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Editor: H. SMITH, ICRP, Didcot, Oxfordshire

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Chairman: Dr. D. Beckross, *Comisión Nacional de Energía Atómica, Avenida Libertador 8250,  
1429 Buenos Aires, Argentina*

Vice-Chairman: Dr. H. Jammot, *Centre d'Etudes Nucléaires, B.P. No. 34, Bâtiment No. 38, 92265 Fontenay-aux-Roses  
Cedex, France*

Scientific Secretary: Dr. H. Smith, ICRP, P.O. Box No. 35, Didcot, Oxfordshire, OX11 0R3, England

## Members of the Main Commission of the ICRP

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H. J. Dunster, *London*

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## RADIATION PROTECTION

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## PREFACE

(1) Since 1977, when the Commission issued its basic recommendations as *ICRP Publication 26*, it has reviewed these recommendations annually and, from time to time, has issued supplementary Statements in the *Annals of the ICRP*. A complete list of the Commission's publications is given in Annex D. Developments in the last few years have now made it necessary to issue a completely new set of recommendations. In doing so, the Commission has had three aims in mind:

- (a) to take account of new biological information and of trends in the setting of safety standards,
- (b) to improve the presentation of the recommendations,
- (c) to maintain as much stability in the recommendations as is consistent with the new information.

(2) The draft of these recommendations was prepared by a Task Group set up by the 1985-89 Commission and comprising:

D. Beninson (Chairman)	Chairman of the Commission
H. Jammel	Vice-Chairman of the Commission
W. K. Sinclair	Chairman of Committee 1
C. B. Meinhold	Chairman of Committee 2
J. Liniecki	Chairman of Committee 3
H. J. Dunster	Chairman of Committee 4 to 1989
R. H. Clarke	Chairman of Committee 4 from 1989
B. Lindell	Emeritus Member of the Commission
H. Smith (Secretary)	Scientific Secretary of the Commission

The draft was discussed and adopted by the 1989-93 Commission in November 1990.

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E. Tajima		W. K. Sinclair	Chairman, Committee 1
H. Smith	Scientific Secretary	H. Smith	Scientific Secretary

## 1. INTRODUCTION

Chapter 1 deals with the history of the Commission and its recommendations. It sets out the aims and form of this report. It indicates why the Commission concerns itself only with the protection of man and only with ionising radiation. A list of the Publications of the Commission is given in Annex D.

### 1.1. The History of the Commission

(3) The International Commission on Radiological Protection, hereafter called the Commission, was established in 1928, with the name of the International X ray and Radium Protection Committee, following a decision by the Second International Congress of Radiology. In 1950 it was restructured and renamed. The Commission still retains a special relationship with the four-yearly Congress meetings and with the International Society of Radiology but, over the years, has greatly broadened its interests to take account of the increasing uses of ionising radiation and of practices that involve the generation of radiation and radioactive materials.

(4) The Commission works closely with its sister body, the International Commission on Radiation Units and Measurements, and has official relationships with the World Health Organisation and the International Atomic Energy Agency. It also has important relationships with the International Labour Organisation and other United Nations bodies, including the United Nations Scientific Committee on the Effects of Atomic Radiation and the United Nations Environment Programme, and with the Commission of the European Communities, the Nuclear Energy Agency of the Organisation for Economic Co-operation and Development, the International Standards Organisation, the International Electrotechnical Commission, and the International Radiation Protection Association. It takes account of progress reported by major national organisations.

(5) The Commission issued its first report in 1928. The first report in the current series, subsequently numbered *Publication 1* (1959), contained the recommendations approved in September 1958. Subsequent general recommendations have appeared as *Publication 6* (1964), *Publication 9* (1966), and *Publication 26* (1977). *Publication 26* was amended and extended by a Statement in 1978 and further clarified and extended by Statements in later years (1980, 1983, 1984, 1985, and 1987). Reports on more specialised topics have appeared as intermediate and subsequent publication numbers (Annex D).

### 1.2. The Development of the Commission's Recommendations

(6) The method of working of the Commission has not changed greatly over the last few decades. Since there is little direct evidence of harm at levels of annual dose at or below the limits recommended by the Commission, a good deal of scientific judgement is required in predicting the probability of harm resulting from low doses. Most of the observed data have been obtained at higher doses and usually at high dose rates. The Commission's aim is to draw on a broad spectrum of expertise from outside sources as well as from its own Committees and Task Groups and thus to reach a reasonable consensus about the outcome of exposures to radiation. It has not thought it appropriate

to use either the most pessimistic or the most optimistic interpretation of the available data, but has aimed at using estimates that are not likely to underestimate the consequences of exposures. The estimation of these consequences and their implications necessarily involves social and economic judgements as well as scientific judgements in a wide range of disciplines. The Commission has aimed to make the basis of such judgements as clear as possible, and recognises that others may wish to reach their own conclusions on many of the issues.

(7) The Commission has found that its recommendations have been used both by regulatory authorities and by management bodies and their specialist advisers. Because of the wide range of situations to which the Commission's recommendations might be applied, the degree of detail has deliberately been restricted. However, the Commission has had historical links with medical radiology and its advice in this area has often been more detailed.

(8) The Commission's recommendations have helped to provide a consistent basis for national and regional regulatory standards. For its part, the Commission has been concerned to maintain stability in its recommendations. It believes that frequent changes would only cause confusion. The Commission reviews the newly published data annually against the background of the much larger accumulation of existing data. It is not likely that dramatic changes would be called for by these reviews, but if new data should show the existing recommendations to be in need of urgent change, the Commission would respond rapidly.

(9) Over the last few decades, there has been a significant change in emphasis in the presentation and application of the system of protection recommended by the Commission. Initially, and into the 1950s, there was a tendency to regard compliance with the limits on individual doses as being a measure of satisfactory achievement. The advice that all exposures should be kept as low as possible was noted, but not often applied conscientiously. Since then, much more emphasis has been put on the requirement to keep all exposures "as low as reasonably achievable, economic and social factors being taken into account". This emphasis has resulted in substantial decreases in individual doses and has greatly reduced the number of situations in which the dose limits play a major role in the overall system of protection. It has also changed the purpose of the dose limits recommended by the Commission. Initially, their main function was the avoidance of directly observable, non-malignant effects. Subsequently, they were also intended to limit the incidence of cancer and hereditary effects caused by radiation. Over the years, the limits have been expressed in a variety of ways, so that comparisons are not easy. In broad terms, however, the annual limit for occupational exposure of the whole body was reduced by a factor of about 3 between 1934 and 1950, and by a further factor of 3, to the equivalent of 50 mSv, by 1958.

### 1.3. The Aims of this Report

(10) The Commission intends this report to be of help to regulatory and advisory agencies at national, regional, and international levels, mainly by providing guidance on the fundamental principles on which appropriate radiological protection can be based. Because of the differing conditions that apply in various countries, the Commission does not intend to provide a regulatory text. Authorities will need to develop their own structures of legislation, regulation, authorisations, licences, codes of practice, and guidance material in line with their usual practices and policies. The Commission

believes that these regulatory structures should be designed to be broadly consistent with the guidance in this report. In addition, the Commission hopes that the report will be of help to management bodies with responsibilities for radiological protection in their own operations, to the professional staff whom they use as their advisers, and to individuals, such as radiologists, who have to make decisions about the use of ionising radiation.

(11) The Commission has therefore set out these recommendations in the form of a main text supported by more detailed annexes. The main text contains all the recommendations, together with sufficient explanatory material to make clear the underlying reasoning. It is intended to be used by those concerned with policy, who can turn to the supporting annexes if they need more detailed information on specific points. Specialists will need to study both the main text and the annexes.

(12) Chapters 2 and 3 deal with the quantities and units used in radiological protection and with the biological effects of radiation. Chapter 4 describes the conceptual framework of radiological protection and leads into Chapters 5 and 6 which deal with the Commission's main recommendations. Chapter 7 discusses the practical implementation of the recommendations. Finally, there is a summary of the recommendations.

### 1.4. The Scope of the Commission's Recommendations

(13) Ionisation is the process by which atoms lose, or sometimes gain electrons and thus become electrically charged, being then known as ions. Ionising radiation is the term used to describe the transfer of energy through space in the form of either electromagnetic waves or subatomic particles that are capable of causing ionisation in matter. When ionising radiation passes through matter, energy is imparted to the matter as ions are formed.

(14) The recommendations of the Commission, as in previous reports, are confined to protection against ionising radiation. The Commission recognises the importance of adequate control over sources of non-ionising radiation, but continues to consider that this is a subject outside its own field of competence. It also recognises that this concentration on a single one of the many dangers facing mankind may cause an unwanted element of anxiety. The Commission therefore wishes to emphasise its view that ionising radiation needs to be treated with care rather than fear and that its risks should be kept in perspective with other risks. The procedures available to control exposures to ionising radiation are sufficient, if used properly, to ensure that it remains a minor component of the spectrum of risks to which we are all exposed.

(15) Ionising radiation and radioactive materials have always been features of our environment, but, owing to their lack of impact on our senses, we became aware of them only at the end of the 19th century. Since that time, we have found many important uses for them and have developed new technological processes which create them, either deliberately or as unwanted by-products. The primary aim of radiological protection is to provide an appropriate standard of protection for man without unduly limiting the beneficial practices giving rise to radiation exposure. This aim cannot be achieved on the basis of scientific concepts alone. All those concerned with radiological protection have to make value judgements about the relative importance of different kinds of risk and about the balancing of risks and benefits. In this, they are no different from those working in other fields concerned with the control of hazards.

(16) The Commission believes that the standard of environmental control needed to protect man to the degree currently thought desirable will ensure that other species are

not put at risk. Occasionally, individual members of non-human species might be harmed, but not to the extent of endangering whole species or creating imbalance between species. At the present time, the Commission concerns itself with mankind's environment only with regard to the transfer of radionuclides through the environment, since this directly affects the radiological protection of man.

## 2. QUANTITIES USED IN RADIOLOGICAL PROTECTION

Chapter 2 explains in simple terms the principal quantities used in radiological protection. The formal definitions and more detailed information are given in Annex A.

### 2.1. Introduction

(17) Historically, the quantities used to measure the "amount" of ionising radiation (subsequently called "radiation" in this report) have been based on the gross number of ionising events in a defined situation or on the gross amount of energy deposited, usually in a defined mass of material. These approaches omit consideration of the discontinuous nature of the process of ionisation, but are justified empirically by the observation that the gross quantities (with adjustments for different types of radiation) correlate fairly well with the resulting biological effects.

(18) Future developments may well show that it would be better to use other quantities based on the statistical distribution of events in a small volume of material corresponding to the dimensions of biological entities such as the nucleus of the cell or its molecular DNA. Meanwhile, however, the Commission continues to recommend the use of macroscopic quantities. These, among others, are described in Annex A and are known as dosimetric quantities. They have been defined in formal terms by the International Commission on Radiation Units and Measurements (ICRU).

(19) Before discussing dosimetric quantities, it is necessary to anticipate some of the information on the biological effects of radiation described in Chapter 3. The process of ionisation necessarily changes atoms and molecules, at least transiently, and may thus sometimes damage cells. If cellular damage does occur, and is not adequately repaired, it may prevent the cell from surviving or reproducing, or it may result in a viable but modified cell. The two outcomes have profoundly different implications for the organism as a whole.

(20) Most organs and tissues of the body are unaffected by the loss of even substantial numbers of cells, but if the number lost is large enough, there will be observable harm reflecting a loss of tissue function. The probability of causing such harm will be zero at small doses, but above some level of dose (the threshold) will increase steeply to unity (100%). Above the threshold, the severity of the harm will also increase with dose. For reasons explained in Section 3.4.1, this type of effect, previously called "non-stochastic", is now called "deterministic" by the Commission.

(21) The outcome is very different if the irradiated cell is modified rather than killed. Despite the existence of highly effective defence mechanisms, the clone of cells resulting from the reproduction of a modified but viable somatic cell may result, after a prolonged and variable delay called the latency period, in the manifestation of a malignant condition, a cancer. The probability of a cancer resulting from radiation usually increases with increments of dose, probably with no threshold, and in a way that is roughly proportional to dose, at least for doses well below the thresholds for deterministic effects. The

severity of the cancer is not affected by the dose. This kind of effect is called "stochastic", meaning "of a random or statistical nature". If the damage occurs in a cell whose function is to transmit genetic information to later generations, any resulting effects, which may be of many different kinds and severity, are expressed in the progeny of the exposed person. This type of stochastic effect is called "hereditary".

### 2.2. Basic Dosimetric Quantities

(22) The fundamental dosimetric quantity in radiological protection is the *absorbed dose*,  $D$ . This is the energy absorbed per unit mass and its unit is the joule per kilogram, which is given the special name gray (Gy). Absorbed dose is defined in terms that allow it to be specified at a point, but it is used in this report, except where otherwise stated, to mean the average dose over a tissue or organ. The use of the average dose as an indicator of the probability of subsequent stochastic effects depends on the linearity of the relationship between the probability of inducing an effect and the dose (the dose-response relationship)—a reasonable approximation over a limited range of dose. The dose-response relationship is not linear for deterministic effects so the average absorbed dose is not directly relevant to deterministic effects unless the dose is fairly uniformly distributed over the tissue or organ.

#### 2.2.1. Radiation weighting factors

(23) The probability of stochastic effects is found to depend, not only on the absorbed dose, but also on the type and energy of the radiation causing the dose. This is taken into account by weighting the absorbed dose by a factor related to the quality of the radiation. In the past, this weighting factor has been applied to the absorbed dose at a point and called the quality factor,  $Q$ . The weighted absorbed dose was called the dose equivalent,  $H$ .

#### 2.2.2. Equivalent dose

(24) In radiological protection, it is the absorbed dose averaged over a tissue or organ (rather than at a point) and weighted for the radiation quality that is of interest. The weighting factor for this purpose is now called the radiation weighting factor,  $w_R$ , and is selected for the type and energy of the radiation incident on the body or, in the case of sources within the body, emitted by the source. This weighted absorbed dose is strictly a dose, and the Commission has decided to revert to the earlier name of equivalent dose in a tissue or organ, using the symbol  $H_T$ . The change of name also serves to indicate the change from quality factor to radiation weighting factor. The equivalent dose in tissue  $T$  is given by the expression

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

where  $D_{T,R}$  is the absorbed dose averaged over the tissue or organ  $T$ , due to radiation  $R$ . The unit of equivalent dose is the joule per kilogram with the special name sievert (Sv).

(25) The value of the radiation weighting factor for a specified type and energy of radiation has been selected by the Commission to be representative of values of the relative biological effectiveness of that radiation in inducing stochastic effects at low doses. The relative biological effectiveness (RBE) of one radiation compared with another is the inverse ratio of the absorbed doses producing the same degree of a defined

biological end-point. The values of  $w_R$  are broadly compatible with the values of  $Q$ , which are related to the quantity linear energy transfer (LET), a measure of the density of ionisation along the track of an ionising particle. This relationship was originally intended to do no more than provide a rough indication of the variation of the values of  $Q$  with changes of radiation, but it was often interpreted to imply a spurious precision which the Commission hopes will not be inferred from the new radiation weighting factors. The Commission has chosen a value of radiation weighting factor of unity for all radiations of low LET, including  $\alpha$  and gamma radiations of all energies. The choice for other radiations is based on observed values of the relative biological effectiveness (RBE), regardless of whether the reference radiation is  $\alpha$  or gamma radiation.

(26) When the radiation field is composed of types and energies with different values of  $w_R$ , the absorbed dose must be subdivided in blocks, each with its own value of  $w_R$  and summed to give the total equivalent dose. Alternatively it may be expressed as a continuous distribution in energy where each element of absorbed dose from the energy element between  $E$  and  $E + dE$  is multiplied by the value of  $w_R$  from the relevant block in Table 1 or, as an approximation, by the value of  $w_R$  from the continuous function given in paragraph A12 of Annex A and illustrated by the continuous curve in Figure 1. The basis for selecting values for other radiations is given in Annex A (paragraph A13). Auger electrons emitted from nuclei bound to DNA present a special problem because it is not realistic to average the absorbed dose over the whole mass of DNA as would be required by the present definition of equivalent dose. The effects of Auger electrons have to be assessed by the techniques of microdosimetry (see Annex B, paragraph B67).

Table 1. Radiation weighting factors<sup>1</sup>

Type and energy range <sup>2</sup>	Radiation weighting factor, $w_R$
Photons, all energies	1
Electrons and muons, all energies <sup>3</sup>	1
Neutrons, energy < 10 keV	5
10 keV to 100 keV	10
> 100 keV to 2 MeV	20
> 2 MeV to 20 MeV	10
> 20 MeV	5
(See also Figure 1)	
Protons, other than recoil protons, energy > 2 MeV	5
Alpha particles, fission fragments, heavy nuclei	20

<sup>1</sup> All values relate to the radiation incident on the body or, for internal sources, emitted from the source.

<sup>2</sup> The choice of values for other radiations is discussed in Annex A.

<sup>3</sup> Excluding Auger electrons emitted from nuclei bound to DNA (see paragraph 26).

### 2.2.3. Tissue weighting factors and effective dose

(27) The relationship between the probability of stochastic effects and equivalent dose is found also to depend on the organ or tissue irradiated. It is therefore appropriate to define a further quantity, derived from equivalent dose, to indicate the combination of different doses to several different tissues in a way which is likely to correlate well with the total of the stochastic effects. The factor by which the equivalent dose in tissue or organ T is weighted is called the tissue weighting factor,  $w_T$ , which represents the relative

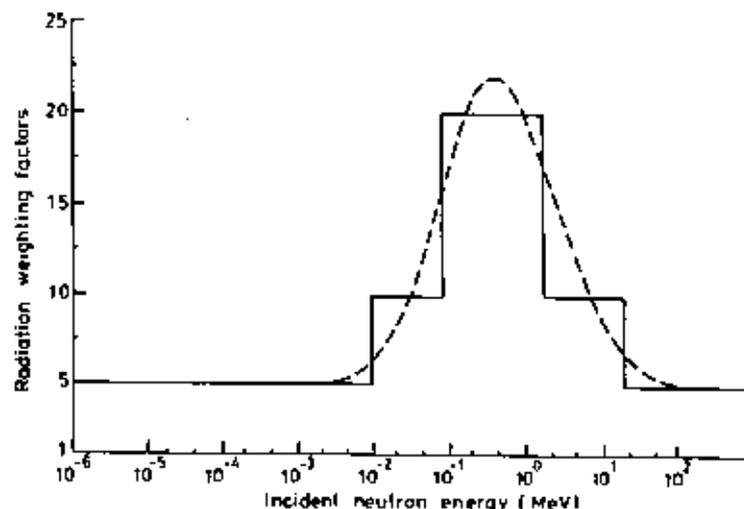


Fig. 1. Radiation weighting factors for neutrons. The smooth curve is to be treated as an approximation.

contribution of that organ or tissue to the total detriment due to these effects resulting from uniform irradiation of the whole body. (See Section 3.5.) The weighted equivalent dose (a doubly weighted absorbed dose) has previously been called the effective dose equivalent but this name is unnecessarily cumbersome, especially in more complex combinations such as collective committed effective dose equivalent. The Commission has now decided to use the simpler name effective dose,  $E$ . The introduction of the name effective dose is associated with the change to equivalent dose, but has no connection with changes in the number or magnitude of the tissue weighting factors. The unit is the joule per kilogram with the special name sievert. The choice of values of the tissue weighting factor is discussed in Section 3.5 and the recommended values are given in Table 2.

(28) The effective dose is the sum of the weighted equivalent doses in all the tissues and organs of the body. It is given by the expression

$$E = \sum_T w_T \cdot H_T$$

where  $H_T$  is the equivalent dose in tissue or organ T and  $w_T$  is the weighting factor for tissue T. The effective dose can also be expressed as the sum of the doubly weighted absorbed dose in all the tissues and organs of the body.

(29) It is desirable that a uniform equivalent dose over the whole body should give an effective dose numerically equal to that uniform equivalent dose. This is achieved by normalising the sum of the tissue weighting factors to unity. The values of the radiation weighting factor depend on the type and energy of the radiation and are independent of the tissue or organ. Similarly, the values of the tissue weighting factor are chosen to be independent of the type and energy of the radiation incident on the body. These simpli-

Table 2. Tissue weighting factors<sup>1</sup>

Tissue or organ	Tissue weighting factor, $w_T$
gonads	0.20
Bone marrow (red)	0.12
Colon	0.12
Lung	0.12
Stomach	0.12
Bladder	0.05
Breast	0.05
Liver	0.05
Oesophagus	0.05
Thyroid	0.05
Skin	0.01
Bone surface	0.01
Remainder	0.05 <sup>2,3</sup>

<sup>1</sup> The values have been developed from a reference population of equal numbers of both sexes and a wide range of ages. In the definition of effective dose they apply to workers, to the whole population, and to either sex.

<sup>2</sup> For purposes of calculation, the remainder is composed of the following additional tissues and organs: adrenals, brain, upper large intestine, small intestine, kidney, muscle, pancreas, spleen, thymus and uterus. The list includes organs which are likely to be selectively irradiated. Some organs in the list are known to be susceptible to cancer induction. If other tissues and organs subsequently become identified as having a significant risk of induced cancer they will then be included either with a specific  $w_T$  or in this additional list constituting the remainder. The latter may also include other tissues or organs selectively irradiated.

<sup>3</sup> In those exceptional cases in which a single one of the remainder tissues or organs receives an equivalent dose in excess of the highest dose in any of the twelve organs for which a weighting factor is specified, a weighting factor of 0.025 should be applied to that tissue or organ and a weighting factor of 0.025 to the average dose in the rest of the remainder as defined above.

cations may be no more than approximations to the true biological situation, but they make it possible to define a radiation field outside the body in dosimetric terms (see Section 2.4) without the need to specify the target organ.

(30) The consequences following an absorbed dose depend not only on the magnitude of the dose, the type and energy of the radiation (dealt with by the radiation weighting factor), and the distribution of the dose within the body (dealt with by the tissue weighting factor), but also on the distribution of the dose in time (dose rate and protraction of exposure). In previous formulations, provision was made for possible weighting factors other than the radiation and tissue weighting factors. The product of these other, unspecified, weighting factors was called  $N$ . Any effect of the time distribution of dose could have been accommodated by assigning a set of values to  $N$ . In practice this has not been attempted and the Commission has decided to drop the use of  $N$ . The effect of all exposure conditions other than those dealt with by the radiation and tissue weighting factors will be covered by using different values of the coefficients relating equivalent dose and effective dose to the probability of stochastic effects, rather than by using additional weighting factors in the definitions of the quantities.

(31) The values of both the radiation and the tissue weighting factors depend on our current knowledge of radiobiology and may change from time to time. Indeed, new values are adopted in these recommendations. Although such changes are infrequent, they can cause confusion. The definitions of equivalent dose (in a single tissue or organ) and effective dose (in the whole body) are not confined to any particular set of values of these weighting factors, so care is needed to avoid ambiguity. When the Commission uses equivalent dose and effective dose, it will be implicit that these contain the values of radiation and tissue weighting factors recommended at the relevant time by the Commission. It is appropriate to treat as additive the weighted quantities used by the Commission but assessed at different times, despite the use of different values of weighting factors. The Commission does not recommend that any attempt be made to correct earlier values. It is also appropriate to add values of dose equivalent to equivalent dose and values of effective dose equivalent to effective dose without any adjustments. If values of weighting factors other than those recommended by the Commission are used, this fact should be clearly stated and the values should be explicitly given when the quantities are introduced. These weighted quantities should not be added to the Commission's quantities.

(32) Both equivalent dose and effective dose are quantities intended for use in radiological protection, including the assessment of risks in general terms. They provide a basis for estimating the probability of stochastic effects only for absorbed doses well below the thresholds for deterministic effects. For the estimation of the likely consequences of an exposure of a known population, it will sometimes be better to use absorbed dose and specific data relating to the relative biological effectiveness of the radiations concerned and the probability coefficients relating to the exposed population.

### 2.3. Subsidiary Dosimetric Quantities

(33) Several subsidiary dosimetric quantities have proved useful. Following an intake to the body of a radioactive material, there is a period during which the material gives rise to equivalent doses in the tissues of the body at varying rates. The time integral of the equivalent-dose rate is called the committed equivalent dose,  $H_T(\tau)$  where  $\tau$  is the integration time (in years) following the intake. If  $\tau$  is not specified, it is implied that the value is 50 years for adults and from intake to age 70 years for children. By extension, the committed effective dose,  $E(\tau)$ , is similarly defined. When the Commission refers to an equivalent or effective dose accumulated in a given period of time, it is implicit that any committed doses from intakes occurring in that same period are included.

(34) The dosimetric quantities referred to above all relate to the exposure of an individual. The Commission uses further quantities related to exposed groups or populations. These quantities take account of the number of people exposed to a source by multiplying the average dose to the exposed group from the source by the number of individuals in the group. The relevant quantities are the collective equivalent dose,  $S_T$ , which relates to a specified tissue or organ, and the collective effective dose,  $S$ . If several groups are involved, the total collective quantity is the sum of the collective quantities for each group. The unit of these collective quantities is the man sievert. The collective quantities can be thought of as representing the total consequences of the exposure of a population or group, but their use in this way should be limited to situations in which the consequences are truly proportional to both the dosimetric quantity and number of people exposed, and in which an appropriate probability coefficient is available (see

Section 2.4). When it is necessary to distinguish between a collective dose and the dose to an individual, the latter is called the individual dose.

(35) The collective effective dose resulting from the presence of radioactive materials in the environment may be accumulated over long periods of time, covering successive generations of individuals. The total collective effective dose to be expected from a given situation is the integral over all time of the collective effective dose rate resulting from, i.e. committed by, a single release (or a unit period of a practice in the case of a continuing operation). If the integration is not over infinite time, the quantity is described as being truncated at a defined time. If the ranges of individual dose or time are large, it may be useful to subdivide the collective quantities into blocks covering more limited ranges of dose and time. When considering the consequences of a unit period of practice, it is sometimes convenient to distinguish between the collective effective dose already delivered and the collective effective dose committed over all time.

(36) The dose commitment ( $H_{c,T}$  or  $E_c$ ) is a calculational tool. It can be assessed for a critical group as well as for the whole world population. It is defined as the infinite time integral of the per caput dose rate ( $\dot{H}_T$  or  $\dot{E}$ ) due to a specified event, such as a unit of practice (e.g. a year of practice):

$$H_{c,T} = \int_0^{\infty} \dot{H}_T(t) dt$$

or

$$E_c = \int_0^{\infty} \dot{E}(t) dt$$

In the case of an indefinite practice at a constant rate, the maximum annual per caput dose rate ( $\dot{H}_T$  or  $\dot{E}$ ) in the future for the specified population will be equal to the dose commitment of one year of practice, irrespective of changes in the population size. If the practice is continued only over a time period  $\tau$ , the maximum future annual per caput dose will be equal to the corresponding truncated dose commitment, defined as

$$H_{c,T}(\tau) = \int_0^{\tau} \dot{H}_T(t) dt$$

or

$$E_c(\tau) = \int_0^{\tau} \dot{E}(t) dt$$

## 2.4. Other Quantities

(37) Several other quantities are of special use in radiological protection. One of these is the activity,  $A$ , of a quantity of a radionuclide. Activity is the average number of spontaneous nuclear transformations taking place per unit time. Its unit is the reciprocal second,  $s^{-1}$ , given, for this purpose, the special name becquerel (Bq).

(38) There are also four operational quantities of particular interest in the measurement of radiation fields for protection purposes. These ICRU quantities, the ambient dose equivalent,  $H^*(d)$ , the directional dose equivalent,  $H'(d)$ , the individual dose equivalent, penetrating,  $H_p(d)$ , and the individual dose equivalent, superficial,  $H_s(d)$  are

defined in Annex A. All these quantities are based on the concept of the dose equivalent at a point and not on the concept of equivalent dose (see paragraph 24).

(39) In relating the probability of stochastic effects to dosimetric quantities, it is convenient to use a probability coefficient. For example, the fatality probability coefficient is the quotient of probability that an increment of dose will cause death and the magnitude of that increment of dose. The dose in question will usually be an equivalent dose or an effective dose. Such coefficients necessarily relate to a specified population.

(40) It is often useful in general statements to use generic terms that can apply to any of the relevant dosimetric quantities. The Commission uses "dose" as one such term in phrases such as "dose limit". This may be a limit applied to equivalent or effective dose. The choice is usually clear from the context. The Commission also uses the term "exposure" in a generic sense to mean the process of being exposed to radiation or radioactive material. The significance of an exposure in this sense is determined by the resulting doses. It seems unlikely that this causes any confusion with the highly specific use of exposure as a quantity defined by ICRU.

(41) The Commission uses the International System of units (SI) and the international convention that the names of units are written with a lower case initial letter. The abbreviations for units are written with a lower case letter, or initial letter, except when the name of the unit is derived from a person's name, e.g. m and mm for metre and millimetre, but Sv and mSv for sievert and millisievert.

## 3. BIOLOGICAL ASPECTS OF RADIOLOGICAL PROTECTION

Chapter 3 provides an introduction to the stochastic and deterministic biological effects of ionising radiation and leads on to a discussion of the problems of establishing a quantitative measure of the detriment associated with an exposure to radiation. More detailed biological information, including that on radiation risks, is provided in Annex B. The use of this information as a basis for radiological protection policy is discussed in Annex C.

### 3.1. Introduction

(42) As explained in Chapter 1, radiological protection is concerned with protecting man against the harmful effects of radiation. In all its work, the Commission has based its approach on the best available information on the biological effects of radiation and has used this to provide a simplified, but adequate, biological basis for radiological protection. This chapter and Annex B therefore deal with the deleterious effects only to the extent necessary to support that approach. To help in achieving clarity, distinction has been made between four terms: change, damage, harm and detriment. Changes may or may not be harmful. Damage represents some degree of deleterious change, for example to cells, but is not necessarily deleterious to the exposed individual. Harm is the term used to denote clinically observable deleterious effects that are expressed in individuals (somatic effects) or their descendants (hereditary effects). Detriment is a complex concept combining the probability, severity and time of expression of harm. It is not easily represented by a single variable and is discussed in Section 3.3.

(43) The term "risk" has previously been used by the Commission to mean the probability of a defined deleterious outcome, but it has also been widely used elsewhere

as the product of the probability and severity of an event and, more generally, in a purely descriptive manner. The Commission now uses risk only descriptively and in well-established expressions such as "risk estimate" and "excess relative risk". It now uses probability when that is what is meant. Aspects of probability and risk are discussed in detail in Annexes B and C.

### 3.2. The Biological Effects of Ionising Radiation

(44) Part of this material has been previewed in Section 2.1, and is discussed here in more detail. The process of ionisation necessarily changes atoms, at least transiently, and may thus alter the structure of the molecules containing them. Molecular changes may also be caused by the excitation of atoms and molecules if the excitation energy exceeds the binding energy between atoms. About half the energy deposited in tissue by ionising radiation is due to excitation, but this is of less consequence than ionisation and has not been considered separately in what follows. If the affected molecules are in a living cell, the cell itself may sometimes be damaged, either directly if the molecule is critical to the cell's function, or indirectly by causing chemical changes in adjacent molecules, e.g. the production of free radicals. Of the various forms of damage that radiation can cause in cells, the most important is that in the DNA. Damage in the DNA may prevent the survival or reproduction of the cell, but frequently the damage is repaired by the cell. If that repair is not perfect, it may result in a viable but modified cell. The occurrence and proliferation of a modified cell may well be influenced by other changes in the cell caused either before or after the exposure to radiation. Such influences are common and may include exposure to other carcinogens or mutagens.

(45) If enough cells in an organ or tissue are killed or prevented from reproducing and functioning normally, there will be a loss of organ function—an effect that the Commission now calls "deterministic". The loss of function will become more serious as the number of affected cells is increased. More details are given in Section 3.4.1. A modified somatic cell may still retain its reproductive capacity and may give rise to a clone of modified cells that may eventually result in a cancer. A modified germ cell in the gonads, with the function of transmitting genetic information to the descendants of an exposed individual, may transmit incorrect hereditary information and may cause severe harm to some of those descendants. These somatic and hereditary effects, which may start from a single modified cell, are called stochastic effects. They are discussed further in Sections 3.4.2 and 3.4.3. Because of the complex processes involved in the development of the conceptus to an embryo and a fetus, it is convenient to discuss both deterministic and stochastic effects of radiation on the unborn child in a separate section (Section 3.4.4).

(46) There is some experimental evidence that radiation can act to stimulate a variety of cellular functions, including proliferation and repair. Such stimulation is not necessarily beneficial. In some circumstances, radiation appears also to enhance immunological responses and to modify the balance of hormones in the body. In particular, radiation may be able to stimulate the repair of prior radiation damage, thus decreasing its consequences, or may be able to improve immunological surveillance, thus strengthening the body's natural defence mechanisms. Most of the experimental data on such effects, currently termed "hormesis", have been inconclusive, mainly because of statistical difficulties at low doses. Furthermore, many relate to biological endpoints other than cancer or hereditary effects. The available data on hormesis are not sufficient to take them into account in radiological protection.

### 3.3. The Concept of Detriment

(47) In *Publication 26* (1977), the Commission introduced the concept of detriment as a measure of the total harm that would eventually be experienced by an exposed group and its descendants as a result of the group's exposure to a radiation source. Health detriment was included as part of the total detriment. In practice, the Commission has used only the health detriment and recommends that a separate allowance should be made for other forms of detriment when decision-aiding techniques are used, for example in optimisation studies. In this report, the Commission uses the term detriment to mean only health detriment.

(48) The Commission's definition of detriment in *Publication 26* used the expected number of cases of a radiation-induced health effect weighted by a factor representing the severity of the effect. It was the expectation value (called more strictly the mathematical expectation) of the weighted number of health effects to be experienced by the group. The weighting factor was taken as 1 for the death of individuals and for severe hereditary effects in their descendants. Smaller weighting factors were implied for other, less severe effects, but were not specified. In relation to an individual, the detriment could also be expressed as the product of the probability of a deleterious effect and a measure of the severity of that effect. If the measure of the severity is normalised to 1 for the most severe effects, and if the values of all the products are small, the products for different outcomes in the same individual can be summed to give the total detriment to that individual. It is implicit in this concept of detriment that the relevant doses are small, well below the thresholds for deterministic effects.

(49) This approach to detriment has proved useful but is somewhat too limited. The Commission now finds it necessary to take a broader view. The general aim is still to find a quantitative way of expressing a combination of the probability of occurrence of a health effect and a judgement of the severity of that effect. Ideally, detriment should be represented as an extensive quantity, i.e. one that allows the detriment to a group to be added as additional exposures occur to individuals and as more individuals are added to the group. This requirement cannot be fully met, at least for the individual, because some of the outcomes of exposure are mutually exclusive and some are not. Death due to one exposure excludes death due to another, but non-fatal conditions may occur concurrently or consecutively. A second problem is posed by the multifarious nature of the possible outcomes, so that probability and severity can be combined in many different ways to represent detriment.

(50) The Commission needs to use detriment for several different purposes. One is to assess the consequences of continued or cumulative exposures in order to recommend dose limits. Another is to compare the consequences of different distributions of equivalent dose within the body and thence to select a set of tissue weighting factors. A third is to provide a basis for assessing the valuation of a unit of effective dose for use, for example, in the optimisation of protection within a practice. These purposes are discussed in Chapter 4.

(51) The Commission has concluded that the many aspects of detriment and its many purposes make the selection of a single approach undesirable. Therefore, the Commission has replaced its previous concept of detriment by a multi-dimensional concept. For recommending dose limits, the detriment from an exposure has been expressed in a variety of ways. This approach is dealt with in Chapter 5 and, in more detail, in Annexes B and C. For this purpose, only a limited attempt is made to aggregate these facets into a

single quantity, called in *Publication 45* (1985) a unified index of harm. However, an aggregative method was preferred in choosing tissue weighting factors because these are used only to make adjustments for the differential sensitivity of tissues and organs. Since it is rare for single tissues, except for the lung and perhaps the thyroid and skin, to be irradiated alone, the choice of tissue weighting factors is not very sensitive to the procedure for aggregating the different aspects of detriment. Details are given in Section 3.5 and in Annex B.

### 3.4. Quantitative Estimates of the Consequences of Radiation Exposures

(52) In order to develop a system of radiological protection, it is necessary to know quantitatively how the probability of stochastic effects and the severity of deterministic effects vary with dose. The most relevant sources of information are those obtained directly from studies of the effects of radiation on man. In addition, a great deal of information about the mechanisms of damage and the relationships between dose and the probability of deleterious effects in man can be inferred from studies on micro-organisms, on isolated cells grown *in vitro*, and on animals. Unfortunately, little, if any, of the available information can be applied directly in radiological protection—it all needs considerable interpretation. The Commission's conclusions on the biological information needed in radiological protection are drawn to the maximum extent possible from data on radiation effects in human beings, with other information used in support.

(53) Data on deterministic effects in man come from the side effects of radiotherapy, from effects on the early radiologists, from the effects of the atomic bombs at Hiroshima and Nagasaki in Japan, and from the consequences of severe accidents, some in the nuclear industry and some involving radiographic sources. At present, the three principal sources of information on stochastic effects are the epidemiological studies on the survivors of the nuclear weapon attacks on Hiroshima and Nagasaki, on patients exposed to radiation for medical treatment or diagnosis, and on some groups of workers exposed to radiation or radioactive substances at work. Studies of this kind are very complex and time-consuming and are not conducted by the Commission itself. The Commission, with the help of its Committees, examines the published accounts of the studies and any reviews carried out by national and international bodies and then draws conclusions relevant to the needs of radiological protection.

#### 3.4.1. Deterministic effects

(54) In many organs and tissues of the body there is a continuous process of loss and replacement of cells. An increase in the rate of loss, for example following exposure to radiation, may be compensated for by an increase in the replacement rate, but there will be a transient, and sometimes permanent, net reduction in the number of cells available to maintain the functions of the organ or tissue. Many organs and tissues are unaffected by small reductions in the number of available cells, but if the decrease is large enough, there will be clinically observable pathological conditions such as a loss of tissue function or a consequential reaction as the body attempts to repair the damage. If the tissue is vital and is damaged sufficiently, the end result will be death. If some individuals in the exposed group are already in a state of health approaching the pathological condition, they will reach that condition as a result of exposure to radiation after a smaller loss of cells than would usually be the case. For healthy individuals, the probability of causing harm will be zero at doses up to some hundreds, or sometimes thousands, of milli-

sieverts, depending on the tissue, and will increase steeply to unity (100%) above some level of dose called the threshold, more strictly, the threshold for clinical effect. The plot on linear axes of the probability of harm against dose is sigmoid. Above the appropriate threshold, the severity of the harm will increase with dose, reflecting the number of cells damaged, and usually with dose rate because a protracted dose will cause the damage to cells to be spread out in time, allowing for more effective repair or repopulation. This type of effect, characterised by a severity that increases with dose above some clinical threshold, was previously called "non-stochastic". Although the initial cellular changes are essentially random, the large number of cells involved in the initiation of a clinically observable, non-stochastic effect gives the effect a deterministic character. For this reason, the Commission now calls such effects "deterministic" effects.

(55) In addition to the loss of functional cells in a tissue or organ, damage to supporting blood vessels may also occur, leading to secondary tissue damage. There may also be some replacement of functional cells by fibrous tissue causing a reduction in organ function. The clinical findings depend on the specific function of the irradiated tissue. For example, opacities may occur in the lens of the eye, sometimes leading to visual impairment (cataract), and, if the gonads are irradiated, there may be a temporary or permanent loss of fertility.

(56) Some deterministic effects are of a functional nature and may be reversible, provided that the damage is not too severe. Some examples of functional effects are: decreasing of glandular secretions (e.g. from the salivary glands or thyroid); neurological effects (e.g. changes in electroencephalograms or retinograms); vascular reactions (e.g. early erythema or subcutaneous oedema).

(57) The equivalent dose is not always the appropriate quantity for use in relation to deterministic effects because the values of radiation weighting factors have been chosen to reflect the relative biological effectiveness (RBE) of the different types and energies of radiation in producing stochastic effects. For radiations with a radiation weighting factor larger than 1, the values of RBE for deterministic effects are smaller than those for stochastic effects. The use of the equivalent dose to predict deterministic effects for high LET radiations, e.g. neutrons, will thus lead to overestimates.

(58) The data for low LET radiation show a wide range of sensitivities for different tissues. However, it can be concluded that few tissues show clinically significant detrimental effects following single (i.e. acute) absorbed doses of less than a few gray. For doses spread out over a period of years, severe effects are not likely in most tissues at annual doses of less than about 0.5 Gy. However, the gonads, the lens of the eye, and the bone marrow show higher sensitivities.

(59) The threshold for temporary sterility in the male for a single absorbed dose in the testes is about 0.15 Gy. Under conditions of prolonged exposure the dose rate threshold is about 0.4 Gy  $y^{-1}$ . The corresponding values for permanent sterility are about 3.5 to 6 Gy and 2 Gy  $y^{-1}$ . The threshold for permanent sterility in women is an acute absorbed dose in the range from about 2.5 to 6 Gy, older women being more sensitive; or a protracted dose rate over many years of more than 0.2 Gy  $y^{-1}$  (see Annex B, Table B-1).

(60) The threshold for opacities sufficient to cause impairment of vision, which occur after some delay, seems to be in the range 2 to 10 Gy for an acute exposure to low LET radiation. For high LET radiation, the absorbed dose thresholds are 2 or 3 times less. The dose rate threshold is less well known for chronic exposure, but for exposure over many years is thought to be somewhat above 0.15 Gy  $y^{-1}$  (see Annex B, Table B-1).

(61) Clinically significant depression of the blood-forming process has a threshold for

acute absorbed doses in the whole bone marrow of about 0.5 Gy. The dose-rate threshold for protracted exposure over many years is more than  $0.4 \text{ Gy y}^{-1}$ . The  $\text{LD}_{50}$  in 60 days due to bone marrow syndrome in a heterogeneous population uniformly and acutely exposed is about 3 to 5 Gy in the absence of a high standard of medical care (see Annex B, Table B-2).

#### 3.4.2. Stochastic effects in exposed individuals

(62) The response of the body to the development of a clone of modified somatic cells is complex. The initial development of such a clone may be inhibited unless its development is promoted by some additional agent and any surviving clone is very likely to be eliminated or isolated by the body's defences. However, if it is not, it may result, after a prolonged and variable delay called the latency period, in the development of a malignant condition in which the proliferation of modified cells is uncontrolled. Such conditions are commonly grouped together and called cancer. The cancers induced by radiation, with or without a contribution from other agents, are not distinguishable from those occurring from other causes. The defence mechanisms are not likely to be totally effective, even at small doses, so they are unlikely to give rise to a threshold in the dose-response relationship. The probability of a cancer resulting from the radiation will be at least partly dependent on the number of clones of modified cells initially created, since this number will influence the probability of at least one clone surviving. It is then the probability of malignancy that is related to dose, while the severity of a particular cancer is influenced only by the type and location of the malignant condition. The process appears to be random, although individuals may differ somewhat in their sensitivities to the induction of cancer by radiation, reflecting genetic and physiological variations. Some individuals with rare genetic diseases may be substantially more sensitive than the mean. It seems that no stochastic effects in the exposed individual other than cancer (and benign tumours in some organs) are induced by radiation. In particular, any life-shortening found in exposed human populations and in experimental animals after low doses has been shown to be due to excess radiation-induced cancer mortality.

(63) Many million million ion pairs are created every year in the total mass of DNA in a human being by the exposure of the body to natural sources of radiation. No more than about one death in four is attributable to cancer and radiation is responsible for only a small fraction of these cancer deaths. Clearly, the process of passing from the creation of an ion pair in the DNA to the manifestation of a cancer is very rarely completed.

(64) The process of drawing conclusions about stochastic effects is not straightforward because epidemiological studies cannot provide exactly the information needed. They can provide only statistical associations, but they are strengthened when the association is clearly dose-related and is supported by corresponding experimental data. The data from Japan are compelling and are extensive, but they relate to a study group of which about 60% now survive, so the total number of stochastic effects eventually occurring has to be estimated. Moreover, most of the cancers yet to appear will occur in individuals who were under the age of 20 years at the time of exposure, and for whom the attributable lifetime fatality probability per unit dose is probably higher than that for older individuals. Although the study group is large (about 80,000), excess numbers of malignancies, statistically significant at the 95% level, can be found only at doses exceeding about 0.2 Sv. Excesses of lower significance can be found at doses in the region of 0.05 Sv. It must also be borne in mind that all the doses to the Japanese study group were incurred at very high dose rates, whereas information is needed in radiological protection for both acute and protracted exposures, almost always at very much

lower dose rates. However, studies on this group have several advantages over other studies. The group contains both sexes and all ages, and was exposed to a very wide range of doses, from trivial to fatal, distributed fairly uniformly through the bodies of those exposed.

(65) The studies on patients also pose problems. In particular, the irradiations were intentionally non-uniform, the selection of patients on medical grounds sometimes makes it difficult to identify comparable control groups, and the patients may not be representative of the general population. Nevertheless, such groups provide valuable sources of information and are the subject of continuing study.

(66) The studies on workers that have so far yielded significant results relate to those who worked with radium-226 in the early decades of the 20th century and to those who inhaled radon and its daughters in mining, mainly uranium mining, in the middle years of the century. In both cases, there were difficulties in estimating the intake of radioactive materials and the uranium miners may also have been exposed to other carcinogens. The exposures were protracted, but the doses were to localised tissues in the bone and lung and were essentially confined to those from alpha particles. Comparison with the effects of gamma radiation is not simple. Studies on the early radiologists show some stochastic effects, but the estimation of dose is not easy, and quantitative risk estimates have not proved possible. Studies on other groups of workers, such as those in atomic energy laboratories in the US and the UK have provided estimates of risk, with however, very wide confidence intervals. Their range of estimates includes the nominal fatal probability coefficients given in this report.

(67) Numerous reports involving the exposure of populations to low doses of radiation appear in the literature from time to time and are carefully examined by the Commission. Some of these arise from exposure to nuclear sources such as fallout, some involve military personnel exposed at weapons tests and some in the environment of nuclear plants. Others include fetuses exposed to diagnostic x rays, other medically irradiated populations and still other populations living in relatively high natural radiation background areas in the world, including those in India, Brazil, Colorado USA and China. Such low-dose studies avoid the need for the application of factors from high dose-rate information to low dose-rate circumstances, i.e. the DDREF (see paragraph 74). On the other hand, these studies suffer from one or more of the following methodological difficulties including small sample size, lack of adequate controls, extraneous effects other than those due to radiation, inadequate dosimetry and confounding social factors. Furthermore "positive" findings tend to be reported while negative studies often are not. Overall, studies at low dose, while potentially highly relevant to the radiation protection problem, have contributed little to quantitative estimates of risk.

(68) If, as seems likely, some types of cancer can result from the damage originating in a single cell, there can be a real threshold in the dose-response relationship for those types of cancer only if the defence mechanisms are totally successful at small doses. The balance of damage and repair in the cell and the existence of subsequent defence mechanisms can influence the shape of the relationship, but they cannot be expected to result in a real threshold.

(69) At small increments of dose above background, the probability of inducing an additional cancer is certainly small and the expectation value of the number of cases attributable to the increment of dose in an exposed group may well be much less than 1, even in a large group. It is then almost certain that there will be no additional cases, but this provides no evidence for the existence of a real threshold.

(70) In almost all situations apart from accidents and the treatment of patients, the

equivalent dose in individuals is incurred over long periods of time and at annual rates that do not add greatly to the dose delivered to the whole body by natural sources. The annual addition from artificial sources ranges typically from a small fraction of the annual dose from natural sources up to about ten times that annual dose. The lung is a special case because the equivalent dose from radon daughters is very variable and is sometimes as much as several thousand times higher than the equivalent dose to other parts of the body from natural sources.

(71) The existence of doses in all parts of the body from natural sources of radiation decreases the importance of the shape of the dose-response relationship at doses close to zero. Small doses are always additions to the natural background dose. For moderate increments above the background, a linear relationship between the incremental dose and the incremental probability of a deleterious effect will be an adequate approximation, whatever may be the true shape of the relationship between equivalent dose and the probability of stochastic effects. Even so, the shape of this relationship is still important because it can change the estimates of the slope of the incremental relationship.

(72) The simplest relationship between an increment in equivalent dose and the resulting increment in the probability of a defined stochastic effect is that of a straight line through the origin. The human epidemiological data are not sufficiently precise to confirm or exclude that relationship. However, almost all the data relating to stochastic changes in cells *in vitro* and in simple biological organisms such as *tradescantia*, and to the induction of many animal tumours, show curvilinear dose-effect relationships for radiations of low linear energy transfer (LET), with the slope at low doses being less than that at high doses. In this context, low doses (and low dose rates) imply situations in which it is very unlikely that more than one ionising event will occur in the critical parts of a cell within the time during which repair mechanisms in the cell can operate. In such situations, the dose-response relationship will be linear. At higher doses and dose rates, two or more events may be able to combine, producing an enhanced effect reflected by a quadratic term in the dose-response relationship. At still higher doses, where cell killing becomes important, the slope again decreases. The results for radiations of high LET are usually more nearly rectilinear over the range of doses below those causing appreciable cell killing. Some cellular studies *in vitro*, however, show an increased slope at the low-dose end of this range.

(73) In short, for low LET radiations, the most characteristic form of the relationship between the equivalent dose in an organ and the probability of a resultant cancer is that of an initial proportional response at low values of equivalent dose, followed by a steeper rate of increase (slope) that can be represented by a quadratic term, followed finally by a decreasing slope due to cell killing. There are no adequate grounds for assuming a real threshold in the relationship. This form of response, while typical, is not necessarily the definitive form for all human cancers. Taken together with the linear approximation for increments over the dose due to natural background, it provides a suitable basis for the Commission's use of a simple proportional relationship at all levels of equivalent dose and effective dose below the dose limits recommended in this report.

(74) The Commission has concluded that, in the context of radiological protection, there is sufficient evidence to justify its making an allowance for non-linearity when interpreting data for low LET radiation at high doses and high dose rates to give estimates of the probability of effects at low doses and low dose rates. On the basis of discussions in Annex B, the Commission has decided to reduce by a factor of 2 the probability co-

efficients obtained directly from observations at high doses and high dose rates, modified if necessary by an allowance for the effects of cell killing. There is a wide spread in the data and the Commission recognises that the choice of this value is somewhat arbitrary and may be conservative. No such factor is used in the interpretation of data for high LET radiation. The reduction factor is called by the Commission the *Dose and Dose Rate Effectiveness Factor*, DDREF. It has been included in the probability coefficients for all equivalent doses resulting from absorbed doses below 0.2 Gy and from higher absorbed doses when the dose rate is less than 0.1 Gy per hour.

(75) Another major difficulty in interpreting the human data is that of estimating the number of stochastic effects yet to appear in the populations being studied. For a few cancers, there is no difficulty because the rate of appearance of new cases has fallen back to, or close to, the expected rate in a matched control population. This is true of leukaemia in the Japanese survivors and the British spondylitics and of bone cancer in the patients injected with radium-224. For the total of other cancers, the rate is still enhanced and, in the Japanese study, still rising, largely as a result of the excess mortality in those exposed as children.

(76) For most types of cancer, the excess mortality seems, after an initial period of zero or very low risk called the minimum latency period, to have the same pattern in time as the natural mortality due to the same type of cancer. If this pattern is continued throughout life, and this is by no means certain, there will be a simple proportion between the natural cancer mortality and the excess due to radiation for the whole time after the minimum latency period. This model, the *multiplicative risk projection model*, is probably too simple, even for the exposure of adults. The Japanese data show that neither it nor the *additive risk projection model* (see below) adequately fits the pattern of mortality following the exposure of young children. The model does not necessarily imply a *multiplicative biological process*—it may only be a convenient description of the way in which the probability of an attributable cancer varies with time after exposure.

(77) An alternative model, the *additive risk projection model*, postulates that the excess mortality would be broadly independent of the natural mortality. After the initial minimum latency period, the rate would rise over a period of years after exposure and then remain fairly constant or, as with leukaemia and bone cancer, fall. This model, with current probability coefficients, produces predictions of eventual total probability of death of about half the values predicted by the multiplicative risk projection. It also predicts more time lost per attributable death. However, it is no longer seen to be consistent with most of the epidemiological observations.

(78) Because of the uncertainties of recording cancer incidence rather than mortality, most of the data on exposed human populations are expressed in terms of excess cancer mortality attributable to the exposures. However, the incidence of cancer is also important and the Commission takes it into account on the basis of currently observed cure rate for the main types of cancer. More generally, the Commission needs a broader basis for expressing the harm expected in an exposed population and has therefore made use of the concept of detriment as discussed in Section 3.3. Hereditary effects are discussed in Section 3.4.3.

(79) All these difficulties introduce uncertainties into the estimation of the cancer risks from exposure to radiation. For this reason, and because the Commission estimates the risks for representative populations with defined exposure patterns, the Commission calls the estimated probability of a fatal cancer per unit effective dose the *nominal fatality probability coefficient*. This applies to low doses at all dose rates and to high doses and

low dose rates (see paragraph 74). In deriving values of the nominal probability coefficient, the Commission has previously used the probability of induction of a fatal cancer without making any allowance for the reduction in that probability resulting from competing causes of death. If a multiplicative, rather than additive, risk projection model is used, that correction is essential. The correction is now used by the Commission in deriving all values of probability coefficients. As will be discussed in Chapter 5, it is very desirable for protection purposes to use the same nominal coefficients for both men and women and for a representative population of a wide range of ages. Although there are differences between the sexes and between populations of different age-specific mortality rates, these are not so large as to necessitate the use by the Commission of different nominal probability coefficients. A small difference is, however, introduced between the nominal probability coefficients for workers and for the whole population. Although small, this difference is very likely to exist because it arises principally from the inclusion of the more sensitive younger age groups in the whole population.

(80) Reviews of the available data are summarised in Annex B. In choosing a value for the nominal probability coefficients, the Commission has had to take account of a wide range of options. Because the data from Japan are derived from a large population of all ages and both sexes, and because the doses are fairly uniformly distributed through the whole body, these data have been taken as the primary source of information. The interpretation of the data from the irradiated spondylitic patients leads to a lower estimate of the annual probability of fatal cancer per unit dose by a factor of about two. Lower estimates can also be derived from studies on patients treated for cervical cancer, although the doses were very non-uniform. These data confirm the Commission's view that the estimates based on the data from Hiroshima and Nagasaki are unlikely to underestimate the risks.

(81) The Commission has also had to select a risk projection model. For leukaemia, the choice of model has little effect because it is likely that almost all the leukaemia deaths have already been observed. The combination of models used by the Commission emphasises the multiplicative model for cancers other than leukaemia, with the understanding that this may overestimate the probability of cancer incidence at older ages because the multiplying factor may not persist over the whole span of life. The effect of competing causes of death reduces the importance of any such error.

(82) Finally, the Commission has had to decide how to transfer conclusions reached about the post-war Japanese population to other populations. Again two models are available. Either the absolute mortality rate per unit dose can be applied to the other populations or the transfer can be made by using the proportional increase in the mortality rate of each type of cancer in turn. In either case, the mortality pattern of the new population has to be used to allow for competing causes of death. The Commission has averaged over five populations to give a reasonable representation of a typical population. There is no adequate basis at present for making a choice between the two transfer models and the Commission has used the average of both methods.

(83) The data in Annex B relating to high doses and high dose rates of low LET radiation, show a lifetime fatality probability coefficient for a reference population of both sexes and of working age, of about  $8 \times 10^{-2} \text{ Sv}^{-1}$  for the sum of all malignancies. This value, combined with the DDREF of 2, leads to a nominal probability coefficient for workers of  $4 \times 10^{-2} \text{ Sv}^{-1}$ . The corresponding values for the whole population, including children, are about  $10 \times 10^{-2} \text{ Sv}^{-1}$  for high doses and dose rates and  $5 \times 10^{-2} \text{ Sv}^{-1}$  for low dose and dose rates (see Table 3). Typically, the multiplicative model shows a mean

loss of life per attributable cancer death of about 13 to 15 years. The additive model gives a corresponding figure of about 20 years.

(84) Extensive data exist on the relationship between the probability of bone cancer and the radium content of workers in the early luminising industry; between the probability of bone cancer in patients and the activity of radium-224 injected; and between the probability of lung cancer and the estimated exposure to radon and its daughters in mining environments. In almost all these cases, it is difficult to estimate the dosimetric quantities and thus these human data do not provide good estimates of the relationship between the stochastic effects from exposure to high LET radiation and the doses to human organs. However, it is known from studies on cells and from work with experimental animals that, per unit absorbed dose, high LET radiations cause more stochastic damage than do low LET radiations.

(85) Values of the relative biological effectiveness do not lead directly to values of the radiation weighting factor. Experimental data from animals and cells are used to estimate the relevant values of RBE for typical stochastic effects at low doses. The experimental studies use either x rays with an energy of a few hundred keV or gamma rays of energy of about 1 MeV. While these radiations are about equally effective at high doses and high dose rates, there is a factor of about two in biological effectiveness between these two energy bands at low doses. Since the values of radiation weighting factor have to apply to all the tissues and organs in the body, a substantial degree of simplification is needed. The Commission has therefore not distinguished between x and gamma radiation and has selected values of radiation weighting factor for other radiations broadly representative of the observed values of RBE relative to either x or gamma radiation. The nominal fatality probability coefficients per unit equivalent dose and per unit effective dose for high LET radiation are then the same as those for low LET radiation. The values are given in Table 1 in Chapter 2.

(86) In the special case of lung cancer from inhaled radon progeny, the epidemiological data from radon-exposed miners yield a direct relationship between their cumulative exposure to radon progeny and the excess probability of lung cancer (see Annex B). In these circumstances it is reasonable to express the attributable risk coefficient per unit of radon exposure and not per unit dose to the lung or the bronchial epithelium.

### 3.4.3. Stochastic effects in progeny

(87) If the damage caused by radiation occurs in the germ cells, this damage (mutations and chromosomal aberrations) may be transmitted and become manifest as hereditary disorders in the descendants of the exposed individual. Radiation has not been identified as a cause of such effects in man, but experimental studies on plants and animals suggest that such effects will occur and that the consequences may range from the undetectably trivial, through gross malformations or loss of function, to premature death. It must be presumed that any non-lethal damage in human germ cells may be further transmitted to subsequent generations. This type of stochastic effect is called "hereditary".

(88) Hereditary effects vary widely in their severity. One such effect is the production of dominant mutations leading to genetic disease in the first generation progeny. Some of these conditions are seriously harmful to the affected individual and are sometimes life-threatening. They occur predominantly in the first and second generations after exposure. Chromosomal aberrations may also result in congenital abnormalities in children.

Recessive mutations produce little effect in the first few generations of descendants, but make a contribution to the general pool of genetic damage in subsequent generations. There are also many deleterious conditions that have a substantial incidence in man and which are due to the interaction of genetic and environmental factors. They are known as multifactorial disorders. A general increase in mutations might increase their incidence, although this has not been demonstrated in either man or animals. In assessing the consequences for exposed individuals, the Commission has previously taken account of the hereditary effects that might occur in their children and grandchildren. This left the effects in later generations to be considered as part of the consequences for society. The Commission now attributes the whole detriment to the dose received by the exposed individual, thus avoiding the need for a two-stage assessment.

(89) For low doses and dose rates, the nominal hereditary effect probability coefficient for severe effects (excluding multifactorial effects, see below) over all generations and related to the gonad doses distributed over the whole population is  $0.5 \times 10^{-2} \text{ Sv}^{-1}$ . About 80% of the effects are due to dominant and X-linked mutations. Of these, about 15% occur in each of the first two generations. No reliable estimate is available for the probability coefficient for the multifactorial conditions, but, weighted for severity, it is probably about  $0.5 \times 10^{-2} \text{ Sv}^{-1}$ . Because of the different age distribution of a working population, the coefficients for workers are slightly smaller than for the general population (a reduction by about 40%). The Commission considers that the nominal hereditary effect probability coefficients of  $1 \times 10^{-2} \text{ Sv}^{-1}$  for the whole population and  $0.6 \times 10^{-2} \text{ Sv}^{-1}$  for workers adequately represent the weighted number of hereditary effects to be expected in all generations (see Table 3). This only includes weighting for severity. With further weighting for years of life lost if the harm occurs (see paragraph 96), the corresponding numbers will be  $1.3 \times 10^{-2} \text{ Sv}^{-1}$  and  $0.8 \times 10^{-2} \text{ Sv}^{-1}$  (see Table 4).

