follow-up period is 41 years (January 1958 – December 1998). These latter data, which were not available in 1990, can provide more reliable estimates of risk principally because cancer incidence allows for more accurate diagnosis. The Commission has therefore placed emphasis on incidence data for its present recommendations. In addition, epidemiological data from the LSS provide further information on the temporal and age-dependent pattern of radiation cancer risk, particularly the assessment of risk amongst those exposed at early ages. Overall, current cancer risk estimates from the LSS are not greatly changed since 1990 but the improved quality of the cancer incidence data provide a more firm foundation for the risk modelling described in Annex A.

(68) The LSS is not, however, the sole source of information on radiation cancer risk and the Commission has considered data from medical, occupational and environmental studies (UNSCEAR 2000, NAS/NRC 2006). For cancers at some sites there is reasonable compatibility between the data from the LSS and those from other sources. However it is recognised by the Commission that for a number of organs/tissues there are indications of differences in radiation risk estimates among the various data sets, with the LSS estimates being generally higher. Most studies on environmental radiation exposures currently lack sufficient data on dosimetry and tumour ascertainment to contribute directly to risk estimation by the Commission but are expected to be a potentially valuable data source in the future.

(69) A dose and dose-rate effectiveness factor (DDREF) has been used by the Commission to project cancer risk determined at high doses and high dose rates to the risks that would apply at low doses and low dose rates. In general, cancer risk at these low doses and low dose rates is judged, from a combination of epidemiological, animal, and cellular data, to be reduced by the value of the factor ascribed to DDREF. In its 1990 Recommendations the Commission made the broad judgement that a DDREF of 2 should be applied for the general purposes of radiological protection.

(70) In principle, epidemiological data on protracted exposure, such as those from environmental and occupational circumstances, should be directly informative on judgements of DDREF. However the statistical precision afforded by these studies and other uncertainties associated with the inability to adequately control for confounding factors (see Annex A), do not allow for a precise estimate of DDREF at this time. Accordingly the Commission has decided to continue to use broad judgements in its choice of DDREF based upon dose-response features of experimental data, the LSS, and the results of probabilistic uncertainty analysis conducted by others (NCRP 1997, EPA 1999, NCI/CDC 2003, Annex A).

(71) The BEIR VII Committee (NAS/NRC 2006) recently undertook probabilistic analyses. The approach taken was a Bayesian analysis of combined dose-response data. The data sets considered were a) solid cancer in the LSS; b) cancer and life shortening in animals; and c) chromosome aberrations in human somatic cells. The modal value of DDREF from these analyses was 1.5 with a range of 1.1 to 2.3 and the BEIR VII Committee chose the value of 1.5. However a DDREF of 2 was compatible with these data and the Committee recognised the subjective and probabilistic uncertainties inherent in this specific choice. Further, the BEIR VII Committee noted that for the induction of gene and chromosomal mutations values of DDREF generally fall in the range of 2-4, and for the induction of cancer in animals and life shortening in animals values of DDREF generally fall in the range of 2-3. The Commission emphasises that a DDREF is considered for

solid cancers and not leukaemia for which a linear-quadratic response is seen, i.e. a lower risk per unit dose at low doses than at high doses.

(72) In considering all the data noted above, and recognising the broad range of experimental animal data showing reduction in carcinogenic effectiveness and life-shortening following protracted exposures, the Commission finds no compelling reason to change its 1990 recommendations of a DDREF of 2. However, the Commission emphasises that this continues to be a broad whole number judgement for the practical purposes of radiological protection which embodies elements of both subjective and probabilistic uncertainty. This risk reduction factor of 2 is used by the Commission to derive the nominal risk coefficients for cancer overall given in Table 1 but the Commission recognises that, in reality, different dose and dose rate effects may well apply to different organs/tissues.

### 3.2.2 Risk of hereditary effects

(73) Although there continues to be no direct evidence that exposure of parents to radiation leads to excess heritable disease in offspring, the Commission judges that there is compelling evidence that radiation causes mutation in reproductive (germ) cells in experimental animals. Accordingly, the risk of hereditary effects continues to be included in the Commission's system of radiological protection. The Commission also notes reports (reviewed in UNSCEAR, 2001) which argue, on the basis of A-bomb survivor and mouse genetic data, that the risk of heritable diseases tended to be overestimated in the past.

(74) There are some post-1990 human and animal data on the quantitative aspects of radiation-induced germ cell mutation that impact on the Commission's judgement on the risk of induction of genetic disease expressing in future generations. There have also been substantial advances in the fundamental understanding of human genetic diseases and the process of germ line mutagenesis including that occurring after radiation. The Commission has re-appraised the methodology used in *Publication 60* for the estimation of hereditary risks including risks of multifactorial diseases (*Publication 83*; ICRP, 1999b). The Commission has now adopted a new framework for the estimation of hereditary risks that employs data from human and mouse studies (UNSCEAR, 2001; NAS/NRC, 2006). Also, for the first time, a scientifically justified method for the estimation of risk of multifactorial disease has been included. Mouse studies continue to be used to estimate genetic risks because of the lack of clear evidence in humans that germline mutations caused by radiation result in demonstrable genetic effects in offspring.

(75) The new approach to hereditary risks continues to be based on the concept of the doubling dose (DD) for disease-associated mutations used in *Publication 60*. However, the methodology differs in that recoverability of mutations in live births is allowed for in the estimation of DD. An additional difference is that direct data on spontaneous human mutation rates are used in conjunction with radiation-induced mutation rates derived from mouse studies. This new methodology (see Annex A, Box 2) is based on the UNSCEAR 2001 report and has also been used recently by NAS/NRC (2006). The present ICRP estimate of the second generation risk of about 0.2% per Gy is essentially the same as that cited by UNSCEAR 2001 (see Annex A and UNSCEAR 2001, Table 46). However, given the major changes in methodology, the close similarity of the present 2nd generation risk to that of *Publication 60* is wholly coincidental. In *Publication 60* genetic risks were expressed at a theoretical equilibrium between mutation and selection. In the light of

further knowledge the Commission judges that many of the underlying assumptions in such calculations are no longer sustainable. The same view has been expressed by UNSCEAR (2001) and NAS/NRC (2006). Accordingly the Commission now expresses genetic risks up to the second generation and judges that this procedure will not lead to a significant underestimation of genetic risk. This issue is discussed in detail in Annex A where it is argued on the basis of UNSCEAR calculations (UNSCEAR 2001) that there are no substantial differences between genetic risks expressed at 2 and 10 generations.

