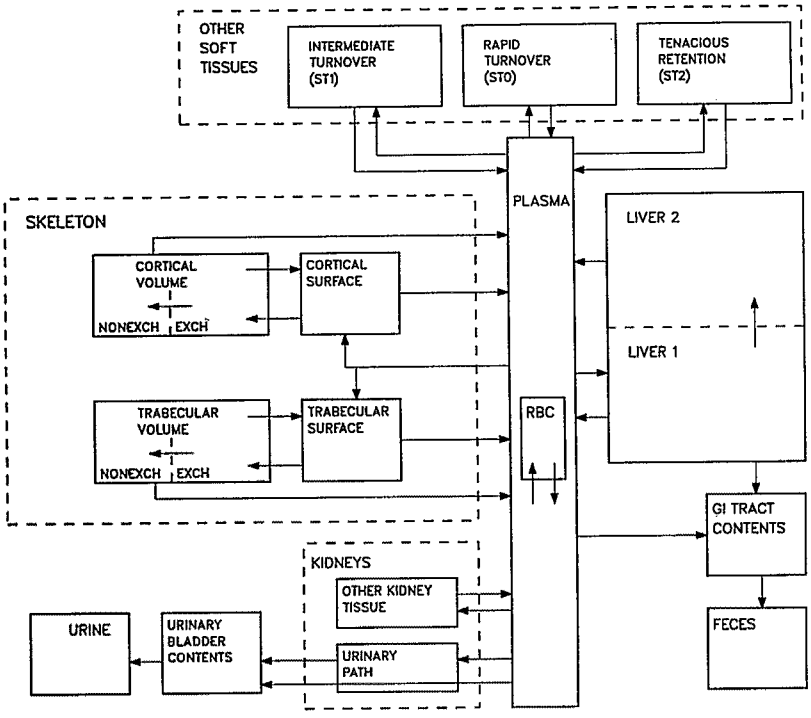

Fig. 4.6. The ICRP's generic model structure for calcium-like elements (ICRP, 1993). RBC = red blood cells, EXCH = exchangeable bone volume, NONEXCH = nonexchangeable bone volume.

assumed to have the same biokinetics as if the radionuclide had been taken in as a parent radionuclide. A radionuclide other than a noble gas formed in bone volume is assigned the biokinetics of the parent. Noble gases produced in soft tissues and bone surfaces are assumed to migrate from the body with a transfer coefficient of 100 d^{-1}. Noble gases produced in exchangeable and non-exchangeable bone volume are assumed to migrate from the body at rates of 1.5 d^{-1} and 0.36 d^{-1}, respectively.

Appendix C describes in detail the treatment of decay chain members produced in the body after absorption of the parent radionuclide, ^{232}Th, to blood.

Radionuclides produced in the respiratory tract are assumed to have the same kinetics as the parent radionuclide while in the respiratory tract. The rate of dissolution of the carrier of the

radionuclide is assumed to control the rate of migration of inhaled radionuclides and their radioactive progeny. An exception is made for ^{222}Rn, which is assumed to escape from the body at a fractional rate of 100 d^{-1} after its production in any segment of the respiratory tract.

Chain members produced in, or migrating to, the gastrointestinal tract after intake of the parent radionuclide are assigned the gastrointestinal absorption fraction (f_1) of the parent in most cases. For consistency with the treatment of the systemic biokinetics of radionuclides formed *in vivo*, exceptions are made if the parent radionuclide is an isotope of lead, radium, thorium, or uranium. In these cases, fractional absorption of a chain member produced *in vivo* is assumed to be the same as if that chain member had been taken in as a parent radionuclide.

Solution of the biokinetic models

The solver used in the DCAL computational system (Eckerman et al., to be published) to track the time-dependent distribution of activity of the parent and the decay chain members in the body is described elsewhere (Leggett et al., 1993).

Uncertainties in the biokinetic models

Quantification of uncertainties in the biological behavior of radionuclides in humans is a complex problem that has received little attention in the literature. However, three major efforts to characterize such uncertainties for environmentally or occupationally important radionuclides currently are underway: the U.S. National Council on Radiation Protection and Measurements (NCRP) is preparing a report on the reliability of the models and dose coefficients of ICRP Publication 30; the ICRP is preparing a report on the reliability of its models and dose coefficients for members of the public; and the Commission of the European Communities (CEC) and the U.S. NRC are preparing a joint report on the uncertainties in the biokinetic, dose, and risk models used in probabilistic risk assessment codes for reactor releases.

The purpose of this section is to provide semi-quantitative descriptions of the expected reliability of the ICRP's age-specific biokinetic models for selected radionuclides as central estimators for the population. The discussion is based on a paper by Leggett et al. (to be published) that summarizes work done by those authors as part of the uncertainty analyses of the NCRP, ICRP, and CEC-NRC. Attention is restricted to a small set of environmentally important radionuclides that serve to illustrate various levels of knowledge concerning biokinetics of radionuclides in humans: ^{3}H as tritiated water (HTO), ^{60}Co, ^{90}Sr, ^{95}Zr, ^{106}Ru, ^{125}Sb, ^{137}Cs, ^{226}Ra, and ^{239}Pu. The paper by

Leggett and coworkers focuses mainly on the age-specific systemic biokinetic models for these radionuclides but provides a brief discussion of the uncertainties in the level of absorption of these radionuclides from the gastrointestinal and respiratory tracts into the systemic circulation of the adult.

The term "uncertainty" refers here to the level of knowledge of a central value for the population, the quantity of interest for these calculations, and should not be confused with the variability in the biological behavior of a radionuclide in the population. Variability refers to the range of values encountered in the population, that is, to quantitative differences between different members of a population. For example, two healthy persons of the same age and gender may exhibit considerably different gastrointestinal uptake or systemic retention of a given radionuclide. While uncertainty and variability are distinct concepts, the variability in biokinetic or dosimetric characteristics of individuals within a population is usually an important factor contributing to the uncertainty in estimates of central values. This is because such variability complicates the problem of identifying the central tendency of these characteristics in the population due to the small number of observations generally available and the fact that subjects usually are not randomly selected from the population of interest.

The uncertainty in a given biokinetic quantity is described here in semi-quantitative terms. Specifically, the uncertainty is described as "low", "low to moderate", "moderate to high", or "high" if the central value is judged to be known within a factor of 2, 2-3, 3-8, or >8, respectively (as defined below). In the fairly common case in which it can be agreed by different experts only that the uncertainty in a biokinetic quantity is somewhere between low (less than a factor of 2) and high (at best an order of magnitude estimate), the uncertainty is described as "moderate".[2]

The level of confidence in the quantity of interest is first estimated in terms of subjective lower and upper bounds, A and B, such that there is judged to be roughly a 90% probability that the true but unknown central value is no less than A and no greater than B. The uncertainty is characterized as low, low to moderate, moderate to high, or high if $(B/A)^{1/2} < 2$, $2 \le (B/A)^{1/2} < 3$, $3 \le (B/A)^{1/2} \le 8$, or $8 < (B/A)^{1/2}$, respectively. The quantity $(B/A)^{1/2}$ is the same as $B/G = G/A$, where the central value G is the geometric mean of A and B; that is, $G = (A \times B)^{1/2}$. Thus, the biokinetic quantity may be described as being known within a factor of about $(B/A)^{1/2}$, because the likely values fall within a factor of $(B/A)^{1/2}$ of the geometric mean. Although the geometric mean of the range of likely values often is not used as the best estimate of the central value for the population, this

[2]This terminology differs from that of Leggett and coworkers, who generally refer to the "reliability" rather than the "uncertainty" of a model and thus would describe a model prediction as being "highly reliable" (for example) rather than as having "low uncertainty".

approach provides a uniform method of characterizing uncertainties in biokinetic models that is consistent with informal statements of uncertainty commonly made by researchers.

For most biokinetic endpoints (e.g., fractional absorption from the gastrointestinal tract or integrated activity in a given organ), assignment of a level of uncertainty is based largely on the quality and completeness of data on the behavior of the element and its physiological analogues in humans and laboratory animals. High confidence in a biokinetic estimate for an element usually is gained from the existence of reasonably complete, high-quality data on that element in human subjects. Confidence decreases with decreasing quality and completeness of the data on humans or with increasing reliance on surrogate information such as data on the behavior of the element in laboratory animals or a chemical analogue of the element in humans. Confidence in estimates based on surrogate data may be particularly low if the surrogate data have inherent weaknesses or if the logical basis for surrogacy is weak.

Depending on the endpoint under consideration, assignment of a level of uncertainty may also be heavily influenced by the radiological half-life or other physical constraints. For example, uncertainties in the long-term biokinetics of zirconium is of little consequence when estimating the integrated activity of ^{95}Zr in bone because virtually no ^{95}Zr atoms will remain in the body beyond two years after intake due to the short radiological half-life of this radionuclide (64 d). Thus, non-biokinetic considerations may lead to a much smaller range of uncertainty for some radionuclides than examination of the biokinetic data alone might indicate.

Based on the considerations described above, each of the selected radionuclides was evaluated with regard to: (1) the fraction of ingested activity reaching blood, assuming ingestion of typical environmental forms of a radionuclide by members of the public; (2) the fraction of inhaled activity reaching blood, assuming inhalation of typical environmental forms of a radionuclide; and (3) the 50-year integrated activity in selected organs, assuming injection of a radionuclide into blood. The first two items were evaluated for a typical adult, and the third item was evaluated for a typical child of age 5 years as well as for a typical adult.

Conclusions drawn for the adult are summarized in Table 4.3. With regard to fractional absorption from the gastrointestinal tract as well as the subsequent behavior of absorbed activity, uncertainties were judged to be low for ^{3}H (as tritiated water), ^{90}Sr, and ^{137}Cs, in view of the extensive measurements that have been made of uptake and retention of tritium, strontium, and cesium in healthy human subjects (ICRP, 1989, 1993). Predictions for ^{125}Sb were judged to be highly uncertain due to the paucity of data on the behavior of antimony in humans and the substantial inconsistencies in findings for laboratory animals (ICRP, 1995a). Fractional absorption of ^{95}Zr from the gastrointestinal tract in humans was judged to be highly uncertain because uptake

Table 4.3. Semi-quantitative assessment of the uncertainty in selected biokinetic models of the ICRP as central estimators for healthy adults.[a]

Radionuclide	Uncertainty in fraction of ingested activity absorbed	Uncertainty in fraction of inhaled activity absorbed	Uncertainty in 50-y integrated activity in selected organs after injection into blood
^{3}H (HTO)	low	low	low (total body)
^{60}Co	moderate	moderate	low to moderate (liver)
^{90}Sr	low	moderate	low (bone)
^{95}Zr	high	high	moderate to high (bone)
^{106}Ru	moderate	high	moderate to high (total body)
^{125}Sb	high	moderate	high (liver)
^{137}Cs	low	low	low (total body)
^{226}Ra	moderate	moderate	low to moderate (bone)
^{239}Pu	moderate	moderate	low to moderate (bone surface)

[a]Based on methods and conclusions of Leggett et al. (to be published).

data on this element are available only for rats, which have sometimes proved to be unreliable models for humans with regard to uptake of metals. The uncertainty in fractional absorption of the other elements was judged to be moderate. With regard to the integrated activity in the main repository after injection into blood, the uncertainty was judged to be low to moderate for ^{60}Co, ^{226}Ra, and ^{239}Pu and moderate to high for ^{95}Zr and ^{106}Ru.

For many radionuclides, fractional absorption of inhaled activity can be estimated only within fairly wide bounds for typical environmental exposures. The main difficulty is that fractional absorption of an inhaled element depends strongly on the physical and chemical form of the carrier (ICRP, 1994a), which, for many elements, cannot be characterized with much confidence. Of the elements addressed here, tritium (as tritiated water) and cesium are reasonably well understood with regard both to characterization of environmental forms and absorption of those forms from the respiratory tract to blood. At least two of the elements, zirconium and ruthenium, appear to be poorly understood in both regards. With regard to fractional absorption of inhaled activity, the uncertainty was judged to be low for ^{3}H and ^{137}Cs, high for ^{95}Zr and ^{106}Ru, and moderate for the other five radionuclides.

With regard to the 50-year integrated activity in selected organs (the same organs as considered for the adult; see the last column of Table 4.3) after injection of a radionuclide into blood

of a 5-year-old child, the uncertainty was judged to be low for ^{137}Cs; low to moderate for ^{3}H (as HTO), ^{90}Sr, and ^{226}Ra; moderate to high for ^{239}Pu; and high for ^{60}Co, ^{95}Zr, ^{106}Ru, and ^{125}Sb. Absorption from the gastrointestinal and respiratory tracts to blood in children was not evaluated.

The semi-quantitative assessments described above provide a useful starting point for addressing the previously neglected problem of characterizing the uncertainties in the ICRP's biokinetic models. In view of the work in progress by the NCRP, CEC, NRC, and ICRP, it seems likely that more quantitative assessments of uncertainty may soon be available for a number of environmentally and occupationally important radionuclides.

CHAPTER 5. DOSIMETRIC MODELS FOR INTERNAL EMITTERS

The dosimetric methodology used in this report is that of the ICRP and is generally consistent with the schema of the Medical Internal Radiation Dose Committee (MIRD) of the U.S. Society of Nuclear Medicine (Loevinger et al., 1988). The methodology considers two sets of anatomical regions within the body. A set of "source regions" is used to specify the location of radioactivity within the body. A set of "target regions" consists of those organs and tissues for which the radiation dose may be calculated.

Both the ICRP and MIRD consider the mean absorbed dose to a target region as the fundamental dosimetric quantity. The principal biological effect of interest in radiation protection, cancer induction, is cellular in origin, and the mean dose in a target is relevant to the extent that dose is representative of the dose to the cells at risk. The cells at risk are assumed to be uniformly distributed in the target region. Thus, the mean dose is assumed to be the relevant quantity.

The source regions selected for a given application consist of explicitly identified anatomical regions and an implicit region, referred to as *Other*, defined as the complement of the set of explicitly identified regions. The radioactivity in each source region is assumed to be uniformly distributed. For most regions the distribution is by volume, but for mineral bone regions and the airways of the respiratory tract the distribution may be by surface area. For all target regions, the relevant quantity is the mean energy absorbed in the target volume averaged over the mass of the target.

A full list of source and target regions currently used by the ICRP is given in Table 5.1. The names of most source or target regions adequately identify the associated organs or tissues of the body, but additional explanation is needed for some regions, such as *Body Tissues*, *Other*, and *Bone Surface*. These and other special source and target regions are defined in Appendix B.

The esophagus is a radiosensitive tissue but has not yet been incorporated explicitly into the mathematical phantom used for internal dosimetric calculations. At present, the dose calculated for the target region *Thymus* is applied to the esophagus.

Age-dependent masses of source and target regions

With the exception of *Urinary Bladder Contents*, masses of source and target regions in children are taken from the phantom series of Cristy and Eckerman (1987), and values for the adult

Table 5.1. Source and target organs used in internal dosimetry methodology.

Organ or Tissue	Source Region	Target Region
Adrenals	Yes	Yes
Blood	Yes	No
Brain	Yes	Yes
Breasts	Yes	Yes
Gall Bladder Contents	Yes	No
Gall Bladder Wall	Yes	Yes
Heart Contents	Yes	No
Heart Wall	Yes	Yes
Kidneys	Yes	Yes
Liver	Yes	Yes
Muscle	Yes	Yes
Ovaries	Yes	Yes
Pancreas	Yes	Yes
Skin	Yes	Yes
Spleen	Yes	Yes
Testes	Yes	Yes
Thymus	Yes	Yes
Thyroid	Yes	Yes
Urinary Bladder Contents	Yes	No
Urinary Bladder Wall	Yes	Yes
Uterus	Yes	Yes
Body Tissues	Yes	No
Soft Tissues of Body Tissues	Yes	No
Other	Yes	No

Table 5.1, continued

Organ or Tissue	Source Region	Target Region
Skeleton:		
Bone Surface	No	Yes
Cortical Bone Surface	Yes	No
Cortical Bone Volume	Yes	No
Trabecular Bone Surface	Yes	No
Trabecular Bone Volume	Yes	No
Red Marrow	Yes	Yes
Gastrointestinal Tract:		
Stomach Contents	Yes	No
Stomach Wall	Yes	Yes
Small Intestine Contents	Yes	No
Small Intestine Wall	Yes	Yes
Upper Large Intestine Contents	Yes	No
Upper Large Intestine Wall	Yes	Yes
Lower Large Intestine Contents	Yes	No
Lower Large Intestine Wall	Yes	Yes
Respiratory Tract:		
Extrathoracic Region 1 – Surface	Yes	No
Extrathoracic Region 1 – Basal Cells	No	Yes
Extrathoracic Region 2 – Surface	Yes	No
Extrathoracic Region 2 – Bound	Yes	No
Extrathoracic Region 2 – Sequestered	Yes	No
Extrathoracic Region 2 – Basal Cells	No	Yes
Lymph Nodes – Extrathoracic Region	Yes	Yes
Bronchial Region – Gel (Fast Mucus)	Yes	No
Bronchial Region – Sol (Slow Mucus)	Yes	No
Bronchial Region – Bound	Yes	No
Bronchial Region – Sequestered	Yes	No
Bronchial Region – Basal Cells	No	Yes
Bronchial Region – Secretory Cells	No	Yes
Bronchiolar Region – Gel (Fast Mucus)	Yes	No
Bronchiolar Region – Sol (Slow Mucus)	Yes	No
Bronchiolar Region – Bound	Yes	No
Bronchiolar Region – Sequestered	Yes	No
Bronchiolar Region – Secretory Cells	No	Yes
Alveolar-Interstitial Region	Yes	Yes
Lymph Nodes – Thoracic Region	Yes	Yes

male are taken from the Reference Man document (ICRP Publication 23, 1975). Masses of *Urinary Bladder Contents* are based on data assembled for the revision of Reference Man and are intended to represent the contents of the bladder averaged over the filling and voiding cycles (Cristy and Eckerman, 1993).

For the adult female, regional masses are mostly reference values from ICRP Publication 23 but, where none are given, are scaled from those for the reference adult male. Masses for the target region *Bone Surface* or for source regions within mineral bone of the adult female are taken as 75% of the values for males. For *Urinary Bladder Contents* and *Urinary Bladder Wall*, values for the 15-y-old male are applied to the adult female.

Age-specific masses of source and target regions are listed in Appendix B.

Dosimetric quantities

The mean energy absorbed in the target region depends on the nature of the radiations emitted in the source regions, the spatial relationships between the source and target regions, and the nature of the tissues between the regions. The details of these considerations are embodied in a radionuclide-specific coefficient called the specific energy or *SE*.

For any radionuclide, source organ S, and target organ T, the specific energy at age t is defined as

$$SE(T \leftarrow S;t) = \frac{1}{M_T(t)} \sum_i Y_i \, E_i \, AF_i(T \leftarrow S;t) \, , \qquad (5.1)$$

where Y_i is the yield of radiations of type i per nuclear transformation, E_i is the average or unique energy of radiation type i, $AF_i(T \leftarrow S;t)$ is the fraction of energy emitted in source region S that is absorbed within target region T at age t, and $M_T(t)$ is the mass of target region T at age t. The age dependence in *SE* arises from the age dependence of the absorbed fraction and the mass of the target region. The quantity $AF_i(T \leftarrow S;t)$ is called the absorbed fraction (*AF*), and when divided by the mass of the target region, M_T, is called the specific absorbed fraction (*SAF*).

Whether one is interested in equivalent dose to a region, effective dose, or assessment of risk, the basic quantity to be computed is the absorbed dose rate at various times. The dose rate in target region T includes contributions from each radionuclide in the body and from each region in which radionuclides are present. The absorbed dose rate at age t in region T of an individual of age t_0 at the time of intake, $\dot{D}_T(t,t_0)$, can be expressed as

$$\dot{D}_T(t,t_0) = c \sum_S \sum_j q_{S,j}(t)\ SE(T \leftarrow S;t)_j \ , \qquad (5.2)$$

where $q_{S,j}(t)$ is the activity of radionuclide j present in source region S at age t, $SE(T \leftarrow S;t)_j$ is the specific energy deposited in target region T per nuclear transformation of radionuclide j in source region S at age t, and c is any numerical constant required by the units of q and SE.

The following shorthand terminology is sometimes used: "photons" for x radiation, gamma radiation, and annihilation quanta; "electrons" for β+ particles, β- particles, internal conversion electrons, and Auger electrons; and "alphas" for alpha particles and alpha recoil nuclei.

Nuclear decay data

In Eq. 5.1, there are two terms from the nuclear decay data: Y_i is the yield of radiations of type i per nuclear transformation, and E_i is the average or unique energy of radiation type i. The radiations that contribute the overwhelming majority of the energy per nuclear transformation are tabulated in ICRP Publication 38 (1983) and in a MIRD publication (Weber et al., 1989).

The decay data files in the DCAL computational system include the beta spectra (Eckerman et al., 1994). The beta spectra files are used in the dosimetry for the ICRP's new respiratory tract model. For other organs, only the average energy of each beta transition is used.

The nuclear decay data files include the kinetic energies of each emitted alpha particle but not the corresponding kinetic energies of the recoiling nucleus. The recoil energy E_r for an alpha transition is computed as

$$E_r = \frac{4.0026\ E_\alpha}{A - 4} \ , \qquad (5.3)$$

where E_α is the kinetic energy of the alpha particle, A is the mass number of the nuclide, and 4.0026 is the atomic mass of an alpha particle.

Specific absorbed fractions for photons

Photon *SAFs* are derived from radiation transport calculations in anthropomorphic phantoms representing newborn, 1 y, 5 y, 10 y, 15-y-old male, and adult male (with breasts, ovaries, and uterus

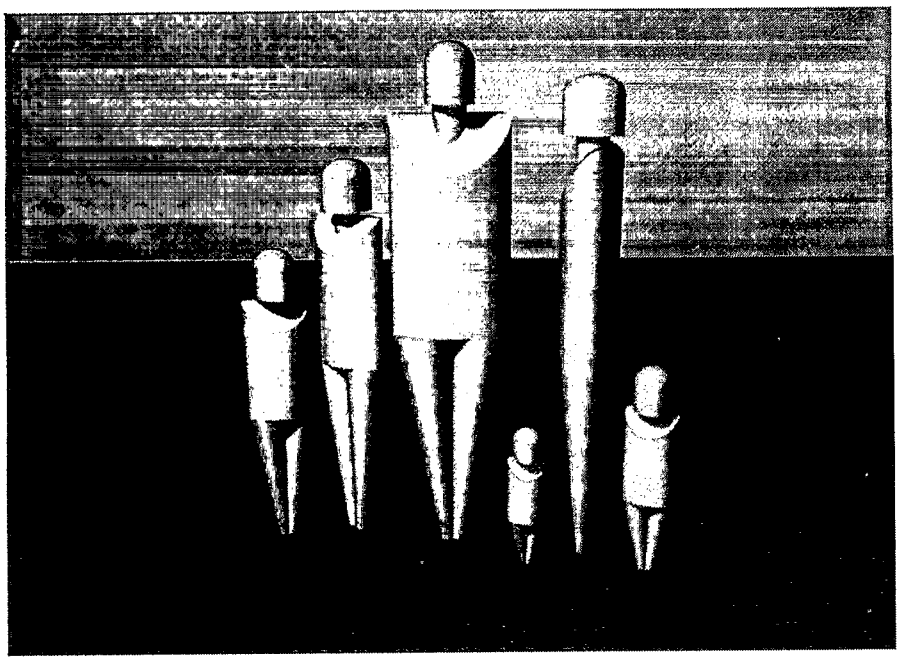

Fig. 5.1. Illustration of phantoms used to derive age-dependent specific absorbed fractions for photons.

added). These phantoms are illustrated in Fig. 5.1. In this report, the specific absorbed fractions for the adult male are also applied to the adult female.

The specific absorbed fractions are tabulated for 12 energies between 10 keV and 4 MeV. *SAFs* at intermediate energies are calculated by interpolating linearly between energies. Photons of energy below 10 keV are treated as nonpenetrating radiations for most regions and are considered to be absorbed in the source region. For bone dosimetry and for sources in the contents of walled organs (e.g., stomach), the dosimetry for photons is analogous to that described below for electrons.

Absorbed fractions for electrons

The kinetic energy of electrons is assumed to be absorbed entirely in the source region, except when the source is in part of the skeleton or when the source is in the contents of a walled organ. Thus, for solid regions,

$$AF(T \leftarrow S; t) = \begin{cases} 1, \ if \ T = S \\ 0, \ if \ T \neq S \ and \ S \neq BT \\ M_T \ / \ M_{BT}, \ if \ S = BT \end{cases} \tag{5.4}$$

where *BT* (*Body Tissues*) indicates the systemic tissues of the body. Note that if the source region is *Body Tissues* of mass M_{BT}, then the fraction of the *Body Tissues* activity in the target region is M_T/M_{BT}, to which an absorbed fraction of 1 is applied.

Appendix B lists absorbed fractions for beta-emitters for cases in which the source organ and target organ are both in bone (ICRP, 1979). The values are assumed to be independent of age.

For contents of walled organs, it is assumed that the dose to the wall is the dose at the surface of a half-space, or half the equilibrium dose to the contents. Thus, the specific absorbed fraction is

$$SAF(wall \leftarrow cont; t) = \frac{0.5}{M_{cont}} \tag{5.5}$$

where M_{cont} is the mass of the contents of the walled organ.

Absorbed fractions for alpha particles and recoil nuclei

For alpha particles and alpha recoil nuclei, the radiation is assumed to be absorbed entirely in the source region, except when the source is in part of the skeleton or when the source is in the contents of a walled organ. Equation 5.4 applies to all solid regions.

Appendix B lists absorbed fractions for alpha-emitters for cases in which the source and target organ are both in bone (ICRP, 1979). The values are assumed to be independent of age. For a source in a bone surface or bone volume compartment and a target consisting either of Bone Surface or Red Marrow, there is assumed to be no contribution to *SE* from alpha recoils.

The assumptions of ICRP Publication 30 are applied to contents of walled organs. That is, for application to alpha particles, the right side of Eq. 5.5 is multiplied by 0.01 to account for the reduced alpha dose to radiosensitive cells in the wall, and an absorbed fraction of zero is applied to alpha recoil nuclei. As discussed later, the value 0.01 is not based on calculations of energy deposition but is a cautiously high value based on comparative studies of radiogenic effects from alpha and beta emitters in the gastrointestinal tracts of rats.

Spontaneous fission

Spontaneous fission occurs in the decay of some isotopes of U, Pu, Cm, Bk, Cf, and Es and results in the emission of photons, electrons, and neutrons, as well as fission fragments. Spontaneous fission products have not yet been incorporated into the internal dosimetry

methodology. However, for radionuclides with spontaneous fission that are addressed in this report, this decay mode represents a relatively small portion of the total emitted energies.

Computation of *SE*

Within the DCAL computational system (Eckerman et al., to be published), the *SEs* are computed by the module SEECAL (Cristy and Eckerman, 1993). These *SE* calculations are based on nuclear decay data files, libraries of specific absorbed fractions for non-penetrating radiations and photons, and age-specific organ masses. The nuclear decay data files and specific absorbed fractions are those currently used by the ICRP (Cristy and Eckerman 1987, 1993). Organ masses for adults are taken from ICRP Publication 23 (1975). For children, age-specific organ masses are taken from the phantoms of Cristy and Eckerman (1987), which are based on data from ICRP Publication 23.

Uncertainties in the internal dosimetric models

SEs for photons

There are two principal computational procedures available for estimating specific absorbed fractions for photon emissions: the Monte Carlo method of simulation of radiation transport and the point-source kernel method. Both of these methods may involve significant sources of error, depending on the energy and the organs under consideration. An examination of the advantages and disadvantages of these two very different methods, together with a comparison of predictions of the two methods for various situations, provides insight into the uncertainties in *SEs* for photons and ways to minimize those uncertainties.

The Monte Carlo method is a computerized approach for estimating the probability of a photon interaction within target organ T after emission from source organ S. This method is carried out for all combinations of source and target organs and for several photon energies. The body is represented by an idealized phantom in which the internal organs are assigned masses, shapes, positions, and attenuation coefficients based on their chemical composition. Hypothetical interactions of numerous photons emanating in randomly chosen directions from points in the source organ are recorded as the photon travels through tissues and escapes from the body or loses its energy. This approach can produce significant statistical errors in situations where few interactions are expected to occur, such as cases involving low initial energies or target organs which are relatively small or remote from important sources of activity.

The second procedure for estimating specific absorbed fractions for photon emissions involves integration of a point-source kernel $\phi(x)$, where x is the distance from the point source. The function ϕ is composed of inverse-square and exponential attenuation factors that reflect the loss of energy from photon interactions and a build-up factor that reflects the contribution of scattered photons to dose. The point-source kernel method technically is valid only for a homogeneous, unbounded medium and hence may lead to large errors (a factor of two or more) in cases involving significant variations in composition or density of body tissue, or to small errors (up to about 10%) in cases where target organs or important sources of activity lie near a boundary of the body.