Table 3. Nominal probability coefficients for stochastic effects

Exposed population	Detriment ( $10^{-2} \text{ Sv}^{-1}$ ) <sup>1</sup>			Total
	Fatal cancer <sup>2</sup>	Non-fatal cancer	Severe hereditary effects	
Adult workers	4.0	0.8	0.8	5.6
Whole population	5.0	1.0	1.3	7.3

<sup>1</sup> Rounded values.

<sup>2</sup> For fatal cancer, the detriment coefficient is equal to the probability coefficient.

#### 3.4.4. Effects of antenatal exposure

(90) The effects on the conceptus of exposure to radiation depend on the time of exposure relative to conception. When the number of cells in the conceptus is small and their nature is not yet specialised, the effect of damage to these cells is most likely to take the form of a failure to implant or of an undetectable death of the conceptus. It is thought that any cellular damage at this stage is much more likely to cause the death of the conceptus than to result in stochastic effects expressed in the live-born. Exposure of the embryo in the first three weeks following conception is not likely to result in deterministic or stochastic effects in the live-born child, despite the fact that the central nervous system and the heart are beginning to develop in the third week. During the rest

of the period of major organogenesis, conventionally taken to be from the start of the third week after conception, malformations may be caused in the organ under development at time of exposure. These effects are deterministic in character with a threshold in man, estimated from animal experiments, to be about 0.1 Gy.

(91) Throughout the period from 3 weeks after conception until the end of pregnancy, it is likely that radiation exposure can cause stochastic effects resulting in an increased probability of cancer in the live-born. The available data are not consistent and considerable uncertainty exists. However, the Commission assumes that the nominal fatality probability coefficient is, at most, a few times that for the population as a whole.

(92) Values of intelligence quotient (IQ) lower than expected have been reported in some children exposed in utero at Hiroshima and Nagasaki. There have been two principal quantitative findings. One is the observation of a general downward shift in the distribution of IQ with increasing dose. The Commission assumes that the shift is proportional to dose. Small shifts cannot be clinically identified. A coefficient of about 30 IQ points  $\text{Sv}^{-1}$  relates to the dose in the fetus in the period from 8 weeks to 15 weeks after conception. A similar, but smaller shift, is detectable following exposure in the period from 16 weeks to 25 weeks. This appears to be a deterministic effect, probably with a threshold determined only by the minimum shift in IQ that can be clinically recognised.

(93) The second finding is of a dose-related increase in the frequency of children classified as "severely retarded". The number of cases is small, but the data indicate an excess probability of severe mental retardation of 0.4 at 1 Sv. As shown in Annex B, this finding is consistent with the general shift in IQ distribution with increasing dose. Because of the Gaussian shape of the IQ distribution, the excess number of cases of severe mental retardation will be very small at small IQ shifts, rising steeply only as the shift approaches 30 IQ points. On this basis, a large change in the IQ of an individual can be caused only by a large dose. At doses of the order of 0.1 Sv, no effect would be detectable in the general distribution of IQ, but at somewhat larger doses the effect might be sufficient to show an increase in the number of children classified as severely retarded. The effects at all levels of dose are less marked following exposure in the period from 16 weeks to 25 weeks after conception and have not been observed for other periods. All the observations on IQ and severe mental retardation relate to high dose and high-dose rates and their direct use probably overestimates the risks.

#### 3.5. Tissue Weighting Factors

(94) The tissue weighting factors introduced in Chapter 2 for defining the quantity effective dose were intended to ensure that a weighted tissue equivalent dose would produce broadly the same degree of detriment irrespective of the tissue or organ involved. The Commission has adopted an aggregated representation of detriment for this purpose. It includes four components: the probability of attributable fatal cancer, the weighted probability of attributable non-fatal cancer, the weighted probability of severe hereditary effects and the relative length of life lost. Since effective dose will be used only over ranges where the total probability of attributable death will be small, even the fatal contribution to detriment can be treated as additive when several organs are irradiated. Each consequence can then be weighted by a factor chosen to represent its severity. As in Publication 26, death and severe hereditary effects are both given a weighting factor of 1.

(95) Discussions in *Publication 45* (1985) suggest a weight for non-fatal cancers relative to fatal cancers equal to the average lethality fraction of the cancer concerned. A type of cancer that is difficult to cure, and thus has a high lethality fraction and usually a reduced quality of life for the survivors, would have a high weighting factor for the non-fatal events, while an easily cured cancer would have a low weighting factor for the non-fatal events. The weights would then range from about 0.01 for non-fatal skin cancer to about 0.99 for non-fatal leukaemia. The weighting factor to be applied to the fatality coefficient is derived in Annex B. The weighting factors for the severity of hereditary effects is already included in the probability coefficients.

(96) A second weighting is applied to take account of the different mean latency time for different types of cancer. This weighting is simply the relative time lost due to an attributable cancer death or, in the case of non-fatal cancers and hereditary effects, the relative time of impaired life taken for cancers as the same as the time lost by death for the same type of cancer. Finally, the products of the mortality coefficient and the weighting factors for morbidity and time lost are normalised to give a total of unity and thus provide a basis for the tissue weighting factors recommended by the Commission. These tissue weighting factors are provided as rounded values for individual tissues and organs and are given in Table 2 on bases set out in Annex B.

(97) The data in Table 4 are representative of those for a nominal population of equal numbers of men and women. Except for the breast, the differences between the sexes are small. The effect on the tissue weighting factors of combining the data is that some weighting factors are slightly higher and some slightly lower than the values that would relate to men and women separately. The effect of confining the population to workers is

Table 4. Nominal probability coefficients for individual tissues and organs<sup>1</sup>

Tissue or organ	Probability of fatal cancer ( $10^{-2} \text{ Sv}^{-1}$ )		Aggregated detriment <sup>2</sup> ( $10^{-2} \text{ Sv}^{-1}$ )	
	Whole population	Workers	Whole population	Workers
Bladder	0.30	0.24	0.29	0.24
Bone marrow	0.50	0.40	1.04	0.83
Bone surface	0.05	0.04	0.07	0.06
Breast	0.20	0.16	0.36	0.29
Colon	0.85	0.68	1.03	0.82
Liver	0.15	0.12	0.16	0.13
Lung	0.85	0.68	0.80	0.64
Oesophagus	0.30	0.24	0.24	0.19
Ovary	0.10	0.08	0.15	0.12
Skin	0.02	0.02	0.04	0.03
Stomach	1.10	0.88	1.90	0.80
Thyroid	0.08	0.06	0.15	0.12
Remainder	0.50	0.40	0.59	0.47
Total	5.00	4.00	5.92	4.74
	Probability of severe hereditary disorders			
Genetics	1.10	0.6	1.23	0.80
Grand total (rounded)			7.3	5.6

<sup>1</sup> The values relate to a population of equal numbers of both sexes and a wide range of ages.  
<sup>2</sup> See paragraphs 95 and 96 and Table B-20 in Annex B.

to decrease the nominal probability coefficient for workers to  $4 \times 10^{-2} \text{ Sv}^{-1}$ , but does not significantly change the values of the tissue weighting factors.

(98) If the equivalent dose is fairly uniform over the whole body, it is possible to obtain the probability of fatal cancer associated with that effective dose from the nominal fatality probability coefficient. If the distribution of equivalent dose is non-uniform, this use of the nominal coefficient will be less accurate because the tissue weighting factors include allowances for non-fatal and hereditary conditions. For example, the contribution of fatalities from the equivalent dose in the lung will be underestimated by about 25%, and the contribution from the skin and thyroid will be overestimated by a factor of about 3. If the tissue equivalent doses are known, the nominal fatality probability coefficients for the individual tissues and organ can be used, but the difference between the two methods will not be significant because the individual tissue coefficients are not known with sufficient accuracy. The necessary data for both methods are provided in Table 4. As an approximation for a wide range of distributions of equivalent dose, the non-fatal somatic detriment adds about 20–30% to the fatal detriment.

#### 4. THE CONCEPTUAL FRAMEWORK OF RADIOLOGICAL PROTECTION

Chapter 4 deals with the general policy of radiological protection. It introduces the idea of source-related and individual-related assessments. It outlines the basic system of protection for occupational, medical, and public exposures and distinguishes between a "practice", which causes exposures to radiation, and "intervention", which decreases exposures.

##### 4.1. The Basic Framework

(99) Everyone in the world is exposed to radiation from natural and artificial sources. Any realistic system of radiological protection must therefore have a clearly defined scope if it is not to apply to the whole of mankind's activities. It also has to cover, in a consistent way, a very wide range of circumstances.

(100) The basic framework of radiological protection necessarily has to include social as well as scientific judgements, because the primary aim of radiological protection is to provide an appropriate standard of protection for man without unduly limiting the beneficial practices giving rise to radiation exposure. Furthermore, it must be presumed that even small radiation doses may produce some deleterious health effects. Since there are thresholds for deterministic effects, it is possible to avoid them by restricting the doses to individuals. On the other hand, stochastic effects cannot be completely avoided because no threshold can be invoked for them. The Commission's basic framework is intended to prevent the occurrence of deterministic effects, by keeping doses below the relevant thresholds, and to ensure that all reasonable steps are taken to reduce the induction of stochastic effects.

(101) Most decisions about human activities are based on an implicit form of balancing benefits against costs and disadvantages, leading to the conclusion that a particular course of action or practice either is, or is not, worthwhile. Less commonly, it is also recognised that the conduct of a practice should be adjusted to maximise the net benefit to the individual or to society. This is not a simple process because the objectives

of the individual and society may not coincide. In radiological protection, as in other areas, it is becoming possible to formalise and quantify procedures that help in reaching these decisions. In doing so, attention has to be paid, not only to the advantages and disadvantages for society as a whole, but also to the protection of individuals. When the benefits and detriments do not have the same distribution through the population, there is bound to be some inequity. Serious inequity can be avoided by the attention paid to the protection of individuals. It must also be recognised that many current practices give rise to doses that will be received in the future, sometimes the far future. These future doses should be taken into account in the protection of both populations and individuals, although not necessarily on the same basis as is used for current doses. Current practices may also give rise to a probability, but not a certainty, that exposures will occur. The probability of incurring the exposures is then important, in addition to the magnitude of the exposures.

(102) To clarify the way in which the Commission has developed its recommendations, it is convenient to think of the processes causing human exposures as a network of events and situations. Each part of the network starts from a source. This term is used by the Commission to indicate the source of an exposure, not necessarily a physical source of radiation. Thus the source of occupational exposures in a hospital might be the x-ray units, rather than the anodes which are the physical source of the x-rays. When radioactive materials are released to the environment as waste, the installation as a whole might be regarded as the source. Radiation or radioactive material then passes through environmental pathways, which may be simple in a workplace, but very complex in the natural environment, with some pathways being common to many sources. Eventually, individuals, possibly many individuals, are exposed as a result of a single original source. Since there can be many sources, some individuals will be exposed to radiation from more than one of them. If natural sources are included, all individuals are exposed to radiation from at least a few sources.

(103) Fortunately, it is rarely necessary to treat this network as a single entity. Provided that the individual doses are well below the threshold for deterministic effects, the contribution to an individual dose from a single source has an effect that is independent of the doses from other sources. For many purposes, each source, or group of sources, can then be treated on its own. Each individual, however, is exposed as a result of several sources. It follows that assessments of the effectiveness of protection can be related to the source giving rise to the individual doses (source-related) or related to the individual dose received by a person from all the relevant sources (individual-related).

(104) Source-related assessments make it possible to judge whether a source is likely to bring benefits sufficient to outweigh any disadvantages that it may have, and whether all reasonable steps have been taken to reduce the radiation exposures that it will cause. The source-related assessment will take account of the magnitude and the probability of occurrence of individual doses attributable to that source, and of the number of individuals so exposed, but will not consider the additional contributions from other sources.

(105) It will therefore be necessary also to consider an individual-related assessment of the total doses in individuals from all the relevant sources, in order to determine whether any individual has too high a probability of stochastic effects and whether any individual dose approaches one of the thresholds for deterministic effects.

(106) Some human activities increase the overall exposure to radiation, either by

introducing whole new blocks of sources, pathways, and individuals, or by modifying the network of pathways from existing sources to man and thus increasing the exposure of individuals or the number of individuals exposed. The Commission calls these human activities "practices". Other human activities can decrease the overall exposure by influencing the existing form of the network. These activities may remove existing sources, modify pathways, or reduce the number of exposed individuals. The Commission describes all these activities as "intervention".

(107) The steps needed to restrict the exposure of individuals, either in the control of a practice or by intervention, can be taken by applying action at any point in the network linking the sources to the individuals. The action may be applied to the source, to the environment, or to the individual. Actions that can be applied at the source will be the least disruptive. They can be made as effective as is required, unless they fail as the result of an accident. They influence all the pathways and individuals associated with that source. In the extreme case, the action may be to avoid the use of the source. Where available, controls applied at the source are to be preferred. Actions applied to the environment or to individuals are more obtrusive and may have social disadvantages, not all of which are foreseeable. Their effectiveness will be limited because they apply only to some of the pathways and individuals.

(108) The Commission's system of protection is intended to be as general as possible, partly for consistency and partly to avoid changes of policy resulting from the demarcation of different situations. However, the various types of exposure and the distinction between practices and intervention give rise to different degrees of controllability and thus influence the judgements about the reasonableness of the various control procedures.

(109) The Commission uses a division into three types of exposure: occupational exposure, which is the exposure incurred at work, and principally as a result of work; medical exposure, which is principally the exposure of persons as part of their diagnosis or treatment; and public exposure, which comprises all other exposures. More detailed descriptions are given in Chapter 5.

(110) In the control of occupational exposure, it is usually possible to apply controls at all three points: at the source, by fixing its characteristics and its immediate shielding and containment; in the environment, by ventilation or additional shielding; and at the individual, by requiring working practices and the use of protective clothing and equipment. Not all these levels of control are needed all the time. In medical exposures, the controls are also applied at all three points, but mainly as part of the primary function of diagnosis or treatment, rather than as part of a separate system of protection. In public exposure, the controls should be applied at the source. Only if these cannot be made effective should controls be applied to the environment or to individuals.

(111) The appropriate control measures also depend on whether they are to be applied to a practice causing exposures or to intervention aimed at reducing exposures. In the case of a new practice, there is the option of accepting the practice, as proposed or with modifications, or of rejecting it outright. Existing practices can be reviewed in the light of new information or changed standards of protection and, at least in principle, can be withdrawn; but the sources and pathways that they involve may persist. Any further changes then require intervention. Accidents, once they have occurred, give rise to situations in which the only available action is some form of intervention. In practices and in intervention, it will often be virtually certain that exposures will occur and their magnitude will be predictable, albeit with some degree of uncertainty. Sometimes, how-

ever, there will be a potential for exposure, but no certainty that it will occur. The Commission calls such exposures "potential exposures". It is often possible to apply some degree of control to both the probability and the magnitude of potential exposures.

#### 4.2. The System of Radiological Protection

(112) The system of radiological protection recommended by the Commission for proposed and continuing practices is based on the following general principles. Details of the system in relation to practices are given in Chapter 5. The system for intervention is discussed in the next paragraph and in Chapter 6.

- (a) No practice involving exposures to radiation should be adopted unless it produces sufficient benefit to the exposed individuals or to society to offset the radiation detriment it causes. (The justification of a practice.)
- (b) In relation to any particular source within a practice, the magnitude of individual doses, the number of people exposed, and the likelihood of incurring exposures where these are not certain to be received should all be kept as low as reasonably achievable, economic and social factors being taken into account. This procedure should be constrained by restrictions on the doses to individuals (dose constraints), or the risks to individuals in the case of potential exposures (risk constraints), so as to limit the iniquity likely to result from the inherent economic and social judgements. (The optimisation of protection.)
- (c) The exposure of individuals resulting from the combination of all the relevant practices should be subject to dose limits, or to some control of risk in the case of potential exposures. These are aimed at ensuring that no individual is exposed to radiation risks that are judged to be unacceptable from these practices in any normal circumstances. Not all sources are susceptible of control by action at the source and it is necessary to specify the sources to be included as relevant before selecting a dose limit. (Individual dose and risk limits.)

(113) The system of radiological protection recommended by the Commission for intervention is based on the following general principles.

- (a) The proposed intervention should do more good than harm, i.e. the reduction in detriment resulting from the reduction in dose should be sufficient to justify the harm and the costs, including social costs, of the intervention.
- (b) The form, scale, and duration of the intervention should be optimised so that the net benefit of the reduction of dose, i.e. the benefit of the reduction in radiation detriment, less the detriment associated with the intervention, should be maximised.

Dose limits do not apply in the case of intervention (see paragraph 131). Principles (a) and (b) can lead to intervention levels which give guidance to the situations in which intervention is appropriate. There will be some level of projected dose above which, because of serious deterministic effects, intervention will almost always be justified.

(114) Any system of protection should include an overall assessment of its effectiveness in practice. This should be based on the distribution of doses achieved and on an appraisal of the steps taken to limit the probability of potential exposures. It is important that the basic principles should be treated as a coherent system. No one part should be taken in isolation. In particular, mere compliance with the dose limits is not a sufficient demonstration of satisfactory performance.

### 4.3. Radiological Protection in Proposed and Continuing Practices

#### 4.3.1. The justification of a practice

(115) Decisions concerning the adoption and continuation of any human activity involve a choice between possible options and are often carried out in two stages. The first stage is the examination of each option separately in order to identify those options which can be expected to do more good than harm. This provides a "short list" from which the preferred option can then be selected. The second stage, the final selection, will often involve the replacement of one existing practice by another. The net benefit of the change will then be the relevant feature rather than the net benefit of each option separately. The Commission recommends that, when practices involving exposure, or potential exposure, to radiation are being considered, the radiation detriment should be explicitly included in the process of choice. The detriment to be considered is not confined to that associated with the radiation—it includes other detriments and the costs of the practice. Often, the radiation detriment will be a small part of the total. The justification of a practice thus goes far beyond the scope of radiological protection. It is for these reasons that the Commission limits its use of the term justification to the first of the above stages, i.e. it requires only that the net benefit be positive. To search for the best of all the available options is usually a task beyond the responsibility of radiological protection agencies.

(116) The process of justification is required, not only when a new practice is being introduced, but also when existing practices are being reviewed in the light of new information about their efficacy or consequences. If such a review indicates that a practice could no longer be claimed to produce sufficient benefit to offset the total detriment, withdrawal of the practice should be considered. This option should be treated in the same way as the justification of a new practice, but it must be remembered that the disadvantages of withdrawing a well-established practice may be more obvious than the advantages of introducing a comparable new one and withdrawal of the practice may not result in the withdrawal of all the associated sources of exposure. Preventing the further extension of an existing practice that is no longer justified may sometimes be a reasonable compromise, but will introduce an anomaly between the past and the present and will not always be seen as logical.

#### 4.3.2. The optimisation of protection

(117) Once a practice has been justified and adopted, it is necessary to consider how best to use resources in reducing the radiation risks to individuals and the population. The broad aim should be to ensure that the magnitude of the individual doses, the number of people exposed, and the likelihood of incurring exposures where these are not certain to be received, are all kept as low as reasonably achievable, economic and social factors being taken into account. Consideration has to be given to any interaction between these various quantities. If the next step of reducing the detriment can be achieved only with a deployment of resources that is seriously out of line with the consequent reduction, it is not in society's interest to take that step, provided that individuals have been adequately protected. The protection can then be said to be optimised and the exposures to be as low as reasonably achievable, economic and social factors having been taken into account. The procedure should also be applied when an existing practice is being reviewed.

(118) These considerations are complicated by the interaction between the various

factors to be included, and the methods for dealing with them are diverse. They range from simple common sense to complex techniques of cost-benefit analysis or multi-attribute analysis. In the Commission's view, all these techniques are aids to deciding when sufficient effort has been applied to the reduction of the detriment associated with a practice or with an identifiable component of a practice. Except when dealing with potential exposures, it is appropriate to use the effective dose as a surrogate for detriment to an individual, because the weighting factors used in calculating the effective dose take account of the whole detriment to the health of individuals and their descendants, not only the fatal detriment. The collective effective dose is an adequate representation of the collective detriment. For potential exposures, the situation is more complicated. (See Section 4.3.4.)

(119) The judgements involved in optimising protection are not purely quantitative—they involve preferences between detriments of different kinds and between the deployment of resources and health effects. Guidance on the necessary techniques has already been published by the Commission in *Publication 37* (1983) and *Publication 55* (1989).

(120) The process of optimising protection should be carefully structured. It is essentially source-related and should first be applied at the design stage of any project. It is here that dose reductions are most likely to be achievable in cost-effective ways. In achieving a design optimised for protection, designers should take account of, and influence, the way the plant or equipment will subsequently be used, although their information and influence on these future operational aspects may be limited. They may also wish to take account of the substantial advantages offered by engineering standardisation. At the design stage, therefore, the process of optimisation of protection will have some generic aspects. Further optimisation of protection should be carried out at the operational level. Operational optimisation is usually informal, involving common-sense changes in procedures, but is often very effective.

(121) Most of the methods used in the optimisation of protection tend to emphasise the benefits and detriments to society and the whole exposed population. The benefits and detriments are unlikely to be distributed through society in the same way. Optimisation of protection may thus introduce a substantial inequity between one individual and another. This inequity can be limited by incorporating source-related restrictions on individual dose into the process of optimisation. The Commission calls these source-related restrictions dose constraints, previously called upper bounds. They form an integral part of the optimisation of protection. For potential exposures, the corresponding concept is the risk constraint. The choice of constraints depends on the circumstances and is discussed further in Chapter 5.

#### 4.3.3. Individual dose limits

(122) If the procedures of justification of practices and of optimisation of protection have been conducted effectively, there will be few cases where limits on individual dose will have to be applied. However, such limits provide a clearly defined boundary for these more subjective procedures and prevent excessive individual detriment, which might result from a combination of practices. The Commission's dose limits should be applied only in the control of practices.

(123) It is the Commission's intention to choose the values of dose limits so that any continued exposure just above the dose limits would result in additional risks from the defined practices that could reasonably be described as "unacceptable" in normal circumstances. Thus the definition and choice of dose limits involve social judgements.

These judgements are difficult, partly because the dose limit has to be set at a defined value and there is no discontinuity in the scale of acceptability. For agents like ionising radiation, for which no threshold can be assumed in the dose-response relationship for some of the consequences of exposure, this difficulty is inescapable and the choice of limits cannot be based on health considerations alone.

(124) In practice, several misconceptions have arisen about the definition and function of dose limits. In the first place, the dose limit is widely, but erroneously, regarded as a line of demarcation between "safe" and "dangerous". Secondly, it is also widely, and also erroneously, seen as the most simple and effective way of keeping exposures low and forcing improvements. Thirdly, it is commonly seen as the sole measure of the stringency of a system of protection. These misconceptions are, to some extent, strengthened by the incorporation of dose limits into regulatory instruments. Causing a dose limit to be exceeded then becomes an infraction of the rules and sometimes a statutory offence. Against this background, it is not surprising that managements, regulatory agencies, and governments all improperly set out to apply dose limits whenever possible, even when the sources are partly, or even totally, beyond their control, and when the optimisation of protection is the more appropriate course of action.

(125) It has also become apparent that dose limits are commonly used in two very different ways. In one application, mainly related to occupational exposure, the dose limit is regarded as a limiting restriction on the design and operation of an installation. In the other way, the dose limit is used in its original function of applying controls on each individual's accumulation of dose. It will never be appropriate to apply dose limits to all types of exposure in all circumstances. In circumstances for which they were not intended, e.g. in emergencies or during special operations of considerable importance, they can often be replaced by specially developed prescriptive limits or by specified levels of dose that call for the initiation of a defined course of action. Such levels, often called action or investigation levels or, in more general cases, reference levels, provide a useful way of structuring the procedures of radiological protection.

(126) For the above reasons the Commission has had to develop a more complex approach to dose limits. The specification of dose limits and the choice of values are discussed in Chapter 5.

#### 4.3.4. Potential exposures

(127) Not all exposures occur as forecast. There may be accidental departures from the planned operating procedures, or equipment may fail. Environmental changes may occur after the disposal of radioactive waste, or there may be changes in the way in which the environment is used. Such events can be foreseen and their probability of occurrence estimated, but they cannot be predicted in detail. The concept of both individual and collective detriment resulting from an exposure then has to be extended to allow for the fact that the exposure may not occur.

(128) Potential exposures need to be considered as part of the assessment of practices, but they may also lead to calls for intervention. Their implications should therefore be considered in both contexts. If the probability of occurrence of the event causing the potential exposures is fairly high, so that several such events might be expected within a year, it should be assumed that the doses resulting from the event will certainly occur.

(129) Dose limits do not apply directly to potential exposures. Ideally, they should be supplemented by risk limits, which take account of both the probability of incurring a dose and the detriment associated with that dose if it were to be received. However, risk

limits differ from dose limits in that the probability of occurrence and the magnitude of the potential exposure cannot be determined—they can only be inferred from an assessment of future scenarios. Furthermore, a potential exposure may become a real exposure and may then call for intervention. The problems are discussed further in Section 5.6.

#### 4.4. Radiological Protection by Intervention

(130) In some situations, the sources, the pathways, and the exposed individuals are already in place when the decisions about control measures are being considered. Sometimes, the new control procedures can be achieved as part of a review of the original practice, but, more commonly, they will constitute intervention. An important group of such situations is that involving exposure to natural sources of radiation. Accidents and emergencies will have been considered as sources of potential exposure when dealing with practices, but if they occur, they may call for intervention. All these cases are dealt with in Chapter 6.

(131) In most situations, intervention cannot be applied at the source and has to be applied in the environment and to individuals' freedom of action. The countermeasures forming a programme of intervention, which always have some disadvantages, should be justified in the sense that they should do more good than harm. Their form, scale and duration should then be optimised so as to maximise the net benefit. The dose limits recommended by the Commission are intended for use in the control of practices. The use of these dose limits, or of any other pre-determined dose limits, as the basis for deciding on intervention might involve measures that would be out of all proportion to the benefit obtained and would then conflict with the principle of justification. The Commission therefore recommends against the application of dose limits for deciding on the need for, or scope of, intervention. Nevertheless, at some level of dose, approaching that which would cause serious deterministic effects, some kind of intervention will become almost mandatory.

#### 4.5. The Assessment of the Effectiveness of a System of Protection

(132) When establishing that a system of protection is satisfactory, it is necessary to assess the overall effectiveness of the system. It is not appropriate merely to examine its component parts separately. When dealing with proposed or continuing practices, the expected or observed distributions of individual doses and the collective effective dose from defined operations should be considered. Comparisons between comparable operations and trends with time will often indicate the possibility of improvements. The assessment is more difficult for potential exposures because it is necessary to depend on an examination of the procedures for estimating the probability of the exposures. The probabilities cannot be directly determined. For intervention, including that resulting from accidents, the assessment should concentrate on the effectiveness of the forward planning and, retrospectively, on the effectiveness of the action taken in particular cases.

## 5. THE SYSTEM OF PROTECTION FOR PROPOSED AND CONTINUING PRACTICES

Chapter 5 indicates how the Commission develops the concepts described in Chapter 4 in the contexts of Occupational Exposure (the exposure of people at work), Medical Exposure (the exposure of people as part of their medical diagnosis or treatment), and

Public Exposure (all other exposures to radiation). It relates to practices, which cause exposure to radiation, and excludes intervention. It sets out the main structure of the recommended control procedures and, where relevant, defines the scope and recommended values of dose limits.

(133) The basic policies underlying the system of protection recommended by the Commission and described in Chapter 4 are developed in this chapter for application to practices. The chapter is subdivided to take account of the several types of exposure identified in Chapter 4, namely Occupational Exposure, Medical Exposure, and Public Exposure. There are many circumstances in which these types of exposure are best treated and discussed separately, as in this Chapter. Nevertheless, this separation is not always appropriate. For example, all types of exposure resulting from a practice have to be considered together in the justification of that practice. The justification of a practice has therefore been discussed fully in Chapter 4. However, some additional aspects of justification relating to medical practices are dealt with in Section 5.4.1. There are also situations in which decisions about public exposure interact with occupational exposures. These interactive situations are discussed in Section 5.7. The practical arrangements suggested for implementing the system of protection are discussed in Chapter 7.

### 5.1. Types of Exposure

#### 5.1.1. Occupational exposure

(134) The Commission has noted the conventional definition of occupational exposure to any hazardous agent as including all exposures incurred at work, regardless of their source. However, because of the ubiquity of radiation, the direct application of this definition to radiation would mean that all workers should be subject to a regime of radiological protection. The Commission therefore limits its use of the phrase "occupational exposure (to radiation)" to exposures incurred at work as the result of situations that can reasonably be regarded as being the responsibility of the operating management.

(135) Of the components of exposure to natural sources, those due to potassium-40 in the body, cosmic rays at ground level, and radionuclides in the earth's crust are all outside any reasonable scope of control. Only radon in workplaces and work with materials containing natural radionuclides can reasonably be regarded as the responsibility of the operating management. Furthermore, there is some exposure to radon in all workplaces, and it is important not to require the use of a formal system of separate decisions to exempt each individual workplace where controls are not needed. They should be excluded from the control of occupational exposure by some general system. Considerable knowledge and judgement is needed to define such a system. The Commission recommends that exposure to radon and the handling of materials containing traces of natural radionuclides should be regarded as excluded from occupational exposure and treated separately, unless the relevant regulatory agency has ruled otherwise, either in a defined geographical area or for defined practices.

(136) To provide some practical guidance, the Commission recommends that there should be a requirement to include exposures to natural sources as part of occupational exposure only in the following cases:

- (a) Operations in workplaces where the regulatory agency has declared that radon needs attention and has identified the relevant workplaces.

- (h) Operations with and storage of materials not usually regarded as radioactive, but which contain significant traces of natural radionuclides and which have been identified by the regulatory agency.
- (c) Operation of jet aircraft.
- (d) Space flight.

The definition of quantified specifications for cases (a) and (b) will depend on the local circumstances; but, as a very general guide, operations in spas, in most uranium mines, including open-cast mines, in many other underground mines and caves, and in some other underground workplaces are likely to constitute examples of case (a). Case (c) will relate principally to the aircraft crew, but attention should also be paid to groups such as coairers who fly more often than other passengers. Case (d) relates to very few individuals and will not be discussed further here.

(137) It is also necessary to consider how exposures to natural sources should be dealt with in workplaces where there is already a need for controls on the exposures directly associated with the work. It will be sufficient to take account of the exposures to natural sources if, and only if, they would be controlled in their own right as indicated in the previous paragraph. Elsewhere, they need not be included in radiation monitoring results, or in statistical reports of occupational exposures.

(138) Any exposure at work (excluding any medical exposure at work) as a result of artificial sources in, or associated with, the workplace should be included in occupational exposure, unless the sources have formally been excluded from regulatory control or exempted from the relevant aspects of regulatory control by the regulatory agency. Guidance on exclusion and exemption is given in Section 7.8.

#### 5.1.2. Medical exposure

(139) Medical exposure is confined to exposures incurred by individuals as part of their own medical diagnosis or treatment and to exposures (other than occupational) incurred knowingly and willingly by individuals helping in the support and comfort of patients undergoing diagnosis or treatment. Exposure of an individual to other sources, such as stray radiation from the diagnosis or treatment of other persons, is not included in medical exposure. Nor is any occupational exposure of staff. Exposures incurred by volunteers as part of a programme of biomedical research are also dealt with in this document on the same basis as medical exposure.

#### 5.1.3. Public exposure

(140) Public exposure encompasses all exposures other than occupational and medical exposures. The component of public exposure due to natural sources is by far the largest, but this provides no justification for reducing the attention paid to smaller, but more readily controlled, exposures to artificial sources.

### 5.2. The Application of the System of Protection

(141) The system of radiological protection described in Chapter 4 can usually be applied in much the same way in all types of exposure. Where there are significant differences, these are discussed in the following Sections. To some extent, different methods of application are needed for potential exposures, which are discussed separately in Section 5.6. Intervention is discussed in Chapter 6.

(142) It is necessary to consider the implications for radiological protection of different coefficients linking effective dose and detriment for different ages and sexes. These differences result from the effect of competing causes of death and the different intrinsic sensitivity of some tissues, notably the breast. However, as indicated in Section 3.5, reflecting these differences would have only a small effect on the definition of effective dose and on the nominal probability coefficient. In addition, many of the most effective methods of controlling exposures are applied without reference to the age and sex of those exposed, so it is desirable to set limits and to optimise protection in ways that are independent of both age and sex.

(143) The dose limits recommended in the following sections apply only to the sum of dose contribution from a relevant set of exposures and not to those from all sources of radiation. Because the identification of the relevant dose contributions cannot easily be generalised, the details are given in the following sections. However, in all cases the limits apply to the sum of all relevant doses from external exposure in the specified periods and the committed doses from intakes during the same periods.

### 5.3. The System of Protection in Occupational Exposure

#### 5.3.1. The optimisation of protection in occupational exposure

(144) An important feature of optimisation is the choice of dose constraints, the source-related values of individual dose used to limit the range of options considered in the procedure of optimisation. For many types of occupation, it is possible to reach conclusions about the level of individual doses likely to be incurred in well-managed operations. This information can then be used to establish a dose constraint for that type of occupation. In the Commission's view, the class of occupation should be specified in fairly broad terms, such as work in x-ray diagnostic departments, the routine operation of nuclear power plants, or the inspection and maintenance of nuclear power plants. Limits prescribed by regulatory agencies and restrictions applied by managements in specific operations as part of the day-to-day control of exposures are not constraints in the sense used here. In general, these limits and restrictions should be established on the basis of the results of optimisation. More information is given in Section 7.3.1.

(145) It will usually be appropriate for dose constraints to be fixed at the national or local level. When using a dose constraint, a designer should specify the sources to which it is linked so as to avoid confusion with other sources to which the workforce might be concurrently exposed.

(146) The optimisation of protection should, in principle, take account of both actual and potential exposures. However, the techniques for potential exposures are less well developed and the decisions about potential exposures often have no implications for actual exposures. They can then be dealt with separately. (See Section 5.6.)

#### 5.3.2. Dose limits in occupational exposure

(147) Dose limits are needed as part of the control of occupational exposure both to impose a limit on the choice of dose constraints (to cover the occasional case where the same individual is employed on several tasks each with its own constraint) and to provide a protection against errors of judgement in the application of optimisation. In practice, occupational dose limits are applied to all occupational exposure as defined in Section 5.1.1, including that resulting from minor mishaps and misjudgements in operations and

from maintenance and decommissioning in circumstances not necessarily envisaged by the designers. This is an extension of the Commission's previous concept of dose limits and represents a significant increase in the stringency of the Commission's recommendations, regardless of any change in the magnitude of the limits.

(148) The basis of choosing a limit on the risks to which an individual may be subjected has always been difficult to specify. In its 1977 recommendations for dose limits applied to occupational exposure, the Commission attempted to use a comparison with the rates of accidental death in industries not associated with radiation. These comparisons are not altogether satisfactory for a number of reasons. For example, standards of industrial safety are neither constant nor uniform world-wide; the mortality data relate to averages over whole industries, whereas dose limits apply to individuals; the quantitative comparisons were limited to mortality data although the inclusion of non-fatal conditions on both sides of the comparison would have led to less restrictive dose limits; and, finally, there are few grounds for believing that society expects the same standard of safety across a wide range of industries.

(149) The Commission has now adopted a more comprehensive approach. The aim is to establish, for a defined set of practices, a level of dose above which the consequences for the individual would be widely regarded as unacceptable. For this purpose, the limiting dose can be expressed as a lifetime dose received uniformly over the working life, or as an annual dose received every year of work, without prejudice to the way in which the dose limit is finally specified. In the past, the Commission has used the attributable probability of either death or severe hereditary conditions as the basis for judging the consequences of an exposure. This quantity is still a major factor, but is no longer regarded by the Commission as sufficient to describe the detriment. Other factors have been considered in the definition of detriment (see Section 3.3). They include the length of life lost due to an attributable death and the incidence of non-fatal conditions.

(150) In principle, a single index representing the detriment, as now defined, could be used to quantify the consequences of an exposure, but it is extremely difficult to judge the implications of a stated detriment expressed as a single aggregated index, and thus to judge its tolerability. The Commission has found it useful to use three words to indicate the degree of tolerability of an exposure (or risk). They are necessarily subjective in character and must be interpreted in relation to the type and source of the exposure under consideration. The first word is "unacceptable", which is used to indicate that the exposure would, in the Commission's view, not be acceptable on any reasonable basis in the normal operation of any practice of which the use was a matter of choice. Such exposures might have to be accepted in abnormal situations, such as those during accidents. Exposures that are not unacceptable are then subdivided into those that are "tolerable", meaning that they are not welcome but can reasonably be tolerated, and "acceptable", meaning that they can be accepted without further improvement i.e. when the protection has been optimised. In this framework, a dose limit represents a selected boundary in the region between "unacceptable" and "tolerable" for the situation to which the dose limit is to apply, i.e. for the control of practices. Levels of exposure that are regarded as unacceptable in this context may still be tolerable in other contexts; if, for example, they can be reduced only by abandoning a desirable practice e.g., space missions.

(151) In order to provide a quantitative basis for the choice of a dose limit, the Commission has taken account of a range of quantifiable factors in its approach to detriment. For none of them is it possible to establish a categorical criterion against

which to define unacceptable and tolerable, but, taken together, they provide a basis for judgement. Data on the factors considered are given in Annexes B and C.

(152) The Commission has considered these quantifiable factors by selecting several possible values of dose that might be adopted as a dose limit. These test values have been expressed as annual doses received each year over a working lifetime of 47 years. The total dose accumulated has also been considered. The relationship between annual and accumulated dose is valid for external sources of exposure and for short-lived internal sources. If the radionuclides in the body are long-lived and have long biological retention times, the dose is spread out in time and may not all be delivered during the lifetime of the individual. The following assessment then somewhat overestimates the consequences of internal exposures expressed in terms of the 50-year committed equivalent dose.

(153) The consequences of the continued uniform exposure to each of the test values in turn are evaluated. A view is then reached as to which value gives rise to a combination of consequences that is judged to be just short of unacceptable, i.e. just tolerable. This value is then selected as the dose limit. This approach is inevitably subjective, but it makes it possible to consider a wide range of inter-related factors, more properly called attributes. The attributes associated with mortality are as follows:

The lifetime attributable probability of death.

The time lost if the attributable death occurs.

The reduction of life expectancy (a combination of the first two attributes).

The annual distribution of the attributable probability of death.

The increase in the age specific mortality rate, i.e. in the probability of dying in a year at any age, conditional on reaching that age.

(154) These attributes relate to mortality. The Commission has decided to allow for morbidity due to non-fatal cancer and hereditary disorders by using the number of non-fatal conditions weighted for severity as discussed in Section 3.5, and for the period of life lost or impaired. For non-fatal cancers, this weighted number amounts to about 20% of the detriment due to fatalities. The weighted figure for hereditary conditions is very uncertain, but is estimated at about 20% of the number of fatalities for workers (about 27% for the whole population). These contributions are included separately in the following comparisons. They are also summed to give an indication of the aggregated detriment.

(155) The test values of annual effective dose selected for review as a possible basis for the dose limit are 10 mSv, 20 mSv, 30 mSv, and 50 mSv, corresponding approximately to lifetime doses of 0.5 Sv, 1.0 Sv, 1.4 Sv, and 2.4 Sv, given that the annual doses are received every working year. It is implicit in this approach that it is not appropriate to make a decision on the basis of a single attribute. Combinations of attributes should be considered and a judgement should be made on the basis of the whole structure. Annex C provides the necessary age specific calculations. The results are adequately representative of the wider range of populations mentioned in Annex B. The attributes for the test values of annual effective dose are shown in Table 5.

(156) The first combination reviewed is that of the probability of an attributable fatal cancer and the average period of life lost if the attributable fatality occurs. For an annual dose, received every working year, this combination can be expressed as a lifetime probability of losing, on average, a stated period of time. This period is almost independent of the annual dose, since, at low doses, it depends only on the time of the

Table 5. Attributes of detriment due to exposure of the working population<sup>1</sup>

Annual effective dose (mSv)	10	20	30	50	50 (1977 data)
Approximate lifetime dose (Sv)	0.5	1.0	1.4	2.4	2.4
Probability of attributable death (%)	1.8	3.6	5.3	8.6	2.9
Weighted contribution from non-fatal cancer (%) <sup>2</sup>	0.4	0.7	1.1	1.7	—
Weighted contribution from hereditary effects (%) <sup>2</sup>	0.4	0.7	1.1	1.7	1.2
Aggregated detriment (%) <sup>3</sup>	2.5	5	7.5	12	—
Time lost due to an attributable death given that it occurs (y)	13	13	13	13	10-15
Mean loss of life expectancy at age 18 years (y)	0.2	0.5	0.7	1.1	0.3-0.5

<sup>1</sup> The values are all derived from Annex C (see paragraph 155); in Annex B, which deals with a wider range of populations, a somewhat higher estimate is given for the time lost due to an attributable death.

<sup>2</sup> Weighted for severity and loss of lifetime.

<sup>3</sup> The sum of the probability of attributable fatal cancer or equivalent detriment (rounded).

attributable death, not on its probability. For the combination of an additive risk projection model for leukaemia and a multiplicative model for other cancers, the loss is slightly less than 13 years. For the additive model, the loss is slightly less than 20 years. Another attribute, itself an aggregation of these data, is the mean loss of life expectancy at age 18 years as a result of subsequent occupational exposure.

(157) In Table 5, results derived from the data available in 1977 for an annual dose of 50 mSv over 40 years are included for comparison. It should be recognised that these numbers were not used as the basis for selection of the dose limit at that time. As indicated in paragraph 148 the selection of the 1977 limit was made on an entirely different basis (comparing the average fatal cancer risk in radiation work with the fatality risk in "safe" non-radiation occupations and assuming a ratio of 10:1 between the maximum and the average risk). Since the Commission no longer considers that method satisfactory, the 1977 results in the table give little guidance for the present choice of dose limit and have not been used for that purpose.

(158) The way in which the annual probability of attributable death varies with time is also of interest and is shown in Figure 2. The combined effect of latency and the extended period of exposure is to produce a distribution sharply peaked in time at older ages for both the additive risk projection model and the multiplicative risk projection model. The curves are for women, but those for men are very similar. The age of maximum (unconditional) annual probability of attributable death following the exposure of a population of equal numbers of men and women over a whole working lifetime occurs at 68 years for the additive model and 78 years for the multiplicative model. This age is almost independent of the annual dose. The term "unconditional" is used to indicate that the probability is not conditional on reaching the age for which the probability is quoted. The conditional probability continues to rise indefinitely.

(159) The changes in the age-specific mortality rate (roughly the probability of dying within a year conditional on reaching the beginning of that year) are best shown graphically. The data are presented in Annex C (Figure C-9). Even for a continued annual dose of 50 mSv, the changes in mortality rate are small compared with the differences in mortality rate between men and women.

(160) Before any attempt is made to choose a dose limit from this quantitative material, it is necessary to remember that the Commission's aim at this stage is to reach a judgement about a level of dose that would reasonably be regarded as being only just short of unacceptable in the control of practices. The levels of dose actually achieved are

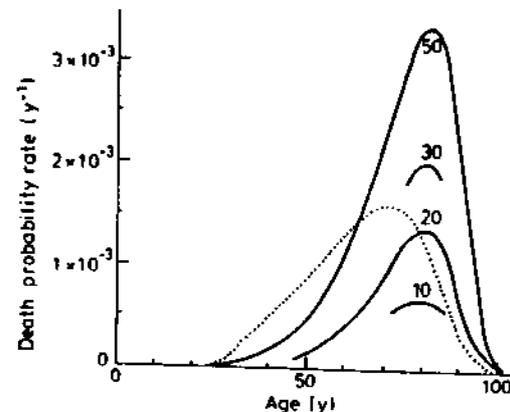


Fig. 2. The unconditional death probability rate (the attributable death age probability density normalised for lifetime risk) for exposure from age 18 to age 65 y. The curves are for females and for present risk estimates. . . . . Additive risk projection model (50 mSv y<sup>-1</sup>) — Multiplicative risk projection model (showing various annual doses in mSv)

not relevant for the purpose of this assessment. The data are expressed in terms of an annual dose over a full working lifetime of 47 years. The form in which the dose limits are best expressed for practical application is discussed later in this section.

(161) The first conclusion drawn by the Commission is that there is no need to extend the range of test doses to be considered in the choice of a dose limit for occupational exposure. The second is that the results indicate that a regular annual dose of 50 mSv, corresponding to a lifetime effective dose of 2.4 Sv, is probably too high, and would be regarded by many as being clearly so. In particular, the reduction of life expectancy at this level (1.1 years) and the fact that there would be a probability exceeding 8% that the radiation hazards in a worker's occupation would be the cause of his death, albeit at a late age, would be widely seen as excessive for a group of occupations many of which are of recent origin and should therefore be setting an example.

(162) On the basis of the data presented above, the Commission has reached the judgement that its dose limit should be set in such a way and at such a level that the total effective dose received in a full working life would be prevented from exceeding about 1 Sv received moderately uniformly year by year and that the application of its system of radiological protection should be such that this figure would only rarely be approached. The final choice of limits and the way in which they should be expressed are influenced by the way in which the limits will be applied in practice. The need to ensure that the limits provide protection against deterministic effects also has to be taken into account.

(163) At the levels of dose incurred in normal situations, excluding doses to the patient in radiotherapy, the control of stochastic effects could be based on the dose accumulated over periods of many years. However, such long control periods can be misused by allowing a rapid accumulation of doses and intakes near the start of a control period in the expectation, not always realised, of smaller doses later in the period. Flexibility of this kind also weakens the emphasis on achieving the control of exposures by design, transferring the emphasis to operational controls.

(164) In recent years, the Commission has recommended a rigid control period of one year: i.e., it has recommended that the effective dose from sources of radiation external to the body and committed by intakes of radioactive substances into the body should be controlled over each year, with no credit taken for any earlier years of low effective dose or intake. This system is very inflexible, and alternatives have been considered.

(165) It has sometimes been suggested that the dose limits for occupational exposure might include a limit on the lifetime effective dose. The Commission sees difficulties in the practical application of lifetime limits. One of these relates to the interpretation of the limit for a worker who is employed in work involving significant occupational exposure for only part of his working life. Decisions have also to be taken about the long-term future employment of workers who exceed the lifetime limit. Short-term limits would also be needed because the Commission's risk estimates are derived for doses distributed fairly uniformly over the occupational age range. Because of these difficulties and the points made in paragraph 163, the Commission does not recommend the use of lifetime limits.

(166) It has also been suggested that flexibility might be provided by setting the limit in the form of the total dose accumulated over a period of a few years, while retaining an annual limit higher than the annual average over the longer period. This would pose some practical problems of the same type as those arising from a lifetime limit, but they would be much less severe. The Commission believes that a period of five years would adequately limit the severity of these difficulties, and would also provide sufficient flexibility. For workers on short-term contracts, the regulatory agency might consider an averaging period not exceeding the period of the contract of employment. The Commission recommends a limit on effective dose of 20 mSv per year, averaged over 5 years (100 mSv in 5 years), with the further provision that the effective dose should not exceed 50 mSv in any single year. The 5-year period would have to be defined by the regulatory agency, e.g. as discrete 5-year calendar periods. The Commission would not expect the period to be introduced and then applied retrospectively. It is implicit in these recommended dose limits that the dose constraint for optimisation should not exceed 20 mSv in a year.

(167) However the control period is defined, the Commission recommends that, following a control period in which the exposure of the individual has exceeded a dose limit, there need be no special restriction applied to the exposure of an individual. Such events should call for a thorough examination, usually by the regulatory agency, of the design and operational aspects of protection in the installation concerned, rather than for restrictions or penalties applied to the exposed individual. If the dose is unknown, or is thought to be high, referral to a physician should be considered.

(168) The recommended limits should apply to all forms of occupational exposure as defined in Section 5.1.1, unless special provisions have been made by the regulatory agency. Because of the difficulties of responding rapidly to an increase in stringency in operations on plant and equipment already in existence, the Commission recognises that regulatory agencies may wish to make temporary use of higher dose limits. Such arrangements should be regarded as transient.

(169) The dose limit forms only a part of the system of protection aimed at achieving levels of dose that are as low as reasonably achievable, economic and social factors being taken into account. It is not to be seen as a target. It represents, in the Commission's view, the point at which regular, extended, deliberate, occupational exposure can reasonably be regarded as only just tolerable.

(170) The Commission's multi-attribute approach to the selection of dose limits

necessarily includes social judgements applied to the many attributes of risk. These judgements would not necessarily be the same in all contexts and, in particular, might be different in different societies. It is for this reason that the Commission intends its guidance to be sufficiently flexible to allow for national or regional variations. In the Commission's view, however, any such variations in the protection of the most highly exposed individuals are best introduced by the use of source-related dose constraints selected by the regulatory agencies and applied in the process of the optimisation of protection rather than by the use of different dose limits.

(171) The restrictions on effective dose, even assuming that the values are at the limit for long periods, are sufficient to ensure the avoidance of deterministic effects in almost all body tissues and organs. However, there are two tissues which will not necessarily be adequately protected by a limit on effective dose, mainly in the case of external exposure. These are the lens of the eye, which makes no contribution to the effective dose, and the skin, which may well be subject to localised exposures. Separate dose limits are needed for these tissues. Internal exposures are dealt with in paragraphs 174 and 175 below.

(172) The previously recommended annual dose limit for the lens of the eye was 150 mSv. The estimated threshold of annual equivalent dose for visual impairment (cataract) was given in *Publication 41* (1984) as " $>0.15$  Sv" and is confirmed in Annex B. The Commission continues to recommend an annual equivalent-dose limit for the lens of the eye of 150 mSv. For external exposure to penetrating radiation over any substantial part of the whole body, the effective-dose limit will be more restrictive.

(173) For the skin, the situation is more complicated. For stochastic effects, the equivalent dose can be averaged over the whole area of the skin. The stochastic effects are expected to arise in the basal layer at a nominal depth of  $7 \text{ mg cm}^{-2}$  (range 2–10  $\text{mg cm}^{-2}$ ). Some deterministic effects also arise at the same depth, others arise in the deeper layers of the dermis (30–50  $\text{mg cm}^{-2}$ ). The limitation on the effective dose provides sufficient protection for the skin against stochastic effects. An additional limit is needed for localised exposures in order to prevent deterministic effects. The recommended annual limit is 500 mSv averaged over any  $1 \text{ cm}^2$ , regardless of the area exposed. The nominal depth is  $7 \text{ mg cm}^{-2}$ . In practice, monitoring is carried out at representative locations for external exposure and over larger areas for contamination. The guidance given in *Publication 35* (1982) on averaging areas is still valid. This limit, applied to the skin of the face, will also provide protection for the lens of the eye against localised exposures to radiation of low penetrating power such as beta particles. The same limit can be applied to all the tissues in the hands and feet.

(174) For internal exposure, annual limits on intake (ALIs) are provided by the Commission as *Publication 61* (1991) and will be based on a committed effective dose of 20 mSv. As indicated in Annex B (paragraph B52) this approach will take adequate account of any non-uniform distributions of dose within organs such as those due to hot particles. The estimated intakes may be averaged over a period of 5 years to provide some flexibility. Revised occupational limits for radon are now under review. Meanwhile the existing recommendations (*Publication 47* (1986)) remain valid.

(175) The restriction of intakes (averaged over 5 years) to the annual limit on intake will, in practice, ensure that the lifetime equivalent dose (not committed equivalent dose) in any single organ will not be such as to result in deterministic effects.

### 5.3.3. *The occupational exposure of women*

(176) The basis for the control of the occupational exposure of women who are not

pregnant is the same as that for men. However, if a woman is, or may be, pregnant, additional controls have to be considered to protect the unborn child. Several factors complicate this matter. The conceptus is at times more prone than the post-natal individual to deterministic injuries caused by radiation and may be more sensitive to the induction of later malignancies. It now seems clear that deterministic effects in the live-born child, including significant mental retardation, will not occur if the exposure of the mother does not exceed the dose limits now recommended for occupational exposure, regardless of the distribution of the exposures in time. Accidental higher exposures of the mother may be more damaging to the conceptus than to the mother.

(177) It is the Commission's policy that the methods of protection at work for women who may be pregnant should provide a standard of protection for any conceptus broadly comparable with that provided for members of the general public. The Commission considers that its policy will be adequately applied if the mother is exposed, prior to a declaration of pregnancy, under the system of protection recommended by the Commission, including the recommended dose limits for occupational exposure. On this basis the Commission recommends no special occupational dose limit for women in general.

(178) Once pregnancy has been declared, the conceptus should be protected by applying a supplementary equivalent-dose limit to the surface of the woman's abdomen (lower trunk) of 2 mSv for the remainder of the pregnancy and by limiting intakes of radionuclides to about 1/20 of the ALI. The Commission wishes to emphasise that the use of its system of protection, particularly the use of source-related dose constraints, will usually provide an adequate guarantee of compliance with this limit without the need for specific restrictions on the employment of pregnant women. The principal criterion will then be that the employment should be of a type that does not carry a significant probability of high accidental doses and intakes. Identification of such situations should be determined by regulatory agencies.

## 5.4. The System of Protection in Medical Exposure

### 5.4.1. The justification of a practice in medical exposure

(179) The justification of a practice leading to medical exposures should be dealt with in the same way as the justification of any other practice. Most of the benefits and detriment accrue to the individuals undergoing diagnosis or treatment, but account should be taken of all the resulting exposures, including the occupational and public exposures, and of any potential exposures. In the first instance, the practice should be defined in broad terms. However, each procedure, either diagnostic or therapeutic, is subject to a separate decision, so that there is an opportunity to apply a further, case-by-case, justification for each procedure. This will not be necessary for simple diagnostic procedures based on common indications, but may be important for complex investigations and for therapy. Guidance is given in *Publications 34* (1982), *44* (1985), and *52* (1987).

### 5.4.2. The optimisation of protection in medical exposure

(180) Because most procedures causing medical exposures are clearly justified and because the procedures are usually for the direct benefit of the exposed individual, less attention has been given to the optimisation of protection in medical exposure than in most other applications of radiation sources. As a result, there is considerable scope for

dose reductions in diagnostic radiology. Simple, low cost, measures are available for reducing doses without loss of diagnostic information, but the extent to which these measures are used varies widely. Doses from similar investigations cover ranges of as much as two orders of magnitude. Consideration should be given to the use of dose constraints, or investigation levels, selected by the appropriate professional or regulatory agency, for application in some common diagnostic procedures. They should be applied with flexibility to allow higher doses where indicated by sound clinical judgement.

(181) Constraints should also be considered in the optimisation of protection when the procedures are not intended to be of direct value to the exposed individual, as in scientific and clinical studies involving the exposure of volunteers.

### 5.4.3. Dose limits in medical exposure

(182) Medical exposures are usually intended to provide a direct benefit to the exposed individual. If the practice is justified and the protection optimised, the dose in the patient will be as low as is compatible with the medical purposes. Any further application of limits might be to the patient's detriment. The Commission therefore recommends that dose limits should not be applied to medical exposures. The question of dose constraints is discussed in Section 5.4.2.

(183) For reasons similar to those given in the previous paragraph, it is not appropriate to include the doses incurred by patients in the course of diagnostic examinations or therapy when considering compliance with dose limits applied to occupational or public exposures. Furthermore, each increment of dose resulting from occupational or public exposure results in an increment of detriment that is, to a large extent, unaffected by the medical doses.

### 5.4.4. Medical exposure of pregnant women

(184) As discussed in Section 3.4.4, exposure of the embryo in the first three weeks following conception is not likely to result in deterministic or stochastic effects in the liveborn child. A pregnant patient is likely to know, or at least suspect, that she is pregnant after one missed menstruation, so the necessary information on possible pregnancy can, and should, be obtained from the patient herself. If the most recent expected menstruation has been missed, and there is no other relevant information, the woman should be assumed to be pregnant. Diagnostic and therapeutic procedures causing exposures of the abdomen of women likely to be pregnant should be avoided unless there are strong clinical indications.

## 5.5. The System of Protection in Public Exposure

(185) The control of public exposure in all normal situations is exercised by the application of controls at the source and the controls applied in one year may lead to continuing exposures or intakes in succeeding years, for example when long lived radionuclides are to be released to the natural environment. As an alternative to the use of long-term equilibrium environmental models linking regular releases to the eventual level of individual and collective doses, the concept of dose commitment is useful. Future individual doses, more strictly the doses to typical members of a critical group, can be limited by the use of the dose commitment. If a limit is set to the effective dose commitment to a critical group from each year of practice that continues at a constant annual level, the average annual individual effective dose will never exceed that limit. If a

truncation time is used in defining the commitment, the guarantee will hold only up to the time of truncation. The collective effective dose per unit of practice can be used in the justification of a practice and in the optimisation of protection. It should be noted that part of the collective dose may be received in the distant future. If that fact is considered to be of significance in judging the importance of the detriment, the full collective dose commitment should be replaced by the collective effective dose delivered in defined periods of time.

#### 5.5.1. *The optimisation of protection in public exposure*

(186) In practice, almost all public exposure is controlled by the procedures of constrained optimisation and the use of prescriptive limits. It is often convenient to class together individuals who form a homogeneous group with respect to their exposures to a single source. When such a group is typical of those most highly exposed by that source, it is known as a critical group. The dose constraint should be applied to the mean dose in the critical group from the source for which the protection is being optimised. Occasionally, the same group will also be critical for other sources, or, if the critical groups are different, each group may incur some dose from the sources for which it is not critical. If the exposures in any critical group are likely to approach the dose limit for public exposure (see Section 5.5.2), the constraints applied to each source must be selected to allow for any significant contribution from other sources to the exposure of the critical group.

(187) The main aim of constrained optimisation in public exposure should be to develop practical restrictions on the sources of exposure, e.g. in the form of restrictions on the release of radioactive waste to the environment.

#### 5.5.2. *Dose limits in public exposure*

(188) With the widespread use of source-related dose constraints and practical restrictions on the sources of public exposure, generally applicable dose limits are rarely limiting in practice. However, because the constraints are source related they might, at least in principle, fail to take adequate account of the exposures from other sources. Although the Commission does not believe that this occurs to a significant extent, it continues to recommend dose limits for public exposure, if only to provide a limit on the choice of constraints.

(189) The Commission defines the scope of its dose limits for public exposure by confining it to the doses incurred as the result of practices. Doses incurred in situations where the only available protective action takes the form of intervention are excluded from the scope of the dose limits. Separate attention has to be paid to potential exposures. (See Section 5.6.) The intended emission of radionuclides from installations, including the emission of naturally occurring radionuclides from installations such as mines and waste disposal sites, should be treated as practices. The resulting doses should be subject to the dose limits. Radon in dwellings and in the open air and radioactive materials, natural or artificial, already in the environment, are examples of situations that can be influenced only by intervention. Doses from these sources are therefore outside the scope of the dose limits for public exposure. Other exposures to natural sources are also outside this scope. Radon in both existing and new dwellings is dealt with in Section 6.2.1. The conduct of intervention involves occupational exposure and should be treated accordingly.

(190) At least two approaches are possible in choosing a dose limit for public

exposure. The first is the same as that used for choosing occupational limits. Assessing the consequences is no more difficult than in the occupational case, but judging the point at which these consequences can reasonably be described as unacceptable is much more difficult. The second approach is to base the judgement on the variations in the existing level of dose from natural sources. This natural background may not be harmless, but it makes only a small contribution to the health detriment which society experiences. It may not be welcome, but the variations from place to place (excluding the large variations in the dose from radon in dwellings) can hardly be called unacceptable.

(191) The consequences of continued additional exposure giving annual effective doses in the range from 1 mSv to 5 mSv are presented in Annex C. They provide no easy basis for a judgement, but do suggest a value of the annual dose limit not much above 1 mSv. On the other hand, the data in Figure C-6 of Annex C show that, even at a continued exposure of 5 mSv  $y^{-1}$ , the change in the age specific mortality rate is very small. Excluding the very variable exposures to radon, the annual effective dose from natural sources is about 1 mSv, with values at high altitudes above sea level and in some geological areas of at least twice this. On the basis of all these considerations, the Commission recommends an annual limit on effective dose of 1 mSv. Averaging over time is discussed in the next paragraph.

