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Aims and Scope

The International Commission on Radiological Protection (ICRP) is the primary body in protection against ionising radiation. ICRP is a registered charity and is thus an independent non-governmental organisation created by the 1928 International Congress of Radiology to advance for the public benefit the science of radiological protection. The ICRP provides recommendations and guidance on protection against the risks associated with ionishing radiation, from artificial sources widely used in medicine, general industry and nuclear enterprises, and from naturally occurring sources. These reports and recommendations are published four times each year on behalf of the ICRP as the journal *Annals of the ICRP*. Each issue provides in-depth coverage of a specific subject area.

Subscribers to the journal receive each new report as soon as it appears so that they are kept up to date on the latest developments in this important field. While many subscribers prefer to acquire a complete set of ICRP reports and recommendations, single issues of the journal are also available separately for those individuals and organizations needing a single report covering their own field of interest. Please order through your bookseller, subscription agent, or direct from the publisher.

ICRP is composed of a Main Commission and five standing Committees on: radiation effects, doses from radiation exposure, protection in medicine, the application of ICRP recommendations, and protection of the environment, all served by a small Scientific Secretariat. The Main Commission consists of twelve members and a Chairman. Committees typically comprise 15–20 members. Biologists and medical doctors dominate the current membership; physicists are also well represented.

ICRP uses Working Parties to develop ideas and Task Groups to prepare its reports. A Task Group is usually chaired by an ICRP Committee member and usually contains a majority of specialists from outside ICRP. Thus, ICRP is an independent international network of specialists in various fields of radiological protection. At any one time, about one hundred eminent scientists are actively involved in the work of ICRP. The Task Groups are assigned the responsibility for drafting documents on various subjects, which are reviewed and finally approved by the Main Commission. These documents are then published as the *Annals of the ICRP*.

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PUBLICATION 103

The 2007 Recommendations of the International Commission on Radiological Protection

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The 2007 Recommendations of the International Commission on Radiological Protection

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Approved by the Commission in March 2007

Abstract–These revised Recommendations for a System of Radiological Protection formally replace the Commission's previous, 1990, Recommendations; and update, consolidate, and develop the additional guidance on the control of exposure from radiation sources issued since 1990.

Thus, the present Recommendations update the radiation and tissue weighting factors in the quantities equivalent and effective dose and update the radiation detriment, based on the latest available scientific information of the biology and physics of radiation exposure. They maintain the Commission's three fundamental principles of radiological protection, namely justification, optimisation, and the application of dose limits, clarifying how they apply to radiation sources delivering exposure and to individuals receiving exposure.

The Recommendations evolve from the previous process-based protection approach using practices and interventions by moving to an approach based on the exposure situation. They recognise planned, emergency, and existing exposure situations, and apply the fundamental principles of justification and optimisation of protection to all of these situations. They maintain the Commission's current individual dose limits for effective dose and equivalent dose from all regulated sources in planned exposure situations. They re-inforce the principle of optimisation of protection, which should be applicable in a similar way to all exposure situations, subject to the following restrictions on individual doses and risks; dose and risk constraints for planned exposure situations, and reference levels for emergency and existing exposure situations. The Recommendations also include an approach for developing a framework to demonstrate radiological protection of the environment.
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Editorial

WE COULD NOT HAVE DONE IT WITHOUT YOUR HELP

The new recommendations of the International Commission on Radiological Protection were adopted on 21 March 2007, Essen, Germany, after eight years of discussions, involving scientists, regulators, and users all around the world.

The Commission is an advisory body that offers its recommendations to regulatory and advisory agencies, mainly by providing guidance on the fundamental principles on which appropriate radiological protection can be based. Since its inception in 1928, the Commission has regularly issued recommendations regarding protection against the hazards of ionising radiation. The first report in the current series, *Publication 1,* contained the recommendations adopted in 1958 (ICRP, 1959). The more recent recommendations have appeared as *Publication 26* (ICRP, 1977), and *Publication 60* (ICRP, 1991b), and contain the recommendations adopted in 1977 and 1990, respectively.

International organisations and national authorities responsible for radiological protection, as well as the users, have taken the recommendations and principles issued by the Commission as a key basis for their protective actions. As such, virtually all international standards and national regulations addressing radiological protection are based on the Commission's recommendations.

Currently, most national regulations are based on the 1990 Recommendations in *Publication 60*. International standards, such as the International Basic Safety Standards, various international labour conventions, and European directives on radiological protection are also based on these recommendations.

In *Publication 26*, the Commission quantified the risks of stochastic effects of radiation and proposed a system of dose limitation with its three principles of justification, optimisation of protection, and individual dose limitation. In *Publication 60*, the Commission revised its recommendations and extended its philosophy to a system of radiological protection while keeping the fundamental principles of protection.

New scientific data have been published since *Publication 60*, and while the biological and physical assumptions and concepts remain robust, some updating is required. The overall estimate of deterministic effects remain fundamentally the same. The estimates of cancer risk attributable to radiation exposure have not changed greatly in the past 17 years, whereas the estimated risk of heritable effects is currently lower than before. The new data provide a firmer basis on which to model risks and assess detriment.

The 2007 Recommendations evolve from the previous process-based approach of practices and interventions to an approach based on the characteristics of radiation exposure situations. The system of radiological protection applies in principle to any situation of radiation exposure. Similar procedures are used for deciding on the extent and level of protective actions, regardless of exposure situation. Specifically, the principles of justification and optimisation apply universally. ICRP is of the opinion that by focusing more on optimisation, the implementation of protection for what has until now been categorised as interventions could be enhanced.

In view of the importance afforded to the Commission's recommendations and to ensure that the new recommendations adequately and appropriately address national issues and concerns, the Commission has initiated a much more open process than that used for the development of the previous recommendations. It should also be noted that the Commission mentions, for the first time, the need to account for the views and concerns of stakeholders when optimising protection.

The Commission has therefore solicited input from a broad spectrum of radiological protection stakeholders, ranging from government institutions and international organisations to scientists and non-governmental organisations. The draft recommendations have been discussed at a large number of international and national conferences and by the many international and national organisations with an interest in radiological protection.

Many of these also arranged particular activities around the Recommendations project. Thus for instance, the International Radiation Protection Association arranged reviews through its member organisations world-wide for their 2000 and 2004 Congresses and in connection with our 2006 public consultation, the Nuclear Energy Agency of the OECD organised seven international workshops and performed four detailed assessments of draft ICRP texts (in 2003, 2004, 2006, and 2007), and the European Commission organised a seminar in 2006 to debate the scientific issues in the Recommendations. The United Nations agencies, with the International Atomic Energy Agency as the lead agency, are using the 2007 ICRP Recommendations as a major input to their project of revising the International Basic Safety Standards, and likewise the European Commission uses the 2007 Recommendations as a major input to their revision of the European Basic Safety Standards.

The Recommendations have been prepared after two phases of international public consultation. By following this policy of transparency and involvement of stakeholders, ICRP is expecting a clearer understanding and wide acceptance of its Recommendations. Although the revised Recommendations do not contain any fundamental changes to the radiological protection policy, they will help to clarify application of the system of protection in the plethora of exposure situations encountered, thereby improving the already high standards of protection.

The Commission is pleased at having arrived at the end of a long but useful gestation phase including numerous consultations and is proud to present these 2007 Recommendations. The extensive consultations resulted in a much improved document and the Commission is grateful to the many organisations, experts, and individual members of the public who have devoted so much of their time and

experience to helping us to improve the Recommendations. Their contributions have been crucial for the future success of the 2007 Recommendations.

LARS-ERIK HOLM
CHAIRMAN, ICRP

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PREFACE

Since issuing its 1990 Recommendations as ICRP *Publication 60* (ICRP 1991b), the Commission has reviewed these Recommendations regularly and, from time to time, has issued supplementary reports in the *Annals of the ICRP*. The extent of these supplementary reports has indicated the need for the consolidation and rationalisation presented here. New scientific data have also been published since *Publication 60*, and while the biological and physical assumptions and concepts remain robust, some updating is required. The overall estimates of deterministic effects and stochastic risk remain fundamentally the same. The overall estimates of cancer risk attributable to radiation exposure have not changed appreciably in the past 16 years. Conversely, the estimated risk of heritable effects is currently lower than before. Overall, the new data provide a firmer basis on which to model risks and assess detriment. Finally, it has also become apparent that the radiological protection of the environment should receive more emphasis than in the past.

Therefore, while recognising the need for stability in international and national regulations, the Commission has decided to issue these revised Recommendations having two primary aims in mind:

- to take account of new biological and physical information and of trends in the setting of radiation safety standards; and
- to improve and streamline the presentation of the Recommendations.

In addition, the Commission has maintained as much stability in the Recommendations as is consistent with the new scientific information and societal expectations.

In its revised System of Protection, the Recommendations of the Commission now evolve from the previous process-based approach of practices and interventions to an approach based on the characteristics of radiation exposure situations. In taking this approach, the Commission wishes to affirm that its system of protection can be applied in principle to any situation of radiation exposure. Similar procedures are used for deciding on the extent and level of protective actions, regardless of exposure situation. Specifically, the principles of justification and optimisation apply universally. The Commission is of the opinion that the implementation of protection for what has until now been categorised as interventions could be enhanced by increasing the attention to these common features.

These Recommendations were produced by the Main Commission of ICRP, based on an earlier draft that was subjected to public and internal consultation in 2004 and again, in revised form, in 2006. By introducing more transparency and by involving the many organisations and individuals having an interest in radiological protection in the revision process, the Commission is expecting a better common understanding and acceptance of its Recommendations.

The membership of the Main Commission during the period of preparation of the present Recommendations was:

(2001–2005)

R.H. Clarke (Chairman)	A.J. González	Y. Sasaki
R.M. Alexakhin	L.-E. Holm (Vice-Chairman)	C. Streffer
J.D. Boice jr	F.A. Mettler jr	A. Sugier (2003–2005)
R. Cox	Z.Q. Pan	B.C. Winkler (✠ 2003)
G.J. Dicus (✠ 2006)	R.J. Pentreath (2003–2005)	

Scientific Secretary: J. Valentin

(2005–2009)

L.-E. Holm (Chairman)	J.-K. Lee	Y. Sasaki
J.D. Boice jr	H. Menzel (2007–2009)	N. Shandala
C. Cousins	Z.Q. Pan	C. Streffer (2005–2007)
R. Cox (Vice-Chairman)	R.J. Pentreath	A. Sugier
A.J. González	R.J. Preston	

Scientific Secretary: J. Valentin

The work of the Commission was greatly aided by significant contributions from P. Burns, J. Cooper, J.D. Harrison, and W. Weiss. It also benefited from discussions at many international meetings on the present Recommendations.

The Commission wishes to express its appreciation to all international and national organisations, governmental as well as non-governmental, and all individuals who contributed in the development of these Recommendations.

EXECUTIVE SUMMARY

(a) On 21 March 2007, the Main Commission of the International Commission on Radiological Protection (ICRP) approved these revised Recommendations for a System of Radiological Protection which formally replace the previous Recommendations issued in 1991 as *Publication 60* (ICRP, 1991b) and update the additional guidance on the control of exposure from radiation sources issued since *Publication 60*. These revised Recommendations consolidate and develop the previous Recommendations and guidance.