(76) The new estimate for genetic risks up to the second generation is around 0.2% per Sv. This value relates to continuous low dose-rate exposures over these two generations, i.e., doses to the parental and child generations and effects observed in children and grandchildren. As a result, these revised estimates of genetic risk have reduced the judged value of the tissue weighting factor for the gonads considerably (see Chapter 4). However, the Commission emphasises that this reduction in the gonadal tissue weighting factor provides no justification for allowing controllable gonadal exposures to increase in magnitude.

### 3.2.3 Detriment-adjusted nominal risk coefficients for cancer and hereditary effects

(77) New information on the risks of radiation-induced cancer and hereditary effects has been used in risk modelling and disease detriment calculations in order to estimate sex-averaged nominal risk coefficients.

(78) It remains the policy of the Commission that its recommended nominal risk coefficients should be applied to whole populations and not to sub-groups therein. The Commission believes that this policy provides for a general system of protection that is simple and sufficiently robust. In retaining this policy the Commission does however recognise that there are significant differences in risk between males and females (particularly for the breast) and in respect of age at exposure. Annex A provides data and calculations relating to these differences.

(79) The calculation of sex-averaged nominal risk coefficients for cancer involves the estimation of nominal risks for different organs and tissues, adjustment of these risks for lethality and quality of life and, finally, the derivation of a set of site-specific values of relative detriment, which includes heritable effects from gonadal exposures. These relative detriments provide the basis of the Commission's system of tissue weighting which is explained in Annex A (Box 1) and summarised in Chapter 4.

(80) On the basis of these calculations the Commission proposes nominal risk coefficients for detriment-adjusted cancer risk as $5.5\ 10^{-2}$ $Sv^{-1}$ for the whole population and $4.1\ 10^{-2}$ $Sv^{-1}$ for adult workers. For hereditary effects, the detriment-adjusted nominal risk in the whole population is estimated as $0.2\ 10^{-2}$ $Sv^{-1}$ and in adult workers as $0.1\ 10^{-2}$ $Sv^{-1}$. These estimates are shown in Table 1, where they are compared with the estimate of detriment used in the 1990 Recommendations in *Publication 60* (ICRP, 1991b).

(81) The most significant change from *Publication 60* is the 6-8 fold reduction in the nominal risk coefficient for hereditary effects. This reduction comes about mainly because the Commission has chosen to express such risks up to the second

generation rather than at a theoretical equilibrium. This change is discussed and justified in Annex A.

Table 1. Detriment-adjusted nominal risk coefficients for cancer and hereditary effects ($10^{-2}$ $Sv^{-1}$)

| Exposed population | Cancer | | Heritable effects | | Total | |
|---|---|---|---|---|---|---|
| | Present[1] | *Publ. 60* | Present[1] | *Publ. 60* | Present[1] | *Publ. 60* |
| Whole | 5.5 | 6.0 | 0.2 | 1.3 | 6.0 | 7.3 |
| Adult | 4.1 | 4.8 | 0.1 | 0.8 | 4.0 | 5.6 |

[1]Values from Annex A.

(82) Note that although all coefficients are presented as fractional values, this presentation is used for the purposes of traceability to Annex A only and does not imply a level of precision (see paragraphs 78 and 79).

(83) The present detriment-adjusted nominal risk coefficient for cancer shown in Table 1 has been computed in a different manner from that of *Publication 60*. The present estimate is based upon lethality and life impairment weighted data on cancer incidence, whereas in *Publication 60* detriment was based upon fatal cancer risk weighted for non-fatal cancer, relative life lost for fatal cancers and life impairment for non-fatal cancer.

(84) In spite of changes in the cancer risk data and their treatment, the present nominal risk coefficients are wholly compatible with those presented by the Commission in *Publication 60* (ICRP 1990). Given the uncertainties discussed in Annex A, the Commission considers that the small reduction in the estimate of nominal risk since 1990 is of no practical significance.

(85) It is therefore the recommendation of the Commission that the approximated overall risk coefficient of 5% per Sv on which current international radiation safety standards are based continues to be appropriate and should be retained for the purposes of radiological protection.

### 3.2.4 Radiation effects in the embryo and fetus

(86) The risks of tissue reactions and malformation in the irradiated embryo and fetus have been reviewed in *Publication 90* (ICRP, 2003a). In the main, this review reinforced the judgements on in-utero risks given in *Publication 60* although on some issues new data allow for clarification of views. On the basis of *Publication 90*, the Commission has reached the following conclusions on the in-utero risks of tissue injury and malformation at doses below about 100 mGy of low LET radiation.

(87) The new data confirm embryonic susceptibility to the lethal effects of irradiation in the pre-implantation period of embryonic developments. At doses under 100 mGy, such lethal effects will be very infrequent.

(88) In respect of the induction of malformations, the new data strengthen the view that there are gestation age-dependent patterns of in-utero radiosensitivity with

maximum sensitivity being expressed during the period of major organogenesis. On the basis of animal data it is judged that there is a true dose-threshold of around 100 mGy for the induction of malformations; therefore, for practical purposes, the Commission judges that risks of malformation after in-utero exposure to doses well below 100 mGy are not expected.

(89) The *Publication 90* (ICRP, 2003a) review of A-bomb survivor data on the induction of severe mental retardation after irradiation in the most sensitive pre-natal period (8-15 weeks post-conception) now supports a true dose-threshold of at least 300 mGy for this effect and therefore the absence of risk at low doses. The associated data on IQ losses estimated at around 25 points per Gy are more difficult to interpret and the possibility of a non-threshold dose response cannot be excluded. However, even in the absence of a true dose-threshold, any effects on IQ following in utero doses under 100 mGy would be of no practical significance. This judgement accords with that developed in *Publication 60* (ICRP, 1991b).