(192) In deriving restrictions on sources of public exposure, some allowance is made for variations in the environmental pathways to man, but there will always be the possibility of larger transient changes. There will also be variations in the effectiveness of control procedures applied at the source and the Commission recommends that the transient increases in dose resulting from such variations should be included in the doses subject to the dose limits. Doses due to major accidents are not subject to the dose limits because they can be dealt with only by intervention. Since the detriment is a function of the accumulation of dose over many years, it would be unduly restrictive to require the controls to be related rigidly to annual dose limits. Some flexibility in the limits is desirable. The Commission's previous recommendations provided for a principal limit on the annual effective dose, with a subsidiary limit on the effective dose in some years, provided that the average effective dose over a lifetime did not exceed the principal limit. This recommendation is still sound in principle, but the Commission has concluded that the very long averaging period in the subsidiary limit gives excessive flexibility. It now recommends that the limit for public exposure should be expressed as an effective dose of 1 mSv in a year. However, in special circumstances, a higher value of effective dose could be allowed in a single year, provided that the average over 5 years does not exceed 1 mSv per year. Because this represents only a slight change from the previous recommendation, the Commission recommends that the 5-year period should be applied retrospectively when the new recommendation is being implemented. For this purpose, values of effective dose may be added to earlier values of effective dose equivalent. It is implicit in this limit that the constraints for the optimisation of protection in the design of new installations should be smaller than 1 mSv in a year.

(193) In selecting the limit on effective dose, the Commission has sought a value that would be only just short of unacceptable for continued exposure as the result of deliberate practices the use of which is a matter of choice. This does not imply that higher doses from other sources, such as radon in dwellings, should be regarded as unacceptable. The existence of these sources may be undesirable, but it is not a matter of choice. The doses can be controlled only by intervention, which will also have undesirable features.

(194) Limits are also needed for the lens of the eye and localised areas of skin since these tissues will not necessarily be protected against deterministic effects by the limit on effective dose. Because the total period of exposure may be nearly twice as long as for occupational exposure, and because the exposed individuals may show a wider range of sensitivity than the more limited population of workers, the recommended annual limits (non-occupational) for the equivalent dose in these tissues are lower than those for workers. The Commission has adopted an arbitrary reduction factor of 10, leading to annual limits of 15 mSv for the lens and 50 mSv averaged over any 1 cm<sup>2</sup> area of skin, regardless of the area exposed. The recommended limits are summarised in Table 6.

Table 6. Recommended dose limits<sup>1</sup>

Application	Dose limit	
	Occupational	Public
Effective dose	20 mSv per year, averaged over defined periods of 5 years <sup>2</sup>	1 mSv in a year <sup>3</sup>
Annual equivalent dose in the lens of the eye	150 mSv	15 mSv
the skin <sup>4</sup>	500 mSv	50 mSv
the hands and feet	500 mSv	—

<sup>1</sup> The limits apply to the sum of the relevant doses from external exposure in the specified period and the 50-year committed dose (to age 70 years for children) from intakes in the same period (see paragraph 143).

<sup>2</sup> With the further provision that the effective dose should not exceed 50 mSv in any single year. Additional restrictions apply to the occupational exposure of pregnant women, which is discussed in Section 5.3.3.

<sup>3</sup> In special circumstances, a higher value of effective dose could be allowed in a single year, provided that the average over 5 years does not exceed 1 mSv per year.

<sup>4</sup> The limitation on the effective dose provides sufficient protection for the skin against stochastic effects. An additional limit is needed for localised exposures in order to prevent deterministic effects (see paragraphs 173 and 194).

### 5.6. Potential Exposures

(195) The initial treatment of potential exposures should form part of the system of protection applied to practices, but it should be recognised that the exposures, if they occur, may lead to intervention. At this stage, there should be two objectives, prevention and mitigation. Prevention is the reduction of the probability of the sequences of events that may cause or increase radiation exposures. It involves maintaining the reliability of all the operating and safety systems and of the associated working procedures. Mitigation is the limitation and reduction of the exposures if any of these sequences do occur. It involves the use of engineered safety features and operational procedures to control each sequence of events with the aim of limiting its consequences, should it occur. The arrangements for mitigation should not be restricted to plans for intervention. A great deal can be accomplished at the stages of design and operation to reduce the consequences of accident sequences so that intervention may not become necessary. It is difficult to compare, and to combine, the benefit of a reduction in probability (prevention) with that of a reduction in dose (mitigation) because a reduction in probability by a factor is not usually seen as equivalent to a reduction in dose by the same factor.

(196) In order to maintain a strict coherence in the treatment of actual and potential exposures, it would be necessary to extend the concept of detriment to include the probability of occurrence of the situation giving rise to the detriment. Techniques for achieving this are still being developed. Meanwhile, emphasis has to be placed on one part of the detriment, the probability of an attributable death. It must also be recognised that the uncertainties in estimating the probability of occurrence will usually be much greater than the uncertainties in estimating the probability of the consequences should the dose occur.

(197) The simplest way of dealing with the potential exposure of individuals is to consider the overall (*a priori*) individual probability of attributable death from cancer, rather than the effective dose, as the quantity to be used in the system of protection. For this purpose, the probability is defined as the product of the probability of incurring the dose and the lifetime conditional probability of attributable death from the dose if it were to have been incurred. A restriction corresponding to a dose limit can then be expressed in the form of a risk limit, i.e. a limit on the fatality probability. (See Section 5.6.3.) If the risk limit is derived from the probability of death attributable to exposure at the relevant dose limit, a corresponding level of protection will also be provided against non-fatal cancer and against deterministic effects.

(198) This use of the overall individual radiation risk is an adequate starting point for use in the system of protection, but it is not sufficient. This is because the situation will change if the event giving rise to the potential exposures actually occurs. At low probabilities of the potential event, an overall individual risk limit might imply doses when the event occurs that would be large enough to call for intervention or might result in deterministic effects. These undesirable outcomes should be borne in mind at the planning stage. They may call for lower risk constraints (analogous to dose constraints) than would be needed for high probability, low dose situations. When assessing the individual risk, it should be remembered that the conditional probability of deleterious effects if a dose is, in fact, incurred may be higher than the nominal probability because the doses and dose rates may be higher than those for which the nominal probability coefficients have been selected and because deterministic effects may become important at these higher doses.

(199) The specification of collective detriment from potential exposures is difficult and controversial, even if the consideration of detriment is limited to attributable deaths. It is not appropriate to depend on the use of the product of the probability of an event and the number of attributable deaths should it occur—the expectation value of the number of deaths—because this conceals the fact that the outcome will be either no consequences if the event does not occur, or the full consequences if it does. It also involves an implicit assumption of reciprocity between reductions in probability and reductions in the scale of consequences: i.e. the assumption that a frequent event with small consequences and a rare event with large consequences are equally detrimental if the expectation values of the consequences are the same.

(200) A more comprehensive approach to the collective detriment from potential exposures is that of multi-attribute analysis. Each characteristic (attribute) of the available options has to be identified and quantified. It is then given a weighting factor judged to represent its importance. The weighted attributes can then be aggregated to provide a figure of merit or compared individually with the weighted attributes in other options. Either method leads to a quantitative, or semi-quantitative, basis for choice between options.

(201) Meanwhile, a simpler approach is possible for both individual and collective

exposures if the doses will be small even if the event occurs. If the doses, should they occur, will not be in excess of dose limits, it is adequate to use the product of the expected dose and its probability of occurrence as if this were a dose that is certain to occur. The conventional procedures of justification and optimisation can then be applied.

### 5.6.1. *Justification of a practice*

(202) If sufficient information is available, the detriment associated with a proposed practice in the assessment of the justification of the practice should include that from the potential exposures. In practice, it may well be that the estimation of the detriment from potential exposures will be improved by operating experience obtained after the introduction of the practice. This will require a re-evaluation of the justification of the practice.

### 5.6.2. *The optimisation of protection*

(203) If the options for applying the system of protection to potential exposures do not alter the other exposures resulting from the practice, the potential detriment can be used in the procedures of optimisation without further complications. Sometimes, however, the two sets of exposure are interdependent and the optimisation of protection must then be carried out for both types of exposure together. (See Section 5.7.) In either case, the procedure must be constrained by an individual risk limit or, more probably, by source-related and sequence-related individual risk constraints.

### 5.6.3. *Individual risk limits and constraints*

(204) Although a risk limit can be defined by analogy with the dose limit, it will have a very different character. The probability of events leading to potential exposures cannot be determined by observation. They are the result of some form of probabilistic safety assessment. These assessments commonly provide estimates of the probability of defined accident sequences.

(205) The total probability from all possible sequences can be obtained only from a further stage of forecasting. It is therefore more useful to define a series of risk constraints applicable to the attributable probability of death, defined as the product of the probability of receiving a dose as the result of a precisely defined sequence and the lifetime conditional probability of attributable death from the dose if it were to have been received. Taken alone, these constraints will not be adequate because an individual will be at risk from more than one sequence. Unless there is one dominating sequence, there will also be a need for a risk limit, despite the difficulty of assessing the total risk to which the limit should apply. The Commission does not yet recommend an annual risk limit for individuals.

(206) There is also the possibility of potential doses in medical exposures. Errors in dosimetry and equipment failures have given rise to injurious, and sometimes fatal, doses to patients. The Commission does not recommend any specific value for risk constraints in this context.

## 5.7. *Interactive Situations*

(207) The bulk of the individual and collective doses often results from a single type of exposure. However, there are some cases where there is a significant contribution from several types of exposure.

(208) The first example is that of an interaction between public and occupational exposure. If the public exposure is due to the release of waste to the environment, a reduction in that exposure may result in increased occupational exposure due to the additional waste processing and storage. The simplest approach to the optimisation of protection is then to use the combined collective effective dose from the two forms of exposure. However, it has sometimes been considered that the detriment due to public exposure should be treated differently from that due to occupational exposure. This is not a view to which the Commission subscribes. The Commission recommends that the sum of the effective doses from each type of exposure from a given source should be used in the optimisation procedures. If the two components were thought to have different weightings, they could be used separately in a multi-attribute analysis.

(209) The second example is the interaction between potential exposure and occupational or public exposure. The mechanical inspection of plant may reduce the probability of failures but only at the expense of additional occupational exposure, and the reduction of public exposure by the increased storage of waste may cause increased potential occupational and public exposures. This form of interaction can be dealt with only by the methods of multi-attribute analysis.

## 6. THE SYSTEM OF PROTECTION IN INTERVENTION

Chapter 6 deals with situations where the sources of exposure and the exposure pathways are already present and the only type of action available is intervention. The chapter deals mainly with intervention applying to public exposure, including intervention following accidents, but includes some material on occupational exposure in emergencies. The practical application of these recommendations for intervention are discussed in Chapter 7.

(210) Before a programme of intervention is initiated, it should be demonstrated that the proposed intervention will be justified, i.e. do more good than harm, and that the form, scale, and duration of the intervention have been chosen so as to optimise the protection. As explained in Section 4.4 the Commission recommends against the use of dose limits for deciding on the need for, or scope of, intervention.

### 6.1. *The Basis of Intervention in Public Exposure*

(211) In judging the benefits and detriments of intervention aimed at reducing public exposure, the comparison should, in the first place, be made for those at risk, but there will also be an impact on the rest of society and the judgements will have to be wide enough to cover these impacts too.

(212) As indicated in Section 4.4, the processes of justification and optimisation both apply to the protective action, so it is necessary to consider them together when reaching a decision. Justification is the process of deciding that the disadvantages of each component of intervention, i.e. of each protective action, are more than offset by the reductions in the dose likely to be achieved. Optimisation is the process of deciding on the method, scale and duration of the action so as to obtain the maximum net benefit. The duration of countermeasures influences the averted dose and therefore the provisional

decision about the withdrawal of the countermeasures should be taken as part of the process of optimisation. In simple terms, the difference between the disadvantages and the benefits, expressed in the same terms, e.g. costs, including social costs with an allowance for anxiety, should be positive for each protective action adopted and should be maximised by settling the details of that action.

(213) The cost of intervention is not just the monetary cost. Some protective or remedial actions may involve non-radiological risks or serious social impacts. For example, the short-term removal of people from their homes is not very expensive; but it may cause the temporary separation of members of a family and result in considerable anxiety. Prolonged evacuation and permanent relocation are expensive and have sometimes been found to be highly traumatic.

(214) It follows from the above paragraphs that it is not possible to define quantitative intervention levels for rigid application in all circumstances. Nevertheless, because some kinds of action may be needed urgently, it is useful to have guidance prepared in advance for use following accidents and emergencies.

## 6.2. Situations in which Remedial Action may be Needed

(215) Many situations in which intervention is being considered are of long standing and do not call for urgent action. Others, resulting from accidents, may cause serious exposures unless immediate action can be taken. They may also cause long-term problems. The long-standing situations are dealt with in this section and the immediate problems of accidents in Section 6.3.

### 6.2.1. Radon in dwellings

(216) Radon in dwellings needs special attention because both the individual and the collective doses from radon are higher than those from almost any other source. In many countries, there are some individual doses substantially higher than those that would be permitted in occupational exposure. If improvements are needed, they have to be achieved by intervention involving modifications to the dwellings or to the behaviour of the occupants.

(217) In *Publication 39* (1984), the Commission recommended the use of action levels to help in deciding when to require or advise remedial action in existing dwellings. The choice of an action level is complex, depending not only on the level of exposure, but also on the likely scale of action, which has economic implications for the community and for individuals. For owner-occupied dwellings, general guidance may be adequate, leaving the final decision to be made by the occupier, on behalf of all the occupants, but in countries with substantial numbers of rented dwellings, it may be desirable to establish firm national action levels, at least for rented properties. In such cases, the best choice of an action level may well be that level which defines a significant, but not unmanageable, number of houses in need of remedial work. It is then not to be expected that the same action level will be appropriate in all countries.

(218) The problem of new dwellings has some similarity to that of existing dwellings because the concentration of radon cannot be determined with confidence until the dwelling has been completed and occupied for a year or so. It is then an existing dwelling. It is therefore dealt with here, rather than in Chapter 5. Guides or codes for the construction of new dwellings in selected areas can be established so that it is highly probable that they will result in exposures in these dwellings below some chosen reference level. The choice of this level may cause marked changes in conventional building practices and this

might have unforeseen effects on structures or living conditions. The Commission therefore wishes to proceed cautiously. It has initiated a further review of current experience with a view to issuing revised recommendations in due course. Meanwhile the guidance in *Publication 39* (1984) should still be used.

### 6.2.2. Radioactive residues from previous events

(219) The most common causes of residues are the burial of long-lived materials from early operations such as mining and luminising with radium compounds. The use of mining spoil as a land-fill material, followed by the construction of dwelling houses, has caused substantial problems. Buildings used for radium work have subsequently been put to other purposes, with the radium being discovered only years later. There have been several accidents in which long-lived radioactive materials have been dispersed in residential and agricultural areas. The necessary remedial actions vary greatly in complexity and scale and may themselves give rise to problems of occupational exposure and waste disposal. These should be dealt with in accordance with the Commission's recommendations for practices. The need for and extent of remedial action has to be judged by comparing the benefit of the reductions in dose with the detriment of the remedial work, including that due to the doses incurred in the remedial work. No general solutions are available, but the methods recommended for the optimisation of protection can be used to give guidance in each individual case.

## 6.3. Accidents and Emergencies

### 6.3.1. Intervention affecting the public

(220) The first step in deciding on the intervention likely to be needed after an accident is to define the type of all the likely protective actions and to consider the costs and the expected reductions in individual and collective doses as functions of the scale and duration of each. A substantial amount of preliminary work on economic and environmental models and on accident forecasting is needed for these assessments.

(221) Because the initial introduction of protective actions on any scale, however small, involves significant costs, it may well be that small-scale, short-duration, intervention is costly without being effective. As the scale and duration are increased, the effectiveness initially increases without a marked increase in costs. Eventually, further increases will fail to achieve increased benefits comparable with their costs and the net benefit again begins to fall. There is then a range of values of the possible intervention level of individual dose averted, within which there is an optimum level. If the net benefit at that optimum is positive, intervention of the defined type, scale and duration will be justified. The initial planning for emergencies should include the choice of intervention levels of dose averted, or a limited range of such intervention levels, that are likely to lead to intervention that is justified and reasonably well optimised.

(222) The benefit of a particular protective action within a programme of intervention should be judged on the basis of the reduction in dose achieved or expected by that specific protective action, the dose averted. Thus each protective action has to be considered on its own merits. For example, decisions about the control of individual foodstuffs are independent of decisions about other foodstuffs and of decisions about sheltering or evacuation. In addition, however, the doses that would be incurred via all the relevant pathways of exposure, some subject to protective actions and some not, should be assessed. If the total dose in some individuals is so high as to be unacceptable even in an emergency, the feasibility of additional protective actions influencing the

major contributions to the total dose should be urgently reviewed. Doses causing serious deterministic effects or a high probability of stochastic effects would call for such a review. For this purpose, an intervention level of dose received by all pathways should be chosen at the planning stage.

(223) The Commission has set out the general principles for planning intervention after an accident and included quantitative guidance on intervention levels in *Publication 40* (1984). This guidance was confined to short and medium term action. The Commission plans to issue further guidance covering the whole subject.

### 6.3.2. *The limitation of occupational exposure in emergencies*

(224) Occupational exposures directly due to an accident can be limited only by the design of the plant and its protective features and by the provision of emergency procedures. Ideally, the aim should be to keep the doses within those permitted in normal conditions, but, while this is usually possible, it may not always be so in serious accidents.

(225) In addition to the exposures resulting directly from the accident, there will be exposures of emergency teams during emergency and remedial action. Even in serious accidents, these can be limited by operational controls. The doses incurred are likely to be higher than in normal situations and should be treated separately from any normal doses. Emergencies involving significant exposures of emergency teams are rare, so some relaxation of the controls for normal situations can be permitted in serious accidents without lowering the long-term level of protection. This relaxation should not permit the exposures in the control of the accident and in the immediate and urgent remedial work to give effective doses of more than about 0.5 Sv except for life-saving actions, which can rarely be limited by dosimetric assessments. The equivalent dose to skin should not be allowed to exceed about 5 Sv, again except for life saving. Once the emergency is under control, remedial work should be treated as part of the occupational exposure incurred in a practice.

## 7. IMPLEMENTATION OF THE COMMISSION'S RECOMMENDATIONS

Chapter 7 emphasises the importance of the operational level of radiological protection and shows how this should be developed from the requirements of regulatory agencies and the recommendations of the Commission. It gives advice on the measurement of doses (monitoring) and on possible bases for exemption from regulatory requirements. It deals with both practices and intervention.

(226) This chapter is concerned principally with organisational features that may help in the implementation of the Commission's recommendations. Although the organisational structures will differ from country to country, and the chapter is therefore intended to be illustrative, the Commission hopes that it will provide useful guidance to managements and regulatory agencies.

(227) In the implementation of the Commission's recommendations, the main practical responsibilities fall on the designers and operators of equipment and installations, who obtain their guidance partly from professional advisors and publications such as those of the Commission and international organisations, and partly from regulatory and advisory bodies. Governments should establish a framework of regulatory and advisory

functions aimed at helping the operating managements to meet their responsibilities and at ensuring that a suitable standard of protection is maintained. This framework should also make provision for any necessary central services, including those for intervention, and for links to regional and international organisations in both normal and emergency situations.

(228) The organisational structures used in the control of practices should, as far as possible, also be used to deal with intervention, although they will have to be modified and extended in some respects. This will help to maintain consistency and will avoid too much dependency on lines of demarcation. Planning for intervention in the event of emergencies should be an integral part of normal operating procedures. Any changes in responsibility, e.g. from the usual line of command to an emergency controller, should be planned in advance. The hand-over should be a formal procedure. More details are given in Section 7.7. When there is no operating management, e.g. for radon in dwellings, intervention should become the responsibility of the regulatory agency or of some other clearly defined body.

(229) The Commission's recommendations have been set out as a sequence of concepts, starting with the primary aims and broadening out to cover more detailed aspects. This structure has been followed in this chapter, which shows how the responsibilities of the various bodies are interrelated. To do this it is necessary to establish a logical sequence of stages, as follows:

- Allocation of responsibility
- Basic recommendations of the Commission
- Requirements of regulatory agencies
- Management requirements
- Validation of performance

To a large extent, these stages are the same for all types of exposure. However, when intervention is required, there may not always be a relevant operating management available and the regulatory agency, or some other designated body, will have to accept some of the responsibilities usually carried by the operating management.

### 7.1. Responsibility and Authority

(230) In radiological protection, as in other matters concerning health and safety, it is often convenient to distinguish between responsibility and authority. The first stage of **responsibility** is the duty to establish objectives, to provide the measures needed to achieve those objectives, and to ensure that these measures are properly carried out. This is essentially a prospective concept. Those bearing responsibility should then have the **authority** to commit the resources needed to meet their responsibilities. There is also a retrospective component of responsibility, sometimes called **accountability**, that requires a continuing review of performance to be made so that failures can be identified and steps taken to prevent recurrence. Accountability implies the need to establish a programme of verification to determine how effectively the original objectives are being achieved.

(231) The primary responsibility for achieving and maintaining a satisfactory control of radiation exposures rests squarely on the management bodies of the institutions conducting the operations giving rise to the exposures. When equipment or plant is designed and supplied by other institutions, they, in turn, have a responsibility to see that the items supplied will be satisfactory, if used as intended. Governments have the

responsibility to set up regulatory agencies, which then have the responsibility for providing a regulatory, and often also an advisory, framework to emphasise the responsibilities of the management bodies while, at the same time, setting and enforcing overall standards of protection. They may also have to take direct responsibility when, as with exposures to many natural sources, there is no relevant management body.

(232) In all organisations, the responsibilities and the associated authority are delegated to an extent depending on the complexity of the duties involved. The working of this delegation should be examined regularly. There should be a clear line of accountability running right to the top of each organisation. The delegation of responsibilities does not detract from that accountability. There is also an interaction between the various kinds of organisation. Advisory and regulatory agencies should be held accountable for the advice they give and any requirements they impose. The imposition of requirements expressed in general terms and the acceptance of advice do not reduce the responsibility, or the accountability, of the operating organisations. This is also true of prescriptive requirements expressed in terms of objectives or limits. Prescriptive requirements concerning the conduct of operations do, however, result in some de facto transfer of responsibility and accountability from the operator to the regulator. The use of such requirements can be very effective, especially where the operating management lacks detailed experience, but such use always needs to be carefully justified.

(233) Requirements, operating instructions, regulatory approvals and licences and other administrative devices are not, of themselves, enough to achieve an appropriate standard of radiological protection. Everyone in an undertaking, from the individual workers and their representatives to the senior management, should regard protection and accident prevention as integral parts of their every-day functions. Success and failure in these areas are at least as important as they are in the primary function of the undertaking.

## 7.2. The Recommendations of the Commission

(234) As indicated in Section 1.3, the recommendations of the Commission are intended, *inter alia*, to provide a useful basis from which to derive the necessary regulatory requirements. Subject to any mandatory requirements of the regulatory agencies, the recommendations also provide guidance to the operating managements. The widespread adoption of the recommendations has the advantage of giving a consistency of aims and standards across a wide range of countries. It also helps to provide an appropriate degree of uniformity of procedures. To assist in this process, the Commission has tried to make clear the reasons for its recommendations and has deliberately included some flexibility, so that consistency can be obtained without rigidity.

(235) Widespread acceptance of the quantities discussed in Chapter 2 and of the proposed values of the nominal probability coefficient, the radiation weighting factors,  $w_R$ , and the tissue weighting factors,  $w_T$ , will greatly simplify world-wide comparisons of doses and practices and will help in the development of engineering standards for instrument design and performance.

## 7.3. Regulatory Requirements

(236) The form of regulatory agencies, their requirements, and their methods of operating differ widely. Regulatory provisions are not an alternative to management

requirements: they are better seen as a bridge between the recommendations of the Commission and the management requirements. In some respects they should go further. In particular, a large part of the duty of assessing the justification of a practice should rest on the regulatory agency or on the government upon which it depends. Provisions may be needed to prohibit practices not regarded as being justified. The regulatory provisions should also set a broad and adequate standard of protection for application to the practices that are regarded as justified.

(237) One important national and international need is to provide adequate resources for the education and training of future professional and technical staff in radiological protection. These resources cannot be provided by the regulatory agencies alone.

### 7.3.1. The regulation of practices

(238) One feature of the regulation of practices is the use of source-related constraints to be applied to the optimisation of protection. It will avoid confusion if it is made clear that these regulatory constraints are not the same as prescriptive regulatory limits. Limits prescribed by regulatory agencies and restrictions applied by managements to specific operations as part of the day-to-day control of exposures are not constraints in the sense used here. In general, they should be established on the basis of the results of optimisation. However, some regulatory agencies use prescribed limits as a form of regulatory constraint, requiring the operating management to achieve further reductions based on optimisation. Prescriptive limits may apply not only to dose but also to any features that are under the direct control of the operating management, such as releases to the environment. The purpose of prescriptive limits should be clarified when they are being set. In any event, they should never be regarded as an alternative to the process of optimising protection. It is not satisfactory to set design or operational limits or targets as an arbitrary fraction of the dose limit, regardless of the particular nature of the plant and the operations.

(239) A high proportion of operations can be conducted in such a way that the standard of protection is set by the process of constrained optimisation and not by the dose limits. Mandatory dose constraints, applicable to selected classes of operation, then provide a useful regulatory tool. Alternatively, the regulatory agency might establish investigation levels for classes of operation. Exceeding an investigation level would require an investigation to be made of the optimisation programme of the operator or designer.

(240) Occasionally, an individual is seen to be consistently exposed at a high level, close to the individual dose limit, so that the accumulated effective dose may be approaching an unacceptable level. Special attention should then be given to the justification of the practice and the optimisation of protection. This may lead to the imposition of a special prescriptive limit aimed at forcing an improvement, or to the use of an investigation level requiring a formal review of the procedures for optimising protection.

(241) The regulatory agencies should be particularly concerned with public exposures because of the possibility of individuals being exposed to more than one source. This makes it particularly important to identify lines of responsibility and to establish clearly to which sources the regulatory provisions apply.

(242) The regulatory provisions may be of a general nature, or they may be related directly to one installation or to a class of installations. In each case, the agency will have to consider both the source-related approach, to ensure the proper optimisation of protection, including the selection of source-related dose constraints, and the individual-

related approach to ensure the adequate protection of individuals in relation to all the relevant sources. If the primary source is not under the jurisdiction of the agency, e.g. when radioactive material is released to a river upstream of the agency's area, it may be useful to consider assessments and controls to be related to a particular sector of the environment. Control cannot then be applied at the source, so that doses can be limited, if at all, only by some form of intervention. It will usually be better to achieve control of the source by inter-state, or inter-agency, collaboration.

(243) The objectives, and to some extent the methods, of regulatory agencies may sometimes be subject to formal international or regional requirements. Most of these are advisory, but some are mandatory, at least as far as objectives are concerned. There is also a range of international engineering standards, some of which have a bearing on radiological protection. The responsible international bodies also issue advisory documents. All these documents provide a valuable input to the process of achieving an appropriate level of protection.

### 7.3.2. Regulation in the context of potential exposures

(244) The first step in regulation in the context of potential exposures is that of establishing a duty on the operating management to conduct assessments of the expected frequency and possible consequences of events, such as accidents and major errors of design and operation, that might give rise to doses substantially higher than those in normal conditions. Account should be taken of a wide range of initiating causes, including those outside the operator's control, e.g. floods and storms. The operator should be required to include a review of the procedures necessary to deal with the events, should they occur. These assessments will necessarily be based on identified sequences of events: it will rarely be possible to ensure that all such sequences have been identified. The possible existence of rare unidentified sequences makes it impossible to justify assessments leading to very low values of the overall probability of accidents.

(245) The second stage is that of regulatory review. Depending on the likely scale of the problems posed by the events giving rise to potential exposures, the regulatory agency should establish a procedure for reviewing the operators' assessments. In most cases, this need be no more than the conventional level of testing for compliance with any regulatory requirement. In the few installations where the consequences of an accident might be severe, the procedure may involve a detailed review of the whole assessment, possibly linked to a system of prior approval or licensing. The use of risk constraints related to individual sequences should be considered. These may make it unnecessary to establish overall risk limits, which are difficult to select and even more difficult to enforce.

(246) Compliance with risk limits and constraints has to be judged from the results of assessments of the quality of the design, operation and maintenance of the plant and equipment and the quality of the management arrangements. Relevant features include the performance and reliability of equipment and the quality of test procedures, operating instructions and training.

## 7.4. Management Requirements

(247) The first, and in many ways the most important, of the practical steps in implementing the Commission's recommendations is the establishment of a safety-based attitude in everyone concerned with all the operations from design to decommissioning.

This can only be achieved by a substantial commitment to training and a recognition that safety is a personal responsibility and is of major concern to the top management. Close links between the management and the representatives of the workforce have a major role to play.

(248) This attitude to safety should be reinforced by the creation of a formal management structure for dealing with radiological protection, including the optimisation of protection, and by the issuing of clear operating instructions. These should take account of any requirements applied to the design of the plant and equipment and of the installation as a whole, and should cover subsidiary operations such as inspection and maintenance. The details of the management structure and of the operating instructions will depend on the form and scale of the operating organisation, but their importance should be recognised even in small or informal organisations. From the point of view of the Commission, it is convenient to consider design requirements and operating instructions as parts of a unified system, to be called the management requirements, even though the two parts may be laid down by different components of the management organisation.

(249) The aims of the management requirements should be to set out the practical basis for protecting all concerned. The detailed techniques cover such aspects as the choice of radiation source or radioactive material, the use of shielding and distance to reduce radiation fields, the restriction of the time spent in the proximity of sources, and the use of containment, usually in several stages, to limit the spread of radioactive materials into workplaces and the public environment. Attention should also be given to the layout of plant and equipment. In addition, the techniques for dealing with potential exposures include safety analysis to identify possible causes of accidents and the methods available to reduce their likelihood and severity, followed by the assessment of the reliability of all the principal systems affecting the probability of accidents. These systems include the plant and equipment, any software used in the equipment or in the operations, the operating and maintenance procedures, and the performance of the human operators. Much of the responsibility for these analyses should fall on the designer, but part of it should rest on the operating management. There should be plans for dealing with accidents should they occur. These plans should be subject to periodic review. All these reviews and assessments should lead to the preparation of written management requirements.

(250) The management requirements should be expressed in clear and unambiguous terms and they should be eminently practical. They will stem, in part, from the requirements of regulatory agencies (see Section 7.3), but they should also draw on the recommendations of the Commission, manuals of good practice, and engineering standards. The task of preparing and implementing management requirements is onerous, but it plays an important part in achieving the correct balance between the protection measures and the effective conduct of the operations.

### 7.4.1. The classification of workplaces and working conditions

(251) One of the most important functions of management requirements is that of maintaining control over the sources of exposure and over the workers who are occupationally exposed. It is usually easy to specify the sources of occupational exposure. They are the artificial radioactive materials and the electrical generators of radiation used in the workplace, together with the natural sources specified in Section 5.1.1. The specification has to be applied with common-sense because artificial radionuclides are

present in trace amounts in most materials. The control of sources is helped by requiring that the workplaces containing them be formally designated. The Commission uses two such designations—controlled areas and supervised areas.

(252) A controlled area is one in which normal working conditions, including the possible occurrence of minor mishaps, require the workers to follow well-established procedures and practices aimed specifically at controlling radiation exposures. A supervised area is one in which the working conditions are kept under review but special procedures are not normally needed. The definitions are best based on operational experience and judgement. Account should be taken both of the expected levels of exposure and of the likely variations in these exposures. In areas where there is no problem of contamination by unsealed radioactive materials, designated areas may sometimes be defined in terms of the dose rates at the boundary. The aim should be to ensure that anyone outside the designated areas will not need to be regarded as occupationally exposed. The dose limits recommended by the Commission are intended to apply to all workers, but the use of designated areas should enable the actual doses received outside the designated areas to be kept below the dose limits for public exposure. The dividing line between controlled areas and supervised areas, if the latter are used, has commonly been set with the aim of ensuring that the doses to workers in the supervised areas can confidently be predicted to be less than 3/10 of the occupational dose limits. The Commission now regards this definition as being too arbitrary and recommends that the designation of controlled and supervised areas should be decided either at the design stage or locally by the operating management on the basis of operational experience and judgement. This judgement has to take account of the expected level and the likely variations of the doses and intakes, and the potential for accidents.

(253) In previous recommendations, the Commission has defined two types of working conditions based on the expected level of individual annual dose. This was originally intended to help in the choice of workers to be subject to individual monitoring and special medical surveillance. In recent years, it has become apparent that neither of these decisions is best linked to a crude classification of working conditions based on expected dose and the Commission no longer recommends such a classification. The design of monitoring programmes is discussed in Section 7.5.1 and medical surveillance in Section 7.4.4.

#### 7.4.2. Operational guides

(254) Generalised exhortations to keep risks low are implicit in radiological protection. They should be supplemented by specific statements that the designers and the operators can use as guides. The operating management is responsible for establishing these guides, which should include an indication of the maximum levels of exposure that the management expects to occur in defined operations.

(255) These guides apply to both the designers and operators of plant and equipment, but they are not targets and are not sufficient. They provide only an envelope within which the designers and operators should work. In addition, there should be an obligation to consider the available options and to establish operational procedures based on more completely optimised levels of protection for the specific circumstances. These operational guides are becoming increasingly common and are to be welcomed, provided that they are soundly based. If operational guides are chosen to be the same for widely diverse operations, they are likely to be arbitrary and will not be consistent with the standards of protection recommended by the Commission.

(256) In principle, the operational guides should include material on the standard of

reliability needed to limit potential exposures. In practice, however, it is proving difficult to establish a sound basis for such material, sometimes known as "safety goals". It is therefore necessary to depend heavily on past experience, often codified in the form of engineering standards.

#### 7.4.3. Reference levels

(257) It is often helpful in the management of operations to establish values of measured quantities above which some specified action or decision should be taken. These values are generally called reference levels. They include recording levels, above which a result should be recorded, lower values being ignored; investigation levels, above which the cause or the implications of the result should be examined; and intervention levels, above which some remedial action should be considered. The use of these levels can avoid unnecessary or unproductive work and can help in the effective deployment of resources. If recording levels are used, the fact that no unrecorded results exceeded the recording level should be made clear.

#### 7.4.4. Occupational services for protection and health

(258) One common responsibility of the operating management is to provide access to occupational services dealing with protection and health. These may be in-house services or consultancy services brought in from outside. The protection service should provide specialist advice and arrange any necessary monitoring provisions, both inside and outside the installation. The head of the protection service should have direct access to the senior operating management. Most of this report has already been concerned with the provisions for protection. This section therefore concentrates on the provision of occupational health services.

(259) The principal role of the occupational health service is the same as it is in any occupation. Physicians supervising the health of a force of radiation workers need to be familiar with the tasks and working conditions of the workforce. They then have to decide on the fitness of each worker for the intended tasks. It is now very rare for the radiation component of the working environment to have any significant influence on that decision. Furthermore, this component should have no influence on the administrative conditions of service of those occupationally exposed.

(260) The supervising physician, sometimes supported by specialists, may also be required to counsel workers in three special categories. The first is women who are, or may become, pregnant. They should be advised to inform the physician as soon as they think they may be pregnant, so that the management can be advised to arrange for any necessary change of duties or special protective provisions.

(261) The second group comprises any individuals who have been exposed substantially in excess of the dose limits or may have been involved in potentially dangerous situations. Only in exceptional conditions will clinical tests or treatment be indicated. Nevertheless, depending on the potential for accidents, the physician should ensure that suitable arrangements for diagnostic tests and treatment can be provided at short notice if they should be required. One laboratory test to be considered in this context is the examination of lymphocytes for chromosome aberrations. This test can often give useful results and reassurance after suspected accidents. In-house provisions are rarely needed because there are laboratories in many countries to which blood samples can be sent.

(262) The third group comprises individual workers who are considering volunteering for deliberate exposures as part of biomedical research programmes. In well-designed

experiments, the doses will be small compared with those commonly incurred in occupational exposure and will be limited by dose constraints applied in the optimisation of protection. The supervising physician can provide reassurance and can exclude any volunteers expressing anxiety. Reference to a properly constituted ethics committee is needed to ensure that the research aims are proper and well defined and that the system for selecting volunteers is satisfactory.

(263) The supervising physician needs information about the working conditions and the exposures of individual workers. Some of this information will come from plant records, and some from the protection service. Some of the data will be transferred to, and then form part of, the individual's medical record. Such records are usually regarded as medically confidential. It is important not to let confidentiality compromise the availability of the original data to the management and to non-medical professionals involved in protection.

### 7.5. The Assessment of Doses

(264) The basis of the Commission's recommendations is the restriction of doses and of the probability of incurring doses. The measurement or assessment of doses is fundamental to the practice of radiological protection. Neither the equivalent dose in an organ nor the effective dose can be measured directly. Values of these quantities must be inferred with the aid of models, usually involving environmental, metabolic, and dosimetric components. Ideally, these models and the values chosen for their parameters should be realistic, so that the results they give can be described as "best estimates". Where practicable, estimates should be made of the uncertainties inherent in these results.

(265) In practice, realistic models are rarely available. If the purposes of the model includes the setting of limits or the subsequent testing for compliance with limits, and if realistic models are not available, it is appropriate to use models that are intended to give results that are not likely to underestimate the consequences of exposure, though without overestimating the consequences excessively. In the justification of a practice, the optimisation of protection, or the decision to use intervention following an accident, any errors of estimation are liable to cause misuse of resources. If the models are to be used solely for these purposes, they should therefore be chosen with the emphasis on realism.

#### 7.5.1. Dosimetry in occupational exposure

(266) In occupational exposure, it is usually feasible to monitor the doses received by individuals. Often, however, there is no clear-cut line between workers closely involved with radiation sources and others who are exposed only casually, either because they are rarely present in the relevant locations or because they are remote and receive only trivial doses. To avoid a wasteful use of resources in monitoring and record keeping, it is necessary to identify groups of workers for whom individual monitoring is needed.

(267) The decision to provide individual monitoring for a group of workers depends on many factors. Some of these are technical and others are concerned more with industrial relations. The decision should be taken by the operating management, but should be subject to review by the regulatory agency. Three major technical factors should influence the decision; the expected level of dose or intake in relation to the relevant limits, the likely variations in the dose and intakes, and the complexity of the measurement and interpretation procedures comprising the monitoring programme. This

third factor results in an approach to the monitoring for external exposure that is different from that for intakes and the resulting committed effective dose. Individual monitoring for external radiation is fairly simple and does not require a heavy commitment of resources. It should be used for all those who are occupationally exposed, unless it is clear that their doses will be consistently low, or, as in the case of air crew, it is clear that the circumstances prevent the doses from exceeding an identified value. In addition to its primary function of providing information for the control of exposures, a programme of individual monitoring may be helpful in confirming the classification of workplaces and in detecting fluctuations in working conditions. It gives useful reassurance and may provide data of use in reviewing optimisation programmes.

(268) Individual monitoring for intakes of radioactive material is usually much more difficult, and should be used routinely only for workers who are employed in areas that are designated as controlled areas specifically in relation to the control of contamination and in which there are grounds for expecting significant intakes. Guidance on the type of work calling for individual monitoring is given in *Publication 35* (1982). Guidance on the interpretation of individual monitoring for intakes is given in *Publication 54* (1988).

(269) When calculating the annual limits on intake (ALIs), the Commission has previously used the 50-year committed effective dose. For workers with a working life from 18 to 65 years (a mean of about 40 years) and an expectation of living to 75 years, a value of 35 years would be more typical. However, the difference is small, even for long-lived, long retained, nuclides, and the Commission recommends the retention of the 50-year period for occupational exposure. (See Section 7.5.3 for Public Exposure.) In discussions with an individual worker of the possible health implications of his monitoring results, account should be taken of the actual age at intake. The intake can be directly related to the annual limit on intake more convincingly than the committed dose can be related to the annual dose limit so it will usually be more satisfactory to discuss estimated intakes rather than committed doses.

(270) The assessment of collective dose from occupational exposure is usually based on the recorded doses from individual monitoring programmes, but will often have to be supplemented by the use of data on low individual doses derived from models based on measurements in the workplace.

(271) In practice, it is usually possible without great difficulty to achieve an accuracy of about 10% at the 95% confidence level for measurements of radiation fields in good laboratory conditions. In the workplace, where the energy and orientation of the radiation field are rarely known, uncertainties by a factor of 1.5 will not be unusual in the estimation of annual doses from the external exposure of individual workers. In view of the other uncertainties, this factor is acceptable. It will rarely be possible to achieve the same standard of accuracy when estimating intakes and the associated committed equivalent and effective doses. Uncertainties by a factor of at least 3 may well have to be recognised and are acceptable. Further guidance is given in *Publication 54* (1988).

#### 7.5.2. Dosimetry in medical exposure

(272) The assessment of doses in medical exposure, i.e. doses to patients, is of critical importance in radiotherapy and is dealt with by the International Commission on Radiation Units and Measurements. Frequent measurements on equipment should form an important part of the quality control programme. In diagnostic radiology, there is rarely a need for routine assessment of doses, but periodic measurements should be made to check the performance of equipment and to encourage the optimisation of

protection. In nuclear medicine, the administered activity should always be recorded and the doses, based on standard models, will then be readily available.

### 7.5.3. Dosimetry in public exposure

(273) Routine individual monitoring of persons subject to public exposure is not necessary in normal situations and is not recommended. Dose assessment is then dependent on models representing the pathways between the source and the exposed individuals, sometimes supplemented by environmental monitoring. This procedure cannot take full account of individual habits and characteristics. For comparisons with limits, the models should relate to real or postulated "critical groups". These groups are chosen to be representative of the individuals most highly exposed as a result of the source under review. They are required to be reasonably homogeneous with respect to the characteristics that influence their doses from that source. When this is achieved, any individual limits should be applied to the mean values for the critical group. The Commission has dealt with the selection of critical groups in *Publication 43* (1985).

(274) For public exposure, the integrating period for committed effective dose for children should be from the age of the intake to 70 years. For adults, the period should be 50 years. The Commission has provided age-specific relationships between intake and committed effective dose in *Publication 56* (1989).

(275) In public exposure, it is rare for the collective dose to be predominantly composed of doses in members of the critical group. Dose assessment for the purposes of justification of a practice or the optimisation of protection has to be based on more general models. For current situations, and those extending only into the near future, such models can sometimes be validated by selective measurements, for example on environmental materials or, more rarely, on individuals. For longer-term predictive models, which are often used to forecast doses over many centuries and over large areas, no direct validation is possible. However, techniques such as sensitivity and uncertainty analysis are useful in indicating the likely degree of error and make it possible to test any proposed choice of action against a range of predictive models.

## 7.6. Compliance with the Intended Standard of Protection

(276) All the organisations concerned with radiological protection should have a duty to verify their compliance with their own objectives and procedures. The operating management should establish a system for reviewing its organisational structure and its procedures, a function analogous to financial auditing. Regulatory agencies should conduct similar internal audits and should have the added duty of, and authority for, assessing both the level of protection achieved by operating managements and the degree of compliance with the regulatory provisions. All these verification procedures should include consideration of potential exposures by a verification of the safety provisions. Verification procedures should include a review of quality assurance programmes and some form of inspection. However, inspection is a form of sampling—it cannot cover all eventualities. It is best seen as a mechanism for persuading those inspected to put, and keep, their own houses in order.

### 7.6.1. Record keeping

(277) Any system of validation includes the keeping of records. The minimum requirements will usually be laid down by the regulatory agencies, but operating manage-

ments should consider the additional requirements for records for their own purposes. The type of record, the degree of detail, and the retention period should all be defined formally. A balance has to be struck between the complexity of the initial entry of data, which may compromise the accuracy or completeness, and the possible future use of the records. The value of most records decreases with time, as does the likelihood of their being needed. As a general guide, and subject to any regulatory requirements, records giving the results of assessments of individual doses should be retained for periods comparable with the expected lifetime of the individual; those giving supplementary information used in the interpretation of monitoring results, e.g. results of monitoring of the workplace, should be retained for a period long enough to keep them available for any likely re-assessment of the interpretation, a few years. The details and retention of personnel records should be in accordance with the normal practice of the employer. The details of releases of waste to the environment should be retained for at least 10 years, with summaries being kept for several decades.

## 7.7. Emergency Planning

(278) When an emergency that may affect the public is declared, there should usually be a shift in the placing of responsibilities. In many cases, there will be an operating management at the scene of the initiating event. The operating management will then be available to take initial control of the event itself, but this may not be regarded as appropriate if the event is outside, or extends beyond, the operator's premises. The wider responsibilities for emergency action will usually have to be carried by the regulatory agency, which will also have to decide who shall be responsible for implementing any action following its decisions.

(279) Accidents or operational misjudgements may call for urgent action. The responsibility for planning local emergency action should fall primarily on the operating management, if this can be identified in advance. More general, and especially national, planning should be the responsibility of the regulatory agency or other body designated by the Government. Local and national plans need to be closely co-ordinated and linked to other plans dealing with accidents not involving radiation. Links to regional and international plans should also be provided. Bilateral agreements with neighbouring states are often needed and are essential where major installations are located near national boundaries. The scale of the detailed plans for dealing with radiation accidents will be influenced by the degree of co-ordination with other plans and by the magnitude and expected frequency of accidents. The establishment, maintenance, and exercising of emergency plans require a substantial commitment of resources, so the choice of the scale of the plans has considerable practical implications.

(280) Experience has identified several key areas of difficulty in emergency planning. The first is the recognition that an accident has occurred and that emergency action is needed. This presents few difficulties if the accident is to major plant, but dangerous situations due to lost or misused radiographic sources have been very difficult to recognise. The second problem area is the rapid acquisition and interpretation of data. It is obvious that data have to be obtained in the area affected by the accident, but it is not always recognised that there will be a widespread demand for data to provide reassurance in unaffected areas. Thirdly, the interpreted data have to lead to decisions and actions, or to a convincing conclusion that no action is needed. The initial decisions will often have to be made by someone on the spot, regardless of the formal chain of responsi-

bilities. This should be recognised in the plans, but provision should also be made for the more formal making of decisions on a longer timescale. The fourth problem area is communications. The demand for information has been consistently underestimated in the past. The communication system for the emergency organisation is not difficult to specify, but it is expensive to establish and maintain. Adequate communications with the public are very much more difficult to achieve. The provision of local instructions and advice in the event of an accident is fairly straightforward, once the content has been settled. It is much more difficult to disseminate reassurance to the much larger areas where no action is called for. Special provisions should be made in national plans.

(281) Because of these special features, there are many parts of emergency plans that are not in routine use. These have to be maintained in a state of readiness by regular exercises. Exercises are often regarded as wasteful of scarce resources, but they should be treated as a necessary part of emergency planning.

(282) It is necessary to initiate emergency procedures by some form of declaration of a state of emergency. This may be local, perhaps applying only to a single installation, or even to a single workplace, or it may be more widespread. Such a declaration has the additional function of establishing that the system of protection is now that relating to intervention. Provision also has to be made for the withdrawal of the state of emergency and of any countermeasures that have been applied.

(283) Although flexibility is a necessary feature of emergency plans, it is very valuable to include in the plans a set of intervention levels to provide an immediate basis for urgent decisions. These intervention levels should be established for the types of action likely to be needed and should be promulgated by, or on behalf of, the regulatory agency. As discussed in Chapter 6, the choice of intervention levels should be based on the dose averted by the proposed action. Since the dose that will be averted cannot easily be estimated in the period immediately after an accident, derived intervention levels should be established for quantities that can be measured or estimated at the time of use. The intervention levels should not be treated as limits, they are guides to action.

(284) To avoid unnecessary restrictions in international trade, especially in foodstuffs, it may be necessary, in this context, to apply derived intervention levels in a different way. They could then indicate a line of demarcation between freely permitted exports or imports and those that should be the subject of special decisions. Any restrictions applied to goods below the intervention levels, better called intervention exemption levels for this purpose, should be regarded as artificial barriers to trade. Trade in materials above an intervention exemption level should not automatically be prohibited, but such materials might be subject to temporary controls. Intervention exemption levels used in this way in international trade should not necessarily have the same quantitative values as the intervention levels used for initiating action in other circumstances.

### 7.8. Exclusion and Exemption from Regulatory Control

(285) In order to avoid excessive regulatory procedures, most regulatory systems include provisions for granting exemptions in cases where it is clear that a practice is justified, but where regulatory provisions are unnecessary. Provision may also be made for the complete exclusion of some situations from the scope of any regulatory instruments.

(286) The Commission believes that the exemption of sources is an important component of the regulatory functions. It notes that the International Atomic Energy

Agency and the Nuclear Energy Agency of OECD issue advice on this subject to their member states.

(287) There are two grounds for exempting a source or an environmental situation from regulatory control. One is that the source gives rise to small individual doses and small collective doses in both normal and accident conditions. The other is that no reasonable control procedures can achieve significant reductions in individual and collective doses.

(288) The basis for exemption on the grounds of trivial dose is much sought after, but very difficult to establish. Apart from the difficulty of deciding when an individual or a collective dose is small enough to be disregarded for regulatory purposes, there is a considerable difficulty in defining the source. For example, if the source is defined as a single smoke detector, both the individual and the collective doses from that source may well be trivial, but the individual may be exposed to many other sources. If the source is taken as smoke detectors in general, the individual doses will still be small, but the collective dose may be substantial. The underlying problem is that exemption is necessarily a source-related process, while the triviality of the dose is primarily individual-related.

(289) When the exempt source comprises a class of devices, it may not be appropriate to exempt the manufacture and large scale storage of the devices. The devices themselves can be made subject to the requirements of approved engineering standards, and their sale and use can then be exempted from all further regulatory requirements. When the use is so exempted, it is necessary also to be able to exempt the eventual disposal of the devices.

(290) The second basis for exemption calls for a study similar to that needed in the optimisation of protection. It provides a logical basis for exemption of sources that cannot be exempted solely on the grounds of trivial doses, but for which regulation on any reasonable scale will produce little or no improvement.

(291) Sources that are essentially uncontrollable, such as cosmic radiation at ground level and potassium-40 in the body, can best be dealt with by the process of exclusion from the scope of the regulatory instruments, rather than by an exemption provision forming part of the regulatory instruments.

(292) One other form of exemption is sometimes considered. Some sources give rise to widespread exposures involving only very small individual doses. It has been suggested that these sources could be exempted from regulatory concern and the small individual doses might be excluded from the calculation of collective dose. In effect, it is argued that the resulting risks to individuals are so insignificant that they can be ignored even if there are many exposed individuals. In the context of waste management, this approach tends to ignore large collective doses delivered at long ranges, often in other countries. This method of exemption is sometimes the result of an implicit form of optimisation of protection. If the doses are individually small and the sources are widespread, it may well be impossible to reduce the doses further with any reasonable deployment of resources. It is unlikely, however, that this argument would lead to a single value of dose for exemption purposes.

(293) The Commission recognises that this method of exemption, i.e. ignoring the collective dose if the individual doses are all very small, is in use, not always explicitly, and that it often leads to conclusions that are broadly consistent with those that would result from the application of the Commission's system of protection. Nevertheless, this consistency is not always achieved and the Commission does not recommend the use of

this technique. The extent to which small individual doses should be included in the estimation of collective doses for the purposes of optimisation depends on the extent to which the contribution from these doses influences the choice between the options under review. Further guidance is given in *Publication 55* (1989).

## SUMMARY OF RECOMMENDATIONS

This summary contains the principal recommendations and new concepts in the 1990 Recommendations of the Commission. Explanatory material is omitted. The order of the summary follows that of the Main Text of the recommendations.

### Introduction

(S1) The Recommendations are intended to be of help to regulatory and advisory agencies and to management bodies and their professional staff. They deal only with ionising radiation and with the protection of man. The Commission emphasises that ionising radiation needs to be treated with care rather than fear and that its risks should be kept in perspective with other risks. Radiological protection cannot be conducted on the basis of scientific considerations alone. All those concerned have to make value judgements about the relative importance of different kinds of risk and about the balancing of risks and benefits.

### Quantities Used in Radiological Protection

(S2) The Commission uses macroscopic dosimetric quantities while recognising that microdosimetric quantities based on the statistical distribution of events in a small volume of material may eventually be more appropriate. The principal dosimetric quantities in radiological protection are the mean absorbed dose in a tissue or organ,  $D_T$ , the energy absorbed per unit mass; the equivalent dose in a tissue or organ,  $H_T$ , formed by weighting the absorbed dose by the radiation weighting factor,  $w_R$ ; and the effective dose,  $E$ , formed by weighting the equivalent dose by the tissue weighting factor,  $w_T$ , and summing over the tissues. The time integral of the effective-dose rate following an intake of a radionuclide is called the committed effective dose,  $E(x)$ , where  $x$  is the integration time (in years) following the intake. The unit of absorbed dose is the gray (Gy), and the unit of both equivalent and effective dose is the sievert (Sv). The values of the radiation and tissue weighting factors are given in Tables S-1 and S-2.

(S3) Another useful quantity is the collective effective dose, which is the product of the mean effective dose in a group and the number of individuals in that group. With some reservations, it can be thought of as representing the total consequences of the exposure of a population or group.

(S4) The Commission uses "dose" as a generic term that can apply to any of the relevant dosimetric quantities. The Commission also uses the term "exposure" in a generic sense to mean the process of being exposed to radiation or radioactive material. The significance of an exposure in this sense is determined by the resulting doses.

### Biological Aspects of Radiological Protection

(S5) Ionising radiation causes both deterministic and stochastic effects in irradiated tissue. Radiological protection aims at avoiding deterministic effects by setting dose limits below their thresholds. Stochastic effects are believed to occur, albeit with low frequency, even at the lowest doses and therefore have been taken into account at all doses.

Table S-1. Radiation weighting factors<sup>1</sup>

Type and energy range <sup>2</sup>	Radiation weighting factor, $w_r$
Photons, all energies	1
Electrons and muons, all energies <sup>1</sup>	1
Neutrons, energy < 10 keV	5
10 keV to 100 keV	10
> 100 keV to 2 MeV	20
> 2 MeV to 20 MeV	10
> 20 MeV	5
(See also Figure 1)	
Protons, other than recoil protons, energy > 2 MeV	5
Alpha particles, fission fragments, heavy nuclei	20

<sup>1</sup> All values relate to the radiation incident on the body or, for internal sources, emitted from the source.

<sup>2</sup> The choice of values for other radiations is discussed in Annex A.

<sup>3</sup> Excluding Auger electrons emitted from nuclei bound to DNA (see paragraph 26).

Table S-2. Tissue weighting factors<sup>1</sup>

Tissue or organ	Tissue weighting factor, $w_T$
Gonads	0.20
Bone marrow (red)	0.12
Colon	0.12
Lung	0.12
Stomach	0.12
Bladder	0.05
Breast	0.05
Liver	0.05
Oesophagus	0.05
Thyroid	0.05
Skin	0.01
Bone surface	0.01
Remainder	0.05 <sup>2,3</sup>

<sup>1</sup> The values have been developed from a reference population of equal numbers of both sexes and a wide range of ages. In the definition of effective dose they apply to workers, to the whole population, and to either sex.

<sup>2</sup> For purposes of calculation, the remainder is composed of the following additional tissues and organs: adrenals, brain, upper large intestine, small intestine, kidney, muscle, pancreas, spleen, thymus and uterus. The list includes organs which are likely to be selectively irradiated. Some organs in the list are known to be susceptible to cancer induction. If other tissues and organs subsequently become identified as having a significant risk of induced cancer they will then be included either with a specific  $w_T$  or in this additional list constituting the remainder. The latter may also include other tissues or organs selectively irradiated.

<sup>3</sup> In those exceptional cases in which a single one of the remainder tissues or organs receives an equivalent dose in excess of the highest dose in any of the twelve organs for which a weighting factor is specified, a weighting factor of 0.025 should be applied to that tissue or organ and a weighting factor of 0.025 to the average dose in the rest of the remainder as defined above.

(S6) Deterministic effects result from the killing of cells which, if the dose is large enough, causes sufficient cell loss to impair the function of the tissue. The probability of causing such harm will be zero at small doses, but above some level of dose (the threshold for clinical effect) the probability will increase steeply to unity (100%). Above the threshold, the severity of the harm will increase with dose. Thresholds for these effects are often at doses of a few Gy or dose rates of a fraction of a Gy per year.

(S7) An important observation in children exposed in utero during a critical 8–15 week period, at Hiroshima and Nagasaki, is a downward shift in the distribution of IQ with increasing dose which can result, after higher doses, in an increase in the probability of severe mental retardation. The effect is presumed to be deterministic with a threshold related to the minimum shift in IQ that can be recognised.

(S8) Stochastic effects may result when an irradiated cell is modified rather than killed. Modified somatic cells may subsequently, after a prolonged delay, develop into a cancer. There are repair and defence mechanisms that make this a very improbable outcome. Nevertheless, the probability of a cancer resulting from radiation increases with increments of dose, probably with no threshold. The severity of the cancer is not affected by the dose. If the damage occurs in a cell whose function is to transmit genetic information to later generations, any resulting effects, which may be of many different kinds and severity, are expressed in the progeny of the exposed person. This type of stochastic effect is called "hereditary".

(S9) The Commission has estimated the probability of a fatal cancer by relying mainly on studies of the Japanese survivors of the atomic bombs and their assessment by bodies such as UNSCEAR and BEIR. These committees have estimated the lifetime cancer risk by considering the accumulated data to 1985, the new dosimetry (DS86) and projection to lifetime by a multiplicative or modified multiplicative model, for high dose, high dose rate exposure. The Commission has concluded, after reviewing the available experimental information on dose-response relationships and the influence of dose and dose rate, that the most probable response is linear quadratic in form for low LET radiation. The linear coefficient at low doses or low dose rates is obtained from the high dose, high dose rate estimates of risk by dividing by a DDREF (dose and dose rate effectiveness factor) of 2. The nominal fatal cancer probabilities for a working population and for a general population, which differ somewhat because of the greater sensitivity of young people, are given in Table S-3. The Commission has made its own estimates of how this fatal cancer risk is distributed among organs and the length of life lost for cancer in each of these organs, by further analysis of the data on the atomic bomb survivors.

(S10) The estimates of severe hereditary effects are also based on the assessments of UNSCEAR and BEIR of experimental data on genetic effects in animals. Evidence suggests that these estimates are not less than the corresponding effects in man. For low dose and dose rates, the probability coefficient for severe hereditary effects in all generations (resulting about equally from dominant and X-linked mutations on the one hand, and multifactorial diseases weighted for severity on the other) are given for both a working population and a general population in Table S-3.

(S11) The Commission uses the term detriment to represent the combination of the probability of occurrence of a harmful health effect and a judgement of the severity of that effect. The many aspects of detriment make it undesirable to select a single quantity to represent the detriment and the Commission has therefore adopted a multi-dimensional concept. The principal components of detriment are the following stochastic quantities: the probability of attributable fatal cancer, the weighted probability of

Table S-3. Nominal probability coefficients for stochastic effects

Exposed population	Detriment ( $10^{-2} \text{ Sv}^{-1}$ ) <sup>1</sup>			Total
	Fatal cancer <sup>2</sup>	Non-fatal cancer	Severe hereditary effects	
Adult workers	4.0	0.8	0.8	5.6
Whole population	5.0	1.0	1.3	7.3

<sup>1</sup> Rounded values.

<sup>2</sup> For fatal cancer, the detriment is equal to the probability coefficient.

attributable non-fatal cancer, the weighted probability of severe hereditary effects and the length of life lost if the harm occurs. The values of this aggregated detriment at low dose for both a working population and a general population are also given in Table S-3.

(S12) The Commission has also assessed the distribution of the detriment in organs and tissues by considering first the fatal cancer probability in each of them, multiplying by an appropriate factor for non-fatal cancer (which is determined by the severity (lethality factor) for that cancer), adding in the probability of severe hereditary effects and adjusting for the relative length of life lost. This distribution of aggregate detriment among organs is represented, after appropriate rounding, by the tissue weighting factors,  $w_T$ , given in Table S-2.

(S13) The effective dose is the sum of the weighted equivalent doses in all the tissues and organs of the body. It is given by the expression

$$E = \sum_T w_T \cdot H_T$$

where  $H_T$  is the equivalent dose in tissue or organ T and  $w_T$  is the weighting factor for tissue T. The effective dose can also be expressed as the sum of the doubly weighted absorbed dose in all the tissues and organs of the body.

### The Conceptual Framework of Radiological Protection

(S14) A system of radiological protection should aim to do more good than harm, should call for protection arrangements that maximise the net benefit, and should aim to limit the inequity that may arise from a conflict of interest between individuals and society as a whole.