(b) The Commission has prepared these Recommendations after two phases of international public consultation, one in 2004 and one in 2006, on draft Recommendations. By following this policy of transparency and involvement of stakeholders, the Commission is anticipating a clearer understanding and wider acceptance of its Recommendations.

(c) The major features of the present Recommendations are:

- Updating the radiation and tissue weighting factors in the quantities equivalent and effective dose, and updating the radiation detriment based on the latest available scientific information of the biology and physics of radiation exposure;
- Maintaining the Commission's three fundamental principles of radiological protection, namely justification, optimisation, and the application of dose limits, and clarifying how they apply to radiation sources delivering exposure and to individuals receiving exposure;
- Evolving from the previous process-based protection approach using practices and interventions, by moving to a situation-based approach applying the fundamental principles of justification and optimisation of protection to all controllable exposure situations, which the present Recommendations characterise as planned, emergency, and existing exposure situations;
- Maintaining the Commission's individual dose limits for effective dose and equivalent dose from all regulated sources in planned exposure situations – these limits represent the maximum dose that would be accepted in any planned exposure situations by regulatory authorities;
- Re-enforcing the principle of optimisation of protection, which should be applicable in a similar way to all exposure situations, with restrictions on individual doses and risks, namely dose and risk constraints for planned exposure situations and reference levels for emergency and existing exposure situations; and
- Including an approach for developing a framework to demonstrate radiological protection of the environment.

(d) The Commission's system of radiological protection applies to all exposures to ionising radiation from any source, regardless of its size and origin. However, the Recommendations can apply in their entirety only to situations in which either the source of exposure or the pathways leading to the doses received by individuals can be controlled by some reasonable means. Some exposure situations are excluded from radiological protection legislation, usually on the basis that they are unamenable to control with regulatory instruments, and some exposure situations are

exempted from some or all radiological protection regulatory requirements where such controls are regarded as unwarranted.

(e) An understanding of the health effects of ionising radiation is central to the Commission's Recommendations. Following a review of the biological and epidemiological information on the health risks attributable to ionising radiation, the Commission has reached the following conclusions. The distribution of risks to different organs/tissues is judged to have changed somewhat since *Publication 60*, particularly in respect of the risks of breast cancer and heritable disease. However, assuming a linear response at low doses, the combined detriment due to excess cancer and heritable effects remains unchanged at around 5% per Sv. Embodied in this current estimate is the use of a dose and dose-rate effectiveness factor for solid cancers which is unchanged at a value of 2. The Commission also judges that, following prenatal exposure, a) cancer risk will be similar to that following irradiation in early childhood and b) threshold dose exists for the induction of malformations and for the expression of severe mental retardation. The Commission has retained the effective dose limits and the equivalent dose limits for the skin, hands/feet, and eye given in *Publication 60* but recognises that further information is needed and revised judgements may be required particularly in respect of the eye. The available data on possible excess in non-cancer diseases (e.g., cardiovascular disorders) are judged to be insufficient to inform on risks at low doses.

(f) The Commission's extensive review of the health effects of ionising radiation has, however, not indicated that any fundamental changes are needed to the system of radiological protection. Importantly, existing numerical recommendations in the policy guidance issued since 1991 remain valid unless otherwise stated. Therefore, these revised Recommendations should not imply any substantial changes to radiological protection regulations that are based on its previous Recommendations and subsequent policy guidance.

(g) The central assumption of a linear dose–response relationship for the induction of cancer and heritable effects, according to which an increment in dose induces a proportional increment in risk even at low doses, continues to provide the basis for the summation of doses from external sources of radiation and from intakes of radionuclides.

(h) The use of equivalent and effective dose remains unchanged, but a number of revisions have been made to the methods used in their calculation. Reviews of the range of available data on the relative biological effectiveness of different radiations, together with biophysical considerations, have led to changes to the values of radiation weighting factors used for neutrons and protons, with values for neutrons given as a continuous function of neutron energy, and the inclusion of a value for charged pions. Radiation weighting factors for photons, electrons, muons, and alpha particles are unchanged.

(i) An important change is that doses from external and internal sources will be calculated using reference computational phantoms of the human body based on medical tomographic images, replacing the use of various mathematical models. For adults, equivalent doses will be calculated by sex-averaging of values obtained using male and female phantoms. Effective dose will then be calculated using revised

age- and sex-averaged tissue weighting factors, based on updated risk data and intended to apply as rounded values to a population of both sexes and all ages. Effective dose is calculated for a Reference Person and not for an individual.

(j) Effective dose is intended for use as a protection quantity. The main uses of effective dose are the prospective dose assessment for planning and optimisation in radiological protection, and demonstration of compliance with dose limits for regulatory purposes. Effective dose is not recommended for epidemiological evaluations, nor should it be used for detailed specific retrospective investigations of individual exposure and risk.

(k) The collective effective dose quantity is an instrument for optimisation, for comparing radiological technologies and protection procedures, predominantly in the context of occupational exposure. Collective effective dose is not intended as a tool for epidemiological risk assessment, and it is inappropriate to use it in risk projections. The aggregation of very low individual doses over extended time periods is inappropriate, and in particular, the calculation of the number of cancer deaths based on collective effective doses from trivial individual doses should be avoided.

(l) In order to assess radiation doses, models are necessary to simulate the geometry of the external exposure, the biokinetics of incorporated radionuclides, and the human body. The reference models and necessary reference parameter values are established and selected from a range of experimental investigations and human studies through judgements. For regulatory purposes, these models and parameter values are fixed by convention and are not subject to uncertainty. The Commission is aware of uncertainties and lack of precision of the models and parameter values. Efforts are undertaken to critically evaluate and to reduce the uncertainties. For individual retrospective dose and risk assessments, individual parameters and uncertainties have to be taken into account.

(m) The Commission's process of consolidation of previous guidance and recommendations has indicated that some changes to the structure and terminology of the system of protection were desirable in order to improve clarity and utility. In particular the distinction between practices and interventions may not have been clearly understood in the wider radiological protection community. Additionally, there were exposure situations which were difficult to categorise in this manner.

(n) The Commission now recognises three types of exposure situations which replace the previous categorisation into practices and interventions. These three exposure situations are intended to cover the entire range of exposure situations. The three situations are:

- *Planned exposure* situations, which are situations involving the planned introduction and operation of sources. (This type of exposure situation includes situations that were previously categorised as practices.)
- *Emergency exposure* situations, which are unexpected situations such as those that may occur during the operation of a planned situation, or from a malicious act, requiring urgent attention.

- *Existing exposure* situations, which are exposure situations that already exist when a decision on control has to be taken, such as those caused by natural background radiation.

(o) The three key principles of radiological protection are retained in the revised Recommendations. The principles of *justification* and *optimisation* apply in all three exposure situations whereas the principle of *application of dose limits* applies only for doses expected to be incurred with certainty as a result of planned exposure situations. These principles are defined as follows:

- *The Principle of Justification*: Any decision that alters the radiation exposure situation should do more good than harm.
- *The Principle of Optimisation of Protection*: The likelihood of incurring exposure, the number of people exposed, and the magnitude of their individual doses should all be kept as low as reasonably achievable, taking into account economic and societal factors.
- *The Principle of Application of Dose Limits*: The total dose to any individual from regulated sources in planned exposure situations other than medical exposure of patients should not exceed the appropriate limits specified by the Commission.

The Commission continues to distinguish amongst three categories of exposure: occupational exposures, public exposures, and medical exposures of patients (and comforters, carers, and volunteers in research). If a female worker has declared that she is pregnant, additional controls have to be considered in order to attain a level of protection for the embryo/fetus broadly similar to that provided for members of the public.

(p) The revised Recommendations emphasise the key role of the principle of optimisation. This principle should be applied in the same manner in all exposure situations. Restrictions are applied to doses to a nominal individual (the Reference Person), namely dose constraints for planned exposure situations and reference levels for emergency and existing exposure situations. Options resulting in doses greater in magnitude than such restrictions should be rejected at the planning stage. Importantly, these restrictions on doses are applied prospectively, as with optimisation as a whole. If, following the implementation of an optimised protection strategy, it is subsequently shown that the value of the constraint or reference level is exceeded, the reasons should be investigated but this fact alone should not necessarily prompt regulatory action. The Commission expects that this emphasis on a common approach to radiological protection in all exposure situations will aid application of the Commission's Recommendations in the various circumstances of radiation exposure.

(q) The relevant national authorities will often play a major role in selecting values for dose constraints and reference levels. Guidance on the selection process is provided in the revised Recommendations. This guidance takes account of numerical recommendations made previously by the Commission.

(r) Planned exposure situations encompass sources and situations that have been appropriately managed within the Commission's previous Recommendations for

practices. Protection during the medical uses of radiation is also included in this type of exposure situation. The process of planning protection in planned exposure situations should include consideration of deviations from normal operating procedures including accidents and malicious events. Exposures arising in such circumstances are referred to by the Commission as potential exposures. Potential exposures are not planned but they can be anticipated. The designer and the user of a source must therefore take actions to reduce the likelihood of a potential exposure happening, such as assessing the probability of an event and introducing engineering safeguards commensurate to this probability. Recommendations for planned exposure situations are substantially unchanged from these provided in *Publication 60* and subsequent publications. The dose limits for occupational and public exposures for practices are retained for application to regulated sources in planned exposure situations.

(s) Radiological protection in medicine includes the protection not only of patients but also of individuals exposed to radiation whilst caring for or comforting patients, and volunteers involved in biomedical research. The protection of all of these groups requires special consideration. The Commission's Recommendations for radiological protection and safety in medicine are given in *Publication 73* (ICRP 1996a) which has been further elaborated in a series of publications. The recommendations, guidance and advice in these publications remain valid and are summarised in the present Recommendations and in *Publication 105* (ICRP, 2007b) which was drafted by ICRP Committee 3 to support these Recommendations.

(t) Emphasis on optimisation using reference levels in emergency and existing exposure situations focuses attention on the residual level of dose remaining after implementation of protection strategies. This residual dose should be below the reference level, which represents the total residual dose as a result of an emergency, or in an existing situation, that the regulator would plan not to exceed. These exposure situations often involve multiple exposure pathways which means that protection strategies involving a number of different protective actions will have to be considered. The process of optimisation will however continue to use the dose averted by specific countermeasures as an important input into the development of optimised strategies.

(u) Emergency exposure situations include consideration of emergency preparedness and emergency response. Emergency preparedness should include planning for the implementation of optimised protection strategies which have the purpose of reducing exposures, should the emergency occur, to below the selected value of the reference level. During emergency response, the reference level would act as a benchmark for evaluating the effectiveness of protective actions and as one input into the need for establishing further actions.