(90) *Publication 90* also reviewed data concerning cancer risk following in-utero irradiation. The largest studies of in-utero medical irradiation provided evidence of increased childhood cancer of all types. The Commission recognises that there are particular uncertainties on the risk of radiation-induced solid cancers following in-utero exposure. Nonetheless, the Commission considers that it is prudent to assume that life-time cancer risk following in-utero exposure will be similar to that following irradiation in early childhood i.e. at most, a few times that of the population as a whole.

### 3.2.5 Genetic susceptibility to cancer

(91) The issue of individual genetic differences in susceptibility to radiation-induced cancer was noted in *Publication 60* and reviewed in *Publication 79* (ICRP, 1999a). Since 1990, there has been a remarkable expansion in knowledge of the various single gene human genetic disorders, where excess spontaneous cancer is expressed in a high proportion of gene carriers – the so-called high penetrance genes which can be strongly expressed as excess cancer. Studies with cultured human cells and genetically altered laboratory rodents have also contributed much to knowledge and, with more limited epidemiological and clinical data, suggest that most of the rare single gene, cancer prone disorders will show greater-than-normal sensitivity to the tumorigenic effects of radiation.

(92) There is also a growing recognition, with some limited supporting data, that variant genes of lower penetrance through gene-gene and gene-environment interactions can result in a highly variable expression of cancer following radiation exposure.

(93) On the basis of the data and judgements developed in *Publication 79* and further information reviewed in the UNSCEAR (2000; 2001) and NAS/NRC (2006) reports, the Commission believes that strongly expressing, high penetrance, cancer genes are too rare to cause significant distortion of population-based estimates of low dose radiation cancer risk. However, there are likely to be implications for individual cancer risks, particularly for second cancers in gene carriers receiving high-dose radiotherapy for a first neoplasm; although the features of low-dose radiation risk are not entirely clear.

(94) Although the Commission recognises that variant cancer genes of low penetrance may, in principle, be sufficiently common to impact upon population-based estimates of radiation cancer risk, the information available is insufficient to provide a meaningful quantitative judgement on this issue.

### 3.3 The induction of diseases other than cancer

(95) Since 1990 evidence has accumulated that the frequency of non-cancer diseases is increased in some irradiated populations. The strongest statistical evidence for the induction of these non-cancer effects at effective doses of the order of 1 Sv derives from the most recent mortality analysis of the Japanese atomic bomb survivors followed after 1968 (Preston et al., 2003). That study has strengthened the statistical evidence for an association with dose – particularly for heart disease, stroke, digestive disorders and respiratory disease. However, the Commission notes current uncertainties on the shape of the dose-response at low doses and that the LSS data are consistent both with there being no dose threshold for risks of disease mortality and with there being a dose threshold of around 0.5 Sv. Additional evidence of the non-cancer effects of radiation, albeit at high doses, comes from studies of cancer patients receiving radiotherapy but these data do not clarify the issue of a possible dose threshold (Annex A). It is also unclear what forms of cellular and tissue mechanisms might underlie such a diverse set of non-cancer disorders.

(96) Whilst recognising the potential importance of the observations on non-cancer diseases, the Commission judges that the data available do not allow for their inclusion in the estimation of detriment following radiation doses less than around 100 mSv.

# 4. QUANTITIES USED IN RADIOLOGICAL PROTECTION

## 4.1. Introduction

(97) Radiological protection is concerned with controlling exposures to ionising radiation, so that the risk of radiation-induced cancer and hereditary disease (stochastic effects) is limited to acceptable levels and tissue reactions (deterministic effects) are prevented. For assessing doses from radiation exposures, special *dosimetric quantities* have been developed. The fundamental *protection quantities* adopted by the Commission are based on measures of the energy deposited in organs and tissues of the human body. For relating the radiation dose to radiation risk (detriment), it is also necessary to take into account variations in the biological effectiveness of radiations of different quality as well as the varying sensitivity of organs and tissues to ionising radiation.

(98) In *Publication 26* (ICRP, 1977) the protection quantities *dose equivalent*, for organs and tissues of the human body, and *effective dose equivalent* were introduced. The definition and method of calculation of these quantities were modified in *Publication 60* (ICRP, 1991b) to give the quantities *equivalent dose* and *effective dose*. The development of the quantities effective dose equivalent and effective dose has made a significant contribution to radiological protection as it has enabled doses to be summed from whole and partial body exposure from external radiation of various types and from intakes of radionuclides.

(99) Equivalent dose and effective dose cannot be measured directly in body tissues. The protection system therefore includes *operational quantities* that can be measured and from which the equivalent dose and the effective dose can be assessed.

(100) The general acceptance of effective dose and the demonstration of its utility in radiological protection are important reasons for maintaining it as the central quantity for dose assessments in radiological protection. There are, however, a number of aspects of the dosimetry system given in *Publication 60* that need to be addressed and clarified as summarised below and given in more detail in Annex B. Care is also needed in describing the situations in which effective dose should be and should not be used. In some situations tissue absorbed dose or equivalent dose are more appropriate quantities.

## 4.2. Considerations of health effects

(101) Radiological protection in the low dose range is primarily concerned with protection against radiation-induced cancer and hereditary disease. These effects are taken to be probabilistic in nature and to increase in frequency in proportion to the radiation dose, with no threshold (see Chapter 3 or Annex A). For the definition and calculation of effective dose the recommended radiation weighting factors, $w_R$, allow for the differences in the effect of various radiations in causing stochastic effects while tissue weighting factors, $w_T$, allow for the variations in radiation sensitivity of different organs and tissues to the induction of stochastic effects (see

Section 4.3.4 and Annex B). The radiation weighting factors for radiations characterised by a high linear energy transfer, so called high-LET radiations (see Section 4.3.3), are derived for stochastic effects at low doses.