(S15) Some human activities increase the overall exposure to radiation. The Commission calls these human activities "practices". Other human activities can decrease the overall exposure by influencing the existing causes of exposure. The Commission describes these activities as "intervention".

(S16) The Commission uses a division into three types of exposure: occupational exposure, which is the exposure incurred at work, and principally as a result of work; medical exposure, which is principally the exposure of persons as part of their diagnosis or treatment; and public exposure, which comprises all other exposures.

(S17) In practices and in intervention, it will often be virtually certain that exposures will occur and their magnitude will be predictable, albeit with some degree of error. Sometimes, however, there will be a potential for exposure, but no certainty that it will occur. The Commission calls such exposures "potential exposures".

### The system of protection in practices

(S18) The system of radiological protection recommended by the Commission for proposed and continuing practices is based on the following general principles.

- No practice involving exposures to radiation should be adopted unless it produces sufficient benefit to the exposed individuals or to society to offset the radiation detriment it causes. (The justification of a practice.)
- In relation to any particular source within a practice, the magnitude of individual doses, the number of people exposed, and the likelihood of incurring exposures where these are not certain to be received should all be kept as low as reasonably achievable, economic and social factors being taken into account. This procedure should be constrained by restrictions on the doses to individuals (dose constraints), or the risks to individuals in the case of potential exposures (risk constraints), so as to limit the inequity likely to result from the inherent economic and social judgements. (The optimisation of protection.)
- The exposure of individuals resulting from the combination of all the relevant practices should be subject to dose limits, or to some control of risk in the case of potential exposures. These are aimed at ensuring that no individual is exposed to radiation risks that are judged to be unacceptable from these practices in any normal circumstances. Not all sources are susceptible of control by action at the source and it is necessary to specify the sources to be included as relevant before selecting a dose limit. (Individual dose and risk limits.)

### The system of protection in intervention

(S19) The system of radiological protection recommended by the Commission for intervention is based on the following general principles.

- The proposed intervention should do more good than harm, i.e. the reduction in detriment resulting from the reduction in dose should be sufficient to justify the harm and the costs, including social costs, of the intervention.
- The form, scale, and duration of the intervention should be optimised so that the net benefit of the reduction of dose, i.e. the benefit of the reduction in radiation detriment, less the detriment associated with the intervention, should be maximised.

Dose limits do not apply in the case of intervention. Principles (a) and (b) can lead to intervention levels which give guidance to the situations in which intervention is appropriate. There will be some level of projected dose above which, because of serious deterministic effects, intervention will almost always be justified.

(S20) Any system of protection should include an overall assessment of its effectiveness in practice. This should be based on the distribution of doses achieved and on an appraisal of the steps taken to limit the probability of potential exposures. It is important that the basic principles should be treated as a coherent system. No one part should be taken in isolation.

### The Control of Occupational Exposure

#### Dose constraints

(S21) An important feature of optimisation is the choice of dose constraints, the source-related values of individual dose used to limit the range of options considered in

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### The Control of Occupational Exposure

#### Dose constraints

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the procedure of optimisation. For many types of occupation, it is possible to reach conclusions about the level of individual doses likely to be incurred in well-managed operations. This information can then be used to establish a dose constraint for that type of occupation. The class of occupation should be specified in fairly broad terms, such as work in x-ray diagnostic departments, the routine operation of nuclear plant, or the inspection and maintenance of nuclear plant. Limits prescribed by regulatory agencies and restrictions applied by managements to specific operations as part of the day-to-day control of exposures are not constraints in the sense used here. In general, they should be established on the basis of the results of optimisation. It will usually be appropriate for dose constraints to be fixed at the national or local level.

#### Dose limits

(S22) The dose limits for application in occupational exposure are summarised in Table S-4.

(S23) Dose limits are needed as part of the control of occupational exposure, both to impose a limit on the choice of dose constraints and to provide a protection against errors of judgement in the application of optimisation.

(S24) In setting dose limits, the Commission's aim is to establish, for a defined set of practices, and for regular and continued exposure, a level of dose above which the consequences for the individual would be widely regarded as unacceptable. In the past, the Commission has used the attributable probability of death or severe hereditary disorders as the basis for judging the consequences of an exposure. This quantity is still a major factor, but is no longer regarded by the Commission as sufficient to describe the detriment.

(S25) The Commission recommends a limit on effective dose of 20 mSv per year, averaged over 5 years (100 mSv in 5 years), with the further provision that the effective

Table S-4. Recommended dose limits<sup>1</sup>

Application	Dose limit	
	Occupational	Public
Effective dose	20 mSv per year, averaged over defined periods of 5 years <sup>2</sup>	1 mSv in a year <sup>3</sup>
Annual equivalent dose in		
the lens of the eye	150 mSv	15 mSv
the skin <sup>4</sup>	500 mSv	50 mSv
the hands and feet	500 mSv	—

<sup>1</sup> The limits apply to the sum of the relevant doses from external exposure in the specified period and the 50-year committed dose (to age 70 years for children) from intakes in the same period (see paragraph 1.4.3).

<sup>2</sup> With the further provision that the effective dose should not exceed 50 mSv in any single year. Additional restrictions apply to the occupational exposure of pregnant women, which is discussed in Section 5.3.3 of the Main Text.

<sup>3</sup> In special circumstances, a higher value of effective dose could be allowed in a single year, provided that the average over 5 years does not exceed 1 mSv per year.

<sup>4</sup> The limitation on the effective dose provides sufficient protection for the skin against stochastic effects. An additional limit is needed for localised exposures in order to prevent deterministic effects. (See paragraphs 1.7.3 and 1.9.4.)

dose should not exceed 50 mSv in any single year. The 5-year period would have to be defined by the regulatory agency, e.g. as discrete 5-year calendar periods. The Commission would not expect the period to be introduced and then applied retrospectively. It is implicit in these recommended dose limits that the dose constraint for optimisation should not exceed 20 mSv in a year.

(S26) Subject to medical advice in individual cases, there need be no special restrictions applied to the exposure of an individual following a control period in which the exposure of the individual has exceeded a dose limit. Such events should call for a thorough examination, usually by the regulatory agency, of the design and operational aspects of protection in the installation concerned, rather than for restrictions or penalties applied to the exposed individual. If the dose is unknown, or is thought to be high, referral to a physician should be considered.

(S27) The recommended limits should apply to all forms of occupational exposure, unless special provisions have been made by the regulatory agency. Because of the difficulties of responding rapidly to an increase in stringency in operations on plant and equipment already in existence, the Commission recognises that regulatory agencies may wish to make temporary use of higher dose limits. Such arrangements should be regarded as transient.

(S28) The dose limit forms only a part of the system of protection aimed at achieving levels of dose that are as low as reasonably achievable, economic and social factors being taken into account. It is not to be seen as a target. It represents, in the Commission's view, the point at which regular, extended, deliberate, occupational exposure can reasonably be regarded as only just tolerable.

(S29) The restrictions on effective dose are sufficient to ensure the avoidance of deterministic effects in all body tissues and organs except the lens of the eye, which makes a negligible contribution to the effective dose, and the skin, which may well be subject to localised exposures. Separate dose limits are needed for these tissues. The annual limits are 150 mSv for the lens and 500 mSv for the skin, averaged over any 1 cm<sup>2</sup>, regardless of the area exposed.

(S30) For internal exposure, annual limits on intake will be based on a committed effective dose of 20 mSv. The estimated intakes may be averaged over a period of 5 years to provide some flexibility. The occupational limits for radon are under review. Meanwhile, the values given in *Publication 47* (1986) remain valid.

#### The occupational exposure of women

(S31) The basis for the control of the occupational exposure of women who are not pregnant is the same as that for men and the Commission recommends no special occupational dose limit for women in general.

(S32) Once pregnancy has been declared, the conceptus should be protected by applying a supplementary equivalent dose limit to the surface of the woman's abdomen (lower trunk) of 2 mSv for the remainder of the pregnancy and by limiting intakes of radionuclides to about 1/20 of the ALI. The Commission wishes to emphasise that the use of its system of protection, particularly the use of source-related dose constraints, will usually provide an adequate guarantee of compliance with this limit without the need for specific restrictions on the employment of pregnant women. The principal criterion will then be that the employment should be of a type that does not carry a significant probability of high accidental doses and intakes. High-dose and high-risk occupations from which pregnant women should be excluded should be defined by regulatory agencies.

### The Control of Medical Exposure

(S33) In the justification of a practice leading to medical exposures, the practice should be defined in broad terms. However, each procedure, either diagnostic or therapeutic, is subject to a separate decision, so that there is an opportunity to apply a further, case-by-case, justification for each procedure. This will not be necessary for simple diagnostic procedures based on common indications, but may be important for complex investigations and for therapy.

(S34) There is considerable scope for dose reductions in diagnostic radiology using the techniques of optimisation of protection. Consideration should be given to the use of dose constraints, or investigation levels, selected by the appropriate professional or regulatory agency, for application in some common diagnostic procedures. They should be applied with flexibility to allow higher doses where indicated by sound clinical judgement.

(S35) Constraints should also be considered in the optimisation of protection for medical exposures when the procedures are not intended to be of direct value to the exposed individual, as in scientific and clinical studies involving the exposure of volunteers.

(S36) Medical exposures are usually intended to provide a direct benefit to the exposed individual. If the practice is justified and the protection optimised, the dose in the patient will be as low as is compatible with the medical purposes. The Commission therefore recommends that dose limits should not be applied to medical exposures. Further, it is not appropriate to include the doses incurred by patients in the course of diagnostic examinations or therapy when considering compliance with dose limits applied to occupational or public exposures.

(S37) Diagnostic and therapeutic procedures causing exposures of the abdomen of women likely to be pregnant should be avoided unless there are strong clinical indications. Information on possible pregnancy should be obtained from the patient herself. If the most recent expected menstruation has been missed, and there is no other relevant information, the woman should be assumed to be pregnant.

### The Control of Public Exposure

(S38) The control of public exposure in all normal situations is exercised by the application of controls at the source rather than in the environment. The controls are achieved almost entirely by the procedures of constrained optimisation and the use of prescriptive limits. It is often convenient to class together individuals who form a homogeneous group with respect to their exposures to a single source. When such a group is typical of those most highly exposed by that source, it is known as a critical group. The dose constraint should be applied to the mean dose in the critical group from the source for which the protection is being optimised.

#### Dose limits

(S39) The scope of dose limits for public exposure is confined to the doses incurred as the result of practices. Doses incurred in situations where the only available protective action takes the form of intervention are excluded from that scope. Separate attention has to be paid to potential exposures. Radon in dwellings and in the open air, radioactive materials, natural or artificial, already in the environment, and other natural sources are

examples of situations that can be influenced only by intervention. Doses from these sources are therefore outside the scope of the dose limits for public exposure. The conduct of intervention involves occupational exposure and should be treated accordingly.

(S40) The Commission now recommends that the limit for public exposure should be expressed as an effective dose of 1 mSv in a year. However, in special circumstances, a higher value of effective dose could be allowed in a single year, provided that the average over 5 years does not exceed 1 mSv per year.

(S41) In selecting the limit on effective dose, the Commission has sought a value that would be only just short of unacceptable for continued exposure as the result of deliberate practices the use of which is a matter of choice. This does not imply that higher doses from other sources, such as radon in dwellings, should be regarded as unacceptable. The existence of these sources may be undesirable but is not a matter of choice. The doses can be controlled only by intervention, which will also have undesirable features.

(S42) Limits are also needed for the lens of the eye and skin since these tissues will not necessarily be protected against deterministic effects by the limit on effective dose. The Commission recommends annual limits of 15 mSv for the lens and 50 mSv for the skin averaged over any 1 cm<sup>2</sup>, regardless of the area exposed. The recommended limits are summarised in Table S-4.

### Potential Exposures

(S43) The initial treatment of potential exposures should form part of the system of protection applied to practices, but it should be recognised that the exposures, if they occur, may lead to intervention. At this stage, there should be two objectives, prevention and mitigation. Prevention is the reduction of the probability of the sequences of events that may cause or increase radiation exposures. Mitigation is the limitation and reduction of the exposures if any of these sequences do occur. A great deal can be accomplished at the stages of design and operation to reduce the consequences of accident sequences so that intervention may not become necessary.

(S44) In order to maintain a strict coherence in the treatment of actual and potential exposures, it would be necessary to extend the concept of detriment to include the probability of occurrence of the situation giving rise to the detriment. Techniques for achieving this are still being developed. A comprehensive approach to this problem calls for the application of multi-attribute analysis.

(S45) A simpler approach is possible for both individual and collective exposures if the doses will be small even if the event occurs. If the doses, should they occur, will not be in excess of dose limits, it is adequate to use the product of the expected dose and its probability of occurrence as if this were a dose that was certain to occur. The conventional procedures of justification and optimisation can then be applied.

### The System of Protection in Intervention

(S46) Before a programme of intervention is initiated, it should be demonstrated that the proposed intervention will be justified, i.e. do more good than harm, and that the form, scale, and duration of the intervention have been chosen so as to optimise the protection. The processes of justification and optimisation both apply to the protective

action, so it is necessary to consider them together when reaching a decision. Justification is the process of deciding that the disadvantages of each component of intervention, i.e. of each protective action, are more than offset by the reductions in the dose likely to be achieved. Optimisation is the process of deciding on the method, scale and duration of the action so as to obtain the maximum net benefit. In simple terms, the difference between the disadvantages and the benefits, expressed in the same terms, e.g. costs, including social costs with an allowance for anxiety, should be positive for each protective action adopted and should be maximised by settling the details of that action.

#### Radon in Dwellings

(S47) Radon in dwellings needs special attention because both the individual and the collective doses from radon are higher than those from almost any other source. If improvements are needed in existing dwellings, they have to be achieved by intervention involving modifications to the dwellings or to the behaviour of the occupants.

(S48) The Commission recommended the use of action levels to help in deciding when to require or advise remedial action in existing dwellings. The choice of an action level is complex, depending not only on the level of exposure, but also on the likely scale of action, which has economic implications for the community and for individuals. For new dwellings, guides or codes for their construction in selected areas can be established so that it is highly probable that exposures in these dwellings will be below some chosen reference level. The Commission has initiated a further review of current experience with a view to issuing revised recommendations in due course. Meanwhile the guidance in *Publication 39* (1984) should still be used.

#### Intervention After Accidents

(S49) The benefit of a particular protective action within a programme of intervention should be judged on the basis of the reduction in dose achieved or expected by that specific protective action, i.e. the dose averted. Thus each protective action has to be considered on its own merits. In addition, however, the doses that would be incurred via all the relevant pathways of exposure, some subject to protective actions and some not, should be assessed. If the total dose in some individuals is so high as to be unacceptable even in an emergency, the feasibility of additional protective actions influencing the major contributions to the total dose should be urgently reviewed. Doses causing serious deterministic effects or a high probability of stochastic effects would call for such a review.

(S50) Occupational exposures of emergency teams during emergency and remedial action can be limited by operational controls. Some relaxation of the controls for normal situations can be permitted in serious accidents without lowering the long-term level of protection. This relaxation should not permit the exposures in the control of the accident and in the immediate and urgent remedial work to give effective doses of more than about 0.5 Sv except for life-saving actions, which can rarely be limited by dosimetric assessments. The equivalent dose to skin should not be allowed to exceed about 5 Sv. Once the immediate emergency is under control, remedial work should be treated as part of the occupational exposure incurred in a practice.

#### Practical Implementation of the Commission's Recommendations

(S51) Chapter 7 of the recommendations emphasises the importance of the operational level of radiological protection and shows how this should be developed from the requirements of regulatory agencies and the recommendations of the Commission. The Commission now recommends that the designation of controlled and supervised areas should be decided either at the design stage or locally by the operating management on the basis of operational experience and judgement. The classification of working conditions based upon expected dose is no longer recommended. The Chapter gives advice on the measurement of doses (monitoring and record keeping) and on medical surveillance. It also discusses emergency planning and the bases for exemption from regulatory requirements. It deals with both practices and intervention.

**ANNEX A**  
**QUANTITIES USED IN RADIOLOGICAL PROTECTION**

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### A.1. Introduction

(A1) Application of the Commission's recommendations requires an understanding of a variety of concepts and quantities. Many of these have application in other fields of science and precision in their definition reflects this broad application. The information on basic radiation units and quantities has been obtained from reports of the International Commission on Radiation Units and Measurements (ICRU, 1980; 1985).

(A2) A somewhat different approach is acceptable and more appropriate for many of the quantities which apply only to radiation protection. These are discussed in terms of weighting factors to be used to allow for the different types and energies of radiation incident upon the body and the relative radio-sensitivities of the different tissues of the body.

### A.2. Absorbed Dose

(A3) Absorbed dose,  $D$ , is defined by the relationship:

$$D = \frac{d\bar{\epsilon}}{dm}$$

where  $d\bar{\epsilon}$  is the mean energy imparted by ionising radiation to the matter in a volume element and  $dm$  is the mass of the matter in this volume element. The SI unit for absorbed dose is joule per kilogram ( $\text{J kg}^{-1}$ ) and its special name is gray (Gy). The time derivative of absorbed dose is the absorbed dose rate,  $\dot{D}$ , i.e.,

$$\dot{D} = \frac{dD}{dt}$$

where  $dD$  is the increment of absorbed dose in the time interval  $dt$ .

### A.3. Organ Dose

(A4) For radiation protection purposes, it is useful to define a tissue- or organ-average absorbed dose,  $D_T$ , i.e.,

$$D_T = \frac{\epsilon_T}{m_T}$$

where  $\epsilon_T$  is the total energy imparted in a tissue or organ and  $m_T$  is the mass of that tissue or organ. For example,  $m_T$  may range from less than 10 g for the ovaries to over 70 kg for the whole body.

### A.4. Linear Energy Transfer

(A5) The unrestricted linear energy transfer is defined by ICRU as

$$L_\infty = \frac{dE}{d\ell}$$

where  $dE$  is the energy lost by a charged particle in traversing a distance  $d\ell$ . In this report  $L_\infty$  is denoted by  $L$ .

### A.5. Lineal Energy

(A6) Lineal energy is defined by ICRU as  $y = \epsilon/\bar{\ell}$  where  $\epsilon$  is the energy imparted to the matter in a volume of interest by an energy deposition event and  $\bar{\ell}$  is the mean chord length in that volume. Since the mean lineal energy represents discrete energy deposition, it is in principle more meaningful than linear energy transfer (LET) as the physical quantity to be used in the specification of radiation quality. Although this characteristic of lineal energy is directly measurable,  $L$  has been used in most of the existing practical radiation protection calculations. Therefore,  $Q$  will be given here as a function of  $L$  although the Commission recognises that the use of lineal energy is also possible.

### A.6. Quality Factor

(A7) Since the probability of stochastic effects is found to be dependent on the quality of the radiation, a weighting factor has been traditionally introduced to modify the absorbed dose and to define the dose equivalent. This dimensionless factor, called the quality factor,  $Q$ , is given as a function of the unrestricted linear energy transfer.

### A.7. $Q$ - $L$ Relationship

(A8) The Commission has modified its recommendations on the formal relationship between the quality factor,  $Q(L)$ , and unrestricted linear energy transfer,  $L$ , to reflect the higher  $\text{RBE}_m$  values for intermediate energy neutrons given in Annex B while maintaining as much simplicity as possible. Simplicity is important to reflect our lack of precise information in man and an appreciation of the practical aspects of radiation protection. For example, the Commission does not believe it is helpful to adopt different quality factor values for different photon energies. The Commission also recognises the reduced effectiveness of heavy ions with  $L$  greater than  $100 \text{ keV } \mu\text{m}^{-1}$ . The following formulation is adopted:

Table A-1. Specified  $Q$ - $L$  relationships

Unrestricted linear energy transfer, $L$ , in water ( $\text{keV } \mu\text{m}^{-1}$ )	$Q(L)^1$
< 10	1
10-100	$0.32L - 2.2$
> 100	$300/\sqrt{L}$

<sup>1</sup> With  $L$  expressed in  $\text{keV } \mu\text{m}^{-1}$ .

### A.8. Radiation Weighting Factor

(A9) The Commission now believes that the detail and precision inherent in using a formal  $Q$ - $L$  relationship to modify absorbed dose to reflect the higher probability of detriment resulting from exposure to radiation components with high LET is not justified because of the uncertainties in the radiobiological information. In place of  $Q$  or more precisely  $\bar{Q}$ , the Commission now selects radiation weighting factors,  $w_R$ , based on a

review of the biological information, a variety of exposure circumstances and inspection of the results of traditional calculations of the ambient dose equivalent.

(A10) The Commission now specifies modifying factors which apply to the tissue or organ absorbed dose and are based on the type and quality of the external radiation field or on the type and quality of the radiation emitted by an internally deposited radionuclide.

(A11) The specified values of  $w_R$  are given in Table A-2.

Table A-2. Radiation weighting factors<sup>1</sup>

Type and energy range <sup>2</sup>	Radiation weighting factor, $w_R$
Photons, all energies	1
Electrons and muons, all energies <sup>3</sup>	1
Neutrons, energy < 10 keV	5
10 keV to 100 keV	10
> 100 keV to 2 MeV	20
> 2 MeV to 20 MeV	10
> 20 MeV	5
(See also Figure A-1)	
Protons, other than recoil protons, energy > 2 MeV	5
Alpha particles, fission fragments, heavy nuclei	20

<sup>1</sup> All values relate to the radiation incident on the body or, for internal sources, emitted from the source.

<sup>2</sup> The choice of values for other radiations is discussed in paragraph A14.

<sup>3</sup> Excluding Auger electrons emitted from nuclei bound to DNA (see paragraph A13).

(A12) To assist in providing consistency in calculations, a smooth fit to the  $w_R$  values for neutrons as a function of energy is given in Figure A-1 (page 83). The mathematical relationship is:

$$w_R = 5 + 17 e^{-\frac{1.67E}{1.67E+1}}$$

where  $E$  is the neutron energy in MeV. There is no intention to imply any biological meaning to this relationship. It is simply a calculational tool.

(A13) Auger electrons emitted from nuclei bound to DNA present a special problem because it is not realistic to average the absorbed dose over the whole mass of DNA as would be required by the present definition of equivalent dose. The effects of Auger electrons have to be assessed by the techniques of microdosimetry (see Annex B, paragraph B67).

(A14) For radiation types and energy which are not included in the table, an approximation of  $w_R$  can be obtained by calculation of  $\bar{Q}$  at a 10 mm depth in the ICRU sphere:

$$\bar{Q} = \frac{1}{D} \int_0^D Q(L) D(L) dL$$

where  $D(L) dL$  is the absorbed dose at 10 mm between linear energy transfer  $L$  and  $L+dL$ ; and  $Q(L)$  is the quality factor of  $L$  at 10 mm. The  $Q$ - $L$  relationships are given in paragraph A8. Figures A-2 (page 84) and A-3 (page 85) demonstrate the application of this formulation to photons and neutrons and can be seen to give values consistent with the recommended values of  $w_R$  in Table A-2. For this reason the Commission recommends this approach for radiations not included in the table.

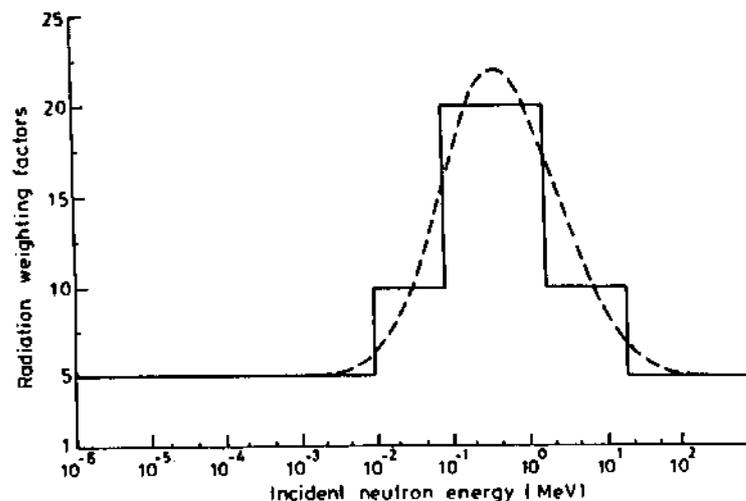


Fig. A-1. Radiation weighting factors for neutrons. The smooth curve is to be treated as an approximation.

### A.9. Equivalent Dose in an Organ or Tissue

(A15) In its previous recommendations, the Commission adopted the quantity dose equivalent at a point,  $H$ , to indicate the biological implications of radiation exposure at the levels of absorbed dose encountered in normal radiation protection. The Commission now recommends a new quantity derived from the absorbed dose averaged over a tissue or organ and named the equivalent dose. The equivalent dose,  $H_{T,R}$ , in tissue or organ T due to radiation R, is given by:

$$H_{T,R} = w_R \cdot D_{T,R}$$

where  $D_{T,R}$  is the average dose from radiation R in the tissue or organ T and  $w_R$  is the radiation weighting factor. Since  $w_R$  is dimensionless, the SI unit of equivalent dose is the same as for absorbed dose, namely  $J kg^{-1}$ , and its special name is sievert (Sv). The time derivative of the equivalent dose is the equivalent dose rate,  $\dot{H}_{T,R}$ .

(A16) When the radiation field is composed of types and energies with different values of  $w_R$ , the absorbed dose must be subdivided in blocks, multiplied by its own value of  $w_R$  and summed to determine the total equivalent dose i.e.:

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

where  $D_{T,R}$  is the average absorbed dose from radiation R in tissue T. Alternatively, the absorbed dose resulting from increments of energy between  $E$  and  $E+dE$  can be multiplied by the  $w_R$  values obtained from Table A-2, or as an approximation from the continuous function given in paragraph A12 illustrated in Figure A-1, and integrated over the energy spectrum to determine the total equivalent dose.

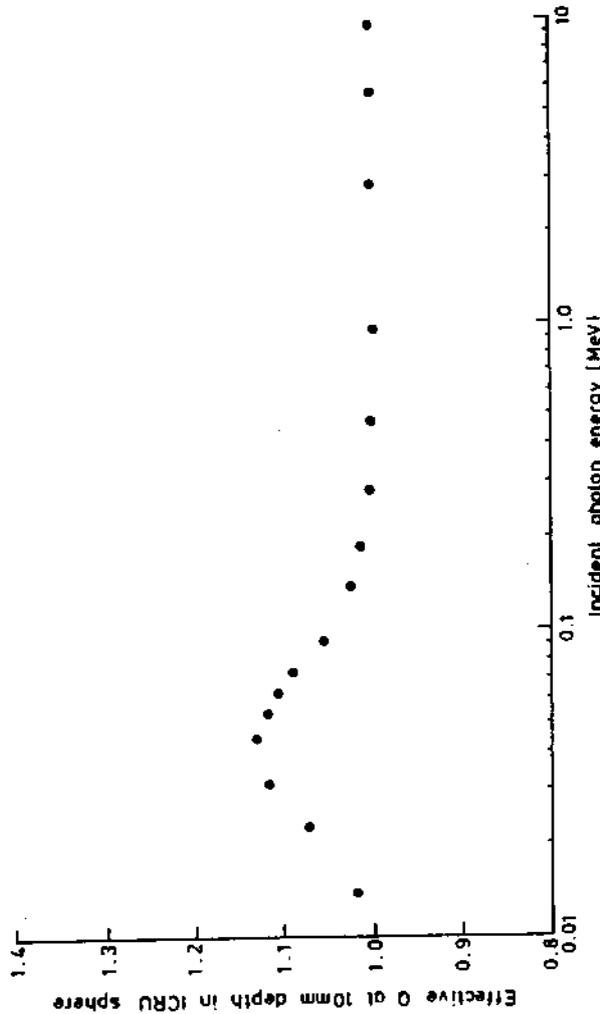


Fig. A-2. Effective  $Q_1(\bar{Q})$  as a function of photon energy. Reference: Dreaxler *et al.* (1990).

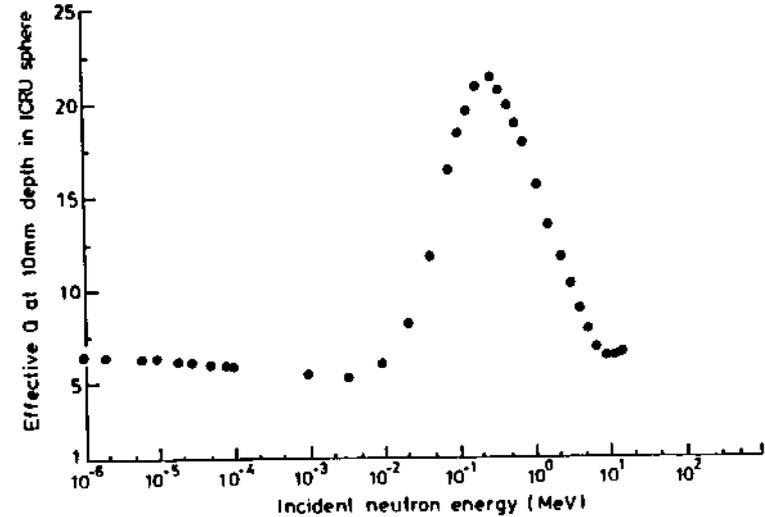


Fig. A-3. Effective  $Q_2(\bar{Q})$  as a function of neutron energy. Reference: Leuthold (1990).

#### A.10. Tissue Weighting Factors and Effective Dose

(A17) The relationship between the probability of stochastic effects and equivalent dose is found also to vary with the organ or tissue irradiated. It is, therefore, appropriate to define a further quantity, derived from equivalent dose, to indicate the combination of different doses to several different tissues in a way which is likely to correlate well with the total of the stochastic effects. The factor by which the equivalent dose in tissue or organ T is weighted is called the tissue weighting factor,  $w_T$ . The values of  $w_T$  are chosen so that a uniform equivalent dose over the whole body gives an effective dose numerically equal to that uniform equivalent dose. The sum of the tissue weighting factors is then unity. This weighted equivalent dose (a doubly weighted absorbed dose) has previously been called the effective dose equivalent but this name is unnecessarily complicated, especially in more complex combinations such as collective committed effective dose equivalent. The Commission has now decided to use the simpler name effective dose,  $E$ . The unit of effective dose is  $J kg^{-1}$ , with the special name sievert (Sv).

(A18) The effective dose,  $E$ , is the sum of the weighted equivalent doses in all the tissues and organs of the body. It is given by the expression:

$$E = \sum_T w_T \cdot H_T$$

where  $H_T$  is the equivalent dose in tissue or organ T and  $w_T$  is the weighting factor for tissue T.

Evidently:

$$E = \sum_R w_R \sum_T w_T \cdot D_{T,R} = \sum_T w_T \sum_R w_R \cdot D_{T,R}$$

where  $D_{T,R}$  is the mean absorbed dose in tissue or organ T delivered by radiation R. In both expressions the radiation is that incident on the body or emitted by a source within the body. The two forms of summation are clearly identical.

(A19) The recommended values for tissue weighting factors are given in Table A-3.

Table A-3. Tissue weighting factors<sup>1</sup>

Tissue or organ	Tissue weighting factor, $w_T$
Gonads	0.20
Bone marrow (red)	0.12
Colon	0.12
Lung	0.12
Stomach	0.12
Bladder	0.05
Breast	0.05
Liver	0.05
Oesophagus	0.05
Thyroid	0.05
Skin	0.01
Bone surface	0.01
Remainder	0.05 <sup>2,3</sup>

<sup>1</sup> The values have been developed from a reference population of equal numbers of both sexes and a wide range of ages. In the definition of effective dose they apply to workers, to the whole population, and to either sex.

<sup>2</sup> For purposes of calculation, the remainder is composed of the following additional tissues and organs: adrenals, brain, upper large intestine, small intestine, kidney, muscle, pancreas, spleen, thymus and uterus. The list includes organs which are likely to be selectively irradiated. Some organs in the list are known to be susceptible to cancer induction. If other tissues and organs subsequently become identified as having a significant risk of induced cancer they will then be included either with a specific  $w_T$  or in this additional list constituting the remainder. The latter may also include other tissues or organs selectively irradiated.

<sup>3</sup> In those exceptional cases in which a single one of the remainder tissues or organs receives an equivalent dose in excess of the highest dose in any of the twelve organs for which a weighting factor is specified, a weighting factor of 0.025 should be applied to that tissue or organ and a weighting factor of 0.025 to the average dose in the rest of the remainder as defined above.

#### A.11. Committed Tissue or Organ Equivalent Dose

(A20) Exposure to a radiation field of penetrating, externally applied radiation results in the simultaneous deposition of energy in a tissue. Tissue irradiation from incorporated radionuclides, however, is spread out in time, energy deposition occurring as the radionuclide decays. The time distribution of energy deposition will vary with the physico-chemical form of radionuclide, and its subsequent biokinetic behaviour. To take account of this time distribution, the Commission recommends the use of committed equivalent dose which is the time integral over time  $\tau$  of the equivalent-dose rate in a particular tissue that will be received by an individual following an intake of radioactive material.

When the period of integration  $\tau$  is not given, a period of 50 years is implied for adults or a period of 70 years for children.

(A21) The committed equivalent dose is defined by:

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

for a single intake of activity at time  $t_0$  where  $\dot{H}_T(t)$  is the relevant equivalent-dose rate in an organ or tissue T at time  $t$  and  $\tau$  is the time period over which the integration is performed. In specifying  $H_T(\tau)$ ,  $\tau$  is given in years.

#### A.12. Committed Effective Dose

(A22) If the committed organ or tissue equivalent doses resulting from an intake are multiplied by the appropriate weighting factors,  $w_T$ , and then summed, the result will be the committed effective dose.

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

In specifying  $E(\tau)$ ,  $\tau$  is given in the number of years over which the integration is made. The dose commitment ( $H_{c,T}$  or  $E_c$ ) is a calculational tool. It can be assessed for a critical group as well as for the whole world population. It is defined as the infinite time integral of the per caput dose rate ( $\dot{H}_T$  or  $\dot{E}$ ) due to a specified event, such as a unit of practice (e.g. a year of practice):

$$H_{c,T} = \int_0^{\infty} \dot{H}_T(t) dt$$

or

$$E_c = \int_0^{\infty} \dot{E}(t) dt$$

In the case of an indefinite practice at a constant rate, the maximum annual per caput dose rate ( $\dot{H}_T$  or  $\dot{E}$ ) in the future for the specified population will be equal to the dose commitment of one year of practice, irrespective of changes in the population size. If the practice is continued only over a time period,  $\tau$ , the maximum future annual per caput dose will be equal to the corresponding *truncated* dose commitment, defined as

$$H_{c,T}(\tau) = \int_0^{\tau} \dot{H}_T(t) dt$$

or

$$E_c(\tau) = \int_0^{\tau} \dot{E}(t) dt.$$

#### A.13. Activity

(A23) The activity,  $A$ , of an amount of radioactive nuclide in a particular energy state at a given time is the quotient of  $dN$  by  $dt$ , where  $dN$  is the expectation value of the number of spontaneous nuclear transitions from that energy state in the time interval  $dt$ .

$$A = \frac{dN}{dt}$$

The unit of activity is the reciprocal second,  $s^{-1}$ , with the special name becquerel (Bq).

#### A.14. ICRU Quantities for Environmental and Individual Monitoring

(A24) The use of the ICRU quantities as given in *ICRU Report 39* (ICRU, 1985) are expected to give reasonable approximations of the effective dose and the equivalent dose to the skin when these quantities are calculated using the  $Q$ - $L$  relationship given in Table A-1. The Commission will be examining these dosimetric quantities in detail as part of a general revision of *ICRP Publication 51* (ICRP, 1987) which will incorporate the new radiation weighting factors.

(A25) It has been convenient to consider the determination of quantities related to the effective dose equivalent and to the dose equivalent in the skin. This has been done separately for environmental (including area) and individual monitoring. For such monitoring purposes, certain conventions have been used. All these quantities are based on the concept of the dose equivalent at a point in the ICRU sphere.

(A26) In defining the quantities associated with these concepts, it is useful to stipulate certain radiation fields that are derived from the actual radiation field. The terms "expanded" and "aligned" are given in *ICRU Report 39* (ICRU, 1985) to characterise these derived radiation fields. In the expanded field, the influence and its angular and energy distribution have the same values throughout the volume of interest as the actual field at the point of reference. In the aligned and expanded field the fluence and its energy distribution are the same as in the expanded field but the influence is unidirectional.

##### A.14.1. Environmental monitoring

(A27) Two concepts linking the external radiation field to the effective dose, and to the equivalent dose in the skin, are introduced here for purposes of environmental and area monitoring. The first of these concepts, the **ambient dose equivalent**,  $H^*(d)$ , is appropriate for strongly penetrating radiation, and the second, the **directional dose equivalent**,  $H'(d)$ , is suitable for weakly penetrating radiation.

(A28) The ambient dose equivalent,  $H^*(d)$ , at a point in a radiation field, is the dose equivalent that would be produced by the corresponding aligned and expanded field, in the ICRU sphere at a depth,  $d$ , on the radius opposing the direction of the aligned field.

(A29) The directional dose equivalent,  $H'(d)$ , at a point in a radiation field, is the dose equivalent that would be produced by the corresponding expanded field in the ICRU sphere at depth,  $d$ , on a radius in a specified direction.

##### A.14.2. Individual monitoring

(A30) Two concepts are introduced for purposes of individual monitoring. The first of these concepts, the **individual dose equivalent, penetrating**,  $H_p(d)$ , is appropriate for organs and tissues deeply situated in the body which will be irradiated by strongly penetrating radiation, and the second, the **individual dose equivalent, superficial**,  $H_s(d)$ , is suitable for superficial organs and tissues which will be irradiated by both weakly and strongly penetrating radiation.

(A31) The individual dose equivalent, penetrating,  $H_p(d)$ , is the dose equivalent in soft tissue, defined as in the ICRU sphere below a specified point on the body at depth,  $d$ , that is appropriate for strongly penetrating radiation.

(A32) The individual dose equivalent, superficial,  $H_s(d)$ , is the dose equivalent in soft tissue below a specified point on the body at a depth,  $d$ , that is appropriate for weakly penetrating radiation.

#### A.15. Collective Equivalent Dose

(A33) The Commission has defined a quantity to express the total radiation exposure of a specific tissue or organ in a group of individuals. The quantity defined by the Commission as the collective equivalent dose in tissue  $T$ , is given by

$$S_T = \int_0^{\infty} H_T \cdot \frac{dN}{dH_T} dH_T$$

where  $(dN/dH_T)dH_T$  is the number of individuals receiving an equivalent dose between  $H_T$  and  $H_T + dH_T$ ; or by

$$S_T = \sum_i \bar{H}_{T,i} \cdot N_i$$

where  $N_i$  is the number of individuals in population subgroup  $i$  receiving mean organ equivalent dose,  $\bar{H}_{T,i}$ . The collective equivalent dose can be subdivided into compartments in which the individual doses lie within specified ranges.

#### A.16. Collective Effective Dose

(A34) If a measure of the radiation exposure in a population is desired, the collective effective dose can be calculated. This quantity has been defined by the Commission as follows:

$$S = \int_0^{\infty} E \cdot \frac{dN}{dE} dE \quad \text{or} \quad \sum_i \bar{E}_i \cdot N_i$$

where  $\bar{E}_i$  is the mean effective dose to population subgroup  $i$ .

(A35) Neither the definition of collective equivalent dose nor the collective effective dose explicitly specify the time over which the dose is delivered. Therefore, the time period and population over which the collective equivalent dose is summed or integrated should be specified.

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**ANNEX B**  
**BIOLOGICAL EFFECTS OF IONISING RADIATIONS**

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### B.1. Introduction

(B1) Not long after the discovery of x rays in 1895 and of natural radioactivity in 1896, clinical evidence, mainly from effects on the skin, indicated that ionising radiation is harmful to human tissues. Later it was realised that not only is ionising radiation damaging to most tissues but exposure of the germinal tissue in plants and animals was found to result in effects in the descendants as well. During almost a century of exploring the uses of ionising radiation, extensive studies of radiation effects on living species have taken place. These explorations and studies received an enormous impetus following the discovery of nuclear fission in 1939 and the subsequent uses, some military, to which fission energy was quickly put. It became evident that human beings must study the biological effects of ionising radiation in order to protect themselves and other species from its harmful effects while at the same time maximising the benefits of its use.

(B2) Diverse studies in many laboratories throughout the world, while by no means complete, have resulted in a wealth of information concerning the biological effects of radiation, possibly greater than that associated with any other environmental hazard. For radiation protection, concerns pertain to two types of effect. The first type, nonstochastic

effects of radiation, now called deterministic effects, involve the malfunctioning or loss of function of tissues in organs due mainly to cell loss. These effects result from high dose exposures and for them there is a threshold. The second type, stochastic effects, express themselves long after the exposure and include increased risk of cancer and, by implication from studies on animals, of hereditary disorders. These stochastic effects appear to have no threshold and may occur after low radiation doses (small fractions of a gray) even though their frequency is then low.

(B3) Deterministic effects are avoided in normal radiation protection procedures by limiting doses to below the threshold dose levels for these effects. Deterministic effects are relevant in accidents and they are also observed in healthy tissues unavoidably irradiated during radiotherapy. Stochastic effects can be reduced in frequency by lowering the dose but cannot be avoided entirely since they are assumed to occur with low frequency even at low doses. On this basis it may be assumed that these effects may be induced by the natural radiation to which we are all exposed and by additional small doses from man-made sources used in society. In this Annex, the subject of biological effects will be treated broadly in relation to radiation protection, describing deterministic effects from high doses at high dose rates as well as the probability of cancer and hereditary disorders, and the special problems associated with the exposure of the embryo and fetus, occurring also after low doses. Many biological effects of radiation such as effects in radiation therapy and the late deterministic effects in specific tissues, such as fibrosis, which may result from such treatment, are not discussed further.

(B4) In the last few decades the risk of cancer has emerged as the primary effect of concern at low doses and, therefore, much of the attention of this Annex will be focused on the probability of cancer induction.

(B5) Note that throughout this text the term probability is used rather than the frequently used term "risk". The term risk is used only in its more general sense as a concept rather than a quantity, roughly equivalent to "hazard" (see Annex C).

### B.2. Interaction between Radiation and Matter

(B6) Matter achieves its extraordinary diversity on earth by being made up of many different molecular species in which the component atoms may be combined in a wide variety of ways. Individual atoms can be thought of as consisting of a positively charged nucleus surrounded by negatively charged electrons. The nucleus in turn is made up of protons (positively charged) and neutrons (electrically neutral); the number of protons determines the nature of the atom and the number of neutrons determines the particular isotope. While many nuclides in nature are stable (depending on their ratio of neutrons to protons) and maintain their form and composition indefinitely, many others are unstable. These unstable nuclides return to stability by the emission of a charged particle (alpha particle, beta particle or positron) from the nucleus at a defined, characteristic rate. They are then called radioactive nuclides or simply radionuclides. The decay rate of a radionuclide is characteristic of that radionuclide and is described by its half-life. Half-lives range from fractions of a second to billions of years. Many different radionuclides exist naturally especially among atoms of high atomic number. The new nucleus formed by the emission of a particle may still be radioactive and emit further particles or may be in an excited state and may return to stability by emitting further radiation (gamma radiation) which leaves the nucleus stable but does not alter its composition.

### B.2.1. Ionisation and ionising radiations

(B7) Atoms can be "ionised" by a variety of interactions which result in an electron being removed from the atom, thus creating an ion pair. An ion pair consists of the removed electron (which may quickly attach itself to another atom to form a negative ion) and the residual nucleus with its complement of remaining electrons constituting a positive ion.

(B8) Ionising radiations are radiations that are capable of causing ionisation in the atoms of any medium through which they may pass. They consist either of high velocity charged particles (e.g. alpha particles, beta particles) which may be emitted from radionuclides or which may arise secondarily when indirectly ionising radiations such as x rays (generated artificially), gamma rays (from nuclear transitions) or neutrons expel them from the atoms of the medium. These secondary charged particles (usually electrons or protons) then cause further ionisation or excitation in the same way as do primary charged particles. The processes by which photons (x and gamma rays) eject electrons from atoms include the photoelectric effect, the Compton effect and pair production. The relative contributions of these processes depend on the energy of the photons and on the properties of the medium through which they pass. These processes are well documented, as also are those by which neutrons eject protons and other particles from nuclei and cause these nuclei to recoil.

### B.2.2. Interaction between radiation and matter

(B9) When ionising radiations traverse a medium, the resulting electrical interactions are random and follow the often haphazard tracks of the charged particles (primary or secondary) bouncing from one interaction event to another as they pass through the medium. At low doses most of the atoms of a medium will be unaffected by the radiation while a small number are ionised or excited. Excitation is a process by which energy is transferred to the atom from the radiation and raises the energy level of the atom to an excited state but does not ionise it. This excitation energy can also cause effects in the medium but these are generally different and considered to be less important than the effects of ionisation.

(B10) Each of the interaction events described above involves the transfer of a small amount of energy from the radiation to the medium which in the case of low LET radiations, are usually in packets of about 100 electron volts (eV) or less. (This includes not only the energy deposited by ionisations but also by excitation.) These transfers occur in a very short time ( $< 10^{-16}$  seconds) but may be broadly distributed spatially in a discontinuous fashion throughout the medium along the tracks of the charged particles. The microdistribution of the ionisations and excitations produced by ionising radiation depends on the type and energy of the incident radiation. While it cannot be fully quantified at present, approximations are used in microdosimetry to represent the microdistribution of energy. For example, the average energy deposited along the track of the particle per unit length depends on the type of particle and its energy and is called the linear energy transfer (LET) of the particle. Thus, a sparsely ionising radiation producing few events per micron of track, is known as a "low-LET" radiation (e.g., x or gamma rays) whereas radiations producing dense ionisations along the track are known as "high LET" radiation (e.g., alpha particles, protons and recoil nuclei from neutrons, heavy ions) (Figure B-1). The actual energy lost by a charged particle is subject to random fluctuations and the energy deposited by the particle in passing through a spherical site of

### Tracks in chromatin fibre

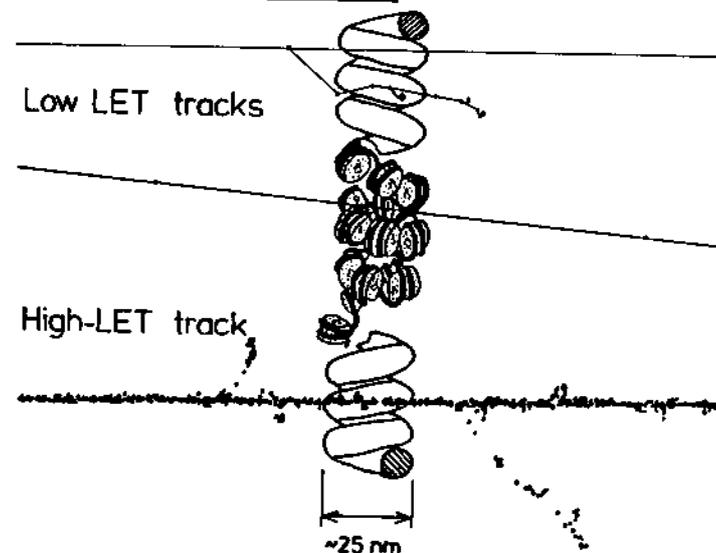


Fig. B-1. Diagram of high and low LET tracks passing through a section of chromatin (a mixture of DNA and protein).

specific diameter in the medium determines the lineal energy  $y$  (ICRU, 1983), which may differ substantially from the LET.

(B11) The energy transferred to an atom or to a small volume of medium, such as a biological target or cell, is not the same for all atoms and targets. It has an average value and a distribution of values about this average. The average energy transferred per unit mass of medium is the absorbed dose. The effects the radiation will have on the medium are related to the amount of energy transferred, i.e., the absorbed dose, but they also depend on the microdistribution of energy i.e., the type of radiation.

(B12) The transfers of energy give rise to further physico-chemical processes such as the induction of free radicals (which may occur in  $\sim 10^{-12}$  seconds). These can move rapidly in the medium some distance from the site of the original event and cause further chemical changes in the molecules of the medium before they are inactivated (in times of the order of  $10^{-6}$  seconds or less). Molecular changes reflecting breakage of chemical bonds can manifest themselves over various periods of time and in a variety of ways depending on the nature of the medium. The changes are of special interest in the tissues of living organisms.

### B.2.3. Biological structure and function

(B13) The basic unit of the living organism is the cell, its nucleus containing coded, genetic information in nuclear DNA that is capable of providing instructions for cellular

reproduction and for intracellular protein synthesis. Other cellular structures (organelles) ensure that protein and energy are produced. A "milieu intérieur" is maintained within the cell and in relation to its extracellular environment, aided by a complex system of semi-permeable membranes surrounding the organelles. These membranes regulate movement of water, nutrients and electrolytes in and out of the cell. Any disturbance of this equilibrium can threaten the cell's viability but the cell has evolved an elaborate system of repair processes, particularly for damage within the nuclear DNA.

(B14) In higher organisms, cells are organised into tissues and organs with specialist roles such as energy production and storage, muscular activity for locomotion, digestion of food and excretion of waste products and oxygen supply. The organism relies upon its nervous and endocrine systems to co-ordinate these body activities. The magnitude of the effect of insults in tissues and organs from noxious agents is influenced by the particular tissue and also the ability to compensate for and repair damage. This ability is dependent upon age at exposure, the health status, sex and genetic predisposition of the individual. Thus, it is not surprising that there is a variation in response amongst individuals in a population exposed to deleterious environmental factors, of which ionising radiation is but one.

#### B.2.4. DNA damage and repair

(B15) Important biological structures can be altered either directly by the disruption caused by ionisation (or perhaps excitation, although this is much less likely) or indirectly by the further changes (such as free radical induction) set in motion by transfers of energy to the medium. The random distribution of energy absorption events produced by radiation may damage vital parts of the double-stranded DNA or other important macromolecules of cells in several ways. Direct effects occur in the DNA in the form of single-strand or double-strand breaks in the molecule. Other effects include a variety of recombinational changes as well as cross-links, alterations in sugar and base fractions, base substitutions, deletions etc. Chromosomal aberrations are a result of DNA damage. These changes can be measured quantitatively as a function of the absorbed dose.

(B16) There is substantial evidence that DNA is a principal target in the irradiated cell. Many of the acute effects observed in the intact organism are mediated through the death of cells when they attempt to divide and can no longer multiply—so-called cell reproductive death. In order to deal with the initial DNA damage that gives rise to these changes, cells have evolved complex, enzyme-mediated repair systems. These are specific for different molecular forms of DNA damage whereby lesions induced in DNA by ionising radiation, ultraviolet and chemical agents are identified and removed, often within a timescale of tens of minutes. When a single strand break occurs, the site of damage is identified and the break easily repaired by simply annealing of the broken ends. If base damage occurs on the single strand, enzymatic excision occurs and the intact complementary strand of the molecule provides a template upon which to reconstruct the bases in the correct sequence. Such induced damage may be removed with high fidelity, returning the DNA structure to its original form (error-free repair). In these circumstances there is no long term cellular consequence of that lesion. Alternatively repair processes may be error-prone in so far as overall DNA integrity is retained but results in small base sequence changes (point mutations) at the site of the initial lesions or more gross changes such as gene deletions or rearrangements (Friberg and Hanawalt, 1988). These misrepair events, if they occur in important regions of DNA, may have long

term consequences for the cell and can result in cell reproductive death or stable genetic changes in surviving cells.

(B17) Double strand breaks may also be repaired by simple annealing, but the consequences are much more serious if base damage occurs simultaneously on both strands. This is because there is no longer an available template for reconstructing the base sequence on either strand. The outcome could be cell reproductive death, or misrepair reflected in a point mutation or more extensive gene deletion. Increased frequency of misrepair of DNA double strand breaks has been observed in radiation-sensitive strains of cultured mammalian cells known to be deficient in DNA repair enzymes and there is also evidence that the fidelity of DNA repair may be a major factor that determines response to variable dose rate and radiation quality (Debenham *et al.*, 1987).

(B18) These molecular changes in DNA are presumably related (although this is not well understood) to the later forms of biological damage (stochastic and deterministic) manifest as observed effects in living organisms.

#### B.2.5. Cell killing

(B19) The killing of somatic cells, resulting from irreparable damage to vital cell structures such as the chromosomes, often becomes manifest in rapidly dividing cell populations a few hours or days after exposure. In slowly dividing cell populations, death may not occur for months or even years. The degree of killing of cells in a population increases with dose. If enough cells are killed in an organ or tissue the function of the organ or tissue is impaired. In extreme cases the organism itself may die. These effects constitute what are defined as deterministic (formerly nonstochastic (ICRP, 1979)) effects.

(B20) Cell killing is not the only process that can lead to alterations in the behaviour and function of organs and tissues. Functional disorders can also result from direct alteration of other cellular processes such as membrane permeability and cell to cell communication.

#### B.2.6. Cell modification

(B21) A second process, taking place in much longer overall times, is the modification of a normal cell, presumably the result of specific molecular DNA changes by a process known as neoplastic transformation. Such changes can be induced by various agents including radiation. One characteristic result of this change is the potential capability of the transformed cells for unlimited cellular proliferation. This change alone does not constitute "malignant" transformation (i.e. the ability of the cells to multiply and form tumours when injected into recipient animals) since other phenotypic changes occur in malignant transformation as well. They are recognised, for example, by the altered behaviour of cells in cell-cell interactions and the invasion of neighbouring tissues and metastasis to distant sites in living organisms. Currently it is believed that the multiple changes that occur in the development of a cancer proceed in sequential stages. The initial events in the genome and the production of a cell or cells with the potential to develop into a cancer are known as initiation. Both endogenous and exogenous factors may influence expression of the initial event. The initiated cell(s) must undergo further changes, usually after a long time and possibly after stimulation by a promoting substance, before becoming a cell with malignant potential. (In at least one theory this promoted cell would be described as a "precancerous" cell and a further step, conversion, would be required before the cell became "cancerous".) Thereafter the division

and multiplication of this cell gives rise to an occult tumour in the stage known as progression. The carcinogenic process including the growth of a primary cancer to a detectable size (e.g. about 1 cm diameter and containing billions of cells) and its spread to other tissues can take months in small animals and many years in humans. The interval between exposure and the detection of a radiation-induced cancer is referred to as the latency period. This period varies with the type of cancer and the age at exposure.

(B22) Changes in the genome, compatible with continued cell division may also take place in the germinal cells of the reproductive tissues. They result in a variety of transmissible lesions, most often deleterious, which are passed on to and may be manifest as hereditary disorders in succeeding generations.

#### B.2.7. Tissue response to cell modification

(B23) Most neoplastic cell transformations do not progress to a cancer. This is thought to be due to a combination of circumstances

- virtually no unrepaired cells remain viable after more than a few divisions
- those capable of several divisions are frequently "programmed" to differentiate into non-dividing functional cells
- the required sequence of promotion and progression events in the cell's environment does not occur
- host defence mechanisms (e.g. competent immuno-surveillance, natural killer cell activity) exist to prevent selective cloning.

#### B.2.8. Definition of stochastic and deterministic effects

(B24) The deposition of energy by ionising radiation is a random process. Therefore even at very low doses it is possible that sufficient energy may be deposited into a critical volume within a cell to result in its modification or even its death. Death of one or a small number of cells will, in most cases, have no consequences in tissue, but modifications in single cells such as genetic changes or transformations leading ultimately to malignancy (see paragraph B21) may have serious consequences. These effects have been termed stochastic. There is a finite probability for the occurrence of such stochastic events even at very small doses, so unless all such events can be repaired up to some level of dose there can be no threshold. As the dose is increased the frequency of such events increases, but in the absence of other modifying factors, the severity of the resultant changes is not expected to increase, in contrast to the case for deterministic effects (see paragraph B25 and Figure B-3).

(B25) With larger doses, there may be a substantial degree of cell killing, sufficient to result in detectable tissue changes. Although other mechanisms may also be involved, cell killing plays a crucial role in the pathogenesis of tissue injury. Hence the response of tissues *in vivo* is determined by the characteristics of cell survival. For any defined nonstochastic injury, a given proportion of cells must be killed in order to reach the level of detection. This constitutes a threshold, the magnitude of which will, of course, depend on the chosen level of injury. This is illustrated in Figure B-3. The pathogenesis of some types of injury, however, later remains unresolved, e.g., mental retardation and lens opacification. These have been regarded as deterministic effects and hence subject to a threshold. Such a conclusion cannot *a priori* be justified without knowledge of the mechanisms leading to the observable defects. A detailed discussion of deterministic effects is found in *Publication 41* (ICRP, 1984a).

(B26) Since cell killing by irradiation is itself a stochastic process, the term non-stochastic for injury resulting from the death of a large number of cells is now considered to be unsuitable. It has been replaced in this report by the term deterministic, meaning "causally determined by preceding events" which is considered to be more appropriate.

### B.3. Deterministic Effects

(B27) Deterministic effects in humans can result from general or localised tissue irradiation causing an amount of cell killing that cannot be compensated for by proliferation of viable cells. The resulting loss of cells can cause severe and clinically detectable impairment of function in a tissue or organ. Thus, the severity of the observed effect can be expected to depend on the dose. There will be a threshold below which the loss of cells is too small to detectably impair tissue or organ function. In addition to cell killing, radiation can damage tissues in other ways: by interfering with a variety of tissue functions including regulation of cellular components, inflammatory reactions involving modifications in permeability of cells and tissues, natural migration of cells in developing organs, and indirect functional effects (e.g., irradiation of the pituitary gland influencing endocrine function in other tissues). All of these play a part in the severity of deterministic effects.

#### B.3.1. Cell killing and *in vitro* survival curves

(B28) Cell killing is the main but not the only process involved in deterministic effects. Unless the dose is high (many gray), most types of cells are not usually killed immediately after exposure but may continue to function until they attempt to divide. The attempt may then fail, probably because of severe chromosome damage and the cell will die. While individual cell death in a tissue may be considered as a random (i.e. stochastic) effect, the composite effect of killing a high proportion of cells in a tissue, or other forms of damage, is deterministic. Studies of cultured mammalian cells demonstrate that cell survival varies as a function of dose which may be described by "survival curves", typified by those given in Figure B-2. For densely ionising radiation (high LET) the dose-response curve may be exponential, i.e. linear on a semi-logarithmic plot (Figure B-2A). It may be characterised by one parameter, the slope, which is usually represented by its reciprocal, the dose ( $D_0$ ) required to reduce survival to 37%. For sparsely ionising radiation (low LET) such as x rays, the dose response (Figure B-2A) usually has an initial shoulder, followed by a portion which is straight, or almost straight on a semi-logarithmic plot. The curve is characterised by any two of three parameters:  $D_0$ , the dose required to reduce survival to 37% on the exponential part of the curve, i.e., the reciprocal slope of the straight portion of the curve; the extrapolation number,  $n$ , as shown; and  $D_q$ , the quasi-threshold dose, being the intercept of the straight line portion of the curve on the dose axis (Figure B-2A).

(B29) Several equations have been used to describe the shapes of survival curves.

(a) The curve illustrated in Figure B-2A for densely ionising (high LET) radiation is given by

$$S = e^{-D/D_0}$$

where  $S$  = survival

$D$  = dose

$D_0$  = dose at 37% survival or the reciprocal of the slope.

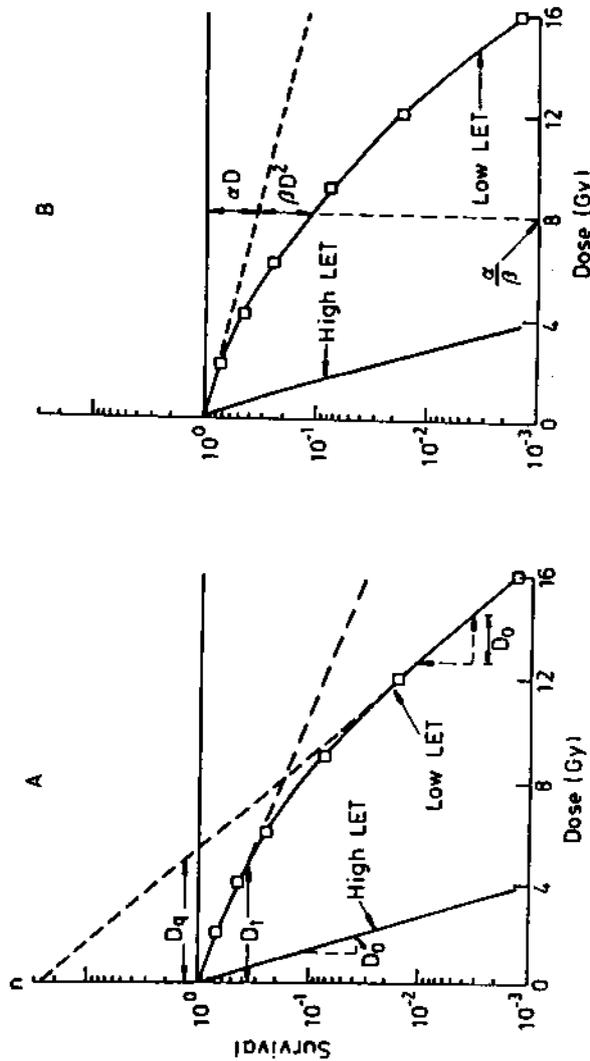


Fig. B-2. Survival curves for mammalian cells exposed to high LET and low LET ionising radiation at high dose rates ( $>0.1$  Gy min $^{-1}$ ). The fraction of surviving cells is plotted on a logarithmic scale against dose on a linear scale.