(v) Existing exposure situations include naturally occurring exposures as well as exposures from past events and accidents, and practices conducted outside the Commission's Recommendations. In this type of situation, protection strategies will often be implemented in an interactive, progressive manner over a number of years. Indoor radon in dwellings and workplaces is an important existing exposure situation and is one where the Commission made specific recommendations in 1994 in

Publication 65 (ICRP 1993b). Since then several epidemiological studies have confirmed the health risk from radon exposure and have generally provided support for the Commission's Recommendations on protection against radon. Consistent with its approach to radiological protection in the revised Recommendations, the Commission now recommends that national authorities should set national reference levels as an aid to optimisation of protection against radon exposures. For the sake of continuity and practicability, the Commission retains the upper value of 10 mSv (effective dose, converted by convention from 600 Bq m^{-3} Rn-222 in dwellings) for the annual dose reference level, as given in *Publication 65*. The Commission reaffirms that radon exposure at work at levels above the national reference level should be considered part of occupational exposure whereas exposures at levels below should not. Nevertheless, optimisation is a requirement below the national reference level.

(w) The revised Recommendations acknowledge the importance of protecting the environment. The Commission has previously concerned itself with mankind's environment only with regard to the transfer of radionuclides through it, mainly in the context of planned exposure situations. In such situations, the Commission continues to believe that the standards of environmental control needed to protect the general public would ensure that other species are not placed at risk. To provide a sound framework for environmental protection in all exposure situations, the Commission proposes the use of Reference Animals and Plants. In order to establish a basis for acceptability, additional doses calculated to these reference organisms could be compared with doses known to have specific biological effects and with dose rates normally experienced in the natural environment. The Commission, however, does not propose to set any form of 'dose limits' for environmental protection.

(x) The Commission anticipates that although the revised Recommendations do not contain any fundamental changes to the radiological protection policy, these Recommendations will help to clarify application of the system of protection in the plethora of exposure situations encountered, thereby further improving the already high standards of protection.

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GLOSSARY

α/β ratio

A measure of the curvature of the cell survival curve and a measure of the sensitivity of a tissue or tumour to dose fractionation. The dose at which the linear and quadratic components of cell killing are equal.

Absorbed dose, D

The fundamental dose quantity given by

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

where $\mathrm{d}\bar{\varepsilon}$ is the mean energy imparted to matter of mass $\mathrm{d}m$ by ionising radiation. The SI unit for absorbed dose is joule per kilogram (J kg^{-1}) and its special name is gray (Gy).

Active (red) bone marrow

The organ system bone marrow contains the cell systems for the formation of blood cells starting from the pluripotent haematopietic stem cells to the mature blood cells.

Activity, A

The expectation value of the number of nuclear transformations occurring in a given quantity of material per unit time. The SI unit of activity is per second (s^{-1}) and its special name is becquerel (Bq).

Activity Median Aerodynamic Diameter (AMAD)

The value of aerodynamic diameter such that 50% of the airborne activity in a specified aerosol is associated with particles greater than the AMAD. Used when deposition depends principally on inertial impaction and sedimentation, typically when the AMAD is greater than about 0.5 μm.

Adaptive response

A post-irradiation cellular response which, typically, serves to increase the resistance of the cell to a subsequent radiation exposure.

Ambient dose equivalent, $H^*(10)$

The dose equivalent at a point in a radiation field that would be produced by the corresponding expanded and aligned field in the ICRU sphere at a depth of 10 mm on the radius vector opposing the direction of the aligned field. The unit of ambient dose equivalent is joule per kilogram (J kg^{-1}) and its special name is sievert (Sv).

Annual intake, *AI*

The amount of a specified radionuclide entering the human body by ingestion or inhalation within one year.

Apoptosis

An active biochemical process of programmed cell death following radiation or other insults.

Averted dose

The dose prevented or avoided by the application of a protective measure or set of protective measures, i.e., the difference between the projected dose if the protective measure(s) had not been applied and the expected residual dose.

Baseline rates

The annual disease incidence observed in a population in the absence of exposure to the agent under study.

Becquerel (Bq)

The special name for the SI unit of activity, 1 Bq = 1 s^{-1} ($\approx$2.7 10^{-11} Ci).

Bioassay

Any procedure used to determine the nature, activity, location, or retention of radionuclides in the body by in vivo measurement or by in vitro analysis of material excreted or otherwise removed from the body.

Biological half-life

The time required, in the absence of further input, for a biological system or compartment to eliminate, by biological processes, half the amount of a substance (e.g., radioactive material) that has entered it.

Brachytherapy

Radiation treatment of a patient using sealed or unsealed sources of radiation placed within the patient's body.

Bystander effect

A response in unirradiated cells that is triggered by signals received from irradiated neighbouring cells.

Categories of exposure

The Commission distinguishes between three categories of radiation exposure: occupational, public, and medical exposures of patients.

Collective dose

See 'Collective effective dose'.

Collective effective dose, S

The collective effective dose due to individual effective dose values between E_1 and E_2 from a specified source within a specified time period ΔT is defined as:

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

It can be approximated as $S = \Sigma_i E_i N_i$ where E_i is the average effective dose for a subgroup i, and N_i is the number of individuals in this subgroup. The time period and number of individuals over which the effective doses are summed should always be specified. The unit of the collective effective dose is joule per kilogram (J kg^{-1}) and its special name is man sievert (man Sv). The number of individuals experiencing an effective dose in the range E_1 to E_2, $N(E_1, E_2, \Delta T)$ is

$$N(E_1, E_2, \Delta T) = \int_{E_1}^{E_2} \left[\frac{\mathrm{d}N}{\mathrm{d}E}\right]_{\Delta T} \mathrm{d}E$$

and the average value of effective dose $\overline{E}(E_1, E_2, \Delta T)$ in the interval of individual doses between E_1 and E_2 for the time period ΔT is:

$$\overline{E}(E_1, E_2, \Delta T) = \frac{1}{N(E_1, E_2, \Delta T)} \int_{E_1}^{E_2} E \left[\frac{\mathrm{d}N}{\mathrm{d}E}\right]_{\Delta T} \mathrm{d}E$$

Committed effective dose, $E(\tau)$

The sum of the products of the committed organ or tissue equivalent doses and the appropriate tissue weighting factors (w_T), where τ is the integration time in years following the intake. The commitment period is taken to be 50 years for adults, and to age 70 years for children.

Committed equivalent dose, $H_T(\tau)$

The time integral of the equivalent dose rate in a particular tissue or organ that will be received by an individual following intake of radioactive material into the body by a Reference Person, where τ is the integration time in years.

Confidence limits

An interval giving the lowest and highest estimate of a parameter that is statistically compatible with the data. For a 95% confidence interval, there is a 95% chance that the interval contains the parameter.

Controlled area

A defined area in which specific protection measures and safety provisions are, or could be, required for controlling normal exposures or preventing the spread of contamination during normal working conditions, and preventing or limiting the extent of potential exposures. A controlled area is often within a supervised area, but need not be.

DD

See 'Doubling dose'.

Derived air concentration (DAC)

This equals the annual limit on intake, ALI, (of a radionuclide) divided by the volume of air inhaled by a Reference Person in a working year (i.e., $2.2\ 10^3\ m^3$). The unit of DAC is Bq m^{-3}.

Designated area

An area that is either 'controlled' or 'supervised'.

Deterministic effect

Injury in populations of cells, characterised by a threshold dose and an increase in the severity of the reaction as the dose is increased further. Also termed tissue reaction. In some cases, deterministic effects are modifiable by post-irradiation procedures including biological response modifiers.

Detriment

The total harm to health experienced by an exposed group and its descendants as a result of the group's exposure to a radiation source. Detriment is a multidimensional concept. Its principal components are the stochastic quantities: probability of attributable fatal cancer, weighted probability of attributable non-fatal cancer, weighted probability of severe heritable effects, and length of life lost if the harm occurs.

Detriment-adjusted risk

The probability of the occurrence of a stochastic effect, modified to allow for the different components of the detriment in order to express the severity of the consequence(s).

Diagnostic reference level

Used in medical imaging with ioning radiation to indicate whether, in routine conditions, the patient dose or administered activity (amount of radioactive material) from a specified procedure is unusually high or low for that procedure.

Directional dose equivalent, $H'(d, \Omega)$

The dose equivalent at a point in a radiation field that would be produced by the corresponding expanded field in the ICRU sphere at a depth, d, on a radius in a specified direction, Ω. The unit of directional dose equivalent is joule per kilogram (J kg^{-1}) and its special name is sievert (Sv).

DMF

Dose modifying factor: the ratio of doses with and without modifying agents, causing the same level of biological effect.

DNA damage signalling

Interacting biochemical processes which recognise and respond to DNA damage in cells, e.g., by causing the arrest of the reproductive cell cycle.

Differentiation

The process whereby stem cells enter a pathway of proliferation during which daughter cells acquire specialised functions.

Dose and dose-rate effectiveness factor (DDREF)

A judged factor that generalises the usually lower biological effectiveness (per unit of dose) of radiation exposures at low doses and low dose rates as compared with exposures at high doses and high dose rates.

Dose coefficient

Used as a synonym for dose per unit intake of a radioactive substance, but sometimes also used to describe other coefficients linking quantities or concentrations of activity to doses or dose rates, such as the external dose rate at a specified distance above a surface with a deposit of a specified activity per unit area of a specified radionuclide.

Dose commitment, E_c

A calculational tool, defined as the infinite time integral of the per caput dose rate $\dot{E}$ due to a specified event, such as a year of a planned activity causing discharges. In the case of indefinite discharges at a constant rate, the maximum annual per caput dose rate $\dot{E}$ 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 activity causing discharges is continued only over a time period, τ, the maximum future annual per caput dose will be equal to the corresponding truncated dose commitment, defined as

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

Dose constraint

A prospective and source-related restriction on the individual dose from a source, which provides a basic level of protection for the most highly exposed individuals from a source, and serves as an upper bound on the dose in optimisation of protection for that source. For occupational exposures, the dose constraint is a value of individual dose used to limit the range of options considered in the process of optimisation. For public exposure, the dose constraint is an upper bound on the annual doses that members of the public should receive from the planned operation of any controlled source.

Dose equivalent, H

The product of D and Q at a point in tissue, where D is the absorbed dose and Q is the quality factor for the specific radiation at this point, thus:

$$H = DQ$$

The unit of dose equivalent is joule per kilogram (J kg^{-1}), and its special name is sievert (Sv).

Dose limit

The value of the effective dose or the equivalent dose to individuals from planned exposure situations that shall not be exceeded.

Dose of record, $H_p(10)$

The effective dose of a worker assessed by the sum of the measured personal dose equivalent $H_p(10)$ and the committed effective dose retrospectively determined for the Reference Person using results of individual monitoring of the worker and ICRP reference biokinetic and dosimetric computational models. Dose of record may be assessed with site-specific parameters of exposure, such as the type of materials and AMAD, but the parameters of the Reference Person shall be fixed as defined by the Commission. Dose of record is assigned to the worker for purposes of recording, reporting and retrospective demonstration of compliance with regulatory dose limits.

Dose-threshold hypothesis

A given dose above background, below which it is hypothesised that the risk of excess cancer and/or heritable disease is zero. (See also Threshold dose for tissue reactions).

Doubling dose (DD)

The dose of radiation (Gy) that is required to produce as many heritable mutations as those arising spontaneously in a generation.

DS02

Dosimetry System 2002, a system for estimating gamma and neutron exposure under a large variety of situations and which allows the calculation of absorbed dose to specific organs for members of the Life Span Study. DS02 improved on the DS86 dose system.

