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# The Use of Effective Dose as a Radiological Protection Quantity

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# [Guest] Editorial

***To be drafted.***

# ABSTRACT

## The Use of Effective Dose as a Radiological Protection Quantity

ICRP PUBLICATION 1XX

Approved by the Commission in MONTH 201X

**Abstract**-The concept of 'effective dose' (*E*) was developed by ICRP as a risk-adjusted dosimetric quantity for the management of protection against stochastic effects, principally cancer, enabling comparison of planned or received doses with dose limits, dose constraints, and reference levels expressed in the same quantity. Its use allows all radiation exposures from external and internal sources to be considered together and summed, relying on the assumptions of a linear-non-threshold dose-response relationship, equivalence of acute and chronic exposures at low doses or low dose rates, and equivalence of external and internal exposures. Considering exposures incurred by patients during medical procedures, *E* is of practical value for comparing: doses from different diagnostic examinations and interventional procedures; the use of similar technologies and procedures in different hospitals and countries; and the use of different technologies for the same medical examination, provided that the representative patients or patient populations for which the effective doses are derived are similar with regard to age and sex. As stated in the 2007 Recommendations (ICRP, 2007a), "… risk assessment for medical diagnosis and treatment … is best evaluated using appropriate risk values for the individual tissues at risk and for the age and sex distribution of the individuals undergoing the medical procedures". *Publication 103* (ICRP, 2007a) provides detailed explanation of the purpose and use of *E* and of equivalent dose to individual organs and tissues. However, questions have arisen regarding practical applications, highlighting a clear need for further guidance on specific aspects. This publication draws on the explanations provided in *Publication 103* and emphasises that *E* has proved a valuable and robust quantity for use in the optimisation of protection, to set dose criteria and verify compliance. Conclusions are drawn that: a) Equivalent dose (*H*) is not required as a protection quantity. It will be more appropriate for limits for the avoidance of tissue reactions for the hands and feet, lens of the eye, and skin, to be set in terms of absorbed dose (Gy) rather than equivalent dose (Sv). b) While risk assessments for individuals based on organ/tissue doses and specific dose-risk models make best use of scientific knowledge, *E* may be used as an approximate indicator of possible risk, recognising that this is a pragmatic, but unintended, application of effective dose. It is made clear in this report that while doses incurred at low levels of exposure may be measured or assessed with reasonable accuracy, the associated risks are increasingly uncertain at lower doses. However, bearing in mind the uncertainties associated with risk projection to low doses, *E* may be considered as an approximate indicator of possible risk, with the additional consideration of variation in risk with age, sex and population group. Use of *E* in this way is not a substitute for risk analysis using best estimates of organ/tissue doses, appropriate information on the relative effectiveness of different radiation types, and age-, sex- and population-specific risk factors, with consideration of uncertainties.

*Keywords:* Absorbed dose; Equivalent dose; Effective dose; Stochastic risks

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# PREFACE

Experience has shown that the quantity 'effective dose' which has been defined and introduced by ICRP for risk management purposes, i.e. for risk limitation and optimisation, is widely used in radiological protection and related fields beyond its original purpose, incorrectly in some cases. Useful guidance on restrictions for the use of the quantity is provided in the 2007 Recommendations (ICRP, 2007a). However, ICRP has recognised the need to expand this guidance with an important focus being medical exposures.

The Task Group has made use of a draft report produced by a Working Party chaired by John Cooper. Task Group membership has included members of Committees 1, 2, 3 and 4.

The membership of Task Group 79 was as follow:

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# MAIN POINTS

The majority of the information provided in this report and the main points listed below have the purpose of clarifying the intended use of the ICRP protection quantities. Changes and extensions to previously endorsed practice are shown in bold.

- The dosimetric quantities used in radiological protection are absorbed dose ($D$), with the special name of gray (Gy), and equivalent dose ($H$) and effective dose ($E$), both with the special name of sievert (Sv); the SI unit is J $kg^{-1}$ in each case.
- Absorbed dose is calculated for radiological protection purposes as an average over organs and tissues and is the primary scientific quantity from which $E$ is calculated. **Absorbed dose is the most appropriate quantity for use in setting limits on organ/tissue doses to prevent tissue reactions (deterministic effects).**
- Equivalent dose to organs and tissues is obtained by multiplying organ/tissue absorbed doses by radiation weighting factors ($w_R$) to account for the relative effectiveness of different radiation types in causing stochastic effects at low levels of exposure can be seen as an intermediate step in the calculation of $E$. **The Commission considers that the use of equivalent dose to set limits on organ/tissue doses to prevent tissue reactions should be discontinued, but that current limits can continue to be applied until new general recommendations are issued.**
- Effective dose is calculated as the weighted average of organ/tissue equivalent doses, summing equivalent doses multiplied by tissue weighting factors ($w_T$) which provide a simplified representation of fractional contributions to total stochastic detriment from cancer and hereditary effects. Detriment-adjusted nominal risk coefficients ($Sv^{-1}$) are calculated as averages from sex-, age-, and population-specific values, to provide internationally applicable coefficients for all workers (18-64 years at exposure) and the whole population (0-84 years at exposure).
- $E$ is accepted internationally as the central radiological protection quantity, providing a risk-adjusted measure of total body dose from external and internal sources in relation to risks of cancer and hereditary effects.
- $E$ has proved to be a valuable and robust quantity for use in the optimisation of protection, the setting of control criteria (limits, constraints and reference levels), and the demonstration of compliance.
- The use of $E$ requires the assumption of a linear-non-threshold dose-response relationship between dose and risk at low doses or low dose rates, of the equivalence of effect of acute and chronic low-level exposures, and of internal and external exposures.
- $E$ is calculated for sex-averaged Reference Persons of specified ages. The *Publication 103* (ICRP, 2007a) definition of $E$ includes the specification of reference male and female anatomical models for radiation transport calculations. While exposures may relate to individuals or population groups, $E$ is calculated for Reference Persons exposed in the same way.
- **Although $E$ will generally be used at doses below 100 mSv, its use exceptionally in emergency exposure situations at acute doses in the range up to around 1 Sv is reasonable, noting that the possibility of occurrence of tissue reactions should also be considered at such doses if a significant contribution is made by non-**

**uniform distribution of external dose or radionuclides concentrated in specific tissues/organs.**

- ICRP provides effective dose coefficients for situations of external and internal exposures of workers and members of the public, and for radiopharmaceutical administrations to patients, as reference coefficients for use in prospective and retrospective dose assessments.
- In general, while dose coefficients change with each new set of general recommendations, there should be no general requirement for the recalculation of previous dose assessments.
- Reference dose coefficients are provided for particular circumstances of exposure, including specific chemical and physical forms of ingested and inhaled radionuclides. Site-specific information on the exposure should be used if available and if the level of exposure warrants more precise estimation of dose.
- In evaluating annual exposures, *E* is calculated as the sum of external dose received in the year and committed dose from internal exposures during the year, where committed dose is integrated over a 50-year period for adults and to age 70 years for children. This procedure introduces an element of conservatism for long-lived radionuclides with long biological half-times.
- Although effective dose coefficients are provided for a number of age groups of children, it is normally sufficient in public dose assessments to use only the groups 1 year, 10 years and adults.
- Effective dose coefficients for the fetus following intakes of radionuclides are provided for comparison with dose for other age groups, showing that it is only in the case of a few radionuclides that fetal doses may need to be considered.
- While age-, sex-, and population-related differences in risks per Gy are recognised, the use of constraints and reference levels set in effective dose and applying to all workers and all members of the public, together with optimisation, provides a pragmatic, equitable and workable system of protection that does not distinguish on an individual basis.
- In medical applications, estimates of *E* to Reference Persons are used for comparing doses from different diagnostic and interventional imaging modalities (e.g. CT and nuclear medicine) and exposure techniques that give different spatial distributions of radiation within the body tissues. In this context, *E* is used to provide a generic indicator for classifying different types of medical procedure into broad risk categories for the purpose of communicating risks to clinicians and patients.
- *E* is also used to inform decisions on justification of patient diagnostic and interventional procedures, planning requirements in research studies, and evaluation of unintended exposures. In each of these cases, *E* provides a measure of detriment. Thus, *E* can be used prospectively as an indicator of radiation detriment in justification decisions and when planning medical research studies involving radiation exposure, or retrospectively in initial assessments of unintended exposures or overexposures of patients.
- **Bearing in mind the uncertainties associated with risk projection to low doses or dose rates, *E* may be considered as an approximate indicator of possible risk, with the additional consideration of variation in risk with age, sex and population group.**
- For medical procedures or other situations in which a single radiosensitive organ receives the majority of the dose, such as the breast in mammography, or the thyroid

from therapeutic administration of iodine, mean absorbed doses to the tissues of interest should be used rather than effective dose. In considering doses to patients having diseases with poor prognoses, life expectancy will be a consideration in evaluating radiation risks.

- The use of *E* as an approximate indicator of possible risk is not a substitute for a risk analysis using best estimates of organ/tissue doses, appropriate information on the relative effectiveness of different radiation types, and age-, sex- and population-specific risk factors, with consideration of uncertainties.
- **Collective effective dose is a valuable tool in the optimisation of protection, particularly for occupational exposures. It is not intended for use in risk projection. Its use to predict potential/possible health effects should be treated with great caution, put into context and judged in relation to baseline lifetime morbidity risks.** For public exposures, components of dose integration in time and space should be considered in estimating collective doses, particularly when considering exposures of large populations over very long periods of time.

# 1. INTRODUCTION

(1) Effective dose was originally introduced in the 1977 Recommendations of the International Commission on Radiological Protection (ICRP, 1977) for the control of occupational and public exposures to external and internal sources of radiation. While the concept has remained essentially unchanged through the 1990 Recommendations (ICRP, 1991b) to the 2007 Recommendations (ICRP, 2007a), its use has been extended to members of the public of all ages, including *in utero* exposures of the fetus (ICRP, 2001, 2004, 2006). In addition, it is widely used in medical applications, which include its interpretation as a measure of risk to individual patients, contrary to its intended use (Martin, 2007a; McCullough et al. 2010; Balonov and Shrimpton, 2012; Brenner, 2008, 2012; Harrison and Ortiz-Lopez, 2015).

(2) The ICRP protection quantities – equivalent dose ($H$) and effective dose ($E$) – enable the summation of doses from internal emitters and from external sources to provide a single number for comparison with dose limits, dose constraints and reference levels that relate to potential stochastic effects of whole-body radiation exposure; that is, risks of developing cancer and of hereditary effects (Streffer, 2007). Thus, the primary application of $E$ is in the planning and demonstration of compliance in various situations of exposure of workers and members of the public. The calculation of $E$ can be seen as a three-step process, starting with the calculation of the mean absorbed dose ($D$) to organs and tissues, in gray (Gy; joules per kg). Because radiation types differ in their ability to cause biological effects including cancer per unit of absorbed dose, the second step is to multiply the calculated values of absorbed dose by radiation weighting factors that take account of the greater effectiveness of radiations including alpha particles and neutrons compared to beta particles and gamma rays. The result is termed equivalent dose, with the unit: sievert (Sv). The final step is to sum the equivalent doses to individual organs and tissues, multiplying each by a tissue weighting factor that represents its contribution to total detriment from uniform whole-body irradiation. Thus, effective dose is a weighted average of organ/tissue doses. The intention is that the overall risk should be comparable irrespective of the type and distribution of radiation exposure; $E$, expressed in Sv, is the well-known quantity that is often referred to simply as "dose".

(3) It is important to recognise that while $E$ is a risk-related construct for use in radiation protection, particularly in planning and optimising protection for workers and members of the public, it does not provide estimates of dose to specific individuals (ICRP, 2007a; Dietze and Menzel, 2004; Harrison and Streffer, 2007; Dietze et al., 2009). Rather, absorbed doses to organs and tissues are calculated in mathematical phantoms and used to provide sex-averaged values of effective dose for a "reference person" (ICRP, 2007a, 2009a, 2010a). Furthermore, the associated risks at low doses (< 100 mGy low-LET radiation) or low dose rates (< 5 mGy/h low-LET radiation) are uncertain and the simplified risk-adjustments made using radiation and tissue weighting factors do not fully reflect our scientific understanding of radiation risks. For example, no account is taken of recognised differences between low energy mammography x-rays and Cobalt-60 gamma rays (Hill, 2004). There is evidence that the relative effectiveness of different radiations is dependent on cancer type and there may, for example, be larger differences for liver cancer than for leukaemia (ICRP, 2003b; Harrison and Muirhead, 2003). Tissue weighting factors are age- and sex-averaged values that conceal differences between cancer risk estimates for males and females, and at different ages, dependent on cancer type (ICRP, 2007a; NRC/NAS, 2006; Harrison and Day, 2008;

UNSCEAR, 2013) so that, for example, the risk of thyroid cancer or leukaemia is greater at younger ages at exposure.

(4) *E* and the tissue weighting factors used in its calculation relate to detriment from radiation induced cancer and hereditary effects following low levels of exposure. Detriment is calculated as defined by ICRP and explained in detail in Annex A of *Publication 103* (ICRP, 2007a). The main source of data on cancer risks is the follow-up studies of the Japanese atomic bomb survivors (A-bomb data), used to derive risk coefficients averaged over seven Western and Asian populations with different background cancer rates. The most recent ICRP (2007a) calculations of detriment use cancer incidence data, adjusted for lethality, loss of quality of life and years of life lost. In applying the risk factors obtained from epidemiological studies to exposures at lower doses and dose rates, ICRP applies a Dose and Dose Rate Effectiveness Factor (DDREF) of two for solid cancers and uses a linear-quadratic model for leukaemia. Weighting for hereditary effects is based on estimates of disease occurring in the first two generations, calculated on the basis of animal data. ICRP publishes nominal values of radiation detriment coefficients for an averaged world population, giving values for all ages (members of the public) and adults (workers).

(5) The application of *E* in the control of stochastic effects for protection purposes requires a number of key assumptions (see Chapter 2), principally that:

- a linear-non-threshold (LNT) relationship between dose and risk applies at low doses or low dose rates
- acute low doses are equally as effective as chronic low-dose-rate exposures
- external dose and internal dose from radionuclides deposited in body tissues can be summed, taking account of radiation quality through simple adjustments using radiation weighting factors.

(6) *Publication 103* provides detailed explanation of the purpose and use of the ICRP protection quantities in Section 3 and Annex B (ICRP, 2007a). However, further clarification and guidance have been sought, with identified issues including the following:

1) Confusion between equivalent dose and effective dose expressed in the same units (Sv) when they are not sufficiently carefully distinguished, particularly when considering doses from internal emitters that concentrate in specific organs, e.g. iodine-131 (Gonzalez et al., 2013).
2) The use of equivalent dose in setting limits for the avoidance of tissue reactions in the cases of irradiation of the hands and feet, lens of the eye, and skin; that is, limits set below thresholds for the occurrence of acute damage to organs and tissues. In general, smaller differences in effects per Gy are observed between radiation types in relation to tissue reactions than stochastic effects (ICRP, 2003b).
3) Confusion between operational quantities used to measure exposures to external sources and the protection quantities: specifically between dose equivalent (the measured quantity for external radiation used as an estimate of effective dose) and equivalent dose (an intermediate quantity in the calculation of effective dose).
4) Apparent inconsistencies in the setting of radiation weighting factors, with a simple approach for all low-LET radiations and alpha particles but greater complexity for neutrons, and the use of a different approach using quality factor in calculating operational quantities for neutron exposures.
5) The use of a single set of tissue weighting factors in the calculation of *E* for all age groups and both sexes, despite recognised age-, sex- and population group-related differences in cancer risks.

6) The calculation of *E* for a sex-averaged reference person rather than separately to males and females, and for children as well as adults, and confusion between reference person and representative person.
7) The dose range over which *E* is applicable, particularly in considering higher doses that may occur in accidents that may involve high equivalent doses to individual organs/tissues (e.g. from iodine-131).
8) The apparent conservatism of calculating committed dose from internal emitters; that is, dose integrated over a 50-year period for adults and to age 70 years for children (ICRP, 2007a). For long-lived radionuclides that have long biological retention times in body organs and tissues (e.g. plutonium-239), absorbed dose to organs/tissues is delivered over the whole time period such that only a small proportion is delivered within the year of intake. In contrast, for external sources, and for internally deposited radionuclides with short half-lives and/or short biological retention times, dose is delivered within the year of exposure/intake.
9) The calculation of *E* to the fetus following maternal exposures to internal emitters.
10) The use of *E* to estimate risks to specific individuals, particularly in evaluating exposure of patients undergoing medical procedures.
11) The use of collective effective dose to estimate risks to population groups.

(7) The following section of this report reviews the scientific background to the use of the ICRP protection quantities, considering the key assumptions listed above that underpin their use. Subsequent sections focus on occupational, public and medical exposures and address the issues enumerated above.

# 2. HEALTH EFFECTS

## 2.1. Categories of effect

(8) *Publication 103* (ICRP, 2007a) provides detailed explanations of the judgements made and approaches taken to the quantification of radiation risks for radiological protection purposes. A distinction is made between two major classes of recognised health effects:

- ***Tissue reactions (Deterministic effects)*** occurring above dose thresholds for impairment of organ/tissue function, with severity increasing with increasing dose.
- ***Cancers and heritable diseases (Stochastic effects)*** assumed to occur with increasing probability (but not severity) with increasing dose, with no threshold below which there is no risk.

## 2.2. Tissue reactions (Deterministic effects)

(9) *Publication 103* (ICRP, 2007a) made no changes to previously recommended dose limits for tissue reactions in relation to planned exposure situations, set in terms of equivalent dose, of 150 mSv/y for the lens of the eye and 500 mSv for skin and the hands and feet for occupational exposures, and 15 mSv for the lens of the eye and 50 mSv for skin for public exposures. However, there was accumulating evidence that the lens of the eye may be more sensitive to induction of opacities than indicated by earlier data (Worgul et al., 2007; Neriishi et al., 2007). *Publication 118* (ICRP, 2012a) provided a comprehensive review and analysis

of tissue reactions caused by radiation that confirmed the judgements made in *Publication 103* (ICRP, 2007a, Annex A) regarding threshold doses in most cases, but more recent epidemiological data indicated a lower threshold for induction of cataracts of around 0.5 Gy compared with the values given in *Publication 103* (ICRP, 2007a) of 2 Gy for acute exposures and 4-5 Gy for fractionated and protracted exposures. The available data suggested that acute and protracted exposures were similarly effective and were consistent with the assumption of a non-threshold relationship as well as a threshold of around 0.5 Gy (ICRP, 2012a; Bouffler et al., 2015). In response to this evidence, ICRP (2012a) issued a Statement on Tissue Reactions recommending that the equivalent dose limit for the lens of the eye for occupational exposures should be reduced to 20 mSv $y^{-1}$ averaged over 5 years, with dose in any year not exceeding 50 mSv.

(10) The epidemiological studies on which judgements on cataract risk were based relate largely to external exposures to gamma rays (Ainsbury et al., 2009; ICRP, 2012a) and in general there is limited information available that can be used to compare the effectiveness of radiations of different qualities in causing tissue reactions. However, the available data indicate that differences between radiation types (e.g. alpha particles and neutrons relative to gamma rays) in their effectiveness per Gy in causing tissue reactions are smaller than differences in their effectiveness in relation to cancer induction (ICRP, 1990, 2003b). It can and has been argued, therefore, that use of equivalent dose limits to prevent tissue reactions is overly conservative and that specific lower radiation weighting factors should be applied. While it is important to recognise this conservatism, it was concluded that this is not of great practical concern in most cases and the complexity of introducing further quantities with different radiation weighting factors was not warranted. A distinction should be drawn here between reasonable conservatism as applied to the use of protection quantities to set limits to prevent tissue reactions in planned exposure situations and scientific judgements of the likelihood of observable effects in specific circumstances. For example, it would not be appropriate to use equivalent dose in the assessment of possible acute effects of an ingested alpha particle emitting radionuclide (e.g., polonium-210).