- (b) The curve illustrated in Figure B-2A for sparsely ionising (low LET radiation) is given by the survival,

$$S = 1 - (1 - e^{-D/D_0})^n$$

where "n" is the extrapolation number at zero dose.

$D_0$  is the reciprocal slope of the exponential portion of the curve.

Typically, for mammalian cells, and for low LET radiation, n is in the range 2-20 (somewhat less than that shown in the figure) and  $D_0$  is in the range 1-2 Gy.

A more complex expression is required to describe the initial slope of the curve which may be given by:

$$S = e^{-D/D_1} [1 - (1 - e^{-D/D_2})^n]$$

where  $D_1$  is the reciprocal of the initial slope of the curve.

- (c) The initial region between 0 and 5 Gy (and often over a broader dose range) can be better described in many biological systems by what is known as the linear-quadratic equation based on the average frequency ( $F$ ) of lethal events

$$F(D) = \alpha D + \beta D^2$$

and the survival ( $S$ ) by

$$S = e^{-(\alpha D + \beta D^2)}$$

This is shown in Figure B-2B.

$\alpha$ , the linear coefficient, may range between  $1 \times 10^{-1}$  and  $5 \times 10^{-1}$  Gy $^{-1}$  and  $\beta$ , the quadratic coefficient between  $1 \times 10^{-1}$  and  $5 \times 10^{-2}$  Gy $^{-2}$ ,  $\alpha/\beta$  being in the range of 1 Gy to 10 Gy (Hall, 1988).

(B30) The initial increase in slope with increasing dose in the survival curve for low LET radiation has been interpreted as demonstrating that cells require to accumulate a certain number of damaging events within a short time in order for the cumulative effect to be lethal to that cell. If time elapses between exposures and thus between events, repair of "sublethal" damage can occur and more radiation will be required to kill the same number of cells. This repair was demonstrated in experiments in mammalian cells involving two doses of radiation separated by intervals of time (Elkind and Sutton, 1960). It is also consistent with the observation that dose rates in excess of 0.1 Gy/min of low LET radiation cause the maximum effect and lower dose rates result in progressively less cell killing until a dose rate of about 0.1 Gy/h or less is reached for mammalian cells (Hall and Bedford, 1964).

(B31) Such quantifiable biological endpoints (typical of cells in culture) are suitable to examine the modifying effects of radiations of high versus low LET and high, versus low dose rate as well as cell modifiers (sensitisers and protective agents) which markedly alter the effectiveness of the radiation (Sinclair, 1969).

### B.3.2. Cell killing and deterministic responses in tissues and organs

(B32) Just as for cells grown in culture, tissues and organs in the body can be impaired by radiation as a result of cell killing and various non-lethal effects (Hewitt and Wilson, 1959; McCullough and Till, 1962; Withers and Elkind, 1970), but in intact tissues there are additional factors. Proliferating cells in a healthy tissue are in dynamic equilibrium and this equilibrium is disturbed by irradiation. Cells vary in sensitivity to cell killing, division delay and other progression changes during the cell cycle (Sinclair,

1968). Consequently the surviving population will not only initially consist mainly of resistant cells but the distribution of cells at each cell cycle stage is modified. At the same time, while the damage in some cells is being repaired, other undamaged cells will repopulate the tissue. Eventually, if the dose is not too large, the tissue should recover completely with virtually intact functional integrity. These changes are dependent on the dose rate at which the dose is delivered.

(B33) Tissues vary in their response to ionising radiation (ICRP, 1984a). Among the most radiosensitive tissues are the ovary and testes, bone marrow and the lens of the eye. In general, the dose-frequency relationship for these tissues will be sigmoid in shape when plotted on linear axes, the effect becoming more frequent as the dose increases. Deterministic effects vary with the dose in severity as well as frequency. The upper panel in Figure B-3 illustrates how the frequency of a particular deterministic effect, defined as a clinically recognisable pathological condition, increases as a function of dose in a population of individuals of varying susceptibilities. The lower panel in Figure B-3 represents the dose-severity relationship for a population of mixed sensitivity. For simplicity, three levels of radiosensitivity are shown in curves a, b, c. The severity of the pathological effect increases most markedly in those individuals in a subgroup who are most susceptible (curve a), reaching the threshold of detectability at a lower dose than in less susceptible subgroups (curves b and c). The range of doses over which the different subgroups cross the same threshold of severity is reflected in the upper panel, which shows the frequency of the pathological condition in a population (i.e. all subgroups), and which reaches 100 per cent only at that dose which is sufficient to exceed the defined threshold of severity in all members of the population.

(B34) Threshold doses for some deterministic effects in the more radiosensitive tissues in the body are as shown in Table B-1. Several formulations describe this change

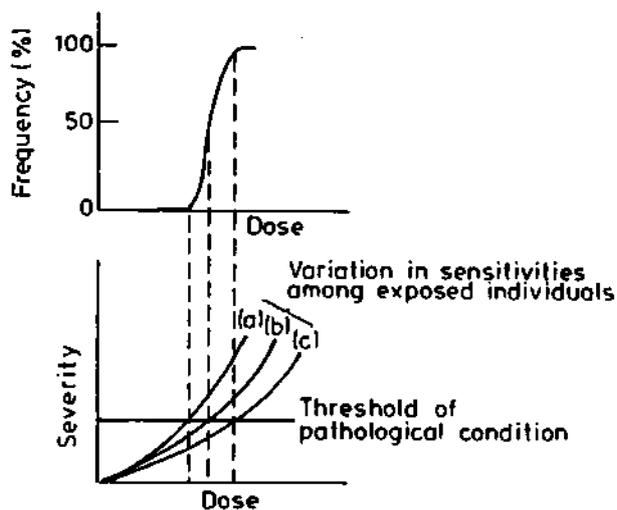


Fig. B-3. Typical dose-effect relationships for deterministic effects expressed in a population (ICRP, 1984a).

Table B-1. Estimates of the thresholds for deterministic effects in the adult human testes, ovaries, lens and bone marrow (from ICRP, 1984a)<sup>1</sup>

Tissue and effect	Threshold		
	Total dose equivalent received in a single brief exposure (Sv)	Total dose equivalent received in highly fractionated or protracted exposures (Sv)	Annual dose rate if received yearly in highly fractionated or protracted exposures for many years (Sv y <sup>-1</sup> )
Testes			
Temporary sterility	0.15	NA <sup>2</sup>	0.4
Permanent sterility	3.5-6.0 <sup>3</sup>	NA	2.0
Ovaries			
Sterility	2.5-6.0	6.0	> 0.2
Lens			
Detectable opacities	0.5-2.0 <sup>4</sup>	5	> 0.1
Visual impairment (cataract)	5.0 <sup>5</sup>	> 8	> 0.15
Bone marrow			
Depression of hematopoiesis	0.5	NA	> 0.4

<sup>1</sup> For further details consult *Publication 41* (ICRP, 1984a).

<sup>2</sup> NA denotes Not Applicable, since the threshold is dependent on dose rate rather than on total dose.

<sup>3</sup> See UNSCEAR, 1988a.

<sup>4</sup> See also Otake and Schull, 1990.

<sup>5</sup> Given as 2-10 Sv (NCRP, 1989a).

Except as noted in footnotes (3, 4, 5) the values in Table B-1 represent current threshold values expressed as equivalent dose.

with the time pattern of the exposure. For the case of dose rate varying with time, a formula (Kirk *et al.*, 1972) has been used in practice to assess the "instantaneous equivalent dose", i.e. the short-time dose producing the same tissue effects as the exposure under consideration (Walinder, 1981). A special important case is the internal exposure at one ALI every year, where the Kirk formula shows that no deterministic threshold is exceeded during and after a working lifetime. It is clear that in general fractionation or protraction of the exposure raises the threshold value. Details on other tissues are available in *Publication 41* (ICRP, 1984a) and other publications (UNSCEAR, 1982; NUREG, 1989).

(B35) Tissues typified by bone marrow have rapidly dividing progenitor (stem) cells and harm is manifest as an early effect, whereas tissues typified by liver have low rates of cell renewal and harm is expressed as a late effect when cells divide. With regard to the mechanism of deterministic damage in selected tissues, Michalowski (1981) and coworkers (Wheldon *et al.*, 1982) have classified tissues into two main types: those containing stem cells that divide and proceed through several stages of division and maturation before they finally become functional (e.g. hematopoietic tissue); and those containing functional cells that are capable of dividing upon demand (e.g. liver parenchyma). Radiation injury develops by different pathways in these tissues because the tissues are organised differently. Alternative models for proliferation in normal tissues and their response to irradiation are described (Wheldon and Michalowski, 1986).

(B36) As an example of a specific deterministic effect, for skin, the threshold for erythema and dry desquamation is about 3-5 Gy, the symptoms appearing after about 3 weeks. Moist desquamation occurs after about 20 Gy, blistering appearing after about 4 weeks. Cell death in the epidermal and dermal layers resulting in tissue necrosis occurs

after a dose of about 50 Gy appearing after about 3 weeks. (To be published as *Publication 59* (ICRP, in preparation).)

(B37) Much new information on deterministic effects is beginning to emerge from the unfortunate experiences during the accident at Chernobyl. These include cytogenetic studies of the doses received by those in the most highly exposed group (Pyatkin *et al.*, 1989) hematological effects (Guskova and Baranov, 1989) and skin effects (Barabonova and Osanov, 1990). Other studies will also come forward and may in the future contribute to our knowledge of the threshold dose values for deterministic effects.

### B.3.3. Death after whole body exposure

(B38) Acute radiation exposure may be so severe in certain unforeseen circumstances that death may result in individual members of a species, including human beings. Death is generally the result of severe cell depletion in one or more vital organ systems in the body, therefore the dose-response relationship, as observed in cellular studies, is in general relevant. The plot on linear axes of the probability of harm against dose is sigmoid in shape (Figure B-4a) while for a probability-linear plot the shape is approximately linear (Figure B-4b).

(B39) In applying this dose-response relationship to predict lethality in an exposed human population, and using the limited human experience of accidental and therapeutic exposure, no individuals would be expected to die at doses below about 1 Gy; then as the dose increases more individuals die until finally, as the dose increases further, all are killed (Figure B-4a). The survival-dose relationship is often described by its midpoint, the  $LD_{50/60}$ , i.e. the dose at which half the individuals would be expected to die in 60 days. Values for the  $LD_{5/60}$  and the  $LD_{95/60}$  are more useful end points in helping to establish the slope of the dose-survival relationship and because of their practical value in protection situations. For a healthy adult human, the  $LD_{50/60}$  after acute exposure is estimated to be between 3 and 5 Gy midline dose (which approximates to the marrow dose for low LET, penetrating radiations such as 1 MeV gamma rays) and the cause of death at this dose is loss of bone marrow function due to the loss of bone marrow stem cells. It is possible to improve the chances of survival of individuals exposed to doses approximating or greater than the  $LD_{50/60}$  by stimulating viable bone marrow stem cells or by substituting new isologous marrow or concentrates of bone marrow stem cells from a suitable donor, together with appropriate medical care (fluid replacement, antibiotics,

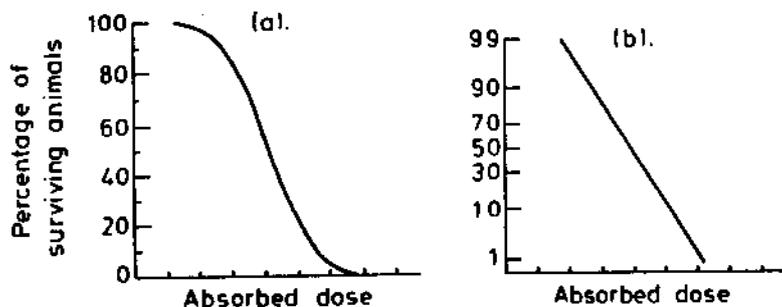


Fig. B-4. Typical dose-response relationship for irradiated mammals. (a) linear ordinate; (b) probability ordinate.

antifungal drugs and barrier nursing) (UNSCEAR, 1988a Annex G). For a discussion of uncertainties in  $LD_{50/60}$  values see Fujita *et al.* (1990).

(B40) At doses in excess of about 5 Gy, additional effects occur, including severe gastrointestinal (stem cell and endothelial capillary cell) damage which, when combined with bone marrow damage, cause death in 1-2 weeks. At about 10 Gy, acute inflammation of the lungs can occur leading to death. At even higher doses, effects on the nervous and cardiovascular systems occur and the individual dies of shock after a few days (NCRP, 1974). Approximate doses for death at different times are given in Table B-2. These are for high dose, low LET radiation given over a short period of time, e.g., a few minutes. It requires a greater total whole body dose for these effects if the dose is given over a period of hours or more (UNSCEAR, 1988a). This report also contains references to much detailed early work on the acute radiation syndrome in man (e.g. Guskova and Baysogolov, 1971). Additional information on high dose effects resulting from accidents has been published (Hubner and Fry, 1980; Ricks and Fry, 1990).

### B.3.4. Functional changes resulting from deterministic effects

(B41) Some deterministic effects are the result of the dysfunction of a tissue or organ after irradiation which is not caused solely by cell killing. The mechanism may be the result of interference with other tissue functions (e.g. pituitary irradiation affecting hormone function in other endocrine glands) as noted earlier. A common characteristic is the reversibility of the transient effects observed if doses are not too high.

(B42) Examples of such functional changes that can occur are: the decrease of salivary gland or endocrine gland secretions; modifications of encephalographic rhythms or of the retinogram; vascular reactions such as early skin erythema (due to histamine release) or subcutaneous edema; and depression of the immunological system. These functional effects can have important consequences clinically, especially in the neurological and immunological systems.

### B.3.5. High LET radiations

(B43) Deterministic effects resulting from exposure to high LET radiation are similar to those from low LET exposure but their frequency and severity are greater per unit absorbed dose of high LET radiation. These differences can be expressed in terms of the relative biological effectiveness (RBE) for the effect under consideration. The RBE of high versus low LET radiation is defined as the ratio of the absorbed dose of the low LET radiation to cause the same level of the same biological effect as that of a dose of high LET radiation.

Table B-2. Range of doses associated with specific radiation induced syndromes and death in human beings exposed to acute low LET uniform whole body radiation

Whole body absorbed dose Gy	Principal effect contributing to death	Time of death after exposure (days)
3-5	Damage to bone marrow ( $LD_{50/60}$ )	30-60
5-15	Damage to the gastrointestinal tract and lungs <sup>1</sup>	10-20
> 15	Damage to nervous system <sup>1</sup>	1-5

<sup>1</sup> Damage to vasculature and cell membranes especially at high doses is important.

(B44) RBEs for deterministic effects are dose dependent and increase with decreasing dose to a presumed maximum value (designated as  $RBE_m$  to distinguish them from those for stochastic effects designated as  $RBE_M$ ) for a given radiation and a given tissue.  $RBE_m$  values are invariably smaller (ICRP, 1989) than the  $RBE_M$  values at low doses (see paragraph B65 and Table B-3) and are therefore less than recommended values of quality factors for these radiations. They also tend to be smaller for hematopoietic and reproductive tissue and larger for gastrointestinal tract and skin. Values of  $RBE_m$  for fission neutrons, for example, rarely exceed 10. Values of  $RBE_m$  are helpful in elucidating dose contributions from mixed fields.

(B45) A broad discussion of RBEs for deterministic effects as a function of dose and type of radiation for many individual tissues is given in *Publication 58* (ICRP, 1989). With the exception of kidney damage caused by 2.5 MeV neutrons,  $RBE_m$  values caused by neutrons and alpha particles are two to five times lower than values of  $RBE_M$  for stochastic effects in corresponding tissues. Thus the use of  $Q$  or  $w_R$  values in cases where deterministic effects are over-riding would result in an overestimate of the contribution to the risk from high LET radiation.

## B.4. Stochastic Effects: Carcinogenesis

### B.4.1. Introduction

(B46) Stochastic effects are those which result from alterations in normal cells caused by an ionising radiation event which is assumed to have a low probability of occurrence in cells at low doses. The probability of such a change occurring in a population of cells in a tissue is proportional to the dose at very low doses where, microdosimetrically, it can be determined that on the average less than one event per sensitive target in a cell occurs. The dose at which this holds depends on the size of the sensitive target and the LET of the radiation, and may be lower than many practical doses in radiation protection. For example, a dose of 1 mGy of 1 MeV gamma rays and 1 mGy of 1 MeV neutrons results in an average of about 1 (and occasionally more than 2) and  $10^{-2}$  tracks per cell nucleus respectively. Thus many cells would remain unirradiated in the tissue exposed to neutrons. More important from the viewpoint of carcinogenic mechanisms, the probability of energy being deposited in a particular 2 nm segment of DNA (there are about  $2 \times 10^7$  such segments in the DNA molecule) is small for both types of radiation, namely, about  $10^{-9}$  or less. However per unit track length, more energy will be deposited for neutrons than for gamma rays. Thus, if alteration of a particular 2 nm segment may play a vital role in the subsequent carcinogenic process, the biological changes resulting from energy deposition in that segment due to neutrons will be greater. This has been confirmed by cellular studies and animal experiments. Raising the dose within the tens of mGy range simply increases proportionally the number of cells that can be affected by single events. At higher doses when more than one event is likely to occur, per sensitive target of dimensions between about 2 and 100 nm, more complex dose-response relationships (such as linear-quadratic or quadratic) can occur. In our present state of knowledge and in the broad terms of radiological protection, the empirical observation that the quantities defined in Annex A correlate reasonably well with the observed biological effect justifies the Commission's use of these quantities.

(B47) Two general types of stochastic effects are well recognised. The first occurs in somatic cells and may result in the induction of cancer in the exposed person; the second

occurs in cells of the germinal tissue and may result in hereditary disorders in the progeny of those irradiated (see Section B.8).

### B.4.2. Induction of cancer

(B48) It is assumed that there is no threshold for the induction of the molecular change at specific DNA sites involved in the initial events that result in malignant transformation and ultimately cancer. Initial events themselves may involve more than one step in which radiation or any other external trigger is not necessarily the first. At some time after the initial events a clone of cells with malignant potential may arise and after further events in the cells, or their environment, a cancer may develop. In the development of some cancers, at least, these later changes are age dependent. The probability of the development of an overt cancer is far lower than that of the initial events because of host defences and the failure of succeeding changes required for the expression of the malignant potential of initiated cells.

(B49) In humans, the period between exposure to radiation and recognition of a cancer lasts a number of years. This period is called the latency period. The median latency period may be about 8 years in the case of induced leukaemia and two or three times longer in the case of many induced solid tumours such as in the breast or lung. The minimum latency period is the shortest time in which a specified radiation-induced tumour is known or believed to occur after exposure. This minimum latency period is about two years for acute myeloid leukaemia (and for  $^{224}\text{Ra}$  induced osteosarcomas) and of the order of 5–10 years for other cancers (Rall *et al.*, 1985). We have assumed an average of 10 years in this text. The frequency of radiation-induced leukaemias (and  $^{224}\text{Ra}$  induced osteosarcoma) declines after a peak at about 5–7 years to small excess values after about 20 years or more. In the case of cancers other than leukaemia and osteosarcoma, the relative risk remains approximately constant with time in those persons irradiated in adulthood. However there is some evidence of a decreasing relative risk in persons exposed in childhood and a decline in frequency with time has been suggested in the case of radon exposure and lung cancer (NCRP, 1984a,b; NAS, 1988). It is also seen in some cancers arising in patients given x-ray therapy for ankylosing spondylitis (Darby *et al.*, 1987), and in radiation-induced thyroid cancer (Shore *et al.*, 1985).

(B50) In experimental systems, which use neoplastic transformation of cells in culture or induced tumours (benign and malignant) in animals as endpoints, it is possible to study the form of the dose response, its relationship with time, and the influence of modifying factors such as dose rate, LET, and sensitising and protective agents. On the assumption that initiation leading to oncogenesis may occur through induced somatic mutation, studies in "in vitro" mutagenesis systems can also give important information on these factors.

(B51) Although experimental studies have their limitations, some generalisations on the data are possible. For low LET radiations, protracted (low dose rate) or fractionated exposures are less effective for many biological endpoints including tumour induction, than single exposures at high dose rates (see later Figure B-6). The protraction time may be important if within it significant changes take place in the susceptibility of the system to radiation exposure. For high LET radiations, low dose rate or fractionation may have effects similar to that of high dose rate single exposures in some cases and in others, low dose rate or fractionation is more effective than high dose rate, single exposures especially at higher doses as shown in Figure B-6. Certain chemical agents may increase

the rate of radiation-induced cell transformation or tumour induction, e.g., 12-O-tetra-decanyl phorbol-13 acetate (TPA, an active component of croton oil) or asbestos, or decrease it, e.g., vitamin A analogue. Their effectiveness depends to some degree on the LET of the radiation, the high LET response being less influenced (Sinclair, 1987), but note effects of TPA and neutrons (Han and Elkind, 1982).

(B52) The risk of cancer induction is assumed to be broadly proportional to the number of irradiated cells at risk (i.e. perhaps to the number of stem cells present) in a given organ or tissue, even though between species the evidence indicates that there is no correlation with body size. Special circumstances arise when an organ or tissue is irradiated non-uniformly, the extreme case of which occurs when "hot" (very active) particles irradiate only a portion of the organ or tissue, such as in the lung or liver. The dose averaged over the whole tissue is then much less than in the vicinity of the high concentration of the radioactive material. Experimental studies have been made of this situation (for example, Little *et al.*, 1970; Little and O'Toole, 1974) and for alpha particles in the lung the subject is reviewed by National Council on Radiation Protection and Measurements (NCRP, 1975) and by the US National Academy of Sciences (NAS, 1976). Generally, high concentrations of radioactive material in "hot spots" have been found less effective carcinogenically than the same amount of material spread uniformly and delivering a lower but uniform dose. This, in the main, is in accord with theoretical predictions (Mayneord and Clarke, 1973).

#### B.4.3. Cancer induction by low LET radiation: dose-response relationships

(B53) If information on the incidence of radiation induced cancer by low LET radiation were directly available in the dose range important in radiation protection, i.e., a few mGy to perhaps a few tens of mGy, questions about the possibility of a threshold, the shape of the dose-response curve, the effect of dose rate, etc. would be irrelevant. But most human information is obtained in a higher dose range (0.1 to 0.2 Gy and above) and only exceptionally at lower doses are significant results observed. The data from Japan however include many individuals exposed at low doses and eventually these data may yield significant information at low doses. Exposures are also often at high dose rates. Consequently these questions become critical for the evaluation of the probability of induced fatal cancer at low doses and dose rates. Therefore theoretical considerations, experimental data and limited human experience need to be taken into account in order to establish credible dose-response relationships for radiation-induced cancer in human beings at low doses.

(B54) Evidence is accumulating that the initiation of cancer is associated with the induction of lesions in genomic DNA that result in specific gene losses and/or changes in gene structure and activity (Bishop, 1987; Ponder, 1988; Reik and Surani, 1989). Also, recent studies with radiation or chemically-induced rodent tumours are beginning to shed light on the genes that might be involved in this initiation process (Janowski *et al.*, 1990; Sloan *et al.*, 1990; Kumar *et al.*, 1990). Mammalian cells are known to possess enzyme systems that have evolved to recognise and remove lesions from DNA and *in vitro* studies indicate that dose-rate effects on cellular low-LET radiation response may be associated with the activity of certain DNA repair systems (paragraph B76). Some cellular repair systems appear therefore to operate more effectively after low dose-rate exposure than after high dose-rate exposure. The more effective removal of DNA lesions following low dose-rate irradiation then predicts that radiation carcinogenesis will, at moderate to high doses, be subject to a dose-rate factor between high and low dose rates.

At very low doses, when the number of energy loss events in critical cellular target volumes is equal to or less than that of the targets themselves (see paragraph B46), dose-rate dependent cellular processes are essentially irrelevant. For radiological protection the central problem is the form in which dose-rate effectiveness factors for carcinogenesis increase with dose from the simple biophysical base line of unity at very low doses to the higher values predicted by our current knowledge of cellular repair and observed directly in many studies (see below paragraphs B55-B59). However, the genetic complexity of the multi-step cellular processes involved in malignant transformation is such that dose-rate effects may vary in different tissues and for different tumour types. For example, dose-rate effects may be influenced by the specific nature of the tumour-initiating lesion in DNA thereby generating dose-rate effect differences between different tumour types. Nevertheless, it is implicit in the preceding discussion that, overall, the dose-rate effectiveness factors to be applied to estimates of cancer induction derived from data at lower doses should be lower than those required to be applied after observations at higher doses.

(B55) Experimental information on dose-response relationships and the influence of dose rate was comprehensively reviewed in a report by the National Council on Radiation Protection and Measurements (NCRP, 1980). The general conclusion was that the shape of the dose-response relationship for high doses, at high dose rate is likely to be linear-quadratic in form (curve A, Figure B-5) in most biological systems. However for exposure to low doses at low dose rate, the response is often effectively linear as is to be expected for a linear-quadratic response at low dose. In the linear-quadratic form,  $E = \alpha D + \beta D^2$ , the effect initially increases linearly with dose i.e. the effect per unit dose  $E/D = \alpha$  is constant. Thereafter the effect increases more rapidly, i.e. the effect per unit dose increases linearly, as the quadratic term becomes operative ( $E/D = \beta D$ ). At higher doses still, the effectiveness often declines again due to the effect of cell killing reducing the number of cells at risk. In the linear-quadratic equation, the ratio of the parameters for the linear and quadratic terms,  $\alpha/\beta$ , has the dimension of dose and its value reflects the respective contributions of the linear and the quadratic term. Thus if  $\alpha/\beta = 1$  Gy, at 1

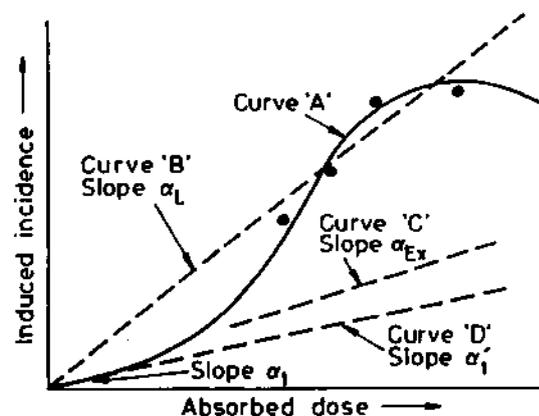


Fig. B-5. Schematic curves of incidence vs. absorbed dose (NCRP, 1980).

Gy the contributions to the response of the linear and quadratic terms (curve A) are equal.

(B56) The NCRP defined a dose-rate-effectiveness factor (DREF), as the ratio of the slope of the linear no threshold fit to high dose, high dose-rate data, to the slope of the linear no threshold fit to low dose-rate data (i.e.  $\alpha_L$ (Curve B) to  $\alpha_L$ (Curve D) in Figure B-5). It is evident from this figure that  $\alpha_L D = \alpha_L D + \beta D^2$  (where curves A and B meet initially) thus the DREF =  $\alpha_L / \alpha_L = 1 + \beta / \alpha_L \cdot D$ . The slope of the experimentally determined curves,  $\alpha_E$ (Curve C), will approximate  $\alpha_L$  when the dose and dose rate are high (and the DREF is high) and  $\alpha_L$  when the dose and dose rate are low (and the experimentally determined DREF is close to unity). Thus the observed DREF in experimental situations will depend on the dose range and the dose rate range over which the studies are performed. It will be smaller if these ranges are small. At the maximum in curve A (which hends over due to cell killing as noted above) the DREF will also be a maximum. The NCRP report provided tables of data on DREF values in a wide variety of experimental biological systems, including tumours and lifeshortening in animals. Some of these experimental data may reflect maximum values of DREF, others may not. The dose ranges involved (and thus the DREFs) are more often greater than is evident in the human experience, for example at Hiroshima and Nagasaki.

(B57) The NCRP concluded that values of DREF in experimental systems varied between 2 and 10 for individual tumour types and for life shortening in animals, as well as for a variety of other experimental endpoints. UNSCEAR (1986) reviewed the available data again and came to the conclusion, based essentially on the same sources of experimental information that responses at low dose and dose rate were less than those at high dose and dose rate by a factor of up to perhaps 5. UNSCEAR (1988b) did not re-evaluate the data but suggested the use of a factor of between 2 and 10, the implication being that the effect varied for different types of tumours. Further discussion (Liniecki, 1989) of this data and some additional experimental information includes data on life-shortening and transformation, confirming the range of 2 to 10 in animal experiments. A recent report on radiation-induced lifeshortening (due to tumours) in mice after single, fractionated and continuous exposures to  $^{60}\text{Co}$  gamma rays gives a maximum DREF of 5 (Thomson and Grahn, 1989), however this number includes "wasted" radiation (i.e. radiation later in the lifespan of the animal which made no further contribution to life-shortening). If this is corrected for, the ratio between single and continuous exposure for this important endpoint is about 2-2.5. (Note: Various terms have been used to describe the ratio called DREF by NCRP. The Commission has decided to call this important ratio, the Dose and Dose Rate Effectiveness Factor (DDREF).)

(B58) Human information on dose-response relationships and dose rate effects is limited and subject to many uncertainties as both NCRP (1980) and UNSCEAR (1986) have commented. Recent information on the A-bomb survivors suggests that for leukaemia the dose response fits a linear-quadratic relationship best with an equivalent DDREF of about 2 (NAS, 1990). For the solid cancers taken together, linearity provides the best fit (NAS, 1990) but individual tumour types show some differences in the slope of the dose response. The most recent reanalysis (Pierce and Vaeth, 1989) however suggests that there is little difference in dose-response relationship for any of the different cancer sites including leukaemia. These authors conclude that a DDREF of up to 2 would be possible from the A-bomb survivor data but greater than 2 would be difficult to justify.

(B59) Clinical data include some studies in which fractionation and single doses are

compared. Data from breast and thyroid studies show little evidence of fractionation effects (Boice *et al.*, 1979; Shore *et al.*, 1984a). A recent study on radiation-induced cancer in the breast shows a possible DDREF of up to 3 (Miller *et al.*, 1989). Recently cancers were found to be induced by  $^{131}\text{I}$  in the thyroid about 4 times less effectively than for acute x rays (Holm *et al.*, 1988) but factors other than dose rate (e.g., spatial distribution of dose and hormone balance) may also be involved. In another study, fractionated exposures in the lung failed to produce lung tumours even after several Gy (but did produce breast tumours) in contradistinction to the A-bomb survivor study, but no DDREF could be derived (Davis *et al.*, 1989). New human information on this question would be extremely valuable.

(B60) It must also be noted that linearity in dose response at doses of 1 Gy or more does not necessarily mean that no dose-rate effects are possible because of the different overall times of exposure involved when the dose is protracted. At such doses more than one ionising event can certainly occur in targets of molecular dimensions. A number of important experimental responses, such as lifeshortening in mice, seem to show linear responses with different slopes for different fractionation or dose rate regimes but mainly over relatively high dose ranges (Thomson and Grahn, 1989). At very low doses, at which less than one event per sensitive target may occur, the response is expected to be linear.

(B61) Theoretical considerations and most of the available experimental and epidemiological data do not support the idea of a threshold for the carcinogenic response to low LET radiation. Nevertheless, on statistical grounds a threshold for individual tumour types cannot be ruled out with certainty in either human or experimental systems. However, if thresholds do exist their values must be less than about 0.2 Gy for most human cancers and perhaps much less.

#### B.4.4. Choice of dose and dose rate effectiveness factor for low LET radiation

(B62) It is evident that theoretical considerations, experimental results in animals and other biological organisms, and even some limited human experience suggest that cancer induction at low doses and low dose rates should be less than that observed after high doses and dose rates. The principal source of risk estimation to be discussed later will be the Japanese survivors of the atomic bombs who were exposed to a range of doses at high dose rate and in whom statistically significant excess of cancer have been observed at doses down to 0.2 Gy. A DDREF should therefore be applied to this data. In making a determination on the value to be used for this purpose the Commission notes: (1) that the full range of DDREF values obtained from studies in animals, namely 2-10, may extend over a broader dose range than human data and therefore include higher values than are relevant; (2) that some human experience shows little evidence of fractionation effects while others indicate possible effects of up to 3 or 4 at most; (3) that direct statistical assessment of the A-bomb survivor data does not seem to allow for much more than a factor of about 2 for the DDREF; (4) that DDREF ratios actually used for risk estimates in the past by others include UNSCEAR, (1977) who used 2 and 2.5; UNSCEAR (1986) who suggested perhaps up to 5; and UNSCEAR (1988b) who recommended 2 to 10. The BEIR III Committee (NAS, 1980) used a DDREF of 2.25 and the BEIR V Committee (NAS, 1990) recommended 2 or more but applied 2 only in the case of leukaemia and 1 for other cancers in deriving their numbers. NUREG (1989) used 3.3 and a U.S. NIH group (Rail *et al.*, 1985) used 2.3. In view of these considerations and especially that limited human information suggests a DDREF in the low region of the

range, the Commission has decided to recommend that for radiation protection purposes the value 2 be used for the DDREF, recognising that the choice is somewhat arbitrary and may be conservative. Obviously this recommendation can be expected to change if new, more definitive information becomes available in the future.

#### B.4.5. Cancer induction after exposure to high LET radiation

(B63) Penetrating high LET radiations such as neutrons and short range high LET radiations in tissue such as alpha particles are generally more damaging per unit absorbed dose than low LET radiations. For cell killing, RBE values are often of the order of 2 or 3 at moderate doses and rise as the dose decreases. For deterministic effects generally, as noted earlier in the text (see paragraph B44), RBE values do not usually exceed 10 (ICRP, 1989). For stochastic effects the RBE of high LET radiations is again a function of dose level determined by the shape of the dose-response relationship. These response curves are typically concave upwards for single doses of low LET radiations and often concave downwards for single doses of high LET radiation as shown in Figure B-6 plotted on linear axes (Sinclair, 1982). In contrast, fractionated doses (or low dose rates) which are well known to be less effective for low LET radiation, are often for high LET radiation either as effective as single doses or more effective than single doses. Evidently, the RBE ( $b/a$  in Figure B-6) increases with decreasing dose but reaches a constant value, denoted by  $RBE_M$  (ICRP-ICRU, 1963), at low doses where both the low LET and high LET dose-response curves become linear.

(B64) In some cases high LET radiation (especially fission neutrons) has been shown to have increased effectiveness due to low dose rate and/or fractionation, even initially i.e., an initial linear slope steeper for low dose rate than high dose rate. This has been

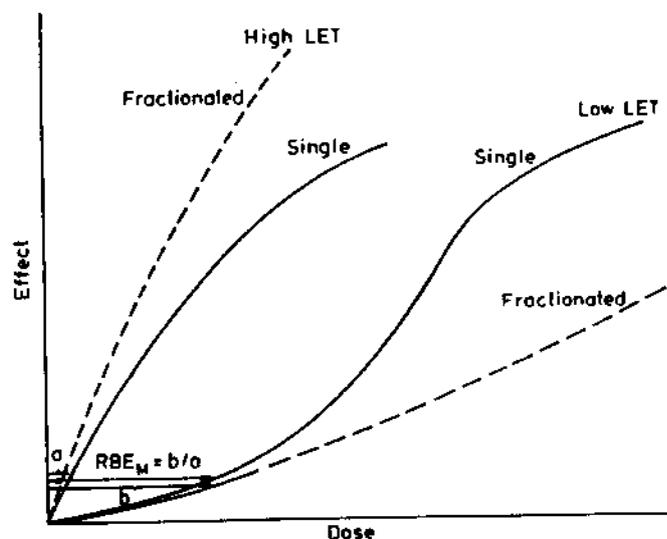


Fig. B-6. Shapes of dose responses for low LET and high LET radiations plotted on linear axes (Sinclair, 1982).

termed "reverse dose-rate effect". The increased effectiveness is usually small (1.5-2.5 times) (Ullrich 1984) but can in some instances *in vitro* be quite large (Hill *et al.*, 1984; Sinclair, 1987). The phenomenon is not always found and is not understood. For purposes of radiation protection however the maximum RBE,  $RBE_M$ , is in any event, that given by the steepest slope for the low dose rate, high LET response vs. the shallowest slope for low dose rate, low LET response.

(B65) Values of  $RBE_M$  vary for different stochastic endpoints and must be determined from experimental information at very low doses. By way of example, values of  $RBE_M$  for fission neutrons versus low dose rate, gamma rays are given in Table B-3 (ICRU, 1986). Similar tables for a broader range of high LET radiations have recently been published (NCRP, 1990). Bearing in mind that each experimental tumour model has its own peculiarities which make generalisations difficult, and that host factors including age and sex have a marked influence on whether or not animals exposed to radiation develop cancer, it is difficult to recommend a typical, single value of  $RBE_M$  for use in deriving quality factors. However values in the range of about 8-50 obtained for a variety of tumour endpoints in mice exposed to fission neutrons vs.  $^{60}\text{Co}$  gamma rays; of 19-70 for lung and mammary tumours in mice; and values of 15-45 for lifeshortening due mainly to tumours could support a range of  $RBE_M$  of about 30-50 for fission neutrons. Alpha particles have  $RBE_M$  values about the same or some what less than those of fission neutrons.

(B66) Values of alpha particle effectiveness have recently been discussed (NAS, 1988; NCRP, 1990). In the latter, for bone sarcoma at low incidence, alpha particles from  $^{226}\text{Ra}$  were found to be 26 times more effective than  $^{90}\text{Sr}$  beta particles in beagles and 25 times more effective in mice. Likewise alpha particles from  $^{239}\text{Pu}$  were about 30 times more effective than beta particles from  $^{144}\text{Ce}$  for the induction of lung cancer. In separate experiments the beta particles of  $^{144}\text{Ce}$  were shown to have the same effectiveness as protracted gamma radiation from  $^{60}\text{Co}$  and each was 15 to 20 times less effective than the alpha particles from  $^{239}\text{Pu}$  and  $^{241}\text{Am}$  in producing chromosome aberrations. In all instances the values of RBE depend on the dose and dose rate being greatest at low doses or dose rate, i.e., at low incidence for the endpoint in question.

(B67) It has recently been appreciated that Auger electrons may have values of RBE considerably higher than those for other electrons. In cases where the radionuclide does not penetrate the cell, Auger electron emitters are very inefficient in producing biological effects because of the short range of the low energy electrons. For those Auger electron emitters which penetrate the cell but are not incorporated into DNA, RBEs for a range of endpoints, including cell killing were found between 1.5 and 8 (Kassis *et al.*, 1988). For Auger emitters incorporated into DNA, such as  $^{125}\text{I}$ , much higher RBE values of 20-

Table B-3.  $RBE_M$  for fission (or optimum energy<sup>1</sup>) neutrons vs. gamma rays (ICRU, 1986; Sinclair, 1985) for stochastic endpoints

Tumour induction	3-200 <sup>2</sup>
Life shortening (due to tumours)	15-45
Transformation	35-70
Cytogenic studies	40-50
Genetic endpoints in mammalian systems	10-45

<sup>1</sup> "Optimum energy" is the most biologically effective energy.

<sup>2</sup> These values have been subsequently modified to 15 to 60 (NCRP, 1990).

40 have been found for endpoints such as cell transformation (Chan and Little, 1986) and calculations of energy deposition patterns have confirmed that those high values of RBE are to be expected (Charlton, 1988; Baverstock and Charlton, 1988).

(B68) It should be noted that at low doses, low LET radiations do not all have the same effectiveness. Conventional x rays (about 200 kV) are about twice as effective as gamma rays based upon studies of mutation in *Tradescantia* cells, aberrations in human lymphocytes and mouse oocyte killing (Bond *et al.*, 1978). Fast electrons may be even less effective than gamma rays. These differences must be taken into account in specifying RBEs from experimental data (ICRU, 1986; Sinclair, 1985).

(B69) Values of  $RBE_w$  for stochastic endpoints are usually specified relative to a particular low LET reference radiation and provide the primary basis for the determination of quality factors for given high LET radiations. These quality factors are appropriate "average" values of RBE for stochastic endpoints involving some judgement as to the overall effectiveness of the radiation in question relative to the "reference radiation" broadly defined to include all low LET radiations and taking the more relevant endpoints into account. The quality factor is applicable only for stochastic effects in the dose range up to tens of mGy. Thus, the applicable RBE values to be accounted for in the assessment of quality factors are usually values of  $RBE_w$  only (Sinclair, 1985; ICRU, 1986; NCRP, 1990). For higher doses (several gray) other sources of material on RBE values related to deterministic effects must be considered (see *Publication 58*, ICRP, 1989).

(B70) In addition to RBEs, other factors must be taken into account in specifying radiation quality or radiation weighting factors ( $w_R$ ) for use in radiological protection practice. The subject is discussed further and a table of values provided in Annex A.

## B.5. Estimates of Probability for Carcinogenic Effects (see Upton, 1991)

### B.5.1. Introduction

(B71) During the years since the publication of the last basic recommendations (ICRP 1977), new information on the risk of radiation-induced cancer in human populations has emerged and new experimental data in both laboratory animals and cultured cells have become available. These developments, summarised in reports by the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR, 1977, 1982, 1986, 1988b) and the Committee on the Biological Effects of Ionizing Radiations of the U.S. National Academy of Sciences, known as the BEIR V Committee (NAS, 1990), make appropriate a reassessment of ICRP's 1977 estimates of the probability of the carcinogenic effects of radiation (ICRP, 1977).

### B.5.2. New information on cancer induction and analytical techniques since 1977

(B72) The principal new information on the probability of radiation-induced human cancer deaths come from the continued assessment of the more than 90,000 survivors of the A-bombs in Japan (76,000 with DS86 dosimetry). Estimates of the probability of cancer death for the period 1950 to 1985 are increased over earlier estimates because of (a) the increase in the number of excess solid cancers observed in the additional 11 year follow-up period (~135 in 1975 compared with ~260 in 1985 for the DS86 cohort<sup>1</sup>) (Pierce, 1989), (b) the new dosimetry for the survivors (DS86 versus the former T65D)

<sup>1</sup> Excess leukaemia increased from 70 in 1950-1975 to 80 in 1950-1985.

(Roesch, 1987) which increases the probability values by between 1 and 2 times depending on the tissue site and the allowance made for neutron RBE<sup>1</sup>, (c) small changes in methods used to compute the age specific probability of cancer (Preston and Pierce, 1988) and (d) preference for multiplicative rather than additive models for projecting the observed numbers of solid cancers to lifetime values.

(B73) Further information is available from two other major populations. These include the 14,106 patients followed up in some cases for 48 years in the U.K. after radiotherapy to alleviate the pain associated with ankylosing spondylitis. Solid cancers (i.e., malignancies other than leukaemia) increased significantly in this population in the 5 to 25 year period following exposure but thereafter the excess appeared to diminish at some specific cancer sites (Darby *et al.*, 1987). These data have particular limitations. Nevertheless the estimates of probability from this study, especially for radiation-induced leukaemias, while lower, are within a factor of 2 of those derived from the survivors of the A-bombs (Table B-4 and see UNSCEAR 1988b, Annex F, Table 56). A parallel analysis of A-bomb survivors and ankylosing spondylitis patients has been published (Darby *et al.*, 1985) which discusses the differences in risk estimates. The differences in risk estimates between the two studies, less than a factor of 2 for leukaemia and about a factor of 2 for all cancer, can presumably be accounted for by the marked differences between the samples and their exposures. These differences include:

- (1) temporal and spatial distribution of the radiation dose and the range of doses involved in the two cases plus the fact that in the ankylosing spondylitic series in only a small subset are individual organ doses available
- (2) age, sex structure and health status of the population at risk
- (3) duration of follow-up
- (4) methods of cancer ascertainment
- (5) nature of the reference population used for comparison
- (6) constitutional differences in susceptibility
- (7) subgroup selection.

As Upton (1991) notes, since the influence of all these factors is not precisely known, it is not clear how to combine these two risk estimates. However, given all these differences they are clearly not incompatible with one another. In a third series, a study of second cancers in women treated for carcinoma of the cervix (Boice *et al.*, 1987, 1988) the results are more difficult to compare and the agreement less satisfactory (Table B-4) but again the differences in so many features of this sample and the A-bomb survivors are very great indeed. Under these circumstances UNSCEAR (1988b) and BEIR V (NAS, 1990) both selected the A-bomb survivors as the most complete set of information on which to base quantitative risk estimates and the Commission will follow this lead.

(B74) A number of other therapeutically irradiated populations provide additional information, e.g., (1) children treated for leukaemia (Tucker *et al.*, 1984; Meadows *et al.*, 1985); (2) patients treated for Hodgkins disease (Tucker *et al.*, 1984); (3) patients treated for ovarian cancer (Reimer *et al.*, 1978); (4) patients treated with <sup>224</sup>Ra for tuberculosis and ankylosing spondylitis (Mays and Spiess, 1984; Spiess, Mays and Chmelevsky, 1989); and (5) patients treated for tinea capitis (Modan *et al.*, 1989; Ron

<sup>1</sup> The difference between probabilities using DS86 vs. T65D dosimetries is based on the UNSCEAR determinations of organ dose equivalent in 1977, which used T65D with a neutron RBE of up to 20; and UNSCEAR in 1988 using DS86 with neutron RBE no longer critical.

Table B-4<sup>1</sup>. Absolute risk (excess deaths per 10<sup>5</sup> PYGy)<sup>2</sup>

Cancer	Atomic bomb survivors	Spondylitis series	Cervical cancer series
Leukaemia	2.94	2.02	0.61
All cancers except leukaemia	10.13	4.67	— <sup>3</sup>
Total	13.07	6.69	—

<sup>1</sup> For further details see UNSCEAR (1988b Annex F, Table 56).

<sup>2</sup> Person Year Gray.

<sup>3</sup> An estimate of the risk of all cancers except leukaemia cannot be made for this series. An estimate of the whole body dose does not exist and probably cannot be estimated given the nature of the exposures.

and Modan, 1984; Ron *et al.*, 1989; Shore *et al.*, 1984b). Exposures of children *in utero* to diagnostic x rays prior to 1958 (Stewart *et al.*, 1958; Stewart and Kneale, 1970) have also recently been reanalysed (Bithel and Stiller, 1988; Harvey *et al.*, 1985). New information has become available on radiation-induced breast cancer (Boice *et al.*, 1979; Land *et al.*, 1980; Howe, 1984); by the study of atomic bomb survivors (Tokunaga *et al.*, 1984); women treated with radiotherapy for acute post-partum mastitis and chronic breast diseases (Shore *et al.*, 1986), and women receiving multiple chest fluoroscopies in the course of therapy for tuberculosis in Massachusetts (Boice and Monson, 1977) and Canada (Howe, 1984; Miller *et al.*, 1989; Boice *et al.*, 1990; Hrubec *et al.*, 1989; Hildreth *et al.*, 1989). New information on the exposure of miners to radon in mines has come from Canada (Muller *et al.*, 1985; Howe *et al.*, 1986), from Czechoslovakia (Sevc *et al.*, 1988) and from the United States (Hornung and Meinhardt, 1987) and these have been reviewed comprehensively in various reports such as the BEIR IV report (NAS, 1988) and are discussed in paragraphs B124–B137. Most of these studies do not provide sufficiently quantitative dose response information for general risk estimation but they provide valuable additional data to support estimates of the probability of induced cancer in specific organs (UNSCEAR, 1988b; NAS, 1990). Other studies involving low dose exposures are discussed in Section B.6.

### B.5.3. New laboratory information since 1977

(B75) New experimental information on the induction of animal tumours by external penetrating radiations of different LET (Broerse, 1989; Upton *et al.*, 1986; Fry and Storer, 1987) and by incorporated alpha-emitting bone-seeking radionuclides (Humphreys, 1989; Taylor *et al.*, 1989) continues to accumulate. There are also new data regarding life shortening in mice (Thomson and Grahn, 1988, 1989; Carnes *et al.*, 1989). These data indicate high RBEs for high LET radiations (Sinclair, 1985; ICRU, 1986; Broerse, 1989; NCRP, 1990) at very low doses and dose rates in concert with the Commission's view of low dose and dose-rate effects for both low LET and high LET radiations. Cytogenetic and molecular studies on radiation- and chemically-induced animal neoplasms have been initiated and are beginning to highlight the importance of specific chromosomal changes in radiation oncogenesis and their possible association with oncogene activation and/or gene losses (Silver *et al.*, 1989). It may be anticipated that such mechanistic studies will lead to more confident interpretation and extrapolation of dose-effect relationships in animal models of induced neoplasia. *In vitro* cellular studies have provided more information on the influence of dose rate, post-irradiation repair/recovery processes, LET and various extrinsic factors on oncogenic transfor-

mation (Han *et al.*, 1980; Han and Elkind, 1982; Hall and Hei, 1985; Harisiadis *et al.*, 1978; Hei *et al.*, 1984). In principle, the utilisation of these cellular systems should facilitate the quantification of low dose response, its modification and the cellular processes involved. However, in conventional cellular systems the interpretation of findings is complicated by the use of established immortalised cell lines and poorly understood factors such as the composition of culture media and the effects of post-irradiation culture conditions (Little, 1989). In this respect, the observation in some laboratories of so-called "reverse-dose rate" effects on cell transformation by certain high LET radiations has been particularly contentious (Hill *et al.*, 1984; Ullrich, 1984). Much emphasis is currently being placed on the development of novel rodent and human epithelial cellular systems that may more accurately represent *in vivo* oncogenesis (Chadwick *et al.*, 1989); these have yet to make significant contribution to our understanding of low dose response. The induction of chromosomal changes in human lymphocytes by radiation have been studied at lower doses (<0.1 Gy) than previously achieved (Edwards *et al.*, 1989). Also, some evidence has been obtained for the induction, by low doses, of an "adaptive response" that reduces the frequency of chromosomal damage (Wolff *et al.*, 1989). The relevance of these findings for low dose oncogenesis remains, however, very uncertain.

(B76) *In vitro* studies with cultured human somatic cells have highlighted the importance of cellular repair/recovery processes in radiation response (e.g. Cox, 1982; Arlett *et al.*, 1989). There is also new information on molecular mechanisms of DNA repair that are directly relevant to cellular radiosensitivity (Thacker, 1991). In particular, recent studies have emphasised the importance of DNA double strand break (dsb) repair in cellular recovery and show that this may have a significant influence on dose-rate effects (Debenham *et al.*, 1987; Kemp *et al.*, 1984; Thacker and Stretch, 1985; Beer *et al.*, 1983; Wlodek and Hittelman, 1987; Evans *et al.*, 1987). In related fields, molecular studies of radiation-induced mutations in cultured cells have shown that mutations in a number of genes principally involve DNA deletion but that DNA base changes (point mutations) are observed in others (Thacker, 1986; Glickman *et al.*, 1987). On the hypothesis that specific gene mutations are responsible for the initiation of oncogenesis, knowledge of induced mutagenic lesions and their dependence on dose, dose rate, radiation quality and repair processes will be of importance to future views of radiological risk (see paragraphs B15–B18 and B54).

### B.5.4. Methodological factors affecting probability estimation

#### Multiplicative and additive models for projection of probabilities

(B77) Since the period of observation of an exposed population sample rarely extends to a full lifetime, it is necessary to project the estimate of probability of cancer induction for the period of observation to the lifetime of the exposed population, in order to obtain the full lifetime risk. Among many possible choices two principal models have been used for that purpose, one the absolute (risk) or additive projection model and the other the relative (risk) or multiplicative projection model. The former predicts, in its simplest form, a constant excess of induced cancer throughout life unrelated to the spontaneous rate of cancer while the latter predicts that the excess of induced cancers will increase with time as a constant multiple of the spontaneous or natural rate of cancer and consequently will increase with age in that population. Both forms of response occur after a minimum latency period. These models are used here to effect a suitable projection of

the data and do not necessarily imply biological mechanisms underlying cancer induction.

### Projection

(B78) The population surviving the Japanese A-bombs still contains many people irradiated in childhood or *in utero* who are now attaining the age when cancer and other diseases become prevalent. About three-fifths of the population survives at the present time. Thus, to obtain an estimate of the lifetime risk,  $U(A_0, D)$ , the experience of the cohort so far must be projected forward in time, taking into account the age structure of the population and the age-dependent force of mortality from causes unrelated to radiation exposure, as well as from radiation-induced cancer. This is done as follows. Let  $q_0(a)$  denote the age-specific death rate from all causes in a particular non-irradiated population and let  $h_{D, A_0}(a)$  denote the age-specific, excess death rate per year associated with exposure to dose  $D$  at age  $A_0$  (note that  $h_{D, A_0}(a) = 0$  for  $a < A_0$ ). The total death rate, then, is given by

$$q_{D, A_0}(a) = q_0(a) + h_{D, A_0}(a)$$

The probability of surviving to age  $a$  (years), given exposure to dose  $D$  at age  $A_0$ , is denoted  $L_{D, A_0}(a)$  and is given by the following algorithm:

$$L_{D, A_0}(a) = 1 \text{ for } a \leq A_0$$

(exposure at age  $A_0$  implies survival until age  $A_0$ )

$$L_{D, A_0}(a) = L_{D, A_0}(a-1) \cdot [1 - q_{D, A_0}(a-1)] \text{ for } a = A_0 + 1, \dots$$

(survival to age  $a$  implies survival to age  $a-1$  and precludes death at age  $a-1$ ).

The annual probability of death from any cause at age  $a$  is

$$L_{D, A_0}(a) q_{D, A_0}(a)$$

and the annual probability of a radiation-induced death at age  $a$  is

$$L_{D, A_0}(a) h_{D, A_0}(a)$$

Thus the lifetime probability of a death due to radiation exposure,  $U(A_0, D)$ , is

$$\sum_{a=A_0}^{\text{max age}} L_{D, A_0}(a) h_{D, A_0}(a)$$

The problem of risk projection arises because in current populations under study the youngest exposed cohorts have been followed barely into middle age. Denoting the follow-up age by the interval  $(A_1, A_2)$ , where  $A_0 \leq A_1 < A_2$ , the observed cumulative mortality is

$$R_{D, A_0}(A_1, A_2) = \sum_{a=A_1}^{A_2} L_{D, A_0}(a) q_{D, A_0}(a)$$

From observations on  $R_{D, A_0}(A_1', A_2')$ , for various subintervals  $(A_1', A_2')$ , where  $A_1 \leq A_1' < A_2' \leq A_2$ , and various doses  $D$  and exposure ages  $A_0$ , it is possible to estimate  $q_{D, A_0}(a)$ , and hence  $h_{D, A_0}(a)$ , as functions of  $D, A_0$  and  $a$  for  $A_1 \leq a \leq A_2$ . Projection involves estimates for values of  $a$  outside the observation interval. For cancers other than leukaemia, two simple models for  $h_{D, A_0}(a)$  have been widely used.

In the simple additive model,  $h_{D, A_0}(a)$  does not vary for  $a \geq A_0 + m$ , where  $m$  is a minimum latent period of 10 years or so:

$$h_{D, A_0}(a) = \begin{cases} 0 & \text{for } a < A_0 + m \\ K_{D, A_0} & \text{for } a \geq A_0 + m \end{cases}$$

In the simple multiplicative model,  $h_{D, A_0}(a)$  varies with  $a$  as a constant multiple of the baseline, age specific cancer rate for a non-exposed population,  $q_{0, \text{cancer}}(a)$ :

$$h_{D, A_0}(a) = \begin{cases} C_{D, A_0} \cdot q_{0, \text{cancer}}(a) & \text{for } a \geq A_0 + m \\ 0 & \text{for } a < A_0 + m \end{cases}$$

In the above formulations  $K_{D, A_0}$  and  $C_{D, A_0}$  depend on  $D$  and  $A_0$ , but not on  $a$ , and  $q_0(a) = q_{0, \text{cancer}}(a) + q_{0, \text{non-cancer}}(a)$ .  $q_{0, \text{cancer}}(a)$  is the component of  $q_0(a)$  that pertains to the specified cancer being considered.

### Projection by a modified multiplicative model

(B79) The U.S. National Academy Committee which produced the BEIR V report (NAS, 1990) used a modified multiplicative projection model which included terms dependent on time since exposure, which enabled a decrease in risk with time at longer times to be included in the formulation, i.e., the age-specific risk due to radiation dose  $D$  at time  $A_0$  for age  $a$  is  $h_{D, A_0} = q_{0, \text{cancer}}(a) \{f(D) \cdot g\}$ .

$f(D)$  is a dose-response function and is either linear ( $\alpha \cdot D$ ) or linear quadratic ( $\alpha D + \beta D^2$ ).

$g$ , the excess risk modifier, includes terms for sex, attained age, age at exposure and time since exposure. These terms were chosen separately for leukaemia, lung cancer and breast cancer.

### Dosimetry of the A-bomb survivors

(B80) The most informative quantity in which to express the dose when estimating the probability of induction of cancer in a given organ is the dose in that organ. In some cases the shielded kerma is quoted. The "shielded kerma" is the estimate of the kerma to each individual after the gamma rays and neutrons have passed through the shielding of house or other structure determined for that individual. The organ dose depends on the shielded kerma but the ratio between them is different for each organ. When uniform whole body irradiation is cited, organ dose equivalent is the quantity involved and this dose is the same to all organs. Uncertainties in the new DS86 dosimetry are discussed (Roesch, 1987).

### Incidence versus mortality

(B81) Most epidemiological data refer to mortality from the induced cancers in relation to that from spontaneous and other causes of cancer. Data on incidence are relatively sparse but incidence is usually a multiple of mortality for tumours, this multiple being strongly dependent on the level of medical care in each country. Incidence is more often inferred from mortality data since reliable data are difficult to obtain directly. For the Japanese survivors the Life Span Study Tumour Registry should provide direct data on incidence vs. dose to complement those hitherto available only from mortality data (Upton, 1991). In some specific sites e.g. thyroid and breast incidence data has been the primary source of information.

(B82) In the succeeding tables of data, results from the evaluation of the A-bomb survivors in Japan will mainly be used because this is the most comprehensive data base. Not only is the Japanese study large (76,000+ in the DS86 cohort) but both sexes and

all ages are represented, there is an internal control group, the dose range is extensive, the exposure is whole body and the dosimetry relatively well evaluated (see NAS, 1990, Table 4-1 for a comparison of data sets). For certain organs, such as thyroid, bone, skin and liver, sources of information other than the atomic bomb survivors will be used.

(B83) Reliance on the Japanese data exclusively for the derivation of quantitative estimates of the risk of radiation induced cancer in man, as both UNSCEAR (1988b) and BEIR V (NAS, 1990) have done, has been criticised in some reports commenting on current risk estimates (report of French Academy of Sciences, 1990). However it should be noted again that other important sources of information, such as the ankylosing spondylitic patients treated with x rays in the U.K. and to a lesser extent, the international cervix study agree well with the Japanese data (paragraph B73) considering the many differences between the exposed samples (Upton, 1991).