Results of Cristy and Eckerman (1987) indicate that the specific absorbed fractions for photons vary substantially with age for some energies, source organs, and target organs. As a rule, uncertainties in *SAFs* are greater for children than adults due to greater uncertainties concerning typical sizes and shapes of organs of children.

Maximal differences between the Monte Carlo and classical point-kernel methods are expected to occur for widely separated organ pairs and for large coefficients of variation for the Monte Carlo estimates. A comparison of the two methods was made for such situations in phantoms representing children of ages 1-15 y (Cristy and Eckerman, 1987). The results of this comparison indicate that the two approaches agree within a factor of two at all energies and within about 20% at energies greater than about 500 keV. The largest differences between the methods occur at very low energies (10 keV or less) and at energies near 100 keV. The disagreement at 10 keV or less probably results from poor data underlying the point-source kernel method at very low energies. The disagreement at energy levels near 100 keV probably is due largely to the inability of the point-source kernel method to account properly for the effects of scattering.

The type of comparisons described above have been used to determine correction factors for values generated by the point-kernel method (Cristy and Eckerman, 1987). It appears that uncertainties associated with photon absorbed fractions can be minimized by applying a weighted average of the specific absorbed fraction $SAF(T,S)$ and the reciprocal $SAF(T,S)$ produced by the Monte Carlo method for most situations, but replacing this value with the corrected point-kernel value whenever $SAF(T,S)$ is statistically unreliable (usually at low energies) (Cristy and Eckerman, 1987).

SEs for beta particles and discrete electrons

Beta particles and discrete electrons usually are not sufficiently energetic to contribute significantly to cross-irradiation doses of targets separated from a source organ. Thus, for these

radiation types it is generally assumed that $SAF(S,S)$ is the inverse of the mass of organ S, and if source S and target T are separated, $SAF(T,S) = 0$. Exceptions occur when the source and target are in close proximity, which can occur in the respiratory tract or in the skeleton.

In the respiratory tract, there are narrow layers of radiosensitive basal and secretory cells in the epithelium. These are irradiated to some extent by beta particles and discrete electrons emanating from nearby "source organs", including the gel layer, the sol layer, and other identified compartments within the epithelium.

The skeleton is generally represented as a uniform mixture of its component tissues: cortical bone, trabecular bone, fatty marrow, red marrow, and connective tissues. Tissues of interest for dosimetric purposes are the red marrow, which lies within the generally tiny cavities of trabecular bone, and osteogenic cells adjacent to the surfaces of both cortical and trabecular bone. For the red marrow the pertinent dose is assumed to be the average dose to the marrow space within trabecular bone. For the osteogenic tissue, the ICRP recommends that the equivalent dose be calculated as an average over tissues up to a distance of 10 μm from the relevant bone surface.

In the vicinity of discontinuities in tissue compositions such as that between bone mineral and soft tissues, the assumption that the skeleton is a uniform mixture of its component tissues can lead to sizable errors in estimates of dose from beta particles and discrete electrons, as well as photons. For example, neglect of energy transferred to electrons by photon interactions in these regions can result in overestimates of dose to bone marrow by as much as 300-400% for photon energies less than 100 keV. Similarly, conventional methods for treating beta emissions in the skeleton may substantially overestimate the dose to soft tissues of the skeleton. With regard to the ICRP's *SE* values, this problem was recently addressed with regard to photons (Cristy and Eckerman, 1993) but conventional methods are still used for treatment of beta emissions.

SEs for alpha particles

The energy of alpha particles and their associated recoil nuclei is generally assumed to be absorbed in the source organ. Therefore, for alpha particles, $SAF(S,S)$ is taken to be the inverse of the mass of the source organ S, and $SAF(T,S) = 0$ if S and T are separated.

If an alpha emitter is uniformly distributed on the surface of trabecular bone then, by simple geometric considerations, the absorbed fraction in the marrow space is one half. Lacking information on the location of the hematopoietic stem cells, the ICRP assumes that the cells are uniformly distributed within the marrow space. If the sensitive cells were located more than 10 μm

from the bone mineral surface, the relevant absorbed fraction would be reduced to 0.23-0.34 for an alpha emitter with energy in the range 5-8 MeV.

For an alpha emitter uniformly distributed in the mineral of trabecular bone, the absorbed fraction in the red marrow depends on the energy of the alpha particle. Calculations for alpha emitters ranging in energy from 5 to 8 MeV indicate that the absorbed fraction in the marrow space ranges between 0.041 and 0.087, which bracket the value of 0.05 recommended by the ICRP. If the sensitive cells were located more than 10 μm from the bone mineral surface, the relevant absorbed fraction would be reduced to 0.015-0.055. Thus, dose estimates to skeletal tissues for alpha emitters are sensitive to assumptions regarding the spatial relationship between the source and target regions.

For an alpha emitter uniformly distributed in bone mineral, estimates of the absorbed fraction in bone surface ranges from less than 0.02 to more than 0.03, depending on the energy of the alpha particle. The nominal value recommended by the ICRP is 0.025.

Special dosimetric problems presented by walled organs

The so-called "walled organs" of the body are the parts of the gastrointestinal tract and the bladder in which the radionuclide may be present in the contents of the organ. In the case of beta radiation, it is assumed that the dose to the wall of the organ is equivalent to the dose at the surface of the contents. For beta particles of low energy this approach overestimates the dose to the wall and to the cells associated with maintaining the epithelial lining of the wall. For alpha radiations the dose to the wall is taken as 1% of the dose at the surface of the contents. This value is not based on calculations of energy deposition but is a cautiously high value based on an acute toxicity study on rats (Sullivan et al., 1960). In this study, the LD_{50} for ingested ^{91}Y was estimated as about 12 Gy while a more than 100-fold greater dose to the mucosal surface from ^{239}Pu had no effect. Continued use of the presumably cautious dosimetry for walled organs is due in part to concerns that some radioelements may be retained in the walls of these organs to a greater extent than commonly modeled. Also, with regard to the intestines, considerable difficulties are encountered in defining the appropriate geometry of the convoluted wall and the contents of this organ.

CHAPTER 6. DOSIMETRIC MODELS FOR EXTERNAL EXPOSURES

Three external exposure scenarios are considered in this report: submersion in a semi-infinite cloud, exposure to ground surface contamination, and exposure to soil contaminated to an infinite depth. Persons are assumed to be exposed throughout their lifetimes to a unit concentration of the radionuclide in air, on the ground surface, or in soil.

Dose rate coefficients from external exposure are taken from Federal Guidance Report No. 12 (EPA, 1993), which tabulates coefficients for external exposure to photons and electrons. The coefficients are based on state-of-the-art methods for calculating the energy and angular distribution of the radiations incident upon the body and the transport of these radiations within the body.

Tabulations in Federal Guidance Report No. 12 are for a reference adult, as defined in ICRP Publication 23. Calculations were based on the 70-kg phantom of Cristy (Cristy and Eckerman, 1987), with two modifications: (1) the head region was made more realistic by including a neck and shortening the right elliptical cylinder comprising the lower portion of the head; and (2) a model of the esophagus was added.

Although there is expected to be some age dependence in organ dose rates from external exposures, comprehensive tabulations of age-specific external doses are not yet available. Therefore, the tabulations for the reference adult in Federal Guidance Report No. 12 are applied to all age groups. As discussed later, the application of these external dose coefficients to other age groups appears to result in relatively small errors (usually <30%) in most cases. In extreme cases, such as for external irradiation of deep organs (e.g., ovaries or colon) of infants at energies less than 100 keV energies, 2- to 3-fold errors may arise. In applications of the derived risk coefficients, however, errors arising from application of age-independent external dose rates are likely to be negligible compared with errors associated with the simplified exposure scenarios used here (e.g., constant placement and position, no shielding, and infinite or semi-infinite source regions). Simplified exposure scenarios are used here because it is not feasible to develop an external dosimetric methodology that applies to arbitrary distributions of contamination or to differences in life styles.

Interpretation of dose coefficients from Federal Guidance Report No. 12

Dose coefficients for external exposure relate the dose to organs and tissues of the body to the concentration of radionuclides in environmental media. The term "external exposure" is used to indicate that the radiations originate outside the body. The radiations of concern are those that

are sufficiently penetrating to traverse the overlying tissues of the body and thus are limited to photons, including bremsstrahlung, and electrons.

Because it is not feasible to develop an external dosimetric methodology that applies to arbitrary distributions of radionuclides in environmental media, it has become common practice to consider simplified and idealized exposure geometries. In particular, a semi-infinite source region generally is assumed for submersion in contaminated air, and an infinite source region generally is assumed for exposure to contaminated soil.

If one assumes an infinite or semi-infinite source region with a uniform concentration $C(t)$ of a radionuclide at time t, then the equivalent dose in tissue T, H_T, can be expressed as

$$H_T = h_T \int C(t)\ dt \tag{6.1}$$

where h_T denotes the time-independent dose coefficient for external exposure. The coefficient h_T represents the dose to tissue T of the body per unit time-integrated exposure (integrated concentration of the radionuclide). That is,

$$h_T = \frac{H_T}{\int C(t)\ dt} \quad . \tag{6.2}$$

Alternatively, one may interpret h_T as representing the instantaneous dose rate in organ T per unit activity concentration of the radionuclide in the environment. Furthermore, since only low-LET radiations are considered in the derivation of external dose coefficients, equivalent and absorbed doses are numerically equal.

In Federal Guidance Report No. 12, h_T is interpreted as the dose per unit time-integrated exposure. In this report, however, h_T is interpreted as a dose rate, because dose rates are required as input into the radiation risk methodology applied here.

Nuclear data files used

The energies and intensities of the radiations emitted in spontaneous nuclear transformations of radionuclides have been reported in Publication 38 of the International Commission on Radiological Protection (ICRP, 1983). That publication is a report of the Task Group on Dose Calculations of ICRP Committee 2 and was assembled at Oak Ridge National Laboratory (ORNL) during the preparation of ICRP Publication 30 (ICRP, 1979). The nuclear decay data of ICRP

Publication 38 are based on the Evaluated Nuclear Structure Data Files (ENSDF) (Ewbank and Schmorak, 1978) of the Department of Energy's Nuclear Data Project as processed by the EDISTR code (Dillman, 1980). The processed data files retained in the ICRP/ORNL dosimetric data base include full tabulations of the average or unique energies and intensities of the radiations and also the beta spectra (Eckerman et al., 1994). The dose coefficients for external irradiation given in Federal Guidance Report No. 12 are based on these data files.

Radiations considered

For external exposures, the radiations of concern are those that are sufficiently penetrating to traverse the overlying tissues of the body and deposit ionizing energy in radiosensitive organs and tissues. Photons and electrons are the most important penetrating radiations produced by radionuclides in the environment.

Some radionuclides produce bremsstrahlung that is sufficiently penetrating to be of potential importance in the estimation of external dose. Bremsstrahlung, from the German for "braking radiation", is produced when deceleration of electrons in a medium results in conversion of a small fraction of their initial kinetic energy into energy in the form of photons. Bremsstrahlung energy is distributed from zero up to the initial electron energy. The bremsstrahlung yield is small (about 0.5% at 1.0 MeV in tissue) but for pure beta emitters can be the only source of radiations of sufficient penetrating nature to irradiate some radiosensitive tissues.

The types of radiations considered in Federal Guidance Report No. 12 are photons, including bremsstrahlung, and electrons. The energy spectrum of emitted radiations can be characterized as either (1) discrete emissions of a unique energy (e.g, gamma radiation), and (2) continuous energy distribution of electrons as in the case of beta particles and bremsstrahlung. The beta spectra are used in Federal Guidance Report No. 12 to evaluate the contribution of the beta particles to the skin dose and to determine the yield of bremsstrahlung.

Spontaneous fission occurs in the decay of several radionuclides in the actinide series and results in the emission of photons, electrons, and neutrons, as well as fission fragments. Spontaneous fission is an important decay mode for only a few radionuclides, e.g., ^{248}Cm, ^{252}Cf, ^{254}Cf, and ^{256}Fm. For these cases (none of which are considered in the present document), the dose coefficients given in Federal Guidance Report No. 12 may considerably underestimate true doses due to neglect of the contribution to dose from spontaneous fission in that document. However, equivalent doses from external exposures associated with spontaneous fission usually will be unimportant in dose assessments for members of the public, either because radionuclides with

significant branching fr`ctions for spontaneous fission will occur in relatively small concentrations in the environment or because equivalent doses from internal exposure will be more important for these nuclides.

Effects of indoor residence

The dose coefficients for air submersion and exposure to contaminated soil are taken from Federal Guidance Report No. 12 (EPA, 1993). These dose coefficients assume that exposed individuals spend all of the time outdoors. Depending on such factors as photon energy, type of structure, fraction of time spent indoors, and degree of disequilibrium in the concentration of a radionuclide in indoor and outdoor air, there could be a substantial reduction in the equivalent dose from external exposures during indoor residence due to shielding by structures.

For noble-gas radionuclides, air submersion is the only external exposure mode of concern. The effects of indoor residence on equivalent doses to skin due to electrons should be negligible during chronic releases, unless the range of the emitted electrons in air is somewhat greater than the interior dimensions of building rooms, because the indoor and outdoor air concentrations for noble gases will be about the same.

A radionuclide-independent dose reduction factor is sometimes applied to external dose coefficients to account for the effects of indoor residence (e.g., NRC, 1977). However, the average reduction in external dose due to indoor residence depends on the radionuclide as well as other factors indicated above and generally cannot be quantified with much certainty. In the present document, the external dose coefficients given in Federal Guidance Report No. 12 are not reduced to account for the effects of indoor residence.

Uncertainties in external dose models

Transport of radiation from the environmental source to humans

In Federal Guidance Report No. 12 (EPA, 1993), the problem of estimating external dose rates from contaminated air, soil, or ground surfaces was divided into two steps: (1) the calculation of the radiation field incident on the surface of the body; and (2) calculation of organ dose rates due to a body surface source. The uncertainties associated with the second step are essentially the same as those discussed in Chapter 5 with regard to internal radiation sources.

The method of calculation of the external radiation field was checked as far as practical against other theoretical methods or experimentally determined values (EPA, 1993). The results of the comparisons suggest that the external radiation fields can be determined with reasonably high accuracy, at least for the idealized geometries generally considered. For example, derived values for the case of a contaminated ground source were checked by comparing the energy and angular dependence of the air kerma above a 1.25 MeV plane source at the air-ground interface with calculations of Beck and de Planque (1968) based on another method and with the calculations and measurements given in the Shielding Benchmark Problems report (Garrett, 1968). Agreement was within a few percent in both cases.

The largest differences between the modeled external radiation fields and real-world situations probably arise from differences between the simplified exposure geometries and real exposure geometries. An important example is exposure to contaminated ground surface, for which the source region is assumed to be a smooth plane. In the real world, external dose rates from sources on the ground surface generally are reduced by the shielding provided by "ground roughness", including terrain irregularities and surface vegetation. Dose-reduction factors for a photon spectrum representative of fallout following releases from nuclear reactors are given by Burson and Profio (1977). The recommended values range from essentially unity for paved areas to about 0.5 for a deeply plowed field, and a representative average value is about 0.7. Such dose-reduction factors for ground roughness should overestimate equivalent doses due to external exposure to contaminated ground surfaces if the radionuclides emit mostly low-energy photons (Kocher, 1980).

Effects of shielding during indoor residence

The dose coefficients for air submersion and exposure to contaminated soil assume that exposed individuals spend all of the time outdoors and have no shielding from the radiation (EPA, 1993). For the typical adult male considered in Federal Guidance Report No. 12, one of the largest uncertainties in the external dose rates as applied in the present report is the question of whether a uniform reduction factor, or possibly radionuclide-specific reduction factors, should be used to account for shielding during indoor residence. In the present document, no reduction factors are applied. This approach may be appropriate for some radionuclides (e.g., for some radioisotopes of noble gases) but probably leads to a substantial overestimate of actual dose rates for external exposures in many cases. It is left to the user to decide whether a reduction factor is appropriate for a given application.

For acute releases of radionuclides into the atmosphere, the relationship between indoor and outdoor airborne concentrations of radionuclides will vary with time during and after a release and will also depend strongly on the air exchange rate inside a building (Wallace, 1996). For such releases, a fixed reduction of external dose rates to account for indoor residence would not appear to be appropriate.

Effects of age and gender

The dose coefficients tabulated in Federal Guidance Report No. 12 were calculated for an anthropomorphic model of the adult body derived by Cristy (Cristy and Eckerman, 1987) from ICRP Reference Man data (ICRP, 1975). For all calculations, the phantom is upright at the air-ground interface. The phantom is a hermaphrodite of design similar to that used in the dosimetric evaluation of ICRP Publication 30 (ICRP, 1979).

Age- and gender-specific aspects of external dose have been considered by Drexler et al. (1989) and Petoussi et al. (1991). Limited calculations indicate that the dose to organs of the body from external radiation increases with decreasing body size. This effect is more pronounced at low photon energy than at high energy and is also more pronounced for organs located deep in the body than for more shallow organs with less shielding by overlying tissues.

Calculated effects of age on the effective dose per unit photon fluence are indicated in Fig. 6.1 for the case of photons uniformly distributed in angle (isotropic field). Estimates for intermediate ages fall between the curves for the adult and infant. Similar effects of age were calculated for the case of a broad parallel horizontal beam uniformly distributed about the phantom (rotational normal beam). The isotropic field corresponds to a photon source uniformly distributed in the air (submersion) and the rotational normal beam is similar to the situation in which the photon source is distributed on the ground surface. For both cases, the dependence of the effective dose on age increases at the low photon energy and exceeds a factor of two at energies less that about 0.050 MeV. It is for low photon

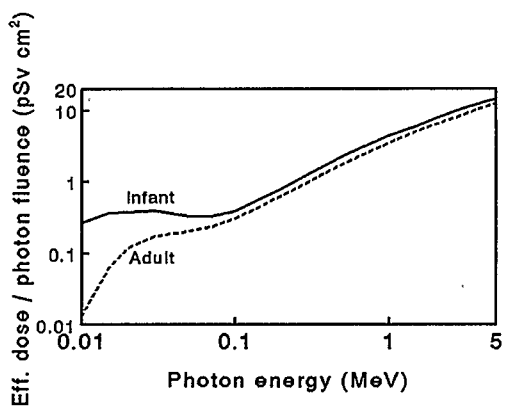

Fig. 6.1. Estimated effects of age on effective dose for photons uniformly distributed in angle.

energies that the reduction in dose by shielding by buildings structures becomes increasingly effective. Uncertainties associated with the use of age-independent external dose rates appear to be overshadowed in most cases by uncertainties associated with shielding and exposure geometries.

CHAPTER 7. RADIOGENIC CANCER RISK MODELS

Calculations of radiogenic risk are based on risk projection models for specific cancer sites. The age- and gender-specific radiation risk models used in this report are taken from a recent EPA report (EPA, 1994) that provides a methodology for calculation of radiogenic cancer risks based on a critical review of data on the Japanese atomic bomb survivors and other study groups. Parameter values in the models have been modified in some cases in the present report to reflect the use of updated vital statistics for the U.S. and to achieve greater consistency in the assumptions made for different age groups and genders. The following age-at-exposure groups are considered in the models: 0-9, 10-19, 20-29, 30-39, and 40+ y.

Types of risk projection models

One of two basic types of radiogenic cancer risk projection models is used for a given cancer site: an absolute risk model or a relative risk model. An absolute risk model is based on the assumption that the age-specific excess force of mortality or morbidity (that is, the mortality or morbidity rate for a given cancer type) due to a radiation dose is independent of cancer mortality or morbidity rates in the population. A relative risk model is based on the assumption that the age-specific excess force of mortality or morbidity due to a radiation dose is the product of an exposure-age-specific relative risk coefficient and baseline cancer mortality or morbidity rate. In this report, risk models for bone, skin, and thyroid cancer are based on an absolute risk hypothesis, and risk models for other sites are based on a relative risk hypothesis.

In the absolute risk models used in this report, the absolute risk $\epsilon(x,x_e)$ at age x due to a unit absorbed dose received at an earlier age x_e ($x_e < x$) is calculated as

$$\epsilon(x,x_e) = \alpha(x_e)\, \zeta(t), \tag{7.1}$$

where:

$\alpha(x_e)$ is a non-negative number, called a "risk model coefficient", that depends on gender as well as age at exposure; and

$\zeta(t)$ is either 0 or 1, depending on the time since exposure, $t = x - x_e$.

The function α defines the potential level of risk of dying from or experiencing a given type of cancer at any given age (and hence time) after the dose is received, and ζ defines the plateau period, that is, the time period during which the risk is expressed.

In the relative risk models used in this report, $\epsilon(x,x_e)$ is calculated as $\epsilon(x,x_e) = \mu(x) \times \eta(x,x_e)$, where $\mu(x)$ is the baseline force of cancer mortality or morbidity at age x and $\eta(x,x_e)$ is the relative risk at age x due to a unit absorbed dose received at age x_e ($x_e < x$); $\eta(x,x_e)$ is calculated as

$$\eta(x,x_e) = \beta(x_e)\, \zeta(t,x_e), \tag{7.2}$$

where

$t = x - x_e$;

$\beta(x_e)$ is a non-negative number, called a "risk model coefficient", that depends on gender as well as age at exposure; and

$\zeta(t,x_e)$ is the relative magnitude of the response at different times after exposure at age x_e.

For all cancers except leukemia, it is assumed that ζ is independent of the exposure age x_e and has a value of either 0 or 1, depending on the time since exposure, $t = x - x_e$. The time-since-exposure response function $\zeta(t,x_e)$ for either chronic granulocytic leukemia or for acute leukemia is given by $\zeta(t,x_e) = 0$ if $t \leq 2$ y and $\zeta(t,x_e) = \phi(t,\xi(x_e),\sigma^2)$ if $t > 2$ y, where

$$\phi(t,\xi(x_e),\sigma^2) = \frac{\exp(-0.5(\ln(t-2) - \xi(x_e))^2 / \sigma^2)}{(t-2)(2\pi\sigma^2)^{0.5}} . \tag{7.3}$$

In this expression, the function $\xi(x_e)$ and the value σ^2 depend on the type of leukemia. For chronic granulocytic leukemia, $\xi(x_e) = 2.68$ and $\sigma^2 = 1.51$. For acute leukemia, $\xi(x_e) = 1.61 + 0.015x_e + 0.0005x_e^2$ and $\sigma^2 = 0.65$ (EPA, 1994). The total leukemia time since response function is a weighted mean of the response function for chronic granulocytic leukemia, which is given a weight of 0.32, and the response function for acute leukemia, which is given a weight of 0.68 (EPA, 1994).

The function β in Eq. 7.2 times the baseline force of cancer mortality or morbidity, $\mu(x)$, at a given age defines the potential level of risk of dying from or experiencing a given type of cancer at that age, and ζ defines the period during which the risk is expressed and, in the case of leukemia, the changes in the level of response during that period. Because the time since response function for leukemia is scaled differently from the time since response functions for other cancers and has a maximum value much less than 1, the risk model coefficients (age- and gender-specific values of β) for leukemia are not directly comparable with the risk model coefficients for other cancers.

The term "risk coefficient" used in the EPA report on radiation risk models (EPA, 1994) has been replaced here with the term "risk model coefficient" to avoid confusion with the radionuclide risk coefficients tabulated in Chapter 2. The risk coefficients given in Chapter 2 refer to risk per unit intake or external exposure to a specific radionuclide in a specific environmental medium.

Epidemiological studies used in the development of risk models

The risk model coefficients given in the EPA report (EPA, 1994) were based in large part on information from studies of the Radiation Effects Research Foundation (RERF) Life Span Study (LSS) cohort of Hiroshima and Nagasaki atomic bomb survivors (Shimizu et al., 1989, 1990). This study has the advantages that it includes a large, relatively healthy population at the time of exposure, a wide range of reasonably well established doses to individual subjects (although some important dosimetric issues remain), a large, well matched control group (that is, people who were present in Hiroshima or Nagasaki at the time of bombing but who received only small doses of radiation), and a detailed, long-term epidemiological follow-up. A statistically significant excess cancer mortality associated with radiation has been found among the bomb survivors for the following types of cancer: leukemia, esophagus, stomach, colon, liver, lung, breast, ovary, urinary tract, and multiple myeloma.

Results of other epidemiological studies on radiation-exposed populations were used for development of risk models for a few sites for which the A-bomb survivor do not appear to provide best available information on radiogenic risk. For example, risk models for the thyroid and breast were based primarily on results of epidemiological studies of medical exposures of these organs. For two other sites, bone and liver, low-LET risk estimates were extrapolated from results of epidemiological studies of humans exposed to ^{224}Ra and thorotrast, respectively (EPA, 1994), together with data on comparative biological effectiveness of alpha and low-LET radiations in laboratory animals. There are additional important epidemiological studies of persons exposed either to low-LET or high-LET radiation, but the main use of these additional studies was for comparison with results for the A-bomb survivors.

Modification of epidemiological data for application to low doses and dose rates

All of the epidemiological studies used in the development of the radiation risk models involve subjects who experienced high radiation doses delivered in a relatively short time. Available evidence indicates that the response per unit dose at low doses and dose rates from low-LET radiation may be overestimated if one extrapolates from observations made at high, acutely delivered doses (NCRP, 1980). The degree of overestimation is commonly expressed in terms of a dose and dose rate effectiveness factor (DDREF): e.g., a DDREF of 2 means the risk per unit dose observed at high acute doses should be divided by 2 before being applied to low doses or low dose rates. "Low

dose" and "low dose rate" are defined here in terms of the range of applicability of a DDREF of 2; "low dose" is defined as <0.2 Gy and "low dose rate" is defined as <0.1 mGy min^{-1} (UNSCEAR, 1993; EPA, 1994). For comparison, the ICRP (1991) used a DDREF of 2 in the calculation of probability coefficients for all equivalent doses below 0.2 Gy and from higher doses resulting from absorbed dose rates less than 0.1 Gy h^{-1} (about 1.7 mGy min^{-1}).

In the EPA report on radiation risk models (EPA, 1994) and hence in the present report, low-LET radiogenic cancer risks for sites other than the breasts are assumed to be reduced by a DDREF of 2 at low doses and low dose rates compared to risks at high acute dose exposure conditions. The DDREF assumed for breast cancer is 1. Risks from high-LET (alpha particle) radiation are assumed to increase linearly with dose and to be independent of dose rate.

Relative biological effectiveness factors for alpha particles

With the exception of radiation-induced breast cancer and leukemia, the EPA has followed the ICRP's recommendation (ICRP, 1991) and assumed that the relative biological effectiveness (RBE) for alpha particles is 20, in comparison to low-LET radiation at low doses and dose rates (EPA, 1994). For leukemia, an effective alpha particle RBE of 1 is used (see discussion of uncertainties of RBE). For breast cancer, an alpha particle RBE of 10 is used.

Where comparison was made in the EPA report (EPA, 1994) against acute high doses of low-LET radiation, a value of 10 was assumed for the alpha particle RBE. This is consistent with the RBE of 20 relative to acute, low-dose, low-LET radiation, given the assumption of a DDREF of 2 for low-LET radiation at low doses and dose rates.

Risk model coefficients for specific organs

Age- and gender-specific risk model coefficients used in this report are summarized in Table 7.1 for cancers other than leukemia and in Table 7.2 for leukemia. Risk model coefficients for esophagus, stomach, colon, lung, ovary, bladder, leukemia, and "residual" are based on updated information on the Japanese atomic bomb survivors and are derived using a slightly modified version of a model of Land and Sinclair (1991). The risk model coefficients for these sites are obtained by taking the geometric mean of model coefficients derived from two equally plausible methods described by Land and Sinclair for transporting risk from one population to another. Both methods assume a constant excess relative risk coefficient beginning 10 y after an exposure and continuing throughout the rest of life for each cancer site, excluding leukemia. One method (multiplicative)

Table 7.1. Revised mortality risk model coefficients[a,b] for cancers other than leukemia, based on the EPA radiation risk methodology (EPA, 1994).