(11) Although equivalent dose can and currently is used to specify limits relating to tissue reactions, absorbed dose (Gy) is the preferable quantity, drawing a clear distinction between limits applying to tissue reactions, set in absorbed dose (Gy), and those applying to stochastic effects, set in effective dose (Sv). The limits for the lens of the eye, skin and hands and feet are relevant mainly to circumstances of exposure to penetrating low LET radiations. However, exposures to neutron and other high LET radiations may require consideration in some situations and it may then be necessary to take account of increased effectiveness per Gy (ICRP, 1990, 2003b).

(12) *Publication 118* (ICRP, 2012a) proposed a threshold dose of 0.5 Gy for radiation-induced circulatory disease and the ICRP Statement on Tissue Reactions (ICRP, 2012a) drew attention to the need for medical practitioners to be aware since doses to patients of this magnitude could be reached during some complex interventional procedures. The meta-analysis of epidemiological data by Little et al. (2012) suggested that a linear-non-threshold (LNT) dose-response relationship could be applied, resulting in risks at low doses/dose rates of a similar magnitude to those inferred for cancer at low doses/dose rates. ICRP will continue to review scientific developments that inform judgements on whether circulatory disease should be included as a component of low dose/dose-rate detriment, but the current view is that different mechanisms of damage are likely to predominate at high and low doses and further mechanistic understanding is required to determine whether stochastic processes are involved in the development of radiation-induced circulatory disease (Hendry, 2015).

## 2.3. Cancers and hereditary diseases (Stochastic effects)

(13) The main stochastic effect of radiation is cancer, with the principal source of information on risk being the epidemiological studies of the Japanese survivors of the atomic bombings at Hiroshima and Nagasaki, although with important information also coming from other studies (ICRP, 2007a). In general, the epidemiological data show a linear dose-response relationship between cancer rates and absorbed dose from gamma rays from around 100 mGy to a few Gy. Attempts are being made to extend observations to lower doses/dose rates, notably studies on large worker cohorts (Muirhead et al., 2009; Haylock et al., 2016; Boice, 2015; Richardson et al., 2015; Leuraud et al., 2015) and studies of children receiving CT scans (Pearce et al., 2012; Mathews et al., 2013; Huang et al., 2014). The CT studies reported statistically significant elevation of cancer rates at doses of a few tens of mSv. However, caution has been advised in the interpretation of these studies (Boice, 2015). A number of problems were identified including lack of information on the reasons for the scans and lack of individual dose reconstruction. It is considered that the patients may well have had underlying conditions that prompted their CT examinations, an example of so-called reverse causation (UNSCEAR, 2013; Walsh et al., 2013, 2014). It will be important that future studies are rigorously controlled to avoid confounding.

(14) A number of assumptions and judgements are made in quantifying low dose/dose-rate cancer risks (ICRP, 2007a). In applying the risk estimates derived from the A-bomb survivor data, a Dose and Dose Rate Effectiveness Factor (DDREF) of two is applied to solid cancers. Epidemiology provides limited evidence of DDREF for solid cancer in humans, although analyses continue (Rühm et al., 2016; Shore et al., 2017), but animal and *in vitro* data indicate curvilinear dose response relationships that support the use of a DDREF. For leukaemia, the A-bomb survivor data are consistent with the use of a linear-quadratic dose response relationship. Having obtained risk estimates for exposures at low doses of a few tens of mGy, a LNT dose-response relationship is assumed. It is the consensus view that for radiological protection purposes this LNT dose-response assumption represents a prudent interpretation of current evidence including mechanistic understanding of radiation-induced cancer at low doses and dose rates (Preston, 2003, 2007; ICRP, 2007a; UNSCEAR, 2012b). Nevertheless, this assumption continues to be controversial, with arguments for supra-linear low dose responses and for thresholds and/or hormetic effects.

(15) The LNT dose-response assumption underpins the use of effective dose as a protection quantity, allowing the addition of external and internal doses of different magnitudes, with different temporal and spatial patterns of delivery. However, it should be recognised that while low doses may be measured or estimated with reasonable reliability, the associated cancer risk is uncertain, and increasingly uncertain as dose decreases.

(16) *Publication 103* (ICRP, 2007a) notes that there is no direct evidence from human epidemiological studies of deleterious heritable effects of radiation but considers the inclusion of heritable risk in overall stochastic risks to be a prudent interpretation of good evidence of heritable effects in experimental animals. Following a detailed analysis by ICRP (2007a) and UNSCEAR (2001), ICRP has applied estimates of heritable risk over two generations in calculations of radiation detriment.

## 2.4. Nominal risk coefficients and Detriment

(17) Annex A of *Publication 103* (ICRP, 2007a) provides a detailed explanation of the methodology applied to the calculation of nominal risk coefficients for radiation-induced

stochastic health effects and associated values of detriment. Nominal risk coefficients are averaged across populations, all ages and both sexes to provide values that can be used as a basis for international protection standards. These risk coefficients are not intended for use in estimating risks to specific individuals. Detriment is a concept used to quantify the harmful effects of radiation at low doses, taking account of the severity of disease in terms of lethality, quality of life and years of life lost. The following summary of the methodological steps in the calculation of detriment is closely based on that provided in Annex A of *Publication 103*:

a) *Determination of lifetime cancer incidence risk estimates for radiation-associated cancers*: For 14 organs or tissues, male and female lifetime excess cancer risks were estimated using both Excess Relative Risk (ERR) and Excess Absolute Risk (EAR) models, largely using analyses of follow-up data for the Japanese A-bomb survivors.

b) *Application of a Dose and Dose Rate Effectiveness Factor (DDREF)*: The lifetime risk estimates were adjusted downward by a factor of two to account for a DDREF except for leukaemia, where the linear-quadratic model for risk already accounts for a reduction in risk per unit dose at low doses.

c) *Transferal of risk estimates across populations*: To estimate radiation risk for each cancer site, a weighting of the ERR and EAR lifetime risk estimates was established that was considered to provide a reasonable basis for generalizing across populations with different baseline risks; for example ERR:EAR weights of 0:100% were assigned for breast, 100:0% for thyroid, 30:70% for lung, and 50:50% for others.

d) *Determination of nominal risk coefficients*: These weighted risk estimates, when applied to and averaged across seven Asian and Western populations and between sexes, provided the nominal risk coefficients given in Table 2.1. The risk coefficients represent averages across selected Asian (Shanghai, Osaka, Hiroshima and Nagasaki) and Euro-American (Sweden, United Kingdom, US SEER) populations.

e) *Adjustment for lethality*: The lifetime risks for respective cancer sites, which were based on excess incident cancers, were converted to fatal cancer risks by multiplying them by their lethality fractions as derived from available cancer survival data.

f) *Adjustment for quality of life*: A further adjustment was applied to account for the morbidity and suffering associated with non-fatal cancers.

g) *Adjustment for years of life lost*: Since the age distributions of types of cancers differ, the years of life lost vary according to cancer type. A weighting factor, relative to the average number of years of life lost due to all solid cancers, was applied to reflect this difference. The result of these calculations was the cancer detriment values shown in Table 2.1.

h) *Inclusion of risks and detriment from heritable effects*: A detailed analysis of laboratory animal data, together with current understanding of heritable effects in humans, led to the conclusion that risk should be defined for the first two generations rather than to equilibrium as done in *Publication 60* (ICRP, 1991). Adjustments were made to risk estimates to provide detriment values, shown in Table 2.1.

i) *Calculation of relative detriment*: Normalising all detriment values to sum to unity gives the values of relative radiation detriment shown in Table 2.1 and used as a basis for specifying tissues weighting factors (see Section 2.5).

(18) Table 2.2 summarises the detriment adjusted risk coefficients derived in *Publication 103* (ICRP, 2007a) and compares them with the values used in *Publication 60* (ICRP, 1991b). The *Publication 103* values for cancer risks are based on considerably improved epidemiological analyses and use of incidence rather than mortality data. The lower values for heritable effects are considered a more scientifically defensible interpretation of the available experimental data – consideration of 5-10 generations instead of two would not materially affect judgements on risk coefficients. While the cancer risk data used to derive the nominal risk coefficients relate almost exclusively to external exposures to gamma rays, the overall population values are expressed in effective dose, Sv, and taken to apply to all radiation exposures (see Section 2.7 and Chapter 3).

Table 2.1. Summary of *Publication 103* Nominal Cancer Risks and Detriment for uniform whole-body exposure to gamma rays.

a) Whole population

| Tissue | Nominal Risk Coefficient (cases per 10,000 persons per Gy)* | Lethality fraction | Nominal risk adjusted for lethality and quality of life* | Relative cancer free life lost | Detriment (relating to column 1) | Relative detriment+ |
|---|---|---|---|---|---|---|
| Oesophagus | 15 | 0.93 | 15.1 | 0.87 | 13.1 | 0.023 |
| Stomach | 79 | 0.83 | 77.0 | 0.88 | 67.7 | 0.118 |
| Colon | 65 | 0.48 | 49.4 | 0.97 | 47.9 | 0.083 |
| Liver | 30 | 0.95 | 30.2 | 0.88 | 26.6 | 0.046 |
| Lung | 114 | 0.89 | 112.9 | 0.80 | 90.3 | 0.157 |
| Bone surface | 7 | 0.45 | 5.1 | 1.00 | 5.1 | 0.009 |
| Skin | 1000 | 0.002 | 4.0 | 1.00 | 4.0 | 0.007 |
| Breast | 112 | 0.29 | 61.9 | 1.29 | 79.8 | 0.139 |
| Ovary | 11 | 0.57 | 8.8 | 1.12 | 9.9 | 0.017 |
| Bladder | 43 | 0.29 | 23.5 | 0.71 | 16.7 | 0.029 |
| Thyroid | 33 | 0.07 | 9.8 | 1.29 | 12.7 | 0.022 |
| Bone Marrow | 42 | 0.67 | 37.7 | 1.63 | 61.5 | 0.107 |
| Other Solid | 144 | 0.49 | 110.2 | 1.03 | 113.5 | 0.198 |
| Gonads (Hereditary) | 20 | 0.80 | 19.3 | 1.32 | 25.4 | 0.044 |
| **Total** | ***1715*** | | ***565*** | | ***574*** | ***1.000*** |

b) Working age population (18-64 years)

| Tissue | Nominal Risk Coefficient (cases per 10,000 persons per Gy)* | Lethality fraction | Nominal risk adjusted for lethality and quality of life* | Relative cancer free life lost | Detriment (relating to column 1) | Relative detriment+ |
|---|---|---|---|---|---|---|
| Oesophagus | 16 | 0.93 | 16 | 0.91 | 14.2 | 0.034 |
| Stomach | 60 | 0.83 | 58 | 0.89 | 51.8 | 0.123 |
| Colon | 50 | 0.48 | 38 | 1.13 | 43.0 | 0.102 |
| Liver | 21 | 0.95 | 21 | 0.93 | 19.7 | 0.047 |
| Lung | 127 | 0.89 | 126 | 0.96 | 120.7 | 0.286 |
| Bone surface | 5 | 0.45 | 3 | 1.00 | 3.4 | 0.008 |
| Skin | 670 | 0.002 | 3 | 1.00 | 2.7 | 0.006 |
| Breast | 49 | 0.29 | 27 | 1.20 | 32.6 | 0.077 |
| Ovary | 7 | 0.57 | 6 | 1.16 | 6.6 | 0.016 |
| Bladder | 42 | 0.29 | 23 | 0.85 | 19.3 | 0.046 |
| Thyroid | 9 | 0.07 | 3 | 1.19 | 3.4 | 0.008 |
| Bone Marrow | 23 | 0.67 | 20 | 1.17 | 23.9 | 0.057 |
| Other Solid | 88 | 0.49 | 67 | 0.97 | 65.4 | 0.155 |
| Gonads (Hereditary) | 12 | 0.80 | 12 | 1.32 | 15.3 | 0.036 |
| **Total** | ***1179*** | | ***423*** | | ***422*** | ***1.000*** |

* Risk coefficients are cases per 10,000 persons per Gy absorbed dose from uniform whole-body gamma ray exposures.

+ The values given should not be taken to imply undue precision but are presented to 3 significant figures to facilitate the traceability of the calculations made and choice of tissue weighting factors.

Table 2.2. Detriment-adjusted nominal risk coefficients per effective dose ($10^{-2}$ $Sv^{-1}$).

| Exposed population | Cancer | | Heritable effects | | Total | |
|---|---|---|---|---|---|---|
| | ICRP103 | ICRP60 | ICRP103 | ICRP60 | ICRP103 | ICRP 60 |
| **Whole** | 5.5 | 6.0 | 0.2 | 1.3 | 5.7 | 7.3 |
| **Adult** | 4.1 | 4.8 | 0.1 | 0.8 | 4.2 | 5.6 |

## 2.5. Tissue weighting factors

(19) Table 2.3 shows the tissue weighting factors, based on the relative detriment values shown in Table 2.1, as used in *Publication 103* (ICRP, 2007a) in the calculation of effective dose. As explained in the Introduction and detailed in Section 2.2, effective dose is calculated as the sum of equivalent doses to individual organs and tissues multiplied by their tissue weighting factors, thus making allowance for their contribution to total detriment. Effective dose is a weighted average of equivalent doses to organs and tissues, used as a measure of whole-body dose. The intention of this procedure is that the overall risk per unit effective dose will approximate the values shown in Table 2.2, irrespective of the contributions made by doses to individual organs and tissues. Because of the uncertainties associated with the calculations of the nominal risk coefficients and detriment values shown in Table 2.1, and their application to low dose/dose-rate exposures to external and internal sources, the tissue weighting factors shown in Table 2.3 are simplified and rounded to avoid any spurious impression of accuracy. Furthermore, a single set of values is used for all ages and both sexes. The tissue weighting factor of 0.08 for gonads applies to detriment from cancer and heritable effects. A tissue weighting factor of 0.01 was applied to salivary gland and brain despite risks not being specifically quantifiable as it was judged that they may be more sensitive to radiation-induced cancer than other tissues constituting the "remainder" group.

Table 2.3. *Publication 103* tissue weighting factors.

| Tissue | $w_T$ | $\Sigma\ w_T$ |
|---|---|---|
| Bone-marrow, Colon, Lung, Stomach, Breast, Remainder Tissues* | 0.12 | 0.72 |
| Gonads | 0.08 | 0.08 |
| Bladder, Oesophagus, Liver, Thyroid | 0.04 | 0.16 |
| Bone surface, Brain, Salivary glands, Skin | 0.01 | 0.04 |

*Remainder Tissues: Mean of doses to Adrenals, Extrathoracic (ET) region, Gall bladder, Heart, Kidneys, Lymphatic nodes, Muscle, Oral mucosa, Pancreas, Prostate (♂), Small intestine, Spleen, Thymus, Uterus/cervix (♀).

## 2.6. Age- and sex- specific cancer risks

(20) The data provided in *Publication 103* (ICRP, 2007a) for the calculation of nominal risk coefficients, relative detriment and tissue weighting factors, for use in the calculation and application of effective dose, do not consider age, sex and population related differences in risk, except for the distinction between the whole population (0–84 years at exposure) and the working age population (18-64 years at exposure). Risks for the working age population are somewhat smaller because risks are generally greater at younger ages. *Publication 103* does present, but does not use, separate risk factors for males and females, averaged over all ages, showing greater nominal risk coefficients and detriment values for females by a few tens of percent.

(21) Wall et al. (2011) examined the variation of lifetime cancer risk with cancer type, sex and age at exposure. Their approach was slightly different from that used in *Publication 103* (ICRP, 2007a), but their results illustrated variations of nominal risks with age and sex. The cumulative risk of cancer incidence per unit organ/tissue dose (Gy) up to age 100 years was calculated separately for males and females and for category of age at exposure (10 age categories of 10 years, from 0-9 years to 90-99 years), for 11 different cancer types (female breast, lung, stomach, colon, bladder, liver, thyroid, oesophagus, ovary, leukaemia, and other solid cancer sites). Risk models were derived from the A-bomb survivor cohort (Preston et al. 2007), using *Publication 103* methodology. To define baseline incidence rates, Wall et al. (2011) used *Publication 103* values for a Euro-American composite population. The values in Table 2.4 are calculated as Lifetime Attributable Risk (LAR) rather than Risk of Exposure-Induced InCidence (REIC) as in Wall et al (2011) for greater consistency with the methodology used in Publication 103. Use of LAR rather than REIC results in somewhat greater risk estimates for exposure at younger ages.

(22) Table 2.5 shows results of identical calculations but with baseline incidence rates from the ICRP Asian composite populations. Comparison of these data shows the same pattern in both populations, with overall risks compared to those in the 30–39 years at exposure group being about two to three times higher in the youngest group (0–9 years at exposure) and about two to three times lower by age 60–69 years at exposure. However, the data also show substantial differences between cancer types, as illustrated in Fig. 2.1 for lung and thyroid cancer, with some differences between the two composite populations in the age at exposure dependence of risk for individual cancers. Note that these variations with age reflect cumulative lifetime risk, so that reduction of risk with increasing age at exposure reflects mainly the reduction in remaining lifetime after exposure rather than a variation of sensitivity with age at exposure. It should be recognised that the values given in Table 2.4 and 2.5 are the results of modelling, based on a set of assumptions that are all subject to uncertainties. However, while it is important to recognise the considerable uncertainties associated with low dose/dose-rate risk estimates (NCRP, 2012; UNSCEAR, 2012b), the overall conclusions regarding age at exposure-related changes in risk remain valid, with differences between individual cancers. Ogino et al (2016) discuss age- and sex- differences in cancer risks for the various organs, applying ICRP methodology to a Japanese population.

(23) With regard to risks of *in utero* irradiation of the unborn child, *Publication 103* (ICRP, 2007a) refers to the review of *Publication 90* (ICRP, 2003a). The overall conclusion from the limited available data, is that it is reasonable to assume that the overall lifetime risk of cancer from *in utero* irradiation is, at most, a few times that of the population as a whole and the *in utero* risk is judged to be no greater than that following exposures in early childhood.

(24) For the practical implementation of the protection system, it is of considerable utility to be able to set protection criteria that apply to all members of the public or all workers, and it is notable, therefore, that the estimated differences in risk between males and females and between age at exposure groups are not large in comparison with the uncertainties associated with their estimation [see NCRP (2012) and UNSCEAR (2012b) for discussion of uncertainties in risk estimates]. The only distinction made between males and females for protection purposes is the treatment of occupationally exposed females during declared pregnancy when the fetus is regarded as a member of the public for the purposes of dose limitation (ICRP, 2007a). The calculation of doses to the fetus is considered in Section 4.2.

(25) Nominal risk coefficients and detriment values are averaged over sex and age at exposure within the public and worker populations. Tissue weighting factors are chosen as simplified and rounded values relating to age- and sex- averaged relative detriment values (Table 2.1a). However, it is important for the purposes of this report to understand potential differences in risk to different population groups and individuals. Particularly in medical applications but also in other applications, there are situations in which there is a requirement for some understanding of risks associated with particular procedures and better information may be required than that conveyed by nominal risk coefficients.