(102) At high doses and especially in emergency situations, radiation exposures may cause tissue reactions (deterministic effects). Such clinically observable damage occurs above threshold doses. The extent of damage depends upon the absorbed dose and dose rate as well as radiation quality (see Annexes A and B) and the sensitivity of the tissue. In general, values of relative biological effectiveness (RBE) for tissue reactions caused by high-LET radiations are found to be lower than those obtained for stochastic effects at low doses and the relative sensitivity of tissues also differs. The quantities equivalent dose and effective dose should not be used in the quantification of higher radiation doses and in making decisions on the need for any treatment related to tissue reactions. For such purposes, doses should be evaluated in terms of absorbed dose (in gray, Gy) and where high-LET radiations (e.g. neutrons or alpha particles) are involved, an absorbed dose weighted with an appropriate RBE, should be used (see Annex B).

## 4.3. Dose quantities

(103) The procedure for the assessment of effective dose adopted by the Commission is to use *absorbed dose* as the fundamental physical quantity, to average it over specified organs and tissues, to apply suitably chosen 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. Values of the equivalent dose to organs and tissues weighted for the radiosensitivity of these organs and tissues are then summed to give the effective dose. This quantity is based on the exposure to radiation from external radiation fields and from incorporated radionuclides as well as on the primary physical interactions in human tissues and on judgements about the biological reactions resulting in stochastic health effects (Annex B).

### 4.3.1. Absorbed dose

(104) 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

$$D = \frac{\mathrm{d}\bar{\varepsilon}}{\mathrm{d}m} \tag{4.1}$$

(105) The SI unit of absorbed dose is $\mathrm{J\,kg^{-1}}$ and its special name is gray (Gy). Absorbed dose is derived from the mean value of the stochastic quantity of energy imparted, $\varepsilon$, 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 $\mathrm{d}m$ 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 (Annex B).

### 4.3.2. Averaging of dose

(106) When using the quantity absorbed dose in practical protection applications, 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 doses in tissues or organs and the summing of weighted mean doses in different organs and tissues of the human body comprise the basis for the definition of the protection quantities which are used for limiting stochastic effects at low doses. This approach is based upon the assumption of a linear, non-threshold, dose-response relationship (LNT) and allows the addition of doses for external and internal exposure.

(107) The averaging of absorbed dose is carried out over the mass of a specified organ (e.g. liver) or tissue (e.g. muscle) or the sensitive region of a tissue (e.g. endosteal surfaces of the skeleton). The extent to which the mean dose value is representative of the absorbed dose in all regions of the organs, tissues or tissue regions depends for external irradiation on the homogeneity of the exposure and on the range of the radiation incident on the body. The homogeneity of the dose distribution in the low dose range depends also upon microdosimetric properties. For radiations with low penetration or limited range (e.g., low-energy photons or charged particles) as well as for widely distributed tissues and organs (e.g. red bone marrow, lymphatic nodes or skin) the absorbed dose distribution within the specified organ or tissue will be even more heterogeneous. In cases of extreme partial body exposure, tissue damage may occur even if the mean organ or tissue dose or the effective dose is below the dose limit. A special limit on local skin dose, for example, takes account of this situation in the case of exposure by low-penetrating radiation.

(108) For radiations emitted by radionuclides retained within body organs or tissues, so-called internal emitters, the absorbed dose distribution in organs depends on the penetration and range of the radiations. Thus, the absorbed dose distribution for radionuclides emitting alpha particles, soft beta particles, low-energy photons or Auger electrons may be highly heterogeneous (see Annex B). This heterogeneity applies in particular to radionuclides in the respiratory and alimentary systems, and the skeleton. Specific dosimetric models have been developed to take account of such heterogeneity in the distribution and retention of activity and of sensitive regions in these particular cases.

### 4.3.3. Equivalent dose and radiation weighting factors

(109) The protection quantities are used to specify exposure limits for keeping the occurrence of stochastic health effects below unacceptable levels and for avoiding tissue reactions in workers and members of the public. The definition of the protection quantities is based on the average absorbed dose, $D_{T,R}$ in the volume of a specified organ or tissue T (see Table 3), due to radiation of type R (see Table 2). The radiation $R$ is 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

$$H_T = \sum_R w_R D_{T,R} \qquad (4.2)$$

where $w_R$ is the radiation weighting factor for radiation 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).

(110) In the early 1960s, radiation weighting in the definition of radiological protection quantities was related to the radiation quality factor 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 was changed for calculating the protection quantities equivalent dose and effective dose. The Commission selected a general set of radiation weighting factors ($w_R$) that were considered to be appropriate for application in radiological protection. The values of $w_R$ were defined largely on the basis of the relative biological effectiveness (RBE) of the different radiations.

(111) A revised set of $w_R$ values has been adopted in these recommendations based upon a re-evaluation of the available data (see Annexes A and B). The values of $w_R$ for neutrons and protons given in these recommendations differ from those given in *Publication 60* (see below and Annex B). A $w_R$ value for charged pions has been included. The value of $w_R$ for photons is the same for x rays and gamma rays of all energies. The numerical values of $w_R$ are specified in terms of type and in the case of neutrons in terms of energy of radiation either incident on the human body or emitted by radionuclides residing in the body (Table 3). The values of $w_R$ are selected by judgement from a broad range of experimental RBE data which are relevant to stochastic effects. The RBE values increase to a maximum ($RBE_M$) with decreasing radiation dose (ICRP, 2003c). The values of $RBE_M$ have been used for $w_R$ selection and are assigned fixed values for radiological protection purposes.

Table 2. Recommended 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 (see Fig. 1 and Equation 4.3) |

All values relate to the radiation incident on the body or, for internal radiation sources, emitted from the incorporated radionuclide(s).

(112) ***Reference radiation.*** Values of RBE obtained experimentally depend on the reference radiation chosen. Generally low-LET photon radiation is taken as the reference although no specific energy has been agreed upon for this purpose. For the selection of radiation weighting factors in *Publication 60*, a broad range of experimental RBE data using either high energy x rays above about 200 kV or $^{60}Co$

or $^{137}Cs$ gamma radiation was considered (see Annex B). This approach is also used in these recommendations.