Cancer type	Risk model type[c]	Age group (x_e) 0-9 y	10-19 y	20-29 y	30-39 y	40+ y
Male:						
Esophagus	R	0.2877	0.2877	0.2877	0.2877	0.2877
Stomach	R	1.223	1.972	2.044	0.3024	0.2745
Colon	R	2.290	2.290	0.2787	0.4395	0.08881
Liver	R	0.9877	0.9877	0.9877	0.9877	0.9877
Lung	R	0.4480	0.4480	0.0435	0.1315	0.1680
Bone	A	0.09387	0.09387	0.09387	0.09387	0.09387
Skin	A	0.06597	0.06597	0.06597	0.06597	0.06597
Breast	R	0.0	0.0	0.0	0.0	0.0
Ovary	R	0.0	0.0	0.0	0.0	0.0
Bladder	R	1.037	1.037	1.037	1.037	1.037
Kidney	R	0.2938	0.2938	0.2938	0.2938	0.2938
Thyroid	A	0.1667	0.1667	0.08333	0.08333	0.08333
Residual	R	0.5349	0.5349	0.6093	0.2114	0.04071
Female:						
Esophagus	R	1.805	1.805	1.805	1.805	1.805
Stomach	R	3.581	4.585	4.552	0.6309	0.5424
Colon	R	3.265	3.265	0.6183	0.8921	0.1921
Liver	R	0.9877	0.9877	0.9877	0.9877	0.9877
Lung	R	1.359	1.359	0.1620	0.4396	0.6047
Bone	A	0.09387	0.09387	0.09387	0.09387	0.09387
Skin	A	0.06597	0.06597	0.06597	0.06597	0.06597
Breast	R	0.7000	0.7000	0.3000	0.3000	0.1000
Ovary	R	0.7185	0.7185	0.7185	0.7185	0.7185
Bladder	R	1.049	1.049	1.049	1.049	1.049
Kidney	R	0.2938	0.2938	0.2938	0.2938	0.2938
Thyroid	A	0.3333	0.3333	0.1667	0.1667	0.1667
Residual	R	1.122	1.122	0.8854	0.3592	0.1175

[a]The tabulated risk model coefficients are the precise values derived from the epidemiological data and used in the calculations. The use of four significant digits should not be interpreted as indicating a low level of uncertainty in the risk model coefficients.

[b]Age-specific risk model coefficients were used to derive composite risk coefficients representing averages over all ages. Application of these risk model coefficients to a specific age group is not recommended due to the high sampling variability in the underlying epidemiological data for some age groups.

[c]A indicates that an absolute risk model is used (coefficient units, 10^{-4} Gy^{-1} y^{-1}), and R indicates that a relative risk model is used (Gy^{-1}). $\alpha(x_e)$ is given for absolute risk model (Eq. 7.1) and $\beta(x_e)$ for a relative risk model (Eq. 7.2).

Table 7.2. Revised mortality risk model coefficients (Gy^{-1}) for leukemia, based on the EPA radiation risk methodology (EPA, 1994).[a]

	Age group (x_e)				
Gender	0-9 y	10-19 y	20-29 y	30-39 y	40+ y
Male	982.3	311.3	416.6	264.4	143.6
Female:	1176	284.9	370.0	178.8	157.1

[a]A relative risk model is used (coefficient units, Gy^{-1}). Risk model coefficients for leukemia are not directly comparable to those for other types of cancer (Table 7.1) due to differences in the scales of the time-since-exposure response functions for leukemia and other cancers (see the discussion following Eq. 7.2).

assumes that the relative risk estimator is the same across populations. The other (NIH, for National Institutes of Health) assumes that the relative risk model coefficients for the target population should yield the same risks as those calculated with the additive risk model coefficients from the original population over the period of epidemiological follow-up, excluding the minimal latency period. These excess relative risk model coefficients are then used to project the risk over the remaining years of life. The data considered in deriving risk model coefficients consisted of cancers observed 10-40 y after exposure for solid tumors and 5-40 y after exposure for leukemia.

As described below, some modifications in the method of calculation of the NIH model coefficients have been made to remove inconsistencies in the derived coefficients. Some but not all of these changes were made in the EPA report on radiation risk models (EPA, 1994); therefore, some of the risk coefficients in Tables 7.1 and 7.2 differ from values given in that report.

An examination of the coefficients for the additive and multiplicative models of Land and Sinclair (1991) reveals that in several instances data for exposures of two or more age groups were combined to calculate a single risk coefficient. In such cases, a single NIH model coefficient has been calculated for use in the present report by combining the risks calculated for the corresponding groups. This was done in the EPA report (EPA, 1994) for model coefficients for lung and colon cancer for two exposure age groups (0-9 y and 10-19 y), and the same principle has been extended in the present report to the coefficients for esophagus, ovary, and bladder cancer. For these three sites, the age-group-specific additive coefficients of Land and Sinclair were based on a single-coefficient multiplicative risk model. For the present report, a NIH model excess relative risk coefficient has been calculated corresponding to the combined risk for exposure for all age-groups, expressed 10-40 years after exposure for the additive risk model.

EPA (1994) noted inconsistencies between ages and between genders in the additive and multiplicative risk models of Land and Sinclair (1991) with regard to coefficients for the residual site for age groups 0-9 y and 10-19 y. These inconsistencies may be the result of uncertain differences between the total observed excess cancers and the sum of those attributed to specific sites. In the EPA report (EPA, 1994), risk model coefficients for the residual site for age group 10-19 y were applied to age group 0-9 y. For the present report, the additive model risks for these two age groups have been combined to calculate gender-specific, single coefficients for the NIH risk model. Single risk coefficients equivalent to the risks projected by the multiplicative model for 10-40 y following exposure of those in this age group were also calculated. These values were used to calculate gender-specific risk model coefficients for these two age groups for the EPA risk model.

For kidney, the LSS data are suggestive of a radiogenic risk but the number of excess cancers is not statistically significant. The existence of a radiogenic kidney cancer risk is indicated by an epidemiological study of subjects receiving radiation treatments for cervical cancer (NAS, 1990; Boice et al., 1988). Given the importance of the kidney as a possible target organ for uranium and some other radionuclides, the EPA (1994) has developed a risk model for this site based on the LSS data. A constant relative risk model independent of age at exposure and sex is used, and a 10-y latency period is assumed.

Risk model coefficients for the liver are based on epidemiological data on patients injected with Thorotrast, an x-ray contrast medium containing isotopes of thorium (NAS, 1980, 1988). To develop risk model coefficients for high-dose, low-LET radiation, an RBE of 10 is assumed for alpha particles. A constant relative risk model independent of age at exposure and sex is used, and a 10-y latency period is assumed.

Estimates of skin cancer risks are highly uncertain, but the mortality risk is known to be relatively low. For acute exposures, the EPA has adopted the mortality risk estimate given in ICRP Publication 60 (1991) but, in contrast to ICRP, has applied a DDREF of 2 in estimating the skin cancer risk at low doses and dose rates. Non-fatal skin cancers, which represent perhaps 99.99% of basal cell carcinomas and about 99% of squamous cell carcinomas, are excluded from the risk model coefficients. A 10-y latency period is assumed.

Thyroid risk estimates are based on NCRP Report 80 (NCRP, 1985). The Nuclear Regulatory Commission (NRC) and the ICRP have also adopted this approach (NRC, 1991, 1993; ICRP, 1991). The mortality risk is assumed to be one-tenth the morbidity risk. The estimated morbidity and mortality risks are each reduced by a factor of 3 in the case of exposures to iodine-125, -129, and -131. This reduction includes the effect of lowered dose rate on the risk, as well as possible other factors. Hence, the DDREF of 2 applied to organ specific risk estimates is not

applied in the case of exposure to one of these radionuclides. A latency period of 5 y is assumed for radiogenic thyroid cancers.

As a basis for estimating radiation-induced bone sarcomas, the EPA has adopted BEIR IV's risk estimate based on alpha irradiation by ^{224}Ra (NAS, 1988). However, this risk estimate refers to *average skeletal dose* and has previously been applied incorrectly as endosteal cell dose. For example, bone cancer risk appears to be substantially overestimated in ICRP Publication 60 (1991) due to a confusion between endosteal and average skeletal doses (Puskin et al., 1992). Because the bone seeker ^{224}Ra decays quickly, the endosteal dose from injected ^{224}Ra is estimated to be an order of magnitude higher than the average skeletal dose. Thus, a risk model coefficient derived in terms of average skeletal dose, if applied to average endosteal dose, would overestimate the radiation-related risk of bone cancer. Risk model coefficients for high-dose, low-LET radiation are derived by dividing values based on alpha irradiation by a factor of 10 and reducing the risk model coefficients by another 30% to account for the fact that about 70% of bone sarcomas are fatal. Following BEIR III (NAS, 1980), a constant absolute risk model is used to project risk, with an expression period extending from 2 to 27 y after exposure.

For breast cancer, the EPA has adopted a model of Gilbert developed for the U.S. Nuclear Regulatory Commission (NRC, 1991, 1993) and based on data for persons receiving medical exposures to radiation. A major issue with regard to breast cancer is in the transport of risk from Japan to the U.S., where the baseline rates are much higher. The model of Gilbert for breast cancer avoids this problem because it is based on North American data.

Site-specific cancer mortality risk estimates from low-dose, low-LET uniform irradiation of the whole body, based on the risk model coefficients in Tables 7.1 and 7.2, are given in Table 7.3. These estimates are age-averaged values for the hypothetical stationary population described in Chapter 3. The method of computation is described in a later section.

Association of cancer type with dose location

The dose locations associated with the different cancer types are shown in Table 7.4. When more than one dose location is associated with a given cancer type, risks are calculated for a weighted mean of the doses at these locations using the weights shown in the table. For specific cancer types, the association of cancer type with dose location follows recommendations in ICRP Publication 60 (1991), except that the weights assigned to regions within the colon and lung are based on more recent recommendations in ICRP Publication 66 (1994a) and 67 (1993), respectively.

Table 7.3. Age-averaged site-specific cancer mortality risk estimates (cancer deaths per person-Gy) from low-dose, low-LET uniform irradiation of the body.

Site	Combined genders	Males	Females
Esophagus	1.17×10^{-3}	7.30×10^{-4}	1.59×10^{-3}
Stomach	4.07×10^{-3}	3.25×10^{-3}	4.86×10^{-3}
Colon	1.04×10^{-2}	8.38×10^{-3}	1.24×10^{-2}
Liver	1.50×10^{-3}	1.84×10^{-3}	1.17×10^{-3}
Lung	9.88×10^{-3}	7.71×10^{-3}	1.19×10^{-2}
Bone	9.50×10^{-5}	9.40×10^{-5}	9.60×10^{-5}
Skin	1.00×10^{-4}	9.51×10^{-5}	1.05×10^{-4}
Breast	5.06×10^{-3}	0.00	9.90×10^{-3}
Ovary	1.49×10^{-3}	0.00	2.92×10^{-3}
Bladder	2.38×10^{-3}	3.28×10^{-3}	1.52×10^{-3}
Kidney	5.15×10^{-4}	6.43×10^{-4}	3.92×10^{-4}
Thyroid	3.24×10^{-4}	2.05×10^{-4}	4.38×10^{-4}
Leukemia	5.57×10^{-3}	6.48×10^{-3}	4.71×10^{-3}
Residual[a]	1.49×10^{-2}	1.35×10^{-2}	1.63×10^{-2}
Total	5.75×10^{-2}	4.62×10^{-2}	6.83×10^{-2}

[a]Residual is a composite of all radiogenic cancers that are not explicitly identified by site in the model.

The residual cancer category represents a composite of primary and secondary cancers that are not otherwise considered in the model. The three dose locations associated with these cancers (skeletal muscle, pancreas, and adrenals) were chosen to be generally representative of doses to soft tissues and are not considered to be the sites where all residual neoplasms originate.

Relation between cancer mortality and morbidity

To obtain estimates of radiation-induced cancer morbidity, each site-specific mortality risk estimate is divided by its respective lethality fraction, that is, the fraction of radiogenic cancers at

Table 7.4. Dose regions associated with cancer types.

Cancer type	Dose region	Weighting factor
Esophagus	Esophagus[a]	1.0
Stomach	Stomach Wall	1.0
Colon	Upper Large Intestine Wall	0.568
	Lower Large Intestine Wall	0.432
Liver	Liver	1.0
Lung	Bronchial Region – Basal Cells	0.1667
	Bronchial Region – Secretory Cells	0.1667
	Bronchiolar Region – Secretory Cells	0.3333
	Alveolar-Interstitial Region	0.3333
Bone	Bone Surface	1.0
Skin	Skin	1.0
Breast	Breasts	1.0
Ovary	Ovaries	1.0
Bladder	Urinary Bladder Wall	1.0
Kidney	Kidney	1.0
Thyroid	Thyroid	1.0
Leukemia	Red Marrow	1.0
Residual	Muscle	0.3334
	Pancreas	0.3333
	Adrenals	0.3333

[a]The esophagus has not yet been incorporated explicitly into the mathematical phantom used for internal dosimetric calculations; at present, the estimated dose to the thymus is applied to the esophagus for internal exposures.

that site which are fatal. Aside from thyroid cancer, the lethality fraction is generally assumed to be the same for radiogenic cancers as for the totality of other cancers at that site. A list of lethality fractions recommended in ICRP Publication 60 (1991) and adopted by the EPA (1994) is reproduced in Table 7.5.

Based on the methods of this report, skin is projected to contribute most of the nonfatal cancers induced by uniform whole body irradiation. At least 83% of all skin cancers are basal cell

Table 7.5. Lethality data for cancers by site in adults.[a]

Cancer site	Lethality fraction *k*
Esophagus	0.95
Stomach	0.90
Colon	0.55
Liver	0.95
Lung	0.95
Bone	0.70
Skin[b]	0.002
Breast	0.50
Ovary	0.70
Bladder	0.50
Kidney	0.65
Thyroid	0.10
Leukemia (acute)	0.99
Residual	0.71

[a]Lethality fractions (mortality-to-morbidity ratios) are from Tables B-19 of and B-20 of ICRP Publication 60 (ICRP, 1991).

[b]At least 83% of skin cancers are basal cell carcinomas (~0.01% lethality) and the remainder are squamous cell carcinomas (~1% lethality). The morbidity estimates for skin cancer given in this report reflect only fatal cases and omit the much larger number of nonfatal cases, most of which are easily curable and result in little trauma for the patient (ICRP, 1992). Left untreated, however, non-fatal skin cancers may require intensive medical treatment or be disfiguring.

carcinomas and the remainder are squamous cell carcinomas. Approximately 99.99% of the former and 99% of the latter are non-fatal. The morbidity estimates for skin cancer given in the present report reflect only fatal cases.

Treatment of discontinuities in risk model coefficients

The radiogenic cancer models described in the preceding sections are discontinuous at some times. For example, the function $\zeta(t)$ that describes the period of expression of risk for solid cancers typically has a value of zero for times between exposure and 10 y after exposure but suddenly jumps to a value of 1 starting at 10 y after exposure.

To calculate a risk coefficient for a given radionuclide and environmental medium, it is necessary to integrate functions that include such discontinuous risk model functions as factors. The integration is accomplished by fitting a smoothly varying spline function to the integrand and performing a straightforward integration of the spline function. The difficulty arises that the integral of the spline function may include unintended contributions to the risk. For example, suppose that the function to be integrated (the integrand) includes the function $\zeta(t)$ described above as a factor, and suppose the integrand is evaluated at one-year increments. Fitting a spline to the integrand provides a continuous transition from the value at 9 y to the value at 10 y but includes an unintended contribution from this interval. The problem is resolved by replacing the value of the discontinuous function at the discontinuity with the average of the values immediately above and below it. For this case, the value of the function $\zeta(t)$ at t =10 y is changed from 1 to $(0 + 1) / 2 = 0.5$.

Uncertainties in risk models

Uncertainties associated with the radiation risk models for low doses and low dose rates are difficult to quantify but are reasonably well understood in a qualitative sense. The purpose of this section is to summarize the main sources and, where feasible, provide some indication of the extent of uncertainties in the radiation risk models used in this report.

Sampling variability

Epidemiologic data on an irradiated population generally can be organized and modeled in many different ways. For example, choices can be made regarding the grouping of cancer sites, the extent of division of the study population by age and gender, the mathematical form of the dose-response, and the general form of the age and temporal dependence. Although interesting features of the data may be revealed by considering small subgroups, there is a concomitant increase in statistical variability that may preclude any meaningful improvement in the model.

For the statistical analysis of the LSS data, the deaths and person-years of survival were aggregated by city, gender, six age groups, seven follow-up intervals, and 10 radiation dose intervals (Shimizu et al. 1989). Site-specific risk coefficients were calculated with a maximum likelihood estimation method that assumes that the numbers of deaths in each group are independent Poisson variates. Based on this analysis, Shimizu and coworkers derived excess relative risk estimates with associated 90% confidence intervals for a number of cancer sites. Their analysis indicates that sampling variability could lead to sizable errors in estimates of excess relative risk, particularly for sites showing relatively small numbers of excess cancer deaths. For example, the analysis indicates a quotient B/A of about 4 for colon, 8 for ovary, and 10 for esophagus, where (A,B) is the 90% confidence interval for excess relative risk.

Diagnostic misclassification

Two types of diagnostic misclassification of cancer can occur: classification of cancers as noncancers (detection error) and erroneous classification of non-cancer cases as cancer (confirmation error). Detection errors lead to an underestimate of the excess absolute risk but do not affect the estimated excess relative risk. Confirmation errors lead to an underestimate of the excess relative risk but do not affect the excess absolute risk (NCRP, 1997). Results of a recent autopsy study by the RERF indicate that the problem of diagnostic misclassification could result in a 10-15% underestimate of excess relative risk and perhaps a 20-40% underestimate of excess absolute risk in the Japanese atomic bomb survivors (Sposto et al., 1992; NCRP, 1997).

Errors in dosimetry

In epidemiological studies of irradiated populations, organ doses generally cannot be determined with high accuracy. For internally exposed subjects, the level or pattern of intake may not be well established, and there is always incomplete information concerning the time-dependent distribution and excretion of the internally deposited radionuclide(s) and any radioactive progeny of those radionuclides produced *in vivo*. For externally exposed subjects, uncertainties in organ doses may arise because the radiation source and/or the position, shielding, or exposure times of the subjects are not well established.

Random errors in the individual dose estimates for the atomic bomb survivor population have been estimated at 25-45% (Jablon, 1971; Pierce et al., 1990; Pierce and Vaeth, 1991). These random errors are likely to result in an overestimate of the average dose in the high dose groups and,

assuming a linear dose response function, a slight underestimate of the dose response (Pierce et al., 1990; Pierce and Vaeth, 1991). More significantly, perhaps, the shape of the dose response will be distorted towards a convex (downward) curvature; hence, a true linear-quadratic dependence may be distorted to look linear (Pierce and Vaeth, 1991).

Measurements of neutron activation products in Hiroshima indicate that neutron doses for Hiroshima survivors may have been underestimated and that the relative magnitude of the error increased with distance from the epicenter (Straume et al., 1992). If neutron doses have been underestimated, then a larger fraction of the radiogenic cancers would be attributable to neutrons, and the estimate of risk from gamma rays should be reduced. Using the tentatively revised estimates of neutron flux derived by Straume and coworkers, Preston et al. (1993) have calculated that the estimated risk from gamma rays for all cancers other than leukemia could be as much as 25% too high, with the calculated overestimate depending on the neutron RBE assumed.

The NCRP Committee identified three additional sources of uncertainty relating to the current dosimetry for the Japanese atomic bomb survivors: (1) bias in gamma ray estimates; (2) uncertainty in the characterization of radiation shielding by buildings; and (3) uncertainty in neutron RBE (NCRP, 1997). Altogether, the dosimetric uncertainties were judged to result in roughly a 15% overestimate of risk model coefficients for combined cancers other than leukemia. This may understate the dosimetric uncertainty for some specific cancer sites.

Uncertainties in the shape of the dose-response curve

The epidemiological studies underlying current radiation risk models generally involve subjects who experienced high radiation doses delivered in a relatively short time. A major issue in radiation risk assessment is how best to extrapolate the results of these epidemiological studies to low doses and/or low dose rates and to quantify the associated uncertainties. A comprehensive examination of this issue was contained in NCRP Report 64 (NCRP, 1980). Primarily on the basis of laboratory studies of cells, plants and animals, the report advocated a linear-quadratic dose response for acute doses up to about 2.5-4 Gy, above which the dose response begins to turn over due to cell killing. At low doses, the quadratic term is negligible compared with the linear term.

A theoretical framework for the linear-quadratic dose response model has been developed by Kellerer and Rossi (1972). In this theory of "dual radiation action", events leading to "lesions" or permanent changes in cellular DNA require the formation of interacting pairs of "sublesions". The interacting pairs can be produced by a single track (traversing particle) or by two tracks, giving rise, respectively, to a linear and a quadratic term in the dose response relationship. According to

the theory, a sublesion may be repaired before it can interact to form a lesion, with the probability of such repair increasing with time. As the dose rate is reduced, the formation of lesions from sublesions caused by separate tracks becomes less important, and the magnitude of the D^2 term decreases. The theory predicts that at sufficiently low doses or dose rates, the response should be linear and, in either limit, should have the same slope.

Results of animal tumorigenesis studies generally are qualitatively consistent with the dual action theory, in that low-LET radiation seems to have reduced effectiveness per unit dose at low dose rates (NCRP, 1980). However, it is usually not possible from the data to verify that the dose response curve has the linear-quadratic form.

Another success of the dual action theory has been in explaining observed differences between the effects of low- and high-LET radiations. In this view, the densely ionizing nature of the latter results in a much greater production of interacting pairs of sublesions by single tracks, leading to a higher biological effectiveness at low doses and a linear dose response relationship (except for deviations at high doses attributable to cell-killing effects).

The dual action theory has nevertheless been challenged on experimental grounds, and observed variations in response with dose, dose rate, and LET can also be explained by other mechanisms, e.g., a theory involving only single lesions and a "saturable" repair mechanism that decreases in effectiveness at high dose rates on the microscopic scale (Goodhead, 1982). One property of such a theory is that, in principle, the effectiveness of repair - and therefore the shape of the dose response curve - can vary widely with cell type, organ system, and species. Hence, results obtained on laboratory animals might not be entirely applicable to humans.

According to either the dual action theory or the saturable repair theory, the dose response should be linear at low doses or low dose rates, and with equal slopes. At higher doses and dose rates, multiple track events become important, and the dose response should bend upward. As a result, the response per unit dose at low doses and dose rates will be overestimated if one extrapolates linearly from observations made at high, acutely delivered doses (NCRP, 1980).

A linear dose response below about 0.2 Gy is consistent with an assumption of maximal DNA repair in that dose range. Repair of radiation-induced DNA damage has been found to be largely complete within a few hours of an acute exposure (Wheeler and Wierowski, 1983; Ullrich et al., 1987). This suggests that maximal repair persists at higher doses, provided the dose received within any time span of a few hours does not exceed 0.2 Gy. Further protraction should have little or no effect on the risk of cancer induction. Thus, the current mechanistic explanations suggest that the DDREF is constant at any dose below about 0.2 Gy and for higher doses received at a low dose

rate. EPA (1994) adopted the recommendation of UNSCEAR (1993) that an hourly averaged dose rate less than 0.1 mGy min^{-1} may be regarded as low in this context.

Until recently, it appeared that the LSS data could not be explained by a linear-quadratic model, because there were inconsistencies for solid tumors or leukemia and also inconsistencies between models developed separately for Hiroshima and Nagasaki. With the revised "DS86" dosimetry, however, these inconsistencies were largely removed (Shimizu et al., 1990; NAS, 1990). The data from the two cities are now in reasonable agreement. The combined leukemia data can be fit by a linear-quadratic dose response function; the slope of the function at low doses is about half that obtained by a linear fit to the data. A statistical analysis of the solid tumor data, on the other hand, is consistent with a linear dose response from low doses up to about 400 rad. Using a linear-quadratic model to fit the data reduces the linear term by, at most, a factor of 2 compared to a simple linear model. Viewing these results through the model used in NCRP 64 (1980) would indicate that: a best estimate of the DDREF is about 2 for leukemia while, for solid tumors, a DDREF of 2 represents *an upper bound*, and the best estimate is about 1. Errors in dose estimation may introduce a negative bias in the dose-squared dependence of the response; this has a relatively minor effect on the best estimate of the DDREF but could increase the upper bound to about 3 or 4. When compared with observed lung cancer risks in the atomic bomb survivors, results of clinical studies suggest that the DDREF may be quite large for lung cancer induction, although the possibility of confounding by the underlying disease process cannot be ruled out.

The results on human solid tumors appear to differ from those obtained through laboratory studies, including studies of radiation-induced tumorigenesis in mice and rats. Most laboratory studies suggest a DDREF of about 2 or 3, and sometimes higher, depending on the end point.

Taken together, current scientific data are generally indicative of a DDREF between 1 and 3 for human cancer induction, except for a possibly higher value for lung. The authors of the EPA report (EPA, 1994) concluded that a value of 2.0 provides a reasonable central estimate. The Agency's Radiation Advisory Committee agreed "that this choice is reasonable and ... consistent with current scientific judgment" (Loehr and Nygaard, 1992). A DDREF of 2 has recently been adopted by the ICRP (1991), as well as by other organizations (NCRP, 1993; CIRRPC, 1992), and is expected to be widely applied for purposes of risk assessment and radiation protection worldwide. The DDREF is applied to all organ-specific risks except for the breast, for which there is epidemiological evidence of a lack of effect of dose fractionation.

Uncertainties in the RBE for alpha particles

Radiobiological data indicate that high-LET alpha radiation has a larger biological effect than an equal absorbed dose of low-LET radiation. However, ranges of estimated values for alpha particle RBE are wide, depending on both the biological system and the observed end-point. The uncertainty in the RBE estimate from an individual study is also usually large, primarily due to the uncertainty in extrapolation of low-LET data to low doses. At relatively high doses, the effectiveness of alpha emitters has been found to be 15 to 50 times that of beta emitters for the induction of bone sarcomas, liver chromosome aberrations, and lung cancers (NCRP, 1990). Since the LET of secondary protons produced by fission neutrons in living tissue is comparable to that for alpha particles, data on the RBE of fission neutrons provides ancillary information relevant to the estimation of alpha particle RBE. Where the dose response data on carcinogenic end-points are adequate to derive an estimate, fission neutrons have been found to have an RBE between 6 and 60 times that of low dose gamma rays (NCRP, 1990).

The data are generally suggestive of a linear nonthreshold dose response for high-LET radiation, except for a possible fall-off in effectiveness at high doses. Under some conditions the effects of high-LET radiation appear to increase with fractionation or with a decrease in dose rate.

Site specific cancer risk estimates for high-LET radiation (neutrons or alpha particles) are often calculated utilizing human epidemiological data on low-LET radiation (e.g., from the LSS) and laboratory data on the relative biological effectiveness (RBE) of the high-LET radiation compared to a reference low-LET radiation (NCRP, 1990). Since the dose response relationship obtained for low-LET radiation is typically linear or concave upward while that for high-LET radiation is linear or concave downward, the RBE is dose dependent. The present report is concerned with risks at relatively low doses and dose rates, where the acute high dose risk for low-LET radiation is reduced by the DDREF. Under these conditions, the dose responses for both low and high LET radiations are thought to be linear, and the RBE takes on a constant (maximum) value: RBE_M.

With the exception of radiation-induced breast cancer and leukemia, the authors of the EPA report (EPA, 1994) followed the ICRP's recommendation (ICRP(1991) and assumed that the RBE for alpha particles is 20, in comparison to low-LET radiation at low doses and dose rates. Where the comparison is made against acute high doses of low-LET radiation, however, a value of 10 is assumed for the alpha particle RBE. Thus the low-LET radiation DDREF of 2 used for these cancers is implicitly incorporated into the RBE value for alpha radiation.

For breast cancer induction, a DDREF of 1 has been adopted. Therefore, the RBE will be independent of dose and dose rate. Since there is no DDREF correction of the low-LET breast

cancer risk estimates at low doses and dose rates, it is assumed that the acute high dose RBE of 10 is also applicable to breast cancer at low doses and dose rates.

There is evidence that alpha particle leukemia risks estimated on the basis of an RBE of 20 are too high (EPA, 1991). For this reason, an alpha particle leukemia risk estimate of 5.0×10^{-3} Gy^{-1} is employed, consistejt with the available high-LET epidemiological data (NAS, 1988; EPA, 1991). Quantitatively, this would correspond to an RBE of 1 for this site (relative to low dose, low-LET radiation). This should not be interpreted as implying that alpha radiation is no more carcinogenic than low-LET radiation in inducing leukemia. At least in part, the lower than expected leukemia risk produced by alpha emitters may result from a nonuniform distribution of dose within the bone marrow. That is, average doses to sensitive target cells of bone marrow may be substantially lower than calculated average marrow doses, to an extent that may vary from one alpha-emitting radionuclide to another. The RBE of 1 for alpha particles is regarded as an "effective RBE" that reflects factors other than just the relative biological sensitivity to high- and low-LET radiations.