(26) In addition to age at exposure- and sex- related differences in radiation risk, there are variations in radiation sensitivity between individuals related to genetic differences that are generally not well understood (ICRP, 2007a; AGIR, 2013; Bouffler, 2016). There are good prospects for increased understanding of such differences with advances in genetic typing and testing but with ethical challenges in the application of such information (Bouffler, 2016). However, current information is insufficient to quantify the effect of such differences in terms of individual risk.

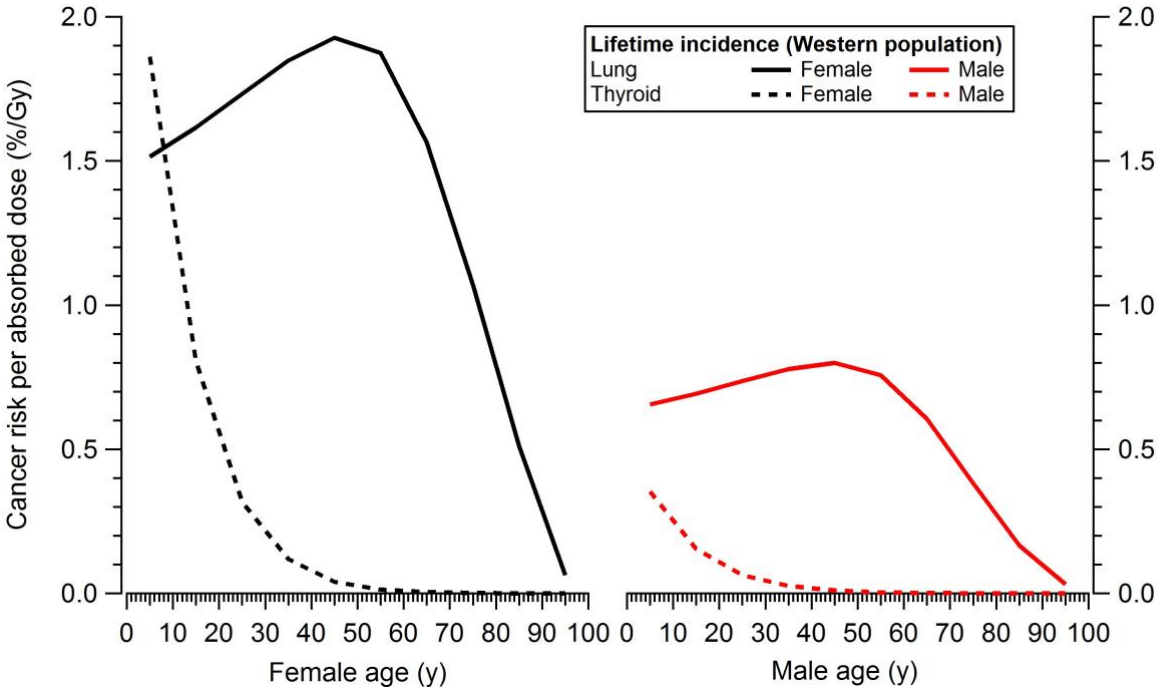


Fig. 2.1. Lifetime attributable risks of cancer incidence per absorbed dose (cases per 100 per Gy; % / Gy) from uniform external exposure to gamma rays for the ICRP (2007a) Euro-American composite population for lung and thyroid cancer (from Table 2.4).

Table 2.4. Lifetime attributable risks of cancer incidence per absorbed dose (cases per 100 per Gy) from uniform external exposure to gamma rays for the ICRP (2007a) Euro-American composite population.

| | Age at exposure (years) | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Organ | 0-9 | 10-19 | 20-29 | 30-39 | 40-49 | 50-59 | 60-69 | 70-79 | 80-89 | 90-99 |
| *Males* | | | | | | | | | | |
| Lung | 0.7 | 0.7 | 0.7 | 0.8 | 0.8 | 0.8 | 0.6 | 0.4 | 0.2 | 0.03 |
| Stomach | 1.0 | 0.8 | 0.6 | 0.4 | 0.3 | 0.2 | 0.1 | 0.05 | 0.02 | 0.0 |
| Colon | 1.6 | 1.3 | 1.1 | 0.8 | 0.6 | 0.4 | 0.2 | 0.1 | 0.04 | 0.0 |
| RBM | 1.3 | 1.3 | 0.8 | 0.7 | 0.7 | 0.4 | 0.3 | 0.1 | 0.07 | 0.02 |
| Bladder | 0.9 | 0.8 | 0.7 | 0.6 | 0.5 | 0.3 | 0.2 | 0.1 | 0.05 | 0.01 |
| Liver | 0.6 | 0.5 | 0.4 | 0.3 | 0.2 | 0.1 | 0.06 | 0.03 | 0.01 | 0.0 |
| Thyroid | 0.4 | 0.2 | 0.06 | 0.03 | 0.01 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Oesophagus | 0.1 | 0.1 | 0.1 | 0.1 | 0.1 | 0.1 | 0.1 | 0.08 | 0.05 | 0.01 |
| Other | 4.9 | 3.2 | 2.4 | 1.4 | 0.9 | 0.5 | 0.3 | 0.1 | 0.03 | 0.0 |
| **All cancers** | **11.5** | **8.8** | **6.8** | **5.0** | **4.0** | **2.9** | **1.9** | **1.0** | **0.4** | **0.08** |
| *Females* | | | | | | | | | | |
| Breast | 6.7 | 4.1 | 2.5 | 1.5 | 0.8 | 0.4 | 0.2 | 0.07 | 0.02 | 0.0 |
| Lung | 1.5 | 1.6 | 1.7 | 1.8 | 1.9 | 1.9 | 1.6 | 1.1 | 0.5 | 0.06 |
| Stomach | 1.7 | 1.3 | 1.0 | 0.7 | 0.5 | 0.3 | 0.2 | 0.1 | 0.05 | 0.0 |
| Colon | 0.8 | 0.7 | 0.5 | 0.4 | 0.3 | 0.2 | 0.1 | 0.08 | 0.03 | 0.0 |
| RBM | 0.5 | 0.5 | 0.5 | 0.4 | 0.5 | 0.3 | 0.2 | 0.1 | 0.04 | 0.01 |
| Bladder | 0.8 | 0.7 | 0.6 | 0.5 | 0.4 | 0.4 | 0.3 | 0.2 | 0.1 | 0.01 |
| Liver | 0.3 | 0.2 | 0.2 | 0.1 | 0.09 | 0.06 | 0.04 | 0.02 | 0.01 | 0.0 |
| Thyroid | 1.9 | 0.8 | 0.3 | 0.1 | 0.04 | 0.01 | 0.0 | 0.0 | 0.0 | 0.0 |
| Oesophagus | 0.1 | 0.1 | 0.1 | 0.1 | 0.1 | 0.2 | 0.2 | 0.2 | 0.2 | 0.03 |
| Ovary | 0.6 | 0.4 | 0.3 | 0.2 | 0.2 | 0.1 | 0.06 | 0.03 | 0.01 | 0.0 |
| Other | 3.7 | 2.5 | 1.7 | 1.2 | 0.8 | 0.5 | 0.3 | 0.1 | 0.05 | 0.0 |
| **All cancers** | **18.5** | **13.0** | **9.4** | **7.1** | **5.7** | **4.4** | **3.2** | **2.1** | **1.0** | **0.1** |

RBM = Red Bone Marrow, the target tissue for leukaemia risk. Risks are calculated using EAR and ERR models and applying a DDREF of 2 for all cancer types other than leukaemia (ERR/EAR of 100/0 for thyroid, 30/70 for lung, 0/100 for breast, 50:50 for all others). Latent periods applied were 2 years for leukaemia and 5 years for solid cancers.

Table 2.5. Lifetime attributable risks of cancer incidence per absorbed dose (cases power 100 per Gy) from uniform external exposure to gamma rays for the ICRP (2007a) Asian composite population.

| | Age at exposure (years) | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Organ | 0-9 | 10-19 | 20-29 | 30-39 | 40-49 | 50-59 | 60-69 | 70-79 | 80-89 | 90-99 |
| *Males* | | | | | | | | | | |
| Lung | 0.7 | 0.8 | 0.8 | 0.8 | 0.9 | 0.8 | 0.7 | 0.4 | 0.2 | 0.04 |
| Stomach | 1.6 | 1.3 | 1.0 | 0.8 | 0.6 | 0.4 | 0.2 | 0.1 | 0.03 | 0.0 |
| Colon | 1.9 | 1.5 | 1.2 | 0.9 | 0.7 | 0.5 | 0.3 | 0.1 | 0.04 | 0.01 |
| RBM | 1.3 | 1.3 | 0.8 | 0.7 | 0.7 | 0.5 | 0.3 | 0.1 | 0.07 | 0.02 |
| Bladder | 0.5 | 0.5 | 0.4 | 0.3 | 0.3 | 0.2 | 0.2 | 0.09 | 0.04 | 0.01 |
| Liver | 1.1 | 0.8 | 0.7 | 0.5 | 0.4 | 0.2 | 0.1 | 0.05 | 0.01 | 0.0 |
| Thyroid | 0.3 | 0.1 | 0.06 | 0.02 | 0.01 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Oesophagus | 0.2 | 0.2 | 0.2 | 0.2 | 0.2 | 0.1 | 0.1 | 0.09 | 0.06 | 0.01 |
| Other | 2.9 | 1.9 | 1.3 | 0.9 | 0.6 | 0.3 | 0.2 | 0.07 | 0.02 | 0.0 |
| **All cancers** | **10.5** | **8.3** | **6.4** | **5.1** | **4.1** | **3.0** | **2.0** | **1.1** | **0.5** | **0.09** |
| *Females* | | | | | | | | | | |
| Breast | 6.8 | 4.1 | 2.5 | 1.5 | 0.8 | 0.4 | 0.2 | 0.06 | 0.02 | 0.0 |
| Lung | 1.4 | 1.4 | 1.5 | 1.6 | 1.7 | 1.6 | 1.4 | 0.9 | 0.5 | 0.09 |
| Stomach | 2.2 | 1.7 | 1.3 | 1.0 | 0.7 | 0.5 | 0.3 | 0.1 | 0.05 | 0.01 |
| Colon | 0.8 | 0.6 | 0.5 | 0.4 | 0.3 | 0.2 | 0.1 | 0.06 | 0.02 | 0.0 |
| RBM | 0.5 | 0.5 | 0.5 | 0.1 | 0.5 | 0.3 | 0.2 | 0.09 | 0.04 | 0.01 |
| Bladder | 0.5 | 0.5 | 0.4 | 0.3 | 0.3 | 0.3 | 0.2 | 0.1 | 0.07 | 0.01 |
| Liver | 0.5 | 0.4 | 0.3 | 0.3 | 0.2 | 0.1 | 0.08 | 0.04 | 0.01 | 0.0 |
| Thyroid | 2.5 | 1.0 | 0.5 | 0.2 | 0.06 | 0.02 | 0.01 | 0.0 | 0.0 | 0.0 |
| Oesophagus | 0.1 | 0.1 | 0.1 | 0.1 | 0.2 | 0.2 | 0.2 | 0.2 | 0.1 | 0.03 |
| Ovary | 0.4 | 0.3 | 0.2 | 0.2 | 0.1 | 0.07 | 0.04 | 0.02 | 0.01 | 0.0 |
| Other | 3.0 | 2.1 | 1.5 | 1.0 | 0.7 | 0.4 | 0.2 | 0.1 | 0.04 | 0.01 |
| **All cancers** | **18.8** | **12.8** | **9.4** | **6.6** | **5.5** | **4.1** | **2.9** | **1.8** | **0.9** | **0.2** |

RBM = Red Bone Marrow, the target tissue for leukaemia risk. Risks are calculated using EAR and ERR models and applying a DDREF of 2 for all cancer types other than leukaemia (ERR/EAR of 100/0 for thyroid, 30/70 for lung, 0/100 for breast, 50:50 for all others). Latent periods applied were 2 years for leukaemia and 5 years for solid cancers.

## 2.7. Risks from alpha particle emitting radionuclides

(27) The epidemiological data used as the basis for the derivation of nominal risk coefficients, detriment values and tissue weighting factors, as discussed above, relate almost entirely to external exposures to gamma rays, principally cancer incidence and mortality data for the Japanese A-bomb survivors (apart from risk of bone cancer; see below). An important question for the implementation of the protection system is the extent to which risk factors derived principally from studies of short-term exposures to penetrating external radiation apply also to protracted irradiation from charged particles, with heterogeneity of exposure between and within organs and tissues. This question is particularly relevant to internal

exposures to alpha particle emitting radionuclides, including radium-224, since alpha particles only travel very short distances (a few tens of micrometres) in tissue.

(28) In relation to the application of external risk factors to internal exposure to alpha particle irradiation, a number of human studies (UNSCEAR, 2000, 2008; WHO, 2001) provide information that has been used to estimate risks of lung, liver, and bone cancer:

- Lung cancer – occupational exposure of underground hard-rock miners to radon-222 and daughters, with consistent data from studies of residential exposure; and occupational exposure of Mayak workers to plutonium-239.
- Liver cancer – patients given intravascular injections of 'Thorotrast', a colloidal thorium oxide preparation ($^{232}$Th is an alpha emitter), as a contrast medium for diagnostic radiography; and occupational exposure of Mayak workers to $^{239}$Pu.
- Bone cancer – occupational exposure of radium dial painters to $^{226}$Ra and $^{228}$Ra; patients given $^{224}$Ra for medical conditions; and occupational exposure of Mayak workers to $^{239}$Pu.

(29) Harrison and Muirhead (2003) compared risk estimates for radiation-induced cancer derived for these exposures to alpha-emitting radionuclides and those derived for the atomic bomb survivors. They showed that, taking account of the greater effectiveness of alpha particles compared to gamma rays by up to a factor of around 20, the human data show consistency between estimates of radiation risk from internal emitters and external radiation. Similar conclusions were reached by Little et al. (2007) in an analysis of epidemiological data for internal emitters and comparison with A-bomb survivor data. Support is also provided by animal and *in vitro* data comparing the effects of different radionuclides and external radiation (UNSCEAR, 2000, 2008; WHO, 2001). However, uncertainties in the dose estimates for internal emitters and in the risk factors should be recognised (Harrison and Muirhead, 2003; ICRP, 2007a; Harrison and Day, 2008).

(30) In the case of bone cancer, the A-bomb survivor data were less informative in the 1990s than epidemiological studies of the effects of internally deposited $^{224}$Ra. The risk factor for bone cancer in Table 2.1 was based on *Publication 60* (ICRP, 1991b) considerations of the $^{224}$Ra data. In this case, the risk per Gy was divided by an assumed value for the relative biological effectiveness (RBE) of alpha particles compared with gamma rays of 20 to obtain an estimate of risk per Gy of low LET radiation.

(31) An excess of leukaemia has been reported in Thorotrast-treated patients, and quantitative estimates of $^{239}$Pu induced lung cancer have been derived for Russian workers at the Mayak nuclear site (WHO, 2001; Harrison and Muirhead, 2003; Gilbert et al., 2004, 2013). Comparison of leukaemia risks in Thorotrast patients and A-bomb survivors suggested a low alpha particle RBE for this disease of around 1 – 2. Animal data provide some support for a low alpha particle RBE for leukaemia induction (Breckon and Cox, 1990; Ellender et al., 2001; ICRP, 2003b). Marsh et al. (2014) undertook a detailed analysis of lung cancer risks per Gy from inhaled $^{222}$Rn progeny and $^{239}$Pu, focussing on the results of a recent epidemiological study of French uranium miners (Rage et al., 2012) and an epidemiological study of lung cancer in Mayak workers which applied the most recently published Mayak Worker Dosimetry System (MWDS, 2008; Khokhryakov et al., 2013). While the alpha particle dose from radon progeny is delivered predominantly in the airways with only a small proportion delivered to the alveolar regions, the opposite is the case for alpha particle decay of $^{239}$Pu. Marsh et al. (2014) compared the published values of ERR from these studies and also calculated values of lifetime excess absolute risk, comparing results with values based on the A-bomb survivor data. Results showed similar values for $^{222}$Rn progeny and $^{239}$Pu despite the very different dose distributions within the lungs, consistent with central RBE values of around 10 – 20 in each case.

(32) It can be concluded that the available epidemiological data, supported by animal data, indicate that it is reasonable for protection purposes to assume equivalence of risk per unit dose, once simple adjustment are made to account for RBE, between short duration exposures to external penetrating low LET gamma rays and protracted internal exposures to alpha particle emitting radionuclides, for which tissue doses will be substantially more heterogeneous.

# 3. DOSIMETRY

## 3.1. Dose quantities

(33) The procedure for the calculation of effective dose adopted by ICRP is to use *absorbed dose* as the fndamental physical quantity; to average it over specified organs and tissues; to apply suitably chosen radiation weighting factors to take account of differences in biological effectiveness of different radiations to give the quantity *equivalent dose*; and to consider differences in sensitivities of organs and tissues to stochastic health effects and their contribution to total detriment. Values of the equivalent dose to organs and tissues are weighted using tissue weighting factors that provide a simplified representation of relative detriment and the weighted equivalent doses are then summed to give the *effective dose*. This quantity is used to sum exposures to radiation from incorporated radionuclides and to external radiation fields. The description below is based on that provided in Section 4 and Annex B of *Publication 103* (ICRP, 2007a).

## 3.2. Absorbed dose

(34) In radiation biology, clinical radiology, and radiological protection, the absorbed dose ($D$) is the basic physical dose quantity and is used for all types of ionising radiation and any irradiation geometry. It is defined as the quotient of mean energy ($\mathrm{d}\bar{\varepsilon}$) imparted by ionising radiation in a volume element and the mass ($\mathrm{d}m$) of the matter in that volume, that is

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

(35) The SI unit of absorbed dose is J kg$^{-1}$ and its special name is gray (Gy). Absorbed dose is derived from the mean value of the stochastic quantity of energy imparted, ε, and does not reflect the random fluctuations of the interaction events in tissue. While it is defined at any point in matter, its value is obtained as an average over a mass element d$m$ and hence over many atoms or molecules of matter. Absorbed dose is a measurable quantity and primary standards exist to determine its value. The definition of absorbed dose has the scientific rigour required for a basic physical quantity.

(36) When using the quantity absorbed dose in radiological protection, doses are averaged over tissue volumes. It is assumed that for low doses, the mean value of absorbed dose averaged over a specific organ or tissue can be correlated with radiation detriment for stochastic effects in that tissue with an accuracy sufficient for the purposes of radiological protection. The averaging of absorbed dose is carried out over the volume of a specified organ (e.g. liver) or tissue (e.g. red bone marrow) or the sensitive region of a tissue (e.g. endosteal surfaces of the skeleton).

## 3.3. Equivalent dose

(37) The definition of the protection quantity, equivalent dose, is based on the average absorbed dose ($D_{T,R}$) due to radiations of type *R* in the volume of a specified organ or tissue *T*. The radiation types *R* are given by the type and energy of radiation either incident on the body or emitted by radionuclides residing within it. The protection quantity *equivalent dose* in an organ or tissue ($H_T$) is then defined by

$$H_T = \sum_R w_R D_{T,R}$$

where $w_R$ is the radiation weighting factor for radiation type *R*. The sum is performed over all types of radiations involved. The unit of equivalent dose is J kg$^{-1}$ and has the special name sievert (Sv).