DS86

Dosimetry System 1986, a system for estimating gamma and neutron exposure under a large variety of situations and which then allowed the calculation of absorbed dose to specific organs for members of the Life Span Study.

Effective dose, E

The tissue-weighted sum of the equivalent doses in all specified tissues and organs of the body, given by the expression:

$$E = \sum_{T} w_T \sum_{R} w_R D_{T,R} \quad \text{or} \quad E = \sum_{T} w_T H_T$$

where H_T or w_R $D_{T,R}$ is the equivalent dose in a tissue or organ, T, and w_T is the tissue weighting factor. The unit for the effective dose is the same as for absorbed dose, J kg^{-1}, and its special name is sievert (Sv).

ELR

See 'Lifetime risk estimates'.

Emergency

A non-routine situation or event that necessitates prompt action primarily to mitigate a hazard or adverse consequences for human health and safety, quality of life, property or the environment. This includes situations for which prompt action is warranted to mitigate the effects of a perceived hazard.

Emergency exposure situation

An unexpected situation that occurs during the operation of a practice, requiring urgent action. Emergency exposure situations may arise from practices.

Employer

An organisation, corporation, partnership, firm, association, trust, estate, public or private institution, group, political or administrative entity, or other persons designated in accordance with national legislation, with recognised responsibility, commitment, and duties towards a worker in her or his employment by virtue of a mutually agreed relationship. A self-employed person is regarded as being both an employer and a worker.

Equivalent dose, H_T

The dose in a tissue or organ T given by:

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

where $D_{T,R}$ is the mean absorbed dose from radiation R in a tissue or organ T, and w_R is the radiation weighting factor. Since w_R is dimensionless, the unit for the equivalent dose is the same as for absorbed dose, J kg^{-1}, and its special name is sievert (Sv).

Excess absolute risk

The rate of disease incidence or mortality in an exposed population minus the corresponding disease rate in an unexposed population. The excess absolute risk is often expressed as the additive excess rate per Gy or per Sv.

Excess relative risk

The rate of disease in an exposed population divided by the rate of disease in an unexposed population, minus 1.0. This is often expressed as the excess relative risk per Gy or per Sv.

Exclusion

The deliberate exclusion of a particular category of exposure from the scope of an instrument of regulatory control.

Exemption

The determination by a regulatory body that a source or practice activity involving radiation need not be subject to some or all aspects of regulatory control.

Existing exposure situation

A situation that already exists when a decision on control has to be taken, including natural background radiation and residues from past practices that were operated outside the Commission's recommendations.

Exposed individuals

The Commission distinguishes between three categories of exposed individuals: workers (informed individuals), the public (general individuals), and patients, including their comforters and carers.

Fluence (particle fluence), Φ

The quotient of $\mathrm{d}N$ by $\mathrm{d}a$, where $\mathrm{d}N$ is the number of particles incident upon a small sphere of cross-sectional area $\mathrm{d}a$, thus:

$$\Phi = \frac{\mathrm{d}N}{\mathrm{d}a}$$

FSU

Functional subunits of tissues, e.g., nephrons in kidney, alveoli in lung.

Gray (Gy)

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

Growth factors

Molecules that act to control cell reproduction and proliferation/differentiation of a population of cells.

Incidence (incidence rate)

The rate of occurrence of a disease in a population within a specified period of time, often expressed as the number of cases of a disease arising per 100,000 individuals per year (or per 100,000 person-years).

Induced genomic instability

The induction of an altered cellular state characterised by a persistent increase over many generations in the spontaneous rate of mutation or other genome-related changes.

Intake, I

Activity that enters the body through the respiratory tract or the gastrointestinal tract or the skin.

– Acute intake

A single intake by inhalation or ingestion, taken to occur instantaneously.

– Chronic intake

An intake over a specified period of time.

Justification

The process of determining whether either (1) a planned activity involving radiation is, overall, beneficial, i.e. whether the benefits to individuals and to society from introducing or continuing the activity outweigh the harm (including radiation detriment) resulting from the activity; or (2) a proposed remedial action in an emergency or existing exposure situation is likely, overall, to be beneficial, i.e., whether the benefits to individuals and to society (including the reduction in radiation detriment) from introducing or continuing the remedial action outweigh its cost and any harm or damage it causes.

Kerma, K

The quotient of the sum of the kinetic energies, $\mathrm{d}E_{\mathrm{tr}}$, of all charged particles liberated by uncharged particles in a mass $\mathrm{d}m$ of material, and the mass $\mathrm{d}m$ of that material.

$$K = \frac{\mathrm{d}E_{\mathrm{tr}}}{\mathrm{d}m}$$

Kerma is defined as a non-stochastic quantity and $\mathrm{d}E_{\mathrm{tr}}$ is the expectation value of the sum of the kinetic energies. The unit for kerma is joule per kilogram (J kg^{-1}) and its special name is gray (Gy).

LAR

See 'Lifetime risk estimates'.

LD50

Dose that is lethal for half of the exposed individuals.

LET

See 'Linear energy transfer'.

Licensee

The holder of a current legal document issued by the regulatory body granting authorisation to perform specified activities related to an installation or activity.

Life Span Study (LSS)

The long-term cohort study of health effects in the Japanese atomic bomb survivors in Hiroshima and Nagasaki.

Lifetime risk estimates

Several types of lifetime risk estimates can be used to calculate the risk, over a lifetime, that an individual will develop, or die from, a specific disease caused by an exposure: 1) the excess lifetime risk (ELR) which is the difference between the proportion of people who develop or die from the disease in an exposed population and the corresponding proportion in a similar population without the exposure; 2) the risk of exposure-induced death (REID) which is defined as the difference in a cause-specific death rate for exposed and unexposed populations of a given sex and a given age at exposure, as an additional cause of death introduced into a population; 3) loss of life expectancy (LLE) which describes the decrease in life expectancy due to the exposure of interest; and 4) lifetime attributable risk (LAR) which is an approximation of the REID and describes excess deaths (or disease cases) over a follow-up period with population background rates determined by the experience of unexposed individuals. The LAR was used in this report to estimate lifetime risks.

Linear dose response

A statistical model that expresses the risk of an effect (e.g., disease or abnormality) as being proportional to dose.

Linear energy transfer (L or LET)

The average linear rate of energy loss of charged particle radiation in a medium, i.e., the radiation energy lost per unit length of path through a material. That is, the quotient of dE by dl where dE is the mean energy lost by a charged particle owing to collisions with electrons in traversing a distance dl in matter.

$$L = \frac{\mathrm{d}E}{\mathrm{d}l}$$

The unit of L is J m^{-1}, often given in keV μm^{-1}.

Linear-non-threshold (LNT) model

A dose-response model which is based on the assumption that, in the low dose range, radiation doses greater than zero will increase the risk of excess cancer and/or heritable disease in a simple proportionate manner.

Linear-quadratic dose response

A statistical model that expresses the risk of an effect (e.g., disease, death, or abnormality) as the sum of two components, one proportional to dose (linear term) and the other one proportional to the square of dose (quadratic term).

LLE
See 'Lifetime risk estimates'.

MC
See 'Mutation component'.

Mean absorbed dose in a tissue or organ (T), D_T
The absorbed dose D_T, averaged over the tissue or organ T, which is given by

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

where ε_T is the mean total energy imparted in a tissue or organ T, and m_T is the mass of that tissue or organ.

Medical exposure
Exposure incurred by patients as part of their own medical or dental diagnosis or treatment; by persons, other than those occupationally exposed, knowingly, while voluntarily helping in the support and comfort of patients; and by volunteers in a programme of biomedical research involving their exposure.

Mendelian diseases
Heritable diseases attributable to single-gene mutations.

Multifactorial diseases
Diseases that are attributable to multiple genetic and environmental factors.

Multistage tumorigenesis
The stepwise acquisition of cellular properties that can lead to the development of tumour from a single (target) cell.

Mutation component (MC)
A quantity that provides a measure of the relative change in disease frequency per unit relative change in mutation rate, i.e., a measure of responsiveness; MC values differ for different classes of heritable disease.

Nominal risk coefficient
Sex-averaged and age-at-exposure-averaged lifetime risk estimates for a representative population.

Non-cancer diseases
Somatic diseases other than cancer, e.g., cardiovascular disease and cataracts.

NORM (naturally occurring radioactive material)
Radioactive material containing no significant amounts of radionuclides other than naturally occurring radionuclides. Material in which the activity

concentrations of the naturally occurring radionuclides have been changed by some process are included in NORM.

Occupational exposure

This refers to all exposure incurred by workers in the course of their work, with the exception of

1) excluded exposures and exposures from exempt activities involving radiation or exempt sources; 2) any medical exposure; and 3) the normal local natural background radiation.

Operating management

The person or group of persons that directs, controls, and assesses an organisation at the highest level. Many different terms are used, including, e.g., chief executive officer (CEO), director general (DG), managing director (MD), and executive group.

Operational quantities

Quantities used in practical applications for monitoring and investigating situations involving external exposure. They are defined for measurements and assessment of doses in the body. In internal dosimetry, no operational dose quantities have been defined which directly provide an assessment of equivalent or effective dose. Different methods are applied to assess the equivalent or effective dose due to radionuclides in the human body. They are mostly based on various activity measurements and the application of biokinetic models (computational models).

Optimisation of protection (and safety)

The process of determining what level of protection and safety makes exposures, and the probability and magnitude of potential exposures, as low as reasonably achievable, economic and societal factors being taken into account.

Particle fluence, Φ

See 'Fluence'.

Personal dose equivalent, $H_{\mathrm{p}}(d)$

An operational quantity: the dose equivalent in soft tissue (commonly interpreted as the 'ICRU sphere') at an appropriate depth, d, below a specified point on the human body. The unit of personal dose equivalent is joule per kilogram (J kg^{-1}) and its special name is sievert (Sv). The specified point is usually given by the position where the individual's dosimeter is worn.

Planned exposure situations

Everyday situations involving the planned operation of sources including decommissioning, disposal of radioactive waste and rehabilitation of the previously occupied land. Practices in operation are planned exposure situations.

Pooled analysis

An analysis of epidemiological data from several studies based on original data from those studies that are analysed in parallel.

Potential exposure

Exposure that is not expected to be delivered with certainty but that may result from an accident at a source or an event or sequence of events of a probabilistic nature, including equipment failures and operating errors.

PRCF (potential recoverability correction factor)

A set of factors that take account of knowledge that different classes of germ line mutation will show different degrees of recoverability in live-born offspring, i.e., through differing capacities to allow completion of embryonic/fetal development.

Principles of protection

A set of principles that apply equally to all controllable exposure situations: the principle of justification, the principle of optimisation of protection, and the principle of application of limits on maximum doses in planned situations.

Progenitor cell

Undifferentiated cell capable of limited proliferation.

Projected dose

The dose that would be expected to be incurred if no protective measure(s) – were to be taken.

Protection quantities

Dose quantities that the Commission has developed for radiological protection that allow quantification of the extent of exposure of the human body to ionising radiation from both whole and partial body external irradiation and from intakes of radionuclides.

Public exposure

Exposure incurred by members of the public from radiation sources, excluding any occupational or medical exposure and the normal local natural background radiation.