Uncertainties in transporting risk estimates across populations

Baseline rates for specific cancer types vary from population to population and also vary over time within a population. For example, stomach cancer rates are substantially higher in Japan than in the U.S., while the reverse is true for lung, colon, and breast cancer. Moreover, the morbidity rates for lung and breast cancer, particularly, have been increasing in both populations during recent years. Despite the observed rough proportionality between radiation risk and baseline cancer rates by age, it should not be inferred that the radiation risk will vary in proportion to the baseline rate as one goes from one population to another.

Information on how to transport risk estimates across populations is limited by the quality of data available on irradiated populations other than the atomic bomb survivors. Two cancer types for which comparative data exist are thyroid and breast. Data on the thyroid suggest that the risk increases with the baseline rate, but it would appear that the opposite may be true for the breast. Some insight into the problem might be gained by looking at subgroups of an irradiated population. For example, lung cancer rates in Japanese males are several times higher than in Japanese females, presumably due in part to the higher smoking rate in males. Nevertheless, the excess absolute risk for lung cancer attributable to radiation does not differ significantly between the male and female bomb survivors. This suggests that, for lung cancer, absolute risk may be more transportable than relative risk.

Land and Sinclair (1991) present two relative risk models, differing in the method of transporting risk estimates from the LSS population to other populations. Both models assume a constant excess relative risk coefficient beginning 10 y after an exposure and continuing throughout the rest of life for each cancer site, excluding leukemia. One model (multiplicative) assumes that the relative risk coefficient is the same across populations. The other (NIH, for National Institutes of Health) assumes that the relative risk model coefficients for the target population should yield the same risks as those calculated with the additive risk model coefficients from the original population over the period of epidemiological follow-up, excluding the minimal latency period. These excess relative risk model coefficients are then used to project the risk over the remaining years of life. Projections made for the U.S. using the NIH model are much less sensitive to differences in site specific baseline rates between Japan and the U.S. than are those using the multiplicative model.

Data on North American women irradiated for medical purposes indicate about the same risk of radiogenic breast cancer per unit dose as the LSS data, despite the substantially higher breast cancer rates found in the U.S. or Canada, compared to Japan. For breast cancer, therefore, the NIH model projection agrees with observation better than the multiplicative model projection. Comparative data on other radiation-induced cancers are generally lacking or are too weak to draw any conclusions regarding the transportation of risk estimates from the LSS population to the U.S.

Both transportation models have a degree of biological plausibility. For example, the multiplicative model is consistent with the hypothesis that radiation acts as an "initiator" while the factors responsible for differences in baseline rates act as "promoters" of cancer. Alternatively, if both radiation and these factors act independently but at the same stage in the carcinogenesis process, their effects should be additive and radiation risks should be similar between populations despite differences in baseline rates. It seems likely that the actual situation is more complex than either of these alternatives and that some mixture of multiplicative and additive effects of radiation and non-radiogenic carcinogens may be involved.

Given the uncertainty in the transportation of risk across populations, the EPA recommends the use of geometric means of the age- and site-specific risk model coefficients derived from the multiplicative and NIH models of Land and Sinclair (EPA, 1994). The use of a geometric mean coefficient tends to de-emphasize extreme values that may reflect large extrapolations based on a few excess cancers observed among those exposed as children.

Uncertainties in age and time dependence of risk per unit dose

Information on the variation of risk of site-specific radiogenic cancers among the atomic bomb survivors with age and time is limited by sampling uncertainties and by the incomplete period of epidemiological follow-up. For a given age at time of the bomb, the excess solid tumor mortality has generally been found to increase with the age at death, roughly in proportion to the age-specific baseline rate for the site of interest. Consequently, models for most tumor sites are now generally framed in terms of relative risk.

For the period of epidemiological follow-up, the highest relative risks are found in the youngest exposure categories. However, the lifetime risks of solid tumors due to exposures before age 20 remain highly uncertain. Individuals exposed as children are only now entering the years of life where the risk of cancer is concentrated, and the observed excess effects represent a small number of cancer deaths. Hence, the sampling error for most types of cancers is large for the younger age cohorts. Moreover, it is not known whether observed high relative risks will persist. Theoretical considerations, arising from carcinogenesis modeling, suggest that the relative risks may decrease over time. Recent epidemiological evidence indicates such a temporal fall-off in groups irradiated as children (UNSCEAR 1988, Little et al. 1991).

Uncertainties in site-specific cancer morbidity risk estimates

The cancer lethality fractions given in Table 7.5 reflect only cancers appearing in adults. Even for adults, the selection of these values relied in part on subjective judgment, because there is no completely reliable way to determine long-term survival based on current (or future) treatment modalities. Moreover, lethality fractions derived for adults may not always be appropriate for children.

It appears that leukemia is now often curable in children. However, most radiogenic leukemias in the atomic bomb survivors occurred before successful treatment became available. Hence, the leukemia mortality risks derived from the Japanese may more properly reflect morbidity than mortality for children.

Computation of radionuclide risk coefficients

The calculations of radiogenic risk in this report account for the possibility that an exposed person who may have eventually died from, or developed, a radiogenic cancer will die at an earlier

age from a competing cause of death. It is assumed that the survival function is not significantly affected by the exposures being assessed, that is, that the number of radiogenic cancer deaths at any age is small compared with the number of deaths at that age from competing causes. Therefore, the risk coefficients tabulated in this document should not be applied to exposure levels that are sufficiently high to cause a substantial increase in the mortality rate at any age.

The age-specific cancer risk attributable to a unit intake of a radionuclide is calculated from the absorbed dose rate due to a unit intake of the radionuclide and the age-specific risk per unit dose model coefficients. The calculation is specific for each cancer and associated absorbed dose site in the risk model. The complete calculation may involve the sum of contributions from more than one target tissue and from both low-and high-LET absorbed doses.

The age-specific *lifetime risk coefficient* (*LRC*), $r(x)$, is the risk per unit absorbed dose of a subsequent cancer death (Gy^{-1}) due to radiation received at age x. In the EPA report on radiation risk models (EPA, 1994), $r(x)$ is referred to as an *attributable lifetime risk (ALR) coefficient*, but the terminology has been changed for use in this report because the term *attributable risk* is defined differently by different authors.

For an absolute risk model, the *LRC* for a given contribution is

$$r(x) = \frac{\int_x^\infty \alpha(x)\, \zeta(z-x)\, S(z)\, dz}{S(x)} \tag{7.4}$$

where α is the risk model coefficient in Eq. 7.1, ζ defines the plateau period (Eq. 7.1), and S is the survival function, that is, the fraction of live-born individuals in an unexposed population expected to survive to a given age. $S(0) = 1$, and S decreases monotonically for increasing values of x. $S(x)$ is obtained by a spline fit to decennial life table values to provide a continuous function of x.

Similarly, for a relative risk model,

$$r(x) = \frac{\int_x^\infty \eta(z,x)\, \mu(z)\, S(z)\, dz}{S(x)} \tag{7.5}$$

where $\eta(z,x)$ is the relative risk at age z due to a dose received at age x and $\mu(z)$ is the baseline force of mortality at age z for the given cancer type.

Following a unit intake of a radionuclide at age x_i, the absorbed dose rate $\dot{D}(x)$ to a given target tissue varies continuously with age $x \geq x_i$. The cancer risk $r_a(x_i)$ resulting from a unit intake of a radionuclide at age x_i is calculated from the continuously varying absorbed dose rate $\dot{D}(x)$ as follows:

$$r_a(x_i) = \frac{\int_{x_i}^{\infty} \dot{D}(x)\, r(x)\, S(x)\, dx}{S(x_i)} \tag{7.6}$$

where $r(x)$ is the cancer risk due to a unit absorbed dose (Gy^{-1}) at the site at age x. The absorbed dose rate is the absorbed dose rate for low-LET radiation, plus the product of the high-LET absorbed dose rate and the RBE applicable to the cancer type.

Age-specific male and female risk coefficients are combined by calculating a weighted mean:

$$r_a(x_i) = \frac{1.05\, r_{ma}(x_i)\, u_m(x_i)\, S_m(x_i) + r_{fa}(x_i)\, u_f(x_i)\, S_f(x_i)}{1.05\, S_m(x_i)\, u_m(x_i) + S_f(x_i)\, u_f(x_i)} \tag{7.7}$$

where

$r_a(x_i)$ is the combined cancer risk coefficient for a unit intake of activity at age x_i,

1.05 is the presumed sex ratio at birth (male-to-female),

$r_{ma}(x_i)$ is the male risk per unit activity at age x_i,

$r_{fa}(x_i)$ is the female risk per unit activity at age x_i,

$S_m(x_i)$ is the male survival function at age x_i,

$S_f(x_i)$ is the female survival function at age x_i, and

$u_m(x_i)$ and $u_f(x_i)$ are the usage rates (see Chapter 3) of the contaminated medium for males and females, respectively.

This formulation weights each sex-specific risk coefficient by the proportion of that sex in a stationary combined population at the desired age of intake.

The average lifetime risk coefficient for a radionuclide intake presumes that the intake rate is proportional to a constant environmental concentration (e.g., the radionuclide concentration in air). However, usage (e.g., the breathing rate) is also age and gender specific and therefore must be included in the averaging process. Defining the average lifetime risk as the quotient of the expected

lifetime risk and the expected lifetime intake from exposure to a constant environmental concentration yields

$$\overline{r_a} = \frac{\int_0^\infty u(x)\, r_a(x)\, S(x)\, dx}{\int_0^\infty u(x)\, S(x)\, dx} \quad . \tag{7.8}$$

The radionuclide concentration in the environmental medium does not appear in the expression because it is a common factor in both the numerator and denominator.

The above description applies to a stationary population that is subject to fixed gender-specific survival functions and fixed cancer mortality rates. In such a population, the age distribution of a given gender is proportional to the survival function for that gender. The derived risk coefficients may be interpreted either as risk per unit exposure to a typical member of the population exposed throughout life to a constant concentration of a radionuclide in an environmental medium, or as average risk per unit exposure to members of the population due to acute exposure to that radionuclide in that environmental medium. As discussed in Appendix D, a similar analysis may be applied to the case of acute exposure of a population with an arbitrary age distribution, if it is assumed that the exposed population is subject to fixed gender-specific survival functions and fixed cancer mortality rates at all times after the exposure. In this case, the survival function $S(x)$ in Eq. 7.8 is replaced by a function $P(x)$ representing the age distribution of the population at the time of acute exposure.

Lifetime risks for external radionuclide exposures are calculated in a manner similar to that for radionuclide intakes. Since the external exposure is not considered to be age dependent, the calculation is simpler. Given the age-specific cancer risk per unit dose, $r(x)$, and the corresponding dose per unit exposure coefficient, d_e, the lifetime risk is simply

$$r_e(x) = d_e\, r(x) \tag{7.9}$$

for an external exposure at age x. Age-specific male and female risk coefficients are combined by calculating a weighted mean as in Eq. 7.7, but with the usage rates $u_m(x_i)$ and $u_f(x_i)$ removed from

that equation. For lifetime external exposure at a constant exposure rate, d_e, the average lifetime risk is

$$\overline{r_e} = \frac{\int_0^\infty r_e(x)\, S(x)\, dx}{\int_0^\infty S(x) dx} \tag{7.10}$$

where $r_e(x)$ is given in Eq. 7.9 and $S(x)$ is the gender-weighted survival function. This equation applies to a specific cancer site. The total risk is the sum over all cancer sites.

APPENDIX A. MODELS FOR MORTALITY RATES FOR ALL CAUSES AND FOR SPECIFIC CANCERS

The life tables used in this report are based on data prepared by the National Center for Health Statistics for the U.S. Decennial Life Tables for 1989-91 (NCHS, 1997). The data are given in terms of $q(x)$, the probability of death in the age interval beginning at age x (NCHS, 1997, Tables 2 and 3). For each gender, tabulations are for age intervals from 0-1, 1-7, 7-28, and 28-365 days, and from 0-1 through 109-110 y in one-year increments. For purposes of this report, these values of $q(x)$ were extended in one year intervals to ages 110 y and above using the same methods that had been used to calculate the values for ages 100 to 109 y (Bell et al., 1992). Briefly, it is assumed that for $x > 109$ y, $q(x)$ for males is the minimum of $1.05\,q(x\text{-}1)$ and 1.0, and $q(x)$ for females is the minimum of $1.06q(x\text{-}1)$ and the value $q(x)$ for males. The completed set of values of $q(x)$ were then used to calculated $S(x)$, the probability of survival to age x [that is, $S(x) = (1\text{-}q(x\text{-}1))S(x\text{-}1)$] and $\mathring{e}(x)$, the expected life time remaining at age x. Values of $S(x)$ and $\mathring{e}(x)$ for a combined population were calculated for a male-to-female live birth ratio of 1.050. The derived values of $S(x)$ and $\mathring{e}(x)$ are shown in Table A.1.

For consistency with the survival data, age- and gender-specific cancer mortality rates (force of mortality) were calculated using NCHS data for reported deaths during 1989-91 (NCHS, 1992, 1993a, 1993b). Because of the small numbers of deaths for specific cancer sites at some ages, reasonably smooth force of mortality curves cannot be obtained by simply fitting the death data in one-year intervals. The method used here combines the one-year interval death data, starting with the first age with at least one death, into intervals of one or more years that contain at least five deaths. Above age 95 y, the one-year intervals are combined into a single group ending at the last age with any reported deaths. Cumulative deaths, expressed as a fraction of the total number of deaths in the interval in a stationary population defined by the gender-specific survival functions, are calculated at the end of each age interval. A third-order hermite polynomial spline (Fritsch and Carlson, 1980) is then fitted to these values. The "force of mortality" associated with a given cancer site and age is calculated as the quotient of the first derivative (with respect to age) of the spline fit to the cumulative deaths and the value of the survival function at that age.

The force of mortality estimate at the maximum reported age is applied to subsequent ages, and a value of zero is applied to ages below the minimum reported age. Finally, the calculated force of mortality data are smoothed by convolution with a gaussian response function with a full-width-half-maximum value of 3 years. Although the reported death data are discrete values for one-year intervals, the derived forces of mortality are continuous functions of age.

Table A.1. Gender- and age-specific values for the survival function, $S(x)$, and the expected remaining lifetime, $\mathring{e}(x)$, used in this report.

Age (y)	$S(x)$			$\mathring{e}(x)$		
	Combined	Male	Female	Combined	Male	Female
0	1.0000	1.0000	1.0000	75.24	71.83	78.81
1	9.9064E-01	9.8961E-01	9.9173E-01	74.94	71.58	78.47
2	9.8992E-01	9.8884E-01	9.9106E-01	74.00	70.64	77.52
3	9.8944E-01	9.8830E-01	9.9064E-01	73.03	69.68	76.55
4	9.8908E-01	9.8789E-01	9.9033E-01	72.06	68.70	75.58
5	9.8878E-01	9.8754E-01	9.9008E-01	71.08	67.73	74.60
6	9.8851E-01	9.8723E-01	9.8984E-01	70.10	66.75	73.61
7	9.8826E-01	9.8696E-01	9.8963E-01	69.12	65.77	72.63
8	9.8804E-01	9.8670E-01	9.8944E-01	68.13	64.78	71.64
9	9.8784E-01	9.8647E-01	9.8928E-01	67.15	63.80	70.65
10	9.8766E-01	9.8628E-01	9.8912E-01	66.16	62.81	69.67
11	9.8750E-01	9.8611E-01	9.8897E-01	65.17	61.82	68.68
12	9.8735E-01	9.8594E-01	9.8882E-01	64.18	60.83	67.69
13	9.8713E-01	9.8570E-01	9.8864E-01	63.20	59.85	66.70
14	9.8682E-01	9.8528E-01	9.8843E-01	62.22	58.87	65.71
15	9.8636E-01	9.8465E-01	9.8815E-01	61.24	57.91	64.73
16	9.8574E-01	9.8377E-01	9.8780E-01	60.28	56.96	63.75
17	9.8498E-01	9.8267E-01	9.8740E-01	59.33	56.03	62.78
18	9.8410E-01	9.8140E-01	9.8694E-01	58.38	55.10	61.81
19	9.8315E-01	9.8000E-01	9.8646E-01	57.44	54.17	60.84
20	9.8217E-01	9.7855E-01	9.8597E-01	56.49	53.25	59.87
21	9.8114E-01	9.7703E-01	9.8545E-01	55.55	52.34	58.90
22	9.8008E-01	9.7546E-01	9.8492E-01	54.61	51.42	57.93
23	9.7897E-01	9.7383E-01	9.8437E-01	53.67	50.51	56.96
24	9.7785E-01	9.7218E-01	9.8381E-01	52.73	49.59	56.00
25	9.7671E-01	9.7050E-01	9.8324E-01	51.79	48.68	55.03
26	9.7556E-01	9.6881E-01	9.8266E-01	50.86	47.76	54.06
27	9.7440E-01	9.6710E-01	9.8207E-01	49.92	46.84	53.09
28	9.7321E-01	9.6536E-01	9.8146E-01	48.98	45.93	52.12
29	9.7197E-01	9.6356E-01	9.8081E-01	48.04	45.01	51.16
30	9.7067E-01	9.6167E-01	9.8013E-01	47.10	44.10	50.19
31	9.6930E-01	9.5970E-01	9.7939E-01	46.17	43.19	49.23
32	9.6786E-01	9.5763E-01	9.7861E-01	45.23	42.28	48.27
33	9.6636E-01	9.5549E-01	9.7778E-01	44.30	41.37	47.31
34	9.6479E-01	9.5325E-01	9.7690E-01	43.38	40.47	46.35
35	9.6314E-01	9.5092E-01	9.7597E-01	42.45	39.57	45.40
36	9.6140E-01	9.4847E-01	9.7498E-01	41.52	38.67	44.44
37	9.5958E-01	9.4591E-01	9.7394E-01	40.60	37.77	43.49
38	9.5767E-01	9.4324E-01	9.7282E-01	39.68	36.88	42.54
39	9.5567E-01	9.4048E-01	9.7162E-01	38.76	35.98	41.59
40	9.5358E-01	9.3762E-01	9.7034E-01	37.85	35.09	40.65
41	9.5140E-01	9.3467E-01	9.6896E-01	36.93	34.20	39.70
42	9.4910E-01	9.3160E-01	9.6748E-01	36.02	33.31	38.76
43	9.4668E-01	9.2840E-01	9.6587E-01	35.11	32.43	37.83
44	9.4409E-01	9.2501E-01	9.6413E-01	34.21	31.54	36.89
45	9.4132E-01	9.2140E-01	9.6223E-01	33.31	30.66	35.97
46	9.3831E-01	9.1752E-01	9.6013E-01	32.41	29.79	35.04
47	9.3502E-01	9.1333E-01	9.5780E-01	31.52	28.93	34.13
48	9.3145E-01	9.0880E-01	9.5524E-01	30.64	28.07	33.22
49	9.2758E-01	9.0392E-01	9.5242E-01	29.77	27.22	32.31
50	9.2339E-01	8.9868E-01	9.4933E-01	28.90	26.37	31.42
51	9.1884E-01	8.9301E-01	9.4595E-01	28.04	25.54	30.53
52	9.1387E-01	8.8686E-01	9.4223E-01	27.19	24.71	29.65
53	9.0844E-01	8.8017E-01	9.3814E-01	26.35	23.89	28.77
54	9.0253E-01	8.7288E-01	9.3367E-01	25.52	23.09	27.91
55	8.9610E-01	8.6494E-01	9.2882E-01	24.70	22.30	27.05
56	8.8913E-01	8.5634E-01	9.2356E-01	23.89	21.52	26.20
57	8.8157E-01	8.4701E-01	9.1785E-01	23.09	20.75	25.36
58	8.7334E-01	8.3687E-01	9.1164E-01	22.30	19.99	24.53
59	8.6437E-01	8.2583E-01	9.0485E-01	21.53	19.25	23.71
60	8.5460E-01	8.1381E-01	8.9744E-01	20.77	18.53	22.90

Table A.1, continued

Age (y)	$S(x)$			$\mathring{e}(x)$		
	Combined	Male	Female	Combined	Male	Female
61	8.4405E-01	8.0086E-01	8.8940E-01	20.02	17.82	22.11
62	8.3274E-01	7.8701E-01	8.8076E-01	19.29	17.13	21.32
63	8.2064E-01	7.7224E-01	8.7147E-01	18.57	16.44	20.54
64	8.0770E-01	7.5649E-01	8.6148E-01	17.86	15.78	19.77
65	7.9390E-01	7.3974E-01	8.5076E-01	17.16	15.12	19.02
66	7.7922E-01	7.2201E-01	8.3930E-01	16.47	14.48	18.27
67	7.6370E-01	7.0334E-01	8.2708E-01	15.79	13.85	17.53
68	7.4729E-01	6.8369E-01	8.1406E-01	15.13	13.23	16.80
69	7.2989E-01	6.6298E-01	8.0014E-01	14.48	12.63	16.09
70	7.1140E-01	6.4109E-01	7.8522E-01	13.84	12.05	15.38
71	6.9173E-01	6.1797E-01	7.6919E-01	13.22	11.48	14.69
72	6.7085E-01	5.9359E-01	7.5197E-01	12.62	10.93	14.02
73	6.4873E-01	5.6801E-01	7.3349E-01	12.03	10.40	13.36
74	6.2547E-01	5.4137E-01	7.1377E-01	11.46	9.89	12.71
75	6.0118E-01	5.1387E-01	6.9286E-01	10.90	9.39	12.08
76	5.7598E-01	4.8565E-01	6.7082E-01	10.36	8.90	11.46
77	5.4988E-01	4.5679E-01	6.4764E-01	9.82	8.44	10.85
78	5.2292E-01	4.2742E-01	6.2321E-01	9.31	7.98	10.26
79	4.9505E-01	3.9763E-01	5.9734E-01	8.80	7.54	9.68
80	4.6622E-01	3.6750E-01	5.6987E-01	8.31	7.12	9.12
81	4.3643E-01	3.3706E-01	5.4077E-01	7.85	6.72	8.59
82	4.0583E-01	3.0647E-01	5.1017E-01	7.40	6.34	8.07
83	3.7468E-01	2.7609E-01	4.7821E-01	6.97	5.98	7.58
84	3.4339E-01	2.4650E-01	4.4512E-01	6.56	5.64	7.10
85	3.1230E-01	2.1816E-01	4.1115E-01	6.17	5.30	6.65
86	2.8153E-01	1.9116E-01	3.7643E-01	5.79	4.98	6.22
87	2.5117E-01	1.6550E-01	3.4113E-01	5.43	4.68	5.81
88	2.2156E-01	1.4140E-01	3.0573E-01	5.09	4.39	5.42
89	1.9307E-01	1.1910E-01	2.7074E-01	4.76	4.12	5.06
90	1.6604E-01	9.8784E-02	2.3666E-01	4.46	3.87	4.72
91	1.4063E-01	8.0549E-02	2.0372E-01	4.17	3.63	4.40
92	1.1706E-01	6.4441E-02	1.7231E-01	3.92	3.42	4.11
93	9.5685E-02	5.0524E-02	1.4310E-01	3.68	3.23	3.85
94	7.6820E-02	3.8824E-02	1.1672E-01	3.47	3.06	3.61
95	6.0582E-02	2.9266E-02	9.3463E-02	3.26	2.90	3.39
96	4.6893E-02	2.1656E-02	7.3392E-02	3.08	2.74	3.18
97	3.5553E-02	1.5693E-02	5.6407E-02	2.90	2.60	2.99
98	2.6410E-02	1.1151E-02	4.2432E-02	2.74	2.47	2.81
99	1.9215E-02	7.7620E-03	3.1241E-02	2.59	2.34	2.65
100	1.3686E-02	5.2851E-03	2.2507E-02	2.44	2.22	2.49
101	9.5253E-03	3.5144E-03	1.5837E-02	2.30	2.10	2.34
102	6.4653E-03	2.2780E-03	1.0862E-02	2.16	1.99	2.20
103	4.2700E-03	1.4365E-03	7.2452E-03	2.03	1.88	2.06
104	2.7372E-03	8.7932E-04	4.6879E-03	1.90	1.77	1.93
105	1.6982E-03	5.2121E-04	2.9340E-03	1.78	1.67	1.80
106	1.0164E-03	2.9833E-04	1.7705E-03	1.66	1.57	1.68
107	5.8477E-04	1.6438E-04	1.0262E-03	1.55	1.47	1.56
108	3.2202E-04	8.6882E-05	5.6892E-04	1.44	1.38	1.45
109	1.6891E-04	4.3873E-05	3.0020E-04	1.34	1.29	1.35
110	8.3912E-05	2.1069E-05	1.4990E-04	1.24	1.21	1.24
111	3.9216E-05	9.5701E-06	7.0343E-05	1.14	1.12	1.15
112	1.7103E-05	4.0859E-06	3.0771E-05	1.05	1.05	1.05
113	6.8928E-06	1.6274E-06	1.2422E-05	0.97	0.97	0.97
114	2.5380E-06	5.9920E-07	4.5737E-06	0.89	0.89	0.89
115	8.5432E-07	2.0170E-07	1.5396E-06	0.82	0.82	0.82
116	2.5924E-07	6.1205E-08	4.6717E-07	0.75	0.75	0.75
117	6.9636E-08	1.6441E-08	1.2549E-07	0.68	0.68	0.68
118	1.6159E-08	3.8150E-09	2.9120E-08	0.62	0.62	0.62
119	3.1292E-09	7.3878E-10	5.6391E-09	0.55	0.55	0.55
120	4.7981E-10	1.1328E-10	8.6466E-10	0.49	0.49	0.49

APPENDIX B. ADDITIONAL DETAILS OF THE DOSIMETRIC MODELS

Definitions of special source and target regions

The source region *Body Tissues* (formerly called *Whole Body*) consists of the entire body, minus the contents of the gastrointestinal (GI) tract, the urinary bladder, the gall bladder, and the heart. Thus, *Body Tissues* consists essentially of the "living tissues" of the body. The source region *Blood* is assumed to be uniformly distributed in *Body Tissues*.

The source region *Soft Tissues* represents *Body Tissues* minus cortical and trabecular bone. This source region is used to describe the distribution of some radionuclides that are distributed throughout the soft tissues of the body but have little deposition in mineral bone.

The target region historically referred to as *Bone Surface* represents radiosensitive endosteal tissue that actually is neither bone in its composition nor is it a surface. This target region is defined as the volume of soft tissue within 10 μm of the endosteal surface of bone. The target region *Bone Surface* should not be confused with the source regions *Cortical Bone Surface* and *Trabecular Bone Surface*, which refer to radioactivity assumed to be associated with infinitely thin surfaces of cortical and trabecular bone, respectively.

Within mineral bone, activity may be distributed within the volume of cortical or trabecular bone as well as on the surfaces of mineral bone. The four source regions *Cortical Bone Surface*, *Cortical Bone Volume*, *Trabecular Bone Surface*, and *Trabecular Bone Volume* are not used as target regions, because mineral bone is not radiosensitive.

Following long-term usage in radiation dosimetry, the source or target region *Red Marrow* is identified with the hematopoietically active marrow. The percentage of active marrow cells (cellularity) within a volume of marrow varies from site to site in the skeleton. The age-specific distribution of marrow within the body and relative cellularity at different sites have been taken into account in the dosimetry.

For a given biokinetic model, the source region *Other* consists of *Body Tissues*, minus the explicit source organs identified in the biokinetic model. The contribution of radiations emitted in *Other* to the energy deposition in a target region T is derived by assuming that the radioactivity is distributed uniformly by mass in *Other*.

Only source regions that are regarded as "volume sources" (that is, that have non-zero volume) may be considered as part of *Other*. Because the source regions *Cortical Bone Surface* and *Trabecular Bone Surface* are considered as infinitely thin surfaces of bone, they are not volume sources and hence cannot be part of *Other*. However, *Cortical Bone Volume* and *Trabecular Bone*

Volume are volume sources and may be part of *Other*. If no source regions in the volume of mineral bone or on its surfaces are explicitly identified in the biokinetic model, then *Other* includes radioactivity uniformly distributed by mass in *Cortical Bone Volume* and *Trabecular Bone Volume*. If any source region in the volume or on the surfaces of mineral bone is explicitly identified in the biokinetic model, then *Other* does not include any activity in mineral bone, that is, neither *Cortical Bone Volume* nor *Trabecular Bone Volume*. The entire mineral bone (*Cortical Bone Volume* plus *Trabecular Bone Volume*) is either included in *Other* or the entire mineral bone is excluded. It is never separated. *Red Marrow* will always be part of *Other* unless it is explicitly identified as a source region in the biokinetic model.

The esophagus is a radiosensitive tissue but has not yet been incorporated explicitly into the mathematical phantom used for internal dosimetric calculations. At present, the dose calculated for the target region *Thymus* is applied to the esophagus.