(38) Radiation weighting in the definition of radiological protection quantities was originally related to the radiation quality factor (Q) as a function of LET and denoted as *L* in the *Q*(*L*) function of *Publication 26* (ICRP, 1977). In *Publication 60* (ICRP, 1991b) the method of radiation weighting for effective dose was changed, with the selection of a set of radiation weighting factors ($w_R$). The values of $w_R$ were selected largely on the basis of measurements of relative biological effectiveness (RBE) of the different radiations. RBE values are experimentally determined and are the ratio of doses of a test radiation and a low LET reference radiation that produce the same level of observed effect. A range of RBE values are observed depending on the biological end-point studies and also on the reference radiation: common references are high energy x-rays above about 200 kV or $^{60}$Co or $^{137}$Cs gamma radiation. Table 3.1 shows the $w_R$ values adopted in *Publication 103* (ICRP, 2007a).

Table 3.1. *Publication 103* radiation weighting factors.

| Radiation type | Radiation weighting factor, $w_R$ |
|---|---|
| Photons | 1 |
| Electrons and muons | 1 |
| Protons and charged pions | 2 |
| Alpha particles, fission fragments, heavy ions | 20 |
| Neutrons | A continuous function of neutron energy (Fig. 3.1) |

All values relate to the radiation incident on the body or, for internal radiation sources, emitted from the source.

(39) The use of $w_R$ = 1 for all emissions of photons, electrons and muons does not imply that there are no differences in biological effectiveness at different energies. This simple approach is considered sufficient for the intended applications of effective dose. For retrospective risk assessments, more detailed information on the radiation field and appropriate RBE values may need to be considered if relevant data are available, but such considerations go beyond the intended application of effective dose. Heterogeneity of the radiation dose within cells, as can occur with Auger emitters incorporated into DNA, for example, may also require specific analysis in risk assessments.

(40) The radiation weighting factor for neutrons reflects the relative biological effectiveness of neutrons following external exposure. The biological effectiveness of neutrons incident on the human body is strongly dependent on neutron energy (see *Publication 103*, Annex B). The energy function shown in Fig. 3.1 takes account of the large contribution of secondary photons to the absorbed dose in the human body at lower energies, and the decrease of $w_R$ at neutron energies above 50 MeV as, for physical reasons, RBE values are assumed to converge with those for protons.

(41) Protons in cosmic radiation fields or fields near high-energy particle accelerators are mainly of very high-energy and it is considered appropriate to adopt a single $w_R$ value for protons of all energies that is mainly based on radiobiological data for high-energy protons above 10 MeV. Pions are negatively or positively charged or neutral particles encountered in radiation fields resulting from interactions of the primary cosmic rays with nuclei at high altitudes in the atmosphere. These particles contribute to exposures in aircraft and are also found as part of the complex radiation fields behind shielding of high-energy particle accelerators.

(42) Alpha particle exposures occur as a result of the inhalation or ingestion of alpha-emitting radionuclides. Information from experimental and epidemiological studies indicate that RBE values differ dependent on the organ and cancer type being considered. The distribution of radionuclides in organs and tissues and the estimation of dose is complex and associated with substantial uncertainties, contributing to observations of a broad range of RBE values (see Section 2.1; ICRP, 2003b, 2007a). A single $w_R$ value of 20 is used for alpha particle irradiation and the same value is used for fission fragments, and also as a conservative value for heavy ions.

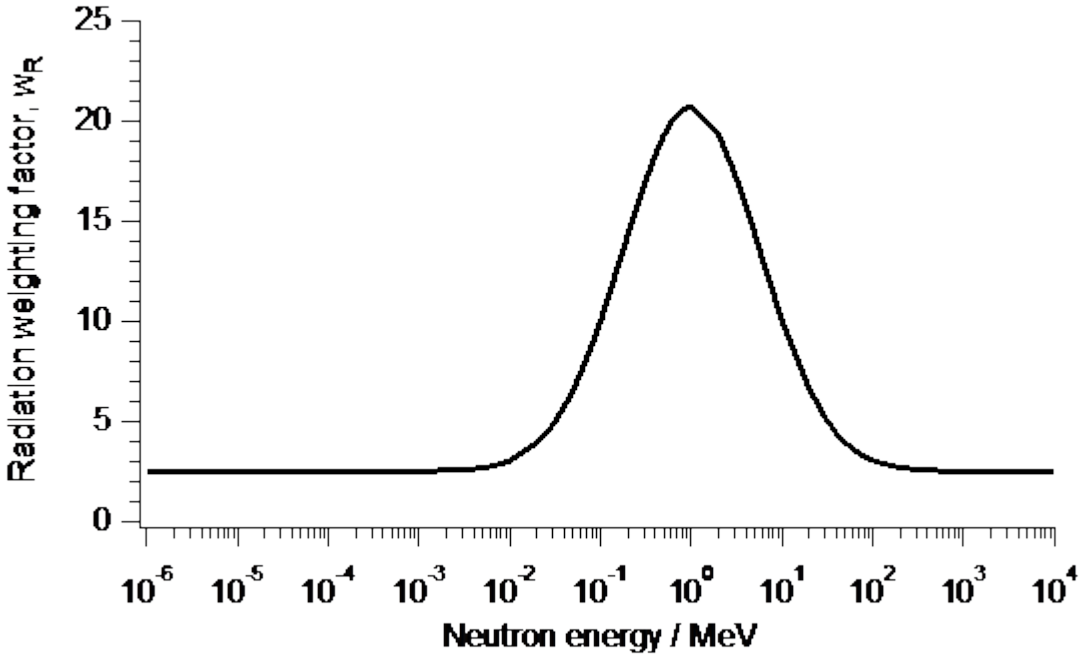


Fig. 3.1. Energy function for radiation weighting factor, $w_R$, for neutrons.

(43) It has been argued [e.g. Thomas and Edwards (2003)] that the ICRP treatment of radiation weighting for the calculation of effective dose exhibits inconsistencies, is unnecessarily complex, and over-interprets the available biological data (ICRP, 2003b). For protection purposes, it would arguably be sufficient to use two $w_R$ values: 1 for low LET radiations and 10 for high LET radiations, including the high LET component of neutron

dose. Such a simplified scheme would not obviate the need for more complex calculations in situations that require the use of best available data to estimate dose and risk as accurately as possible – an example is the calculation of doses and estimation of risk to astronauts which can be substantial and involves consideration of exposures to complex radiation fields (ICRP, 2013). However, the current system of radiation weighting as specified in *Publication 103* (ICRP, 2007a) has the advantage of providing continuity of approach, and an important consideration is the relationship between effective dose and measurements made using operational quantities (see below).

(44) Equivalent dose can be seen as an intermediate step in the calculation of effective dose. Dose limits, dose constraints and reference levels in relation to stochastic health effects are set in terms of effective dose. Equivalent dose has been used to specify limits for the avoidance of tissue reactions but, as discussed in Section 2.2, these will be more appropriately set in terms of absorbed dose (Gy). Communication difficulties have arisen in situations where equivalent dose and effective dose expressed in the same units (Sv) have not been adequately distinguished, for example in explaining doses for intakes of iodine-131 for which the equivalent dose to the thyroid is more than twenty times the effective dose (Gonzalez et al., 2013). There is also scope for confusion between equivalent dose and the operational quantity, dose equivalent (Sv). Such difficulties will be avoided if organ and tissue doses are referred to in terms of absorbed dose, if necessary specifying low and high LET components. For example, an intake of iodine-131 might result in an effective dose of 10 mSv, with a thyroid dose of 240 mGy (low LET). The use of equivalent dose as a distinct protection quantity is not required.

## 3.4. Effective dose

(45) The effective dose, $E$, as introduced in *Publication 60* (ICRP, 1991b) and applied in *Publication 103* (ICRP, 2007a) is defined as:

$$E = \sum_{\mathrm{T}} w_{\mathrm{T}} \sum_{\mathrm{R}} w_{\mathrm{R}} D_{\mathrm{T,R}}$$
$$= \sum_{\mathrm{T}} w_{\mathrm{T}} H_{\mathrm{T}}$$

where $w_{\mathrm{T}}$ is the tissue weighting factor for tissue, T and $\Sigma\, w_{\mathrm{T}} = 1$. The sum is performed over all organs and tissues of the human body for which specific radiation detriment values can be calculated (Table 2.1) and tissue weighting factors can be specified (Table 2.3). Mathematically, effective dose is a weighted average of equivalent doses to organs / tissues. As outlined above, the $w_{\mathrm{T}}$ values are chosen to represent the contributions of individual organs and tissues to overall radiation detriment from stochastic effects, averaged over all ages and both sexes. The $w_{\mathrm{T}}$ values are rounded and have only four different numerical values (Table 2.3), despite the greater differentiation possible on the basis of relative detriment (Table 2.1), to avoid the impression of unwarranted accuracy in relation to effects of low dose radiation.

(46) The unit of effective dose is J $kg^{-1}$ with the special name sievert (Sv). It applies over the dose range of concern for the induction of stochastic effects and in this connection, questions have arisen regarding the upper limit to the applicability of effective dose. *Publication 103* (ICRP, 2007a) refers to setting of reference levels in relation to emergency planning and management in the range of 20-100 mSv effective dose. In principle, there is no reason why effective doses should not be used as a quantity at doses above 100 mSv: for

example, as might be required as a short-term relaxation of worker doses in order to control an accident situation. However, two factors need to be taken into consideration at higher doses:

1) The potential for the occurrence of tissue reactions should be considered and avoided. For effective doses up to a few hundreds of mSv and for which irradiation is reasonably uniform throughout the body, severe tissue reactions would not be expected to occur. However, if there was a significant contribution to the effective dose from radionuclides concentrated in particular organs (e.g. iodine-131 in the thyroid, inhaled insoluble radionuclides in the lung), tissue damage could occur. Notably, for $^{131}$I, for example, an effective dose of 250 mSv would correspond to an absorbed dose to the thyroid of > 6 Gy.
2) A secondary consideration is that for doses in excess of 100 mSv (or more precisely doses to organs and tissues > 100 mGy low LET radiation) delivered at high dose rate, the DDREF of two applied in determining solid cancer risk at low doses/dose rates will not apply, so that risks may be somewhat greater than might be assumed on the basis of *Publication 103* (ICRP, 2007a) nominal risk coefficients.

## 3.5. Dose coefficients

(47) For internal exposures, ICRP has published dose coefficients (Sv Bq$^{-1}$) for intakes of individual radionuclides by workers and members of the public, giving both equivalent doses to organs and tissues, and effective dose for adults and children (ICRP, 1979, 1980, 1981, 1987, 1989, 1993, 1994a,b, 1995a,b, 1996a, 1999, 2002a). Dose coefficients have also been provided for radiopharmaceutical doses to patients (ICRP, 1987, 1998, 2008). For consideration in relation to occupational and environmental exposures, doses to the fetus following maternal intakes have been calculated and also doses to infants from radionuclides transferred to breast-milk (ICRP, 2001, 2004). In each case, biokinetic models are provided, used to describe the behaviour of radionuclides in the body and calculate energy deposition and absorbed dose in target organs (for which doses contribute to the calculation of effective dose) for transformations occurring in source organs (sites of radionuclide retention).

(48) *Publication 119* (ICRP, 2012b) provides a compilation of internal dose coefficients for workers and members of the public, calculated according to *Publication 60* methodology (ICRP, 1991b). It also includes conversion coefficients for occupational exposures to external radiation, abstracted from *Publication 74* (ICRP, 1996c), calculating the protection quantities from estimates of absorbed dose per unit air kerma or fluence, assuming whole-body irradiation by mono-energetic photons, electrons and neutrons in a number of idealised standard exposure geometries. *Publication 128* (ICRP, 2015a) provides a compilation of dose coefficients for radiopharmaceuticals calculated using *Publication 60* (ICRP, 1991b) methodology.

(49) Revisions of ICRP recommendations invariably require recalculation of dose coefficients because changes are made to the radiation and tissue weighting factors used in the calculation of equivalent and effective dose. In addition, improvements to the models used to calculate doses also lead to revised values. Work is currently in progress to provide replacement dose coefficients based on the 2007 Recommendations (ICRP, 2007a), incorporating a number of important methodological improvements, including revised and updated biokinetic and dosimetric models. It should be noted, however, that while dose coefficients are revised following each new set of ICRP recommendations, these changes should be regarded as evolution and improvement as scientific knowledge improves rather

than fundamental change, and there should be no general requirement for the recalculation of previous dose assessments.

(50) Computational phantoms (or mathematical models) of the human body are used to model energy deposition in organs and tissues from internal and external radiation exposures. These phantoms have generally been based on mathematical expressions representing geometric shapes that provide reasonable approximations to the shapes of body structures. This type of phantom was developed at the US Oak Ridge National Laboratory (Cristy, 1980; Cristy and Eckerman, 1987) for the Medical Internal Radiation Dose (MIRD) Committee of the Society of Nuclear Medicine. From the original adult MIRD phantom, several paediatric phantoms were developed to represent infants and children of various ages (Cristy, 1980). MIRD type models were developed by Stabin et al. (1995) for three stages of pregnancy. These models have been used in the calculation of ICRP dose coefficients.

(51) More recently, a number of groups have developed so-called tomographic or voxel models based on medical imaging data, providing a more realistic representation of human anatomy. *Publication 110* (ICRP, 2009a), a joint report with International Commission on Radiation Units and Measurements (ICRU), provided reference phantoms for the adult male and female derived in this way from imaging data for individuals. The individuals were chosen for their similarity to the external dimensions and organ masses of the reference adult male and female (ICRP, 2002a) and the models were subsequently adjusted for consistency with these data. The use of male and female phantoms rather than the hermaphrodite MIRD phantoms requires explicit sex-averaging in the calculation of effective dose. Thus, in calculations relating to the 2007 Recommendations (ICRP, 2007a), equivalent dose is calculated separately for males and females and averaged in the calculation of effective dose to the sex-averaged reference person (Fig. 3.2). ICRP will issue a set of reference phantoms for children of different ages and for the pregnant woman and fetus.

(52) *Publication 116* (ICRP, 2010) provided the first set of dose coefficients calculated using *Publication 103* (ICRP, 2007a) methodology and *Publication 110* (ICRP, 2009a) anatomical models, considering occupational exposures to external radiation. The radiations considered are external beams of monoenergetic photons; electrons and positrons; neutrons; protons; pions (negative/positive); muons (negative/positive) and He ions. The organ dose conversion coefficients tabulated in the report represent ICRP/ICRU recommended values. Comparisons of the protection quantities, equivalent and effective dose, with corresponding operational quantities (see Section 3.7) showed the latter to provide conservative estimates of dose in the majority of cases. Annexes and a CD provide detailed supporting information, including dose coefficients for the lens of the eye and skin.

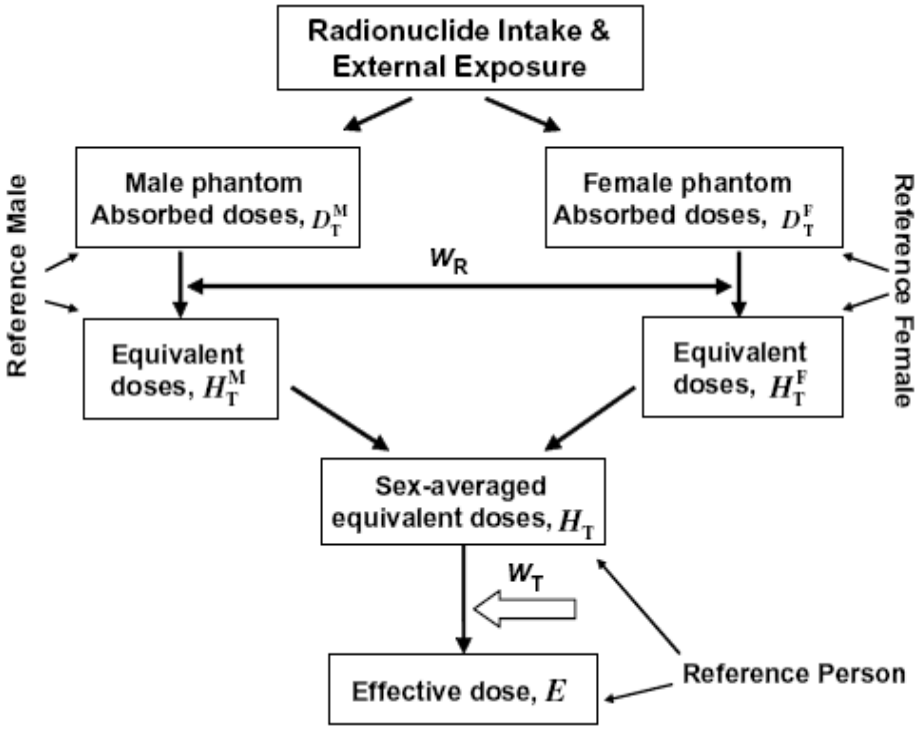


Fig. 3.2. Sex-averaging in the calculation of effective dose using *Publication 110* (ICRP, 2009a) reference phantoms.

(53) Work is in progress to replace internal dose coefficients and provide associated bioassay data for occupational exposures (ICRP, 2015b, 2016, 2017) and replace dose coefficients for members of the public and for radiopharmaceutical administrations to patients. A report is also in preparation to provide, for the first time, dose coefficients for exposures of members of the public, including children, to external sources.

## 3.6. Skin dose

(54) The first concern with regard to doses to skin is the avoidance of tissue reactions. As discussed in Section 2.1, the dose limits set to avoid such damage are equivalent doses of 500 mSv for workers and 50 mSv for members of the public. The standard approach to the calculation of skin doses is to determine the average dose to the most exposed 1 $cm^2$ at a depth of 70 µm (ICRP, 1991a, 2007a). ICRP (1991a) refers to a range in epidermal thickness of from 20 µm to 100 µm for the majority of body sites but both ICRP and ICRU (1997) use a nominal average value of 70 µm for general dosimetric purposes. However, ICRP (2002a) has published reference values for the thickness of epidermis of 45 µm for the newborn child, and 1-year-old and 5-year-old children, 50 µm for 10-year-old children and 60 µm for 15-year-old children as well as 70 µm for adults. A legitimate question raised therefore, particularly in connection with environmental contamination with radioactive particles [e.g. COMARE (2014)], is whether skin doses should be calculated at shallower depths for the younger age groups. Such considerations are important when calculating doses from radionuclides with low energy beta or alpha particle emissions. However, for a number of reasons, it appears most appropriate to continue to determine dose as an average over 1 $cm^2$ at a depth of 70 µm for all ages:

- Threshold doses and $ED_{50}$ values (dose causing an effect in 50% of individuals) for skin damage are calculated in relation to a depth of 70 µm; different values are obtained for calculations relating to other assumed depths (Charles and Harrison, 2007). The cautious limit for workers of 500 mSv is calculated at 70 µm, as is the highly cautious value of 50 mSv for members of the public;
- The variations in skin thickness for different regions of the body substantially exceed the differences implied by the reference epidermal thickness values given in *Publication 89* (ICRP, 2002a);
- The ICRP Task Group on the biological basis for skin dose limitation considered that for normalising effects of different energy beta particle emissions from radioactive particles, the best measure was an average over 1 $cm^2$ at a depth of 150 µm (ICRP, 1991a). A depth of 150 µm corresponds approximately to the depth of the basal cell layer of the epidermis around hair follicles.