(113) ***Photons, electrons, and muons.*** Photons, electrons, and muons are radiations with LET values of less than 10 keV/μm. These radiations have always been given a radiation weighting of 1. There are good arguments (see Annex B) to continue to use a $w_R$ of 1 for all low-LET radiations (Annex B, Table 3). This does not, however, imply that there are no differences in radiation quality of photons of different energies. The proposed simplification is sufficient only for the intended application of equivalent dose and effective dose, e.g. for dose limitation, assessment and controlling of doses in the low dose range. In cases where individual retrospective risk assessments have to be made, more detailed information on the radiation field and appropriate RBE values may need to be considered if relevant data are available. Heterogeneity of the radiation dose within cells, as can occur with tritium or Auger emitters incorporated into DNA, may also require specific analysis (see Annex B).

(114) ***Neutrons.*** 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 Annex B).

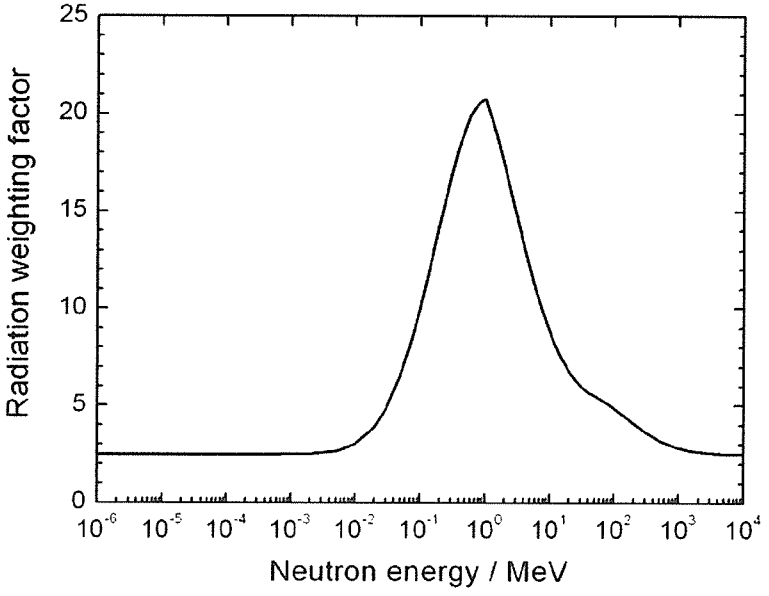


Fig. 1. Radiation weighting factor, $w_R$, for neutrons versus neutron energy.

(115) In *Publication 60* (ICRP, 1991b), the radiation weighting factor for neutrons was defined by a step function. It is now recommended that the radiation weighting factor for neutrons be defined by a continuous function (Fig. 1). It should be noted, however, that the use of a continuous function is based on the practical consideration that most neutron exposures involve a range of energies. The recommendation of the function does not imply a higher precision of the basic data. A detailed discussion on the selection of the $w_R$-function for neutrons is given in Annex B. The most significant changes compared to the data in *Publication 60* are the decrease of $w_R$ in the low-energy range, which takes account of the large contribution of secondary photons to the absorbed dose in the human body, and the decrease of $w_R$ at neutron energies above 100 MeV. The following continuous

function in neutron energy $E_n$ (MeV) is recommended for the calculation of radiation weighting factors for neutrons:

$$w_R = \begin{cases} 2.5 + 18.2\, e^{-[\ln(E_n)]^2/6} & , \quad E_n < 1 \text{ MeV} \\ 5.0 + 17.0\, e^{-[\ln(2E_n)]^2/6} & , \quad 1 \text{ MeV} \le E_n \le 50 \text{ MeV} \\ 2.5 + 3.25 e^{-[\ln(0.04E_n)]^2/6} & , \quad E_n > 50 \text{ MeV} \end{cases} \qquad (4.3)$$

This function, i.e., equation (4.3) and Fig. 1, has been derived empirically and is consistent with existing biological and physical knowledge (Annex B).

(116) ***Protons and pions.*** When considering exposure to protons, only external radiation sources are of importance in practical radiological protection. In the proton component of cosmic radiation fields or fields near high-energy particle accelerators, very high-energy protons dominate and protons with energies of few MeV are of minor significance even when their increased biological effectiveness at low energies is taken into account. For radiological protection, it is judged to be sufficiently accurate 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. The range of 10 MeV protons in tissue is 1.2 mm and decreases with lower energies. These protons will be absorbed in skin. (Annex B). A single radiation weighting factor of 2 is recommended for external proton radiation for general use (ICRP 2003c). It replaces the value of 5 recommended in *Publication 60* (ICRP 1991b).

(117) 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 the exposure in aircraft. They are also found as part of the complex radiation fields behind shielding of high-energy particle accelerators and thus contribute to the occupational exposure of accelerator staff. Considering that the energy distribution of pions in radiation fields is very broad, the use of a single weighting factor of 2 is recommended for all charged pions.

(118) ***Alpha particles.*** Humans may be exposed to alpha particles from internal emitters, e.g. from inhaled radon progeny or ingested alpha-emitting radionuclides such as isotopes of plutonium, polonium, radium, thorium and uranium. There are a number of epidemiological studies, as well as animal data, that provide information on the risk from incorporated alpha emitters. However, the distribution of radionuclides in organs and tissues is complex and the estimation of dose is dependent on the models used. Hence the calculated doses are associated with substantial uncertainties and result in a broad range of RBE values from epidemiological as well as experimental studies (*Publication 92*, ICRP 2003c, and Annex B).

(119) Despite substantial uncertainties in estimates of dose and risk from intakes of alpha emitting radionuclides, the available human and animal data indicate that the RBE depends on the biological end-point under consideration. The limited human data that allow estimation of alpha particle RBE values suggest values of around 10 – 20 for lung and liver cancer and lower values for bone cancer and leukaemia. Judgements on the available data and the selection of a $w_R$ value for alpha particles have been reviewed in *Publication 92* (ICRP, 2003c). As recent data do not provide compelling evidence for a change of the radiation weighting factor

for alpha particles, the $w_R$ value of 20 adopted in *Publication 60* (ICRP, 1991b) is retained.