### B.5.5. Biological factors affecting cancer induction

#### Age

(B84) The incidence of radiation-induced fatal cancer varies with age at exposure and age at attainment depending upon the tumour type considered. In general, younger persons are more susceptible. For the female breast, for example, susceptibility is greatest in the very young female, declining throughout life and virtually disappearing if exposure occurs after menopause. Susceptibility to thyroid cancer shows a similar age related trend but in any event, lifetime incidence in children is 2 to 3 times greater than in adults. This pattern is also seen in the estimates of relative probability of death for all cancers except leukaemia (Table B-5). (For example, at ages <10 years ATB the total column indicates that those exposed had a 2.32 times greater relative risk of a solid cancer than the controls for all attained ages, but looking across the table, the ratio is less at older

Table B-5. Relative probability of fatal cancer after 1 Gy (shielded kerma) by age ATB<sup>1</sup> and attained age at death for various sites of cancer (extract from Shimizu *et al.*, 1988, Table 6)

Age ATB (y)	Total	Attained age (y) <sup>2</sup>						
		<20	20-29	30-39	40-49	50-59	60-69	70+
<b>Leukaemia</b>								
<10	17.05	44.16	3.41	8.64	0.95			
10-19	4.76	54.74	— <sup>3</sup>	2.45	1.02	0.82		
20-29	5.06		5.33	3.54	43.09	1.02	0.82	
30-39	3.99			0	24.05	10.58	1.47	3.89
40-49	2.55				0.83	3.82	0.82	3.10
50+	6.50					15.63	5.18	6.90
All ages	4.02	46.47	9.81	4.75	3.68	3.98	1.70	4.40
<b>All cancers except leukaemia</b>								
<10	2.32	(70.07)	5.89	1.96	1.86			
10-19	1.65	(40.90)	(0.82)	1.66	1.39	1.68		
20-29	1.65			(1.38)	2.09	1.74	1.37	
30-39	1.26			(0.84)	(1.12)	1.11	1.23	1.48
40-49	1.24				(1.25)	(1.12)	1.13	1.33
50+	1.11					(2.58)	(0.95)	1.15
All ages	1.29	75.32	2.22	1.60	1.58	1.39	1.13	1.29

<sup>1</sup> ATB = at time of bomb.

<sup>2</sup> Attained age, i.e. at death.

<sup>3</sup> No convergence.

Numbers in parentheses are the relative probabilities before the assumed minimum latent period of 10 years.

attained ages and more at younger attained ages. Furthermore this ratio declines for those older at the time of irradiation (ATB). The same pattern is seen initially with acute and chronic myeloid and acute lymphatic leukaemias but in this case susceptibility rises again for those exposed later in life (see Table B-5, total column). (For more detail on individual sites, see Shimizu *et al.*, 1988, Table 6.)

#### Sex

(B85) Females have been considered in the past somewhat more likely to develop radiation-induced cancers than males, for all cancers except leukaemia and especially for breast and thyroid cancers. For radiation-induced leukaemias, males are more sensitive, at least when expressed on an absolute risk basis. In the recent data, at least over the period of observation differences between the sexes overall are not large, the excess deaths for all cancers including leukaemia being only about 20% higher for women than men (Table B-6). The sex difference may be due to interactions between other factors such as hormone dependent promoting factors rather than a difference in radiation sensitivity. Differences in spontaneous cancer incidence such as in the thyroid (for which females are approximately 3 times more susceptible than males) or in co-factors may be more important.

#### Sensitive subpopulations

(B86) There are no epidemiological data currently available which identify adult subpopulations that are hypersensitive to the induction of cancer by ionising radiation although such groups are known to exist. In the case of exposure to UV light, patients with the DNA repair deficient genetic disorder, xeroderma pigmentosum (XP) show substantially increased susceptibility to sunlight (UV)-induced skin carcinoma. In general, *in vitro* studies show that cells from XP patients are not hypersensitive to ionising radiation. Patients with the leukaemia-prone genetic disorder ataxia-telangiectasia (A-T) are, however, extremely sensitive to the effects of low LET radiations. Cellular studies implicate DNA repair deficiency as the cause (Cox, 1982; Debenham *et al.*, 1987; Arlett *et al.*, 1989). It is important to recognise that even if all A-T patients were more likely than healthy persons to develop leukaemia, the very low frequency of the homozygous A-T mutation in the population implies an extremely small contribution

Table B-6. Relative risk and fatality probability coefficients by sex (shielded kerma) (from Shimizu *et al.*, 1988, Table 12)

Site of cancer	Estimated RR at 1 Gy			Excess deaths per 10 <sup>3</sup> PYGy		
	Male	Female	M/F	Male	Female	M/F
Leukaemia <sup>1</sup>	4.96	4.92	1.00	3.14	1.80	1.74 <sup>2</sup>
All cancers except leukaemia	1.17	1.44	0.81	5.76	8.78	0.66
Oesophagus	1.19	2.99	0.40	0.30	0.40	0.75
Stomach	1.15	1.36	0.85	2.01	2.18	0.92
Colon	1.45	1.67	0.87	0.60	0.51	1.18
Lung	1.26	1.86	0.68	1.07	1.47	0.73
Urinary tract <sup>3</sup>	2.00	2.15	0.93	0.81	0.42	1.93
Multiple myeloma	5.29	2.32	2.28	0.23	0.21	1.10

<sup>1</sup> Does not include lymphoma.

<sup>2</sup>  $p < 0.05$ .

<sup>3</sup> Mainly bladder.

to population risk. Additionally, the A-T mutation in the more frequent heterozygous form has also been suggested to confer a degree of spontaneous cancer susceptibility, particularly that of breast (Swift *et al.*, 1987). Consequently A-T heterozygotes could, in principle, constitute a small but possibly significant sensitive subpopulation, although this is not yet established. Other human genetic disorders such as retinoblastoma where tissue specific cancers may be associated with heterozygosity for so called "cancer suppressor genes" could also be considered to carry increased risk (Knudsen, 1986; Reik and Surani, 1989). Our current lack of knowledge on the frequency of all such mutations and their implications for induced cancers preclude, however, any quantitative estimate of their cancer yield in an irradiated human population.

#### Other factors

(B87) Other carcinogenic factors can also play a role and a wide variety of interactive responses have qualitatively been observed. One important example is the carcinogenic action of radiation on the skin which can be enhanced by ultraviolet light (Shore *et al.*, 1984b). Another is the influence of smoking on the induction of lung cancer by radon observed in miners (NAS, 1988).

#### Age at expression

(B88) Radiation-induced tumours, such as breast cancer in women, tend to be expressed later in life when tumours from other causes also occur regardless of age at exposure. This fact suggests that radiation may initiate the process at a young age but completion requires additional steps later in life, some of which are hormone dependent.

#### B.5.6. Estimates of fatal cancer probabilities

(B89) In the Japanese A-bomb survivors, the excess cancer deaths are estimated to be  $13.1 \times 10^{-4}$  per person year gray (Shimizu *et al.*, 1988, Table 4) for a follow up period from 1950-1985 (equivalent to 2.2 million person years). By comparison the excess probability of fatal cancer for all neoplasms (except carcinoma of the colon which is excluded because this type of cancer is thought to be related to the spondylitis) among the ankylosing spondylitis is  $6.7 \times 10^{-4}$  per person year gray for a mean follow up period of 13.0 y (equivalent to 184,000 person years). Considering the various differences between the two sets of data including the age of the individuals exposed, the time of delivery of the radiation and the partial body character of the exposure, this is quite good agreement, see paragraph B7.3 (Upton, 1991) and UNSCEAR, 1988b, Annex F, Table 56). Because the data base is so much more comprehensive for the Japanese A-bomb survivors and is a measure of excess cancers after uniform whole body irradiation, these have been used primarily by the UNSCEAR (1988b) and also by NAS (1990) for projecting estimates of the probability of fatal cancer from the period of observation to the lifetime of the Japanese population.

#### B.5.7. UNSCEAR estimates

(B90) UNSCEAR used both models, additive and multiplicative, for projection to the full lifetime of the exposed population. These two models result in somewhat different estimates of lifetime probability of fatal cancer, however these differences have become smaller with time (see Table B-10).

#### Age and projection

(B91) Age at the time of exposure is an important parameter and it influences the projection of fatal cancer probability to lifetime. Some estimates were made by UNSCEAR in the Japanese study population using age specific coefficients for each 10 y age interval, others were made more approximately by using an "age-averaged" coefficient. The estimates made by UNSCEAR for (a) the entire population, (b) all adults over age 25, (c) a working population of ages (25-64), which yield somewhat different estimates of the probability of fatal cancer, are given in Table B-7.

Table B-7. Projections of lifetime probability of fatal cancer and life lost for 1 Gy whole-body, low-LET radiation (UNSCEAR, 1988b)

	Projection model	Excess fatal cancers <sup>1</sup> ( $10^{-2}$ )	Period of life lost <sup>2</sup> (year)
Total population <sup>3</sup>	Additive	4.0 <sup>1</sup> - 5.0 <sup>1</sup>	0.95 <sup>1</sup> - 1.20 <sup>1</sup>
	Multiplicative	7.0 <sup>1</sup> - 11.0 <sup>1</sup>	0.95 <sup>1</sup> - 1.40 <sup>1</sup>
Working population <sup>2</sup> (aged 25 <sup>4</sup> -64 years)	Additive	4.0 <sup>1</sup> - 6.0 <sup>1</sup>	0.88 <sup>1</sup> - 1.33 <sup>1</sup>
	Multiplicative	7.0 <sup>1</sup> - 8.0 <sup>1</sup>	0.82 <sup>1</sup> - 0.97 <sup>1</sup>
Adult population <sup>2</sup> (over 25 years)	Additive	5.0 <sup>1</sup>	0.84 <sup>1</sup>
	Multiplicative	6.0 <sup>1</sup>	0.62 <sup>1</sup>

<sup>1</sup> Based on cancer mortality rates for the population of Japan.

<sup>2</sup> Equal numbers of males and females.

<sup>3</sup> Age-specific coefficient of probability.

<sup>4</sup> Adult age-averaged coefficient of probability.

<sup>5</sup> Age 25 y is the mean of age 20-29 y.

#### Cancers in specific sites

(B92) The estimates of relative probability and excess probability for each cancer site for the observation period (Shimizu *et al.*, 1988, Table 4) as a function of age at irradiation can be projected by either the additive or multiplicative projection model to estimate lifetime excess probability of fatal cancer at each site. Both estimates based on the age-averaged coefficient are given in Table B-8 (Upton, 1991, Table 12; UNSCEAR, 1986, Table 69).

#### B.5.8. BEIR V estimates

(B93) The BEIR V Committee adopted a somewhat different approach as noted earlier. They used a modified multiplicative projection model which included a term allowing for a decrement in the probability of a fatal cancer with time when appropriate. Different parameters were used for different cancer types so that the form of the decrement could be varied to fit the data available. The analysis then provided for age specific coefficients in 10 year intervals to be projected according to the model for each cancer or cancer group separately, as indicated for a dose equivalent of 0.1 Sv in Table B-9. The BEIR committee expressed the result per Sv because the neutron component of dose equivalent, with an RBE of 20, was included. The results show a very substantial variation with age at exposure for most cancer groups, a steady decline with age for cancer in digestive organs and breast for example, but an increase in the middle age range for respiratory cancers. Overall, the difference between the sexes is less than estimated by UNSCEAR, females being more sensitive than males by only about 6 percent. The total risk (average for males and females) for all cancers for 0.1 Sv is  $0.79 \times 10^{-2}$ . In this estimate the contribution for leukaemia has already been reduced by a factor of 2 (using

Table B-8. Excess probability of a fatal cancer (specific) after acute whole body exposure, 1 Gy organ absorbed dose of low-LET radiation (UNSCEAR, 1988a)<sup>1</sup>. (Based on the population of Japan, 90% confidence intervals in parentheses)

Malignancy	Probability of fatal cancer ( $10^{-2}$ )	
	Multiplicative risk projection model	Additive risk projection model
Red bone marrow	0.97 (0.71-1.32)	0.93 (0.77-1.10)
All cancers except leukaemia	6.10 (4.80-7.50)	3.60 (2.80-4.40)
Bladder	0.39 (0.16-0.73)	0.23 (0.11-0.40)
Breast <sup>2</sup>	0.60 (0.28-1.05)	0.43 (0.22-0.69)
Colon	0.79 (0.36-1.34)	0.29 (0.14-0.46)
Lung	1.51 (0.84-2.30)	0.59 (0.34-0.88)
Multiple myeloma	0.22 (0.06-0.51)	0.09 (0.03-0.17)
Ovary <sup>2</sup>	0.31 (0.09-0.68)	0.26 (0.08-0.48)
Oesophagus	0.34 (0.08-0.72)	0.16 (0.03-0.31)
Stomach	1.26 (0.66-1.99)	0.86 (0.45-1.31)
Remainder	1.14 <sup>3</sup>	1.03 <sup>3</sup>
	1.18 <sup>4</sup>	0.66 <sup>4</sup>
Total	7.07 <sup>5</sup>	4.53 <sup>5</sup>
	7.12 <sup>6</sup>	4.16 <sup>6</sup>

<sup>1</sup> Estimates based on age averaged coefficients.

<sup>2</sup> These values have to be divided by 2 to calculate the total and other organ probability values. Values are similar for Japanese survivors and other sources.

<sup>3</sup> This value is derived by subtracting the sum of the probabilities at the sites specified from the probabilities for all cancers except leukaemia.

<sup>4</sup> This value is derived by fitting a linear relative probability model to the basic cancer data after the exclusion of those cases of cancer at the specific sites listed. (Coefficients 0.19 excess relative probability per Gy and  $1.87 \times 10^{-2}$  per person year gray.)

<sup>5</sup> Red bone marrow plus all other cancers.

<sup>6</sup> Red bone marrow plus other individual sites including remainder.

a linear-quadratic response) whereas for solid tumours a linear response was used. For high dose, high dose rate the leukaemia contribution should be doubled, giving a total average risk for all cancers of  $8.85 \times 10^{-2} \text{ Sv}^{-1}$ . [It should also be noted that in the BEIR V approach early cancer deaths (i.e. cancer deaths due to exposure in persons who would have died of spontaneous cancer later) are not included in the estimates of total excess lifetime mortality. Thus those estimates are about 20% lower than would be obtained by the UNSCEAR approach for the same population.]

#### B.5.9. Comparison of UNSCEAR and BEIR V with earlier estimates

(B94) Over the years, starting in about 1972, the UNSCEAR and BEIR Committees and some other sources, (e.g. a risk evaluation sponsored by the Nuclear Regulatory Commission of the United States, NUREG) have made major risk evaluations resulting in estimates of the risk associated with 1 Gy of acute low-LET uniform whole-body irradiation. Some representative values are listed below (Table B-10). For data from the Japanese A-bomb survivors, the first four of these refer to T65 dosimetry, the last two, to DS86 dosimetry.

(B95) It is evident that estimates based on the additive model and the multiplicative model have come closer together with time. Furthermore the estimates based on the multiplicative model have changed the least, i.e. they have remained the most robust.

Table B-9. Excess lifetime mortality (specific organ systems) after exposure to 0.1 Sv acute uniform whole-body low LET radiation (US population) (NAS, 1990)<sup>1</sup>

Age at exposure (year)	Total	Probability of death ( $10^{-3}$ )				
		Males				
		Leukaemia <sup>1</sup>	Nonleukaemia <sup>2</sup>	Respiratory	Digestive	Other
5	12.76	1.11	11.65	0.17	3.61	7.87
15	11.44	1.09	10.35	0.54	3.69	6.12
25	9.21	0.36	8.85	1.24	3.89	3.72
35	5.66	0.62	5.04	2.43	0.28	2.33
45	6.00	1.08	4.92	3.53	0.22	1.17
55	6.16	1.66	4.50	3.93	0.15	0.42
65	4.81	1.91	2.90	2.72	0.11	0.07
75	2.58	1.65	0.93	0.90	0.05	—
85	1.10	0.96	0.14	0.17	—	—
Average	7.70	1.10	6.60	1.90	1.70	3.00

Age at exposure (year)	Total	Females					
		Leukaemia <sup>1</sup>	Nonleukaemia <sup>2</sup>	Respiratory	Digestive	Breast	Other
5	15.32	0.75	14.57	0.48	6.55	1.29	6.25
15	15.66	0.72	14.94	0.70	6.53	2.95	4.76
25	11.78	0.29	11.49	1.25	6.79	0.52	2.93
35	5.57	0.46	5.11	2.08	0.73	0.43	3.87
45	5.41	0.73	4.68	2.77	0.71	0.20	1.80
55	5.05	1.17	3.88	2.73	0.64	0.06	0.45
65	3.86	1.46	2.40	1.72	0.52	—	0.16
75	2.27	1.27	1.00	0.72	0.26	—	0.03
85	0.90	0.73	0.17	0.15	0.04	—	—
Average	8.10	0.80	7.30	1.50	2.90	0.70	2.20

<sup>1</sup> Based on a single exposure in radiation and on a life-table weighted average over each of the age groups listed, in a stationary population having U.S. mortality rates.

<sup>2</sup> Based on the sum of cancers of respiratory tract, digestive tract, breast and other organs, linear dose response assumed.

<sup>3</sup> Based on linear-quadratic dose response which reduces high dose, high dose rate value by a factor of 2. Models used to derive numbers are in Upton (1991).

varying by less than a factor of 2 since 1972. A few years ago the results obtained by the additive model were preferred and it is for this reason among others, that the previous risk estimates used as the Commission's basis for radiation protection (ICRP, 1977) appear now to have changed, overall by about 3-4 times, since 1977.

#### B.5.10. Probability of fatal cancer in organs vs. sex, age and population (see Land and Sinclair, 1991)

(B96) Especially for determining the effective dose in the case of non-uniform irradiation of the body, the distribution of fatal cancer risk among organs needs to be known. The list of fatal cancer probability in organs given in Table B-8 was derived by UNSCEAR using age averaged risk coefficients and for both additive and multiplicative projection models. While quite useful, these tabulations do not provide enough detail to examine the effect on the distribution of risks of fatal cancer in the more important organs (i.e. the basis for weighting factors) of important variables such as sex, different age ranges and for different population characteristics as well as for different models. These factors must be examined in order to determine whether it is reasonable to use a single set of weighting factors for a wide variety of exposure circumstances. More

Table B-10. Excess lifetime mortality from all cancer, attributable to 1 Gy acute uniform whole-body low LET irradiation of the general population (Upton, 1991)<sup>1</sup>

Source of estimate	Probability of death ( $10^{-3}$ )	
	Additive risk projection model	Multiplicative risk projection model
BEIR I, 1972	1.2	6.2
UNSCEAR, 1977	2.5	—
BEIR III, 1980	0.8–2.5	2.3–5.0
NUREG, 1985	2.9	5.2
UNSCEAR, 1988	4.0 <sup>2</sup> –5.0 <sup>3</sup>	7.0 <sup>2</sup> –11.0 <sup>3</sup>
BEIR V, 1990	—	8.85 <sup>4,5</sup>

<sup>1</sup> Population of Japan.

<sup>2</sup> Estimate based on age-specific coefficients of probability.

<sup>3</sup> Estimate based on constant (age-averaged) coefficient of probability.

<sup>4</sup> U.S. population—adjusted to high dose using values from Table B-9.

<sup>5</sup> Modified multiplicative model.

<sup>6</sup> "Low dose" leukaemia component multiplied by 2.

detailed calculations of the probability of fatal cancer in these organs were undertaken in order to do this.

(B97) The starting point is the age specific coefficients available from the A-homb study (Shimizu *et al.*, 1988, Tables 5A and B) for most of the organs in the UNSCEAR list. Results for the oesophagus, ovary and bladder were derived separately, because the information is too scant to provide detailed variation of fatal cancer probability with age. Cancers other than those in the eight organs listed, i.e. the remainder, were held at a constant fraction of the total, 0.15 (explained in Land and Sinclair, 1991). Calculations made for the Japanese population involve first a transfer from the observed data and then projection in time using three different models, the additive risk model, the multiplicative risk model and the model used by an ad hoc working group of the U.S. National Institute of Health to develop radioepidemiological tables ("NIH model") and also used earlier in the BEIR III report (NAS, 1980). The first two were described earlier. The latter (Rall *et al.*, 1985) involves estimating the absolute risk for the period of observation (in the Japanese population) then transferring to the new population as an absolute risk before converting to relative risk in the new population and projecting over time in the same way as for the multiplicative model.

(B98) Estimates of the fatal cancer probability after 1 Gy of acute low LET whole-body radiation have been made in each of eight organs plus the remainder tissues; for all cancer for males and females; for four age ranges 0–90 y, 0–19 y, 20–64 y and 65–90 y; for five populations (those of Japan, the U.S., Puerto Rico, the U.K. and China); and for each of three models. One representative sample of these calculations of risk is shown for the population of Japan and for one age range (0–90 y) in Table B-11 for 3 models and both sexes. Similar information on fatal cancer probability is available for other age ranges and for years of life lost for the Japanese population. Hereafter, in this section results are presented for the different variables involved in the form of relative values of the fatal cancer probability totalling 1.00. The actual total risk is also given in each case. The various factors involved are separated as follows.

Table B-11. Excess mortality from cancer after acute whole-body low LET radiation (Japanese population, age 0–90 y)

	Excess mortality ( $10^{-2} \text{ Sv}^{-1}$ )					
	Additive		Multiplicative		NIH	
	Male	Female	Male	Female	Male	Female
Oesophagus	0.118	0.234	0.217	0.467	0.217	0.467
Stomach	0.680	0.799	2.241	2.768	2.041	2.237
Colon	0.201	0.236	0.894	2.451	1.008	0.929
Lung	0.358	0.572	1.293	1.732	1.788	1.732
Breast	—	0.272	—	0.491	—	0.439
Ovary	—	0.32	—	0.306	—	0.306
Bladder	0.277	0.123	0.566	0.251	0.566	0.251
Bone marrow	1.063	0.649	0.859	0.587	1.157	0.688
Remainder	0.756	0.955	1.951	4.421	1.879	3.656
All cancer	3.452	4.071	8.022	13.470	8.659	10.687

#### Sex and projection model

(B99) Results for the relative probabilities of fatal cancer in the organs and the total risk for the Japanese population, ages 0–90 y, both sexes and three different models are presented in Table B-12. It is evident that the total risks are similar to those found by UNSCEAR for the additive and multiplicative models (see Table B-7). Furthermore the results using the NIH model are close to those for the multiplicative model (within less than a factor of 2). (Also the ratios for an average of males and females are similar, for the additive and multiplicative models, to those which can be derived from the UNSCEAR values of Table B-8.) The largest differences in the relative probabilities for a given model between males and females (ignoring the breast and ovary) for any given important contributor organ are about a factor of 2 (e.g. for bone marrow and for colon, especially in the multiplicative model). Less important contributor organs such as the bladder may differ by up to a factor of 3. The total risk for all cancers differs between male and female at most by about 50% for the multiplicative model, females having the greater risk. Thus in the final assessment of weighting factors for radiation protection purposes if a difference of about 30–50% in total risk between females and males and a factor of 2 difference between any important organ is acceptable, this is a useful guide with which to test the importance of other variables such as age and population mix. An inspection of the overall data available (Land and Sinclair, 1991) indicates that these sex differences in important organs are not greater (indeed about the same) for the populations of the U.S., Puerto Rico, the U.K. and China.

#### Age

(B100) The relative probabilities of fatal cancer in the different organs and the total risks for the Japanese population, sexes averaged, ages 0–90 y, 0–19 y, 20–64 y, two models (multiplicative and NIH) are presented in Table B-13. The additive model is not a preferred model and therefore is not considered further here, although results using it are available elsewhere (Land and Sinclair, 1991). It is evident that the relative probabilities vary with age group for a given model by a factor of 2 or 3 in the case of both models (for leukaemia and colon). However, the differences, for either of the two models for the different age groups are not much greater than the differences for sex. (But note that the total risk determined as the sum of the individual organ risks differs by a factor of about 3 for young (0–19 y) vs. older (20–64 y) age groups.)

Table B-12. Relative probabilities of fatal cancer in organs vs. sex and projection model (Japanese population, age 0-90 y)

Organ	Projection model					
	Additive		Multiplicative		NIH	
	M	F	M	F	M	F
Oesophagus	0.039	0.065	0.031	0.044	0.028	0.057
Stomach	0.225	0.223	0.319	0.262	0.261	0.274
Colon	0.067	0.066	0.127	0.232	0.129	0.113
Lung	0.118	0.160	0.184	0.164	0.229	0.212
Breast	—	0.076	—	0.046	—	0.054
Ovary	—	0.065	—	0.029	—	0.037
Bladder	0.092	0.034	0.081	0.024	0.073	0.031
Bone marrow	0.307	0.158	0.106	0.040	0.129	0.071
Remainder	0.150	0.150	0.150	0.150	0.150	0.150
All cancer	1.000	1.000	1.000	1.000	1.000	1.000
Total probability ( $10^{-2}$ Sv $^{-1}$ )	3.45	4.07	7.99	13.5	8.64	10.7

Table B-13. Relative probabilities of fatal cancer in organs vs. age group (0-90 y, 0-19 y, 20-64 y) Japanese population, average of male and female

Organ	Projection model					
	Multiplicative			NIH		
	0-90 y	0-19 y	20-64 y	0-90 y	0-19 y	20-64 y
Oesophagus	0.038	0.021	0.061	0.042	0.024	0.063
Stomach	0.291	0.266	0.305	0.268	0.225	0.301
Colon	0.180	0.255	0.089	0.121	0.171	0.066
Lung	0.174	0.191	0.159	0.221	0.297	0.129
Breast	0.023	0.025	0.022	0.027	0.034	0.019
Ovary	0.014	0.009	0.023	0.019	0.013	0.025
Bladder	0.052	0.030	0.082	0.052	0.028	0.080
Bone marrow	0.077	0.052	0.109	0.100	0.055	0.165
Remainder	0.150	0.150	0.150	0.150	0.150	0.150
All cancer	0.999	1.000	1.000	0.998	1.000	1.000
Total probability ( $10^{-2}$ Sv $^{-1}$ )	10.7	24.6	7.8	9.7	21.5	7.3

#### National populations and transfer models

(B101) The results for the relative probabilities of fatal cancer for males and females averaged, for age 0-90 y, using the multiplicative model both for transfer and projection for Japan, U.S., Puerto Rico, the U.K. and China are given in Table B-14A. Large differences are evident in the contributions for the oesophagus, stomach and breast among the five national populations. All organs are however, within a factor of about 3 of the average value. In order to examine the effect of the method of transfer, the NIH model which transfers by absolute risk and then projects multiplicatively, was used to determine relative probabilities of fatal cancer in the same way. A few of the results shown in Table B-14B are dramatically different from those of Table B-14A. The risk for cancer of the stomach makes a higher contribution in each of the populations other than in the Japanese, whereas in Table B-14A these contributions were much less. Overall the NIH model gives less variation between different populations, no more than factor of 2 for

Table B-14A. Relative probabilities of fatal cancer in organs vs. population type. (Average of male and female, age 0-90 y, multiplicative model)

Organ	Japan	United States	Puerto Rico	United Kingdom	China	Average
Oesophagus	0.038	0.014	0.098	0.030	0.269	0.090
Stomach	0.291	0.033	0.136	0.050	0.224	0.144
Colon	0.180	0.320	0.206	0.225	0.103	0.207
Lung	0.174	0.205	0.141	0.274	0.097	0.179
Breast	0.023	0.075	0.048	0.085	0.022	0.051
Ovary	0.014	0.031	0.016	0.031	0.019	0.022
Bladder	0.052	0.076	0.078	0.090	0.036	0.067
Bone marrow	0.077	0.096	0.127	0.064	0.079	0.089
Remainder	0.150	0.150	0.150	0.150	0.150	0.150
All cancer	0.999	1.000	1.000	0.999	0.999	0.999
Total probability <sup>1</sup> ( $10^{-2}$ Sv $^{-1}$ )	(10.7)	(11.2)	(9.5)	(12.9)	(6.3)	(10.1)

<sup>1</sup> In the process of transfer between populations calculations based on individual organ, transfers vary more and give higher total risks, by up to 20% than calculations of risk based on all nonleukaemia sites transferred together, especially for the multiplicative model.

any organ as might be expected since the transfer is additive. The estimates of total risk, determined as the result of transferring the estimation for nonleukaemia as a group, for all populations vary more for the multiplicative than for the NIH model. Note also that where comparisons are possible the relative organ risks for a U.S. population, multiplicative model (column 2, Table B-14A) agree quite well with BEIR V results (see Table B-9).

(B102) Unfortunately, there is no general agreement on which, if any, transfer method is to be preferred or indeed whether the same method should apply to each cancer site (see NAS, 1990, p. 218 and Land, 1991). Nor is there any specific reference population with which ICRP should deal. (The populations used here are, of course, representative of various different parts of the world but were included primarily because of their diversity among the available populations with the requisite information.) Therefore, to reduce the effects of national population characteristics, the relative probabilities of the fatal cancers in organs will simply be averaged (the populations could be weighted, but a simple average might well be as good a representation of a "world" population as any other, furthermore adding further populations would not change the average greatly). This is done in the sixth column of Tables B-14A and B-14B. It is evident now (Table B-14A) that the deviation of any population from this average ratio is within a factor of about 3-4 for any organ and that for the average in Table B-14B the deviations are less.

(B103) This examination clearly shows that while the effects of sex, age and projection model on relative probabilities of fatal cancer in organs are considerable, i.e. up to about a factor of 3, they are nevertheless rather less than the effect of the choice of transfer model, and some differences in national population characteristics, i.e. compare Tables B-14A and B. Consequently it is reasonable to consider only a single set of relative probabilities of fatal cancer in organs at least until definite conclusions can be made about transfer models and differences in national populations, at least for the multiplicative model. The differences are much less for the NIH model.

(B104) In view of the difficulty of choosing between transfer models and to minimise further the effects of statistics in national populations, the ratios obtained by the two

Table B-14B. Relative probabilities of fatal cancer in organs vs. population type. (Average of male and female, age 0-90 y, NIH projection model)

Organ	Japan	United States	Puerto Rico	United Kingdom	China	Average
Oesophagus	0.042	0.025	0.030	0.023	0.037	0.032
Stomach	0.268	0.317	0.346	0.336	0.291	0.309
Colon	0.121	0.188	0.138	0.147	0.113	0.142
Lung	0.221	0.121	0.137	0.183	0.132	0.160
Breast	0.027	0.034	0.027	0.028	0.044	0.032
Ovary	0.019	0.023	0.027	0.019	0.022	0.022
Bladder	0.052	0.048	0.054	0.037	0.052	0.049
Bone marrow	0.100	0.093	0.092	0.077	0.158	0.104
Remainder	0.150	0.150	0.150	0.150	0.150	0.150
All cancer	0.998	0.999	1.001	1.000	0.999	1.000
Total probability <sup>1</sup> ( $10^{-2}$ Sv <sup>-1</sup> )	(9.7)	(8.7)	(10.2)	(9.7)	(6.0)	(8.9)

<sup>1</sup> In the process of transfer between populations calculations based on individual organ, transfers vary more and give higher total risks, by up to 20% than calculations of risk based on all nonleukaemia sites transferred together, especially for the multiplicative model.

Table B-15. Distribution of probabilities of fatal cancer in organs (Average of males and females, five national populations, two models, age 0-90 y)

Organ	Average
Oesophagus	0.061
Stomach	0.229
Colon	0.174
Lung	0.168
Breast	0.041
Ovary	0.022
Bladder	0.058
Bone marrow	0.096
Remainder	0.150
All cancer	0.999
Total probability ( $10^{-2}$ Sv <sup>-1</sup> )	9.5

methods, multiplicative transfer Table B-14A and additive transfer (NIH model, Table B-14B) will be averaged again. This yields the values given in Table B-15. These values will be used as the basis of the relative probabilities of cancer in organs for a nominal "world" population of all ages from which to derive the detriment.

(B105) It would be most useful if one could compare the results obtained for cancer induction per unit dose in specific organs from the Japanese survivors with cancer induced in specific organs per unit dose in other populations and circumstances. This comparison is possible however only in rather few cases. One such would appear to be cancer of the breast, in which the risk for women in different age groups has been compared in a detailed analysis of the atomic bomb survivors, New York mastitis series and Massachusetts fluoroscopy series (Land *et al.*, 1980). The results show that absolute risk in the three series agree quite well, much better than for relative risk. On the other hand UNSCEAR gives a table (UNSCEAR, 1988b, Annex F, Table 36) which seems to

imply that relative risks agree quite well not only in the above three groups but also in the Canadian fluoroscopy series. BEIR V (NAS, 1990) finds in the two mortality series (the life span study in Japan and the Canadian cohort without the Nova Scotia patients) that absolute risks agree while in the three incidence series, relative risks agree better and they preferred a relative risk model. Apparently the information is insufficient, when separated out according to age, to provide definitive answers even in the case of cancer of the breast.

#### B.5.11. Expected years of life lost from fatal cancer in organs vs. sex, age and population

(B106) Calculations can be made of expected years of life lost (e.g. see UNSCEAR, 1988b, Table 70) for different sexes, ages, populations, etc. for site specific and total cancers. A set of tables parallel to those for cancer deaths are obtained. A summary table of ratios based on expected years of life lost, average for males and females, five national populations, two models, age 0-90 y is given in Table B-16. The ratios are broadly similar to those in Table B-15 except that leukaemia is higher, reflecting the shorter latency for leukaemia.

Table B-16. Relative values of expected life lost due to induced cancer among organs averaged for sex, five national populations and two models (multiplicative and NIH), age 0-90 y

Organ	Relative life lost
Oesophagus	0.048
Stomach	0.190
Colon	0.148
Lung	0.154
Breast	0.049
Ovary	0.025
Bladder	0.039
Bone marrow	0.197
Remainder	0.150
All cancer	1.000

#### B.5.12. Fatal cancer in other selected organs

(B107) Not accounted for in the list of organs for which fatal risks are derived from the Japanese data are some organs which are often selectively irradiated and therefore specific information on probability of induced cancer is available and for which relative fatal probabilities are especially useful. Included among these are the thyroid, bone, skin and liver. Each of these tissues shows elevated but nonsignificant relative risks in the Japanese data but additional risk information is available from other sources.

##### Thyroid

(B108) UNSCEAR (1988b, Annex F, p. 493) and BEIR V (NAS, 1990, p. 294) agree that the most current estimates of risk to the thyroid are those presented in *NCRP Report 80* (NCRP, 1985). These estimates give a lifetime risk estimate for fatal cancer of  $0.075 \times 10^{-2}$  Gy<sup>-1</sup>. The fatality rate is stated to be 0.1, thus the incidence is  $0.75 \times 10^{-2}$  Gy<sup>-1</sup>. The value for total cancer is estimated for the high dose range but will be included in Table B-17 as it is because of the presumed linear nature of the thyroid response for external radiation. <sup>131</sup>I was estimated to be about one-fourth to one-third as effective as external radiation (NCRP, 1985; UNSCEAR 1988b).

*Bone surface*

(B109) UNSCEAR (1988b, p. 493) was unable to provide a new estimate of either high or low LET radiation lifetime risk estimates for bone. However, they cited BEIR III (NAS, 1980), (UNSCEAR, 1988b, Table 33, p. 510) as  $27 \times 10^{-4} \text{ Gy}^{-1}$  lifetime for high LET radiation and  $1.4 \times 10^{-4} \text{ Gy}^{-1}$  lifetime for low LET radiation. BEIR V (NAS, 1990) citing BEIR IV (NAS, 1988, p. 237) derives a lifetime incidence of  $2 \times 10^{-2} \text{ Gy}^{-1}$  for  $^{224}\text{Ra}$ , appreciably higher than earlier estimates. However, it appears that a better value, BEIR IV (NAS, 1988, p. 208), allowing for life table analysis, is about  $133 \times 10^{-4} \text{ Gy}^{-1}$ . With a lethality fraction of 0.70, this becomes  $93 \times 10^{-4} \text{ Gy}^{-1}$  or about  $4.7 \times 10^{-4} \text{ Sv}^{-1}$  for a quality factor of 20. Since these are derived from high LET radiation sources with a  $Q$  of 20 the low LET radiation value will be presumed to apply to low doses, i.e.,  $0.047 \times 10^{-2} \text{ Sv}^{-1}$  will be entered in Table B-17.

*Skin*

(B110) The report of the *ICRP Task Group on Skin* (ICRP, in preparation) finds the incidence of cancer in skin to be  $10^{-1} \text{ Sv}^{-1}$ , while the fatality (or lethality) fraction is 0.2% or  $2 \times 10^{-3}$ . This fatal skin cancer risk is presumed to be applicable at low doses and  $0.02 \times 10^{-2} \text{ Sv}^{-1}$  will be entered in Table B-17.

*Liver*

(B111) UNSCEAR (1988b, p. 484) points out that neither the Japanese A-bomb studies nor the spondylitis patients provide definitive risks for induced primary liver cancer and the situation is complicated by metastatic liver cancer (Upton, 1991). The

Table B-17. Lifetime mortality in a population of all ages from specific fatal cancer after exposure to low doses

	Fatal probability coefficient ( $10^{-2} \text{ Sv}^{-1}$ )	
	ICRP (1977)	This report
Bladder	—	30
Bone marrow	20	50
Bone surface	5	5
Breast	25	20
Colon	—	85
Liver	—	15
Lung	20	85
Oesophagus	—	30
Ovary	—	10
Skin	—	2
Stomach	—	110
Thyroid	5	8
Remainder <sup>1</sup>	50	50
Total	125 <sup>2</sup>	500 <sup>3</sup>

<sup>1</sup> The composition of the remainder is quite different in the two cases.

<sup>2</sup> This total was used for both workers and the general public.

<sup>3</sup> General public only. The total fatal cancer risk for a working population is taken to be  $400 \times 10^{-2} \text{ Sv}^{-1}$ .

data from thorotrast studies in West Germany, Portugal, Japan and Denmark yield about 300 fatal liver cancers  $\times 10^{-4} \text{ Gy}^{-1}$  (NAS, 1990, p. 306). With a  $Q$  of 20, one obtains a risk estimate of  $0.15 \times 10^{-2} \text{ Sv}^{-1}$  which can be applied also for low LET radiation. This estimate is somewhat less than the value for the bladder or breast, and is comparatively poorly known.

(B112) The fatal cancer rates attributed to these four organs have been subtracted from the remainder tissues also given in Table B-17.

#### B.5.13. Recommended estimates of probability of fatal cancer for low dose, low dose rate, low LET radiation

(B113) The estimate of probability for total fatal cancer given by UNSCEAR (1988b), for the preferred multiplicative projection model is  $11 \times 10^{-2} \text{ Sv}^{-1}$ , for the Japanese total population (Table B-7). The various estimates of relative probability of fatal cancers available in Tables B-11 to B-15, for the multiplicative or NIH model, yield values for the general population of different countries (0-90 y) of  $6-13 \times 10^{-2} \text{ Sv}^{-1}$  with an average of  $9.5 \times 10^{-2} \text{ Sv}^{-1}$  (Table B-15). The corresponding value obtained from the BEIR V committee for the U.S. population is  $9 \times 10^{-2} \text{ Sv}^{-1}$  or possibly some 20% higher if calculated in the same way as UNSCEAR (see paragraph B93). The "average" of these various values is broadly about  $10 \times 10^{-2} \text{ Sv}^{-1}$  and this value will be used as the nominal risk for acute high dose exposure. Applying the dose and dose-rate effectiveness factor of 2 (see paragraph B62) yields a nominal value of  $5 \times 10^{-2} \text{ Sv}^{-1}$  for the probability of induced fatal cancer in a population of all ages. A smaller value would be obtained for a working population of age 20-64 years, at about  $4 \times 10^{-2} \text{ Sv}^{-1}$  (Table B-7). With the appropriate choice of  $w_R$ , these values apply also to high LET radiation.

(B114) The probability of fatal cancer induction after low dose, low dose-rate irradiation of the total population,  $5 \times 10^{-2} \text{ Sv}^{-1}$ , is distributed among the organs as shown in Table B-17, second column. These values are derived from the distribution of fatal cancers given in Table B-15 multiplied by  $5 \times 10^{-2} \text{ Sv}^{-1}$  with the addition of fatal probabilities for thyroid, bone surface, skin and liver subtracted from the remainder. The values are compared with those given in *Publication 26* (ICRP, 1977) for fatal cancer induction in specific sites in the first column. Evidently there is much uncertainty and a certain arbitrariness in the determination of the distribution of fatal cancer probability among tissues and organs resulting primarily from the transfer between populations and some of their characteristics. More time and information is needed to reduce these uncertainties. The total risk of fatal cancer, on the other hand (Tables B-11 to B-14) is comparatively robust.

#### B.5.14. Detriment

(B115) The detriment must include not only the estimates of fatal cancer but also other deleterious effects of radiation. In what follows the Commission considers four main components of the detriment due to radiation exposure of the whole body at low doses. These include the risk of fatal cancer in all relevant organs, a specific allowance for differences in latency which result in different values of expected life lost for fatal cancer in different organs, an allowance for the morbidity resulting from induced non-fatal cancers and finally an allowance for the risk of serious hereditary disease in all future generations descended from the irradiated individual.

*Life lost*

(B116) In order to make allowance for the differences in expected years of life lost for induced cancer in different organs, it is necessary to obtain the expected years of life lost ( $l$ ) for each fatal cancer as an average for sex, exposure age, national population and both the multiplicative and NIH models. The  $l$  values for bladder, bone marrow, breast, colon, lung, oesophagus, ovary, stomach and remainder can be derived from data in Land and Sinclair (1991, Table 4), and are presented in their Table 10 and here in Table B-18. It should be noted that in those cancers occurring only in females (e.g. breast and ovary) the length of life lost per specific cancer is based on the female data only and is not averaged for males and females. Furthermore  $\bar{l}$ , the average for all cancers, is obtained by dividing the expected years of life lost for all cancers by the total number of fatal cancers as a group. This yields a value of 15.0 years. The values for  $l$  for bone surface, liver, skin and thyroid cannot be obtained in the same way and therefore were arbitrarily set at the same value as  $\bar{l}$ . The values for the correction factor  $l/l$  for each cancer are also shown in Table B-18. The gonads are assigned a period of 20 years of life lost on average for severe genetic disorders, i.e., a correction factor of 1.33.

*Morbidity and detriment*

(B117) The Commission has previously provided a comprehensive discussion on morbidity in *Publication 45* (ICRP, 1984b). While the process is inevitably judgemental, the Commission notes that in any attempt to attach weight to the detriment due to the induction of a curable cancer, importance must be attached to the ease of curing some cancers such as skin, the extreme difficulty of curing some others and the trauma associated with the curative procedures. Some cancers like the breast are probably intermediate between these two situations. Thus the ICRP concluded that to allow for the detriment associated with non-fatal cancers, the detriment of each cancer type includes a

Table B-18. Relative expected life lost per fatal cancer in different organs, averaged for two models, sex and five national populations, age 0-90 y, or per fatal genetic effect

	Life lost (years) $l$	Factor $l/\bar{l}$
Bladder	9.8	0.65
Bone marrow	30.9	2.06
Bone surface	15.0	1.00
Breast	18.2	1.21
Colon	12.5	0.83
Liver	15.0	1.00
Lung	13.5	0.90
Oesophagus	11.5	0.77
Ovary	16.8	1.12
Skin	15.0	1.00
Stomach	12.4	0.83
Thyroid	15.0	1.00
Remainder	13.7	0.91
Gonads	20.0	1.33

$\bar{l}$  is derived from the expected years of life lost for all cancers divided by the total number of fatal cancers, given as a group, and equals 15.0 years.

non-fatal component weighted according to the lethality fraction  $k$ . Thus, if in a given tissue there are  $F$  fatal cancers, the total number of cancers is  $F/k$ . The number of non-fatal cancers is then  $(1-k)F/k$  and the total weighted detriment is  $(F+k(1-k)F/k)$  or  $F(2-k)$ . The nominal weighted effect probability coefficient is then given by multiplying the corresponding fatality probability coefficient by  $(2-k)$ .

(B118) Lethality fractions for cancers in adults were obtained from the latest data available from the National Cancer Institute of the United States (U.S. DHHS, 1989) which gives 5 year survival rates by site (SEER programme) for 1980-85 (Table B-19 column 1). These are too low for full expression of lethality. Also available however are lethality rates for the period 1950-70 (Table B-19 column 2), which are too high by today's standards, because cure rates for this earlier period have now been improved upon. Lethality fractions have been derived as judgement based averages of these two sets of data (column 3 of Table B-19) reflecting the improved treatment for some types of cancer. These lethality fractions are very similar to data obtained recently from Sweden.

(B119) The total detriment is then assessed as outlined in Table B-20. The first column is the fatal cancer probability ( $F$ ) for each organ (Table B-17). The second column includes the contribution for severe genetic disorders (from Section 8, later). The third column lists the relative length of life lost for each fatal cancer (Table B-18) and for genetic effects (see paragraph B116). The fourth column lists the estimates of  $(2-k)$  where values of  $k$  are from Table B-19. The fifth column provides the estimates of detriment as defined by  $F \cdot l/l (2-k)$  for each organ and for the total. The units are in terms of numbers of detrimental occurrences per 10,000 people of all ages per Sv of low dose radiation. The final column represents the relative contributions of each of the organs to the total detriment. [Note: For a working population the total fatal cancer risk is taken to be  $4 \times 10^{-2} \text{ Sv}^{-1}$  and the values of  $F$  for organs are 80% of those listed in Table B-20.

Table B-19. Lethality data for cancers in adults by site (U.S. DHHS, 1989)<sup>1</sup>

	5 year 1980-85	20 year lethality 1950-70	Proposed lethality fraction $k$
Bladder	0.22	0.58	0.50
Bone	—	0.72	0.70
Brain	0.75	0.84	0.80
Breast	0.24	0.62	0.50
Cervix	0.33	0.50	0.45
Colon	0.45	0.62	0.55
Kidney	0.48	0.78	0.65
Leukaemia (acute)	0.98	0.99	0.99
Liver	0.95	0.98	0.95
Lung and Bronchus	0.87	0.96	0.95
Oesophagus	0.92	0.97	0.95
Ovary	0.62	0.74	0.70
Pancreas	0.97	0.99	0.99
Prostate	0.26	0.84	0.55
Skin	—	—	0.002
Stomach	0.85	0.90	0.90
Thyroid	0.06	0.15	0.10
Uterus	0.17	0.35	0.30

<sup>1</sup> Numbers were derived from tables and graphical data of U.S. by F. A. Mettler and W. K. Sinclair.

Table B-20. Relative contribution of organs to the total detriment

	Probability of fatal cancer $F$ (per 10,000 people/Sv)	Severe genetic effects (per 10,000 people/Sv)	Relative length of life lost $l/l$	Relative non-fatal contribution $(2-k)$	Product $F(l/l)(2-k)$ (per 10,000 people/Sv)	Relative contribution
Bladder	30		0.65	1.50	29.4	0.040
Bone marrow	50		2.06	1.01	104.0	0.143
Bone surface	5		1.00	1.30	6.5	0.009
Breast	20		1.21	1.50	36.4	0.050
Colon	85		0.83	1.45	102.7	0.141
Liver	15		1.00	1.05	15.8	0.022
Lung	85		0.90	1.05	80.3	0.111
Oesophagus	30		0.77	1.05	24.2	0.034
Ovary <sup>1</sup>	10		1.12	1.30	14.6	0.020
Skin	2		1.00	2.00	4.0	0.006
Stomach	110		0.83	1.10	100.0	0.139
Thyroid	8		1.00	1.90	15.2	0.021
Remainder	50		0.91	1.29	58.9	0.081
Gonads <sup>1</sup>		100	1.33	—	133.3	0.183
Total	500				725.3	1.000

<sup>1</sup> Gonads (including cancer in ovary).

The severe genetic effects are estimated to be  $0.6 \times 10^{-2} \text{ Sv}^{-1}$  (see later paragraph B159).]

#### B.5.15. Tissue weighting factors

(B120) The relative contributions of the organs to the total detriment (Table B-20, last column) form the basis of the Commission's weighting factors. In considering these relative contributions and recognising that the process of deriving them, let alone the uncertainties in the original data themselves, has large uncertainties, the Commission decided that the values in Table B-20 could be rounded and grouped into a simple system of weights of adequate accuracy for calculations of effective dose. Among many possible systems considered, the Commission selected a very simplified system of weights which would use no more than four groups of weights and require no more than about a factor of 2 rounding between the relative contributions in Table B-20 and the assigned weight. The assigned tissue weighting factors are as follows:

$w_T$		$\sum w_T$
0.01	bone surface, skin	0.02
0.05	bladder, breast, liver, oesophagus, thyroid, remainder	0.30
0.12	bone marrow, colon, lung, stomach	0.48
0.20	gonads	0.20
	Total	1.00

These weighting factors will be used for both a working population and the general population.

#### B.5.16. Uncertainties in risk estimates

(B121) The nominal values of fatal cancer risk, which form the basis of the detriment

following radiation exposure, are not to be regarded as precise and immutable. They are, unfortunately, at this time still subject to many specific uncertainties and to many assumptions involving factors which may be subject to change. Even greater uncertainties arise in the attribution of portions of the total risk to individual organs. It is hoped, and indeed expected, that these uncertainties will diminish in the future as the accumulated experience in exposed populations such as the Japanese survivors increases and as more information develops from a broader variety of human experiences. In the meantime it is useful to consider some of the factors that enter into uncertainties in current estimates and how these have been considered by other bodies involved in the evaluation of cancer risk.

(B122) UNSCEAR discussed uncertainties in risk estimates and in risk projections and treated most of the factors involved but in a general non-quantitative way (UNSCEAR, 1988b, Annex F, paragraphs 513–525). Quantitation of uncertainties is much more difficult but the NIH Ad Hoc Working Group on the Radioepidemiological Tables made some of the first quantitative estimates of uncertainties in their evaluation of probabilities of causation for specific cancer sites (Rall *et al.*, 1985). They considered each of the factors involved and assigned values of the geometric standard deviation (G.S.D) to each of them before deriving a combined G.S.D from all sources for the probability of causation at each cancer site. The BEIR V Committee took a similar approach in developing general estimates of the G.S.D for risk estimates of leukaemia and cancers other than leukaemia, and for males and females. Some of the factors involved are model mis-specification, population differences, dosimetry, sex, age and latency, shape of dose-response relationship and, of course, uncertainties in the base data themselves. Surprisingly the overall estimate of G.S.D for total risk is only about 1.3 although much larger values are indicated for individual organs and individual age groups. However this estimate did not include the shape of the dose-response relationship or the effect of transfer model between populations both of which introduce very considerable uncertainties in the estimates of low dose risk.

(B123) Each of the steps necessary to evaluate overall uncertainty involves the exercise of judgement and is therefore open to debate. At this time it is very difficult to arrive, in any precise way, at a satisfactory measure of overall uncertainty in the nominal values of risk used by the Commission for low dose exposure. The many factors involved and the magnitude of some of these factors mean that the uncertainties can be large both for the nominal total risk and especially for individual organ risks. In view of this, it is perhaps surprising that the Commission distinguishes between the nominal value of  $5 \times 10^{-2} \text{ Sv}^{-1}$  for a population of all ages and  $4 \times 10^{-2} \text{ Sv}^{-1}$  for an adult working population when the uncertainties are clearly greater than this difference. However, in fact, the precise values of the risk are probably not as well known as the strong likelihood that there is a difference between the two populations, the risk for an adult population being less than that for a population of all ages.

#### B.6. Probability of Induced Lung Cancer from Exposure to Radon Progeny

(B124) The induction of lung cancer by long term exposure to radon progeny is a subject of concern because these internally-deposited alpha-emitters contribute the largest fraction of the effective dose from natural background radiation and because of the association between radon exposure and lung cancer. Recent reviews and analyses of epidemiological studies of underground miners and animal laboratory data summarise

the current state of knowledge of the demonstrated and potential health effects of exposure to radon and its progeny. This information is needed to characterise the lung cancer risk associated with exposure to radon and its short-lived daughters in indoor domestic environments (NCRP, 1984a,b; NAS, 1988, 1990; ICRP, 1987; UNSCEAR, 1988b; IARC-WHO, 1988).

(B125) By convention, the concentration of radon daughters is measured in working levels (WL) and cumulative exposures over time are measured in working level months (WLM). The WL is defined as any combination of short-lived radon daughters in 1 litre of air that results in the ultimate release of  $1.3 \times 10^5$  MeV of potential alpha energy; this is approximately the amount of energy emitted by the short-lived daughters in equilibrium with 3.7 Bq (100 pCi) of radon. A WLM is the exposure resulting from inhalation of air with a concentration of 1 WL of radon daughters for 170 working hours. (In the SI system,  $1 \text{ WLM} = 3.5 \times 10^{-3} \text{ Jhm}^{-3} = 3.5 \text{ mJhm}^{-3}$ .)

(B126) The relationship between exposure, measured in WLM, and dose to the target cells and tissues in the respiratory tract is complex and depends on both physical and biological factors, including the physical characteristics of the inhaled air, breathing patterns, and the biological characteristics of the human lung. Radon progeny are formed as condensation nuclei; most attach to aerosols immediately, but a proportion remain unattached. The unattached fraction is an important determinant of the dose received by the target cells in the respiratory tract, because of the efficient deposition of the unattached daughters in the airways. The particle size distribution in the inhaled air also influences the dose to the airways. A further large uncertainty in the estimation of alpha dose results from the unknown depth distribution of the proliferating epithelial cells beneath the mucus sheet in the different bronchial airways.

(B127) Based on different dosimetry models of the lung, the mean absorbed alpha dose to the target cells and tissue in the tracheobronchial region per unit of indoor exposure range from about 4 to 13 mGy per WLM or about 1.2 to 3.7 Gy per  $\text{Jhm}^{-3}$  (NCRP, 1984a; ICRP, 1987; NAS, 1988; James *et al.*, 1988). Because of differences in circumstances of exposure and in the biological and nonbiological factors influencing the dose to target cells in the respiratory tract from radon exposure, it cannot be assumed that exposure to 1 WLM in a home and to 1 WLM in an underground mine results in the same dose of alpha radiation to the cells in the target tissues of the respiratory tract (NCRP, 1984b; NAS, 1988; ICRP, 1987).

(B128) The evidence for lung cancer induction following exposure to radon and its progeny comes from studies of underground miners and extensive animal experiments. Studies have been carried out (and are continuing) on a number of uranium mining cohorts, including Colorado (Hornung and Meinhardt, 1987), Ontario (Muller *et al.*, 1985), Saskatchewan (Howe *et al.*, 1986) and Czechoslovakia (Sevc *et al.*, 1988), and an iron mining cohort in Sweden (Radford and Renard, 1984). The characteristics of cohort size, exposure and lung cancer mortality are indicated in Table B-21. A retrospective study in Newfoundland miners (fluorspar cohort) (Morrison *et al.*, 1988) and a prospective study of miners in New Mexico (Samet *et al.*, 1984; Samet, 1989) provide additional information.

(B129) All these studies indicate a proportional increase of the excess lung cancer frequency with the cumulative exposure to radon progenies, up to exposure levels of about 500 WLM; such a proportional relationship is in agreement with the findings from animal experiments. The data from these cohorts of miners yield a statistically significant excess at cumulative exposures of somewhat less than 50 WLM. This level of statistical

Table B-21. Mortality from lung cancer in underground miners (1976-82)

	Number	Mean exposure (WLM) <sup>1</sup>	Person-years at risk	Number of lung cancer deaths	
				Observed	Expected
Colorado, U.S. (1951-82)	3,347	882	73,642	256	59.1
Ontario, Canada (1955-81)	11,076	37	217,810	87	57.9
Saskatchewan, Canada (1950-80)	6,847	22	114,170	65	28.7
Czechoslovakia (1948-80)	4,043	226	83,836	484	98
Malmberget, Sweden (1951-76)	1,292	98	27,397	51	14.9

<sup>1</sup> 1 WLM = 3.5 mJhm<sup>-3</sup>.

detectability is only about a factor of 2 to 5 higher than the mean lifetime exposure of populations from indoor radon.

#### Lifetime risk

(B130) A number of different lung cancer risk projection models have been used to describe the pattern of risk for the miner cohorts and factors that modify risk. These exposure-time-response models have been used to analyse the epidemiological data obtained from the miner studies. These models require projection of the miner experience during the period of observation to the lifetime of the population at risk. Both additive and multiplicative risk projection models have been applied (Table B-22). Both the NCRP (NCRP, 1984b) and the BEIR IV Committee (NAS, 1988) have demonstrated that excess lung cancer risk varies with time since exposure. In these models, radon exposures more distant in time have a smaller impact on the age-specific excess risk than more recent exposures. Also both the Commission (ICRP, 1987) and BEIR IV (NAS, 1988) include a dependence of risk on age at exposure. In the BEIR IV (NAS, 1988) model the age-specific excess relative risk is higher for younger persons and declines at

Table B-22. Lifetime probability of fatal lung cancer due to lifetime exposure to radon progeny

Evaluation	Projection	Probability of cancer death		
		per unit exposure <sup>1</sup> ( $10^{-6}$ /WLM)	per unit exposure <sup>1</sup> ( $10^{-3}$ /Jhm <sup>-3</sup> )	unit energy inhaled <sup>2</sup> ( $10^{-2}$ /joule)
NCRP (1984b) ICRP (1987)	Modified absolute	130	37	31
	Constant absolute	150	43	36
EPA (1986)	constant relative	230 <sup>3</sup>	66 <sup>3</sup>	55 <sup>3</sup>
	Constant relative	115-400 <sup>4</sup>	33-110 <sup>4</sup>	27-95 <sup>4</sup>
(Puskin and Yang, 1988; Puskin and Nelson, 1989)	Arithmetic estimate	150-450	43-128	36-110
	BEIR IV (1988)	Modified relative	350 <sup>3</sup>	100 <sup>3</sup>

<sup>1</sup> Potential alpha energy exposure.

<sup>2</sup> Potential alpha energy inhaled.

<sup>3</sup> Referring to a global reference population with a baseline lung cancer rate of 400 cases/10<sup>6</sup> persons per year averaged over all ages and both sexes.

<sup>4</sup> Referring to the population of the U.S.A. only.

higher ages. In both *Publication 50* (ICRP, 1987) and BEIR IV (NAS, 1988) reports the primary risk data for miners (i.e. males) have been used for both males and females.

(B131) Comparisons of estimates of lifetime probability of lung cancer mortality due to lifetime exposure to radon progeny in terms of WLM made by different committees are listed in Table B-22. Lifetime probabilities of fatal lung cancer based on constant or modified, relative risk projection models yield primarily values of the lifetime excess relative risk. Their conversion to values of the absolute lifetime risk depends on the spontaneous or baseline lung cancer rate of the study population. The data in *Publication 50* (ICRP, 1987) and the BEIR IV report (NAS, 1988) both yield nearly the same excess relative lifetime risk per unit exposure. One reason for the difference in the absolute lifetime risk estimates is the difference in baseline lung cancer rates in the different populations (see footnotes to Table B-22).

(B132) The absolute lifetime risk coefficients refer to populations with high life expectancies (70–80 years at birth) and represent population-averaged values over all ages, both sexes and over non-smokers and smokers. They indicate lung cancer probability coefficients in the broad range of  $1-4 \times 10^{-4}$  WLM<sup>-1</sup> or  $3-10.0 \times 10^{-3}$  mJ<sup>-1</sup> inhaled potential alpha energy of radon progeny, respectively. This range is due in part to the different time projection models applied, and the different baseline lung cancer rates of the reference populations.

(B133) The different risk approaches should be regarded as an attempt to quantify the possible lung cancer risk associated with the indoor exposure to radon progeny. In the future, these risk projection models and the values they generate will improve as more realistic modifications are introduced on the basis of the continuing analysis of data from radon-exposed miners and from other epidemiological studies on radiation-induced lung cancer. This should narrow the uncertainties inherent in the present approaches.

#### *Smoking and radon exposure*

(B134) Smoking is the most important single causal factor in lung cancer, and the smoking habits of the Rn-exposed miners are important for the interpretation and evaluation of the associated risk from inhaled radon progeny. A description of the interaction between radon daughters and cigarette smoking for the induction of lung cancer is required. To date, the epidemiological evidence allows no firm, quantitative conclusion on the combined carcinogenic effect of inhaled radon progeny and cigarette smoke. Certain of the larger studies on lung cancer in Rn-exposed miners suggest a multiplicative or promoting effect of smoking, this finding is supported by animal experiments, but not by some smaller epidemiological studies. The Colorado uranium miner study (Hornung and Meinhard, 1987) is the largest case-control study on lung cancer in miners with reliable known smoking history; analysis yields a somewhat less than multiplicative interaction, and rejects an additive model.

(B135) The BEIR IV Committee (NAS, 1988) chose a multiplicative interaction for its risk projection which leads to the conclusion that the lifetime lung cancer risk for heavy smokers from exposure to radon progeny might be 6–10 times higher than that for non-smokers. The risk analysis in the Commission's study (ICRP, 1987) indicates that for equal radon progeny exposure conditions the attributable lifetime risk of non-smokers might be about a factor of 4 lower than population-averaged risk coefficient given in Table B-22. Thus exposure to radon progeny not only increases the lung cancer risk in smokers but also causes a significant risk in non-smokers.