Age-dependent masses of source and target regions

Age-specific masses of source and target regions are given in Table B.1. With the exception of *Urinary Bladder Contents*, values for children are taken from the phantom series of Cristy and Eckerman (1987), and those for the adult male are taken from the Reference Man document (ICRP, 1975). Masses of *Urinary Bladder Contents* are based on data assembled for the revision of Reference Man and are intended to represent average contents of the bladder (Cristy and Eckerman, 1993).

For the adult female, regional masses are mostly reference values from ICRP Publication 23 but, where none are given, are scaled from those for the reference adult male. Masses for the target region *Bone Surface* or for source regions within mineral bone of the adult female are taken as 75% of the values for males. For *Urinary Bladder Contents* and *Urinary Bladder Wall*, values for the 15-y-old male are applied to the adult female.

Absorbed fractions for radiosensitive tissues in bone

For electrons, the radiation is usually assumed to be absorbed entirely in the source region. Exceptions are made for alpha and beta-emitters when the source and target regions are parts of the skeleton. The absorbed fractions in Table B.2 are taken from ICRP Publication 30, Part 1 (1979), and are applied to all ages.

Table B.1. Age-specific masses (g) of source and target organs.

Organ	Newborn	1 y	5 y	10 y	15 y	Adult female[a]	Adult male[a]
Adrenals	5.83	3.52	5.27	7.22	10.5	14.0	14.0
Brain	35.2	88.4	1260	1360	1410	1200	1400
Breasts	0.107	0.732	1.51	2.60	360	360	a
Gallbladder Contents	2.12	4.81	19.7	38.5	49.0	50.0	62.0
Gallbladder Wall	0.408	0.910	3.73	7.28	9.27	8.00	10.0
Lower Large Intestine Contents	6.98	18.3	36.6	61.7	109	135	135
Lower Large Intestine Wall	7.96	20.6	41.4	70.0	127	160	160
Small Intestine Contents	20.3	53.1	106	179	322	375	400
Small Intestine Wall	32.6	84.9	169	286	516	600	640
Stomach Contents	10.6	36.2	75.1	133	195	230	250
Stomach Wall	6.41	21.8	49.1	85.1	118	140	150
Upper Large Intestine Contents	11.2	28.7	57.9	97.5	176	210	220
Upper Large Intestine Wall	10.5	27.8	55.2	93.4	168	200	210
Heart Contents	36.5	72.7	134	219	347	410	500
Heart Wall	25.4	50.6	92.8	151	241	240	330
Kidneys	22.9	62.9	116	173	248	275	310
Liver	121	292	584	887	1400	1400	1800
Muscle	760	2500	5000	11,000	22,000	17,000	28,000
Ovaries	0.328	0.741	1.73	3.13	11.0	11.0	a
Pancreas	2.80	10.3	23.6	30.0	64.9	85.0	100
Red Marrow	47.0	150	320	610	1050	1300	1500
Cortical Bone Volume	0.0	299	875	1580	3220	3000	4000
Trabecular Bone Volume	14.0	20.0	219	396	806	750	1000
Bone Surface	15.0	26.0	37.0	68.0	120	90.0	120
Skin	118	271	538	888	2150	1790	2600
Spleen	9.11	25.5	48.3	77.4	123	150	180
Testes	0.843	1.21	1.63	1.89	15.5	0.0	35.0
Thymus	11.3	22.9	29.6	31.4	28.4	20.0	20.0
Thyroid	1.29	1.78	3.45	7.93	12.4	17.0	20.0
Urinary Bladder Contents	10.4	26.0	67.6	78.0	88.4	88.4	120

Table B.1, continued

Organ	Newborn	1 y	5 y	10 y	15 y	Adult female[a]	Adult male[a]
Urinary Bladder Wall	2.88	7.70	14.5	23.2	35.9	35.9	45.0
Uterus	3.85	1.45	2.70	4.16	80.0	80.0	80.0
Body Tissues	3535.7	9543.1	19,458	32,620	55,825	56,912	68,831
Extrathoracic 1 - Basal Cells	0.00173	0.00413	0.00828	0.0126	0.0185	0.0170	0.0200
Extrathoracic 2 - Basal Cells	0.0389	0.0930	0.186	0.284	0.416	0.390	0.450
Lymph Nodes - Extrathoracic	0.701	2.05	4.11	6.78	11.7	12.3	15.0
Bronchial - Basal Cells	0.0938	0.155	0.234	0.311	0.408	0.390	0.432
Bronchial - Secretory Cells	0.187	0.310	0.469	0.622	0.816	0.780	0.864
Bronchiolar - Secretory Cells	0.385	0.596	0.946	1.30	1.76	1.90	1.94
Alveolar-Interstitial	51.4	151	301	497	859	904	1100
Lymph Nodes - Thoracic	0.701	2.05	4.11	6.78	11.7	12.3	15.0

[a]In this report, dosimetric calculations are not performed separately for adult males and females but are based on a reference adult formed by adding the breasts, ovaries, and uterus of the adult female phantom to the adult male phantom.

Table B.2. Absorbed fractions for alpha- and beta-emitters in bone (ICRP, 1979, 1980).

Source Region	Target Region	α-emitter	β-emitter, average energy < 0.2 MeV	β-emitter, average energy ≥ 0.2 MeV
Cortical Bone Surface	Red Marrow	0.0	0.0	0.0
Cortical Bone Volume	Red Marrow	0.0	0.0	0.0
Trabecular Bone Surface	Red Marrow	0.5	0.5	0.5
Trabecular Bone Volume	Red Marrow	0.05	0.35	0.35
Cortical Bone Surface	Bone Surface	0.25	0.25	0.015
Cortical Bone Volume	Bone Surface	0.01	0.015	0.015
Trabecular Bone Surface	Bone Surface	0.25	0.25	0.025
Trabecular Bone Volume	Bone Surface	0.025	0.025	0.025
Red Marrow	Red Marrow	1	1	1
Red Marrow	Bone Surface	(fraction endosteal tissue associated with Red Marrow) • (mass of endosteal tissue) ÷ (mass of Red Marrow)[a]		

[a]This equation corresponds to the assumption that the specific absorbed fraction in endosteal tissue is the same as that in Red Marrow itself. The fraction of endosteal tissue in whole skeleton associated with Red Marrow is assumed to be 1.0, 0.83, 0.65, 0.65, 0.65, and 0.5 for ages newborn, 1-y, 5-y, 10-y, 15-y, and adult, respectively. Adult value is from ICRP Publication 30, and other values are from Cristy and Eckerman (1987).

APPENDIX C. AN ILLUSTRATION OF THE MODELS AND METHODS USED TO CALCULATE RISK COEFFICIENTS FOR INTERNAL EXPOSURE

This appendix provides an example to illustrate the models and computational steps involved in the derivation of a risk coefficient for ingestion or inhalation of a radionuclide. A secondary purpose is to illustrate some differences between the updated models applied here and the older models still commonly used by regulatory agencies, particularly the models of ICRP Publication 30 (1979, 1980, 1981, 1988).

The radionuclide chosen is ^{232}Th. This radionuclide was selected because it represents nearly all of the different types of changes that have been made recently in the ICRP's biokinetic and dosimetric models. For example, age-dependent f_1 values have been introduced for thorium and the f_1 value for the adult has been changed (ICRP, 1995a); a new, age-specific systemic biokinetic model has been adopted for thorium (ICRP, 1995a); the treatment of ingrowing radioactive progeny of ^{232}Th and other thorium isotopes has been revised (ICRP, 1995a); and a new model of the biokinetics of inhaled radionuclides, including ^{232}Th, has been adopted (ICRP, 1994a).

To keep the analysis to a reasonable length, the discussion focuses on estimating the risk, per unit intake of ^{232}Th, of dying from a single cancer type. Leukemia is considered because of the relatively high degree of sophistication and detail provided in the risk model for this type of cancer. Because radiogenic leukemia is assumed to arise from irradiation of the bone marrow, discussion of the dosimetric models focuses on this tissue.

Gastrointestinal tract model and f_1 values

The ICRP's model for transit of material through the gastrointestinal tract is described in Chapter 4. This model has not been changed since its appearance in ICRP Publication 30 (1979). However, applications of the model have changed in recent ICRP publications in the following ways: the model is now applied to all age groups; some of the ICRP's updated systemic biokinetic models depict secretion of activity from the systemic tissues and fluids into compartments of the gastrointestinal tract model; new f_1 values have been adopted for several elements, for application to environmental intakes by the adult; and age-specific f_1 values have been adopted for several elements, for application to environmental intakes.

In ICRP Publication 69 (1995a), an f_1 value of 5×10^{-4} is recommended for calculation of doses from ingestion of environmental thorium by persons of age ≥1 y. This f_1 value, which is 2.5

times the value recommended in ICRP Publication 30 (1979) for consideration of occupational exposures to thorium, is based on experimental data on gastrointestinal absorption of thorium, neptunium, plutonium, americium, and curium in human subjects. On the basis of experimental results indicating that gastrointestinal absorption of actinide elements typically is several times higher in newborn than adult animals, an f_1 value of 5×10^{-3} is assigned to infants (ICRP, 1995a).

Respiratory tract model

The ICRP's new respiratory tract model is described in Chapter 4. The present discussion focuses on predictions of the model for three hypothetical forms (absorption types) of inhaled thorium, including the distribution of thorium in the respiratory tract, its absorption to blood, and its movement from the respiratory tract to excreta, as a function of time after inhalation.

Although the respiratory tract model was designed to allow consideration of compound-specific kinetics, parameter values have been developed for only a few general situations. In current applications of the model, a given compound of an element usually is assigned to one of three default absorption types: Type F, representing fast dissolution and a high level of absorption to blood; Type M, representing an intermediate rate of dissolution and an intermediate level of absorption to blood; and Type S, representing slow dissolution and a low level of absorption to blood. Ideally, the user bases an absorption type on data on the form of material expected to be encountered. In practice, the form of the inhaled material often cannot be characterized with much confidence.

Predictions of the fate of inhaled ^{232}Th of Type F, M, or S based on the ICRP's new respiratory tract model are shown on the left side of Fig. C.1. The assumed particle size is 1 μm (AMAD). Because it is assumed in the model that the behavior of material in the respiratory tract depends only on particle size and absorption type, the predictions apply to all long-lived radionuclides whose gastrointestinal absorption is negligible compared with the indicated levels of absorption from the respiratory tract to blood. For short-lived radionuclides, the curves for the *extrathoracic* (*ET*), *alveolar-interstitial* (*AI*), *bronchial* (*BB*), and *bronchiole* (*bb*) regions may decline faster and those for *Gastrointestinal* (*GI*) *excretion*, *Nasal excretion*, and *Absorption* may have lower maximum values than the curves shown in Fig. C.1 due to radioactive decay in the respiratory tract. Here, *GI excretion* represents the cumulative activity transferred from the respiratory tract to the GI Tract, and *Nasal excretion* refers to removal of material from the *ET* region directly to the environment by such mechanisms as nose blowing.

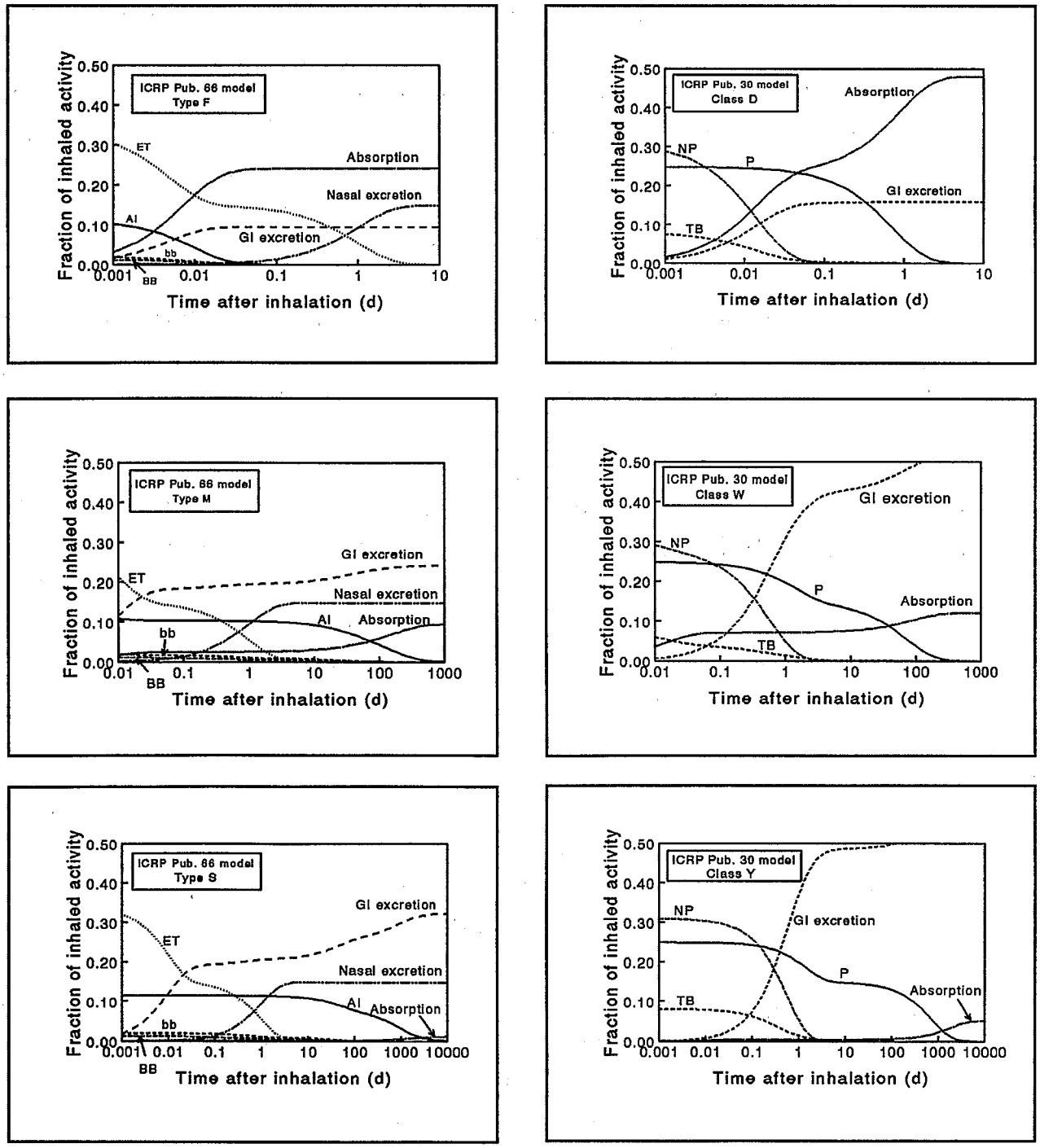

Fig. C.1. Predictions of the ICRP's updated (ICRP, 1994a) and previous (ICRP, 1979) respiratory tract models, for inhalation of ^{232}Th in soluble, moderately soluble, or insoluble 1-μm particles (AMAD).

The three absorption types, F, M, and S, correspond roughly to the three lung clearance classes D (days), W (weeks), and Y (years) used in the ICRP's previous respiratory tract model (ICRP, 1979). Predictions of the previous model for inhaled ^{232}Th of particle size 1 μm and clearance classes D, W, and Y are shown on the right side of Fig. C.1 for comparison with predictions of the new model. Although there is not an exact correspondence between the different regions of the two models, the *nasal-pharyngeal* (*NP*) region may be compared with the *ET* region, the *tracheobronchial* (*TB*) region with the *bronchi* (*BB*) plus *bronchioles* (*bb*), and the *pulmonary* (*P*) region with the *alveolar-interstitial* (*AI*) region of the new model. Compared with the new model, the previous model predicts higher total deposition in the respiratory tract, greater deposition in the lower lungs, faster removal from the extrathoracic regions, and greater absorption to blood.

Biokinetics of absorbed thorium

Structure of the systemic biokinetic model for thorium

A new biokinetic model for thorium was introduced in ICRP Publication 69 (ICRP, 1995a). The model is developed within a generic model framework adopted by the ICRP for application to a class of "bone-surface-seeking" radionuclides (Fig. C.2). To this point, the generic model framework has been applied by the ICRP to thorium, plutonium, americium, curium, and neptunium.

While the model structure is generic, many of the transfer coefficients are not. Some transfer coefficients associated with compartments within the skeleton are expressed in terms of bone remodeling rates and thus are independent of the bone-surface seeker, but element-specific transfer coefficients are required for most of the paths shown in Fig. C.2.

The generic model structure divides systemic tissues and fluids into six main parts: *BLOOD*, *SKELETON*, *LIVER*, *KIDNEYS*, *GONADS*, and *OTHER SOFT TISSUES*. *BLOOD* and *GONADS* are treated as uniformly mixed pools, but each of the other major parts is further divided into a minimal number of compartments needed to explain available biokinetic data on thorium and chemically similar elements.

SKELETON is divided into cortical and trabecular fractions, and each of these is subdivided into fractions associated with bone surface, bone volume, and bone marrow. Activity entering *SKELETON* initially deposits in compartments of bone surface but is transferred gradually to bone marrow by bone resorption or to compartments of bone volume by bone formation. Activity in bone volume compartments is transferred gradually to bone marrow compartments by resorption. Activity

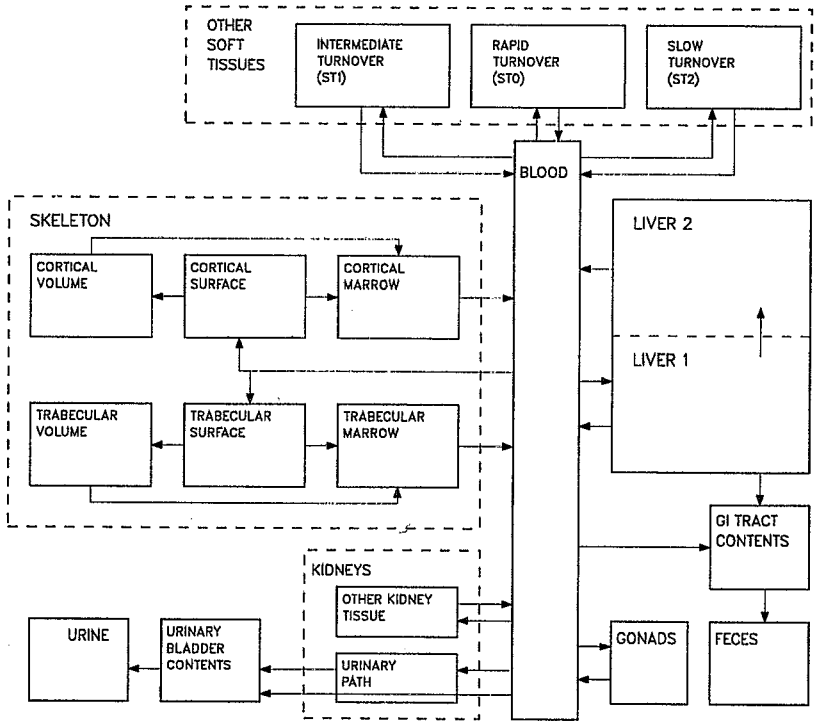

Fig. C.2. The ICRP's generic framework for modeling the systemic biokinetics of a class of bone-surface-seeking elements, including thorium.

moves from bone marrow compartments to *BLOOD* over a few months and is subsequently redistributed in the same pattern as the original input to blood.

LIVER is viewed as consisting of two compartments, called *LIVER 1* and *LIVER 2*. *LIVER 1* represents relatively short-term retention and *LIVER 2* represents relatively long-term retention in the liver. Activity entering the liver is assigned to *LIVER 1*. Activity removed from *LIVER 1* by biological processes is divided among blood, *LIVER 2*, and the contents of the GI tract. Activity leaving *LIVER 2* is assigned to blood.

KIDNEYS consists of two compartments, one that loses activity to urine and another that returns activity to blood. *URINARY BLADDER CONTENTS* is considered as a separate pool that receives all material destined for urinary excretion.

Compartment *ST0* is a soft-tissue pool that includes the extracellular fluids and exchanges material with blood over a period of hours or days. Soft-tissue compartments *ST1* and *ST2* represent intermediate-term retention (up to a few years) and tenacious retention (many years), respectively, in the massive soft tissues (for example, muscle, skin, and subcutaneous fat).

Parameter values for the systemic model for thorium

Movement of material in the body is depicted as a system of first-order processes. Parameter values are expressed as transfer coefficients (fractional transfer per day) between compartments. Age-specific transfer coefficients for thorium are listed in Table C.1 for the six ages considered in the ICRP series on age-dependent dosimetry (ICRP, 1989, 1993, 1995a, 1995b, 1996). Rates for intermediate ages are obtained by interpolating linearly with age between the listed values. For example, a given transfer coefficient for age 4 y is calculated as 0.25 times the rate given for age 1 y plus 0.75 times the rate given for age 5 y. For consideration of the biokinetics of thorium, the age of the mature adult is assumed to be ≥25 y.

Transfer coefficients for the adult were based largely on experimental, occupational, and environmental data on the behavior of thorium in humans, but it was necessary to use data on laboratory animals (mainly beagles) to fill gaps in the human data. For example, the model was required to be consistent with data on early retention, excretion, and blood clearance of thorium in healthy, elderly human subjects who received radiothorium by intravenous injection (Maletskos et al., 1966, 1969). However, the early distribution of thorium in the body was based mainly on experimental data on the early distribution of thorium in beagles (Stover et al. 1960), in the absence of such information for humans. Parameter values controlling predictions of the long-term distribution and retention of thorium were developed mainly on the basis of bioassay or autopsy measurements on occupationally or environmentally exposed humans (Rundo, 1964; Newton et al., 1981; Wrenn et al., 1981; Singh et al., 1983; Ibrahim et al., 1983; Dang et al., 1992), together with consideration of bone restructuring rates in humans (ICRP, 1995c).

Due to the paucity of age-specific data on the biokinetics of thorium, default assumptions concerning the relative kinetics of bone seekers in children and adults were used in ICRP Publication 69 (1995a) to extend parameter values from adults to children. These assumptions are based on numerous observations of the age-specific biokinetics of various bone seekers in laboratory animals and, to a lesser extent, human subjects (Leggett, 1992a, 1992b; ICRP, 1993, 1995b). It is postulated that differences with age in the biokinetics of a radionuclide that accumulates mainly in the skeleton is dominated by three events: (1) increased fractional transfer from plasma to bone in children in

Table C.1. Age-specific transfer coefficients (d^{-1}) in the systemic biokinetic model for thorium (ICRP, 1995a).

	Age (y)					
Pathway[a]	Infant (100 d)	1 y	5 y	10 y	15 y	Adult
Blood to Liver 1	0.0647	0.0647	0.0647	0.0647	0.0647	0.0970
Blood to Cort Surf	0.7763	0.7763	0.7763	0.7763	0.7763	0.6793
Blood to Trab Surf	0.7763	0.7763	0.7763	0.7763	0.7763	0.6793
Blood to UBC	0.0711	0.0711	0.0711	0.0711	0.0711	0.1067
Blood to Urinary Path	0.0453	0.0453	0.0453	0.0453	0.0453	0.0679
Blood to OKT	0.0129	0.0129	0.0129	0.0129	0.0129	0.0194
Blood to LI Contents	0.00647	0.00647	0.00647	0.00647	0.00647	0.00970
Blood to Testes	0.000039	0.000058	0.000066	0.000077	0.00062	0.00068
Blood to Ovaries	0.000023	0.000030	0.000076	0.00013	0.00023	0.00021
Blood to ST0	0.832	0.832	0.832	0.832	0.832	0.832
Blood to ST1	0.162	0.162	0.162	0.162	0.162	0.243
Blood to ST2	0.0259	0.0259	0.0259	0.0259	0.0259	0.0388
ST0 to Blood	0.462	0.462	0.462	0.462	0.462	0.462
Urinary Path to UBC	0.0462	0.0462	0.0462	0.0462	0.0462	0.0462
OKT to Blood	0.00038	0.00038	0.00038	0.00038	0.00038	0.00038
ST1 to Blood	0.00095	0.00095	0.00095	0.00095	0.00095	0.00095
ST2 to Blood	0.000019	0.000019	0.000019	0.000019	0.000019	0.000019
Trab Surf to Trab Vol	0.00822	0.00288	0.00181	0.00132	0.000959	0.000247
Trab Surf to Bone Marrow	0.00822	0.00288	0.00181	0.00132	0.000959	0.000493
Cort Surf to Cort Vol	0.00822	0.00288	0.00153	0.000904	0.000521	0.0000411
Cort Surf to Bone Marrow	0.00822	0.00288	0.00153	0.000904	0.000521	0.0000821
Trab Vol to Bone Marrow	0.00822	0.00288	0.00181	0.00132	0.000959	0.000493
Cort Vol to Bone Marrow	0.00822	0.00288	0.00153	0.000904	0.000521	0.0000821
Bone Marrow to Blood	0.0076	0.0076	0.0076	0.0076	0.0076	0.0076
Liver 1 to Liver 2	0.00095	0.00095	0.00095	0.00095	0.00095	0.00095
Liver 1 to SI Contents	0.000475	0.000475	0.000475	0.000475	0.000475	0.000475
Liver 1 to Blood	0.000475	0.000475	0.000475	0.000475	0.000475	0.000475
Liver 2 to Blood	0.000211	0.000211	0.000211	0.000211	0.000211	0.000211
Testes/Ovaries to Blood	0.00019	0.00019	0.00019	0.00019	0.00019	0.00019

[a]Cort = Cortical, Trab = Trabecular, Surf = Surface, Vol = Volume, UBC = Urinary Bladder Contents, OKT = Other Kidney Tissue, LI = Large Intestine, SI = Small Intestine.

association with elevated bone formation rates in the maturing skeleton; (2) decreased fractional transfer from plasma to soft tissues and excreta in children due to relatively greater competition from immature bone; and (3) an elevated rate of transfer from bone to plasma in children due to an elevated rate of bone turnover. For actinide elements, the additional assumption is made that fractional deposition in the gonads at a given age depends on the mass of the gonads at that age. Except where there is evidence to the contrary, removal half-times from soft tissues, bone surfaces, and exchangeable bone volume are assumed to be independent of age.

In the model for thorium, the deposition fraction on all bone surfaces combined is set at 0.8 for ages ≤15 y compared with 0.7 for adults, and the deposition fractions in soft tissues and excretion pathways are reduced by one-third for application to ages ≤15 y to maintain mass balance. Of greater importance for dose estimates for thorium isotopes in children, however, is the generic assumption that the removal rate of thorium from bone surfaces, its rate of burial in bone volume, and its rate of removal from bone volume to blood (via bone marrow) are all directly related to the bone remodeling rate, which is estimated to be several-fold higher in children than in adults. For example, ICRP Publication 70 (1995c) gives reference values for the remodeling rate of trabecular bone of more than 100% y^{-1} for ages ≤1 y, 48% y^{-1} for age 10 y, and an average of 18% y^{-1} for ages ≥25 y.

Predicted differences with age in systemic biokinetics of thorium

Predicted differences with age in the biokinetics of thorium are illustrated in Fig. C.3, which shows the estimated retention of ^{232}Th on trabecular surfaces as a function of time after intravenous injection at each of three injection ages: infancy (100 d), age 10 y, and age 25 y. The model predicts that there is greater deposition on trabecular surfaces in children than adults but that the bone surface activity declines at a considerably higher rate in children than in adults due to elevated bone turnover rates in children. Part of the activity removed from bone surfaces is assumed to be buried in bone volume. The remainder is assumed to be removed to bone marrow and then to blood, after which a small fraction is excreted and the remainder is recycled to bone surfaces and soft tissues. Activity in bone volume is also assumed to be recycled in the same manner after its gradual release due to bone remodeling.

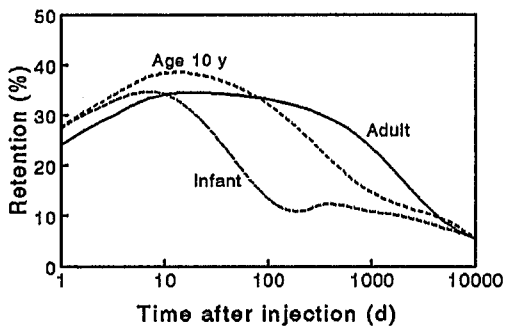

Fig. C.3. Retention of ^{232}Th on trabecular surfaces for three ages at injection, as predicted by the updated model for thorium (ICRP, 1995a).

Table C.2 gives model predictions of the 50-y integrated activity of ^{232}Th in different regions of the body after injection of a unit activity of ^{232}Th into blood at age 100 d (infant), 10 y, or 25 y.

The indicated differences with age at injection result from some combination of three assumptions: elevated uptake of thorium by immature bone, an elevated rate of remodeling of immature bone, and

Table C.2. Predictions of 50-y integrated activity of ^{232}Th (nuclear transformations per Bq injected), following injection into blood at age 100 d, 10 y, or 25 y.