(120) ***Fission fragments and heavy ions.*** Doses from fission fragments are of importance in radiological protection, mainly in internal dosimetry, and the situation regarding radiation weighting factors is similar to that for α-particles. The short ranges of heavy ions and fission fragments in organs and tissues and the resulting ionisation density have a strong influence on their biological effectiveness. A radiation weighting factor of 20 (see Table 2), which equals that for α-particles, is recommended (see Annex B).

(121) Heavy ions are encountered in external radiation fields in air flight at high altitudes and in space exploration. There are very limited RBE data for heavy ions and most of these are based on *in vitro* experiments. For heavy charged particles incident on and stopped in the human body the radiation quality of the particle changes markedly along its track. The selection of a single $w_R$ value of 20 for all types and energies of heavy charged particles is a conservative estimate and is recommended as sufficient for general application in radiological protection. For applications in space, where these particles contribute significantly to the total dose in the human body, a more realistic approach may have to be used.

### 4.3.4. Effective dose and tissue weighting factors

(122) The effective dose, $E$, introduced in *Publication 60* (ICRP, 1991b) is defined as:

$$E = \sum_T w_T \sum_R w_R D_{T,R} = \sum_T w_T H_T \qquad (4.4)$$

where $w_T$ is the tissue weighting factor for tissue, T and $\Sigma w_T = 1$. The sum is performed over all organs and tissues of the human body considered to be sensitive to the induction of stochastic effects. These $w_T$ values are chosen to represent the contributions of individual organs and tissues to overall radiation detriment from stochastic effects. The unit of effective dose is J $kg^{-1}$ with the special name sievert (Sv). The unit is the same for equivalent dose and effective dose as well as for some operational dose quantities (see Section 4.3.7). Care must be taken to ensure that the quantity being used is clearly stated.

(123) The organs and tissues for which $w_T$ values are specified are given in Table 3 (also see Annex A). They represent mean values for humans averaged over both sexes and all ages and thus do not relate to the characteristics of particular individuals.

(124) On the basis of epidemiological studies on cancer induction in exposed populations and risk assessments for hereditary effects a set of $w_T$ values were chosen for the new recommendations (Table 3) based on the respective values of relative radiation detriment (see Table 5, Annex A). In addition, the following judgements were applied:

- The detriments for heritable effects and cancer following gonadal irradiation (i.e., to ovaries or testes) were combined to give a $w_T$ of 0.08.

- The thyroid weighting factor was set to 0.04 due to the higher risk of thyroid cancer in childhood, i.e., young children are considered to be a particularly radiosensitive sub-group.

- Cancer risks in salivary glands and brain, whilst not precisely quantified, are judged to be greater than that of the other tissues and organs comprising the remainder fraction, and for this reason each is ascribed a $w_T$ of 0.01 (see Annex A)

- For the purposes of radiological protection, the $w_T$ values are assumed to be valid for both sexes and all age groups.

(125) The $w_T$ for the remainder tissues (0.12) applies to the weighted mean dose of the 13 organs and tissues for each sex listed in the footnote to Table 3. The so-called splitting rule in the treatment of the remainder in *Publication 60* (ICRP 1991b) is no longer used and hence the effective dose is additive. The sum of the $w_T$ values is 1 by definition (see explanations below and Annex B for further details).

Table 3. Recommended tissue weighting factors.

| Tissue | $w_T$ | $\sum w_T$ |
|---|---|---|
| Bone-marrow (red), 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: Adrenals, Extrathoracic (ET) region, Gall bladder, Heart, Kidneys, Lymphatic nodes, Muscle, Oral mucosa, Pancreas, Prostate (♂), Small intestine, Spleen, Thymus, Uterus/cervix (♀).

### 4.3.5. Sex averaging

(126) In radiological protection it is useful to determine a single value of effective dose for both sexes (see paragraph 33). Therefore, the tissue weighting factors of Table 3 are sex-averaged values and are valid for the male and female breast, testes and ovaries (carcinogenic and hereditary effects) taken together in the value for the gonads, and other organs and tissues with assigned explicit $w_T$ values. The effective dose is computed from the equivalent dose assessed for organ or tissue $T$ of the male, $H_T^M$, and female, $H_T^F$, including the remainder tissues, as in the following equation (see Annex B)

$$E = \sum_T w_T \left[ \frac{H_T^M + H_T^F}{2} \right] \quad (4.5)$$

(127) Analogous to the approach for other organs and tissues the equivalent dose to the remainder is defined separately for males and females and these values are included in Equation (4.5). The equivalent dose to the remainder tissues is computed as the arithmetic mean of the equivalent doses to the tissues listed in the footnotes to

Table 3. The equivalent dose to the tissues of remainder of the male, $H_{rem}^{M}$, and female, $H_{rem}^{F}$, are computed as

$$H_{\text{rem}}^{\text{M}} = \frac{1}{13}\sum_{\text{T}}^{13} H_{\text{T}}^{\text{M}} \quad \text{and} \quad H_{\text{rem}}^{\text{F}} = \frac{1}{13}\sum_{\text{T}}^{13} H_{\text{T}}^{\text{F}} \quad . \qquad (4.6)$$

The summation in Equation (4.5) extends over the equivalent dose to remainder tissues in the male and female (Annex B).

(128) The effective dose for protection purposes is based on the mean doses in organs or tissues of the human body. It is defined and estimated in a reference person. The quantity provides a value which takes account of the given exposure situation but not of the characteristics of a specific individual. In particular, the weighting factors are mean values representing an average over many individuals of both sexes. The reference person can be either a worker or a member of the public represented by defined individual exposure conditions, habits and age group(s).

### 4.3.6. Reference Phantoms

(129) The equivalent dose and effective dose are not measurable quantities. Their values are generally determined using coefficients relating them to measurable quantities. For the calculation of conversion coefficients for external exposure, computational phantoms are used for dose assessment in various radiation fields. For the calculation of dose coefficients from intakes of radionuclides, biokinetic models for radionuclides, reference physiological data, and computational phantoms are used (see Annex B).