(B136) Many epidemiological investigations of the lung cancer risk associated with

radon-daughter exposure in homes have been carried out, but the populations have not been sufficiently large, and the results have been inconclusive; these studies are presently inadequate for purposes of risk estimation for the general population. The risk projection models are therefore based on occupational exposure data. The transfer of risk estimates from the occupational setting to the indoor domestic environment requires several assumptions, primarily concerning the different distributions by age and sex of the population, the differences in durations of exposure, breathing rates, smoking habits and other biological, physical and physiological factors. Of greater importance seem to be the differences between the mining and domestic environments with respect to the physical characteristics of the inhaled air (including the possibility of uranium dust in mine air), the fraction of radon daughters unattached to particles, the aerosol characteristics as regards the particle size and distribution, and the equilibrium of radon with its daughters.

(B137) The overall influence of these factors that modify lung cancer risk is apparently smaller than the uncertainties of the dosimetry and the limitations of the primary epidemiological input data from the radon-exposed miners. Therefore, the range of risk coefficients given in Table B-22 may be representative also for the lung cancer risk in the general population in the domestic environment of exposure to radon progeny. At present, this is considered to be the case provided assumptions are made concerning the extension of the epidemiological findings in miners across the entire lifespan, the interaction of cigarette smoking and exposure to radon daughters, the application of risk projection models, the factors affecting the values estimated, and the unit dose per WLM to the bronchial epithelium in the occupational and environmental settings, and until more direct and reliable information becomes available. For all of these reasons the exposure of the public from radon is under further study by the Commission.

#### **B.7. Examination of the Evidence of Induced Cancer in Humans after Low Doses**

(see NAS, 1990; MacMahon, 1989; Modan 1991)

(B138) The risk factors derived from the Japanese A-bomb survivors (and for that matter most often from therapeutically irradiated populations also) relate to high dose, high dose rate exposures. One of the largest uncertainties in the estimation of the probability of cancer induction at low doses is extrapolating this information to the low dose, often low dose rate, circumstances (e.g. a few mGy y<sup>-1</sup>) most often encountered in routine radiation protection. This is usually done by applying a dose and dose rate effectiveness factor (paragraph B62) which reduces the risk coefficient per unit dose derived from high dose, high dose rate exposure. It would be extremely valuable if quantifiable information were available in human populations directly for low dose exposure.

(B139) Numerous studies of low dose exposure exist in the literature. It is helpful to group them into categories although this is not simple. Nevertheless, the categories may include:—

- (a) Studies of people exposed to nuclear sources such as fallout, presence at weapons tests or around reactors. These include well known studies on persons in counties in Utah believed to have shown higher incidences of leukaemia following fallout from weapons tests (Lyon *et al.*, 1979; Machado *et al.*, 1987), U.S. and U.K. veterans exposed during weapons testing and subsequently examined for cancer incidence (Caldwell *et al.*, 1983; Robinette and Jablon 1983; Jablon 1987; Darby

*et al.*, 1988) and the leukaemia clusters apparently observed around nuclear sites in the U.K. about which much has been written in recent years (Black, 1984; Cook-Mozaffari *et al.*, 1989a,b; Kinlen, 1988; Gardner *et al.*, 1990; Forman *et al.*, 1987).

- (b) Occupational exposure sources include the studies of the Hanford workers (Mancuso *et al.*, 1977; Gilbert and Marks, 1979; Gilbert *et al.*, 1989a,b) shipyard workers (Najarian and Colton, 1978; Rinsky *et al.*, 1981; Stern *et al.*, 1986); UKAEA and UK Atomic Weapons Establishment workers (Beral *et al.*, 1985, 1988); and a recent study of USSR workers involving relatively high doses (Wainson *et al.*, 1990).
- (c) Fetal exposures during diagnostic x-ray examinations of the mother. The original studies (Stewart *et al.*, 1958; MacMahon, 1962), have been followed by further appraisals (Kneale and Stewart, 1980; Monson and MacMahon, 1984). They have also been reassessed (Bithel and Stiller, 1988) and additional studies have been made (Harvey *et al.*, 1985). (See also Section B.9.)
- (d) Medically irradiated populations such as in the x-ray treatment of tinea capitis in which other organs such as the thyroid or breast were also irradiated (Modan *et al.*, 1989).
- (e) Studies of "high" background areas, in India (Gopal-Ayengar *et al.*, 1971), in Brazil (Barcinski *et al.*, 1975), in Colorado, Denver (NAS, 1980) and in China (Wang *et al.*, 1990; Wei *et al.*, 1990).

(B140) Studies at low doses have the advantage that no uncertain dose reduction factors are needed and more suitable population characteristics may exist in the study population than for some highly exposed groups. However several problems and sources of bias may confound their interpretation. These include one or more of the following: (a) small sample size, (b) lack of adequate controls, (c) extraneous effects other than those of radiation, (d) inadequate dosimetry, (e) confounding social factors and (f) "positive" reporting i.e., lack of reporting of negative results. Furthermore, a range of doses is rarely available to establish the strength of the association. Some of these problems occur also in high dose studies but their importance is less in such circumstances. Attention should be drawn here especially to the critical importance of sample size, signal to noise ratio and lack of information about confounding factors (Land *et al.*, 1980). These sources of bias are discussed in more detail, as are many of the studies themselves. (see NAS, 1990; Modan, 1991).

(B141) Some of the low dose studies, (e.g. Beral *et al.*, 1985; Gilbert *et al.*, 1989b) provide risk estimates, but with rather wide confidence limits which however include the values derived from high dose studies as well as zero or below. In the recent study of Soviet workers (Wainson *et al.*, 1990), relative risks are derivable which are quite similar to those from high dose studies.

(B142) A significant proportion of the reported low dose studies yield risk estimates higher, for certain sites, than those derived from high dose studies. Many of these are undoubtedly spurious because of one or more of the various methodological problems discussed above. Some remain puzzling nevertheless. On the other hand, some of the studies cited show significant deficits in the response in certain sites relative to the risk estimates derived from high dose data. Some even show negative correlations between the induction of cancer (all cancer and some selected sites) and dose in the low dose range. In addition many negative studies are not reported. In summary, none of the findings for specific sites are sufficiently strong to provide a quantitative basis for reassessing the current estimates of fatal cancer probability derived from high dose studies.

## B.8. Stochastic Effects: Hereditary (see Sankaranarayanan, 1991)

### B.8.1. Introduction

(B143) Since the publication of the basic recommendations in *Publication 26* (ICRP, 1977), new information that bears on the estimation of the probability of radiation-induced hereditary effects in human populations has become available. However, direct human radiation genetic data continue to remain limited (since only studies in the Japanese survivor progeny provide direct human data and that only in the form of upper bounds to the estimate of risk). Data from experimental mammalian (chiefly the mouse) radiation genetic studies, as in the past, constitute the principal basis for these calculations. What the experimental data provide however, are estimates of mutation rates; these are converted, using certain assumptions, into estimates of probability of radiation-induced hereditary disorders in the human population. Such extrapolations inevitably involve a number of assumptions and associated uncertainties.

(B144) Two kinds of radiation-induced genetic damage are considered important: gene mutations (alterations in the elementary units of heredity, namely the genes) and gross chromosomal aberrations (alterations in the structure or number of chromosomes). In a broad sense, a mutation is considered dominant when its effect is manifest in the first generation progeny (and inheritance of the mutation from one of the two parents will suffice) and recessive when its effect is not so manifest. For the effect of recessive mutations to be expressed, the same gene mutation needs to be inherited from both the parents. Depending on location, mutations are called X-linked if they are in genes located on the X-chromosome and autosomal if they are in genes located on any of the other chromosomes. Structural chromosomal aberrations include, among others, deletions and duplications of parts of chromosomes and exchanges of segments between different chromosomes (e.g., translocations). Numerical chromosomal aberrations include loss and gain of whole chromosomes. A small proportion of these chromosomal aberrations result in congenital abnormalities.

### B.8.2. Methods for estimation of radiation-induced hereditary disorders

(B145) The methods that are used in estimating the probability of hereditary disorders can be broadly grouped under two headings: the "doubling dose method" and the "direct method". These are roughly comparable respectively, to the "relative risk method" and "absolute risk method" used in estimation of cancer probabilities. It is the doubling dose method that is favoured by the Commission.

(B146) The doubling dose is the amount of radiation necessary to produce as many mutations as those that occur naturally in a generation. The estimate of doubling dose used (UNSCEAR, 1977, 1982, 1986, 1988c), and in *Publication 26* (ICRP, 1977) is 1 Gy and is based on mouse data and low dose rate exposure. In its 1990 report, the BEIR V Committee (NAS, 1990) used the same estimate for the doubling dose, this being also the lower 95% confidence limit of the estimate based on the negative findings of the genetic studies in Hiroshima and Nagasaki.

(B147) With the doubling-dose method, the probability of excess cases of hereditary disorders due to radiation exposure is estimated relative to the prevalence of naturally-occurring disorders in the population and thus implies equal induced mutation rates in both sexes. For a population continuously irradiated at low doses, this probability at equilibrium (see below) per unit dose is equal to the prevalence of naturally-occurring hereditary disorders divided by the doubling dose. The rationale is that, under normal conditions, there is an equilibrium in the population between those mutations that arise

and those that are eliminated by selection every generation. With continuous irradiation (and the influx of new mutations that it entails), the population will eventually reach a new equilibrium, and it is the expected additional probability at the new equilibrium that the method allows one to estimate. The increased probability of disorders in the first generation progeny is then estimated from that at equilibrium by making certain assumptions.

(B148) When the population is exposed to radiation only once, there will be an increase in the proportion of mutant genes, but the number will gradually (over a number of generations) decay back to the original equilibrium value. Population genetic theory (Crow and Denniston, 1985) predicts that, numerically, the integrated probability of excess genetic damage over all future generations following a single radiation exposure will be the same as that at equilibrium under conditions of continuous irradiation with that same dose in every generation. Thus the estimate of probability of disorders under equilibrium conditions can be taken to represent the total probability following a single generation radiation exposure.

(B149) Implicit in the use of the doubling-dose method is the assumption that there is a known proportional relationship between mutation and disease. This is the case for autosomal dominant and X-linked disease, but not for disorders of complex aetiology (i.e., multifactorial disorders, see below). It is also assumed that the spectrum of induced mutations is similar to that for spontaneous mutations.

(B150) With the direct method, the absolute probability of occurrence of genetic disorders due to radiation-induced gene mutations in the first generation progeny is estimated from mouse data on rates of induction of dominant (skeletal and cataract) mutations; likewise, the absolute risk of congenitally malformed births due to induced chromosomal aberrations is estimated from cytogenetic data obtained in primate species. These calculations do not rely upon knowledge on the natural prevalence of genetic disorders in the population. However, assumptions are needed to bridge the gap between the experimental animal data on germinal mutational changes and estimates of genetic disorders in the progeny. These assumptions must take into account the radiosensitivity differences between the species, the germ cell stages in both sexes, the transmission rates, doses and dose rate relationships and relative viabilities of the aberration types.

(B151) The term "hereditary disorder" as used here denotes a pathological condition arising as a consequence of a mutation or chromosomal aberration transmitted from one human generation to the next. Conventionally, these disorders are classified into three groups (i) mendelian (i.e., those due to mutations in single genes and which follow Mendel's laws of inheritance; they include autosomal dominant, autosomal recessive and X-linked disorders); (ii) chromosomal (due to either numerical or structural abnormalities of chromosomes) and (iii) multifactorial (resulting from the joint action of multiple genetic and environmental factors) (Czeizel and Sankaranarayanan, 1984). The third group includes congenital abnormalities present at birth and common disorders of adult life (Czeizel *et al.*, 1988). (For examples for these different kinds of disorders see Sankaranarayanan, 1991.)

(B152) The prevalences of naturally-occurring genetic disorders in a typical western-type population is currently estimated as follows: autosomal dominant and X-linked, 1.0% (0.9%+0.1%); autosomal recessive, 0.25%; chromosomal (including those associated with structural and numerical chromosomal aberrations), 0.38%. Earlier studies indicated 4.3% and 4.7%, respectively, for congenital abnormalities and for the other multifactorial disorders, but these figures were revised upwards to 6.0% (con-

genital abnormalities) and about 65% (other multifactorial disorders) (Trimble and Doughty, 1974; UNSCEAR, 1986, 1988c). The last of these figures refers to the total number of disorders per 100 individuals (i.e., a given individual may have more than one condition).

(B153) The degree of severity of these different disorders varies over a wide range. Most autosomal recessive, X-linked and chromosomal disorders appear in infancy or childhood. Autosomal dominant disorders identified at birth or childhood constitute only a small proportion of the total prevalence of 0.9% and the commoner forms first appear in adult life. Congenital abnormalities are present at birth. The other common multifactorial disorders have onset in adulthood. About one-third to one-half of all the known naturally-occurring hereditary disorders may be deemed severe and equivalent in severity to the fatal cancers, either because they occur in early life or because they are regarded as detrimental as lethal diseases in adult life (e.g. Huntington's disease).

### B.8.3. Estimates of probability of hereditary disorders

(B154) During the past decade or so the most widely cited estimates of the probability of hereditary disorders were made by UNSCEAR and the BEIR Committees and are summarised in rounded numbers in Table B-23. Details of the estimates for the various categories of genetic effects can be found in the original reference (Sankaranarayanan, 1991). The BEIR and UNSCEAR estimates have not differed greatly and the major components have changed little over this period. However in the latest reports, the bulk of the probabilities for the inducible multifactorial disorders have not been estimated. Consequently, a component of the total genetic detriment has not been included in the estimates by these Committees.

(B155) In *Publication 26* (ICRP, 1977) estimates of probability used were somewhat higher than those given in UNSCEAR (1977). It was stated that the probability of serious hereditary disorders within the first two generations was  $1 \times 10^{-2} \text{ Sv}^{-1}$ . When account is taken of exposure likely to be genetically significant, i.e., exposures of the younger section of the population that is capable of producing children, this probability became  $0.4 \times 10^{-2} \text{ Sv}^{-1}$ .

Table B-23. Probability of severe hereditary effects estimated using the doubling dose method after 1 Gy low dose rate, low LET radiation to the parental population. The doubling dose assumed is 1 Gy

	Doubling dose (Gy)	Natural prevalence of genetic disorders ( $10^{-2}$ )	Radiation-induced probability ( $10^{-2} \text{ Gy}^{-1}$ )		
			First generation	Second generation	All generations
UNSCEAR 1977	1	10.51	0.63	—	1.85
UNSCEAR 1982	1	10.63	0.22	—	1.50
UNSCEAR 1986	1	1.63	0.18	—	1.04
(excl. multifactorial)					
UNSCEAR 1988	1	~1.30	~0.18	0.14	~1.20
(excl. multifactorial and numerical chromosomal)					
BEIR 1980	0.5-2.5	10.70	0.15-0.75	—	0.60-1.10
BEIR 1990	1	3.6-4.6	0.15-0.40	—	1.15-2.15
(incl. congen. abnorm., excl. common multifactorial)					

#### B.8.4. Current status of the Commission's assessment of hereditary disorders

(B156) The Commission takes into account the most recent information and assessments (UNSCEAR, 1988c; NAS, 1990) which are basically in agreement with one another. The UNSCEAR (1988c) value (which excludes the multifactorial disorders) is 120 cases of hereditary disorders per Gy of low LET radiation at equilibrium (i.e. for all generations) in  $10^4$  live births, i.e.,  $1.2 \times 10^{-2} \text{ Sv}^{-1}$ . However, the genetically significant exposure in a population will be less. If the mean age at reproduction is 30 years and the average life expectancy is 70 to 75 years, the dose received at 30 y is about 40% of that delivered to the entire population in a lifetime, i.e., the probability of genetically significant harm is  $0.5 \times 10^{-2} \text{ Sv}^{-1}$ .

(B157) The corresponding probability per caput in the first two generations is  $0.1 \times 10^{-2} \text{ Sv}^{-1}$  which is only 1/4 of the  $0.4 \times 10^{-2} \text{ Sv}^{-1}$  assessed by the Commission in 1977. The main reason for this difference is that the multifactorial diseases are not included in the present estimate so far. The contribution attributable to this class of highly heterogeneous diseases can only be very approximately and tentatively estimated, as follows.

(B158) With the assumption of a prevalence of about 70%, a mutation component of 5% (an assumed "reasonable value"), and a doubling dose of 1 Gy (as derived for other heritable diseases), the incidence probability over all generations per caput of the total population is about  $1.4 \times 10^{-2} \text{ Gy}^{-1}$  ( $3.5 \times 10^{-2} \text{ Gy}^{-1} \times 30/70$ ). Because some of the multifactorial diseases are less detrimental than those discussed in paragraph B153, this probability should not be added as such without some weighting for the severity of the effects. This weighting is necessarily somewhat arbitrary. It is proposed that the probability is reduced to  $0.5 \times 10^{-2} \text{ Gy}^{-1}$ , i.e. weighted by a factor of about 1/3. The total probability of severe hereditary effects is then assessed at  $1.0 \times 10^{-2} \text{ Gy}^{-1}$ . A further weighting, in proportion to the number of years lost if the effect occurs, is needed to make the detriment comparable to death from radiation-induced cancer.

(B159) The probability per caput in the total population is assumed to be about 40% of the corresponding probability in a reproductive population, 40% being approximately the ratio of the reproductive/total population, 30/70. For a working population, the reproductive fraction is  $(30 - 18)/(65 - 18) \approx (12)/(47) \approx 0.25$ . The probability per caput for workers is therefore  $(12)/(47) \cdot (70)/(30) \approx 60\%$  of  $1 \times 10^{-2} \text{ Sv}^{-1}$ , which is  $0.6 \times 10^{-2} \text{ Sv}^{-1}$ .

### B.9. Effects on the Embryo and Fetus (see UNSCEAR, 1986b; Schull, 1991)

(B160) The principal effects of irradiation on the mammalian fetus include (a) lethal effects in the embryo, (b) malformations and other growth and structural changes, (c) mental retardation, (d) induction of malignancies including leukaemia and (e) hereditary effects (UNSCEAR, 1986b).

#### B.9.1. Lethal effects in the embryo

(B161) Lethal effects can be induced in experimental animals by relatively small doses (such as 0.1 Gy) before or immediately after implantation of the embryo into the uterine wall (UNSCEAR, 1986b, Table 15). They may also be induced after higher doses during all stages of intra-uterine development.

(B162) Although pregnancy loss is known to occur following exposure to ionising radiation (see e.g. Yamazaki *et al.*, 1954) data on the probability of fetal death at a

particular stage in human pregnancy, for different doses, are sparse. It is difficult therefore to develop projections of risk to the human embryo or fetus that encompass all of the recognised hazards.

#### B.9.2. Malformations

(B163) Malformations may be induced which are characteristic of the period of organogenesis at the time of exposure and especially during the most active phase of cell multiplication and differentiation in the structures of concern. Growth disturbances without malformations may also occur at all stages of development especially in the latter phase of pregnancy. These changes appear to result mainly from the killing of cells. Dose-effect relationships for the induction of major teratological malformations in the embryo and fetus of experimental animals are usually curvilinear and become more complex in shape as the development of the relevant structure proceeds. Dose thresholds may well apply to these effects. Such thresholds have been observed in rats and mice (UNSCEAR, 1986, Annex C, Table 15) and similar thresholds may obtain in man. Malformations can, also, of course, arise spontaneously (UNSCEAR, 1986, Annex A, Table 1).

#### B.9.3. Mental retardation

(B164) Modified development of the human brain structures after radiation exposure has been described. It has been found to result in a dose-related increase in mental impairment of varying degree, up to severe mental retardation of cognitive functions. In accordance with events in other anatomical malformations, it is most effectively induced at the time when the relevant tissue, i.e. the brain cortex is being formed. The induction is thought to be associated with impaired proliferation, differentiation and migration of neural elements. The process is described in *Publication 49* (ICRP, 1986). A more recent report (Schull *et al.*, 1989) takes account of some reassessment of the cases and the effect of changes in the dosimetry (from T65D to DS86) at Hiroshima and Nagasaki, where most of the human information is derived.

(B165) Briefly, mental retardation was not observed to be induced by radiation prior to 8 weeks from conception, or after 25 weeks. During the most sensitive period, 8-15 weeks after conception, the fraction of those exposed which became severely mentally retarded increased by approximately  $0.4 \text{ Sv}^{-1}$ . For exposure during weeks 16-25, it increased by about  $0.1 \text{ Sv}^{-1}$ . By applying the DS86 dosimetry, and eliminating two cases of severe mental retardation for which causal association with *in utero* irradiation (8-15 weeks) could be discarded with high probability, it was demonstrated that the dose-response relationship, including a threshold with a lower bound of 0.12 to 0.2 Gy, was more likely than the linear, non-threshold one (Otake *et al.*, 1990). The linear, non-threshold response appeared, *a priori*, unlikely, in view of the presumed deterministic nature of the phenomenon considered. Whether the apparent absence of effects on mental retardation in the first two months after conception merely reflects the fact that embryos exposed at this time commonly fail to survive to an age when mental retardation would be recognised, is unclear.

(B166) Mental impairment of lower severity is also apparent in children exposed *in utero*. This is manifested as a dose-related decrease in intelligence test scores, changes in the occurrence of major features of physical development, impaired school performance, susceptibility to seizure, and possibly other effects. Evidence of such impairment is still

being collected among those exposed *in utero* at the time of the A-bombs in Hiroshima and Nagasaki.

(B167) Since the average IQ score decreases as dose increases without an increase in the variance of the test scores, the decrease in intelligence test scores can be described as a uniform downward shift of the IQ curve (Figure B-7). For exposures *in utero* during the most sensitive period (weeks 8–15), the shift has been estimated to be about 30 IQ units, (i.e. about two standard deviations) per Sv (Otake *et al.*, 1988). This shift of the IQ curve towards lower values must increase the fraction of mentally retarded individuals with increasing dose and suggests that the fall in IQ and the increase in severely mentally retarded with dose are interrelated. If the shift can be quantitatively related to the dose, then the resulting increase of the fraction can be calculated for given doses and compared with the fraction observed.

(B168) If individuals with IQ less than  $100 - x_m\sigma$  are considered mentally retarded, the corresponding fraction  $f$  (without radiation exposure) will be

$$f(x_m) = \frac{1}{\sqrt{2\pi}} \int_{-\infty}^{-x_m} e^{-x^2/2} dx$$

With a shift of 30 IQ units (i.e.  $2\sigma$ ) per Sv, the shift after a dose  $H$  will be  $\Delta x = -2H$ . This shift will bring an additional fraction,  $\Delta f$ , below IQ  $100 - x_m\sigma$ .  $\Delta f$  can be calculated as

$$\Delta f(x_m, H) = \frac{1}{\sqrt{2\pi}} \int_{-\infty}^{-x_m - 2H} e^{-x^2/2} dx$$

If, for example,  $H = 1$  Sv, then  $\Delta f$  would be 0.4 if  $-x_m = 2.205$ , corresponding to IQ 67. This shows that the observation of an increase of the fraction mentally retarded by  $0.4 \text{ Sv}^{-1}$  can be consistent with a shift of 30 IQ units per Sv.

(B169) The increase of  $f$  by  $0.4 \text{ Sv}^{-1}$  can also be presented as a probability of 40% per Sv of being classified as severely mentally retarded. However, the stochasticity is then not in the biological event but in the uncertainty as to whether the individual, without radiation, would have had an IQ low enough to be reduced below  $100 - x_m$  by the dose.

(B170) At low doses (although here not assuming any DDREF > 1), and assuming the

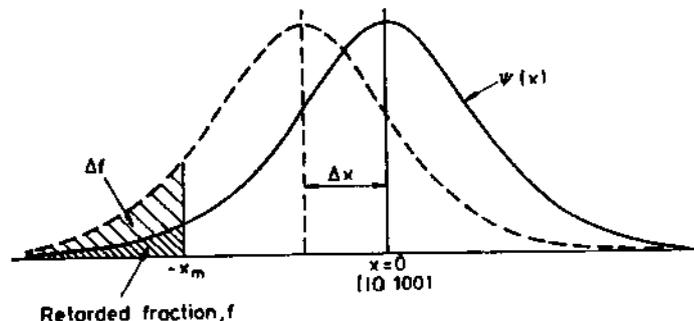


Fig. B-7. The shift of the IQ curve by 30 IQ units or  $2\sigma$  per Sv, i.e.  $\Delta x = 2H$  if  $H$  is the dose equivalent expressed in Sv. The variable  $x$  is the number of standard deviations below (-) or above (+) IQ 100.  $-x_m$  denotes the number of standard deviations below IQ 100 to classify an individual as mentally retarded.

IQ shift is proportional to the dose (an assumption which will overestimate the risk), the increase of  $f$  can be calculated as

$$\Delta f = \Delta x \cdot \psi(x_m) = 2H \cdot \frac{1}{\sqrt{2\pi}} \cdot e^{-x_m^2/2}$$

This will give the following values for  $\Delta f/H$ :

$-x_m = 2$	$\Delta f/H = 10.8\%/Sv$	at IQ 70
2.205	7 %/Sv	67
2.5	3.5%/Sv	62.5
3	0.9%/Sv	55

(B171) The mathematical illustrations in the previous paragraphs are merely intended to demonstrate that during the 8–15 week sensitive period:—

- the two relationships derived from the observations are (a) an increase of  $f$  by  $0.4 \text{ Sv}^{-1}$  and (b) an IQ shift of 30 units per Sv can be quite compatible;
- the increase of  $f$  should not be expected to be linear with dose;
- at low doses, the increase of  $f$  per unit dose would be expected to be substantially less than  $0.4 \text{ Sv}^{-1}$ ;
- the observed shift of 30 IQ units per Sv is best suited to describe the risk;
- if both observations are correct, the most likely interpretation is that the dose required to cause an IQ change large enough to make an otherwise normal individual mentally retarded would be high, while the dose that would bring an individual with potentially low IQ over the borderline may be a few tenths of a Sv (the magnitude of the required doses follows from the shift of 30 IQ units per Sv).

#### B.9.4. Cancer induction including leukaemia

(B172) Irradiated fetuses seem to be susceptible to childhood leukaemias and other childhood cancers which are expressed during approximately the first decade of life. The evidence for this, which comes mainly from the exposure of the mothers to diagnostic x radiation is only marginally at variance with direct observations on the Japanese survivors. Thus at the present time it is considered wise to regard the special susceptibility as real even at very low doses. The risk of fatal childhood cancer due to prenatal exposure has been estimated to be  $2.8 \times 10^{-2} \text{ Sv}^{-1}$  (NAS, 1990). Constancy of risk throughout pregnancy was assumed. A different estimate, based on essentially the same data (Gilman *et al.*, 1989) seems substantially higher ( $13 \times 10^{-2} \text{ Sv}^{-1}$ ). The authors stress that the risk in the first trimester appears substantially larger than that found in the 2nd and 3rd trimester, but this is not established and different views are also held (Muirhead and Kneale, 1989).

(B173) The development of excess cancers later in life following *in utero* irradiation by the A-bombs, has evidently not reached completion. Recent results (Yoshimoto *et al.*, 1988) for the period 1950–1984 and using the absorbed dose (DS86) to the mother's uterus, indicate an increased incidence of cancers in later life in those irradiated *in utero*. This incidence is comparable with the values for those irradiated postnatally, but the study of neither group is complete.

## B.10. Effects on the Skin

### B.10.1. Introduction

(B174) Ionising radiation causes both deterministic effects and cancer induction in the

exposed skin and both must be considered in the radiation protection of the skin. The Commission's current recommendation (ICRP, 1977) on dose limit for the skin is based on deterministic effects. However, in 1978 (ICRP, 1978) the Commission derived a weighting factor of 0.01 for stochastic effects on the skin, based on a skin cancer mortality of  $10^{-4} \text{ Sv}^{-1}$  compared with a total stochastic risk of  $1.65 \times 10^{-2} \text{ Sv}^{-1}$ .

(B175) Effects on the skin have recently been the subject of a detailed examination by a Task Group of Committee 1 of ICRP. The results of that study will be published by the Commission (ICRP, in preparation). A summary of the main findings of the study is presented in the following paragraphs.

#### B.10.2. Deterministic effects

(B176) The effects in the skin of greatest concern and importance in radiation protection are those from exposures to beta particle radiation of various energies and low energy gamma rays, because damage that may be caused by more penetrating x and gamma rays will generally be limited by dose limits to other organs. Exposure to very high doses over a very small area from moderate to high energy beta rays, such as can occur with radioactive particles, in particular, the so-called "hot particle", pose a special problem. Because of the very low penetration of alpha particles, radiation doses from alpha particles could be high in the superficial layers of the skin without appreciable dose to the cells of the basal layers. There have not been any reports of deterministic effects resulting from alpha particle exposure.

##### Acute effects

(B177) The major acute deterministic effects are: (1) moist desquamation which results from damage to cells of the basal layer of the epidermis after high dose acute exposure of the skin to moderate to high energy radiations or low energy x rays. With lower doses only erythema and dry desquamation may occur; (2) acute ulceration which results from interphase death of fibroblasts and vascular endothelial cells may be seen with irradiation from "hot particles"; and (3) acute epithelial necrosis which is caused by interphase death of post mitotic suprabasal cells in the epidermis after exposure to low energy beta particles of energies  $\leq$  about 0.2 MeV maximum energy.

(B178) The assessment of effects is complicated by the multiplicity of targets at different depths which makes it difficult to select a single depth at which to specify the dose to the skin. Some deterministic effects occur at shallow depths but the depths at which the most serious effects arise are estimated to be 300–500  $\mu\text{m}$ . Nevertheless conservatism suggests that the shallow depths be chosen for monitoring specifications. It is also difficult to select an area over which to average the dose since the probability of occurrence of deterministic effects is influenced by the size of the area exposed, as well as the energy of the radiation, the uniformity of exposure and the dose.

(B179) It is accepted clinically that to prevent deterministic effects in the skin, the dose must be reduced as the size of the radiation field is increased. For example,  $^{90}\text{Sr}/^{90}\text{Y}$  sources cause moist desquamation in 50% of the irradiated fields ( $\text{ED}_{50}$ ) after 70 Gy for 5 mm diameter sources but after only 27 Gy for sources of 23 mm diameter or up to 40 mm diameter (Hopewell *et al.*, 1986).

(B180) The influence of the energy of the beta radiation is also marked. The  $\text{ED}_{50}$  doses to produce acute radiation effects were about 30, 70 and 340 Gy measured at a depth of 16  $\mu\text{m}$ , for beta particle radiation of maximum energy 2.27, 0.97 and 0.225 MeV, respectively (Charles *et al.*, 1989).

(B181) The threshold for acute exposures of large areas is about 20 Gy. Protraction of the irradiation decreases the effect and at a dose rate of  $0.4 \text{ Gy h}^{-1}$  no acute tissue breakdown was found with total doses of about 100 Gy (Hopewell, personal communication).

##### Late effects

(B182) Dermal atrophy and damage to the vasculature (including telangiectasia) are the main late effects of acute exposures (Reinhold *et al.*, 1989) and also chronic exposures from moderate to high energy radiations. Dermal atrophy, detected as induration of the skin, a minor detriment, can occur at doses below the threshold for acute breakdown of the skin and thus could be considered the limiting effect. The appropriate depth for specifying the dose for these effects is therefore 300–500  $\mu\text{m}$  (see NCRP, 1989b).

(B183) With fractionated exposures (in man) the threshold dose for telangiectasia and late dermal atrophy 5 years post irradiation is about 30–40 Gy. These doses may be compared with the Commission's current annual dose limit which corresponds to a lifetime dose of about 20 Gy.

##### Effects of radiation from radioactive ("hot") particles

(B184) The characteristic of "hot particle" exposure is that very high doses can occur over a very small area. The number of cells at risk is so small that the risk of cancer induction is considered minor (NCRP, 1989b). The lesion of concern is ulceration or breakdown with subsequent infection that leads to ulceration. The threshold dose for  $\leq 1 \text{ mm}$  particles is estimated to be 70 Gy measured over an area of 1.1  $\text{mm}^2$  or about 1 Gy when averaged over 1  $\text{cm}^2$  at a depth of 100–150  $\mu\text{m}$  (Hopewell *et al.*, 1986). However, below 250 Gy the ulcers are transient, lasting less than a week. Erythema over a larger area is detectable at these doses. Other estimates (NCRP, 1989b) based on the number of beta particles emitted from the source (which is approximately independent of beta energy) suggest threshold values, at least for more severe or more persistent ulceration, of about  $10^{10}$  particles or Bq sec. This emission level corresponds to a dose of about 5 Gy when averaged over 1  $\text{cm}^2$  at 100–150  $\mu\text{m}$ , i.e., a somewhat higher threshold than those values proposed above (Hopewell *et al.*, 1986).

#### B.10.3. Stochastic effects

(B185) Two types of skin cancer, basal and squamous cell carcinomas have been associated with exposure to ionising radiation. As yet, the evidence of an association between ionising radiation and melanoma, the most malignant type of epidermal cancer, is inconclusive. The ratio of basal cell to squamous cell carcinomas that occur in Caucasian populations exposed only to ultraviolet radiation (UVR) in sunlight is about 5:1 but for those exposed to ionising radiation it is 10:1 or greater. The lethality of skin cancer is very low; for basal cell carcinomas perhaps as low as 0.01% compared to about 1% for squamous cell carcinomas.

(B186) The risk of excess skin cancer induced by ionising radiation is influenced by exposure to UVR and is dependent on the degree of skin pigmentation. The greatest risk is in those with a light complexion, the extreme example being Albinism. There is a fifty-fold range of susceptibility among races. The risk of both naturally occurring and ionising radiation-induced skin cancer is low in black-skinned races. The risk estimates given here apply to Caucasians. Evidently it is necessary to make separate risk estimates for areas of

the skin exposed to sunlight such as head and neck, and for areas such as the trunk that receive much less UVR.

(B187) The incidence of basal cell carcinomas, assumed to be caused by UVR, is high in persons with the genetic condition, Nevroid Basal Cell Syndrome (NBCS). Patients with this condition are not more sensitive to cell killing by ionising radiation but they show a high susceptibility to the induction of cancer by ionising radiation, in areas of skin both exposed and shielded from UVR.

(B188) Until recently induction of cancer by doses less than about 10 Gy was not observed, but recent experiments indicate an excess risk at much lower doses, perhaps below 1 Gy. The dose-response relationship is dependent on whether or not exposure to UVR is involved. The data, although incomplete, suggest that a relative risk model is appropriate, but the choice of this model may result in an overestimate of the risk.

#### B.10.4. Risk estimates

(B189) The incidence of skin cancer is proportional to the area of skin exposed to ionising radiation and also to UVR. The absolute risk estimate for the UVR exposed skin of the body, a total area of about 3,000 cm<sup>2</sup>, is  $6.7 \times 10^{-4}$  per person year gray. For the skin shielded from UVR, representing a total area of about 15,000 cm<sup>2</sup>, this risk is estimated to be  $2.0 \times 10^{-4}$  per person year gray. The total risk is estimated to be  $8.7 \times 10^{-4}$  per person year gray when all of the skin of the body is exposed to ionising radiation (Shore, 1990).

(B190) Risks have been estimated by summing of the risks for UVR exposed and shielded areas, averaging risks for both sexes, and assuming a lethality of induced skin cancers of 0.2%. No reduction in risk is assumed for protracted exposures even though such a reduction is very likely. The average whole-body cancer risks for a working lifetime of age 18 to 64 years are shown in Table B-24. These are selected from the report of the Task Group on Skin (ICRP, Table 10, in preparation) in which risk estimates are given for a variety of circumstances.

Table B-24. Absolute and relative risk of induced skin cancer<sup>1</sup>

	Probability ( $10^{-2} \text{ Sv}^{-1}$ )	
	Incidence	Mortality
Absolute risk model	2.3	0.005
Relative risk model	9.8	0.02

<sup>1</sup> Working lifetime from age 18-64 y.  
Mortality based on a frequency ratio of 5:1 for basal cell carcinomas (0.01% mortality) to squamous cell carcinomas (1% lethality).

(B191) The estimate of fatal cancer risk derived from the relative risk model, viz.  $2 \times 10^{-4} \text{ Sv}^{-1}$ , is preferred and the value is used in this annex, earlier (paragraph B110 and Table B-17). The dose should be evaluated at the depth of the basal cell layer which varies between 20  $\mu\text{m}$  and 100  $\mu\text{m}$  over the whole body.

### B.11. Summary of Estimates of Probabilities of Effects

Effect	Population	Exposure period	Exposure modes	Probability
<b>Low-LET radiation</b>				
Mental effects Reduction in IQ	Fetus	8-15 weeks of gestation	High dose, high dose rate	30 IQ points $\text{Sv}^{-1}$
Severe mental retardation	Fetus	8-15 weeks of gestation	High dose, high dose rate	$40 \times 10^{-2}$ at 1 Sv
Hereditary Severe hereditary effects, including multifactorial diseases	Whole population	All generations	Low dose, low dose rate	$1.0 \times 10^{-2} \text{ Sv}^{-1}$
Cancer Fatal cancers (total)	Workers	Lifetime	Low dose, low dose rate	$4.0 \times 10^{-2} \text{ Sv}^{-1}$
Fatal cancers (total)	General population	Lifetime	Low dose, low dose rate	$5.0 \times 10^{-2} \text{ Sv}^{-1}$
Fatal cancer (in specific organs)	Workers General population	Lifetime	Low dose, low dose rate	See Table B-17
Skin (fatal)		Lifetime	High or low dose, low dose rate	$2 \times 10^{-4} \text{ Sv}^{-1}$
Aggregated health detriment (in specific organs)		Lifetime	Low dose, low dose rate	See Table B-20
Tissue weighting factors				Paragraph B120
<b>High-LET radiation</b>				
Cancer and hereditary risks are the same as for low-LET radiation using $\frac{1}{10}$ to assess equivalent or effective dose.				See main text for $\frac{1}{10}$ values, Table 1
Radon: Fatal lung cancers	Workers	Lifetime		$(1-4) \times 10^{-4} \text{ WLM}^{-1}$ (3-10) per $\text{Jhm}^{-1}$

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**ANNEX C**  
**BASES FOR JUDGING THE SIGNIFICANCE OF THE EFFECTS**  
**OF RADIATION**

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**C.1. Introduction**

(C1) This Annex discusses the risk concept and ways of expressing quantities describing various aspects of a potentially hazardous situation. The main emphasis is on the probability of serious or lethal radiation effects, particularly death from cancer, and ways of indicating the severity of such effects.

(C2) The expression of cancer mortality risk in the Annex is based on primary risk

coefficients given in the 1988 United Nations Scientific Committee on the Effects of Atomic Radiation report (UNSCEAR, 1988), and the application of either the simple additive or the simple multiplicative projection model as described in the text. The risk expression (not only the attributable lifetime probability of death but also the distribution of risk over age after exposure) will depend on demographic data such as background cancer incidence and total mortality rates by age. A detailed discussion on how this will influence the cancer risk for specific organs in populations with different characteristics is presented in Annex B. In Annex C, the calculations have been made for a hypothetical population, using, for convenience of calculation, cancer incidence data from Japan and survival data from Sweden. The results are very close to the nominal risk assessments in Annex B. The summary tables and diagrams, showing various alternative ways of expressing radiation risk, can therefore be used as background material for the multi-attribute approach required for the conclusions in the main text. The reader is reminded of the large uncertainties of the primary risk coefficients on which the calculations are based. Compared with these uncertainties, the influence of demographic assumptions is negligible.

### C.2. The Meaning and Expression of "Risk"

(C3) In previous publications, for the sake of simplicity, the Commission has used "risk" as a synonym for probability of a harmful effect (mainly lethal cancer and severe hereditary harm). However, outside the field of radiation protection, "risk" has several other meanings, including the common, loose meaning in everyday language, i.e. the threat of an undesirable event, including both the probability and the character of the event. With this latter meaning, "risk" is almost synonymous with "hazard". In reactor safety, "risk" usually means the mathematical expectation of the magnitude of the undesirable consequence, i.e. the product of the probability and the consequence of the event. These different meanings of the word have caused considerable confusion in transdisciplinary communications.

(C4) With special definitions of "risk", as in reactor safety and in the Commission's previous recommendations, risk becomes a quantity which can be characterised by a magnitude expressed in a unit which is dimensionless if risk means probability, but has some dimension if risk means the mathematical expectation of consequence. However, it has become increasingly recognised that such limited presentation of risk is insufficient to describe a "risk situation". Therefore, in many areas of hazard assessment, specific meanings of the word "risk" are avoided and preference is given to words which more directly indicate the relevant quantity, e.g. "probability", "consequence", and "mathematical expectation" (which is a synonym for the average) of the consequence. This leaves the word "risk" free to be used in the everyday meaning and makes it possible to include in the risk concept a number of factors which, in addition to those more readily quantifiable, influence decisions on risk acceptance. Such factors are, for example, if the risk is imposed or voluntary, if the consequence is new or familiar, but also the severity of the consequence and when it will occur in time.

(C5) With this wider meaning of the word, "risk" is a concept rather than a quantity, although it may also be seen as a multi-attribute quantity. This has led Fischhoff *et al.* (1984) to suggest what they call a "vector" presentation to describe the total risk situation. In such a multi-attribute presentation, different elements represent each quantifiable attribute, such as probabilities and magnitudes of specified consequences

but also some rating of not so easily quantifiable attributes. Preferences in risk comparisons would then have to be made on the basis of a multi-attribute analysis.

(C6) Being aware of this development, the Commission has decided to abandon its practice of always strictly using "risk" with the specific meaning of probability and to attempt to use, where practicable, the more direct term "probability". This should reduce the ambiguity when describing probabilities and consequences and make it easier to communicate the recommendations to regulatory agencies and others who also deal with non-radiation risks. "Risk assessment", in this report, is therefore not necessarily synonymous with "probability assessment" but may include assessments of other aspects of risk, e.g. the nature and severity of the harmful consequences. "Risk of death" may refer both to the probability of death attributable to a defined radiation exposure, but also to the attributable death age probability density (see paragraph C26) or derived quantities such as reduction of life expectancy. In many cases, however, when misunderstanding is unlikely or when the exact meaning of the word is not important, the convenient word "risk" is still used, e.g. in expressions such as "risk acceptance" or "radiation risks".

(C7) The symbols and quantities used in this Annex sometimes deviate somewhat from those in Annex B and from those used in demographic and epidemiological texts. For example, the concept of "death probability rate" is used rather than "mortality rate". The reason in that particular case is that the rates will be integrated and the integral to be used by the Commission is the attributable lifetime probability of death, related to the average individual, rather than the observed or expected number of deaths per 100,000. Since the Commission uses probability as one aspect of risk, it has chosen to use a probabilistic presentation in this Annex where various ways of describing risk are discussed. However, in order to help the reader, references to the corresponding presentation in Annex B are given where appropriate.

(C8) For the purpose of this report, the Commission is mainly concerned with two quantifiable risk quantities, namely:

$P_i$  = the probability of each harmful effect (i). The effect will have to be specified, e.g. lethal cancer or curable cancer, severe hereditary harm, etc.;

$W_i$  = the consequence if the effect occurs. The consequence can be described in a variety of ways, indicating the severity of the effect and its distribution in time.

(C9) The mathematical expectation of consequence, identical to the average consequence is

$$\bar{W} = \sum P_i \cdot W_i$$

when averaging is relevant, a quantity which is sometimes used in the effort to express the magnitude of the "risk" by one single measure. In the collective case, i.e. the number of affected persons in a large population,  $N$ , the mathematical expectation is not far from a likely result unless the individual probability ( $p$ ) of harm is very small. If the possible consequence for each individual is  $w = 1$  case of harm, the expectation will be

$$\bar{W} = N \cdot \bar{w} = N \cdot (p \cdot w) = N \cdot p.$$

Weighted for the severity of the harm, it has been used by the Commission under the name radiation health detriment (ICRP, 1977a). In the individual case, however, the mathematical expectation ( $\bar{w} = p \cdot w$ ) is not an "expected" result, because the only

possible outcomes are 0 or  $w$  measures of harm. The use of the expectation in this case masks the fact that it is composed of the two components  $p$  and  $w$ . For example,  $p = 10^{-4}$  may be the probability of losing, on average, 20 years of life because of cancer. The expectation of loss of life is then  $2 \cdot 10^{-3}$  years, i.e. about 10 minutes. However, the real loss of life is either 0 (almost certain) or about 20 years (with a very small probability) and never 10 minutes.

(C10) The probability of death (as defined later) may be assumed to be the major factor in the multi-attribute concept of risk. This particular component of risk, therefore, will be discussed more thoroughly here. Other attributes should also be considered, such as illness, hereditary disease, risks to any fetus, economic losses, anxiety and other societal impacts. However, too little is known about these to make a full treatment possible. Therefore, the Commission has had to take account of these attributes by means of simplified approaches in order to modify any conclusions that may be drawn from the assessment of the risk of death from cancer. One of the additional components of radiation risk, the hereditary risk, is discussed in Annex B and in the main text.

### C.3. Conventions on Acceptable Risks

(C11) A risk-free society is Utopian. All human activities (or lack of activity) carry some risks albeit that many risks can be kept very low. Some activities are accepted by most even though risks have not been reduced "as far as reasonably achievable". The corresponding risks, however, e.g. traffic risks, are not necessarily acceptable, and there is a growing opinion that unnecessary risks should be reduced wherever reasonably achievable. Other activities are not accepted, because the risks are considered unjustifiably high in relation to the ensuing benefits even after reasonable efforts of risk reduction.

(C12) There seems to be an unspoken convention that we are willing to accept certain levels of risk in order to enjoy the benefits of a modern society, provided that the risks are not unnecessary or easily avoided (see, for example, Fried, 1970). The obvious question is: what levels?

(C13) Many attempts have been made to set an upper limit of risk to an individual (with "risk" often not well defined), i.e. a level of risk which would not be acceptable even if it could not reasonably be further reduced. This limit will undoubtedly depend on the general life situation and the urgency or desire (as in voluntary risk taking) of taking the risk; for the Commission's purposes the relevant circumstances would be the daily normal occupational or private life in what is usually considered to be a safe society.

(C14) A report of a Study Group of the British Royal Society (1983) concluded that imposing a continuing annual occupational probability of death of 1 in 100 would be unacceptable, while they found the situation less clear with regard to an annual probability of death of 1 in 1000. They felt that the latter probability level could "hardly be called totally unacceptable provided the individual at risk knew of the situation, judged he had some commensurable benefit as a result, and understood that everything reasonable had already been done to reduce the risk". However, the annual probability of death is only one of the attributes which are appropriate to take into account. In the following, a number of other aspects will be considered.

(C15) Travis *et al.* (1987a, 1987b) retrospectively reviewed how cancer risk estimates for the public had been used by U.S. federal agencies in the regulation of 132 different chemical carcinogens. Among the risk measures they examined was the individual

attributable lifetime probability of death. Their conclusion was that all substances with an attributable lifetime cancer death probability above approximately  $4 \times 10^{-3}$  appeared to have been regulated regardless of cost. At lower individual probabilities, substances with regulatory costs above 2 million US\$ per life saved, with one exception, were not regulated.

(C16) The dose limits recommended in the Commission's *Publication 26* (ICRP, 1977a) were put forward with the implied assumption that an annual occupational death probability of about  $10^{-3}$  to the most exposed individuals would be at the border of being unacceptable. The corresponding extra imposed annual death probability for members of the public at the annual limit of 1 mSv would be about  $10^{-5}$ .

(C17) A problem for the Commission is therefore that, even if in the unlikely case that it were possible to reach an agreement on the level of total controllable risk that might be considered unacceptable, this would not necessarily give the answer to the question of which level of radiation risk should be said to be unacceptable. There are many sources of risk in life and the question of their addition has to be kept in mind, even though no individual would be expected ever to be exposed at each one of the various limits that authorities may impose.

### C.4. The Risk of Death

(C18) The attributable lifetime probability of death from radiation exposure has been used by the Commission in earlier reviews of the justification of various levels of dose limitation, and radiation risks have been expressed as so and so many "per cent per sievert". However, our total probability of death, which is 100%, cannot be increased. The introduction of a new risk source will not change our lifetime probability of death but only the distribution of the probable causes of death. Any increment that a new risk source causes, is an increment to our death probability rate at any given age, provided that the person is alive at that age (i.e. a conditional probability rate).

(C19) The total conditional death probability rate from all causes, for an average person (i.e. given that the individual is alive at every age  $u$ ), can usually be described by the Gompertz-Makeham expression (Gompertz, 1825; Makeham, 1870):

$$G_n(u) = A e^{B \cdot u} + C$$

where  $u$  is age and  $A$ ,  $B$  and  $C$  are parameters which can be derived from demographic tables (it should be remembered that there is a distinction between the probability in a year—which can never exceed 100%—and the probability per year, i.e. the probability rate, which will exceed 100% at very high ages).

(C20) A defined exposure scenario (e.g. a constant dose rate from age 18 to age 65) may add a conditional source-related increment of probability rate,  $dp/du$ , to the background rate:

$$G(u) = G_n(u) + dp/du$$

This corresponds to the expression which, in Annex B (see paragraph B78), is denoted

$$q_{n,h}(a) = q_n(a) + h_{n,h}(a)$$

From this increment, an unconditional probability rate,  $d/du$ , may be calculated once a reference time (age) has been defined, e.g. the age at the onset of the exposure period

(see paragraph 24). On this assumption, we may define a number of quantities which can be used to express "risk".

#### C.4.1. The conditional death probability rate ( $dp/du$ )

(C21) The first quantity of interest is the radiation-induced death probability rate,  $dp/du$ , which a given source or practice is assumed to cause over the rest of the life of the exposed individual. Assuming that the dose rate is known as a function of age, this can be calculated on the basis of postulated dose-response relations (including assumptions on minimum latent periods, plateau lengths, etc.)

(C22) One of several radiation protection requirements is that this conditional probability rate should be kept acceptably low. The rate is conditional, because it will only be expressed if the individual is alive at the ages ( $u$ ) for which it is defined. One pertinent question is whether it is its absolute value  $dp/du$  which should be kept low, or its relative value  $(dp/du)/G_n(u)$ . It seems reasonable to assume that all requirements on this particular quantity are fulfilled if  $(dp/du)/G_n(u) \ll 1$  for all ages of concern. It can be shown that, for exposure patterns of practical significance, the maximum value of  $(dp/du)/G_n(u)$  occurs at ages below 60 years, irrespective of whether the additive or the multiplicative projection model is used in the assessment (see Tables C-4 a and b and Figure C-7).

#### C.4.2. The unconditional death probability rate ( $dr/du$ )

(C23) The conditional incremental death probability rate,  $dp/du$ , due to a given risk source, cannot be used for calculation of the total attributable lifetime probability of death, because the expression is "open-ended" with no well defined integration limit. The attributable lifetime probability of death from the source under consideration must therefore be calculated from the unconditional incremental death probability rate,  $dr/du$ , taking account of the probability of reaching each age ( $u$ ) by considering the likelihood of dying from other causes as well as from radiation. The unconditional incremental probability rate is obtained as the product of the conditional incremental probability rate  $dp/du$  and the survival probability modified by the incremental risk  $S(T,u)$ :

$$dr/du = S(T,u) \cdot (dp/du)$$

This corresponds to the quantity which, in Annex B (see paragraph B78), is denoted  $I_{D,A}(a) \cdot h_{D,A}(a)$ , although in that expression  $A_n$  is the time of exposure in the case of a single exposure, while  $T$  has a more general meaning (see below).

(C24) The modified survival probability  $S(T,u)$  is related to the age ( $T$ ) from which the probability is calculated. Hence,  $T$  must be defined. The choice of  $T$  is obvious in the case of one single exposure, in which case  $T$  should be the age at the time of the exposure ( $T = A_n$ ). However, with a prolonged exposure pattern (e.g. age 18 to age 65), the choice is less obvious. In this document,  $T$  has been chosen to be the age at the onset of the exposure period. The unconditional ("expressed" or "a priori") incremental death probability rate can then be calculated as above.

#### C.4.3. The attributable lifetime probability of death ( $R$ )

(C25) The attributable lifetime probability of death ( $R$ ) can be calculated as the integral of the unconditional incremental death probability rate:

$$R = \int_0^{\infty} (dr/du) du$$

#### C.4.4. The probability density of the age of death

(C26) The magnitude of the attributable lifetime probability of death alone gives no indication of when death will occur, being merely the probability of dying from cancer due to one particular cause rather than dying from any other cause. Somewhat fuller information is offered by presentation of the variation of  $dr/du$  with age. This is the **probability density of the age of death**, normalised so that the area under the curve is not unity but the attributable lifetime probability of death (see Figures C-3, C-4, C-9 and C-10).

#### C.4.5. Mean loss of lifetime if radiation death occurs ( $Y$ )

(C27) Given the unconditional incremental death probability rate  $dr/du$  over all ages, and the normal remaining life expectancy as a function of age, it is possible to calculate the **mean loss of lifetime**, ( $Y$ ), in the case of death from radiation. The pair of values: the attributable lifetime probability of death ( $R$ ) and the mean loss of lifetime ( $Y$ ) if radiation causes death, is the minimum of information needed to express the incremental "risk".

#### C.4.6. The reduction of life expectancy ( $\Delta L$ )

(C28) It is also possible to calculate the **reduction of life expectancy**, i.e. the mathematical expectation ( $\Delta L$ ) of the loss of lifetime due to a particular exposure pattern. This is simply

$$\Delta L = R \cdot Y$$

i.e. the product of the attributable lifetime probability of death and the mean loss of lifetime if the radiation causes death. As long as  $R$  is  $\ll 1$ , this expectation value is not very informative in the individual case, and may even be misleading because it may wrongly be interpreted as a loss of life that will actually occur. In reality, if  $R$  is small, the most likely loss of lifetime is zero, and there is the small probability,  $R$ , of losing the life period  $Y$ . The expectation value  $\Delta L$  will then never occur.

(C29) In the collective case, however, the situation is different. In a cohort of  $N > 1/R$  individuals, a lifetime expectation of  $N \cdot \Delta L$  (in e.g. man years) is a very likely outcome.

#### C.4.7. Probabilistic "aging"

(C30) There is one alternative to presenting the expectation of the loss of lifetime in the individual case. The shift in the age-specific death probability rate may be described as equivalent to an aging in the sense that the increased probability rate equals that at a higher age. This "aging" with regard to death probability rate is of the same order of magnitude as the expectation of loss of lifetime, but varies with age also because the excess probability rate  $dp/du$  varies with age.

### C.5. The Background Conditional Death Probability Rate ( $G_n(u)$ )

(C31) The lowest conditional death probability rate from all causes usually occurs around the age of 10 years, when the annual probability of death is about 1-2 in 10,000 in most industrialised countries, although it may exceed 1 in 1000 in developing countries.

(C32) In many countries a peak around the age of 20 years apparently reveals juvenile risktaking, particularly in the case of males. At ages above 30-40 years, the death

probability rate doubles about every seven years, i.e. increases by about 10 per cent per year and is of the order of 1 in 100 per year at the age of 60 years, see Figure C-1. In industrialised countries, the age-specific mortality rate has decreased substantially over the last century, as the result of a number of improvements such as improved hygiene, cleaner water, better living conditions, and advances in medicine and public health (Statistics Sweden, 1969, 1988).

(C33) It may be assumed that any particular new justifiable risk source with optimised protection causes a risk which might be seen as acceptable as long as the age-specific mortality rate for those most heavily exposed will not significantly increase at any age, and that any existing risk might be seen as acceptable if it does not contribute signifi-

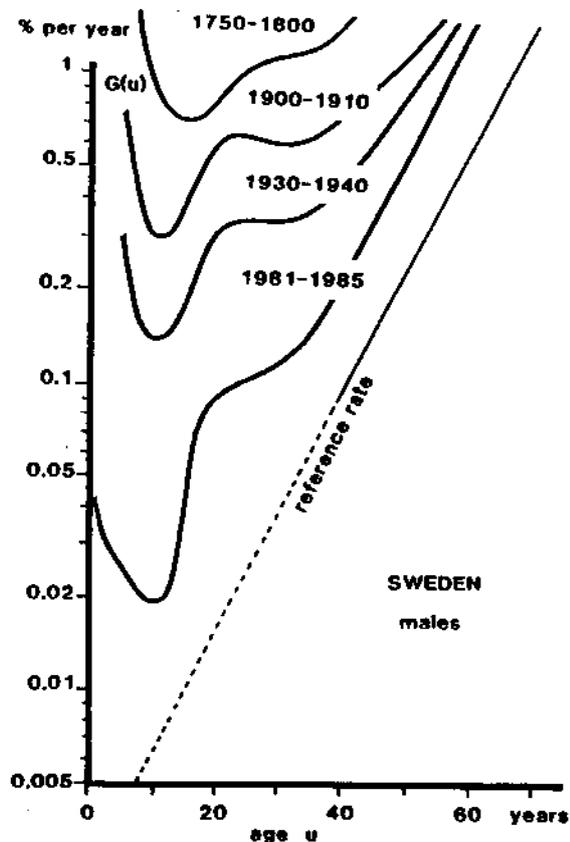


Fig. C-1. Gompertz-Mitcheam curves (the age-specific mortality rate) for Swedish males from 1750-1800 to 1981-1985. The "reference rate" is the lowest age-specific mortality rate currently found in any country for the various ages.

cantly to age-specific mortality rates which are higher than would be expected in comparison with countries or regions which are usually considered "safe". However, the achievement of average safety reflected in health statistics gives little help in judging the appropriate individual risk limitation. It should also be recognised that the justification of a source does not necessarily follow from acceptably low individual risks.

(C34) If a nation wants to control the total impact of a number of new or developing risk sources, the necessary degree of limitation for each single source or practice, including radiation practices, is a problem beyond the realms of radiation protection alone. The choice of each risk limit (e.g. limits for attributable lifetime death probability), including that for the purposes of radiation protection, always involves subjective considerations in addition to the scientific conclusions. Other requirements, such as comparisons with normal variations in exposures from noncontrollable natural sources of radiation and the necessity of maintaining an appropriate margin of safety to dose levels causing deterministic effects, may well determine the appropriate dose limit (although not the justification of the source) within a more narrow range than would the uncertain derivation from a somewhat arbitrarily chosen risk limit.

(C35) Even if an agreement on a reference risk and the derivation of the corresponding dose limit would be possible, the Commission feels that the validity of the exercise would be more apparent than real. The Commission, therefore, now prefers a multi-attribute approach to the choice of dose limits. For this purpose it is necessary to examine the overall risk picture that would be the consequence of various options of dose limits.

#### C.6. Primary "Risk Coefficients"

(C36) A radiation dose, when delivered, will involve a risk commitment, i.e. a commitment of an increased cancer death probability rate in the future, after a minimum latent period which may be from a few years in the case of leukaemia to tens of years for other malignant conditions. Any change in the age-specific death probability rate would therefore occur first later in life, when the risk of death from other causes is also higher. The risk committed by a radiation dose at a given age can therefore not be added to the background risk at the same age. This is different from accidental death which will usually occur at the same age as the primary event.

(C37) In the case of internal exposure, the actual dose (the committed effective dose) may sometimes be partly delivered long after the intake of the radioactive substance. This will even further delay the actual expression of harm.

(C38) An increased cancer death probability rate ( $dp/du$ ) will not occur until after a minimum latent period of time from a radiation exposure. Two models have been used to describe the subsequent excess probability rate as a function of time. In the simple "additive" or "absolute" model, the excess probability rate is dose-dependent but independent of age. In the simplest version of the "multiplicative" or "relative" model, the excess rate increases with age at the same rate as the background cancer rate. The multiplicative model is now considered to fit epidemiological observations best (see Annex B). However, the additive model is also used in this Annex, for purposes of comparison, because it was the model that was used in 1977. The difference between the two models is illustrated in Figure C-2.

(C39) The calculations for this Annex have been made on the basis of primary "risk coefficients" presented by UNSCEAR (1988) and, in Annex B, denoted  $K_{DA}$  and  $C_{DA}$ .