Compartment	Age at injection		
	100 d	10 y	25 y
Trabecular surfaces	9.8×10^{7}	1.2×10^{8}	1.4×10^{8}
Cortical surfaces	2.8×10^{8}	4.3×10^{8}	6.3×10^{8}
Trabecular volume	8.8×10^{7}	9.3×10^{7}	6.4×10^{7}
Cortical volume	1.8×10^{8}	2.7×10^{8}	1.6×10^{8}
Red marrow	3.1×10^{7}	2.0×10^{7}	1.3×10^{7}
Liver	5.7×10^{7}	4.2×10^{7}	3.8×10^{7}
Kidneys	1.1×10^{7}	8.4×10^{6}	7.5×10^{6}
Testes	2.9×10^{5}	4.3×10^{5}	4.6×10^{5}
Ovaries	1.5×10^{5}	1.8×10^{5}	1.4×10^{5}

an age-independent removal half-time for soft tissues. For example, the time-integrated activity in red marrow decreases with age at injection, mainly as a result of rapid recycling of activity from trabecular bone to red marrow in children and an age-independent removal half-time from bone marrow. For gonads, the elevated feedback of activity from bone at younger ages is offset by relatively low deposition fractions for gonads at these ages, resulting in little change with age at injection in the cumulative activity of ^{232}Th.

Treatment of ^{232}Th chain members produced in systemic tissues

In ICRP Publication 30 (1979), decay chain members produced in the body after intake of a parent radionuclide generally were assigned the biokinetic model of the parent; this is the so-called assumption of "shared kinetics" of decay chain members. In a subsequent critical review of experimental data on the fate of radionuclides formed *in vivo*, it was suggested that the following assumption of "independent kinetics" of chain members may be more realistic than the assumption of shared kinetics in most cases (Leggett et al., 1985): (1) a radionuclide born in soft tissues or on bone surfaces behaves as if taken into the body as a parent radionuclide; (2) a radionuclide born in bone volume has the same kinetics as the parent until removed from bone volume and then behaves as if taken into the body as a parent radionuclide.

There is some experimental evidence to support the assumption of independent kinetics for thorium chains (Leggett et al., 1985). For example, activity ratios ^{224}Ra:^{228}Th in tissues and excreta of beagles injected with ^{228}Th are consistent with the assumption that ^{224}Ra born on bone surfaces migrated from ^{228}Th over a period of days and then behaved as if injected directly into blood (Van Dilla et al., 1956, 1957; Stover et al., 1965a, 1965b). Time-dependent activity ratios of subsequent members of the ^{228}Th chain also suggest redistribution consistent with the characteristic biokinetic models of individual members, although the extent of migration of these chain members and hence the interpretation of the data are limited by the short half-lives of the chain members (Stover et al., 1965a, 1965b).

The assumption of independent kinetics was applied in ICRP Publication 69 (1995a) to chain members produced *in vivo* after absorption of thorium isotopes to blood, except that some simplifying assumptions were made in cases where there was little difference, in effect, between the assumptions of shared and independent kinetics. Parameter values for individual chain members can be found in Appendix C of ICRP Publication 71 (ICRP, 1995b). The models for members of various thorium chains are summarized in the following:

1. Radium isotopes formed *in vivo* are assumed to follow the model for radium as a parent (Leggett, 1992a; ICRP, 1993). This requires that the model structure for thorium (Fig. C.2) be expanded to include compartments that are in the radium model (see Chapter 4, Fig. 4.6) but not in the thorium model. For example, each bone volume compartment in the thorium model must be divided into exchangeable and nonexchangeable bone volume compartments to describe the behavior of radium after its movement from plasma to bone surfaces to bone volume. According to the radium model, bone contains about 30%, soft tissues about 15%, and excreta plus excretion pathways (mainly intestinal contents) about 55% of the injected amount at 1 d after injection of long-lived radium into blood of an adult. Most radium atoms entering bone or soft tissues return to plasma within a few days. By 100 d after injection, bone retains less than 5% and soft tissues less than 1% of the injected amount, the rest having been lost in excreta.
2. Radon produced in soft tissues or bone surfaces is assumed to be removed to plasma at a fractional rate of 100 d^{-1}. Radon produced in the exchangeable and nonexchangeable bone volume compartments is assumed to migrate to plasma at rates of 1.5 and 0.36 d^{-1}, respectively. Radon entering plasma is assumed to be removed from the body by exhalation at a fractional rate of 1 min^{-1}.
3. Lead isotopes formed *in vivo* are assumed to follow the model for lead as a parent (Leggett, 1993; ICRP, 1993). Therefore, the model structure used to address a thorium chain that

includes lead as a daughter must include compartments such as red blood cells and exchangeable and nonexchangeable bone volume that are in the lead model (Fig. 4.6) but are not identified separately in the thorium model. According to the lead model, the approximate contents of various regions at 1 d after injection of long-lived lead into an adult are as follows: red blood cells, 59% (of the injected amount); bone, 15%; liver, 11%; kidneys, 5%; other soft tissues, 3%; and excreta plus excretion pathways, 7%. Over the next few weeks there is a gradual shift of lead from red blood cells to bone, soft tissues, and excreta. By 100 d after injection, the predicted contents of the regions are as follows: red blood cells, 4% (of the injected amount); bone, 22%; liver, 5%; kidneys, 2%; other soft tissues, 5%; and excreta, 62%.

4. The model for polonium as a decay chain member is based on the non-recycling model for polonium as a parent given in ICRP Publication 67 (1993), but the latter model is converted into a recycling model to fit into the framework used for thorium, radium, and lead. Removal of polonium from all tissues except bone volume is assumed to occur at a fractional rate of 0.1 d^{-1}, with activity going to plasma. Removal from bone volume to plasma is assumed to occur at the rate of bone turnover. Of polonium reaching plasma, 10% goes to the gastrointestinal tract contents and subsequently to feces and 5% goes to the urinary bladder contents and then to urine. The unexcreted amount is divided as follows: 30% to liver, 10% to kidneys, 5% to spleen, 10% to red marrow, and 45% to other tissues.
5. Bismuth is assumed to be removed from all tissues except bone volume at a fractional rate of 0.035 d^{-1}, with activity going to plasma. From plasma, 35% goes to urine, 7% to feces via the intestines, 35% to the kidneys, 5% to the liver, and 18% to other tissues.
6. Isotopes of thallium appearing in important thorium chains are short-lived and are assumed to decay at their point of origin, and isotopes of actinium, protoactinium, and thorium produced *in vivo* are assigned the model for thorium.

The treatment of decay chain members is a particularly important consideration in the internal dosimetry of ^{232}Th due to the fact that the radioactive progeny of ^{232}Th emit substantially more alpha energy than the parent over a period of a few years. The estimated alpha activity of the total chain is reduced substantially if it is assumed, as indicated by available experimental data, that ^{228}Ra and subsequent chain members migrate over a period of hours or days from sites of production on bone surfaces and in soft tissues and then behave as if injected directly into blood (Table C.3).

Comparison of updated and previous systemic models for thorium

The ICRP's new systemic biokinetic model for thorium differs substantially from its previous model (ICRP, 1979) with regard to basis, structure, and predictions. The previous model consists of three tissue compartments fed by a transfer compartment (Fig. C.4). On the basis of observations

Table C.3. Comparison of estimated 50-y integrated activities of ^{232}Th and its decay chain members, assuming (A) independent or (B) shared kinetics of decay chain members, for the case of injection of ^{232}Th into blood of an adult[a].

Radionuclide	Ratio of integrated activities, A:B						
	Cortical bone surface	Trabecular bone surface	Cortical bone volume	Trabecular bone volume	Red bone marrow	Liver	Testes, ovaries
^{232}Th	1.0	1.0	1.0	1.0	1.0	1.0	1.0
^{228}Ra, ^{228}Ac	0.001	0.003	0.9	0.7	0.08	0.04	0.05
^{228}Th	0.02	0.06	0.8	0.5	0.2	0.06	0.05
^{224}Ra through ^{208}Tl	~0.005	~0.02	~0.8	~0.5	~0.1	~0.05	~0.05

[a]The biokinetic model for thorium given in ICRP Publication 69 (ICRP, 1995a) is applied to ^{232}Th. For the case of independent kinetics, the models and assumptions of ICRP Publication 69 are applied to the radioactive progeny of ^{232}Th.

of the fate of ^{228}Th in beagles (Stover et al., 1960), it is assumed in that model that activity leaves the transfer compartment with a half-time of 0.5 d, with 70% depositing on bone surfaces, 4% depositing in the liver, 16% depositing in other soft tissues, and 10% lost in excreta. Thorium is assumed to be removed from bone surfaces to excretion with a biological half-time of 8000 d and from liver and other soft tissues to excretion with a biological half-time of 700 d. The assumption that skeletal deposits remain on bone surfaces until removed to excretion is generally applied in ICRP Publication 30 to actinide elements.

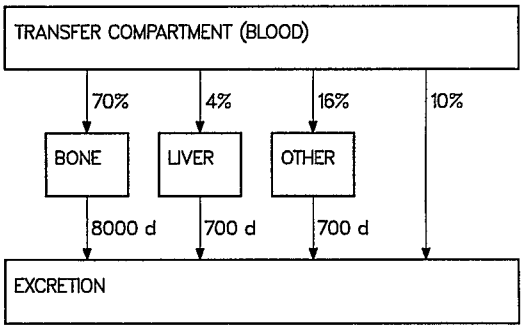

Fig. C.4. Biokinetic model for thorium given in ICRP Publication 30 (1979).

Compared with the model of ICRP Publication 30, the new model predicts considerably longer retention of thorium in the skeleton, liver, and other soft tissues, and consequently much longer retention in the total body of the adult. For example, the model of Publication 30 predicts that

about 75% of an amount injected into blood at time zero will be excreted in 10,000 days, compared with a prediction of about 30% based on the new model (Fig. C.5).

In the model of ICRP Publication 30, the time-dependent concentration of thorium in kidneys and gonads is assumed to be the same as that in all soft tissues other than liver. In the new model, the kidneys and gonads are addressed separately from other soft tissues and are depicted as relatively important repositories for thorium. This is demonstrated in Table C.4, where comparisons are made of the updated and previous models as predictors of the 50-y cumulative activity of ^{232}Th and ^{228}Ra in selected organs of an acutely exposed adult. Three types of acute intake are considered in this table: injection of ^{232}Th into blood, ingestion of ^{232}Th, and inhalation of a moderately soluble form of ^{232}Th (Type M or class W, respectively, in the updated and previous respiratory tract models). The assumption of independent kinetics of decay chain members is used in conjunction with the updated systemic model, and the assumption of shared kinetics is used with the systemic model of ICRP Publication 30.

Conversion of activity to estimates of dose rates to tissues

SE values

The dose rate to a target region *T* due to activity in a source region *S* depends on the amount of activity in *S*, the nature of the radiations emitted in the source region, the spatial relationships between the source and target regions, the nature of the tissues between the regions, and the mass of *T*. As discussed in Chapter 5, the details of these considerations are embodied in a coefficient called the specific energy or *SE*.

The ICRP's updated *SE* values for the adult male generally do not differ substantially from those applied to Reference Man in ICRP Publication 30 (1979), but there are notable exceptions. The most important exception is for the lung as a target region. In ICRP Publication 30, the dose to the lung is an average dose over the entire lung tissue. In ICRP Publication 66 (1994a), the dose to the lung is redefined as a weighted average of doses to sensitive cells of the bronchi, bronchioles, and alveolar-interstitium, with the relatively small mass of cells of the bronchi and bronchioles receiving greater weight that the relatively large mass of the alveolar-interstitium. The two definitions of lung dose can result in substantially different estimates, particularly for radionuclides that emit mainly non-penetrating radiations. This is because the new definition assigns much greater importance to the generally small fraction of the total activity in the lungs that is associated with the

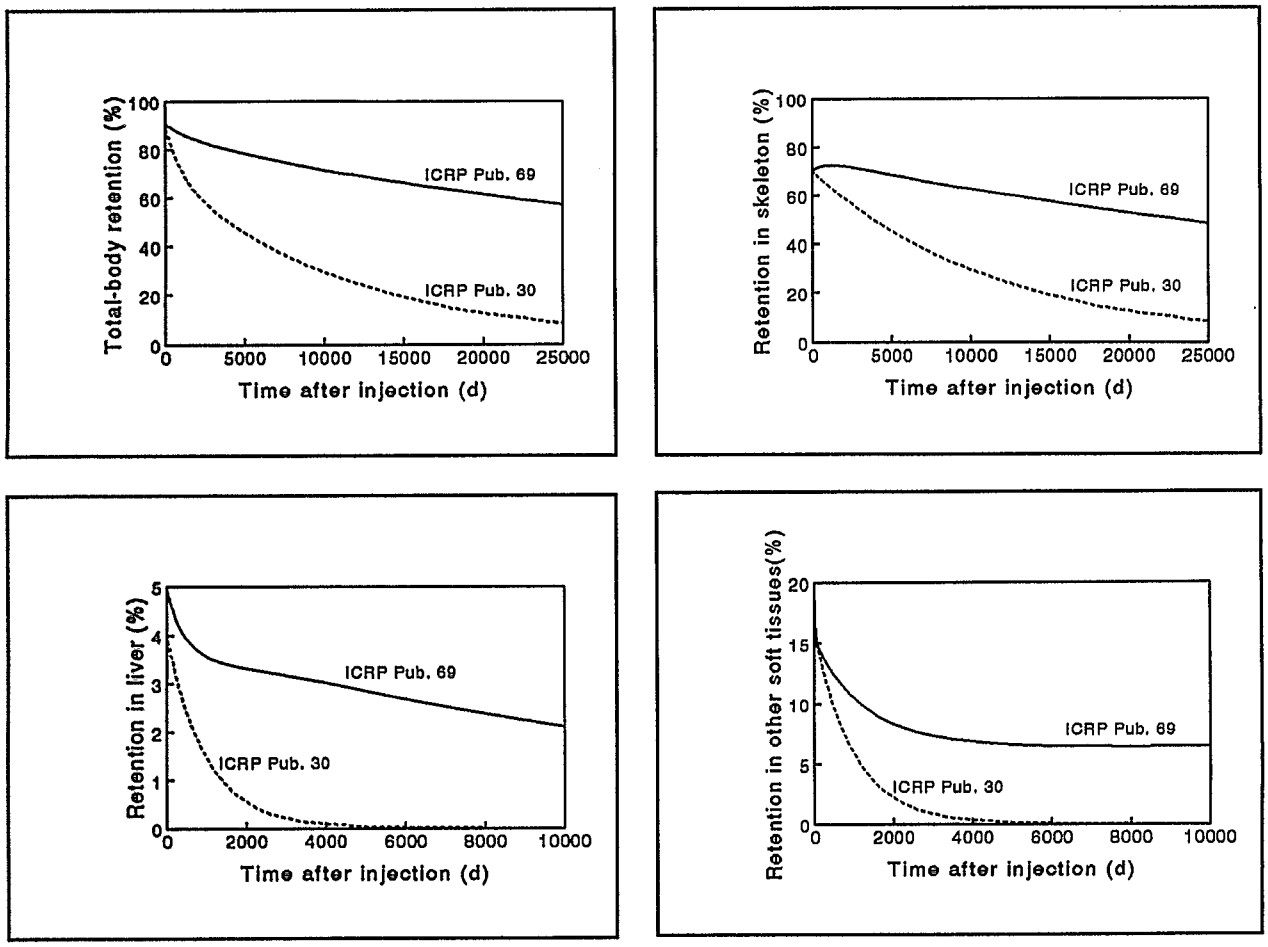

Fig. C.5. Comparison of predictions of ICRP's updated (ICRP, 1995a) and previous (ICRP, 1979) systemic biokinetic models for thorium.

radiosensitive cells of the bronchi and bronchioles. For example, for the case of acute inhalation of a moderately soluble form of ^{232}Th (Type M) by an adult, the estimated activity of all chain members in the alveolar-interstitium (region *AI*) at 50 d after inhalation is about a factor of 40 greater than that in the bronchioles (region *bb*, Fig. 4.1). Yet the estimated dose rate to bronchiolar secretory cells from high-LET radiation at that time is nearly twice as great as that to *AI* as a result of the small mass assigned to the bronchiolar secretory cells.

For purposes of calculating radiogenic risk to members of the public, an important advance in internal dosimetry in recent years has been the introduction of age-specific *SE* values. *SE* values for most pairs of source and target organs vary substantially with age due to changes with age in the

Table C.4. Comparison of ICRP's updated (ICRP, 1995a) and previous (ICRP, 1979) models as predictors of 50-y integrated activity after acute intake of ^{232}Th by an adult.

	Ratio of integrated activities (updated models : previous models)					
	Injection		Ingestion		Inhalation	
Compartment	^{232}Th	^{228}Ra[a]	^{232}Th	^{228}Ra[a]	^{232}Th	^{228}Ra[a]
Trabecular surfaces	0.49	0.0014	1.2	0.0036	0.39	0.0011
Cortical surfaces	2.3	0.0026	5.7	0.0064	1.8	0.0020
Liver	11	1.4	27	3.5	8.4	1.0
Kidneys	110	9.0	280	23	86	7.0
Gonads	60	8.9	150	22	47	7.0
Other systemic activity	25	56	62	140	19	42

[a]Refers to ^{228}Ra produced in the body after intake of ^{232}Th.

masses of target organs and, in some cases, in the relative geometries of the source and target organs during growth.

Changes with age in *SE* values for ^{232}Th are illustrated in Fig. C.6 for the red marrow as a target organ and for each of three source organs: *Trabecular Bone Surface* (*TS*), *Red Marrow* (*RM*), and *Trabecular Bone Volume* (*TV*). In Fig. C.6, *SE*(*T*,*S*) indicates the *SE* value for target organ *T* and source organ *S*. The indicated *SE* values are for high-LET (alpha) radiation, which is the dominant radiation type for ^{232}Th. The decrease with age in the *SE* values result from an increase with age in the mass of *Red Marrow* (Fig. C.7).

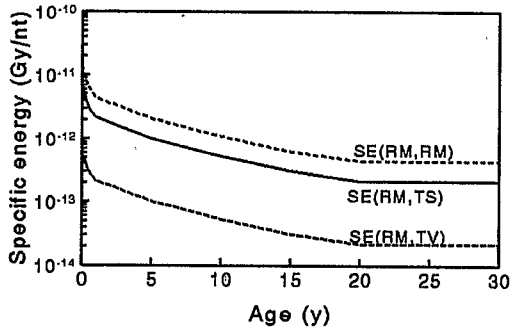

Fig. C.6. Age-specific *SE* values (high-LET) for ^{232}Th. *RM = Red Marrow, TS = Trabecular Bone Surface, TV = Trabecular Bone Volume.*

Use of *SE* values to calculate dose rates

The calculation of dose rates is illustrated for the case of high-LET (alpha) irradiation of *Red Marrow* from internally deposited ^{232}Th. Due to the short range of the alpha particles, the contributing source organs in this case are those in intimate contact with *Red Marrow*, namely, *Red Marrow*, *Trabecular Bone Surface*, and *Trabecular Bone Volume*.

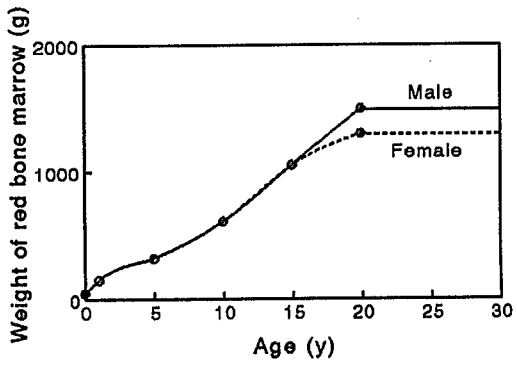

Fig. C.7. Estimated weight of red marrow as a function of age.

Recall that for a given type of radiation, the absorbed dose rate, $\dot{D}_T(t)$, at age t in target region T can be expressed as:

$$\dot{D}_T(t) = \sum_S \sum_j q_{S,j}(t)\ SE(T \leftarrow S;t)_j\ , \qquad \text{(C.1)}$$

where $q_{S,j}(t)$ is the activity of radionuclide j present in source region S at age t and $SE(T \leftarrow S;t)_j$ is the specific energy deposited in target region T per nuclear transformation of radionuclide j in source region S at age t. Therefore, the high-LET dose rate $\dot{D}_{RM}(t)$ to *Red Marrow* from ^{232}Th (excluding radioactive progeny) at age t due to intake of ^{232}Th at age t_0 is the sum

$$\dot{D}_{RM}(t) = q_{RM}(t)\ SE(RM \leftarrow RM;t) + q_{TS}(t)\ SE(RM \leftarrow TS;t) + q_{TV}(t)\ SE(RM \leftarrow TV;t), \qquad \text{(C.2)}$$

where the three *SE* values are as indicated (with abbreviated notation) in Fig. C.6. According to the biokinetic and dosimetric models used here, the right side of Eq. C.2 usually is dominated by the term involving *Trabecular Bone Surface* as a source organ (second term). Although all alpha particles emitted in *Red Marrow* are assumed to be absorbed by *Red Marrow*, the contribution to $\dot{D}_{RM}(t)$ from *Red Marrow* typically is much smaller than the contribution from *Trabecular Bone Surface* for the case of ^{232}Th because the predicted number of thorium atoms contained in *Red*

Marrow at a given time typically is much smaller than the number of thorium atoms in *Trabecular Bone Surface*. Although the predicted number of thorium atoms in *Trabecular Bone Volume* may be larger than that in *Trabecular Bone Surface* at some ages, the contribution to $\dot{D}_{RM}(t)$ from *Trabecular Bone Volume* (third term in Eq. C.2) typically is smaller than the contribution from *Trabecular Bone Surface* because *SE(RM←TV;t)* is much smaller than *SE(RM←TS;t)* (Fig. C.6). The relationship between the three terms on the right side of Eq. C.2 as a function of time after acute injection of ^{232}Th is illustrated in Fig. C.8 for the adult. It is emphasized that the curves in Fig. C.8 represent only the contribution of the parent, ^{232}Th, to the high-LET dose rate to *Red Marrow*. The total high-LET dose rate to *Red Marrow* will also include contributions from the radioactive progeny of ^{232}Th contained in the *Red Marrow*, *Trabecular Bone Surface*, and *Trabecular Bone Volume*.

Calculated high-LET dose rates to *Red Marrow* for the cases of acute ingestion and acute inhalation of 1 Bq of ^{232}Th are shown in Figs. C.9 and C.10, respectively, for three ages at intake: infancy (100 d), 10 y, and 25 y. The dose rates indicated in these figures include contributions from radioactive progeny of ^{232}Th as well as from the parent radionuclide. Due to migration of ^{228}Ra and subsequent chain members from the parent, however, ^{232}Th is the major contributor to the indicated dose rates.

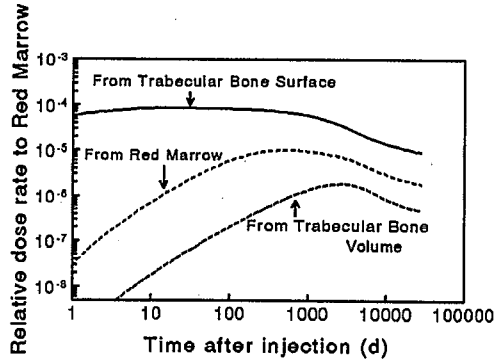

Fig. C.8. Contributions of ^{232}Th in *Trabecular Bone Surface*, *Trabecular Bone Volume*, and *Red Marrow* to the high-LET dose rate to *Red Marrow* in the adult.

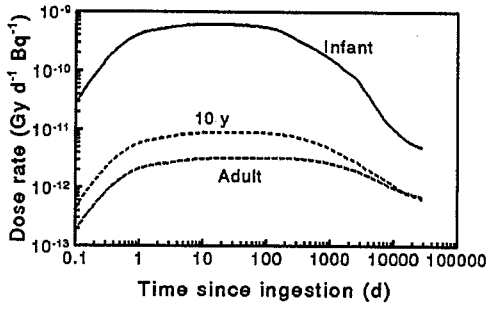

Fig. C.9. Estimated dose rates to *Red Marrow* following acute ingestion of ^{232}Th, for three ages at ingestion.

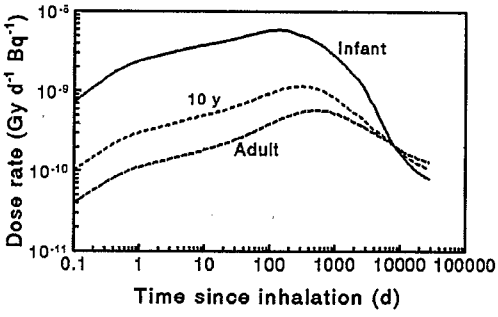

Fig. C.10. Estimated dose rates to *Red Marrow* following acute inhalation of moderately soluble ^{232}Th, for three ages at inhalation.

Conversion of dose rates to estimates of radiogenic cancers

The age-specific cancer risk attributable to a unit intake of a radionuclide is calculated from the absorbed dose rate due to a unit intake of the radionuclide and the age-specific risk per unit dose model coefficients. The calculation is specific for each cancer and associated absorbed dose site in the risk model. The complete calculation for each cancer and associated dose site may involve the sum of contributions from more than one target tissue and from both low-and high-LET absorbed doses.

In the following, attention is focused on the problem of estimating the risk of dying from radiogenic leukemia following intake of ^{232}Th. That is, the problem is one of deriving a mortality risk coefficient for leukemia for ingestion or inhalation of ^{232}Th. In this case, the target organ of interest is red marrow. The risk model used in this report for leukemia is a relative risk model, with age- and gender-specific risk model coefficients.

Recall that the age-specific *lifetime risk coefficient (LRC)*, $r(x)$, is the risk per unit dose of a subsequent cancer death (Gy^{-1}) due to radiation received at age x. For a relative risk model, the *LRC* for a given contribution is

$$r(x) = \frac{\int_x^\infty \eta(u,x)\, \mu(u)\, S(u)\, du}{S(x)} \qquad \text{(C.3)}$$

where $\eta(u,x)$ is the relative risk at age u due to a dose received at age x, $\mu(u)$ is the force of mortality at age u for the given cancer type, and $S(x)$ is the survival function. Because the *LRC* for a given cancer type is independent of the radionuclide and exposure scenario, the *LRC*s need not be recalculated in each derivation of a radionuclide risk coefficient but can be calculated once, stored, and used as input data in the calculation of all radionuclide risk coefficients.

The excess relative risk, $\eta(u,x)$, is the product of a risk model coefficient, $\beta(x)$, and a time-since-exposure response function, $\zeta(t,x)$, that defines the period during which the risk is expressed and (in the radiogenic risk model for leukemia) changes with time in the level of response during that period. The age- and gender-specific risk model coefficients $\beta(x)$ for leukemia are given in Table 7.2, and the time-since-exposure response function $\zeta(t,x)$ for leukemia is described in Eq. 7.3 and the text accompanying that equation.

Relative risk functions $\eta(u,x)$ for radiogenic leukemia in males are shown in Fig. C.11 for three ages at irradiation: infancy (100 d), 10 y, and 25 y. The functions for females are similar to those for males but are not identical because the risk model coefficients, $\beta(x)$, differ slightly for the two genders (Table 7.2).

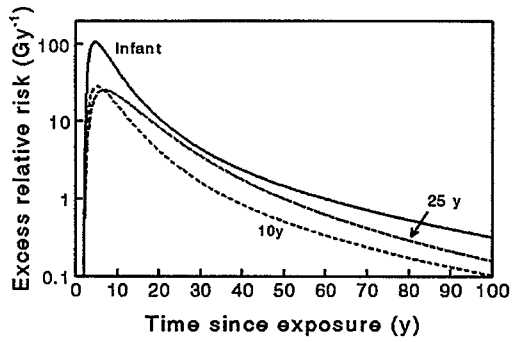

Fig. C.11. Relative risk functions, $\eta(u,x)$, for leukemia in males for three ages at irradiation.

The gender-specific force of mortality functions for leukemia are shown in Fig. C.12 (NCHS, 1992, 1993a, 1993b), and the gender-specific survival functions $S(x)$ (all causes of death) are shown in Fig. C.13 (NCHS, 1997). The *LRC* functions $r(x)$ for radiogenic leukemia in males and females, calculated by integrating the product of the functions $\eta(u,x)$, $\mu(u)$, and $S(x)$ from age x to infinity (age 120 y), are shown in Fig. C.14. The sharp changes in direction in the *LRC* functions at some ages stem mainly from jumps in the risk model coefficients $\beta(x)$ for leukemia at those ages (Table 7.2).