(55) On the basis of these considerations, it is proposed that the most appropriate approach for general protection purposes is to continue to calculate dose averaged over 1 $cm^2$ at a depth of 70 µm in all cases. This has been interpreted for the purposes of dose calculations as a layer of tissue at a depth of 50-100 µm (ICRP, 2010a). In the evaluation of possible effects in individual cases, it may be appropriate to consider the effect of variations in skin thickness and uncertainties regarding locations of target cells.

(56) In evaluating risks of stochastic effects, ICRP (1991b, 2007a) relates the risk of skin cancer to the average doses to the total area of skin, 1.9 $m^2$ in adult man and, for example, 0.48 $m^2$ for a 1-year-old child (ICRP, 2002a). A number of animal studies, mainly involving skin exposures of mice and rats, have compared effects caused by radioactive particles irradiating small areas of skin with effects of spatially uniform radiation exposures. For the same average doses, there is little evidence of any dependence of cancer risk on spatial dose distribution (Charles et al., 2003), supporting the ICRP approach of averaging dose in the assessment of cancer risks.

## 3.7. Operational quantities and dose assessments

(57) For the monitoring of external exposures, operational dose equivalent quantities for area and individual monitoring have been defined by ICRU. Dose equivalent quantities are measurable and instruments for radiation monitoring are calibrated in terms of these quantities. In routine monitoring, the values of these dose quantities are taken as a sufficiently precise assessment of effective dose, and doses to the eye lens and skin.

(58) For individual monitoring for occupational exposures to external radiation, the operational quantity is the personal dose equivalent ($H_p(d)$) which is the dose equivalent in ICRU (soft) tissue at an appropriate depth (d) below a specified point on the human body. The specified point is normally taken to be where the individual dosimeter is worn. For the assessment of effective dose from measurement of personal dose equivalent, a depth $d$ = 10 mm and $H_p(10)$ has been chosen and if the dosimeter is worn on a position of the body that is representative of whole-body exposure, it is assumed that the value of $H_p(10)$ provides an effective dose value that is sufficiently precise for protection purposes. For the assessment of the dose to the skin and to the extremities, the personal dose equivalent ($H_p(0.07)$) with a depth $d$ = 0.07 mm, is recommended for use as an operational quantity. For the case of monitoring the dose to the lens of the eye, a depth $d$ = 3 mm has been proposed. Although *Publication 103* (ICRP, 2007a) considered that measurement of $H_p(3)$ may be unnecessary,

the increased importance of the lens of the eye with the reduction in the dose limit to 20 mSv per year (ICRP, 2012a) has led to a re-evaluation of its application (ICRP, 2010a; Bolch et al., 2015). In some situations in which individual monitoring is not carried out, an assessment of effective dose may be performed by area monitoring applying the quantity ambient dose equivalent ($H^*(10)$).

(59) The set of ICRU operational dose quantities in current use was defined more than 30 years ago. Following from *Publication 116* (ICRP, 2010a) providing updated dose coefficients for occupational exposures to external sources (see Section 3.5), ICRU has reviewed the definition of the operational quantities. There are some shortcomings in their definition including that the published conversion coefficients were calculated using the kerma approximation, i.e. without consideration of energy transport by secondary charged particles and that the operational quantities are not good approximations for effective dose at low energies and high energies. The review resulted in suggestions for new definitions of operational quantities for area and individual monitoring. The proposal is to define them as the product of fluence or air kerma and conversion coefficients derived from the maximum of the conversion coefficient curves for effective dose as function of particle energy for all particles considered in *Publication 116*. As a consequence, the operational quantities are implicitly defined in reference anthropomorphic phantoms, resulting in improved coherence and simplification of the system (ICRU, in preparation).

(60) Dose assessment for intakes of radionuclides in occupational settings can be done by estimating intakes either from direct measurements (e.g. external monitoring of the whole-body or of specific organs and tissues) or indirect measurements (e.g. urine, faeces or environmental samples) and using the same biokinetic models used to calculate dose coefficients.

(61) Radionuclides incorporated into the human body irradiate tissues over time periods determined by their physical half-life and their biological retention within the body. Radionuclides used in radiopharmaceutical preparations invariably have short half-lives but general occupational and public exposures can include radionuclides with long physical half-lives and biological half-times and may give rise to doses to body tissues for many months or years after the intake. The need to regulate exposures to radionuclides and the accumulation of radiation dose over extended periods of time has led to the definition of committed dose quantities. The committed dose from an incorporated radionuclide is the total dose expected to be delivered within a specified time period. The committed equivalent dose ($H_T(\tau)$) in a tissue or organ T is defined by:

$$H_T(\tau) = \int_{t_0}^{t_0+\tau} \dot{H}_T(t)\mathrm{d}t$$

where $\tau$ is the integration time following the intake at time $t_0$. The quantity committed effective dose $E(\tau)$ is then given by:

$$E(\tau) = \sum_T w_T H_T(\tau)$$

(62) For compliance with dose limits, the Commission recommends that the committed dose is assigned to the year in which the intake occurred. For workers, the committed dose is normally evaluated over the 50-year period following the intake. The committed effective dose from intakes of radionuclides is also used in prospective dose estimates for members of the public. In these cases a commitment period of 50 years is considered for adults. For infants and children the dose is evaluated to age 70 years.

(63) It has been argued that the use of committed dose introduces hidden conservatism into calculations of doses from annual intakes (Gonzalez et al., 2013). For some radionuclides, with long half-lives and long biological retention times, only a small proportion of the committed dose is delivered in the year of intake. For plutonium-239, for example, effective dose in the first year after intake will be generally less than 10% of the total committed dose. For most radionuclides, however, this effect will be much less significant and for many, including iodine-131 and caesium-137, dose will be delivered entirely or very largely in the year of intake. For practical purposes, the use of committed dose ensures that longer term exposures from intakes of radionuclides are taken into account.

(64) Effective doses for medical exposures are calculated using dose coefficients that relate measurable quantities to the protection quantities, although note that ICRP has not published reference values. These measurable quantities for radiography and fluoroscopy include entrance surface air kerma (ESAK, $K_e$), which is a measure of the dose to the skin surface relative to air, and kerma-area product (KAP, $P_{KA}$), which is the product of the air kerma incident on the patient and the area of the X-ray beam at the skin surface and provides a measure of radiation entering the patient (Jones and Wall, 1985; Hart et al., 1994, Ranniko et al., 1997, Kramer et al., 2004). For computed tomography (CT) examinations, the dose quantity is the dose-length product (DLP, $P_{DL}$) which is the dose within individual slices of the scan multiplied by the scan length (IMPaCT, CTExpo, Wall et al., 2011; Lee et al., 2012). For nuclear medicine procedures the amounts of radioactivity in radiopharmaceuticals administered to patients is used (ICRP 1987, 1998, 2008; Stabin, 1996: Stabin et al., 2005). Tabulated conversion factors are available in the above references, to allow effective doses for a reference adult or reference paediatric patients of ages 0 year, 1 year, 5 years, 10 years and 15 years to be calculated from the measured quantities for a wide range of procedures. Such assessments give an indication of the radiation doses to patients that are sufficient for most requirements.

## 3.8. Collective dose

(65) For the purpose of optimisation of radiological protection, the Commission has introduced the collective dose quantities (ICRP, 1977, 1991b, 2007a). These quantities take account of the group of persons exposed to radiation and the period of exposure. They represent the sum of all individual doses from a source over a specified time period. The specified quantities have been defined as the collective equivalent dose ($S_T$) which relates to a tissue or an organ T, and the collective effective dose ($S$) (ICRP, 1991b, 2007a). The special name used for the collective dose quantity is the ‘man sievert’. Since the intention of the collective dose is to serve as an instrument in the optimisation of radiological protection only the collective effective dose is retained in the present system.

(66) The use of collective effective dose relies on the validity of the application of the LNT dose-response relationship, and the additivity of different types of radiation exposure. Collective effective dose is mainly an instrument for optimisation, for comparing radiological technologies and protection procedures. It is used, for example, by UNSCEAR (2008, 2010, 2012a) to compare doses from different sources of radiation. Collective effective dose is not intended as a tool for epidemiological risk assessment and it is inappropriate to use it in formal risk projections for such studies. In particular, the computation of cancer deaths based on collective effective doses involving trivial exposures to large populations is not reasonable and should be avoided (ICRP, 2007a).

(67) To avoid aggregation of, e.g., very low individual doses over extended time periods and wide geographical regions, ideally limiting conditions need to be set. Where possible, the dose range and the time period should be stated. The collective effective dose due to individual effective dose values between $E_1$ and $E_2$ is defined as:

$$S(E_1, E_2, \Delta T) = \int_{E_1}^{E_2} E \frac{\mathrm{d}N}{\mathrm{d}E} \mathrm{d}E$$

where d$N$/d$E$ denoted the number of individuals who are exposed to an effective dose between $E$ and $E$ + d$E$ and $\Delta T$ specifies the time period within which the effective doses are summed. The use of collective effective dose is considered further in Section 4.3.

# 4. OCCUPATIONAL AND PUBLIC EXPOSURES

(68) The use of effective dose is well established for controlling and monitoring occupational and public exposures. It provides a robust approach to enable external and internal exposures from a variety of different sources and types of radiation to be summed and compared with appropriate dose limits, dose constraints and reference levels. These limits, constraints and reference levels are set for all workers and all members of the public, recognising differences in risk between individuals and population groups, and also recognising that exposures may continue over a whole or working lifetime. The following sections consider the use of effective dose for occupational and public exposures, covering planned, existing and emergency exposure situations, considering individual and collective doses.

## 4.1. Occupational Exposures

(69) Effective dose is an important tool for the management of all types of occupational exposure situation. In planned exposures, it is used in prospective assessments for optimisation of radiological protection and to ensure that operations will be carried out within the relevant dose limits and dose constraints. The sum of prospective external and internal exposures is used in such assessments to consider both individual and collective exposures. The collective effective dose is a useful tool for operational radiation protection, notably when planning complex work involving multiple workers where it is important to consider collective exposures as well as the exposure to the individual workers. Prospective assessments are based on estimations of the likely exposures from particular types of work and take into account experience in similar situations elsewhere. Collective and individual effective dose estimates can then be used to optimise protection, ensuring that the reductions in exposures for some workers are balanced against the potential increase in the number of workers exposed to smaller doses (ICRP, 2007a).

(70) Retrospective assessments of effective dose for occupational exposures in planned exposure situations are used for demonstrating compliance with regulatory requirements, documentation of exposures for regulatory purposes (e.g. workers' dose records) and demonstrating that the system of protection has been adequately implemented. The effective dose is calculated for both external and internal irradiation and will often be based on specific measurements, for example, from a personal dosimeter or of radionuclides in urine. However, it is important to note that although effective dose is estimated for a specific individual, it remains a formal protective quantity in the system of radiological protection. It is defined for

the reference person with a fixed set of anatomical and biokinetic parameters for the human body (ICRP, 2007a). The definition of effective dose precludes any type of individualisation (e.g. taking into account body size or sex) and, as noted earlier, dose limits, dose constraints and reference levels were set to apply to all workers. Therefore, a value of effective dose given for an occupationally exposed person is generalised with respect to the human body properties, but may, however, be more specific with respect to the exposure conditions (see below).

(71) For external irradiation, while effective dose is the primary quantity that should be evaluated, it may also be necessary to explicitly evaluate annual doses to the lens of the eye, the skin and to the hands and feet. The specific occupational dose limits for these organs and tissues (Section 2.2) may be limiting depending on the particular situation, notably for non-uniform irradiation or where there is a significant beta dose component resulting in irradiation of the skin and/or lens of the eye. Occupational doses from external exposures are normally determined by individual monitoring using personal dosimeters worn on the body. The main operational quantities for individual monitoring are $H_P(10)$, $H_P(3)$ and $H_P(0.07)$, as discussed in Section 3.7, and personal dosimeters can be set to measure all of these quantities. Provided that the personal dosimeter is worn in a position on the body that is shown to be representative of whole-body uniform exposure, $H_P(10)$ provides a sufficiently precise estimate of effective dose for protection purposes for most exposure situations. Similarly, $H_P(0.07)$ can be used as a sufficiently precise assessment of equivalent dose to the skin in most circumstances, and while $H_P(0.07)$ also provided an adequate measure of equivalent dose to the eye lens for photons, $H_P(3)$ provides a better measure for electrons of lower energies (ICRP, 2010a; Bolch et al., 2015). In situations where the dose to the body is known to be non-uniform, dosimeters may be worn in positions to determine doses to the most exposed organs, such as the eye lens. Where appropriate, adjustment factors may be used to provide approximate evaluations indicative of likely levels of effective dose. For example, lead/rubber protective aprons worn in radiology departments to protect sensitive organs within the trunk leave the head and neck unshielded. A single unprotected dosimeter worn at the collar of the apron can give indicative dose levels for both the eye and body, from which an assessment can be made of whether any additional monitoring is required (Martin and Magee, 2013). Clinicians performing interventional procedures would wear two dosimeters, one beneath and the other above the apron, and various formulae are applied to estimate effective dose. More specific information may be required on dose to the eye lens, or dose to the protected tissues to enable a more realistic value to be determined for effective dose. In the rare cases of a significant contribution to external exposure of weakly-penetrating radiation, the contribution of the skin dose to effective dose also needs to be considered.

(72) For internal exposures, committed effective doses are determined retrospectively based on the results of individual monitoring or, in exceptional circumstances, monitoring of radionuclide concentrations in air or other media such as surface contamination. Information may be obtained by individual monitoring of radiation emitted from the whole body using a whole-body counter or from specific organs and tissues using other external counting devices (eg. thyroid counter), and by measurements of excretion in urine and faeces. These measurements are interpreted using the biokinetic models used in the calculation of dose coefficients to provide estimates of intake by inhalation or ingestion (or both). Dose coefficients then give values of effective dose for the estimated intakes. Calculations are done using reference biokinetic models and reference dose coefficients as published by ICRP (see Section 3.5). If sufficient information is available and assessed doses warrant a detailed assessment, changes can be made to the assumed particle size distribution of an inhaled material and its solubility and absorption characteristics in the respiratory and alimentary

tracts. Since such changes relate to exposure conditions in the workplace, it is appropriate to apply them in the estimation of intake and the calculation of effective dose. Examples of the use of material-specific data on solubility in the calculation of doses from inhaled radionuclides have been given by ICRP (2002b).

(73) ICRP has stated that changes should not be made in biokinetic assumptions that relate to individuals in the calculation of effective dose (ICRP, 2007a). However, internal radiation doses may be based on a series of measurements of radionuclides in urine for a particular individual. The standard models used to estimate effective doses may not give a particularly good fit to the observed excretion data and it may be possible to obtain a better fit by changing the reference model parameters. The resulting estimated doses should be clearly distinguished from the standard calculation of 'effective dose' and if it is agreed that such dose information should be added in the individual's dose record, this difference should be clearly noted.

(74) In specific circumstances it may be necessary to consider the incorporation of radionuclides through the skin or wounds for occupational exposures. However, this should not be a normal consideration for planned exposure situations where the situation is controlled; for example, protective clothing might be worn and any wounds or abrasions would be covered. The possible intake of radionuclides via wounds may need to be considered as part of any assessment of potential exposures where unplanned events lead to such intakes (see below).

(75) Existing exposure situations are those that are already in existence when a decision on control has to be made. They include situations involving exposures from naturally occurring radionuclides in the workplace and from man-made radionuclides, such as land contaminated by previous nuclear site operations. In addition, the management of long-term contamination resulting from an emergency situation should also be treated as an existing exposure situation. The treatment of occupational exposures due to radon isotopes, primarily radon-222, and their decay products is addressed in *Publication 126* (ICRP, 2014). A report on the use of naturally occurring radioactive materials (NORM) in various industries is in preparation. For existing exposure situations, the use of effective dose is a firm basis for decisions on whether control measures are required. Similar considerations apply to those addressed above for planned exposures.

(76) Emergency exposure situations may arise in the workplace during the operation of a planned exposure situation and any other unexpected situation might result in the emergency exposure of workers. There are two situations of relevance for emergency exposure situations. Firstly, if there is an accident or failure in control in the workplace, workers may be exposed to higher than normal radiation exposures. It is important to quickly assess what such exposures might have been in order to determine if medical intervention is required. Effective dose can provide an initial indication of whether exposures are such that tissue reactions could be observed and individual organ doses need to be considered in the control of any further exposures. At a later stage, a full retrospective risk assessment may be required following over-exposures in which effective dose will have only an initial role; risk to individuals should be evaluated in such circumstances using best estimates of organ doses, appropriate RBE data and age-, sex- and population-specific risk factors (See Section 2.6 and Section 5).

(77) The second situation is in the immediate aftermath of an accidental release or in an on-going emergency where intervention by workers may be required to bring the situation under control or to introduce protective measures to safeguard others. In these situations, it may be possible to plan the exposures to some extent and it is appropriate to use effective dose as part of this process. However, it may also be important to take into account exposures

of the skin, or of other organs if there are significant intakes by inhalation (ideally the use of personal protective equipment should minimise internal exposures in such circumstances). As discussed in Section 3.4, there is no reason in principle why effective dose should not be used as a protection quantity at doses above 100 mSv in accident situations. However, caution would be required in such circumstances to avoid tissue reactions, particularly when considering doses from external exposures of the skin and lens of the eye and internal exposures from radionuclides that concentrate in particular organs.

(78) The presence of wounds, abrasions, burns or other pathological damage to the skin may greatly increase the ability of radioactive materials to reach subcutaneous tissues and thence the blood and systemic circulation. Although much of the material deposited at a wound site may be retained at the site, and can be surgically excised, soluble (transportable) material can be transferred to the blood and hence to other parts of the body. These events occur only as a result of accidents, each event will, therefore, be unique and will need to be assessed by occupational health physicists and medical staff. ICRP has not given advice on the interpretation of wound monitoring data. The biokinetic models that have been developed for various radionuclides are, however, applicable to the soluble component of any deposit in cuts or wounds that enters the blood circulation. To provide a means for calculating doses resulting from radionuclide-contaminated wounds, the National Council on Radiation Protection and Measurements, in collaboration with the ICRP, has developed a biokinetic and dosimetric model for such exposures (NCRP, 2007). The dose coefficients and data given by ICRP could therefore be used in conjunction with the NCRP wound model parameter values to obtain estimates of organ doses and effective dose for radionuclides that have entered the blood from the wound site.

## 4.2. Public Exposures

(79) Planned exposures to external and internal sources occur in a range of situations, including the following:

- visits to controlled or supervised areas
- access to areas accessible to members of the public adjacent to controlled areas,
- controlled discharges of radioactive material to the environment,
- environmental releases following disposal of solid radioactive waste,
- use of consumer products containing radioactive material.

(80) Both prospective and retrospective assessments are carried out for planned exposure situations. Prospective assessments are carried out for optimisation purposes, ensuring that effective doses to the "representative person" (see below) are below the relevant dose constraint for the public; such assessments are necessarily carried out using modelling. Retrospective assessments may be carried out to demonstrate compliance with dose limits and for comparison with dose constraints. Ideally such assessments would be based on monitoring of people and the environment but this is not always possible as the levels are too small to be detected. The uncertainties associated with assessments should be recognised. Collective effective doses may also be estimated as an input to the optimisation process or for comparative purposes as discussed below.

(81) Existing exposure situations arise from:

- contamination of areas by residual radioactive material originating from past nuclear operations, nuclear or radiological emergencies or
- residual contamination from past activities that were subject to regulatory control but not in accordance with current requirements,

- use of commodities, including food, feed, drinking water and construction materials, that incorporate natural or residual man-made radioactive material,
- exposure to natural sources, including radon indoors.