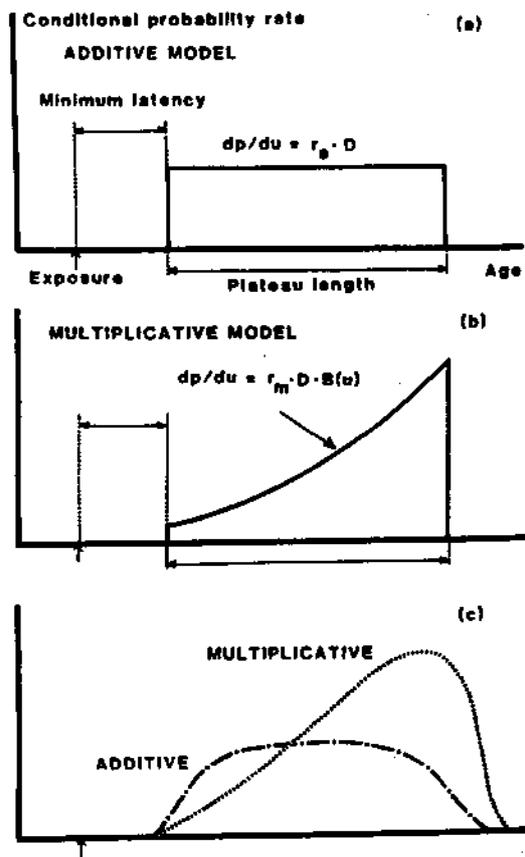


Fig. C-2. Illustration of the two simple projection models. Figures (a) and (b) show the stylised models which have been used for the calculations in this Annex; Figure (c) indicates possible curve shapes under more realistic assumptions.

(a) The simple *additive* model: The excess conditional probability rate (of death from cancer) after a single radiation dose,  $D$ , is assumed to be proportional to the dose, but first after a minimum latency period and over a "plateau" period of time. (b) The simple *multiplicative* model: The excess probability rate is also assumed to be proportional to the background rate of cancer death,  $B(u)$ .

These coefficients are summarised in Table C-1. During the preparation of the Annex, further risk estimates have become available, e.g. the "BEIR-V" report (U.S. National Academy of Sciences, 1989) and the assessments made by Committee I for Annex B. Annex B shows that the differences caused by different demographic assumptions are small. The calculations in this Annex give attributable life time cancer death probabilities which are virtually equal to the probability of fatal cancer after low dose, low dose rate low LET radiation to the total population ( $5 \times 10^{-2} \text{ Sv}^{-1}$ ) assessed in Annex B. For these

reasons, the various attributes of risk shown in this Annex may be taken as representative for the total cancer risk. They serve the double purpose of illustrating the various ways in which "the risk of death" may be expressed and providing background data for a multi-attribute approach to the selection of dose limits.

### C.7. Increment of Death Probability Rate after a Single Dose

(C40) On the basis of the primary risk coefficients used by UNSCEAR, the age-dependent increment of the conditional death probability rate ( $dp/du$ ) after a single dose at various ages can be assessed. Then also the unconditional rate ( $dr/du$ ) can be derived, and its integral, the attributable lifetime probability of death, can be calculated.

(C41) The results are uncertain to the same degree as the primary risk coefficients. The main merit of the exercise is to illustrate the character of the different consequences of the two projection models. The results for the two most relevant exposure situations are summarised in Figure C-9 (see Section C.8).

#### C.7.1. Assessment based on the additive model

(C42) The assessment based on the additive projection model is straightforward. The minimum latent period assumed by UNSCEAR for leukaemia is 2 years and for other cancers 10 years. A plateau length of 40 years was assumed by UNSCEAR for leukaemia and of infinite length for other cancers ("non-leukaemia"). The general shape of the variation of the conditional death probability rate ( $dp/du$ ) with age for males after a single small dose is then as shown in the examples in Figures C-3 (a) and C-4 (a) for exposure at age 5 and 35 years, assuming a DDREF (dose and dose rate effectiveness factor, see Annex B) of 2. The discontinuities are caused by the crude assumption of no risk within the minimum latent period and full risk over the assumed plateau lengths. This is obviously not realistic but will suffice for the purposes of this Annex.

(C43) Figures C-3 (a) and C-4 (a) also show the variation of the unconditional death probability rate ( $dr/du$ ) with age for the example of exposure at ages 5 and 35 years. The area under the curves for  $dr/du$  represents the attributable lifetime probability of death. The curves are derived on the assumption of modified survival probabilities,  $S(5,u)$  and  $S(35,u)$ , based on the normal survival probabilities for the Swedish population in 1986 (Statistics Sweden, 1988), the modification being the correction for the additional radiation risk.

(C44) Figure C-5 (a) shows the variation of the attributable lifetime probability of death with the age at the time of exposure. Here, the discontinuities are the result of the UNSCEAR risk coefficients given as averages for wide ranges of exposure ages.

(C45) From the data represented by Figure C-5 (a), it is possible to calculate a mean value of the attributable lifetime probability of death per unit (single) dose over all ages in a population with a normal age distribution. If the age distribution is taken to have the same shape as the survival probability to age  $u$  at birth,  $S(0,u)$ , which would be the case in a population in growth equilibrium, the mean values would be 1.8%/Sv for males and 2.1%/Sv for females, with the assumed DDREF of 2. On these assumptions, the mean attributable lifetime risk per unit single dose (effective dose) is therefore about 2%/Sv as an average for both sexes.

#### C.7.2. Assessment based on the multiplicative model

(C46) For an assessment on the basis of the multiplicative projection model, assump-

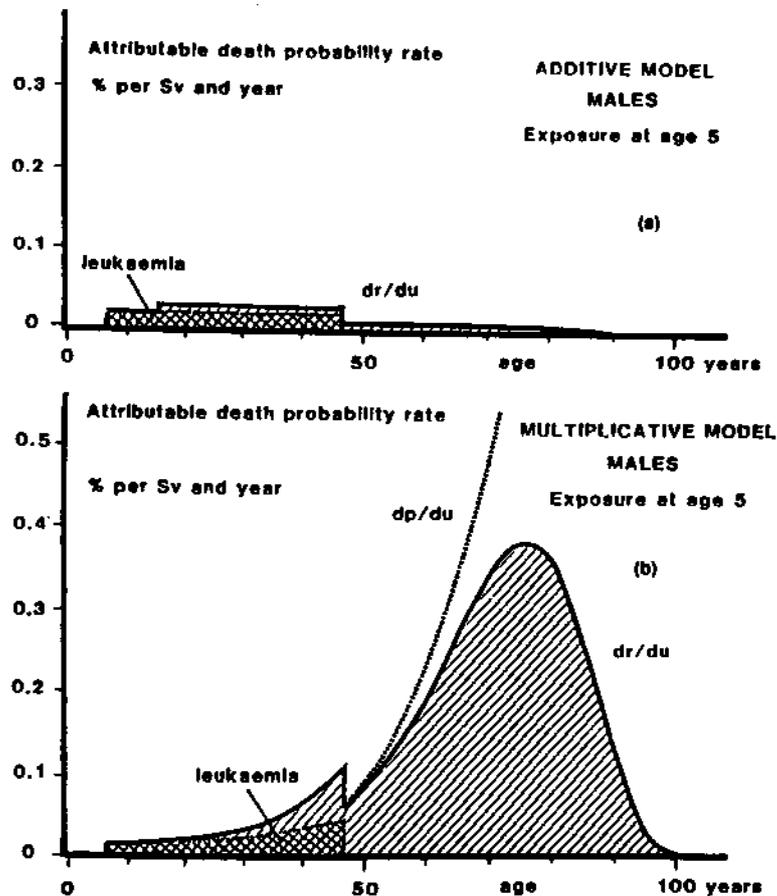


Fig. C-3. Variation with age of the attributable death probability rates  $dp/du$  (conditional) and  $dr/du$  (unconditional) after a single small dose at age 5 years, assuming a DDREF of 2. The discontinuities reflect the simplified assumptions on minimum latency periods and plateau shapes (cf. Figure C-2).

tions must be made on the background age-specific death rates for leukaemia and non-leukaemia. In the assessment for the purposes of this Annex, as for the UNSCEAR summary tables, the background rate has been taken to be that for each sex of the Japanese population in 1986 as presented in the WHO World Health Statistics (World Health Organization, 1986). These data can reasonably well be approximated by a simple power function:

$$B(u) = a \cdot u^b + c$$

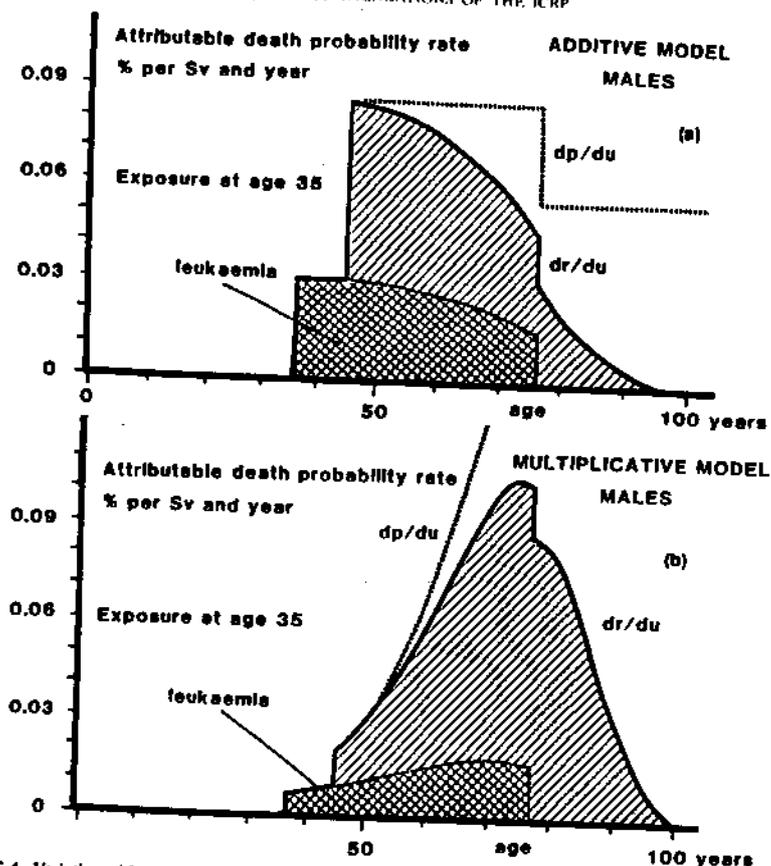


Fig. C-4. Variation with age of the attributable death probability rates after a small single dose at age 35 years, assuming a DDREF of 2. The discontinuities reflect the simplified assumptions on minimum latency periods and plateau shapes (cf. Figures C-2 and C-3).

(C47) The following values for  $a$ ,  $b$  and  $c$  were found to give a sufficiently good approximation (within about  $\pm 15\%$  for ages above 45 years which contribute most to the total risk). All numbers are expressed to give  $B(u)$  per year if the age,  $u$ , is given in years:

Cancer type	Sex	$a$	$b$	$c$
Leukaemia	Males	$4.4 \times 10^{-10}$	3.00	$15 \times 10^{-6}$
	Females	$3.0 \times 10^{-10}$	2.90	$15 \times 10^{-6}$
Non-leukaemia	Males	$3.0 \times 10^{-10}$	5.14	$15 \times 10^{-6}$
	Females	$3.9 \times 10^{-10}$	4.90	$15 \times 10^{-6}$

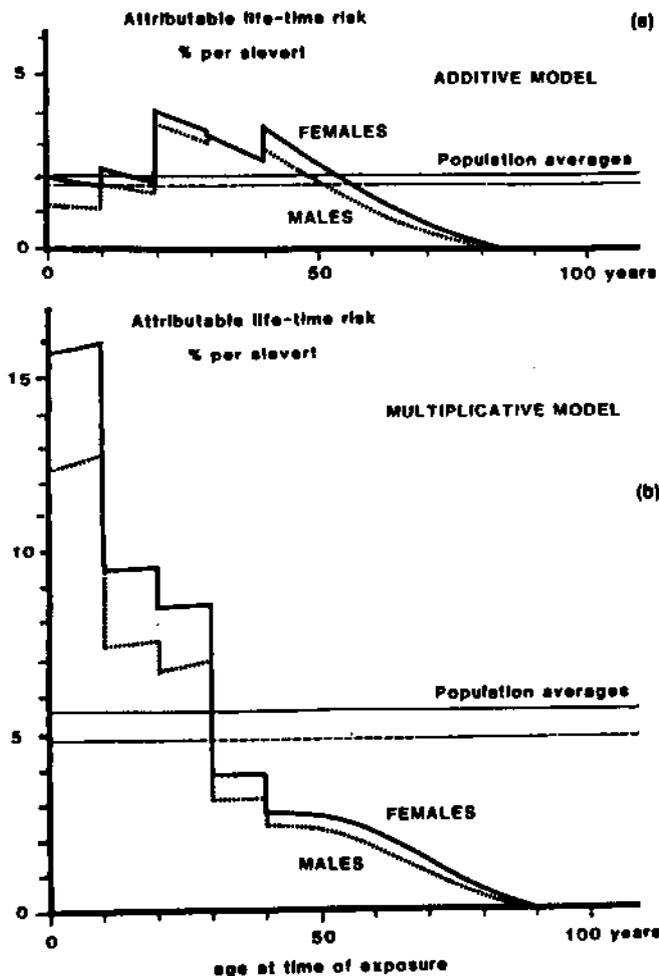


Fig. C-5. The attributable lifetime risk from a single small dose at various ages at the time of exposure, assuming a DDREF of 2. The discontinuities are the result of the use of constant annual values for the primary risk coefficients within 10-year age intervals (cf. Table C-1). The higher risk for the youngest age group will not be expressed until late in life.

(C48) Figures C-3 (b) and C-4 (b) show the conditional and unconditional death probability rates as a function of age, again in the case of single exposures at the age of 5 and 35 years. It can be seen that the attributable lifetime probability of death (represented by the area under the curves for  $dr/du$ ), for exposure of children, is one order of

magnitude higher than in the case of the additive model, but death will occur at higher ages. For exposures at adult ages, the differences are minor.

(C49) Figure C-5 (b) shows the variation of the attributable life-time probability of death with the age at the time of exposure. The substantially higher risk for the youngest age group is notable, but it must be remembered that neither model may be true for this age group, since they do not fit the actual data. Since the UNSCEAR primary risk coefficient is given as an average for the age group 0-9 years, there is no indication on whether the risk is even higher for the youngest ages in the group. This is of particular importance in the case of single exposure of infants, e.g. in accidents. Here, more epidemiological evidence (which could only be expected at high or medium doses) is urgently needed. However, it must be recognised that most of this higher risk will be expressed first at high ages. If the multiplicative projection model is truly valid, therefore, any epidemiological study of exposed infants must involve a follow-up into high ages.

(C50) The mean attributable lifetime probability of death, averaged over all ages and calculated on the same assumptions as for the additive model, i.e. for a small single dose, assuming a DDREF of 2, would be  $4.8 \times 10^{-2} \text{ Sv}^{-1}$  for males and  $5.6 \times 10^{-2} \text{ Sv}^{-1}$  for females. The average for both sexes would be  $5.2 \times 10^{-2} \text{ Sv}^{-1}$ . This value is sufficiently consistent with the estimate of Annex B ( $5.0 \times 10^{-2} \text{ Sv}^{-1}$ ) to make the results in this Annex useful for their intended purposes, in spite of the simplified assumptions.

#### C.8. Increment of Death Probability Rate at Prolonged Exposures

(C51) With the risk coefficients shown in Table C-1, the conditional attributable annual death probability in two exposure situations has been calculated. In the first situation it is assumed that a constant annual dose is received from age 0 and for every year in the future. In the second situation, it is assumed that a constant annual dose is

Table C-1. Primary risk coefficients for annual cancer death (UNSCEAR, 1988). These risk coefficients have been derived on the basis of observations on the cancer death rate among the survivors from the atomic bombing of Hiroshima and Nagasaki. They relate to high doses and high dose rates and are strictly applicable to the Japanese survivors only. "ERR" = excess relative risk. The symbols are those used in Annex B

Age at exposure (years)	Males		Females	
	Additive $10^{-2} \text{ Sv}^{-1}$ and year $K_{D,A}$	Multipl. ERR/Sv $C_{D,A}$	Additive $10^{-2} \text{ Sv}^{-1}$ and year $K_{D,A}$	Multipl. ERR/Sv $C_{D,A}$
<b>(a) Leukaemia</b>				
0-9	0.0384	18.7	0.0300	19.5
10-19	0.0203	4.4	0.0104	4.6
20-29	0.0434	5.6	0.0249	5.8
30-39	0.0631	3.9	0.0196	4.1
40+	0.0472	3.3	0.0318	3.4
<b>(b) All cancer but leukaemia</b>				
0-9	0.0148	1.06	0.0407	2.06
10-19	0.0526	0.65	0.0707	1.27
20-29	0.126	0.57	0.137	1.11
30-39	0.114	0.24	0.137	0.48
40+	0.164	0.18	0.186	0.34

received from age 18 to, and including, age 64. These situations represent the most extreme non-accidental exposures of a member of the public and at work, respectively. For these calculations, again, a DDREF of 2 has been assumed.

(C52) The result of the calculations is shown in Table C-2. For comparison, a reference age-specific mortality rate (from all causes), is shown (for Sweden, which is a typical low-risk country).

Table C-2a. The conditional death probability rate ( $dp/du$ ), total and attributable to various annual doses from birth over lifetime. Assumed DDREF = 2. The values are given as annual numbers per million of each sex. The total death probability rate  $G_0(u)$  is for the Swedish population; these values have been adjusted to reduce random and occasional variations

Age	$G_0(u)$ (per million)	Annual doses (mSv)								
		Additive model				Multiplicative model				
		1	2	3	5	1	2	3	5	5
Males										
0	7200	0	0	0	0	0	0	0	0	0
5	230	1	2	2	4	1	1	2	3	3
10	180	2	4	5	9	1	3	4	7	7
15	400	3	5	8	14	2	3	5	9	9
20	860	4	8	11	19	2	3	7	11	11
25	970	6	12	18	31	3	6	9	16	16
30	1080	9	18	27	44	5	9	14	24	24
35	1360	13	27	40	67	8	15	23	38	38
40	2000	18	36	55	91	12	25	38	63	63
45	2950	21	43	64	108	18	36	55	91	91
50	5300	25	50	75	124	27	53	80	133	133
55	8500	30	59	89	148	42	83	125	208	208
60	13,500	34	69	103	172	64	129	193	322	322
65	22,000	39	77	116	193	97	194	291	485	485
70	35,000	43	86	128	214	143	286	429	714	714
75	57,000	47	93	140	233	207	414	620	1034	1034
80	90,000	50	101	151	252	294	587	880	1470	1470
85	145,000	54	109	163	272	409	819	1230	2050	2050
90	220,000	58	117	175	292	561	1120	1680	2810	2810
95	340,000	62	125	187	313	758	1520	2270	3790	3790
100	520,000	67	133	200	333	1010	2020	3030	5040	5040
Females										
0	6370	0	0	0	0	0	0	0	0	0
5	140	1	1	2	3	1	1	2	3	3
10	115	2	3	5	8	1	3	4	7	7
15	300	3	6	9	15	2	3	5	9	9
20	350	4	9	13	22	2	4	7	11	11
25	400	7	13	20	33	3	6	9	15	15
30	525	9	19	28	47	4	9	13	22	22
35	760	13	27	40	67	7	14	21	35	35
40	1100	17	35	52	86	11	22	34	57	57
45	1650	21	42	62	104	17	33	50	83	83
50	2750	25	49	74	123	25	49	74	124	124
55	4600	30	59	89	148	38	77	115	191	191
60	6900	35	70	104	174	59	117	176	293	293
65	11,000	40	79	119	198	87	174	262	436	436
70	19,000	45	89	134	223	127	254	381	636	636
75	34,000	49	99	148	247	182	363	545	908	908
80	57,000	54	109	164	272	255	509	764	1270	1270
85	100,000	59	118	177	296	350	701	1050	1750	1750
90	165,000	64	128	191	319	475	949	1420	2370	2370
95	280,000	68	137	205	342	633	1270	1900	3170	3170
100	480,000	73	146	219	365	833	1670	2500	4170	4170

Table C-2b. The conditional death probability rate ( $dp/du$ ), total and attributable to various annual doses from age 18 to age 65. Assumed DDREF = 2. The values are given as annual numbers per million of each sex. The total death probability rate  $G_0(u)$  is for the Swedish population; these values have been adjusted to reduce random and occasional variations

Age	$G_0(u)$ (per million)	Annual doses (mSv)								
		Additive model				Multiplicative model				
		10	20	30	50	10	30	50	100	500
Males										
0	7200	0	0	0	0	0	0	0	0	0
5	230	0	0	0	0	0	0	0	0	0
10	180	0	0	0	0	0	0	0	0	0
15	400	0	0	0	0	0	0	0	0	0
20	860	1	2	3	5	0	1	1	2	2
25	970	11	21	32	54	3	7	10	17	17
30	1080	33	66	99	166	9	18	26	44	44
35	1360	79	159	238	397	19	38	58	96	96
40	2000	126	252	378	630	39	78	117	195	195
45	2950	167	334	502	836	70	140	209	349	349
50	5300	210	420	630	1050	121	241	362	603	603
55	8500	263	526	788	1310	199	397	596	993	993
60	13,500	315	629	944	1570	315	631	946	1580	1580
65	22,000	358	715	1070	1790	475	951	1430	2380	2380
70	35,000	390	780	1170	1950	696	1390	2090	3480	3480
75	57,000	408	816	1220	2040	999	2000	3000	5000	5000
80	90,000	392	785	1180	1960	1350	2710	4060	6770	6770
85	145,000	380	760	1140	1900	1810	3620	5430	9050	9050
90	220,000	368	736	1100	1840	2390	4780	7170	11,900	11,900
95	340,000	356	712	1070	1780	3110	6230	9340	15,600	15,600
100	520,000	344	689	1030	1720	4010	8030	12,000	20,100	20,100
Females										
0	6370	0	0	0	0	0	0	0	0	0
5	140	0	0	0	0	0	0	0	0	0
10	115	0	0	0	0	0	0	0	0	0
15	300	0	0	0	0	0	0	0	0	0
20	350	0	1	2	3	0	1	1	2	2
25	400	6	12	18	30	3	6	9	15	15
30	525	26	52	78	131	8	16	24	39	39
35	760	66	131	197	328	17	35	52	87	87
40	1100	105	209	314	524	35	71	106	176	176
45	1650	146	293	439	732	62	125	187	312	312
50	2750	191	382	573	955	107	214	320	534	534
55	4600	245	491	736	1230	174	348	522	869	869
60	6900	299	599	898	1500	274	548	821	1370	1370
65	11,000	348	697	1040	1740	412	828	1240	2060	2060
70	19,000	390	780	1170	1950	604	1210	1810	3020	3020
75	34,000	422	844	1270	2110	865	1730	2590	4320	4320
80	57,000	417	835	1250	2090	1160	2330	3490	5820	5820
85	100,000	410	820	1230	2050	1540	3090	4630	7720	7720
90	165,000	402	804	1210	2010	2020	4040	6070	10,100	10,100
95	280,000	394	788	1180	1970	2610	5230	7840	13,100	13,100
100	480,000	386	772	1160	1930	3340	6670	10,000	16,700	16,700

(C53) It can be seen from Table C-2 that the higher absolute values of the incremental annual probabilities with the multiplicative model appear first at ages higher than 50 years in the case of exposure from birth and 60 years at exposure from age 18 years.

(C54) Since the total background risk increases with age somewhat more rapidly than the cancer background, the relative increments of the death probability rate in relation to

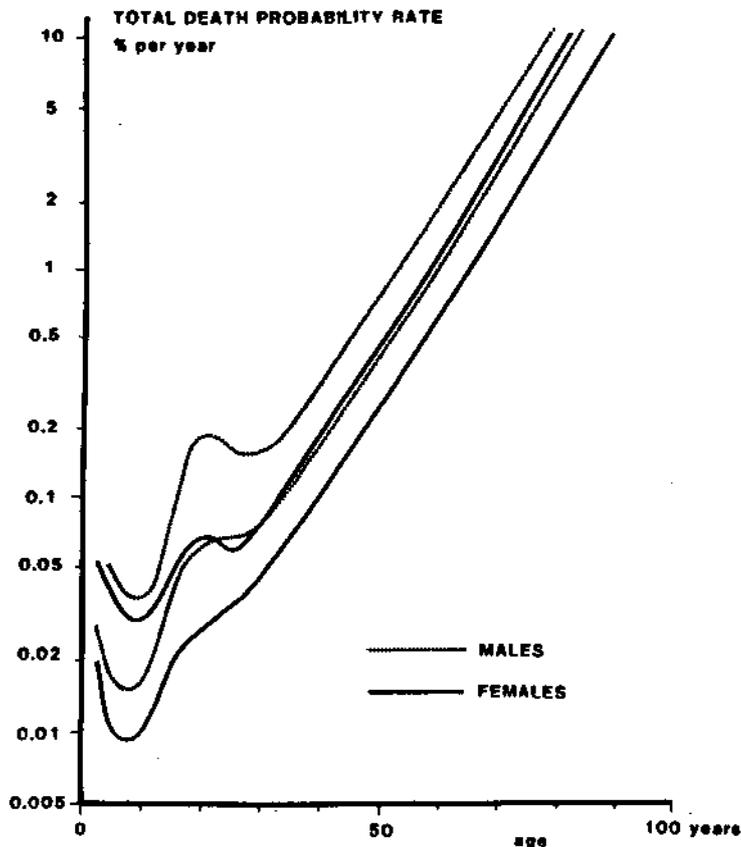


Fig. C-6. Variation (extreme values) of the age-specific mortality rate, approximating the conditional death probability rate, for 18 industrialised countries usually considered "safe": Australia, Austria, Belgium, Canada, Denmark, Finland, France, Germany (GDR), Germany (FRG), Italy, Japan, the Netherlands, New Zealand, Norway, Sweden, Switzerland, United Kingdom and USA.

the background rate do not increase at high ages. In the case of exposure at 1 mSv/year from age zero, the maximum increase is about 1.4% with the additive model and about 0.9% with the multiplicative model (see Table C-4), higher for females than for males because of their lower background risk. The corresponding changes in the Gompertz-Makeham curves for 5 mSv/year are shown in Figure C-7, while the changes caused by 50 mSv/year from age 18 to age 64 are shown in Figure C-8. The changes for 50 mSv/year have maximum values of about 40% in the case of the additive model and 17% with the multiplicative model. These changes may be compared with the variation of the age-

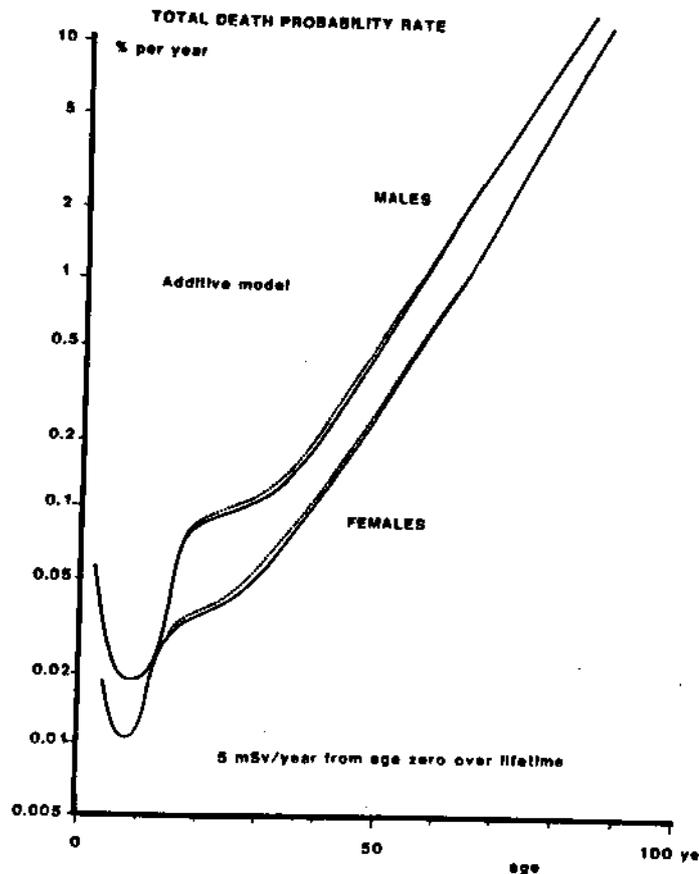


Fig. C-7. Change in the total conditional death probability rate (reference: the Swedish population 1986) after an exposure of 5 mSv per year from birth over lifetime, assuming a DDREF of 2. The change is only shown for the additive projection model. With the multiplicative model the change is smaller for ages below 50 years. At higher ages it is less than 4.5% for females and less than 2.5% for males; these changes are too small to illustrate in this diagram.

specific mortality rate (from all causes) within some industrialised countries which are usually considered "safe" (Figure C-6).

(C55) Figure C-8 also illustrates the fact that the largest increases in relative risk occur earlier in life if the additive projection model is valid.

(C56) Table C-3 gives the unconditional annual death probability rates, i.e. the values in Table C-2 multiplied by the modified (Swedish) survival probabilities, related to the age at the onset of the exposure period (i.e. age zero and age 18 years in the two cases) but corrected for the increased risk because of the radiation exposure.

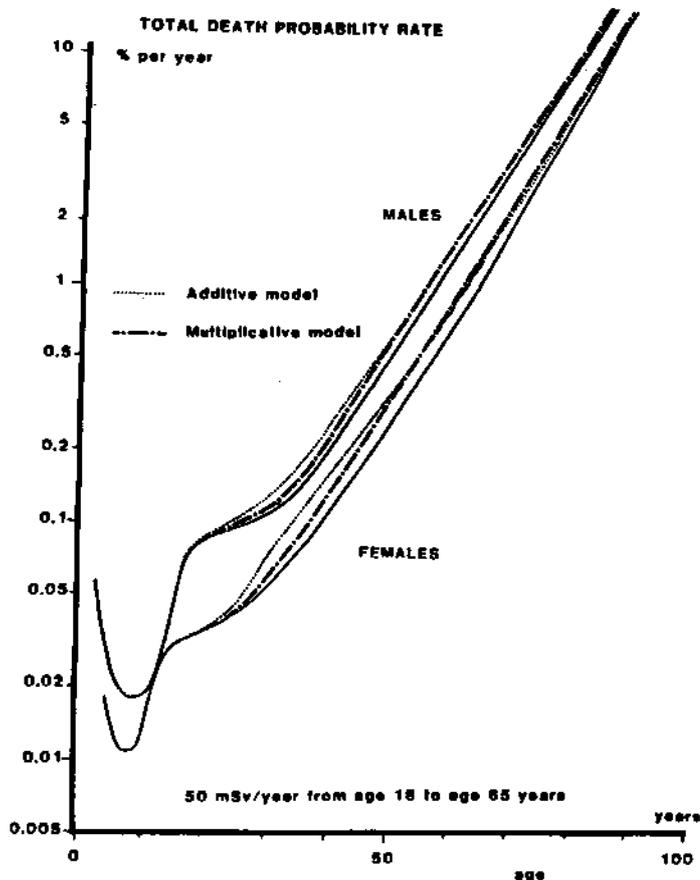


Fig. C-8. Change in the total conditional death probability rate (reference: the Swedish population 1986) after an exposure of 50 mSv per year from age 18 to age 65 years, assuming a DDREF of 2. The change is shown for each of the two projection models.

### C.9. Summary of the Risk Description

(C57) The unconditional death probability rates for females are presented in Figure C-9. The curves can also be seen to represent the normalised probability density of the age of death from radiation-induced cancer. The areas under the curves represent the lifetime probability of dying from cancer caused by the irradiation. These values are given in Table C-4.

(C58) Figure C-9 also illustrates the difference in time distribution of the radiation-induced cancer deaths. With the multiplicative model, the deaths, on the average, will occur significantly later than with the additive model.

Table C-3a. The unconditional ("expressed" or *a priori*) death probability rate ( $dr/du$ ), attributable to various annual doses from birth over lifetime. Assumed DDREF=2. The values are given as annual numbers per million of the initial population (age 18) of each sex. The sum of all annual numbers is the attributable lifetime risk as assessed at age 18. For comparison, the Swedish normalised death age probability density for age 18, i.e.  $S(18,u)G_0(u)$  is also given for each sex

Age	$S(18,u)G_0(u)$ (per million)	Annual doses (mSv)							
		Additive model				Multiplicative model			
		1	2	3	5	1	2	3	5
<b>Males</b>									
0	7200	0	0	0	0	0	0	0	0
5	230	1	2	2	4	1	1	2	3
10	180	2	4	5	9	1	3	4	7
15	400	3	5	8	14	2	3	5	9
20	850	4	8	11	19	2	3	7	11
25	950	6	12	18	30	3	6	9	15
30	1050	9	17	26	43	5	9	14	23
35	1320	13	26	39	66	7	15	22	37
40	1930	18	35	53	87	12	24	36	60
45	2810	20	41	61	102	17	35	52	87
50	4950	23	46	70	116	25	50	75	124
55	7690	27	53	80	133	38	75	113	188
60	11,600	29	59	88	147	55	111	166	276
65	17,400	31	61	91	152	77	153	229	381
70	24,100	29	59	88	147	98	196	294	488
75	31,400	26	51	77	127	114	227	339	562
80	34,000	19	38	57	94	111	220	329	544
85	29,700	11	22	33	55	83	166	247	408
90	17,200	5	9	14	23	43	86	128	210
95	5960	1	2	3	5	13	26	38	62
100	720	0	0	0	0	1	3	4	6
<b>Females</b>									
0	6370	0	0	0	0	0	0	0	0
5	140	1	1	2	3	1	1	2	3
10	115	2	3	5	8	1	3	4	7
15	300	3	6	9	15	2	3	5	9
20	345	4	9	13	22	2	4	7	11
25	395	7	13	20	33	3	6	9	15
30	520	9	19	28	46	4	9	13	22
35	750	13	26	39	66	7	14	21	34
40	1080	17	34	51	84	11	22	33	55
45	1600	20	40	61	101	16	32	48	81
50	2650	24	47	71	118	24	48	71	119
55	4350	28	56	84	140	36	72	109	181
60	6360	32	64	96	160	54	108	162	269
65	9740	35	70	105	175	77	154	231	385
70	15,800	37	74	110	183	105	210	315	523
75	25,100	36	73	109	181	134	267	399	662
80	33,900	32	64	97	160	151	301	449	744
85	39,900	24	47	70	117	139	277	413	682
90	32,200	12	25	37	62	92	183	273	448
95	16,100	4	8	12	19	36	71	106	173
100	3000	0	1	1	2	5	10	15	24

(C59) Figure C-10 supplements Figure C-9 in that the unconditional attributable death probability rates are compared with the total unconditional death probability rate related to age 18 years for females. Figure C-11 gives the net change in the unconditional death probability rate. The fact that radiation causes some premature death gives a negative net attributable unconditional death probability rate at higher ages, because the

Table C-3b. The unconditional ("expressed" or *a priori*) death probability rate ( $dr/du$ ), attributable to various annual doses from age 18 to age 65. Assumed DDREF = 2. The values are given as annual numbers per million of the initial population (age 18) of each sex. The sum of all annual numbers is the attributable lifetime risk as assessed at age 18. For comparison, the Swedish normalized death age probability density for age 18, i.e.  $S(18,u)G_A(u)$ , is also given for each sex

Age	$S(18,u)G_A(u)$ (per million)	Annual doses (mSv)							
		Additive model				Multiplicative model			
		10	20	30	50	10	20	30	50
<b>Males</b>									
0	0	0	0	0	0	0	0	0	0
5	0	0	0	0	0	0	0	0	0
10	0	0	0	0	0	0	0	0	0
15	0	0	0	0	0	0	0	0	0
20	860	1	2	3	5	0	1	1	2
25	965	11	21	32	53	3	7	10	16
30	1070	33	65	98	164	9	17	26	43
35	1340	78	156	234	389	19	38	57	94
40	1950	123	245	368	612	38	76	114	190
45	2840	161	321	480	798	67	134	201	335
50	5010	198	395	590	979	114	227	341	566
55	7780	240	477	713	1180	181	362	542	901
60	11,700	272	541	808	1330	274	545	816	1350
65	17,600	284	564	841	1380	378	753	1120	1860
70	24,400	270	534	795	1300	481	955	1420	2330
75	31,800	225	445	660	1080	550	1090	1610	2620
80	34,400	148	293	433	703	508	999	1470	2360
85	30,000	78	153	226	365	365	712	1040	1640
90	17,400	29	56	83	133	181	349	503	774
95	6030	6	12	18	29	52	98	139	207
100	730	0	1	1	2	5	9	13	18
<b>Females</b>									
0	0	0	0	0	0	0	0	0	0
5	0	0	0	0	0	0	0	0	0
10	0	0	0	0	0	0	0	0	0
15	0	0	0	0	0	0	0	0	0
20	350	0	1	2	3	0	1	1	2
25	400	6	12	18	30	3	6	9	15
30	525	26	52	78	130	8	16	24	39
35	750	65	130	195	325	17	34	52	86
40	1090	103	207	310	516	35	70	104	174
45	1620	143	286	429	713	61	122	183	305
50	2670	185	369	553	917	104	207	310	516
55	4390	234	466	696	1150	166	331	496	824
60	6420	277	552	825	1360	254	507	759	1260
65	9840	309	615	917	1510	367	731	1090	1800
70	15,900	324	643	956	1570	502	998	1490	2450
75	25,300	311	616	914	1490	637	1260	1870	3060
80	34,200	247	489	724	1180	687	1350	2000	3230
85	40,300	163	321	474	768	608	1190	1740	2780
90	32,500	78	154	226	365	387	749	1090	1700
95	16,200	22	44	65	104	145	277	396	601
100	3000	2	5	7	11	20	37	52	75

integrated rate (the total change in death probability) must be zero—the probability of final death is always 100%.

(C60) The summary Table C-4 lists values for a number of quantities which may be used to express the radiation risk. In the individual case, apart from diagrams such as those in Figure C-9, the most informative presentation is by the combination of the

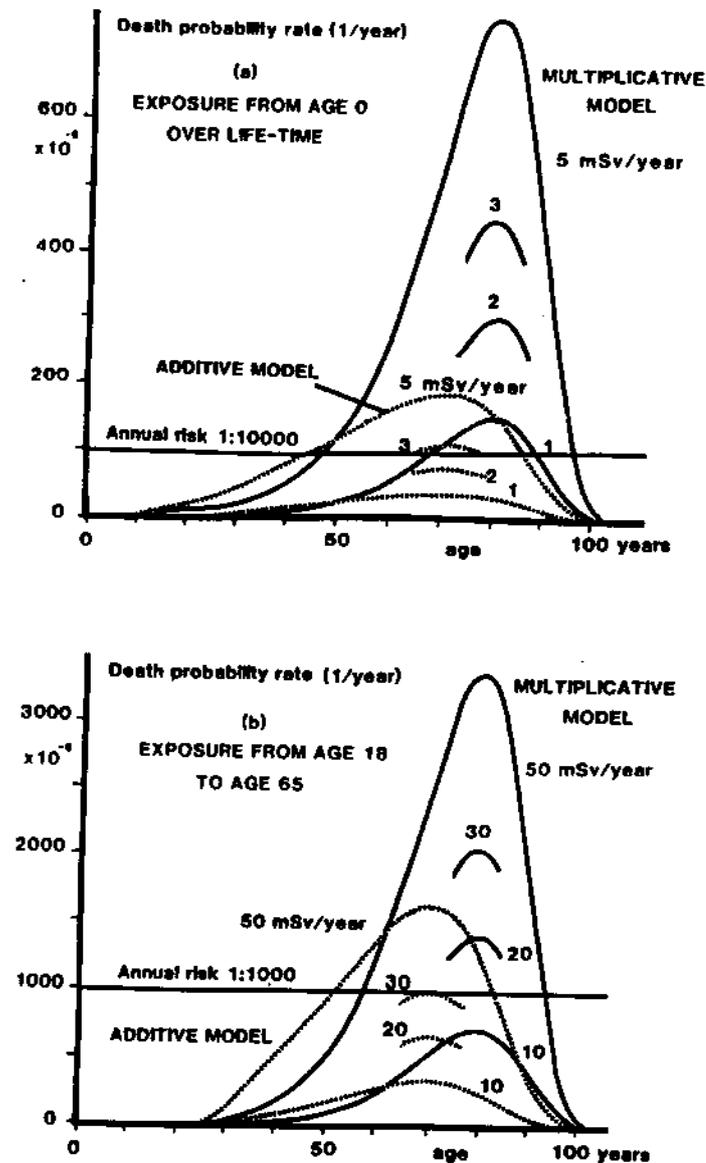


Fig. C-9. The unconditional death probability rate (the attributable probability density of the age of death, normalised for lifetime risk) for two exposure situations: (a) exposure from birth over lifetime, and (b) exposure from age 18 to age 65 years. The curves are for females, assuming a DDREF of 2.

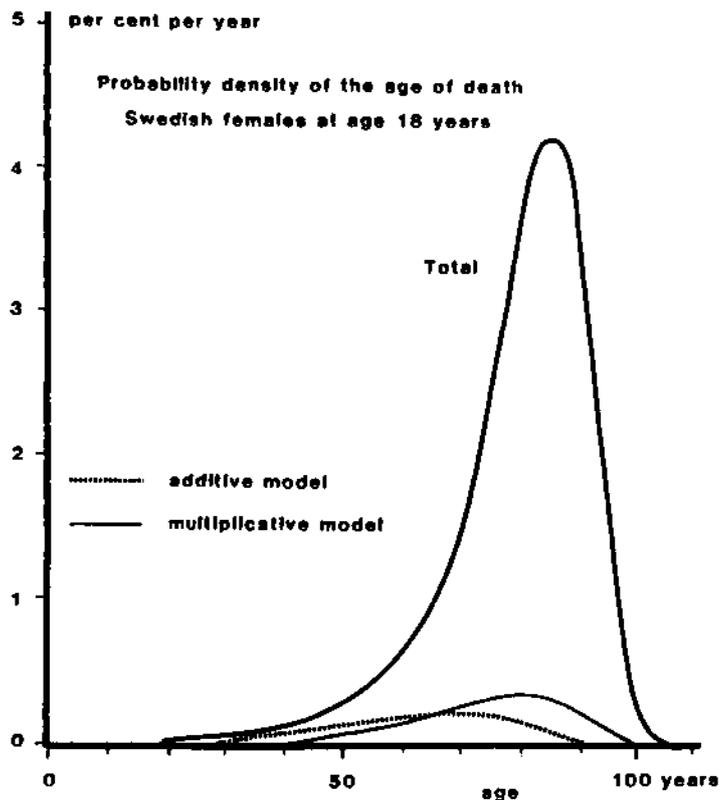


Fig. C-10. Comparison between the probability density of the age of death from all causes and the probability density of the age of attributable radiation death normalised to make the attributable lifetime risk of death directly comparable with the 100% total lifetime risk. For females exposed at 50 mSv per year from age 18 to age 65 years. DDREF assumed to be 2. (cf. Figure C-11 for the net change of the probability density.)

attributable lifetime probability of death and the mean loss of lifetime in the case of death from radiation-induced cancer. In the collective case, for a population exceeding the inverse value of the per caput attributable lifetime probability of death, the detriment represented by the expected number of cancer deaths or by the collective loss of man years is informative although not relevant for individual risk limitation.

(C61) The values in Table C-4 should be used with great caution. They are valid only if the risk coefficients used in the 1988 UNSCEAR report (Table C-1 in this Annex) are valid for all ages in each age group. This is surely not the case; the values are averages for each age group and there is not enough information to indicate the variation within the age groups. This is particularly important with regard to the youngest age group.

(C62) Table C-4 is the most comprehensive indication of the various consequences of

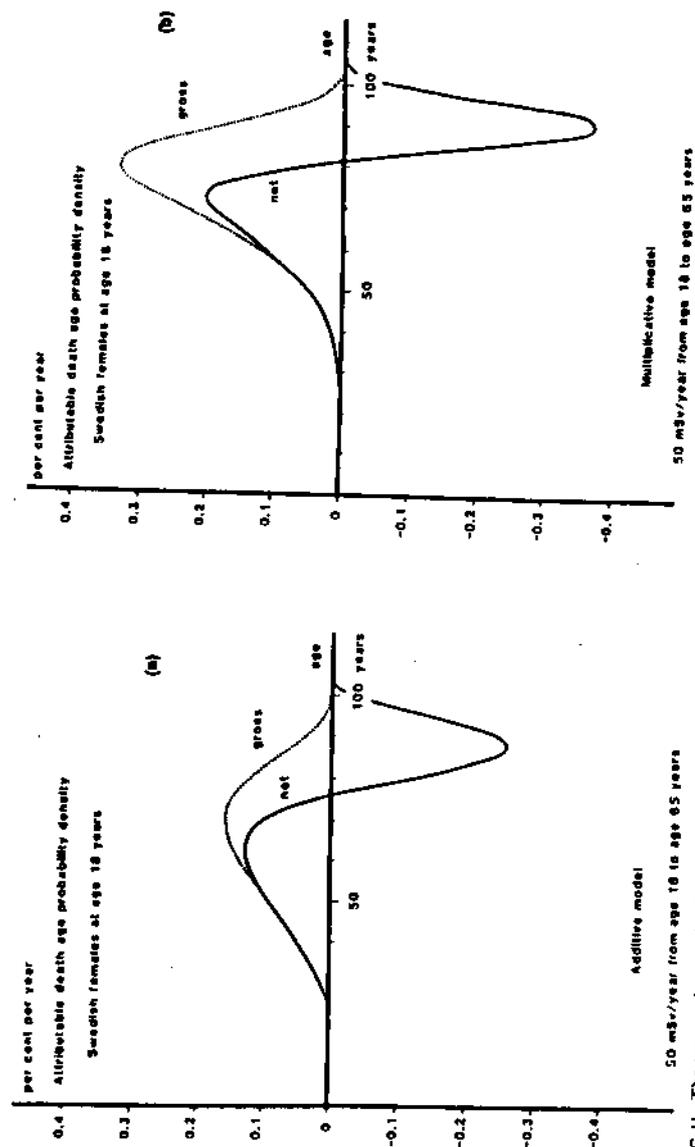


Fig. C-11. The gross and net probability density of the age of attributable death. The integrated change in total probability density of the age of death must be zero since the total probability of death from all causes will remain 100%.

Table C-4a. Summary table (averages for both sexes). Exposure from age zero over lifetime. Assumed DDREF = 2. Uniform whole-body dose. The upper numbers relate to the additive projection model, the lower numbers to the multiplicative model

Quantity to describe "risk"	Annual dose (mSv)			
	1	2	3	5
Attributable lifetime probability of cancer death (%)	0.15	0.31	0.46	0.77
	0.40	0.80	1.12	1.99
Loss of lifetime if cancer death (years)	22.6	22.6	22.6	22.6
	13.4	13.4	13.4	13.5
Loss of life expectancy at age 0 (man years per caput)	0.03	0.07	0.10	0.17
	0.05	0.11	0.16	0.27
Mean annually committed probability of attributable cancer deaths 0-70 years (per million)	21	44	66	110
	57	115	160	280
Annual extra probability of cancer death at age 70 years (per million)	44	87	131	218
	135	270	405	675
Most probable age at attributable death (years)	68	68	68	68
	79	79	79	79
Maximum relative death probability rate (%)	1.4	2.7	4.0	7.0
	0.9	1.8	2.7	4.4
Age at maximum relative rate (years)	34	34	34	34
	42	42	42	42
Maximum risk equivalent aging (years)	0.3	0.6	0.9	1.5
	0.2	0.3	0.5	0.8
Deaths* per million and year in a mixed population at 10% of the dose level	2	4	6	10
	5	10	15	25

\* Attributable cancer deaths.

Table C-4b. Summary table (averages for both sexes). Exposure from age 18 to age 65 years. Assumed DDREF = 2. Uniform whole-body dose. The upper numbers relate to the additive projection model, the lower numbers to the multiplicative model

Quantity to describe "risk"	Annual dose (mSv)				
	3	10	20	30	50
Attributable lifetime probability of cancer death (%)	0.35	1.16	2.31	3.44	5.66
	0.55	1.81	3.57	5.28	8.56
Loss of lifetime if cancer death (years)	19.8	19.8	19.8	19.9	20.0
	12.6	12.7	12.7	12.8	13.0
Loss of life expectancy at age 18 (man years per caput)	0.07	0.23	0.46	0.69	1.12
	0.07	0.23	0.46	0.68	1.11
Mean annually committed probability of attributable cancer deaths, 18-65 years (per million)	74	250	490	730	1200
	120	385	760	1120	1820
Annual extra probability of cancer death at age 70 years (per million)	120	390	780	1200	2000
	200	650	1300	2000	3300
Most probable age at attributable death (years)	68	68	68	68	68
	78	78	78	77	77
Maximum relative death probability rate (%)	2.5	9	17	25	41
	1.0	3	7	10	17
Age at maximum relative rate (years)	39	39	39	39	39
	57	57	57	57	57
Maximum risk equivalent aging (years)	0.2	0.8	1.6	2.3	3.9
	0.07	0.2	0.5	0.7	1.2

life-long exposures at various dose levels. These data have also been used, together with other relevant information, for the multi-attribute approach to selection of the dose limits recommended in the main text. The reader may use them in order to see the possible consequences of applying these recommendations to individuals exposed at the limits over their normal lifetime. They may be compared with similar expressions of risk in other areas.

### C.10. A Multi-attribute Approach to the Selection of Dose Limits

(C63) If all radiation risks were of a deterministic nature, with a comparatively high threshold dose, the selection of dose limits would, to a high degree, be a scientific task and the outcome would heavily depend on the magnitude of the dose threshold. Unfortunately, there is an additional risk of stochastic effects at doses below the thresholds for known deterministic effects. As long as the dose-response relationship for the stochastic effects is without great discontinuities the selection of a dose limit is only partially a scientific decision. It is mainly a value judgment which would need to be based not only on the scientific information but also on knowledge of the level of risk that is usually considered unacceptable under normal conditions. This is a policy matter for the Commission which is discussed in the main text. This Annex can only provide some of the necessary background information relating to the radiation risks.

(C64) The Annex has shown that the radiation "risk" can be presented in a number of ways. This means that, by different modes of presentation, the description may cause quite different impressions. If only the conditional death probability rate is shown, e.g. by the shift of the Gompertz curves, even relatively high annual doses would not seem to change the exposed person's overall risk situation significantly, and the change may be small in comparison with the risk differences between the sexes or between countries which are not usually considered to differ much in risk to their residents. However, the same risk expressed as reduction of life expectancy or (at about the same magnitude) as statistical aging, may amount to several years and therefore perhaps look less acceptable.

(C65) In the case of exposure over many years, such as over an occupational lifetime, the annual incremental death probability expressed at different ages will vary considerably. If a dose limit is to be derived from the annual risk, what is then the appropriate age for which the risk should be assessed? How should the probability increment be expressed? In absolute terms or in relative terms?

(C66) Some readers might ask what the annual dose to workers or members of the public would be, which, with the new risk estimates, would cause the same cancer risk as the old dose limits, in 1977, were assumed to have caused. This question cannot be answered unambiguously since the answer would depend on the age for which one would like to make the comparison. This is illustrated in Table C-5 by data derived from Table C-2 and the multiplicative projection model. The table shows the conditional incremental cancer death probability rate, averaged over the sexes, at various ages and annual doses, for workers exposed from age 18 to age 65 and for members of the public exposed from birth. For comparison, an annual risk of  $1.25 \times 10^{-2}$  Sv<sup>-1</sup>, as assumed in 1977, is given for all ages (although a latent period was recognised, it was not taken into account).

(C67) The information in Table C-5 is not sufficient basis for judging the appropriateness of a new dose limit. One reason is that it would first be necessary to judge whether the cancer risk assumed at the limit in 1977 was appropriately limited at that time and whether the same views on an appropriate risk limit still prevail. Another reason is that

Table C-5. Comparison of present (multiplicative model and a DDREF of 2) and 1977 risk estimates for the annual conditional cancer death probability (per million) at various ages and annual doses

Annual dose (mSv)	Age at risk, years						
	30	40	50	60	65	70	75
<i>Workers</i>							
50	42	190	570	1500	2200	3200	4700
30	25	110	340	880	1300	2000	2800
20	17	75	230	590	890	1300	1900
15	13	55	170	440	650	1000	1400
10	8	37	114	295	445	650	930
50 (1977)	625	625	625	625	625	625	625
<i>Public</i>							
5	4	20	60	150	220	320	470
3	2	12	35	90	130	200	280
2	2	8	24	60	90	130	190
1	1	4	12	30	45	65	95
0.5	0.4	2	6	15	22	32	47
1 (1977)	12	12	12	12	12	12	12

the risk of lethal cancer is only part of the total radiation risk and that other parts, such as the risks of curable cancer and hereditary harm, should also be taken into account.

(C68) The nominal probability coefficients are composed of three components, namely, fatality coefficient, weighted coefficient for curable cancer, and weighted coefficient for hereditary effects. In the 1977 report, only severe hereditary effects in the first two generations were included and curable cancer was not counted in the risk coefficient, although it was crudely assessed in the Commission's 1980 Brighton statement (ICRP, 1980) as a detriment of about 10% of the fatality detriment. Any comparison between new limits and the 1977 limits should consider all these components. This leads to the comparison shown in Table C-6. With the new estimates, according to Annex B, the weighted coefficient for curable cancer is about 20% of the fatality coefficient for both workers and the public. The weighted coefficient for hereditary effects is  $1.33 \times 10^{-2} \text{ Sv}^{-1}$  for the public and  $0.80 \times 10^{-2} \text{ Sv}^{-1}$  for workers. These figures are based upon 47 years for the worker and 75 year lifetime for the public. The nominal cancer fatality coefficient is  $5 \times 10^{-2} \text{ Sv}^{-1}$  for the public and  $4 \times 10^{-2} \text{ Sv}^{-1}$  for workers, but the attributable lifetime cancer death probabilities at the various annual doses have been taken from Table C-4.

(C69) The comparisons in paragraph C66 relate to cancer death probabilities, as if all deaths from cancer involved one and the same degree of harm. However, the probability of death alone does not give enough indication of the risk. For example, a very high probability, assessed at birth, that a person will die at a high age would be seen as positive information, while the same probability of death in childhood would be negative information. In 1977, the Commission assumed that a radiation induced death from cancer would, on the average, mean a loss of life of 10–15 years (ICRP, 1977b). On the additive projection model, the loss of lifetime is about 20 years, but on the multiplicative model only about 13–15 years (see Tables C-4 and B-18). A more realistic estimate in 1977, when the additive model was used, would have indicated a longer loss of lifetime than was then assumed. In that respect, the present higher probability estimate with the multiplicative model is to some degree compensated by the shorter loss of lifetime.

Table C-6. The detriment at various annual doses, as assessed at present on the basis of the multiplicative projection model, compared with the total risk assessed in 1977 at the old dose limits

Annual effective dose (mSv)	Probability ( $10^{-3}$ )			
	Fatal cancer	Weighted curable cancer <sup>1</sup>	Weighted hereditary <sup>1</sup>	Aggregated detriment <sup>2</sup>
<i>Workers (exposure from age 18 to age 65)</i>				
50	8.6	1.72	1.72	12.0
30	5.3	1.06	1.06	7.4
20	3.6	0.72	0.72	5.0
10	1.8	0.36	0.36	2.5
50 (1977)	2.9	—	—	—
<i>Public (exposure from birth over lifetime)</i>				
5	2.0	0.40	0.53	2.93
3	1.1	0.22	0.29	1.61
2	0.8	0.16	0.21	1.17
1	0.4	0.08	0.11	0.59
0.5	0.2	0.04	0.05	0.29
1 (1977)	0.1	—	—	—

<sup>1</sup> The weighting is for severity and length of life lost.

<sup>2</sup> The sum of columns 2, 3 and 4.

(C70) In 1977, the Commission assumed that an annual occupational fatality probability of  $10^{-3}$  might be taken as a reference risk for the dose limit. This was made on the assumption that, in "safe" non-radiation occupations, the average annual fatality rate was about 100 per million workers and that subgroups with high risks might run a risk ten times the average. It can be seen from Table C-5 that an annual cancer death probability of  $10^{-3}$  is not exceeded before age 65 years for annual doses below 20 mSv, nor before age 75 years for annual doses below 10 mSv. For 50 mSv per year, it is exceeded above age 55 years.

(C71) The Commission, in 1977, expressed the view that "the calculated rate at which fatal malignancies might be induced by occupational exposure to radiation should in any case not exceed the occupational fatality rate of industries recognised as having high standards of safety". This implies an ambition to design a system of radiation protection that will keep the average doses to radiation workers low, as, in fact, the recommended principle of optimisation of protection has achieved. However, it must be seen as irrelevant to those receiving the highest doses to know how many workers receive low doses.

(C72) Comparisons with risks in other occupations are therefore difficult, because information on risks usually refer to average risks. There is the additional problem with the relevance of comparisons with accident fatality probabilities. One reason is that there are significant risks not related to radiation in radiation work, for example in mines and factories. Another reason is that there is also, in all industries, occupational disease which may cause untimely death. There may also be differences in the mean loss of life when death occurs; it has been estimated that the mean loss of life from an accidental death in industries may be as high as 35 years (ICRP, 1985).

(C73) The choice of an appropriate dose limit for members of the public is even more

difficult because of the many sources of risk, in addition to radiation risks, to which the public is exposed (see paragraphs C31-C35), and the arbitrariness in allotting some fraction of a (non-existing) total risk limit to radiation risks. The observation by Travis *et al.* (1987a,b) (see paragraph C15), that chemical carcinogens exposing the US public to an attributable life time cancer death probability of more than  $4 \times 10^{-3}$  seem to have been regulated regardless of cost, has no direct relevance to the radiation case. The regulatory action was source-related, i.e. related to individual substances, while the ICRP dose limits are individual-related. With the multiplicative model and DDREF=2, an annual dose of 1 mSv will cause an attributable lifetime fatality probability of  $4 \times 10^{-3}$ . However, the limit of 1 mSv is not intended to apply to each radiation practice but to the total dose from all regulated practices.

(C74) The natural radiation background must be assumed to cause risks which are related to the radiation dose in the same way as described in Annex B and in this Annex. Irrespective of the uncertainties in the assessment of the absolute magnitude of a risk, the relative magnitude of any radiation risk in relation to the risk from background radiation is described by the ratio of the annual effective doses. The fact that a man-made practice involving radiation causes doses which are small in comparison with the background doses does not necessarily imply that the practice is justified, but it does imply that the radiation risk situation of the exposed individual is not significantly changed by the new practice.

(C75) The risk data in this Annex are only part of the information needed for the selection of dose limits. A number of additional attributes has to be considered. However, since these do not describe the biological risk, they do not belong in this Annex but are discussed in the main text. The reader is warned not to draw hasty conclusions from the risk information alone. Other attributes also have to be considered, which determine what would be a risk that under normal conditions would be considered to be at the borderline of unacceptability.

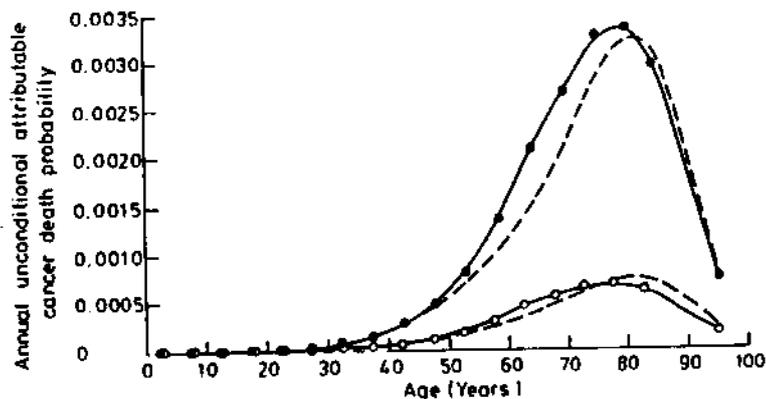


Fig. C-12. Comparison of the data in Table C-2 (and Figure C-9) and results from a UK assessment (see paragraph C76). ●—● females exposed at 50 mSv per year from age 18 to age 65; ○—○ females exposed at 5 mSv per year from birth over lifetime; --- corresponding data from Table C-2.

(C76) The validity of the calculations in this Annex, provided that the primary risk coefficients from UNSCEAR (1988), are valid, has been confirmed by independent assessments. One example is calculations carried out within the United Kingdom National Radiological Protection Board (NRPB, 1990) for the British population. Some results, in comparison with data from Table C-2, are shown in Figure C-12. The somewhat lower unconditional attributable cancer death probability and earlier mean age of death is what might be expected because of the difference between the British and Swedish Gompertz curves. The shape of the curves in Figure C-12 is primarily determined by the conditional background total death probability rate.

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