Quality factor, $Q(L)$

The factor characterising the biological effectiveness of a radiation, based on the ionisation density along the tracks of charged particles in tissue. Q is defined as a function of the unrestricted linear energy transfer, L_∞ (often denoted as L or LET), of charged particles in water:

$$Q(L) = \begin{cases} 1 & L < 10 \text{ keV}/\mu\text{m} \\ 0.32L - 2.2 & 10 \leqslant L \leqslant 100 \text{ keV}/\mu\text{m} \\ 300/\sqrt{L} & L > 100 \text{ keV}/\mu\text{m} \end{cases}$$

Q has been superseded by the radiation weighting factor in the definition of equivalent dose, but it is still used in calculating the operational dose equivalent quantities used in monitoring.

Radiation detriment

A concept used to quantify the harmful health effects of radiation exposure in different parts of the body. It is defined by the Commission as a function of several factors, including incidence of radiation-related cancer or heritable effects, lethality of these conditions, quality of life, and years of life lost owing to these conditions.

Radiation weighting factor, w_R

A dimensionless factor by which the organ or tissue absorbed dose is multiplied to reflect the higher biological effectiveness of high-LET radiations compared with low-LET radiations. It is used to derive the equivalent dose from the absorbed dose averaged over a tissue or organ.

Radioactive material

Material designated in national law or by a regulatory body as being subject to regulatory control because of its radioactivity, often taking account of both activity and activity concentration.

Radiological attack

The use of radioactive or nuclear materials for malicious purposes, such as blackmail, murder, sabotage, or terrorism.

Random error

Errors that vary in a non-reproducible way. These errors can be treated statistically by use of the laws of probability.

RBE

See 'Relative biological effectiveness'.

Reference Animals and Plants

A Reference Animal or Plant is a hypothetical entity, with the assumed basic characteristics of a specific type of animal or plant, as described to the generality of the taxonomic level of Family, with defined anatomical, physiological, and life-history properties, that can be used for the purposes of relating exposure to dose, and dose to effects, for that type of living organism.

Reference Male and Reference Female (Reference Individual)

An idealised male or female with characteristics defined by the Commission for the purpose of radiological protection, and with the anatomical and physiological characteristics defined in the report of the ICRP Task Group on Reference Man (*Publication 89*, ICRP 2002).

Reference Person

An idealised person for whom the organ or tissue equivalent doses are calculated by averaging the corresponding doses of the Reference Male and Reference Female. The equivalent doses of the Reference Person are used for the calculation of the effective dose by multiplying these doses by the corresponding tissue weighting factors.

Reference phantom

Voxel phantoms for the human body (male and female voxel phantoms based on medical imaging data) with the anatomical and physiological characteristics defined in the report of the ICRP Task Group on Reference Man (*Publication 89*, ICRP 2002).

Reference value

The value of a parameter recommended by the Commission for use in a biokinetic model in the absence of more specific information, i.e., the exact value used to calculate the dose coefficients presented in the report. Reference values may be specified to a greater degree of precision than that which would be chosen to reflect the uncertainty with which an experimental value is known, in order to avoid the accumulation of rounding errors in a calculation.

Reference level

In emergency or existing controllable exposure situations, this represents the level of dose or risk, above which it is judged to be inappropriate to plan to allow exposures to occur, and below which optimisation of protection should be implemented. The chosen value for a reference level will depend upon the prevailing circumstances of the exposure under consideration.

Relative biological effectiveness (RBE)

The ratio of a dose of a low-LET reference radiation to a dose of the radiation considered that gives an identical biological effect. RBE values vary with the dose, dose rate, and biological endpoint considered. In radiological protection, the RBE for stochastic effects at low doses (RBE_M) is of particular interest.

Relative life lost

The ratio of the proportion of observed years of life lost among people dying of a disease in an exposed population and the corresponding proportion in a similar population without the exposure.

REID

See 'Lifetime risk estimates'.

Relative survival

The ratio of the proportion of cancer patients who survive for a specified number of years (e.g., 5 years) following diagnosis to the corresponding proportion in a comparable set of cancer-free individuals.

Representative Person

An individual receiving a dose that is representative of the more highly exposed individuals in the population (see *Publication 101*, ICRP 2006a). This term is the equivalent of, and replaces, 'average member of the critical group' described in previous ICRP Recommendations.

Residual dose

The dose expected to be incurred after protective measure(s) have beenfully implemented (or a decision has been taken not to implement any protective measures).

Risk constraint

A prospective and source-related restriction on the individual risk (in the sense of probability of detriment due to a potential exposure) from a source, which provides a basic level of protection for the individuals most at risk from a source and serves as an upper bound on the individual risk in optimisation of protection for that source. This risk is a function of the probability of an unintended event causing a dose, and the probability of detriment due to that dose. Risk constraints correspond to dose constraints but refer to potential exposures.

Safety

The achievement of proper operating conditions, prevention of accidents, or mitigation of accident consequences.

Security

The prevention and detection of, and response to, theft, sabotage, unauthorised access, illegal transfer, or other malicious acts involving nuclear material, other radioactive substances, or their associated installations.

Sensitivity analysis

This aims to quantify how the results from a model depend upon the different variables included in it.

Sievert (Sv)

The special name for the SI unit of equivalent dose, effective dose, and operational dose quantities. The unit is joule per kilogram (J kg^{-1}).

Source

An entity for which radiological protection can be optimised as an integral whole, such as the x-ray equipment in a hospital, or the releases of radioactive materials from an installation. Sources of radiation, such as radiation generators and sealed radioactive materials, and, more generally, the cause of exposure to radiation or to radionuclides.

Source region, S_i

An anatomical region within the reference phantom body which contains the radionuclide following its intake. The region may be an organ, a tissue, the contents of the gastrointestinal tract or urinary bladder, or the surfaces of tissues as in the skeleton, the alimentary tract, and the respiratory tract.

Specific absorbed fraction

The fraction of energy of that emitted as a specified radiation type in a source region, S, that is absorbed in 1 kg of a target tissue, T.

Statistical power

The probability that an epidemiological study will detect a given level of elevated risk with a specified degree of confidence.

Stem cell

Non-differentiated, pluripotent cell, capable of unlimited cell division.

Stochastic effects of radiation

Malignant disease and heritable effects for which the probability of an effect occurring, but not its severity, is regarded as a function of dose without threshold.

Supervised area

A defined area not designated as a controlled area but for which occupational exposure conditions are kept under review, even though no specific protection measures or safety provisions are normally needed.

Systematic error

Errors that are reproducible and tend to bias a result in one direction. Their causes can be assigned, at least in principle, and they can have constant and variable components. Generally these errors cannot be treated statistically.

Target region, T_i

Anatomical region within the body (reference phantom) in which radiation is absorbed. The region may be an organ or a specified tissue as in the gastrointestinal tract, urinary bladder, skeleton, and respiratory tract.

Threshold dose for tissue reactions
: Dose estimated to result in only 1% incidence of tissue reactions.

Tissue reaction
: See 'Deterministic effect'.

Tissue weighting factor, w_T
: The factor by which the equivalent dose in a tissue or organ T is weighted to represent the relative contribution of that tissue or organ to the total health detriment resulting from uniform irradiation of the body (ICRP 1991b). It is weighted such that:

$$\sum_T w_T = 1$$

Track structure
: Spatial patterns of energy deposition in matter along the track from the passage of ionising radiation.

Transport of risk (also called transfer of risk)
: Taking a risk coefficient estimated for one population and applying it to another population with different characteristics.

Voxel phantom
: Computational anthropomorphic phantom based on medical tomographic images where the anatomy is described by small three-dimensional volume elements (voxels) specifying the density and the atomic composition of the various organs and tissues of the human body.

Worker
: Any person who is employed, whether full time, part time or temporarily, by an employer, and who has recognised rights and duties in relation to occupational radiological protection.

References for the Glossary

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1. INTRODUCTION

(1) This chapter deals with the history of the Commission and its Recommendations. It sets out the aims and form of this report and indicates why the Commission concerns itself only with protection against ionising radiation.

1.1. The history of the Commission

(2) The International Commission on Radiological Protection, hereafter called the Commission, was established in 1928 by the International Congress of Radiology, with the name of the International X-Ray and Radium Protection Committee (IXRPC), following a decision by the Second International Congress of Radiology. In 1950 it was restructured and renamed as now.

(3) The Commission is an independent charity, i.e., a non-profit-making organisation. The Commission works closely with its sister body, the International Commission on Radiation Units and Measurements (ICRU), and has official relationships with the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), the World Health Organization (WHO), and the International Atomic Energy Agency (IAEA). It also has important relationships with the International Labour Organization (ILO), the United Nations Environment Programme (UNEP), and other United Nations bodies. Other organisations with which it works include the Commission of the European Communities ('European Commission', EC), the Nuclear Energy Agency of the Organization for Economic Co-operation and Development (OECD/NEA), the International Organization for Standardization (ISO), and the International Electrotechnical Commission (IEC). The Commission also maintains contact with the professional radiological community through its strong links with the International Radiation Protection Association (IRPA). The Commission also takes account of progress reported by national organisations.

1.2. The development of the Commission's Recommendations

(4) The first general Recommendations of the Commission were issued in 1928 and concerned the protection of the medical profession through the restriction of working hours with medical sources (IXRPC, 1928). This restriction is now estimated to correspond to an individual dose of about 1000 millisievert (mSv) per year. The early Recommendations were concerned with avoiding threshold effects, initially in a qualitative manner. A system of measurement of doses was needed before protection could be quantified and dose limits could be defined. In 1934, Recommendations were made implying the concept of a safe threshold about ten times the present annual occupational dose limit (IXRPC, 1934). The tolerance idea continued and, in 1951, the Commission proposed a limit that can now be estimated to be around 3 mSv per week for low-LET radiation (ICRP, 1951). By 1954 the support for a threshold was diminished because of the epidemiological evidence emerging of excess malignant disease amongst American radiologists and the first indication of excess leukaemia in the Japanese A-bomb survivors (ICRP, 1955).

(5) The development of both the military and industrial uses of nuclear energy led the Commission in the early 1950s to introduce recommendations for the protection of the public. In the Commission's 1956 Recommendations (ICRP, 1957), limits on weekly and accumulated doses were set that corresponded to annual dose limits of 50 mSv for workers and 5 mSv for the public. Recognising the possibility of what are now termed stochastic effects, and the impossibility of demonstrating the existence or non-existence of a threshold for these types of effects, the Commission's 1954 Recommendations advised '*that every effort* [should] *be made to reduce exposures to all types of ionising radiation to the lowest possible level*' (ICRP, 1955). This was successively formulated as the recommendation to maintain exposure 'as low as practicable' (ICRP, 1959), 'as low as readily achievable' (ICRP, 1966), and later on 'as low as reasonably achievable, economic and social considerations being taken into account' (ICRP, 1973).

(6) The Commission's first report in the current series, numbered *Publication 1* (1959), contained the Recommendations approved in 1958. Subsequent general Recommendations have appeared as *Publication 6* (1964), *Publication 9* (1966), *Publication 26* (1977), and *Publication 60* (1991b). These general Recommendations have been supported by many other Publications providing advice on more specialised topics.