(82) For existing exposure situations, prospective assessments are carried out to determine the annual effective dose to the hypothetical person as an input to optimisation studies using the relevant reference level of dose established for the situation of interest. Existing exposure situations can continue for many years and radiation conditions may change slowly enabling past monitoring data to be used to estimate future effective doses. Measurements of people and the environment can be used, if available, for retrospective assessments of annual effective dose for comparison with the relevant reference level of effective dose.

(83) Emergency exposure situations may occur during the operation of a planned exposure situation, from a malicious act or from any other unexpected situation, and may require precautionary and/or urgent protective actions in order to avoid or reduce radiation doses. Members of the public may be subject to external or internal exposure through various pathways from radionuclides dispersed in natural or inhabited environments. Prospective assessments may be carried out as part of emergency planning for possible future accidents or in relation to an accident that has occurred to determine what actions are required. Effective doses are estimated as input to the optimisation process and for comparison with relevant reference levels. Depending on the nature of the release, it might also be important to consider estimates of dose to specific organs or tissues; e.g. for accidents involving releases of iodine-131, it is important to specifically consider doses to the thyroid. Emergency exposures are usually of short duration and it is important to take account of differences in dose as a function of age at exposure. Consideration of exposures of pregnant and breast-feeding women may also be important. Retrospective assessments of effective dose due to emergency exposures may be required to assess the need for medical follow-up. In such cases, individual monitoring data (external and internal exposures) and/or biological dosimetry measurements would be required as well as measurements of radionuclides in various environmental media. It is important to recognise uncertainties associated with the assessment of doses for emergency exposure situations, including those associated with measurements of people and the environment as well as in modelling results. In such situations, measurements may have been carried out for public reassurance purposes and so have relatively high limits of detection and significant uncertainties in conversion to dose. Retrospective assessments can also be used to refine the prospective dose assessments to reduce uncertainties and to improve the optimisation process.

(84) Effective dose is the key quantity used for the purposes of radiological protection of the public (ICRP, 2007a). The annual effective dose to members of the public is the sum of the effective dose obtained within the year from external exposure and the committed effective dose from the intake of radionuclides during the year. External exposures may occur from proximity to controlled areas where sources of external radiation are used in industry, hospitals, research establishments and nuclear plants. External exposures of individuals may also occur from radionuclides released from installations and which are present in the air, soil, or water. Internal exposures can occur by inhalation of radionuclides in the air or by ingestion of radionuclides in food or water.

(85) For protection purposes, i.e. for optimisation of radiological protection and for comparison with dose limits, dose constraints or reference levels, effective dose is usually assessed for a real or, more frequently, hypothetical person receiving a dose that is representative of the more highly exposed individuals in the population (the right tail of a distribution of individual doses within a particular cohort) termed the ‘representative person’. The concept of the ‘representative person’ was introduced in *Publication 101* (ICRP, 2006) to

replace the less quantitatively defined concept of the 'critical group'. A number of possible cohorts containing people of various ages with different occupations, habits and food consumption rates would generally be considered to define the representative person.

(86) In the dose assessment process, a number of reference persons of different age and sex can be considered, as specified in *Publication 89* (ICRP, 2002a). The full set of six age-groups are the 3 month-old infants, 1 year, 5 years, 10 years, and 15 years old children and adults. In addition, ICRP considers doses to the embryo/fetus and to the breast-fed infant following intakes of radionuclides by the mother (see Section 3.5). In *Publication 103* (ICRP, 2007a), it is noted that in most cases the dose to the embryo/fetus and breast-fed infant will be small compared to doses received by the adult. However, this is not always the case and for four radionuclides, phosphorus-32 and -33, calcium-45 and strontium-89, the fetus/breast-fed infant may receive significantly higher doses than other age groups in some exposure situations and therefore may be designated as the representative person. Although doses in a year are required for comparison with dose criteria, it may be adequate to carry out a simplified dose assessment using an annual intake of radionuclides by the mother and applying the dose coefficient for chronic exposure of the fetus throughout pregnancy. If a more detailed assessment is required, the annual intake by the mother should be assumed to occur over the nine months of pregnancy and three months of breastfeeding. ICRP has not provided dose coefficients for exposure of the fetus or children to external sources of radiation. External doses to the fetus are taken to be the same as to the maternal uterus; work is in progress to provide dose coefficients for children for external exposures (see section 3.5). *Publication 101* (ICRP, 2006) concludes that consideration of three age groups, 1 year and 10 years old children and adults, is sufficient for most dose assessments, especially for long-term exposures when individual cohort members will naturally proceed through age groups. In general, uncertainties in estimating exposures are large in comparison with differences in dose coefficients for different age-groups. It is recognized that stakeholders may make requests for calculation of additional age groups, and such calculations are appropriate to facilitate dialogue.

(87) Concern has been expressed regarding the use of a single set of tissue weighting factors in the calculation of effective dose, applied to all age groups including the embryo/fetus and infant (Streffer, 2004). The weighting factors are used to allow for the contribution of individual organs and tissues to total stochastic detriment while not over-interpreting knowledge of risks of low dose radiation exposure. They do not represent scientific best judgements for any specific age group. Application to the embryo/fetus is an extension of their application to infants; as discussed above, overall cancer risk following *in utero* exposure is judged to be no greater than that following exposure in early childhood (ICRP, 2003a). Dose control criteria – dose constraints and reference levels – can be set in the knowledge of potential differences between age groups. The use of dose constraints and reference levels that apply to all members of the public (or all workers), together with optimisation, provides a pragmatic, equitable and workable system of protection that recognises age-, sex-, and population-related differences in risks per Sv but does not distinguish on an individual basis. The corollary is that, for practical radiation protection purposes, the use of a single set of tissue weighting factors remains entirely appropriate.

(88) In many situations, direct measurements of external and internal exposures of the public are not available and the assessment of effective dose is carried out using modelling techniques, supported where possible by measurements of ambient dose equivalent rate and concentrations of radionuclides in the environment. Rarely, information is also available from personal dosimeters or from measurements of the radionuclide content of individuals through techniques such as whole-body counting. Methodologies for assessing doses to the public

often adopt cautious parameter values to ensure that doses are not underestimated and therefore to ensure compliance with the relevant dose limits and for comparison with dose constraints and reference levels. It is important that the degree of caution is recognised and care is needed in using the results of such methodologies for optimisation purposes as this might lead to bias in the assessment. This is particularly important when determining whether actions, such as evacuation or decontamination, are required in an emergency exposure situation. It is important to balance the reduction in doses with any deleterious effects of the action and a cautious assessment of doses could lead to unnecessary actions with adverse consequences for the affected population.

(89) In modelling of radionuclide transfer in the environment and internal doses received by members of the public, an important issue is selection of the most appropriate physical and chemical characteristics of radionuclides. This consideration is of particular importance for prospective assessment of pre-operational facilities and for emergencies. Previous experience of similar situations is likely to be instructive when monitoring data and information on radionuclide characteristics are available. ICRP advise that dose coefficients relevant to specific chemical forms of radionuclides should be used whenever the relevant information is available and the assessment warrants such consideration. When no monitoring data are available, the cautious approach for dose assessment is the selection of those radionuclide characteristics and dose coefficients that result in higher dose estimations. Some guidance on this issue is given in *Publication 72* (ICRP, 1996a).

## 4.3. Collective dose assessments

(90) As discussed in Section 3.8, collective effective dose is intended for use in the optimisation of protection. The quantity is particularly valuable in occupational radiological protection, for use, for example, in planning complex work involving varying numbers of workers. Collective effective dose can be used to determine the optimum balance between relatively large exposures to a few workers and smaller exposures to a larger number of workers (ICRP, 2007a).

(91) For public exposures, collective effective doses can be used as part of the optimisation process for planned, existing or emergency exposure situations. The quantity also has a useful role in comparative studies to consider the radiological impact of different sources of exposure.

(92) As discussed in Section 3.8, collective effective dose is not intended as a tool for epidemiological analysis and the prediction of health effects in populations and particular care is needed in interpreting collective dose data made up of very low (μSv or nSv) levels of individual dose received over long time periods by large numbers of people (ICRP, 2007a). However, there can be situations where the estimation of health effects from collective effective doses can be useful for planning of radiation protection actions if treated with appropriate caution. For example, following a severe nuclear accident or in advance planning for such events, an assessment of collective effective dose could be used to give an indication of possible health impact to help with planning and selecting from various protection options. In retrospective assessments of planned or existing exposure situations, assessments of collective effective dose can provide initial screening evaluations of possible health impact to inform medical and epidemiological evaluation. It is essential that such analyses using collective effective dose include consideration of background rates of health effects in the population, including morbidity and mortality, and consider uncertainties, recognising that health effects in individuals exposed to low levels of radiation are highly unlikely to be

attributable to radiation exposure (UNSCEAR, 2012a) and that comparisons with natural disease incidences determine whether epidemiological analyses may provide statistically significant results for populations.

(93) As discussed in *Publication 101* (ICRP, 2006) and *Publication 103* (ICRP, 2007a), it is recommended that when exposures occur over large populations, areas and time periods, such that individual doses range over several orders of magnitude, the collective effective dose should be split according to ranges of individual dose, also taking account of geographical locations and the time-course of dose delivery. *Publication 101* (ICRP, 2006) discusses the use of a collective dose matrix approach to the disaggregation of collective effective dose on the basis of levels of individual dose, and distribution in space (local, regional, global), and time (short-, medium- and long-term). However, there are problems in implementing such recommendations for public exposures if, as is usually the case, ingestion of food is an important exposure pathway. In general, the food that people consume is not produced in the immediate area but rather it is sourced over large areas on a changing basis. It is generally not possible to gain specific information on where people obtain their food, rather collective dose estimates are based on food production data, and the distribution of individual doses is not known (Smith et al., 2006). However, collective effective doses can be estimated for specific population groups living in defined geographical areas over different time periods as discussed in *Publication 103* (ICRP, 2007a). Per-caput doses can also be estimated corresponding to the collective effective doses for different population groups which can provide useful input to optimisation and comparative studies (Smith et al., 2006). Assessments of collective effective dose into the far future are particularly uncertain due to the impact of factors including climate change, changes to human behaviour and population numbers. Therefore, collective effective dose assessments involving integration of doses over thousands of years into the future, as might be done in assessing the radiological impact of solid waste disposal, cannot usefully inform protection decisions, and are not considered useful. As discussed in *Publication 101* (ICRP, 2006), current knowledge suggests that such dose assessments can contribute appropriately to decision making for periods spanning a few generations but should not play a major part in planning for longer time frames.

# 5. MEDICAL EXPOSURES

(94) Radiation is used in a wide range of applications in medical diagnosis and therapy. The radiation doses received by patients in diagnostic and interventional procedures are recorded in terms of quantities that can be measured for each technique. Examples of such quantities are entrance surface air kerma (Ka,e) and kerma-area product ($P_{KA}$) for radiography and fluoroscopy, and volume averaged CT dose index ($CTDI_{vol}$) and dose length product (DLP) for CT (see Section 3.7). These measured quantities can be applied through straightforward methods for assessment of dose levels and are used for comparisons of doses for particular types of examination among different healthcare facilities and around the world. Surveys are made to establish diagnostic reference levels (DRLs) in terms of these measurable quantities (Martin, 2008, 2011; ICRP, 2016). These measured dose quantities are suitable for making comparisons between facilities, machines, and techniques that deliver exposures with similar relative distributions of absorbed dose inside the body.

(95) Because stochastic risks vary substantially according to the organs and tissues irradiated in different medical procedures, measurable dose quantities are unable to convey a meaningful indication of the associated relative health detriments from alternative techniques

that result in different distributions of dose within the body. Effective dose can be used to make such comparisons between doses from medical procedures that expose different regions of the body. It has been instrumental in raising awareness of dose levels from diagnostic procedures and providing a broad understanding of possible risks associated with these radiation exposures. It is used commonly in training medical professionals in radiological protection. It is employed in making informed judgements to aid in justification of medical procedures and in establishing dose constraints for patient carers and for volunteers in medical research. Effective dose has provided a useful reference for the improvement of radiological protection in medical practice, and gives a means of conveying an indication of radiation dose relating to possible risk to health that can be understood by clinicians and non-specialists in radiological protection.

(96) Effective dose has proved to be a useful tool for characterising medical exposures, but using it to provide estimates of risk to individual patients goes beyond its intended applications (ICRP, 2007a; Menzel and Harrison, 2012; Harrison et al., 2016). Brenner (2008, 2012) suggested that effective dose should be replaced by 'effective risk' as a more scientifically based quantity. Effective risk is calculated as the sum of the product of the equivalent dose to each organ/tissue and the corresponding life-time risk per unit equivalent dose, using age- and sex-averages risk factors or age- and sex-specific data. An example of its use applied to CT examinations is provided by Andrade et al. (2012). However, this approach ignores the uncertainties associated with risk inference at low doses based on epidemiological observations of populations exposed to higher doses. While doses can be measured or estimated with reasonable reliability down to very low levels, the inferred risk that may be associated with the dose is increasingly uncertain as dose decreases (Dietze et al., 2009; UNSCEAR, 2012a). However, evidence is presented in this chapter in support of the use of effective dose as an approximate indicator of possible risk associated with medical procedures, showing that difference between estimates of risk based on effective dose and estimates based on the use of organ doses and age-, sex- and cancer-specific risk estimates are predictable and generally not large.

## 5.1. Effective dose from medical procedures

(97) Effective doses from medical procedures are calculated using dose coefficients that relate measurable quantities to the protection quantities (see Section 3.7). Daily decisions for justifying individual patient imaging exposures, or for optimising protection through selecting the most appropriate technique, require approximate estimates of dose relating to inferred risks to health. Generic values of effective dose for a reference person derived using these coefficients provide a straightforward tool with enough information about general radiation exposure levels linked to detriment for the purpose of making these everyday decisions. Ideally these generic values should be based on data that apply to the country and facility under consideration. Examples of the range of values for a selection of examinations in different countries is given in Table 5.1. Variations result from differences in equipment, techniques, and patient selection (weight range), and help to emphasise the importance of using results that apply to the local facility and country wherever possible. When these values are used, it should be understood that they relate to a reference person, and not to any individual patient.

Table 5.1. Examples of typical effective doses (mSv) for adults in 3 countries from some common examinations

| Procedure | UK[a] | USA[b] | Russian Federation[c] |
|---|---|---|---|
| **Radiography** | | | |
| Chest PA | 0.014 | 0.03 | 0.1 |
| Chest Lat | 0.038 | 0.07 | 0.18 |
| Lumbar spine AP | 0.39 | 2.0 | 0.6 |
| Lumbar spine Lat | 0.21 | 2.0 | 0.6 |
| Abdomen AP | 0.43 | 0.7 | 1.0 |
| Pelvis AP | 0.28 | 1.25 | 0.7 |
| **Interventional** | | | |
| Coronary angiography | 3.9 | 15 | 15 |
| Femoral angiography | 2.3 | 7 | 5-10 |
| **Computed tomography** | | | |
| CT Head | 1.8 | 2.1 | 1.8 |
| CT Chest | 14 | 11 | 6.3 |
| CT Abdomen | 16 | | 9 |
| CT Abdomen + Pelvis | 13 | 17 | |
| CT Chest+Abdomen+Pelvis | 19 | 29 | 25 |
| **Nuclear Medicine** | | | |
| Bone scan: Tc-99m | 3 | 5 | 3 |
| PET tumour imaging (F-18 FDG) | 7 | 10 | 5 |

[a]Wall et al., 2011; Shrimpton et al., 2016; ARSAC, 2018. [b]Mettler et al., 2008; Smith-Bindman, 2015; Alessio et al. 2015; Becker et al. 2016. [c]Chipiga & Bernhardsson, 2016; Vodovatov et al., 2016; Zvonova et al. 2015; Balonov et al. 2018.

(98) When imaging is limited predominantly to one anatomic area, such as in mammography of the breast, estimates of organ or tissue dose should be used instead of effective dose. Similarly, assessments of doses from imaging procedures involving radioiodine uptake by the thyroid should primarily be quoted in terms of absorbed dose to the thyroid, which is the predominant organ irradiated. Gonad dose should be used for evaluation of examinations in which doses to the reproductive organs make up the majority of the dose, noting that the calculation of effective dose includes averaging of doses to the gonads of both sexes (see Sections 2.4 and 2.5).

## 5.2. Justification of procedures

(99) ICRP (1996b, 2007b, 2008) recommends justification of medical exposures at three levels: 1) that use of radiation in medicine should do more good than harm, 2) that a given type of procedure is justified for a particular clinical indication as it will improve the diagnosis or treatment of patients; and 3) that a medical examination for an individual patient will do more good than harm, by contributing to the management of the patient's treatment.

(100) The first level of justification occurs at the national level when radiation equipment and techniques are approved for purchase and use in hospitals and other medical installations. The second level is reflected in referral guidelines produced by professional societies and health authorities, and here effective dose to a reference person is used to provide information on the relative magnitudes of doses from different kinds of examinations (Reference American College of Radiology Appropriateness Criteria; EC, 2000; EANM guideline series). Clinicians (e.g. referring clinicians and radiologists) are responsible for carrying out the third level of justification for every patient for whom an imaging procedure that uses ionising radiation is requested, based on the patient's clinical condition and history. In this process, in addition to sex and age, the medical risk of a proven or suspected disease has to be considered, with the implications of radiation exposure varying according to the life expectancy of the patient (Loose et al., 2010).

(101) Values of effective dose for a reference person are included in many guidelines for referral and justification. This information can be used as an additional refinement to justification to help identify the most suitable examination for a given patient and minimise the risk of harm. In addition to values for adults, effective doses are also available for limited ranges of paediatric examinations linked to x-ray exposure factors or administered activity and based on reference paediatric phantoms, as dose distributions within the smaller bodies of children can differ considerably from values obtained for adults. More precise estimates are unnecessary for the purpose of guiding referrals.

## 5.3. Optimisation and reporting of doses

### *Choice of technique*

(102) Patient imaging procedures typically involve partial body radiation exposures, and exposure of tissues with differing sensitivities in terms of radiation-associated cancer risk. The amount of radiation and its distribution within the tissues of the body can be very different with different imaging modalities, even when a similar region of the body is being imaged. Since dose distributions from machine-produced x-ray and nuclear medicine procedures are very different, the effective dose is suitable for use in straightforward comparisons of doses from different techniques.

(103) When two different x-ray imaging modalities are considered, comparison of effective dose can be of value in guiding a referral test selection. For example, a chest CT examination and a conventional chest x-ray both irradiate the lungs, but the effective dose from CT can be a few hundred times that of chest radiography, depending on the protocol technique. Importantly, the spatial distribution of radiation dose within the body is also different. The dose to the breasts from scattered radiation with postero-anterior (PA) chest radiography could be a factor of many thousand times less than that from a chest CT, and the effective dose could be a factor of five hundred lower. If the necessary information can be provided by both chest CT and chest radiography for a particular clinical question, the differences in effective dose (even if crudely estimated) supports the choice of chest radiography, although the importance of clinical guidelines is recognised and physicians may opt for CT, despite the possible increased risk, because of its greater diagnostic capability.