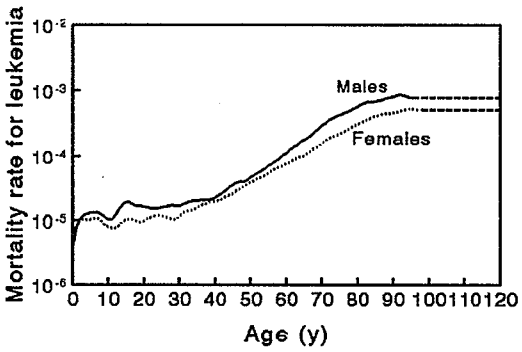

Fig. C.12. Age- and gender-specific mortality rates for leukemia, based on U.S. data for 1989-91 (NCHS, 1992, 1993a, 1993b).

The *LRC* function $r(x)$ is based on a unit dose received at age x. Following intake of a radionuclide at age x_i, the absorbed dose rate $\dot{D}(x)$ to a given target tissue varies continuously with age $x \geq x_i$. The cancer risk $r_a(x_i)$ resulting from a unit intake of a radionuclide at age x_i is calculated from the continuously varying absorbed dose rate $\dot{D}(x)$ using the equation:

$$r_a(x_i) = \frac{\int_{x_i}^{\infty} \dot{D}(x)\, r(x)\, S(x)\, dx}{S(x_i)} \qquad \text{(C.4)}$$

where $r(x)$ is the cancer risk due to a unit absorbed dose (Gy^{-1}) at the site at age x. The functions $S(x)$ and $r(x)$ in the integrand are shown in Figs. C.13 and C.14, respectively. The dose rate function $\dot{D}(x)$ in the integrand is illustrated in Fig. C.9 for ingestion of ^{232}Th and in Fig. C.10 for inhalation of moderately soluble ^{232}Th at age 100 d, 10 y, or 25 y.

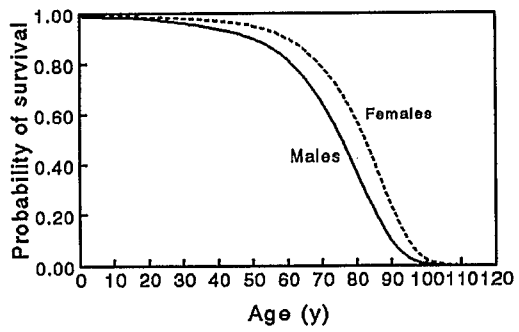

Fig. C.13. Gender-specific survival functions based on U.S. life tables for 1989-91 (NCHS, 1997).

Derived gender-specific risks $r_a(x_i)$ of dying from radiogenic leukemia due to acute ingestion of ^{232}Th are shown in Fig. C.15 for ingestion ages from birth through old age. Model predictions for the case of acute inhalation of ^{232}Th are shown in Fig. C.16. The derived values $r_a(x_i)$ for males and females are combined into a risk estimate for the total population of age x_i by calculating a weighted mean that accounts for the proportion of each sex in a stationary combined population at the desired age of intake (see Chapter 7, Eq. 7.7).

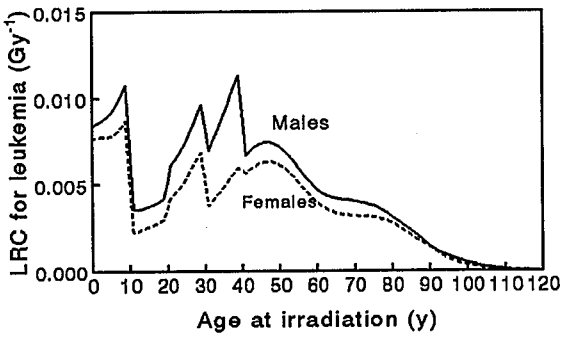

Fig. C.14. Gender-specific *lifetime risk coefficient* (*LRC*) functions for radiogenic leukemia.

For a given gender, the average lifetime leukemia risk coefficient for a ingestion or inhalation of ^{232}Th is calculated from the derived age- and gender-specific values, $r_a(x)$. Because $r_a(x)$ is based on acute intake of 1 Bq of ^{232}Th at age x, $r_a(x)$ must be scaled by (that is, multiplied by) the age-specific intake rate, $C\,u(x)$, where C is the constant radionuclide concentration in the environmental medium and $u(x)$ is the usage rate at age x as specified in the usage scenario. The product $u(x)\,r_a(x)$ must be further scaled by the value of the survival function at x, $S(x)$, to account for the possibility that the exposed person will die from a competing cause before reaching age x. Therefore, for a given gender, the estimated risk of dying from leukemia due to lifetime intake of ^{232}Th is the integral over age from birth to the maximum possible value of x (assumed here to be 120 y) of the product $C\,u(x)\,r_a(x)\,S(x)$. Because a risk coefficient is expressed as risk per unit intake, the integral of $C\,u(x)\,r_a(x)\,S(x)$ must be divided by

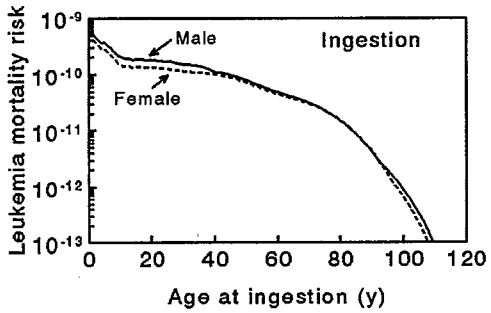

Fig. C.15. Derived gender-specific risk $r_a(x_i)$ of dying from leukemia due to ingestion of 1 Bq of ^{232}Th in food at age x_i.

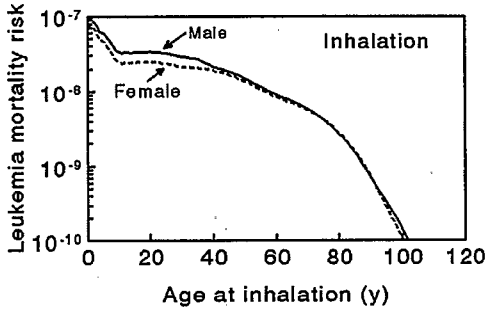

Fig. C.16. Derived gender-specific risk $r_a(x_i)$ of dying from leukemia due to inhalation of 1 Bq of ^{232}Th (Type M) at age x_i.

the probable lifetime intake of ^{232}Th. Because the probable intake rate at age x is $Cu(x)$ times the probability $S(x)$ of surviving to age x, the probable lifetime intake of ^{232}Th is the integral over age of the product $Cu(x)S(x)$. Therefore, for a given gender, the average lifetime leukemia risk coefficient for ingestion or inhalation of ^{232}Th is given by:

$$\overline{r_a} = \frac{\int_0^\infty u(x)\, r_a(x)\, S(x)\, dx}{\int_0^\infty u(x)\, S(x)\, dx} \quad . \tag{C.5}$$

The radionuclide concentration in the environmental medium, C, disappears from the equation because it is a factor in both numerator and denominator.

Gender-weighted average lifetime risk coefficients for ingestion of ^{232}Th are indicated in the bar graph in Fig. C.17, and risk coefficients for inhalation of a moderately soluble form of ^{232}Th are indicated in the bar graph in Fig. C.18. These two figures show the relative contributions of some cancer-specific risk coefficients, including that for leukemia, to the total combined risk coefficient for ingestion or inhalation of ^{232}Th. Shown for comparison are risk coefficients for ^{232}Th based on the risk methodology described in this report but using the biokinetic models and assumptions of ICRP Publication 30 (1979).

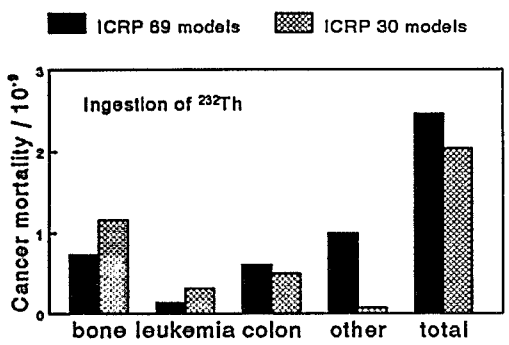

Fig. C.17. Gender-weighted average lifetime risk coefficients for ingestion of ^{232}Th in food, using updated (ICRP, 1995a) and previous (ICRP, 1979) biokinetic models for thorium.

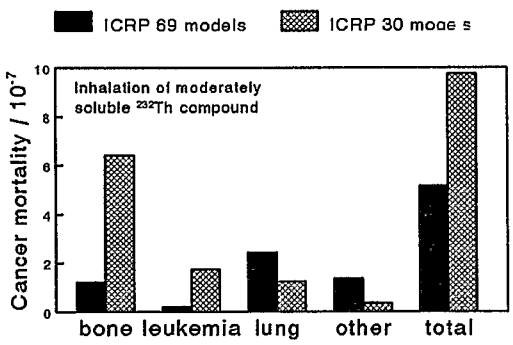

Fig. C.18. Gender-weighted average lifetime risk coefficients for inhalation of moderately soluble ^{232}Th, using updated (ICRP, 1995a) and previous (ICRP, 1979) biokinetic models for thorium.

Comparison with risk estimates based on effective dose

As a measure of the risk from intake of radionuclides, the ICRP uses a quantity called the effective dose. The effective dose is a weighted sum of equivalent doses (that is, integrated equivalent dose rates) to radiosensitive tissues, with tissue weighting factors representing the relative contribution of each tissue to the total detriment for the case of uniform irradiation of the whole body. The effective dose is based on an integration period of 50 years for intake by adults and to age 70 years for intake by children.

The ICRP relates the effective dose to the probability of a fatal cancer through a multiplicative factor called a "nominal fatality probability coefficient". This coefficient is referred to as "nominal" because of the uncertainties inherent in radiation risk estimates and because the ICRP's estimated relation of effective dose and fatal cancers is based on an idealized population receiving a uniform equivalent dose over the whole body. A nominal fatality probability coefficient of 0.05 Sv^{-1} is given in ICRP Publication 60 (1991) for all cancer types combined. According to ICRP Publication 60, "If the equivalent dose is fairly uniform over the whole body, it is possible to obtain the probability of fatal cancer associated with that effective dose from the nominal fatality probability coefficient. If the distribution of equivalent dose is non-uniform, this use of the nominal coefficient will be less accurate because the tissue weighting factors include allowances for non-fatal and hereditary conditions." Another difficulty with the effective dose as a measure of risk is that

it cannot accurately reflect the contribution of competing risks for the many different temporal patterns of dose rates to tissues that occur for various long-lived, tenaciously retained radionuclides.

Despite such limitations in the effective dose, it is common practice to use the nominal fatality probability coefficient to convert effective doses from internally deposited radionuclides to estimates of fatal radiogenic cancers. The effective dose is taken by some analysts as the effective dose equivalent of ICRP Publication 30 (1979, 1980, 1981, 1988) as tabulated in Federal Guidance Report No. 11 (1988), and is taken by others from tabulations in the ICRP's recent series of documents on doses to the public from intake of radionuclides (see summary report, ICRP Publication 72, 1996). Because the latter documents provide the effective dose as a function of age at acute intake, the effective dose may be represented by a weighted average of age-specific effective doses, where the weights reflect assumed levels of intake at different ages. Because such weighted effective doses typically differ by <30% from the effective dose for intake by the adult, the latter is generally applied.

Cancer mortality risk for ingestion of ^{232}Th in food and for inhalation of a moderately soluble form (Type M) of ^{232}Th of particle size 1 μm (AMAD), as derived by the methods of this report, are compared in Table C.5 with estimates derived from the effective dose, E (that is, as $E \times 0.05$ Sv^{-1}). Two different estimates of effective dose are considered, one derived using the committed effective dose coefficient from Federal Guidance Report No. 11 (1988) and the other derived using the effective dose coefficient from ICRP Publication 72 (1996). (ICRP Publication 72 is a summary of the tabulations of the ICRP's series of documents on age-dependent doses to members of the public from intake of radionuclides.) Both estimates are based on an intake of 1 Bq. The two estimates are abbreviated as 0.05 Sv^{-1} × *E(FGR11)* and 0.05 Sv^{-1} × *E(ICRP72)*, respectively. For simplicity, *E(ICRP72)* is taken to be the effective dose for intake by the adult.

For the case of ingestion of ^{232}Th in food, 0.05 Sv^{-1} × *E(FGR11)* is about three-fold higher than 0.05 Sv^{-1} × *E(ICRP72)*, and 15-fold higher than the risk based on the coefficient derived here (Table C.5). The discrepancies between 0.05 Sv^{-1} × *E(FGR11)* and 0.05 Sv^{-1} × *E(ICRP72)* result in part from differences in the new and previous biokinetic models for thorium (discussed earlier), and in part from recent changes in the ICRP's tissue weighting factors (ICRP, 1991). The discrepancies between 0.05 Sv^{-1} × *E(ICRP72)* and the risk coefficient are the net result of a variety of factors, including the limitations of the effective dose as a measure of risk for non-uniformly distributed radionuclides such as ^{232}Th and its radioactive progeny, differences between the high-LET RBEs for leukemia and breast cancer used in the present methodology and those used by the ICRP, and the failure of the effective dose to account adequately for competing risks when the organ doses are received over several decades.

Table C.5. Comparison of cancer mortality risk coefficients with risk estimates based on effective dose, for ingestion (food) or inhalation of ^{232}Th (Type M, 1 μm AMAD).

Method	Ingestion of ^{232}Th		Inhalation of ^{232}Th, Type M, 1 μm	
	Cancer mortality risk (Bq^{-1})	Multiple of value derived in this report	Cancer mortality risk (Bq^{-1})	Multiple of value derived in this report
0.05 Sv^{-1} × *E(FGR11)*[a]	3.69E-08	15	2.22E-05	43
0.05 Sv^{-1} × *E(ICRP72)*[b]	1.15E-08	4.7	2.25E-06	4.3
This report	2.45E-09	–	5.18E-07	–

[a] *E(FGR11)* is the effective dose given in Federal Guidance Report No. 11 (1988), which is based on models and methods of ICRP Publication 30 (1979).

[b] *E(ICRP72)* is the effective dose for intake by the adult, based on models and methods of the ICRP's recent series of documents on age-dependent dosimetry (ICRP 1989, 1993, 1995a, 1995b, 1996). Use of intake-weighted average of age-dependent effective doses typically yields <30% difference from indicated values for commonly used age-specific intake scenarios.

The discrepancies in the three methods of estimation of fatal cancers are even greater for the case of inhalation of moderately soluble ^{232}Th, for which 0.05 Sv^{-1} × *E(FGR11)* is 10-fold higher than 0.05 Sv^{-1} × *E(ICRP72)* and 40-fold higher than the risk coefficient. The reasons for these discrepancies are essentially the same as those described above for the ingestion case. The main reason that relative differences between 0.05 Sv^{-1} × *E(FGR11)* and the other two estimates are smaller in the ingestion case than in the inhalation case is that the new, higher f_1 values for thorium offset part of the reduction in the estimate of effective dose for ingestion of ^{232}Th implied by other recent changes in the biokinetic models and tissue weighting factors. By contrast with model revisions concerning the level of absorption of ingested thorium, the new respiratory tract model predicts slightly lower absorption of inhaled material to blood than does the previous respiratory tract model.

APPENDIX D. ADJUSTMENT OF RISK COEFFICIENTS FOR SHORT-TERM EXPOSURE OF THE CURRENT U.S. POPULATION

A risk coefficient given in Chapter 2 may be interpreted in terms of either chronic or acute (short-term) exposures. That is, a coefficient may be viewed as the average risk per unit exposure to persons exposed throughout life to a constant concentration of a radionuclide in an environmental medium, or as the average risk per unit exposure in populations exposed over a short period of time to the radionuclide in the environmental medium.

The assumed gender and age distributions in the exposed population are those that would eventually occur in a closed, steady-state population with male-to-female birth ratios characteristic of recent U.S. data and with time-invariant survival functions defined by the 1989-91 U.S. decennial life tables. Because of the uncertainty in the future composition of the U.S. population, the use of a stationary or steady-state population based on recent U.S. vital statistics is judged to be appropriate for consideration of long-term, chronic exposures to the U.S. population. However, these age distributions differ substantially from those of the current U.S. population (Fig. D.1). Hence, the question arises as to the applicability of the risk coefficients to short-term exposures of the U.S. population that might occur in the near future.

The purpose of this appendix is to compare the risk coefficients tabulated in Chapter 2 with coefficients derived for a short-term exposure of a hypothetical population with demographics based on the current U.S. population and, on the basis of this comparison, develop scaling factors for conversion of risk coefficients between the steady-state and current populations. As is the case for the stationary population considered in the main body of the report, total mortality rates in this hypothetical current population are defined by the 1989-91 U.S. decennial life table, and cancer mortality rates are defined by U.S. cancer mortality rates for the same period. In contrast to the stationary population, however, it is assumed that the gender-specific age distribution at the time of exposure is the same as that of the U.S. population of 1996 (U.S. Bureau of the Census, Population Division, 1997).

Computation of risk coefficients for the hypothetical current population

Short-term exposures are treated in the calculations as instantaneous exposures. For example, in the solution of the biokinetic models, ingestion or inhalation of a radionuclide is represented as an initial activity in the stomach compartment or in appropriate compartments of the

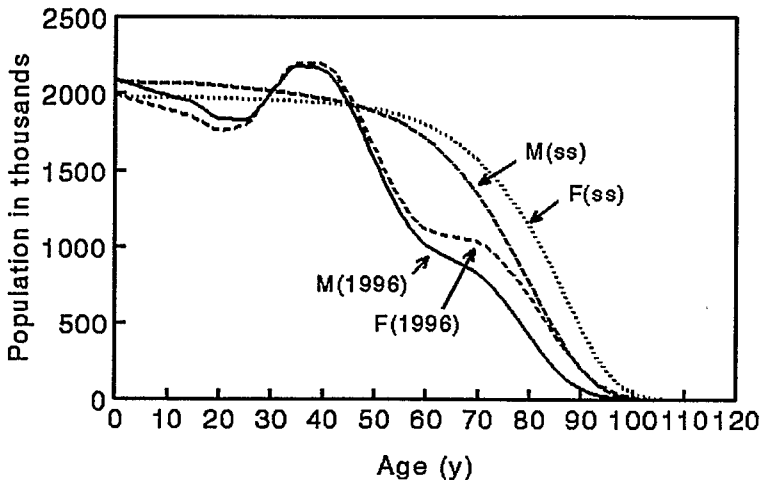

Fig. D.1. Comparison of gender-specific age-distributions in 1996 U.S. population with hypothetical stationary (ss, for steady-state) distributions based on 1989-91 U.S. life table. Normalized to values for age 0 y in 1996 U.S. population. M = males, F = females. Average age: M(1996) = 34.2 y; M(ss) = 38.1 y; F(1996) = 36.9 y; F(ss) = 41.1 y. Average life expectancy: M(1996) = 41.3 y; M(ss) = 38.1 y; F(1996) = 44.7 y; F(ss) = 41.1 y.

respiratory tract, respectively. However, the derived risk coefficients are applicable to any short-term exposure period (e.g., several days, weeks, or months) over which there are only small changes in the gender and age distributions in the population. The coefficients for the hypothetical current population should not be applied to exposure periods longer than a few years because of substantial changes in the age distribution over long periods.

As described in Chapter 7, the average lifetime risk coefficient, $\overline{r_a}$, for continuous intake of a radionuclide is calculated from the age- and gender-specific cancer risk coefficient, $r_a(x)$, by the equation:

$$\overline{r_a} = \frac{\int_0^\infty u(x)\, r_a(x)\, S(x)\, dx}{\int_0^\infty u(x)\, S(x)\, dx} \qquad \text{(D.1)}$$

where $u(x)$ is the gender-weighted usage rate, and $S(x)$ is the gender-weighted survival function. This equation was derived for a stationary population that is subject to fixed gender-specific survival functions and cancer mortality rates. In such a population, the age distribution of a given gender is proportional to the survival function $S(x)$ for that gender. The derived risk coefficients may be interpreted either in terms of lifetime exposure or acute exposure of this population to a radionuclide.

A similar analysis may be applied to the case of acute exposure of a population with an arbitrary age distribution, if it is assumed that the exposed population is subject to fixed gender-specific survival functions and fixed cancer mortality rates at all times after the exposure. In this case, the relative age distribution, $S(x)$, in Eq. D.1 is replaced by a function $P(x)$ representing the age distribution of the population at the time of acute exposure. This change is needed because usage of an environmental medium by members of age x in the hypothetical current population is proportional to $u(x)P(x)$ rather than $u(x)S(x)$. The equation for the current population corresponding to Eq. D.1 for the stationary population is then

$$\overline{r_a} = \frac{\int_0^\infty u(x)\, r_a(x)\, P(x)\, dx}{\int_0^\infty u(x)\, P(x)\, dx} \quad . \tag{D.2}$$

In applications of risk coefficients, it is sometimes necessary to estimate the average usage of environmental media by the population (see Appendix E). Average daily usage values for the hypothetical current population are given in Table D.1 for the four environmental media considered

Table D.1. Average daily usage of environmental media by the two hypothetical populations.

Medium	Males		Females		Combined	
	Stationary	Current	Stationary	Current	Stationary	Current
Air (m^3)	19.2	19.8	16.5	16.3	17.8	18.0
Tap water (L)	1.29	1.25	0.93	0.90	1.11	1.07
Diet (kcal)	2418	2450	1695	1717	2048	2075
Cow's milk (L)	0.282	0.292	0.207	0.214	0.243	0.252

in the internal exposure scenarios. Corresponding values for the stationary population are provided for comparison.

Lifetime risks for acute external exposures are calculated in a manner similar to that for radionuclide intakes. Since the external exposure is not considered to be age dependent, the calculation is simpler. As described in Chapter 7, the average lifetime risk, $\overline{r_e}$, to members of a stationary population from external exposure at a constant exposure rate can be calculated by removing the usage function from Eq. D.1. That is,

$$\overline{r_e} = \frac{\int_0^\infty r_e(x)\, S(x)\, dx}{\int_0^\infty S(x)\, dx} \qquad \text{(D.3)}$$

where $r_e(x)$ is the cancer risk coefficient at age x and $S(x)$ is the survival function and hence the relative age distribution in the stationary population. For the hypothetical current population, the relative age distribution, $S(x)$, is replaced by the function $P(x)$ representing the age distribution of the population at the time of acute exposure. This change is needed because the total exposure to members of the current population of age x is proportional to $P(x)$ rather than $S(x)$. The equation for the current population corresponding to Eq. D.3 for the stationary population is then

$$\overline{r_e} = \frac{\int_0^\infty r_e(x) P(x)\, dx}{\int_0^\infty P(x) dx} \quad . \qquad \text{(D.4)}$$

Comparison of coefficients for the current and stationary populations

Risk coefficients for short-term exposure of the hypothetical current (1996) population were derived for all of the radionuclides and exposure scenarios considered in the main text (Chapters 1-7) and compared with the values tabulated in Chapter 2. Risk coefficients for the current population are consistently greater than the corresponding coefficients for the stationary population, with a maximum difference of 16%. (Table D.2). For a given exposure scenario, the ratios of risk

coefficients for the current and stationary populations are relatively insensitive to the radionuclide, with all ratios falling within 3% and most falling within 1% of the mean ratio (Table D.2).

Therefore, the risk coefficients for the stationary population are reasonably good approximations of the corresponding risk coefficients for short-term exposure of the current population. A closer approximation may be obtained by scaling the coefficients for the stationary population by the exposure-specific mean ratio given in Table D.2. For example, for consideration of short-term inhalation of a radionuclide by the current population, the risk coefficient given in Table 2.1 should be multiplied by 1.11, the mean ratio of inhalation risk coefficients for the current and stationary populations (Table D.2). The scaled coefficient will usually be within 1%, and will always be within 3%, of the risk coefficient derived directly for the current population.

Table D.2. Comparison of risk coefficients for the two hypothetical populations.

Environmental medium	Ratio of risk coefficients for acute exposure current population : stationary population		
	Mean	Standard deviation	Range
Air (inhalation)	1.11	0.008	1.08-1.13
Tap water (ingestion)	1.14	0.013	1.11-1.16
Food (ingestion)	1.10	0.008	1.08-1.11
Milk (ingestion of radioiodine)	1.09	0.006	1.08-1.10
External exposure by submersion in contaminated air	1.11	0.007	1.10-1.14
External exposure to contaminated ground plane	1.11	0.008	1.10-1.13
External exposure to soil contaminated to infinite depth	1.11	0.005	1.10-1.13

APPENDIX E. SAMPLE CALCULATIONS

This appendix provides several sample calculations that illustrate how the tabulated risk coefficients may be applied to different types of exposure. The simplistic exposure scenarios considered here were selected for didactic purposes and are not intended to suggest or endorse assumptions regarding the behavior of radionuclides in the environment.

The risk coefficients in this report represent estimated radiogenic cancer risk, either to a stationary population defined by the 1989-91 U.S. decennial life tables (see Chapter 3) or (when scaled as described in Appendix D) to a hypothetical current population with gender and age distributions based on the total U.S. population in 1996. Risk coefficients for the stationary population are intended mainly to apply to lifetime exposures to radionuclides but, as explained in Chapters 1 and 3, may also be interpreted in terms of acute exposures. Because risk coefficients for the hypothetical current population reflect actual age and gender distributions in the U.S. population in 1996, these coefficients may be appropriate for consideration of short-term exposures (1 y or less) to the current U.S. population or to a representative subpopulation.

For a selected exposure scenario, the computation of risk R involves multiplication of the applicable risk coefficient r by the *per capita* intake I or (external) exposure X for external exposure. That is, $R = r \cdot I$ for intake by inhalation or ingestion and $R = r \cdot X$ for external exposures, where X denotes the time-integrated concentration of the radionuclide in air, on the ground surface, or within the soil, and I is the activity inhaled or ingested *per capita*. Ingestion intakes in tap water and diet are considered. A risk coefficient r is specific to the radionuclide and the mode of exposure or intake. Usage rates for the examples in this appendix are taken from Table D.1.

Some radionuclides considered in this report form radioactive progeny, or daughter products, when undergoing radioactive decay. A series of radionuclides formed by successive radioactive decays is referred to as a decay chain, and the first member of the chain is referred to as the parent. A risk coefficient given in this document does not include the contribution to dose from exposure or intake of other radionuclides that might be present as daughter products in the environment. However, for each radionuclide considered in this document, separate risk coefficients are provided for all radioactive progeny that are considered to be of potential dosimetric significance. Thus, the user may combine risk coefficients for different members of a radionuclide chain to derive a risk coefficient that reflects growth of radioactive progeny in the environment over a user-selected time period.

For example, when considering external exposure to ^{137}Cs on the ground surface, it should be assumed that its short-lived radioactive daughter, ^{137m}Ba ($T_{1/2}$ = 2.552 m) is also present.

Although the risk coefficient for external exposure to ^{137}Cs on the ground surface does not consider the presence of ^{137m}Ba, a separate risk coefficient is provided for external exposure to ^{137m}Ba on the ground surface. As illustrated later in this appendix, an estimate of the risk from the mixture of ^{137}Cs and ^{137m}Ba present on the ground surface may be obtained as a linear combination of the separate risk coefficients for the two radionuclides.

For intake of a relatively long-lived radionuclide, the contribution to dose from its short-lived radioactive progeny (defined here as radioactive progeny with a half-life shorter than 1 h) present in the environment usually is insignificant compared with the dose from the parent. For this reason, separate risk coefficients for ingestion and inhalation are not given for short-lived radioactive progeny of the radionuclides considered in the internal exposure scenarios. For example, risk coefficients are given for ingestion and inhalation of ^{137}Cs but not for ingestion or inhalation of ^{137m}Ba.

On the other hand, after intake of a parent radionuclide, the production and decay of even short-lived radioactive progeny in the body may contribute significantly to organ doses. For this reason, risk coefficients for ingested or inhaled radionuclides include all contributions to dose from growth of chain members in the body.

Example 1. Suppose the concentration of ^{85}Kr in the atmosphere in the environs of a fuel reprocessing plant is 10^3 Bq m^{-3}. Compute the average cancer risk (mortality and morbidity) associated with lifetime external exposure to this level of airborne activity, assuming no shielding by structures.