(7) In *Publication 26*, the Commission first quantified the risks of stochastic effects of radiation and proposed a System of Dose Limitation (ICRP, 1977) with its three principles of justification, optimisation of protection, and individual dose limitation. In 1990, the Commission largely revised the Recommendations partly because of upward revisions of the estimates of risk from exposure to radiation, and partly to extend its philosophy to a System of Radiological Protection from the system of dose limitation (ICRP, 1991b). The principles of justification, optimisation and individual dose limitation remained, and a distinction between 'practices' and 'interventions' was introduced to take into account the differences in the various types of exposure situations. Moreover, more emphasis was put on the optimisation of protection with constraints so as to limit the inequity that is likely to result from inherent economic and societal judgements.

(8) The annual dose limit of 50 mSv for workers[1] set in 1956, was retained until 1990, when it was further reduced to 20 mSv per year on average based on the revision of the risk for stochastic effects estimated from the life-span study of the Hiroshima–Nagasaki atomic bomb survivors (ICRP, 1991b). The annual dose limit of 5 mSv for members of the public was reduced to 1 mSv per year on average in the Commission's 'Paris statement' (ICRP, 1985b) and in *Publication 60* (ICRP, 1991b) the dose limit was given as 1 mSv in a year with the possibility of averaging over 5 years 'in special circumstances'.

(9) Since *Publication 60*, there has been a series of publications that have provided additional guidance for the control of exposures from radiation sources (see the list of all references). When the 1990 Recommendations are included, these reports

[1] Some terms and units used in older reports have been converted to current terminology for consistency.

specify some 30 different numerical values for restrictions on individual dose for differing circumstances. Furthermore, these numerical values are justified in many different ways (ICRP, 2006b). In addition the Commission began to develop policy guidance for protection of the environment in *Publication 91* (ICRP, 2003b).

(10) The Commission has now decided to adopt a revised set of Recommendations while at the same time maintaining stability with the previous Recommendations.

(11) The Commission's extensive review of the vast body of literature on the health effects of ionising radiation has not indicated that any fundamental changes are needed to the system of radiological protection. There is, therefore, more continuity than change in these Recommendations; some recommendations are to remain because they work and are clear; others have been updated because understanding has evolved; some items have been added because there has been a void; and some concepts are better explained because more guidance is needed.

(12) The present Recommendations consolidate and add to previous Recommendations issued in various ICRP publications. The existing numerical recommendations in the policy guidance given since 1991 remain valid unless otherwise stated. Thus, these recommendations should not be interpreted as suggesting major changes to radiological protection regulations that are appropriately based on its previous Recommendations in *Publication 60* and subsequent policy guidance. The Recommendations reiterate and strengthen the importance of optimisation in radiological protection and extend the successful experience in the implementation of this requirement for practices (now included in planned exposure situations) to other situations, i.e., emergency and existing exposure situations.

(13) The Commission plans to follow up these Recommendations with reports applying the process of optimisation in different situations.

(14) These consolidated Recommendations are supported by a series of supporting documents, which elaborate on important aspects of the Commission's policy and underpin the Recommendations:

- Low-dose extrapolation of radiation-related cancer risk (*Publication 99*, ICRP, 2005d).
- Biological and epidemiological information on health risks attributable to ionising radiation: A summary of judgements for the purposes of radiological protection of humans (Annex A to these Recommendations).
- Quantities used in radiological protection (Annex B to these Recommendations).
- Optimisation of radiological protection (in *Publication 101*, ICRP, 2006a, Part 2).
- Assessing dose to the Representative Person (in *Publication 101*, ICRP, 2006a, Part 1).
- A framework for assessing the impact of ionising radiation on the environment (*Publication 91*, ICRP, 2003b).
- In addition the Commission is providing guidance on the scope of radiological protection (*Publication 104*, ICRP 2007a), and on radiological protection in medical practice (*Publication 105*, ICRP 2007b).

(15) The principal objective of the Commission has been, and remains, the achievement of the radiological protection of human beings. It has, nevertheless, pre-

viously regarded the potential impact on other species, although it has not made any general statements about the protection of the environment as a whole. Indeed, in *Publication 60* (ICRP, 1991b) the Commission stated that, at that time, it concerned itself with mankind's environment only with regard to the transfer of radionuclides through the environment, because this directly affects the radiological protection of human beings. The Commission did, however, express the view that the standards of environmental control needed to protect humans to the degree currently thought desirable would ensure that other species are not put at risk.

(16) The Commission continues to believe that this is likely to be the case in general terms under *planned exposure situations* (see Section 5.2 for the definition of planned exposure situations), and that the human habitat has therefore been afforded a fairly high degree of protection. There are, however, other environments to consider, where the Commission's Recommendations for protection of humans have not been used or where humans are absent, and other exposure situations will arise where environmental consequences may need to be taken into account. The Commission is also aware of the needs of some national authorities to demonstrate, directly and explicitly, that the environment is being protected even under planned exposure situations. It therefore now believes that the development of a clearer framework is required in order to assess the relationships between exposure and dose, between dose and effect, and the consequences of such effects for non-human species, on a common scientific basis. This is discussed further in Chapter 8.

(17) The advice of the Commission is aimed principally at regulatory authorities, organisations, and individuals that have responsibility for radiological protection. The Commission's Recommendations have helped in the past to provide a consistent basis for national and regional regulatory standards, and the Commission has been concerned to maintain stability in its Recommendations. The Commission provides guidance on the fundamental principles on which appropriate radiological protection can be based. It does not aim to provide regulatory texts. Nevertheless, it believes that such texts should be developed from, and be broadly consistent with, its guidance.

(18) There is a close connection between the Commission's Recommendations and the International Basic Safety Standards for Protection against Ionizing Radiation and the Safety of Radiation Sources (usually simply called 'the BSS'), which are co-sponsored by the relevant international organisations within the UN family and issued by the IAEA. The governing body of the IAEA has decided that the BSS have to take the Commission's Recommendations into account. The BSS therefore have always followed the establishment of new Recommendations from the Commission; for example, the 1977 and the 1990 ICRP Recommendations were the basis for the revised International Basic Safety Standards published in 1982 and 1996, respectively.

(19) These Recommendations, 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. The International Commission on Non-Ionizing Radiation Protection, ICNIRP, provides recommendations concerning such sources (ICNIRP, 2004).

1.2.1. The evolution of dose quantities and their units

(20) The first dose unit, roentgen (r), was established for x rays in 1928 by the International X-ray Unit Committee, which was later to become ICRU (IXRUC, 1928). The first official use of the term 'dose' together with an amended definition of the unit r occurred in the 1937 recommendations of the ICRU (ICRU, 1938). The ICRU suggested the concept of absorbed dose and officially defined the name and its unit 'rad' in 1953 to extend the concept of dose to certain materials other than air (ICRU, 1954).

(21) The first dose quantity incorporating relative biological effectiveness (RBE) of different types of radiation used by the ICRU was the 'RBE dose in rems', which was an RBE-weighted sum of absorbed dose in rads prescribed in the 1956 recommendations of the ICRU. This dose quantity was replaced by the dose equivalent, a result of joint efforts between the ICRU and the Commission, which was defined by the product of absorbed dose, quality factor of the radiation, dose distribution factor and other necessary modifying factors (ICRU, 1962). The 'rem' was retained as the unit of dose equivalent. Furthermore, the ICRU defined another dose quantity, kerma, and changed the name of exposure dose to simple 'exposure' in its 1962 recommendations.

(22) In its 1977 Recommendations (ICRP, 1977), the Commission introduced a new dose equivalent quantity for limitation of stochastic effects by defining a weighted sum of dose equivalents of various tissues and organs of the human body, where the weighting factor was named 'tissue weighting factor' (ICRP, 1977). The Commission named this new weighted dose equivalent quantity 'effective dose equivalent' at the 1978 Stockholm meeting (ICRP, 1978). At the same time, the SI units of dose were adopted, replacing rad by gray (Gy) and rem by sievert (Sv).

(23) In its 1990 Recommendations (ICRP, 1991b), the Commission redefined the body-related dose quantities. For protection purposes, the absorbed dose averaged over a tissue or organ was defined as the basic quantity. In addition, considering that biological effects are not solely governed by the linear energy transfer, the Commission decided to use 'radiation weighting factors', which were selected based on the RBE in inducing stochastic effects at low doses, instead of the quality factors used in calculation of the dose equivalent of the 1977 Recommendations. To distinguish the resulting quantity from the dose equivalent, the Commission named the new quantity 'equivalent dose'. Accordingly, the effective dose equivalent was renamed 'effective dose'. There were some modifications in the tissue weighting factors to take into account the new information on health effects of radiation.

(24) More details of the dosimetric quantities and their units currently in use appear in Chapter 4.

1.3. Structure of the Recommendations

(25) Chapter 2 deals with the aims and the scope of the Recommendations. Chapter 3 deals with biological aspects of radiation, and Chapter 4 discusses the quantities and units used in radiological protection. Chapter 5 describes the conceptual framework of the system of radiological protection and Chapter 6 deals with the implementation of the Commission's Recommendations for the three different types

of exposure situations. Chapter 7 describes the medical exposure of patients and Chapter 8 discusses protection of the environment.

1.4. References

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2. THE AIMS AND SCOPE OF THE RECOMMENDATIONS

2.1. The aims of the Recommendations

(26) The primary aim of the Commission's Recommendations is to contribute to an appropriate level of protection for people and the environment against the detrimental effects of radiation exposure without unduly limiting the desirable human actions that may be associated with such exposure.

(27) This aim cannot be achieved solely on the basis of scientific knowledge on radiation exposure and its health effects. It requires a model for protecting humans and the environment against radiation. The Recommendations are based on scientific knowledge and on expert judgement. Scientific data, such as those concerning health risks attributable to radiation exposure, are a necessary prerequisite, but societal and economic aspects of protection have also to be considered. All of 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, radiological protection is not different from other fields concerned with the control of hazards. The Commission believes that the basis for, and distinction between, scientific estimations and value judgements should be made clear whenever possible, so as to increase the transparency, and thus the understanding, of how decisions have been reached.

(28) Radiological protection deals with two types of harmful effect. High doses will cause deterministic effects (harmful tissue reactions, see Chapter 3), often of an acute nature, which only appear if the dose exceeds a threshold value. Both high and low doses may cause stochastic effects (cancer or heritable effects), which may be observed as a statistically detectable increase in the incidences of these effects occurring long after exposure.

(29) The Commission's system of radiological protection aims primarily to protect human health. Its health objectives are relatively straightforward: to manage and control exposures to ionising radiation so that deterministic effects are prevented, and the risks of stochastic effects are reduced to the extent reasonably achievable.

(30) In contrast, there is no simple or single universal definition of 'environmental protection' and the concept differs from country to country and from one circumstance to another. Other ways of considering radiation effects are therefore likely to prove to be more useful for non-human species – such as those that cause early mortality, or morbidity, or reduced reproductive success. The Commission's aim is now that of preventing or reducing the frequency of deleterious radiation effects to a level where they would have a negligible impact on the maintenance of biological diversity, the conservation of species, or the health and status of natural habitats, communities and ecosystems. In achieving this aim, however, the Commission recognises that exposure to radiation is but one factor to consider, and is often likely to be a minor one. The Commission will give guidance and advice to ensure that its approach is commensurate with the level of risk, and compatible with efforts being made to protect the environment from the impacts of other human activities.