### *Optimisation of technique*

(104) Once a decision is made regarding an imaging procedure, the next step is to ensure its optimisation. The optimisation of radiological protection for patients is applied to the design, appropriate selection, and construction of equipment and installations; and to the day-to-day choice of techniques and procedure parameters (i.e. the clinical protocols). The basic aim of optimisation of protection is to adjust the protective measures in a way that adequately addresses the clinical question while keeping the radiation dose to a minimum or to as low as reasonably achievable (the 'ALARA' principle) (ICRP, 2007a).

(105) Effective dose is not the best quantity for making comparisons between doses for similar techniques applied in different departments or institutions. Modality-specific dose quantities (e.g. $P_{KA}$, $CTDI_{vol}$) should be used for this purpose. However, in circumstances in which the dose distributions within the body may be substantially different between procedures, effective dose provides an appropriate measure for comparison.

(106) If a single tissue such as the breast is irradiated, with substantially lower doses to other tissues, comparisons should be based on dose to that tissue. Often, however, doses to a number of organs and tissues within the trunk need to be considered and the use of effective dose is appropriate. Examples are when using different radiographic projections (e.g. PA as opposed to anteroposterior (AP)) (Martin et al., 1999; Martin and Sutton, 2014), using different tube potentials (kV) (Martin et al., 1993; Huda et al., 2004), or very different x-ray tube filtration – for example in paediatric radiology or interventional procedures. In comparing AP and PA projections for abdominal radiography (Martin et al., 1999), the stomach, colon and liver lie closer to the surface in the AP than the PA projection and so receive a higher dose and make a greater contribution to effective dose. In the selection of tube potential (kV) or filtration for an x-ray examination, increasing the kV will give more penetrating radiation, so that the exposure level can be reduced, lowering the dose to more superficial tissues, while the effect on doses to tissues deep within the body near to the image receptor will be minor (Martin et al., 1993; Huda et al., 2004; Martin, 2007b; Martin, 2008; Martin and Sutton, 2014).

### *Doses to volunteers*

(107) Exposures incurred by volunteers as part of a programme of biomedical research are considered medical exposures (ICRP, 1991b, 2007b; IAEA, 1995, 2011). Before a research proposal is approved, an evaluation of possible detriment for the individuals involved must be made and recorded. Effective dose is the appropriate quantity to use for summing the possible radiation-related health detriments that may accrue from the various procedures, that are to be performed to support the research objectives, each of which may have a different dose distribution within the body (IAEA, 2011). However, it should be recognised that effective dose is estimated for a reference person. When considering potential radiation-related risks in research subjects, cognisance should be taken of age, sex and health status (see Section 5.4).

### *Reporting of unintended exposures*

(108) Unintended exposures and overexposures of patients in diagnostic procedures provide examples of situations where effective dose for a reference person could provide sufficient information for the incident investigation and report, and inform decisions regarding whether a more detailed assessment may be required. An unintended exposure

could occur in various situations, such as when there has been an error in the referral process or the wrong patient or body part was examined. An overexposure might occur when there has been a mistake in the procedure technique, or where an equipment fault has occurred (Martin, 2005; Martin et al. 2017). In situations of unintended exposure, where the dose level is low, a broad assessment in terms of effective dose will usually be sufficient. If the unintended exposure is known to be similar to the dose for the standard examination of that type, then generic values of effective dose for that procedure can be used if the generic value is a few mSv or less. When the effective dose is greater or exposure conditions do not equate to a standard examination, it is more appropriate to calculate the effective dose for the reference person from the available exposure data. If the effective dose is greater than about a few tens mSv, there is likely to be a perceived need for a more in-depth evaluation involving an assessment of risk for the individual. In these circumstances, it will be more appropriate to estimate doses for all radiosensitive organs and tissues and apply age-, sex- and organ-specific risk coefficients to derive a best estimate of risk (see Section 5.4).

### *Tracking of patient doses*

(109) As the use of radiation for medical imaging has increased, the number of patients who receive repeated imaging procedures has also risen (Sodickson et al., 2009). Dose tracking methods are being developed for recording patients' accumulated radiation exposure from medical imaging procedures over time in order to provide more formal ways to quantify these doses (Rehani et al., 2014; Rehani, 2015). These data are best recorded using measured dose quantities (Rehani and Berris, 2013), but if evaluations are required in the review of doses for specific individuals, calculations of organ and effective dose will aid understanding of potential risks.

### *Doses to carers*

(110) Exposures (other than occupational) incurred knowingly and willingly by individuals helping in the support and comfort of patients undergoing diagnosis or treatment are considered under medical exposures for convenience. A typical example is the exposure of family members of a patient discharged after a thyroid treatment with unsealed $^{131}$I, or patients who have implanted sealed sources. Assessments of potential exposures and doses received will need to be made from time to time, and the appropriate quantity, as for occupational and public exposures, is effective dose to a reference person. The acceptability of doses and risks will depend on the individual circumstances.

## 5.4. Effective dose and risk communication

(111) Although effective dose is not intended as a measure of risk to individuals, it is considered reasonable to use effective dose to a reference person as an approximate indicator for risk communication in general terms, with appropriate caveats for individual patients.

### *Education and training*

(112) Clinicians who refer and other medical professionals who perform medical procedures involving radiation may have little understanding of the potential health detriment from radiation exposure, because it is so small compared to the benefits of medical exposures (ICRP, 2009b; Loose et al., 2010; Zanzonica and Stabin, 2014). Consequently, it is difficult for them to take these potential risks into account when requesting or justifying patient diagnostic or interventional exposures, or when explaining possible risks to their patients. Effective dose is a useful quantity in this context because it is a single value which can be used to compare various exposure scenarios. The concept of effective dose and a knowledge of typical effective doses from common procedures should therefore be included in the education and training of medical practitioners.

(113) Medical practitioners are also one of the first groups approached by members of the public for advice and reassurance in the event of a radiation exposure or an accident involving potential radiation exposure of the public. When only the possibility of stochastic effects is involved (the majority of cases), effective dose is an appropriate quantity for straightforward communication and to facilitate comparisons of the possible health risks of an exposure with risks from other exposure scenarios.

### *Communication of doses and associated health risks*

(114) For discussions regarding justification and optimisation of examinations and for communication with patients, clinicians need language to describe radiation dose that reflects a broad perspective of risk. This can be provided through effective dose. Table 5.2 gives a scale linked to effective dose, with general terms to describe the dose linked to possible levels of risk and examples of procedures within different dose ranges. The terms used for effective doses of 1 mSv and greater are the same as applied by UNSCEAR (2012a) to whole-body absorbed doses (mGy) in the same ranges. Thus, the inferred risk from an exposure giving an effective dose of 10 to 100 mSv can be termed low, while that for effective doses in the range of 1 mSv to 10 mSv can be considered to be ‘very low’, equating to the exposures that individuals get every year simply from living on earth through exposure to natural background radiation. The excess risk from an effective dose less than 0.1 mSv, which includes examinations such as chest x-rays, is categorised in this scheme as negligible; an alternative term might be extremely low.

(115) Clinicians and patients will sometimes need more information in order to put radiation exposures and possible risks into context. For this purpose, comparisons can be helpful with those radiation doses from situations with which the individuals are familiar, and for which they accept the risk. Examples of everyday exposures are those from natural background radiation and the dose that an individual might receive from cosmic rays during an airplane flight. These comparisons can be particularly useful for patients who have concerns about the procedures that they are undergoing, but who have little or no knowledge about radiation and may as a result have an unrealistic fear of the potential harm from a radiation exposure. The quantity effective dose to a reference person can be instrumental in educating medical practitioners, patients and the public, by helping to provide a broader perspective of possible risks from radiation exposure. The potential risk from medical exposures is generally lower than for a reference population due to the higher average age of patients and competing disease related risks with reduced life expectancy, although paediatric populations serve as an exception. Furthermore the risk of radiation exposures in

interventional radiology replaces in many cases the higher risks of alternative surgical therapies.

Table 5.2. Dose ranges and terminology for describing risks from different medical procedures for adult patients of average age (30-39 years) based on UK data (Martin, 2007a; Wall et al., 2011; Martin and Sutton, 2014).

| Effective doses (mSv) | Risk of cancer | Proposed term for dose level | Examples of medical radiation procedures within different dose categories[b] |
|---|---|---|---|
| < 0.1 | Inferred $< 10^{-5}$ | Negligible | Radiographs of chest, femur, shoulder limbs, neck, and teeth, $^{99m}$Tc sentinel node imaging, radionuclide labelling for in vitro counting with $^{14}$C and $^{57}$Co. |
| 0.1–1 | Inferred $10^{-5} – 10^{-4}$ | Minimal | Radiographs of spine, abdomen, pelvis, head and cervical spine, radionuclide labelling for in vitro counting with $^{51}$Cr. $^{99m}$Tc for imaging lung ventilation and renal imaging. |
| 1–10 | Inferred $10^{-4} – 10^{-3}$ | Very low | Barium meals, CT scans of the head and combinations of chest, abdomen, and pelvis, barium enemas, cardiac angiography, interventional radiology; $^{99m}$Tc myocardial imaging, lung perfusion $^{99m}$Tc for imaging lung perfusion, $^{99m}$Tc imaging of bone lesions, cardiac stress tests and $^{99m}$Tc SPECT imaging; imaging with $^{18}$F, $^{123}$I, and $^{111}$In. |
| 10–100 | $10^{-3} – 10^{-2}$ based on LNT model[a] | Low | CT scans of chest, abdomen, and pelvis, double CT scans for contrast enhancement, interventional radiology; $^{67}$Ga tumour, and $^{201}$Tl myocardial imaging; multiple procedures to give doses of 10s mSv, endovascular aneurysm repair. (10-35 mSv).<br>Renal/visceral angioplasty, Iliac angioplasty, follow-up of endovascular aneurysm repair. (35-100 mSv). |
| 100s | $>10^{-2}$ based on epidemiology[a] | Moderate | Multiple procedures and follow-up studies. |

[a]Risk bands are lifetime detriment adjusted incidence to nearest order of magnitude.
[b]Effective doses based on UK for diagnostic procedures and ICRP (2010b) for interventional radiology.

### *Age- and sex-specific cancer risks and effective dose*

(116) As discussed in Section 2.6, epidemiological data used to provide risk estimates for radiation-induced cancer show differences in risk between males and females, and as a function of age at exposure. Depending on the risk projection models used, there are also differences between populations. While estimated risks of lifetime cancer incidence were shown to be similar for males and females for some cancers, including stomach, bladder, liver and leukaemia, risks for females are greater than for males in a number of cases, notably breast cancer, but also lung and thyroid cancers (see Section 2.6). Considering all cancer sites combined, lifetime risks compared with those for the 30-39 years age-group were estimated to be greater by a factor of about two to three for exposures of young children, aged 0-9 years, and less by a factor of two to three for exposures of older adults aged 60-69 years (see

Section 2.6). Within this general trend, some cancer types showed greater age-dependence, notably thyroid cancer, while others show little or no age-dependence, including lung cancer.

(117) Based on the methodology described in section 2.6 to calculate lifetime risk of cancer incidence per unit organ/tissue absorbed dose, and using UK estimates of organ/tissue doses from a range of medical procedures, Wall et al. (2011) derived age- and sex-specific risks per unit effective dose for such procedures. This comparison involved calculation of risk using information on organ/tissue absorbed doses and organ specific risks as a function of age and sex for a specified procedure and expressing the estimated risk per unit effective dose from that procedure. The approach used by Wall et al. (2011) to calculate lifetime risks was slightly different from that used in *Publication 103* (see details in section 2.6), but their results can be used to illustrate variations of lifetime risks with age and sex. For illustration, a selection of their results for an ICRP Euro-American composite population has been recalculated using the risk data in Table 2.4 and presented in Table 5.3. Similarly, using the risk data presented in Table 2.5 for the ICRP Asian composite population, calculated values of age- and sex-specific lifetime risks per Sv are shown in Table 5.4. For males and females and each population, variations in lifetime risk per Sv reflect the combination of organ/tissue doses relating to each procedure. Fig. 5.1 presents the data from Table 5.4, together with the lifetime risks per Sv for uniform whole-body irradiation from Table 2.5. For most procedures, the estimates of lifetime risk of cancer incidence per Sv are within about ± 50% of those for uniform whole-body irradiation for the particular age and sex, noting that the cancer types involved will differ between procedures.

(118) It is important that the precision that might be inferred from the values presented in Tables 5.3 and 5.4 does not give a false impression of the reliability of estimates of cancer risk from low dose radiation exposures. The detailed data are included here to illustrate the overall pattern of age at exposure and sex differences in estimated risk. On the basis of these data, it can be concluded that when considering most x-ray examinations, lifetime risks of cancer incidence per Sv may be around twice as great for the 0-9 years age at exposure group than the 30-39 years group. For patients exposed in their 60s, the estimated lifetime risks are about half those for patients in their 30s, falling to less than one-third for patients in their 70s and about one-tenth for those in their 80s. Bearing in mind the substantial uncertainties associated with projections of low dose risk, it is considered reasonable to reflect such variations in possible risk per Sv effective dose in conveying information to clinicians and patients. While health risk assessments using organ/tissue absorbed doses and site-specific risk models represent best use of scientific knowledge, in most circumstances it will be sufficient to use simple risk terminology as illustrated in Table 5.2. In considering such information, clinicians will wish to take account of factors including the potential benefits of the procedure and the prognosis of the patient's illness.

Table 5.3. Total lifetime risks of cancer incidence (cases per 100) per Sv effective dose as a function of age at exposure and sex for a range of x-ray examinations, calculated using risk data for the ICRP Euro-American composite population (based on Wall et al., 2011).

| | | Age at exposure (y) | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Examination | Sex | 0-9 | 10-19 | 20-29 | 30-39 | 40-49 | 50-59 | 60-69 | 70-79 | 80-89 | 90-99 |
| Head | M | 21 | 14 | 10 | 6 | 4 | 3 | 1 | 0.6 | 0.2 | 0 |
| (AP+PA+Lat) | F | 24 | 14 | 9 | 6 | 4 | 2 | 1 | 0.7 | 0.3 | 0 |
| Cervical spine | M | 13 | 8 | 5 | 3 | 2 | 1 | 0.6 | 0.3 | 0.1 | 0 |
| (AP+Lat) | F | 38 | 18 | 8 | 4 | 2 | 1 | 0.9 | 0.5 | 0.2 | 0 |
| Chest | M | 10 | 8 | 7 | 5 | 5 | 4 | 3 | 2 | 0.7 | 0.1 |
| (PA) | F | 16 | 13 | 11 | 9 | 9 | 8 | 6 | 4 | 2 | 0.3 |
| Thoracic spine | M | 9 | 7 | 6 | 4 | 4 | 3 | 2 | 1 | 0.6 | 0.1 |
| (AP+Lat) | F | 23 | 16 | 12 | 9 | 8 | 7 | 5 | 3 | 2 | 0.2 |
| Abdomen | M | 14 | 11 | 9 | 6 | 5 | 3 | 2 | 1 | 0.4 | 0.1 |
| (AP) | F | 13 | 10 | 8 | 6 | 5 | 4 | 2 | 1 | 0.7 | 0.1 |
| Pelvis | M | 12 | 9 | 8 | 6 | 4 | 3 | 2 | 1 | 0.4 | 0.1 |
| (AP) | F | 10 | 8 | 6 | 5 | 4 | 3 | 2 | 1 | 0.6 | 0.1 |
| Lumbar spine | M | 13 | 10 | 8 | 6 | 4 | 3 | 2 | 0.8 | 0.3 | 0.1 |
| (AP+Lat) | F | 13 | 10 | 7 | 6 | 4 | 3 | 2 | 1 | 0.6 | 0.1 |
| IVU | M | 14 | 10 | 8 | 6 | 4 | 3 | 2 | 0.9 | 0.3 | 0.1 |
| | F | 13 | 10 | 8 | 6 | 5 | 3 | 2 | 1 | 0.6 | 0.1 |
| Ba swallow | M | 10 | 7 | 5 | 4 | 3 | 2 | 1 | 0.8 | 0.3 | 0.1 |
| | F | 27 | 17 | 11 | 7 | 5 | 4 | 3 | 2 | 0.9 | 0.1 |
| Ba follow | M | 15 | 11 | 9 | 6 | 5 | 3 | 2 | 0.9 | 0.3 | 0.1 |
| | F | 13 | 10 | 8 | 6 | 5 | 3 | 2 | 1 | 0.6 | 0.1 |
| Ba enema | M | 13 | 10 | 8 | 6 | 5 | 3 | 2 | 1 | 0.4 | 0.1 |
| | F | 11 | 8 | 7 | 5 | 4 | 3 | 2 | 1 | 0.6 | 0.1 |
| Coronary | M | 10 | 8 | 7 | 6 | 5 | 4 | 3 | 2 | 0.9 | 0.2 |
| angiography | F | 13 | 11 | 10 | 10 | 10 | 9 | 7 | 5 | 3 | 0.3 |
| Femoral | M | 14 | 11 | 8 | 6 | 5 | 3 | 2 | 0.9 | 0.4 | 0.1 |
| angiography | F | 11 | 8 | 7 | 5 | 4 | 3 | 2 | 1 | 0.5 | 0.1 |
| CT head | M | 22 | 15 | 11 | 7 | 5 | 3 | 2 | 0.8 | 0.3 | 0.1 |
| | F | 17 | 12 | 8 | 6 | 4 | 3 | 2 | 0.9 | 0.4 | 0 |
| CT chest | M | 9 | 7 | 6 | 4 | 4 | 3 | 2 | 1 | 0.5 | 0.1 |
| | F | 22 | 15 | 11 | 9 | 7 | 6 | 5 | 3 | 1 | 0.2 |
| CT abdomen | M | 13 | 10 | 8 | 5 | 4 | 3 | 2 | 0.8 | 0.3 | 0 |
| | F | 13 | 10 | 7 | 6 | 4 | 3 | 2 | 1 | 0.5 | 0.1 |
| CT abdomen + | M | 14 | 11 | 9 | 6 | 5 | 3 | 2 | 0.9 | 0.3 | 0.1 |
| pelvis | F | 13 | 10 | 8 | 6 | 5 | 3 | 2 | 1 | 0.6 | 0.1 |
| CT chest + | M | 11 | 8 | 7 | 5 | 4 | 3 | 2 | 1 | 0.5 | 0.1 |
| abdo + pelvis | F | 18 | 13 | 10 | 8 | 6 | 5 | 4 | 2 | 1 | 0.1 |

Note that the methodology used in these calculations is based on but slightly different from that of *Publication 103* (see section 2.6). Tabulated values are risk coefficients (per Sv), not absolute measures of risks from the various procedures from which the doses delivered are in the mSv range or lower.