From Table 2.4, the mortality and morbidity risk coefficients for external exposure to ^{85}Kr in air (submersion) are 7.23×10^{-18} and 1.00×10^{-17} m^3 Bq^{-1} s^{-1}. The years of life lived (the life expectancy at birth) in the stationary population is about 75.2 y (Table A.1). The lifetime exposure resulting from this airborne concentration is

$$10^3 \frac{Bq}{m^3} \cdot 75{,}2y \cdot 3.15 \times 10^7 \frac{s}{y} = 2.37 \times 10^{12} \frac{Bq-s}{m^3} .$$

Therefore, the estimated lifetime risks from the external exposure are

$$\text{Mortality:}\quad 2.37 \times 10^{12}\,\frac{\text{Bq-s}}{\text{m}^3} \cdot 7.23 \times 10^{-18}\,\frac{\text{m}^3}{\text{Bq-s}} = 1.7 \times 10^{-5}$$

$$\text{Morbidity:}\quad 2.37 \times 10^{12}\,\frac{\text{Bq-s}}{\text{m}^3} \cdot 1.00 \times 10^{-17}\,\frac{\text{m}^3}{\text{Bq-s}} = 2.4 \times 10^{-5}\ .$$

Example 2. As in Example 1, suppose the concentration of ^{85}Kr in the atmosphere in the environs of a fuel reprocessing plant is 10^3 Bq m^{-3}. Compute the average cancer risk (mortality and morbidity) associated with a one-year (3.15×10^7 s) external exposure to this level of airborne activity, assuming no shielding by structures and that the age distribution of the population is similar to that of the 1996 U.S. population.

Because the age distribution of the population is similar to that of the 1996 U.S. population, risk coefficients for the stationary population given in Table 2.4 will be scaled as indicated in Appendix D for application to the hypothetical current population. From Table 2.4 the mortality and morbidity risk coefficients for external exposure to ^{85}Kr in air are 7.23×10^{-18} and 1.00×10^{-17} m^3 Bq^{-1} s^{-1}, respectively. From Table D.2, the scaling factor (mean ratio of risk coefficients for hypothetical current and stationary populations) for this exposure scenario is 1.11. The scaled mortality and morbidity risk coefficients for external exposure to ^{85}Kr in air are 8.03×10^{-18} and 1.11×10^{-17} m^3 Bq^{-1} s^{-1}, respectively. The exposure (time-integrated concentration) is

$$10^3\,\frac{\text{Bq}}{\text{m}^3} \cdot 3.15 \times 10^7\,\text{s} = 3.15 \times 10^{10}\,\frac{\text{Bq-s}}{\text{m}^3}\ .$$

The estimated lifetime risks to the population as a consequence of the 1-y external exposure are

$$\text{Mortality:}\quad 3.15 \times 10^{10}\,\frac{\text{Bq-s}}{\text{m}^3} \cdot 8.03 \times 10^{-18}\,\frac{\text{m}^3}{\text{Bq-s}} = 2.5 \times 10^{-7}$$

$$\text{Morbidity:}\quad 3.15 \times 10^{10}\,\frac{\text{Bq-s}}{\text{m}^3} \cdot 1.11 \times 10^{-17}\,\frac{\text{m}^3}{\text{Bq-s}} = 3.5 \times 10^{-7}\ .$$

Example 3. Suppose the ground surface was uniformly contaminated at time zero with ^{137}Cs at a level of 2 Bq m^{-2}. Assume that radioactive decay is the only mechanism by which contamination is reduced. (Reduction of the time-integrated exposure due to weathering is ignored here for simplicity.) Compute the average lifetime cancer risk (mortality and morbidity) resulting from external exposures during the first year following the initial deposition, assuming no shielding and assuming that the age distribution of the exposed population is similar to that of the 1996 U.S. population.

Cesium-137 ($T_{1/2}$ = 30 y) forms ^{137m}Ba ($T_{1/2}$ = 2.552 m) in 94.6% of its decays (see Appendix A of EPA, 1993). Due to the short half-life of ^{137m}Ba, the concentration of ^{137m}Ba on the ground surface will reach 1.89 Bq m^{-2} (0.946 · 2 Bq m^{-2}) within a half hour after time zero and will decline with the half-life of ^{137}Cs.

From Table 2.4 the mortality and morbidity risk coefficients for external exposure to ^{137}Cs distributed on the ground surface are 3.96×10^{-20} and 4.57×10^{-20} m^2 Bq^{-1} s^{-1}. For ^{137m}Ba the corresponding coefficients are 3.12×10^{-17} and 4.60×10^{-17} m^2 Bq^{-1} s^{-1}, respectively. From Table D.2, the scaling factor (mean ratio of risk coefficients for hypothetical current and stationary populations) for external exposure from ground surface contamination is 1.11. The scaled mortality and morbidity risk coefficients for ^{137}Cs are 4.40×10^{-20} and 5.07×10^{-20} m^2 Bq^{-1} s^{-1}, respectively, and the scaled values for ^{137m}Ba are 3.46×10^{-17} and 5.11×10^{-17} m^2 Bq^{-1} s^{-1}, respectively. The exposures (time-integrated concentration) for each radionuclide during the first year are

$$\text{Exposure} = A_0 \int_0^T e^{\frac{-\ln 2\, t}{T_{1/2}}}\, dt = \frac{A_0\, T_{1/2}}{\ln 2}\left(1 - e^{\frac{-\ln 2\, T}{T_{1/2}}}\right)$$

$$\text{Cs-137:}\quad \frac{2\,\frac{Bq}{m^2}\cdot 30y\cdot 3.15\times 10^7\,\frac{s}{y}}{0.693}\left(1 - e^{\frac{-0.693\ 1y}{30y}}\right) = 6.23\times 10^7\,\frac{Bq\text{-}s}{m^2}$$

$$\text{Ba-137m:}\quad \frac{1.89\,\frac{Bq}{m^2}\cdot 30y\cdot 3.15\times 10^7\,\frac{s}{y}}{0.693}\left(1 - e^{\frac{-0.693\ 1y}{30y}}\right) = 5.89\times 10^7\,\frac{Bq\text{-}s}{m^2}\ .$$

The lifetime risks resulting from external exposures during the first year are

Mortality:

$$6.23 \times 10^{7} \frac{\text{Bq-s}}{\text{m}^2} \cdot 4.40 \times 10^{-20} \frac{\text{m}^2}{\text{Bq-s}}$$

$$+ 5.89 \times 10^{7} \frac{\text{Bq-s}}{\text{m}^2} \cdot 3.46 \times 10^{-17} \frac{\text{m}^2}{\text{Bq-s}} = 2.0 \times 10^{-9}$$

Morbidity:

$$6.23 \times 10^{7} \frac{\text{Bq-s}}{\text{m}^2} \cdot 5.07 \times 10^{-20} \frac{\text{m}^2}{\text{Bq-s}}$$

$$+ 5.89 \times 10^{7} \frac{\text{Bq-s}}{\text{m}^2} \cdot 5.11 \times 10^{-17} \frac{\text{m}^2}{\text{Bq-s}} = 3.0 \times 10^{-9}.$$

Note that the radiations emitted by ^{137m}Ba are the main contributors to risk.

Example 4. Assume that measurements of the photon radiation field indicate an average exposure rate of 4 μR/h and that no information is available regarding the energy of the radiation or its origin. Compute the average lifetime risk to a population living in this radiation field, assuming no shielding by structures.

Table 7.3 gives a mortality risk of 5.75×10^{-2} Gy^{-1} for uniform irradiation of the body by low-LET radiation. Assuming an average lifetime of 75.2 years (Table A.1) and, for estimation purposes, equating the radiation unit R with rem, the expected lifetime dose due to this radiation field is

$$4 \times 10^{-6} \frac{\text{R}}{\text{h}} \cdot 1 \frac{\text{rem}}{\text{R}} \cdot 75.2 \text{ y} \cdot 8.76 \times 10^{3} \frac{\text{h}}{\text{y}} \cdot 0.01 \frac{\text{Gy}}{\text{rem}} \approx 2.64 \times 10^{-2} \text{ Gy}$$

and the mortality risk is about

$$2.64 \times 10^{-2} \text{ Gy} \cdot 5.75 \times 10^{-2} \text{ Gy}^{-1} \approx 1.5 \times 10^{-3} .$$

Example 5. Calculate the average lifetime risk to the stationary population associated with ingestion of ^{210}Pb and its radioactive progeny, assuming that the *per capita* dietary intake rates of ^{210}Pb and ^{210}Po are 1.4 and 1.8 pCi d^{-1}, respectively.

Lead-210 decays to ^{210}Bi ($T_{1/2}$ = 5.012 d), which decays to ^{210}Po ($T_{1/2}$ = 138.8 d) . Because of the relatively short half-life of ^{210}Bi, it seems reasonable to assume that ^{210}Bi is in equilibrium with ^{210}Pb in diet.

From Table A.1, the average life expectancy is 27,448 d (75.2 y). Therefore, lifetime intakes of ^{210}Pb, ^{210}Bi, and ^{210}Po in the diet are estimated to be

$$\text{Pb-210/Bi-210:}\quad 1.4\ \frac{\text{pCi}}{\text{d}} \cdot 3.7 \times 10^{-2}\ \frac{\text{Bq}}{\text{pCi}} \cdot 27{,}448\ \text{d} = 1.4 \times 10^{3}\ \text{Bq}$$

$$\text{Po-210:}\quad 1.8\ \frac{\text{pCi}}{\text{d}} \cdot 3.7 \times 10^{-2}\ \frac{\text{Bq}}{\text{pCi}} \cdot 27{,}448\ \text{d} = 1.8 \times 10^{3}\ \text{Bq}$$

The following mortality and morbidity risk coefficients for ^{210}Pb, ^{210}Bi, and ^{210}Po in diet are taken from Table 2.3a: ^{210}Pb, 2.31×10^{-8} and 3.18×10^{-8}, respectively; ^{210}Bi, 1.95×10^{-10} and 3.52×10^{-10}, respectively; and ^{210}Po, 4.44×10^{-8} and 6.09×10^{-8}, respectively. The estimated risks are

$$\begin{aligned}\text{Mortality:}\quad & 1.4 \times 10^{3}\ \text{Bq} \cdot 2.31 \times 10^{-8}\ \frac{1}{\text{Bq}} + \\ & 1.4 \times 10^{3}\ \text{Bq} \cdot 1.95 \times 10^{-10}\ \frac{1}{\text{Bq}} + \\ & 1.8 \times 10^{3}\ \text{Bq} \cdot 4.44 \times 10^{-8}\ \frac{1}{\text{Bq}} = 1.1 \times 10^{-4}\end{aligned}$$

$$\begin{aligned}\text{Morbidity:}\quad & 1.4 \times 10^{3}\ \text{Bq} \cdot 3.18 \times 10^{-8}\ \frac{1}{\text{Bq}} + \\ & 1.4 \times 10^{3}\ \text{Bq} \cdot 3.52 \times 10^{-10}\ \frac{1}{\text{Bq}} + \\ & 1.8 \times 10^{3}\ \text{Bq} \cdot 6.09 \times 10^{-8}\ \frac{1}{\text{Bq}} = 1.5 \times 10^{-4}\end{aligned}$$

Note that ^{210}Bi makes an insignificant contribution to the total risk and that ^{210}Po accounts for about two-thirds of the risk.

Example 6. Assume a concentration of tritium in drinking water of 10 pCi L^{-1}. Compute the average lifetime risk (mortality and morbidity) associated with use of tap water at this concentration, assuming that all tritium in tap water is in the form of tritiated water.

The average intake of tap water is 1.11 L d^{-1} (Table D.1), and the average life expectancy is 27,448 d (75.2 y, Table A.1), giving a lifetime intake of tap water of 3.0×10^4 L. From Table 2.2, the mortality and morbidity coefficients for ^3H (as tritiated water) in tap water are 9.44×10^{-13} and 1.37×10^{-12} Bq^{-1}, respectively. Therefore, the estimated risks are

$$\text{Mortality:}\quad 10\ \frac{pCi}{L}\cdot 0.037\ \frac{Bq}{pCi}\cdot 3.0\times10^4\ L\cdot 9.44\times10^{-13}\ \frac{1}{Bq} = 1.0\times10^{-8}$$

$$\text{Morbidity:}\quad 10\ \frac{pCi}{L}\cdot 0.037\ \frac{Bq}{pCi}\cdot 3.0\times10^4\ L\cdot 1.37\times10^{-12}\ \frac{1}{Bq} = 1.5\times10^{-8}$$

Example 7. Suppose there is a short-term release of 40 mCi of ^{131}I as a vapor from a reactor and that observed atmospheric conditions indicate an atmospheric dispersion factor of about 1×10^{-6} s m^{-3} for a nearby population. Compute the risk associated with inhalation of ^{131}I as the cloud passes over the population, assuming that the age distribution of the population is similar to that of the stationary population considered in the main text.

The time integrated airborne concentration in the cloud is

$$40\ mCi\cdot 3.7\times10^7\ \frac{Bq}{mCi}\cdot 1.0\times10^{-6}\ \frac{s}{m^3} = 1.48\times10^3\ \frac{Bq\text{-}s}{m^3}\ .$$

The average inhalation intake rate is 17.8 m^3 d^{-1} (Table D.1). The mortality and morbidity coefficients for inhalation of ^{131}I in vapor form are 1.48×10^{-10} and 1.36×10^{-9} Bq^{-1} (Table 2.1). Therefore, the estimated risks are

$$\text{Mortality:}\quad 1.48\times10^3\ \frac{Bq\text{-}s}{m^3}\cdot 17.8\ \frac{m^3}{d}\cdot\frac{1\ d}{8.64\times10^4\ s}\cdot 1.48\times10^{-10}\ \frac{1}{Bq} = 4.5\times10^{-11}$$

$$\text{Morbidity:}\quad 1.48\times10^3\ \frac{Bq\text{-}s}{m^3}\cdot 17.8\ \frac{m^3}{d}\cdot\frac{1\ d}{8.64\times10^4\ s}\cdot 1.36\times10^{-9}\ \frac{1}{Bq} = 4.1\times10^{-10}\ .$$

GLOSSARY

Absolute risk hypothesis: The assumption that the excess risk from radiation exposure adds to the underlying (baseline) risk by an increment dependent on dose but independent of the underlying risk.

Absorbed dose (*D*): The microscopic quantity is the differential $d\bar{\epsilon}/dm$, where $d\bar{\epsilon}$ is the mean energy imparted by ionizing radiation to matter of mass *dm*. The macroscopic quantity used in internal dosimetry is tissue-averaged; that is, the absorbed dose to a tissue is the total energy absorbed by the tissue, divided by the mass of the tissue. The special name for the SI unit of absorbed dose ($J\ kg^{-1}$) is gray (Gy). The conventional unit of absorbed dose is the rad. 1 rad = 0.01 Gy.

Absorption type: In the ICRP's respiratory tract model introduced in 1994, a classification scheme for inhaled material according to its rate of absorption from the deep lungs to blood. Three main absorption types are considered: Type F (fast rate), Type M (moderate rate), and Type S (slow rate).

Absorbed fraction (*AF*): The fraction of energy emitted as a specified radiation type in a specified source region that is absorbed in a specified target region.

Activity: The quantity of a radioactive nuclide present at a particular time, expressed in terms of the mean rate of nuclear transformations. The special name for the SI unit of activity (s^{-1}) is becquerel (Bq). The conventional unit of activity is the curie (Ci). $1\ Ci = 3.7\times10^{10}$ Bq.

Activity Median Aerodynamic Diameter (AMAD): The diameter of a unit density sphere with the same terminal settling velocity in air as that of an aerosol particle whose activity is the median for the entire aerosol.

Acute exposure: For purposes of computing risk coefficients, an instantaneous exposure. For practical applications of risk coefficients, any relatively short-term exposure period over which there are numerically trivial changes in the body mass, biokinetic parameters, usage functions, and mortality rates of all, or nearly all, members of the population.

Alpha particle: Two neutrons and two protons bound as a single particle (helium nucleus), emitted from the nucleus of certain radionuclides during nuclear transformations.

Baseline cancer rate: The observed cancer mortality (or morbidity) rate in a population in the absence of the specific radiation exposure being studied.

Becquerel (Bq): The special name for the SI unit of activity. $1\ Bq = 1\ s^{-1}$.

Beta particle: A particle having the charge and mass of an electron, emitted from the nucleus of certain radionuclides.

Biokinetic model: A mathematical description of the time-dependent distribution and translocation of a substance in the body.

Body Tissues (BT): The entire body, minus the contents of the gastrointestinal tract, the urinary bladder, the gall bladder, and the heart. Formerly called Whole Body (WB).

Bone Surface: The soft tissues within 10μm of the endosteal (interior) surfaces of bone.

Bremsstrahlung: Electromagnetic radiation produced when deceleration of electrons in a medium results in conversion of a small fraction of their initial kinetic energy into photons.

Chain members: The sequence of radionuclides formed by successive nuclear transformations, beginning with a radionuclide referred to as the parent.

Chronic exposure: In this report, protracted exposure to a constant concentration of a radionuclide in a given environmental medium.

Committed equivalent dose: The time integral of the equivalent dose rate.

Committed effective dose: Sometimes shortened to "effective dose"; the time integral of the effective dose rate.

Competing cause of death: Any cause of death other than radiogenic cancers attributed to the radionuclide intake or external radiation exposure under consideration.

Cortical bone, compact bone: Bone with a surface-to-volume ratio less than 60 $cm^2\ cm^{-3}$.

Curie (Ci): The conventional unit of activity. 1 Ci = 3.7×10^{10} Bq.

Daughter radionuclide: A radionuclide formed by the nuclear transformation of another radionuclide referred to, in this context, as its parent.

DCAL: Acronym for DOSE CALCULATION System, the software used to compute the risk coefficients tabulated in this document.

DDREF: A factor used to account for an apparent decrease of the risk of cancer per unit dose at low doses or low dose rates for most cancer sites compared with observations made at high, acutely delivered doses.

Dose coefficient, dose factor: The committed equivalent dose to a tissue, or the committed effective dose, per unit intake of a radionuclide.

DOE: U.S. Department of Energy.

Effective dose (E): The sum over specified tissues of the products of the equivalent dose in a tissue or organ (T) and the weighting factor for that tissue, w_T, that is, $E = \Sigma_T w_T H_T$. Lower-case e is used in ICRP documents to denote an effective dose coefficient, that is, effective dose per unit intake of a radionuclide at a given age. The special name for the SI unit of effective dose (J kg^{-1}) is sievert (Sv). The conventional unit of effective dose is the rem. 1 rem = 0.01 Sv.

EPA: U.S. Environmental Protection Agency.

Equivalent dose (H): The product of the absorbed dose (D) and the radiation weighting factor (w_R). Lower-case h is used in ICRP documents to denote a dose coefficient, that is, a committed equivalent dose per unit intake of a radionuclide at a given age. The special name for the SI unit of equivalent dose (J kg^{-1}) is sievert (Sv). The conventional unit of equivalent dose is the rem. 1 rem = 0.01 Sv.

External exposure: Exposure to radiations emitted by radionuclides outside the body.

f_1: The fraction of a radionuclide reaching the stomach that would be absorbed to blood during passage through the gastrointestinal tract without radiological decay.

Federal Guidance: Principles, policies, and numerical primary guides, approved by the President upon recommendation of the Administrator of EPA, for use by Federal agencies as the basis for developing and implementing regulatory standards.

Force of mortality: The age- and gender-specific mortality (or hazard) rate coefficient, μ (y^{-1}), for a cause of death. The probability that an individual alive at age x will die of that cause before attaining age $x + dx$ is equal to μdx.

FRC: The former U.S. Federal Radiation Council, whose functions now reside with the Administrator of EPA.

Gamma radiation, gamma rays: Short wavelength electromagnetic radiation of nuclear origin, similar to x rays but usually of higher energy.

Gastrointestinal tract model: A model of the translocation of swallowed material through the stomach and intestines.

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

Half-time, biological: Time required for the quantity of a radionuclide in a compartment representing all or a portion of the body to diminish by 50% without radiological decay or any additional input to the compartment.

Half-life, radioactive: Time required for a radionuclide to lose 50% of its activity by spontaneous nuclear transformations (radiological decay).

HTO: Tritiated water.

ICRP: International Commission on Radiological Protection.

Independent kinetics of decay chain members: The assumption that each decay chain member produced in the body may have biokinetic behavior that is different from that of the radionuclide taken into the body.

Internal exposure: Exposure to radiations emitted by radionuclides distributed within the body.

Ionizing radiation: Any radiation capable of removing electrons from atoms or molecules, thereby producing ions.

***In utero* exposure:** Radiation exposure received in the womb, that is, before birth.

***In vivo*:** In the living organism.

I-S: Inorganic sulfur.

Isotopes: Nuclides that have the same number of protons in their nuclei and hence the same atomic number but differ in the number of neutrons and therefore in mass number.

Kerma: The kinetic energy transferred to charged particles per unit mass of irradiated medium when indirectly ionizing (uncharged) particles such as photons or neutrons traverse the medium. The special name for the SI unit of kerma ($J\ kg^{-1}$) is gray (Gy).

LET: Average amount of energy lost per unit track length of an ionizing charged particle. *Low LET* refers to radiation characteristic of light charged particles such as electrons produced by x rays and gamma rays where the distance between ionizing events is large on the scale of a cellular nucleus. *High LET* refers to radiation characteristic of heavy charged particles such as protons and alpha particles where the distance between ionizing events is small on the scale of a cellular nucleus.

Lethality fraction: The fraction of radiogenic cancers of a given type that are fatal.

Life Table: A table showing the number of persons who, for a given number of live born, survive to successively higher ages.

Lifetime risk coefficient (LRC): The risk per unit dose of a subsequent cancer death due to radiation received at a given age.

Linear model, Linear dose-effect relationship: A model describing a radiogenic effect as a linear function of dose.

Linear-quadratic model, Linear-quadratic dose-effect relationship: A model describing a radiogenic effect as a quadratic function of dose, D (that is, as $a \cdot D + b \cdot D^2$, where a and b are constants).

Low dose rate: For this report, an hourly averaged absorbed dose rate less than 0.1 mGy min^{-1}.

Low dose: For this report, an acute absorbed dose less than 0.2 Gy.

Minimal latency period: The minimal time following a radiation dose before expression of a radiogenic cancer.

Mortality rate: The age- and gender-specific or total rate at which people die from a specified cause of death, or all causes combined.

MIRD: Medical Internal Radiation Dose; a committee of the Society of Nuclear Medicine.

Morbidity: The age- and gender-specific or total incidence of a specified disease in the population.

Multiplicative transport model: The assumption that the excess relative risk coefficient for a radiogenic cancer is the same across populations.

NCHS: U.S. National Center for Health Statistics.

NCRP: U.S. National Council on Radiation Protection and Measurements.

Neutron: Uncharged subatomic particle capable of producing ionization in matter by collision with protons and through nuclear reactions.

NHANES III: A national dietary, health, and nutrition survey conducted by the National Center for Health Statistics (NCHS) during the period 1988-1994.

NIH: U. S. National Institutes of Health.

NIH transport model: The assumption that the relative risk model coefficients for the target population should yield the same risks as those calculated with the additive risk model coefficients from the original population over the period of epidemiological follow-up, excluding the minimal latency period.

NRC: U.S. Nuclear Regulatory Commission.

Nuclear transformation: The spontaneous transformation of one radionuclide into a different nuclide or into a different energy state of the same nuclide.

OBT: Organically bound tritium.

OBS: Organically bound sulfur.

Other: In internal radiation dosimetry, an implicit source region, defined as the complement of the set of explicitly identified regions, that is, *Body Tissues* minus the explicit source organs identified in the biokinetic model.

Parent radionuclide: The first member of a chain of radionuclides. In an internal exposure scenario, the radionuclide assumed to be taken into the body.

***Per capita*:** Averaged over the population.

Phantom: A mathematical model of the human body, used in radiation dosimetry to derive specific absorbed fractions for penetrating radiations.

Plateau period: The time period following a radiation dose during which radiogenic cancers are likely to occur.

Probability coefficient (for radiological risk): A multiplicative factor used to convert a measure of cumulative dose to a probability of a detrimental effect of radiation. As used by the ICRP, an estimate of the radiation risk per unit effective dose. A probability coefficient is generally based on an idealized population receiving a uniform dose over the whole body.

Rad: The conventional unit for absorbed dose of ionizing radiation. 1 rad = 0.01 Gy.

Radiation risk model: A mathematical model used to estimate the probability of experiencing a radiogenic cancer as a function of time after a radiation dose is received.

Radiation weighting factor (w_R): The principal modifying factor employed in deriving equivalent dose, H, from absorbed dose, D; chosen to account for the relative biological effectiveness (RBE) of the radiation in question, but to be independent of the tissue or organ under consideration, and of the biological endpoint.

Radioisotope: A radioactive atomic species of an element with the same atomic number and usually identical chemical properties.

Radionuclide: A radioactive species of atom characterized by the number of protons and neutrons in its nucleus.

RBE: The relative biological effectiveness of a given type of radiation in producing a specified biological effect, compared with 200-kV x rays.

Reference Man: A hypothetical average adult person with the anatomical and physiological characteristics defined in the report of the ICRP Task Group on Reference Man (ICRP Publication 23).

Relative risk hypothesis: The assumption that the age-specific force of mortality or morbidity due to a radiation dose is the product of an exposure-age-specific excess relative risk coefficient and the corresponding baseline cancer mortality or morbidity rate.

Rem: The conventional unit of equivalent dose. 1 rem = 0.01 Sv.

RERF: Radiation Effects Research Foundation; a bi-nationally funded Japanese foundation chartered by the Japanese Welfare Ministry under an agreement between the U.S. and Japan.

Residual cancers: A composite of all primary and secondary cancers not explicitly identified in a radiogenic risk model.

Respiratory tract model: A model of the deposition, retention, and translocation of particles in the respiratory tract.

Risk model coefficient: An age- and gender-specific multiplicative factor appearing in a radiogenic risk model and indicating the magnitude of the risk of dying from or experiencing a given type of cancer at any given time after the dose is received.

Risk coefficient: For a given radionuclide, environmental medium, and mode of exposure, the estimated probability of radiogenic cancer mortality or morbidity, per unit activity intake for internal exposures or per unit exposure for external exposures.

SEECAL: A computer code used to calculate age-dependent specific energies based on standard nuclear decay data files, libraries of specific absorbed fractions for photons and non-penetrating radiations, and organ masses of reference humans of different ages.

Shared kinetics of decay chain members: The assumption that decay chain members produced in the body have the same biokinetic behavior as the radionuclide taken into the body.

Shielding: Material between a radiation source and a potentially exposed person that reduces the radiation field incident on the exposed person.

Short-lived radionuclide: In this report, a radionuclide having a half-life less than 1 h.

Sievert (Sv): The special name for the SI unit of equivalent dose. $1\ \mathrm{Sv} = 1\ \mathrm{J\ kg^{-1}}$.

Soft Tissues: Body Tissues minus cortical and trabecular bone.

Source organ, source region, source tissue (S): Any tissue or organ of the body, or the contents of any organ, which contains a sufficient amount of a radionuclide to irradiate a target tissue (T) significantly.

Specific energy $SE(T \leftarrow S)_R$: The energy per unit mass of target tissue (T), deposited in that tissue as a consequence of the emission of a specified radiation (R) per nuclear transformation of a specified radionuclide occurring in a source tissue (S).

Stationary population, Steady-state population: A hypothetical closed population whose gender-specific birth rates and survival functions remain invariant over time.

Submersion: External exposure to a radionuclide uniformly distributed in the air surrounding the exposed person.

Surface-seeking radionuclides: Radionuclides that deposit on and remain for a considerable period on the surface of bone structure.

Survival function: The fraction $S(x)$ of live-born individuals in an unexposed population expected to survive to age x.

Systemic biokinetic model: A model describing the distribution and translocation of a substance after its absorption or injection into the systemic circulation.

Tap water: Drinking water, water added to beverages, and water added to foods during preparation but not including water intrinsic in food as purchased.

Target organ, target region, target tissue (T): Any tissue or organ of the body in which radiation is absorbed.

Threshold hypothesis: The assumption that no radiation injury occurs below a specified dose.

Time-since-exposure (TSE) function: A function that defines the period during which radiogenic risk is expressed and any changes in the level of response during that period.

Tissue (organ) weighting factor (w_T): A factor indicating the relative level of risk of cancer induction or heredity defects from irradiation of a given tissue or organ; used in calculation of effective dose and committed effective dose.

Trabecular bone, cancellous bone: Bone with a surface-to-volume ratio greater than 60 $cm^2\ cm^{-3}$.

Transportation of risk estimates: Extrapolation of radiogenic dose-response data from one population to another.

Transfer coefficient: In the context of a compartmental model, fractional flow per unit time from one compartment to another.

Time since response function: A function describing the likely pattern of response as a function of time after irradiation of a large population.

Usage rate: The age- and gender-specific average intake rate of a specified environmental medium (air, food energy, tap water, or milk).

Volume-seeking radionuclides: Radionuclides that enter bone and exchange with bone mineral over the entire mass of bone.

Volume source: Relative to a given biokinetic model, a source region that has non-zero volume.

x radiation, x rays: Penetrating electromagnetic radiation, usually produced by bombarding a metallic target with fast electrons in a high vacuum, or emitted during rearrangement of the electrons about the nucleus following nuclear transformation of a radionuclide.

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