2.2. The basis and structure of the system of protection

(31) Because of the variety of radiation exposure situations and of the need to achieve a consistency across a wide range of applications, the Commission has established a formal system of radiological protection aimed at encouraging a feasible and structured approach to protection. The system has to deal with a number of sources of exposure, some already being in place, and others that may be introduced deliberately as a matter of choice by society or as a result of emergencies. These sources are linked by a variety of interconnected events and situations leading to exposure of individuals, groups, or entire populations, both in the present and in the future. The system of protection has been developed to allow this complex network to be treated by a logical structure.

(32) The system of protection of humans is based on the use of a) reference anatomical and physiological models of the human being for the assessment of radiation doses, b) studies at the molecular and cellular level, c) experimental animal studies, and d) epidemiological studies. The use of models has resulted in the derivation of tabulated, standardised data on the committed 'dose per unit intake' of different radionuclides for internal exposures and 'dose per unit air kerma or fluence' for external exposures of workers, patients, and the public. Epidemiological and experimental studies have resulted in the estimation of risks associated with the external and internal radiation exposure. For biological effects, the data come from human experience supported by experimental biology. For cancer and heritable effects, the Commission's starting points are the results of epidemiological studies and of studies on animal and human genetics. These are supplemented by information from experimental studies on the mechanisms of carcinogenesis and heredity, in order to provide risk estimates at the low doses of interest in radiological protection.

(33) In view of the uncertainties surrounding the values of tissue weighting factors and the estimate of detriment, the Commission considers it appropriate for radiological protection purposes to use age- and sex-averaged tissue weighting factors and numerical risk estimates. The system of protection is sufficiently robust to achieve adequate protection for both sexes. Moreover, this obviates the requirement for sex- and age-specific radiological protection criteria which could prove unnecessarily discriminatory. However, for the purposes of retrospective evaluation of radiation-related risks, such as in epidemiological studies, it is appropriate to use sex- and age-specific data and calculate sex- and age-specific risks. The details of the Commission's methods for calculating detriment are discussed in Annexes A and B.

(34) The Commission's risk estimates are called 'nominal' because they relate to the exposure of a nominal population of females and males with a typical age distribution and are computed by averaging over age groups and both sexes. The dosimetric quantity recommended for radiological protection, effective dose, is also computed by age- and sex-averaging. There are many uncertainties inherent in the definition of nominal factors to assess effective dose. The estimates of fatality and detriment coefficients are adequate for radiological protection purposes, but, as with all estimates derived from epidemiology, the nominal risk coefficients do not apply to

specific individuals. For the estimation of the likely consequences of an exposure of an individual or a known population, it is necessary to use specific data relating to the exposed individual.

(35) Situations in which the dose thresholds for deterministic effects in relevant organs could be exceeded should be subjected to protective actions under almost any circumstances, as already recommended by the Commission (ICRP, 1999a). It is prudent to take uncertainties in the current estimates of thresholds for deterministic effects into account, particularly in situations involving prolonged exposures. Consequently, annual doses rising towards 100 mSv will almost always justify the introduction of protective actions.

(36) At radiation doses below around 100 mSv in a year, the increase in the incidence of stochastic effects is assumed by the Commission to occur with a small probability and in proportion to the increase in radiation dose over the background dose. Use of this so-called linear-non-threshold (LNT) model is considered by the Commission to be the best practical approach to managing risk from radiation exposure and commensurate with the 'precautionary principle' (UNESCO, 2005). The Commission considers that the LNT model remains a prudent basis for radiological protection at low doses and low dose rates (ICRP, 2005d).

(37) Even within a single class of exposure, an individual may be exposed by several sources, so an assessment of the total exposure has to be attempted. This assessment is called '*individual-related*'. It is also necessary to consider the exposure of all the individuals exposed by a source or group of sources. This procedure is called a '*source-related*' assessment. The Commission emphasises the primary importance of source-related assessments, because action can be taken for a source to assure the protection of individuals from that source.

(38) The probabilistic nature of stochastic effects and the properties of the LNT model make it impossible to derive a clear distinction between 'safe' and 'dangerous', and this creates some difficulties in explaining the control of radiation risks. The major policy implication of the LNT model is that some finite risk, however small, must be assumed and a level of protection established based on what is deemed acceptable. This leads to the Commission's system of protection with its three fundamental principles of protection:

- Justification.
- Optimisation of protection.
- Application of dose limits.

These principles are discussed in more detail in Section 5.6.

(39) In protecting individuals from the harmful effects of ionising radiation, it is the control (in the sense of restriction) of radiation doses that is important, no matter what the source.

(40) The principal components of the system of radiological protection can be summarised as follows.

- A characterisation of the possible situations where radiation exposure may occur (planned, emergency, and existing exposure situations).

- A classification of the types of exposure (those that are certain to occur and potential exposures, as well as occupational exposure, medical exposure of patients and public exposure).
- An identification of the exposed individuals (workers, patients, and members of the public).
- A categorisation of the types of assessment, namely source-related and individual-related.
- A precise formulation of the principles of protection: justification, optimisation of protection, and application of dose limits.
- A description of the levels of individual doses that require protective action or assessment (dose limits, dose constraints, and reference levels).
- A delineation of the conditions for the safety of radiation sources, including their security and the requirements for emergency preparedness and response.

(41) The implementation of the system of radiological protection as described in these Recommendations and summarised above should be monitored and assessed. Periodic reviews are important with a view to learning from experience and identifying any areas for improvement.

(42) In these Recommendations, the Commission uses the same conceptual approach in the source-related protection, and emphasises the optimisation of protection regardless of the type of source, exposure situation, or exposed individual. Source-related restrictions on doses or risks are applied during the optimisation of protection. In principle, protective options that imply doses above the level of such restrictions should be rejected. The Commission has previously used the term 'constraint' for these restrictions for practices. For reasons of consistency, the Commission will continue to use this term in the context of planned exposure situations because such situations encompass the normal operation of practices. The Commission recognises, however, that the word 'constraint' is interpreted in many languages as a rigorous limit. Such a meaning was never the Commission's intention, as their application must depend upon local circumstances.

(43) Levels for protective action may be selected on the basis of generic considerations including the Commission's general Recommendations (see Table 8, Section 6.5) or best practice. In any specific set of circumstances, particularly in an emergency or an existing exposure situation, it could be the case that no viable protective option can immediately satisfy the level of protection selected from generic considerations. Thus interpreting a constraint rigorously as a form of limit could seriously and adversely distort the outcome of an optimisation process. For this reason, in emergency or existing exposure situations, the Commission proposes to use the term 'reference level' for the restriction on dose or risk, above which it is judged to be inappropriate to plan to allow exposures to occur, and below which optimisation of protection should be implemented. The Commission wishes to emphasise, however, that the difference in name between planned exposure situations and the other two exposure situations does not imply any fundamental difference in the application of the system of protection. Further guidance on the application of the optimisation

principle in planned exposure situations, emergency exposure situations, and existing exposure situations is provided in Chapter 6.

2.3. The scope of the Recommendations

(44) The Commission's system of radiological protection applies to all radiation exposures from any source, regardless of its size and origin. The term *radiation* is used to mean ionising radiation. The Commission has been using the term *radiation exposure* (or *exposure* in short) in a generic sense to mean the process of being exposed to radiation or radionuclides, the significance of exposure being determined by the resulting radiation dose (ICRP, 1991b). The term '*source*' is used to indicate the cause of an exposure, and not necessarily a physical source of radiation (see Section 5.1). In general, for the purposes of applying the Recommendations, a source is an entity for which radiological protection can be optimised as an integral whole.

(45) The Commission has aimed to make its Recommendations applicable as widely and as consistently as possible. In particular, the Commission's Recommendations cover exposures to both natural and man-made sources. The Recommendations can apply in their entirety only to situations in which either the source of exposure or the pathways leading to the doses received by individuals can be controlled by some reasonable means. Sources in such situations are called *controllable sources*.

(46) There can be many sources, and some individuals may be exposed to radiation from more than one of them. Provided that doses are below the threshold for deterministic effects (harmful tissue reactions), the presumed proportional relationship between the additional dose attributable to the situation and the corresponding increase in the probability of stochastic effects makes it possible to deal independently with each component of the total exposure and to select those components that are important for radiological protection. Furthermore, it is possible to subdivide these components into groups that are relevant to various purposes.

(47) The Commission has previously distinguished between practices that add doses, and interventions that reduce doses (ICRP, 1991b). The Commission now uses a situation-based approach to characterise the possible situations where radiation exposure may occur as *planned, emergency*, and *existing exposure situations*; it applies one set of fundamental principles of protection to all of these situations (see Section 5.6).

(48) The term '*practice*' has, however, become widely used in radiological protection. The Commission will continue to use this term to denote an activity that causes an increase in exposure to radiation or in the risk of exposure to radiation.

(49) Practices can be activities such as a business, trade, industry or any other productive activity; it can also be a government undertaking, or a charity. It is implicit in the concept of a practice that the radiation sources that it introduces or maintains can be controlled directly by action on the source.

(50) The term '*intervention*' has also become widely used in radiological protection and has been incorporated into national and international standards to describe situations where actions are taken to reduce exposures. The Commission believes

that it is more appropriate to limit the use of this term to describe protective *actions* that reduce exposure, while the terms 'emergency' or 'existing exposure' will be used to describe radiological *exposure situations* where such protective actions to reduce exposures are required.

2.4. Exclusion and exemption

(51) The fact that the Commission's Recommendations are concerned with any level and type of radiation exposure does not mean that all exposures, all sources, and all human actions, can or need to be equally considered when establishing the legal and regulatory systems for their application. Instead, a graded burden of obligation must be foreseen according to the amenability of a particular source or exposure situation to regulatory controls, and the level of exposure/risk associated with that source or situation.

(52) There are two distinct concepts that delineate the extent of radiological protection control, namely (i) the exclusion of certain exposure situations from radiological protection legislation, usually on the basis that they are not amenable to control with regulatory instruments (cannot be regulated), and (ii) the exemption from some or all radiological protection regulatory requirements for situations where such controls are regarded as unwarranted, often on the basis that the effort to control is judged to be excessive compared to the associated risk (need not be regulated). A legislative system for radiological protection should first establish what should be within the legal system and what should be outside it and therefore excluded from the law and its regulations. Secondly, the system should also establish what could be exempted from some or all regulatory requirements because regulatory action is unwarranted. For this purpose, the legislative framework should permit the regulatory authority to exempt situations from specified regulatory requirements, particularly from those of an administrative nature such as notification and authorisation or exposure assessment and inspection. While exclusion is firmly related to defining the scope of the control system, it may not be sufficient as it is just one mechanism. Exemption, on the other hand, relates to the power of regulatory authorities to determine that a source or practice need not be subject to some or all aspects of regulatory control. The distinction between exclusion and exemption is not absolute; regulatory authorities in different countries may take different decisions about whether to exempt or exclude a specific source or situation.