Table 5.4. Total lifetime risks of cancer incidence (cases per 100) per Sv effective dose as a function of age at exposure and sex for a range of x-ray examinations, calculated using risk data for the ICRP Asian population (based on Wall et al., 2011).

| | | Age at exposure (y) | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Examination | Sex | 0-9 | 10-19 | 20-29 | 30-39 | 40-49 | 50-59 | 60-69 | 70-79 | 80-89 | 90-99 |
| Head | M | 14 | 9 | 6 | 4 | 3 | 2 | 1 | 0.5 | 0.2 | 0 |
| (AP+PA+Lat) | F | 24 | 14 | 8 | 5 | 4 | 2 | 1 | 0.6 | 0.2 | 0 |
| Cervical spine | M | 10 | 6 | 3 | 2 | 1 | 0.9 | 0.5 | 0.3 | 0.1 | 0 |
| (AP+Lat) | F | 47 | 21 | 10 | 5 | 3 | 1 | 0.8 | 0.4 | 0.2 | 0 |
| Chest | M | 10 | 8 | 7 | 6 | 5 | 4 | 3 | 2 | 0.9 | 0.2 |
| (PA) | F | 16 | 12 | 10 | 8 | 8 | 7 | 6 | 4 | 2 | 0.4 |
| Thoracic spine | M | 9 | 7 | 6 | 5 | 4 | 4 | 3 | 2 | 0.7 | 0.1 |
| (AP+Lat) | F | 24 | 16 | 12 | 9 | 8 | 6 | 5 | 3 | 1 | 0.3 |
| Abdomen | M | 14 | 11 | 9 | 7 | 5 | 3 | 2 | 1 | 0.4 | 0.1 |
| (AP) | F | 13 | 10 | 8 | 6 | 5 | 3 | 2 | 1 | 0.6 | 0.1 |
| Pelvis | M | 10 | 8 | 6 | 5 | 4 | 3 | 2 | 0.8 | 0.3 | 0.1 |
| (AP) | F | 8 | 6 | 5 | 4 | 3 | 2 | 2 | 0.9 | 0.4 | 0.1 |
| Lumbar spine | M | 14 | 11 | 9 | 7 | 5 | 3 | 2 | 0.9 | 0.4 | 0.1 |
| (AP+Lat) | F | 13 | 10 | 8 | 6 | 5 | 3 | 2 | 1 | 0.5 | 0.1 |
| IVU | M | 15 | 11 | 9 | 7 | 5 | 3 | 2 | 1 | 0.4 | 0.1 |
| | F | 14 | 11 | 9 | 6 | 5 | 3 | 2 | 1 | 0.5 | 0.1 |
| Ba swallow | M | 10 | 7 | 5 | 4 | 3 | 2 | 2 | 0.9 | 0.4 | 0.1 |
| | F | 31 | 18 | 12 | 8 | 6 | 4 | 3 | 2 | 0.8 | 0.2 |
| Ba follow | M | 14 | 11 | 8 | 6 | 5 | 3 | 2 | 0.9 | 0.4 | 0.1 |
| | F | 12 | 10 | 7 | 5 | 5 | 3 | 2 | 1 | 0.5 | 0.1 |
| Ba enema | M | 11 | 9 | 7 | 5 | 4 | 3 | 2 | 0.9 | 0.4 | 0.1 |
| | F | 9 | 7 | 6 | 4 | 4 | 3 | 2 | 0.9 | 0.4 | 0.1 |
| Coronary | M | 9 | 8 | 7 | 6 | 6 | 5 | 4 | 2 | 1 | 0.2 |
| angiography | F | 13 | 11 | 10 | 9 | 9 | 8 | 7 | 4 | 2 | 0.5 |
| Femoral | M | 12 | 10 | 7 | 6 | 5 | 3 | 2 | 0.9 | 0.4 | 0.1 |
| angiography | F | 10 | 8 | 6 | 4 | 4 | 3 | 2 | 0.9 | 0.4 | 0.1 |
| CT head | M | 14 | 11 | 7 | 5 | 4 | 3 | 1 | 0.7 | 0.3 | 0.1 |
| | F | 15 | 10 | 7 | 4 | 4 | 3 | 1 | 0.7 | 0.3 | 0.1 |
| CT chest | M | 9 | 8 | 6 | 5 | 4 | 3 | 2 | 1 | 0.6 | 0.1 |
| | F | 22 | 16 | 12 | 9 | 7 | 6 | 4 | 3 | 1 | 0.3 |
| CT abdomen | M | 14 | 11 | 9 | 7 | 5 | 3 | 2 | 0.9 | 0.3 | 0.1 |
| | F | 14 | 10 | 8 | 6 | 5 | 3 | 2 | 1 | 0.5 | 0.1 |
| CT abdomen + | M | 14 | 11 | 8 | 7 | 5 | 3 | 2 | 1 | 0.4 | 0.1 |
| pelvis | F | 13 | 10 | 8 | 6 | 5 | 3 | 2 | 1 | 0.5 | 0.1 |
| CT chest + | M | 11 | 9 | 7 | 6 | 5 | 3 | 2 | 1 | 0.5 | 0.1 |
| abdo + pelvis | F | 19 | 13 | 10 | 7 | 6 | 5 | 3 | 2 | 1 | 0.2 |

Note that the methodology used in these calculations is based on but slightly different from that of *Publication 103* (see section 2.6). Tabulated values are risk coefficients (per Sv), not absolute measures of risks from the various procedures from which the doses delivered are in the mSv range or lower.

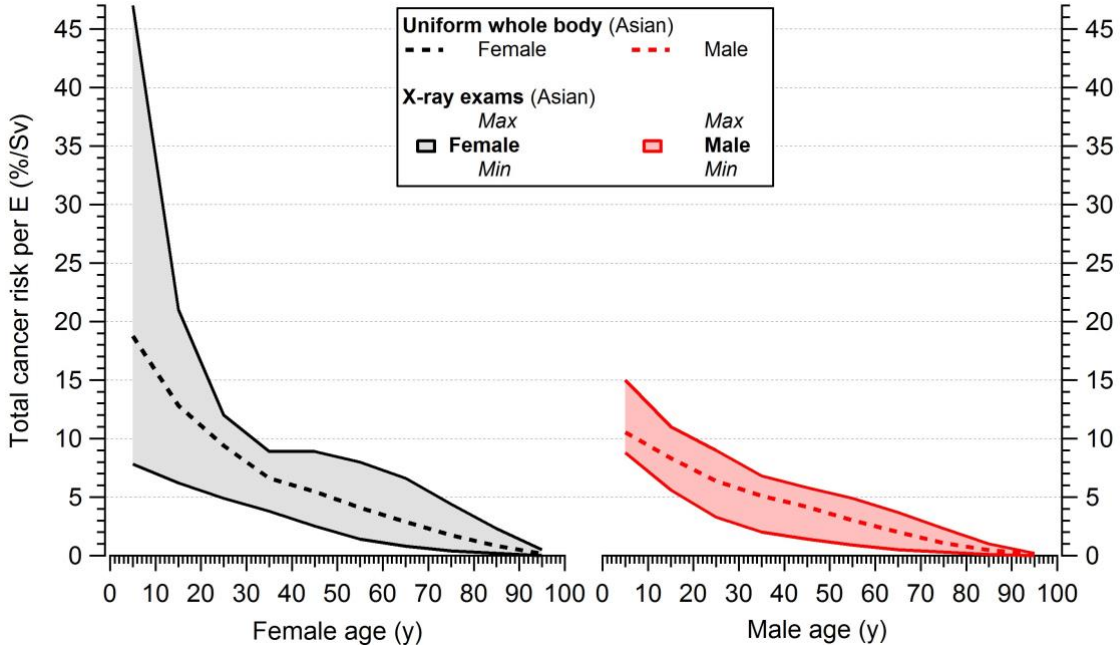


Fig. 5.1. Total lifetime risk of cancer incidence per unit effective dose (cases per 100 per Sv: %/Sv) as a function of age at exposure and sex for a range of x-ray examinations (Table 5.4) and for uniform whole-body exposure of a composite Asian population (Table 2.5). Note that the upper and lower curves show the maximum variation in overall lifetime risks per Sv resulting from the various combinations of organ / tissue doses for the different procedures and the application of specific risk models.

(119) The use of effective dose to provide an approximate indication of lifetime risk of cancer incidence associated with medical procedures is not a substitute for detailed assessments of risk for individuals or specific population groups. Risk assessment will always be based on measurements or estimates of mean absorbed doses to individual organs and use age at exposure- and sex-specific risk coefficients for the most appropriate population group. In cases of exposures involving high LET radiations, appropriate RBE values should be considered. For detailed analyses, absorbed dose estimates should take account of the size of the patient and other factors influencing the distribution of radiation dose within the patient's organs/tissues. For CT scans, doses to larger organs and ones that are located centrally within the scanned region decrease exponentially with trunk diameter (Li et al., 2011). Particular care should be taken when deriving doses for organs and tissues which lie near the boundary of the exposed region of the body, since these can vary substantially with small changes in exposure conditions. Patient-specific organ/tissue doses for CT may be calculated from sectional image data for the examination (Li et al., 2011) or adjustments to organ/tissue doses made based on patient dimensions or weight (Huda and He, 2012). Uncertainties in both dose and risk estimates should be considered.

# 6. SUMMARY AND CONCLUSIONS

(120) The Introduction to this report raised a number of issues for which clarification and guidance were required. This final section expands on the information provided as main points in the front of the document, answering the issues raised in the Introduction and draws together the conclusions reached.

(121) Effective dose (*E*) in sievert (Sv) is accepted internationally as the central radiological protection quantity, providing a risk-adjusted measure of total body dose from external and internal sources in relation to stochastic risks of cancer and hereditary effects, expressed in terms of detriment. *E* has proved to be a valuable and robust quantity for use in the optimisation of protection and setting of dose criteria to control exposures: dose limits, dose constraints and reference levels. The use of *E* relies on the prudent assumption of a linear-non-threshold (LNT) dose-response relationship between dose and risk at low doses/dose-rates, and the equivalence of effect of acute and chronic exposures at low doses/dose-rates, and of internal and external exposures. The LNT dose-response assumption, together with radiation and tissue weighting factors, underpin the use of effective dose as a protection quantity, allowing the addition of external and internal doses of different magnitudes, with different temporal and spatial patterns of delivery. However, it should be recognised that while low doses may be measured or estimated with reasonable reliability, the associated risk of stochastic health effects is uncertain, and increasingly uncertain as dose decreases. The available scientific evidence supports the assumptions of equivalence of acute and chronic exposures at low doses/dose-rates from external and internal sources of radiation. Notably, epidemiological data, supported by animal data, indicate that it is reasonable for protection purposes to assume equivalence of risk per unit dose, once simple adjustments are made to account for RBE, between short duration exposures to external penetrating low LET gamma rays and protracted internal exposures to alpha particle emitting radionuclides, for which tissue doses will be substantially more heterogeneous.

(122) Absorbed dose averaged over organs and tissues is the primary scientific quantity from which *E* is calculated. Absorbed dose (*D*) in gray (Gy) should be used to set limits on organ/tissue doses to prevent tissue reactions (deterministic effects) rather than equivalent dose (*H*) in Sv which relates to cancers and hereditary diseases (stochastic effects). The limits set to prevent tissues reactions, for the lens of the eye, skin and hands and feet, are relevant mainly to circumstances of exposure to penetrating low LET radiations. However, exposures to neutrons and other high LET radiations may require consideration in some situations and it may then be necessary to take account of increased effectiveness per Gy. This change to the use of absorbed dose rather than equivalent dose would not require changes to the numerical values of dose limits for tissue reactions and will be considered by the Commission when new general recommendations are formulated.

(123) The control of stochastic effects relies almost entirely on the use of effective dose. To the extent that it is necessary to consider organ and tissue doses, they are better expressed in terms of absorbed dose in gray (Gy), avoiding any potential confusion with effective dose in sievert (Sv). For example, an intake of iodine-131 might result in an effective dose of 10 mSv, largely contributed by a thyroid dose of 250 mGy (low LET). The discontinuation of the use of equivalent dose as a separate protection quantity will also avoid confusion between this quantity and dose equivalent (Sv), the measured operational quantity for external radiation used as an estimate of effective dose; the words dose and equivalent used together will then more readily be understood to refer to the operational quantity.

(124) Nominal stochastic risk coefficients and corresponding detriment values, to which *E* relates, are calculated for a composite of seven Euro-American and Asian populations,

applying to uniform whole-body exposures to external (low-LET) radiation of a population of both sexes and all ages. Internationally applicable values are provided for all workers (18-65 years) and the whole population. Tissue weighting factors ($w_T$) used in the calculation of effective dose are a simplified representation of relative detriment values, relating to detriment for the whole population; that is, simplified adjustments to take account of the contribution of individual organs and tissues to overall stochastic detriment. *E* is calculated for sex-averaged Reference Persons of specified ages. *Publication 103* (ICRP, 2007a) definition includes the specification of reference male and female anatomical models for radiation transport calculations. While exposures may relate to individuals or population groups, *E* is calculated for Reference Persons exposed in the same way.

(125) For the practical implementation of the radiological protection system, it is of considerable utility to be able to set dose criteria that apply to all members of the public or all workers. It has been argued that this approach does not adequately protect women and younger children and that differences between males and females and greater risks at younger ages should be reflected more explicitly in the ICRP system, including the use of different detriment values and $w_T$ values. In this context, it is notable that estimated differences in lifetime risk of cancer incidence between males and females and between age groups, as illustrated in Tables 2.4 and 2.5, are not large in the context of the practical application of the system of protection at low doses and uncertainties associated with estimates of risk at low doses. Central to the system is optimisation below dose constraints and reference levels, which should ensure protection of all groups within populations. Protection would not be improved by introducing separate considerations for males and females and for children of different ages, with different nominal risk coefficients and associated sets of tissue weighting factors. A distinction should be drawn between the use of scientific information to construct a workable and acceptable protection system and the use of science to provide best estimates of dose and risk to individuals and specific population groups (see below). The use of dose constraints and reference levels that apply to all workers and all members of the public, together with optimisation, provides a pragmatic, equitable and workable system of protection that recognises age-, sex-, and population-related differences in risks per Sv but does not distinguish on an individual basis. The only distinction made between males and females for protection purposes is the treatment of occupationally exposed females during declared pregnancy when the fetus is regarded as a member of the public for the purposes of dose limitation (ICRP, 2007a). Doses to children and the fetus are considered below.

(126) *Publication 103* (ICRP, 2007a) refers to setting of reference levels in relation to emergency planning and management in the range of 20-100 mSv effective dose. In principle, there is no reason why effective doses should not be used as a quantity at doses in the order of several 100 mSv: for example, as might be required to temporarily accept higher doses in order to control an accident situation. However, the potential for the occurrence of tissue reactions should be considered. For effective doses of up to a few hundreds mSv and for which irradiation is reasonably uniform, harmful tissue reactions would not be expected to occur, but if there was a significant contribution to the effective dose from radionuclides concentrated in particular organs (e.g. iodine-131 in the thyroid, inhaled insoluble radionuclides in the lung), tissue damage could occur. Notably, for $^{131}$I, for example, an effective dose of 250 mSv could correspond to a thyroid dose of > 6 Gy. A secondary consideration is that for doses in excess of 100 mSv (or more precisely, absorbed doses to organs and tissues > 100 mGy) delivered at high dose rate, the DDREF of 2 applied in determining solid cancer risk at low doses will not apply, so that risks may be somewhat greater than might be assumed on the basis of *Publication 103* (ICRP, 2007a) nominal risk coefficients.

(127) *E* is generally calculated for annual exposures, relating to external dose received in the year and committed dose from internal exposures, where committed dose is integrated over a 50 years period for workers and to age 70 years for members of the public. As discussed in *Publication 103* (ICRP, 2007a), committed dose is assigned to the year in which the intake occurred. For some radionuclides, with long half-lives and long biological retention times, only a small proportion of the committed dose is delivered in the year of intake. For plutonium-239, for example, effective dose in the first year after intake will be generally less than 10% of the total committed dose. For most radionuclides, however, this effect will be much less significant and for many, including iodine-131 and caesium-137, dose will be delivered entirely or very largely in the year of intake. For practical purposes, the use of committed dose ensures that longer term exposures from intakes of radionuclides are taken into account.

(128) Although effective dose coefficients are provided for a number of age groups of children, it is normally sufficient in public dose assessments to use only the groups 1 year, 10 years and adults. Representative Person is the term introduced in *Publication 101* (ICRP, 2006) to replace the concept of "critical group" and is an estimate of effective dose to a hypothetical person of specified age receiving a dose that is representative of the more highly exposed individuals in a population. Effective dose coefficients for the embryo/fetus following intakes of radionuclides are provided for comparison with dose for other age groups to ensure protection of the fetus, showing that it is only in the case of a few radionuclides that fetal doses may need in some circumstances to be considered.

(129) *E* is in widespread use in medical practice as an approximate indicator of risk. It is made clear in this report that while doses incurred at low levels of exposure may be measured or assessed with reasonable accuracy, the associated risks are uncertain. However, bearing in mind the uncertainties associated with risk projection to low doses/dose-rates, *E* may be considered as an approximate indicator of possible risk, with the additional consideration of variation in risk with age, sex and population group. In the majority of situations, simple qualitative descriptors of the possible risk associated with effective dose will be sufficient to inform judgements. It is emphasized that use of *E* as an approximate measure of possible risk is not a substitute for risk analysis using best estimates of organ/tissue doses, appropriate information on the relative effectiveness of different radiation types, and age-, sex- and population-specific risk factors applying to the organs/tissues at risk, with consideration of uncertainties.

(130) *E* can be used in medical applications to: compare doses from different diagnostic and interventional imaging modalities that give different spatial distributions of radiation within the body; provide a generic indicator for classifying different types of medical procedure into broad risk categories for the purpose of risk communication; informing decisions on justification of patient diagnostic and interventional procedures; planning requirements for research studies; and, initial evaluation of unintended exposures or overexposures of patients. However, for comparisons of doses from the same procedure in different facilities and for setting diagnostic reference levels, measurable dose quantities are preferable.

(131) Data are presented in this report to illustrate variation in cancer detriment per Sv for a range of medical x-ray procedures, assessed using age at exposure- and sex-specific risk factors calculated for the ICRP composite Euro-American and Asian populations. It should be recognised that these data are subject to substantial uncertainties inherent in their derivation and application to low dose radiation exposures. With this important caveat, it can be concluded that when considering most x-ray examinations, lifetime risk of cancer incidence per Sv may be around twice as great for the 0-9 years age at exposure group than

for the 30-39 years group. For patients in their 60s, the lifetime risks from most examinations are estimated to be about half those for patients in their 30s, falling to less than one-third for patients in their 70s and about one-tenth for those in their 80s. Used appropriately, such information is of value in helping clinicians understand the possible risks associated with examinations and assist in communication with patients. In considering doses to patients having diseases with poor prognoses, life-expectancy will be a consideration in evaluating radiation risks.

(132) The use of effective dose as an approximate indicator of stochastic risks can be reasonably extended beyond medical applications to, for example, consideration of protection options for accidental exposures of workers and members of the public. The same caveats apply, including the uncertainties in inferring risks at low doses. In all cases, exposures that are largely limited to a single organ/tissue should be assessed using organ/tissue dose and organ/tissue-specific risk coefficients, as for example, in exposures of the thyroid following intakes of radioactive iodine.

(133) Collective effective dose is a valuable tool in the optimisation of protection, particularly for occupational exposures. Collective effective dose can be used to determine the optimum balance between relatively large exposures to a few workers and smaller exposures to a larger number of workers. For public exposures, collective effective doses can be used as part of the optimisation process for planned, existing and emergency exposure situations. They also have a useful role in comparative studies to consider the effects of adopting different systems of treatment for radioactive wastes or the radiological impact of different sources of exposure.

(134) Collective dose is not intended as a tool for the prediction of health effects in populations and epidemiological analysis and particular care is needed in interpreting collective dose data made up of extremely low (μSv or nSv) levels of individual dose received over long time periods by large numbers of people (ICRP, 2007a). However, there can be situations where the estimation of health effects from collective doses can be useful if treated with appropriate caution, for example, to inform judgements on the need for medical or epidemiological follow-up. It is essential that such analyses using collective dose include consideration of background rates of health effects in the population, including morbidity and mortality, and consider uncertainties.

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