(130) Previous calculations of dose coefficients have used various sex-invariant mathematical models such as the MIRD phantom (Snyder et al., 1969) or the Cristy age-specific phantoms (Cristy, 1980; ICRP, 1994b; 1996). The Commission has now defined the anatomical and physiological characteristics of reference persons reported in *Publication 89* (ICRP, 2002), which supplements and supersedes those given in *Publication 23* (ICRP, 1975). The Commission now uses reference computational phantoms of the adult male and female body that are based on medical tomographic images. The phantoms are made up of 3-dimensional **vo**lume pixels (voxels). The voxels that make up defined organs have been adjusted to approximate the organ masses assigned to the reference adult male and female in *Publication 89* (ICRP, 2002). These models are used, for example, to compute the mean absorbed dose, $D_T$, in an organ or tissue T, from reference radiation fields external to the body and the relationship of the effective dose to the operational quantities specific to the radiation field. They will be used in future calculations of dose coefficients for external radiation fields and for the intake of radionuclides (see Annex B).

### 4.3.7. Operational quantities

(131) As the body-related protection quantities, equivalent dose and effective dose, are not measurable in practice, operational quantities are used for the assessment of effective dose or mean equivalent doses in tissues or organs. These quantities aim to provide a conservative estimate for the value of the protection

quantities related to an exposure, or potential exposure of persons under most irradiation conditions. They are often used in practical regulations or guidance. Different types of operational quantities are used for internal and external exposures as summarised below. More details are given in Annex B.

(132) For the monitoring of external exposures, operational quantities for area and individual monitoring have been defined by ICRU (see Annex B). For area monitoring, the operational quantities are the ambient dose equivalent, $H^*(10)$ and the directional dose equivalent, $H'(0.07,\Omega)$, For individual monitoring, 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 dosemeter is worn. For the assessment of effective dose, $H_p(10)$ with a depth $d = 10$ mm is chosen and 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. For the rare case of monitoring the dose to the lens of the eye, a depth $d = 3$ mm has been proposed. In practice, however, $H_p(3)$ has rarely been used and personal dosemeters are usually not available that allow this to be measured (see Annex B). Operational dose equivalent quantities are measurable quantities 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 skin dose, respectively, in particular, if their values are below the protection limits.

(133) The system of dose assessment for intakes of radionuclides relies on the calculation of the intake of a radionuclide which can be considered as an operational quantity for the dose assessment from internal exposure. The intake can be estimated 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 the application of biokinetic models. The effective dose is then calculated from the intake using dose coefficients recommended by the Commission for a large number of radionuclides. Dose coefficients are given for members of the public of various ages and for adults who are occupationally exposed.

## 4.4. Assessment of radiation exposure

### 4.4.1. External radiation exposure

(134) The assessment of doses from exposure to radiation from external sources is usually performed either by individual monitoring using personal dosemeters worn on the body or e. g. in cases of prospective assessments, by measuring or estimating $H^*(10)$ and applying appropriate conversion coefficients. The operational quantities for individual monitoring are $H_p(10)$ and $H_p(0.07)$. If the personal dosemeter is worn on a position of the body representative for its exposure, the value of $H_p(10)$ provides at low doses and under the assumption of a uniform whole body exposure an effective dose value sufficiently precise for radiological protection practices.

### 4.4.2. Internal radiation exposure

(135) Radionuclides incorporated in the human body irradiate the tissues over time periods determined by their physical half-life and their biological retention within the body. Thus they 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:

$$H_T(\tau) = \int_{t_0}^{t_0+\tau} \dot{H}_T(t)\,dt \qquad (4.7)$$

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

$$E(\tau) = \sum_T w_T H_T(\tau) \qquad (4.8)$$

(136) For compliance with dose limits, the Commission continues to recommend 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-y period following the intake. The commitment period of 50 y is a rounded value considered by the Commission to be the life expectancy of a young person entering the workforce. 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 the age of 70 years.

(137) For assessing doses from occupational intakes of radionuclides the effective dose is based on the worker's intake and the reference dose coefficient. The calculations of dose coefficients for specified radionuclides (Sv $Bq^{-1}$) use defined biokinetic and dosimetric models. Models are used to describe the entry of various chemical forms of radionuclides into the body and their distribution and retention after entering the blood. The computational male and female phantoms are also used to compute, for a series of sources, the fraction of the energy emitted from a source region S that is absorbed in target region T. These approximations are considered to be adequate for the main tasks in radiological protection.

(138) Sex-averaged committed effective dose coefficients $e(\tau)$[1] for the intake of specified radionuclides are calculated according to the equation:

$$e(\tau) = \sum_T w_T \left[ \frac{h_T^M(\tau) + h_T^F(\tau)}{2} \right] \qquad (4.9)$$

[1] The lower case symbols *e* and *h* are used by convention to denote coefficients of the effective dose *E* and the equivalent dose *H*

where $w_T$ is the tissue weighting factor for tissue T, and $h_T^M(\tau)$ and $h_T^F(\tau)$ are the committed equivalent dose coefficients for tissue T of the male and female, respectively for the commitment period $\tau$. The summation in Equation (4.9) also extends over the committed equivalent dose coefficients for the remainder tissues in both the male and female.

### 4.4.3. Occupational exposure

(139) In monitoring occupational exposures to external radiation, individual dosemeters measure the personal dose equivalent $H_P(10)$. This measured value is taken as an assessment of the effective dose under the assumption of a uniform whole body exposure. For internal exposure, committed effective doses are generally determined from an assessment of the intakes of radionuclides from bioassay measurements or other quantities (e.g. activity retained in the body or in daily excreta). The radiation dose is determined from the intake using recommended dose coefficients (see Annex B).

(140) The doses obtained from the assessment of occupational exposures from external radiation and from intakes of radionuclides are combined for the assignment of the value of total effective dose, $E$ for demonstrating compliance with dose limits and constraints using the following formula:

$$E \cong H_p(10) + E(50) \qquad (4.10)$$

where $H_p(10)$ is the personal dose equivalent from external exposure and $E(50)$, the committed effective dose from internal exposure, which is assessed by:

$$E(50) = \sum_j e_{j,inh}(50) \cdot I_{j,inh} + \sum_j e_{j,ing}(50) \cdot I_{j,ing} \qquad (4.11)$$

where $e_{j,inh}(50)$ is the committed effective dose coefficient for activity intakes by inhalation of a radionuclide $j$, $I_{j,inh}$ is the activity intake of a radionuclide $j$ by inhalation, $e_{j,ing}(50)$ is the committed effective dose coefficient for activity intakes of a radionuclide $j$ by ingestion, and $I_{j,ing}$ is the activity intake of a radionuclide $j$ by ingestion. In the calculation of the effective dose from specific radionuclides, allowance may need to be made for the characteristics of the material taken into the body. The dose coefficients used in eqn. (4.11) are those specified by ICRP with no departure from the anatomical, physiological, and biokinetic characteristics of the reference person. Account may be taken of the physical and chemical characteristics of the intake, including the activity medium aerodynamic diameter (*AMAD)* of the inhaled aerosol and the chemical form of the particulate matter to which the specified radionuclide is attached. The effective dose assigned in the worker's dose record, is that value the reference person would experience due to the radiation fields and activity intakes encountered by the worker. The commitment period of 50 years represents the period of possible dose accumulation over a life-time (this is only relevant for radionuclides with long physical half-lives and long retention in body tissues).

(141) The radiation dose from radon isotopes and their decay products may also need to be taken into account in the overall dose assessment (ICRP 1994c).

(142) The incorporation of radionuclides through uncontrolled events involving wounds has implications beyond compliance with work practices and thus these events are not included in eqn. (4.11). The significance of these events must be evaluated and recorded, appropriate medical treatment provided, and further restriction of the worker's exposure considered if warranted.

(143) External exposures to airborne noble gas radionuclides in the workplace may need to be assessed beyond that indicated by $H_p(10)$. In such cases it is necessary to include in eqn. (4.11) a term representing the product of the time-integrated airborne concentration of the noble gas and an effective dose coefficient for so-called submersion exposure. Such dose coefficients are specified by ICRP for both prospective and retrospective applications. In the rare case of a significant contribution to external exposure of weakly-penetrating radiation, the term $0.01H_p(0.07)$ should be added in eqn. (4.10) for the assessment of effective dose.

(144) In certain situations, such as exposure of aircrew or where individual monitoring is not performed, an assessment of effective dose may be performed by area monitoring applying the quantity ambient dose equivalent, $H^*(10)$, and calculating effective dose using appropriate conversion coefficients. The Commission reaffirms its recommendation in *Publication 60* (ICRP, 1991b) that exposures to aircrew by cosmic radiation during aviation should be regarded as occupational exposure.

### 4.4.4. Public exposure

(145) The annual effective dose to members of the public is the sum of the effective dose obtained within one year from external exposure and the committed effective dose from radionuclides incorporated within this year. The dose is not obtained by direct measurement of individual exposures as for occupational exposure but is mainly determined by environmental measurements, habit data and modelling. It can be estimated by effluent monitoring for existing facilities or simulation and prediction of effluents from the technical installation or source during the design period. Information on concentrations of radionuclides in the environment are used in conjunction with radioecological modelling (pathway analysis of environmental transport, e.g. from the release of radionuclides and transport through soil – plants – animals to humans) to assess doses from external radiation exposure and intakes of radionuclides (see Annex B).

### 4.4.5. Medical exposure of patients

(146) The use of effective dose for assessing the exposure of patients has severe limitations that must be considered when quantifying medical exposure. Effective dose can be of value for comparing doses from different diagnostic procedures and for comparing the use of similar technologies and procedures in different hospitals and countries as well as the use of different technologies for the same medical examination. For planning the exposure of patients and risk-benefit assessments, however, the equivalent dose or the absorbed dose to irradiated tissues is the more relevant quantity.

(147) Medical exposures of patients to external radiation are commonly concerned with limited parts of the body only, and it is important that medical staff are fully aware of the doses to normal tissue in the irradiated fields. Considering the low tissue weighting factors for skin and relatively low values for a number of other

tissues, very localised partial body exposures can result in appreciable equivalent doses to local tissues. Similar considerations apply to doses from intakes of radionuclides. Care has to be taken in such situations so that undesirable tissue reactions occur are avoided as best possible under the circumstances.

(148) The assessment and interpretation of effective dose from medical exposure of patients is very problematic when organs and tissues receive only partial exposure or a very heterogeneous exposure which is the case especially with x-ray diagnostics.

### 4.4.6. Application of the effective dose

(149) The main and primary uses of effective dose in radiological protection for both occupational workers and the general public to exposures from controlled sources are as follows:

- prospective dose assessment for planning and optimisation of protection;
- retrospective dose assessment for demonstrating compliance with dose limits. Effective dose provides an instrument for demonstrating compliance with dose limits or dose constraints in radiological protection.

(150) In this sense effective dose is used for regulatory purposes worldwide. In practical radiological protection applications, effective dose is used for controlling possible stochastic effects in workers and the public. The calculation of effective dose or corresponding conversion coefficients for external exposure, as well as dose coefficients for internal exposure, are based on absorbed dose, weighting factors ($w_R$ and $w_T$) and reference values for the human body and its organs and tissues. Effective dose is not based on data from individual persons (see Annex B). In its general application, effective dose does not provide an individual-specific dose but rather that for a reference person under a given exposure situation.

(151) There may be some circumstances in which parameter values may be changed from the reference values in the calculation of effective dose. It is, therefore, important to distinguish between those reference parameter values that might be changed in the calculation of effective dose under particular circumstances of exposure and those values that cannot be changed under the definition of effective dose (e.g. the weighting factors). Thus, in the assessment of effective dose in occupational situations of exposure, changes may be made that, for example, relate to the characteristics of an external radiation field (e.g., direction of exposure) or to the physical and chemical characteristics of inhaled or ingested radionuclides. In such cases it is necessary to clearly state the deviation from the reference parameters.

(152) For retrospective assessments of doses in specified individuals that may substantially exceed dose limits, effective dose can provide an approximate first measure of the overall detriment. If radiation dose and risk need to be assessed in a more accurate way, further specific estimates of organ or tissue doses are necessary, especially if organ-specific risks for the specified individuals are needed.

(153) Effective dose is a quantity developed for radiological protection that is not suitable for use in epidemiological studies of radiation risks. Epidemiological