(53) Exposures that may be excluded from radiological protection legislation include uncontrollable exposures and exposures that are essentially not amenable to control regardless of their magnitude. Uncontrollable exposures are those that cannot be restricted by regulatory action under any conceivable circumstance, such as exposure to the radionuclide potassium–40 incorporated into the human body. Exposures that are not amenable to control are those for which control is obviously impractical, such as exposure to cosmic rays at ground level. The decision as to what exposures are not amenable to control requires a judgment by the legislator, which may be influenced by cultural perceptions. For instance, national attitudes to the reg-

ulation of exposures to natural occurring radioactive materials are extremely variable.

(54) Further guidance on exclusion and exemption is provided in *Publication 104* (ICRP, 2007a).

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3. BIOLOGICAL ASPECTS OF RADIOLOGICAL PROTECTION

(55) Most adverse health effects of radiation exposure may be grouped in two general categories:

- deterministic effects (harmful tissue reactions) due in large part to the killing/ malfunction of cells following high doses; and
- stochastic effects, i.e., cancer and heritable effects involving either cancer development in exposed individuals owing to mutation of somatic cells or heritable disease in their offspring owing to mutation of reproductive (germ) cells.

Consideration is also given to effects on the embryo and fetus, and to diseases other than cancer.

(56) In *Publication 60* (ICRP, 1991b) the Commission classified the radiation effects that result in tissue reactions as deterministic effects and used the term stochastic effects for radiation-induced cancer and heritable disease. Effects caused by injury in populations of cells were called non-stochastic in *Publication 41* (ICRP, 1984), and this was replaced by the term deterministic, meaning 'causally determined by preceding events' in *Publication 60* (ICRP, 1991b). The generic terms, deterministic and stochastic effects, are not always familiar to those outside the field of radiological protection. For this and other reasons (given in Annex A), Chapter 3 and Annex A also use the directly descriptive terms tissue reactions and cancer/heritable effects respectively. However, the Commission recognises that the generic terms, deterministic and stochastic effects, have a firmly embedded use in its system of protection and will use the generic and directly descriptive terms synonymously, according to context.

(57) In this respect the Commission notes that some radiation-associated health consequences, particularly some non-cancer effects (see Section 3.3), are not yet sufficiently well understood to assign to either of the generic categories. Since 1990, the Commission has reviewed many aspects of the biological effects of radiation. The views developed by the Commission are summarised in this Chapter with emphasis on effective doses of up to about 100 mSv (or absorbed doses of about 100 mGy of low-LET radiation) delivered as a single dose or accumulated annually. A more detailed summary of the post-1990 developments in radiation biology and epidemiology is provided in Annex A and *Publication 99* (ICRP, 2005d) together with explanations of the judgements that underpin the recommendations made in this Chapter.

3.1. The induction of deterministic effects (harmful tissue reactions)

(58) The induction of tissue reactions is generally characterised by a threshold dose. The reason for the presence of this threshold dose is that radiation damage (serious malfunction or death) of a critical population of cells in a given tissue needs to be sustained before injury is expressed in a clinically relevant form. Above the threshold dose the severity of the injury, including impairment of the capacity for tissue recovery, increases with dose.

(59) Early (days to weeks) tissue reactions to radiation in cases where the threshold dose has been exceeded may be of the inflammatory type resulting from the release of cellular factors, or they may be reactions resulting from cell loss (*Publication 59*, ICRP, 1991a). Late tissue reactions (months to years) can be of the generic type if they arise as a direct result of damage to that tissue. By contrast other late reactions may be of the consequential type if they arise as a result of early cellular damage (Dörr and Hendry, 2001). Examples of these radiation-induced tissue reactions are given in Annex A.

(60) Reviews of biological and clinical data have led to further development of the Commission's judgements on the cellular and tissue mechanisms that underlie tissue reactions and the dose thresholds that apply to major organs and tissues. However, in the absorbed dose range up to around 100 mGy (low LET or high LET) no tissues are judged to express clinically relevant functional impairment. This judgement applies to both single acute doses and to situations where these low doses are experienced in a protracted form as repeated annual exposures.

(61) Annex A provides updated information on dose thresholds (corresponding to doses that result in about 1% incidence) for various organs and tissues. On the basis of current data the Commission judges that the occupational and public dose limits, including the limits on equivalent dose for the skin, hands/feet and eyes, given in *Publication 60* (ICRP, 1991b) remain applicable for preventing the occurrence of deterministic effects (tissue reactions); see Section 5.10 and Table 6. However, new data on the radiosensitivity of the eye are expected and the Commission will consider these data when they become available. In addition, in Annex A, reference is made to the clinical criteria that apply to dose limits on equivalent doses to the skin.

3.2. The induction of stochastic effects

(62) In the case of cancer, epidemiological and experimental studies provide evidence of radiation risk albeit with uncertainties at doses about 100 mSv or less. In the case of heritable diseases, even though there is no direct evidence of radiation risks to humans, experimental observations argue convincingly that such risks for future generations should be included in the system of protection.

3.2.1. Risk of cancer

(63) The accumulation of cellular and animal data relevant to radiation tumorigenesis has, since 1990, strengthened the view that DNA damage response processes in single cells are of critical importance to the development of cancer after radiation exposure. These data, together with advances in knowledge of the cancer process in general, give increased confidence that detailed information on DNA damage response/repair and the induction of gene/chromosomal mutations can contribute significantly to judgements on the radiation-associated increase in the incidence of cancer at low doses. This knowledge also influences judgements on relative biological

effectiveness (RBE), radiation weighting factors, and dose and dose-rate effects. Of particular importance are the advances in understanding radiation effects on DNA such as the induction of complex forms of DNA double strand breaks, the problems experienced by cells in correctly repairing these complex forms of DNA damage, and the consequent appearance of gene/chromosomal mutations. Advances in microdosimetric knowledge concerning aspects of radiation-induced DNA damage have also contributed significantly to this understanding (see Annexes A and B).

(64) Although there are recognised exceptions, for the purposes of radiological protection the Commission judges that the weight of evidence on fundamental cellular processes coupled with dose-response data supports the view that, in the low dose range, below about 100 mSv, it is scientifically plausible to assume that the incidence of cancer or heritable effects will rise in direct proportion to an increase in the equivalent dose in the relevant organs and tissues.

(65) Therefore, the practical system of radiological protection recommended by the Commission will continue to be based upon the assumption that at doses below about 100 mSv a given increment in dose will produce a directly proportionate increment in the probability of incurring cancer or heritable effects attributable to radiation. This dose-response model is generally known as 'linear-non-threshold' or LNT. This view accords with that given by UNSCEAR (2000). Other estimates have been provided by various national organisations, some in line with the UNSCEAR view (e.g., NCRP, 2001, NAS/NRC, 2006) while a report from the French Academies (2005) argues in support of a practical threshold for radiation cancer risk. However, from an analysis conducted by the Commission (*Publication 99*, ICRP, 2005d), the Commission considers that the adoption of the LNT model combined with a judged value of a dose and dose rate effectiveness factor (DDREF) provides a prudent basis for the practical purposes of radiological protection, i.e., the management of risks from low-dose radiation exposure.

(66) However, the Commission emphasises that whilst the LNT model remains a scientifically plausible element in its practical system of radiological protection, biological/epidemiological information that would unambiguously verify the hypothesis that underpins the model is unlikely to be forthcoming (see also UNSCEAR, 2000, NCRP 2001). Because of this uncertainty on health effects at low doses, the Commission judges that it is not appropriate, for the purposes of public health planning, to calculate the hypothetical number of cases of cancer or heritable disease that might be associated with very small radiation doses received by large numbers of people over very long periods of time (see also Sections 4.4.7 and 5.8).

(67) In arriving at its practical judgement on the LNT model, the Commission has considered potential challenges associated with information on cellular adaptive responses, the relative abundance of spontaneously arising and low-dose-induced DNA damage and the existence of the post-irradiation cellular phenomena of induced genomic instability and bystander signalling (*Publication 99*, ICRP, 2005d). The Commission recognises that these biological factors, together with possible tumour-promoting effects of protracted irradiation, and immunological phenomena, may influence radiation cancer risk (Streffer et al., 2004), but that current uncertainties

on the mechanisms and tumorigenic consequences of the above processes are too great for the development of practical judgements. The evidence is reviewed in *Publication 99* and in UNSCEAR (2008). The Commission also notes that since the estimation of nominal cancer risk coefficients is based upon direct human epidemiological data, any contribution from these biological mechanisms would be included in that estimate. Uncertainty with regard to the role of these processes in cancer risk will remain until their relevance to cancer development in vivo is demonstrated and there is knowledge of the dose dependence of the cellular mechanisms involved.

(68) Since 1990, further epidemiological information has accumulated on the risk of organ-specific cancer following exposure to radiation. Much of this new information has come from the continuing follow-up of survivors of the atomic bomb explosions in Japan in 1945 – the Life Span Study (LSS). For cancer mortality (Preston et al., 2003) the follow-up is 47 years (October 1950–December 1997); for cancer incidence (Preston et al., 2007) the follow-up period is 41 years (January 1958 to December 1998). These latter data, which were not available in 1990, can provide more reliable estimates of risk principally because cancer incidence can allow for more accurate diagnosis. The Commission has therefore placed emphasis on incidence data for its present Recommendations. In addition, epidemiological data from the LSS provide further information on the temporal and age-dependent pattern of radiation cancer risk, particularly the assessment of risk amongst those exposed at early ages. Overall, current cancer risk estimates derived from the LSS are not greatly changed since 1990, but the inclusion of the cancer incidence data provides a firmer foundation for the risk modelling described in Annex A.

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

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

(71) In principle, epidemiological data on protracted exposure, such as those from environmental and occupational circumstances, should be directly informative on judgements of DDREF. However, the statistical precision afforded by these studies

Table 1. Detriment-adjusted nominal risk coefficients (10^{-2} Sv^{-1}) for stochastic effects after exposure to radiation at low dose rate.

Exposed population	Cancer		Heritable effects		Total	
	Present[1]	*Publ. 60*	Present[1]	*Publ. 60*	Present[1]	*Publ. 60*
Whole	5.5	6.0	0.2	1.3	5.7	7.3
Adult	4.1	4.8	0.1	0.8	4.2	5.6

[1] Values from Annex A.

and other uncertainties associated with the inability to adequately control for confounding factors (see Annex A), do not allow for a precise estimate of DDREF at this time. Accordingly the Commission has decided to continue to use broad judgements in its choice of DDREF based upon dose-response features of experimental data, the LSS, and the results of probabilistic uncertainty analysis conducted by others (NCRP, 1997, EPA, 1999, NCI/CDC, 2003, Annex A).

(72) The BEIR VII Committee (NAS/NRC, 2006) recently combined radiobiological and epidemiological evidence concerning DDREF via a Bayesian statistical analysis. The data sets used were: a) solid cancer in the LSS; and b) cancer and life shortening in animals. The modal value of DDREF from these analyses was 1.5 with a range of 1.1 to 2.3 and from this the BEIR VII Committee chose a value of 1.5. The BEIR VII Committee recognised the subjective and probabilistic uncertainties inherent in their specific choice, and a DDREF of 2 remains compatible with the data used and analyses conducted. Further to this, the Commission notes from Annex A that, for the induction of gene and chromosomal mutations, values of DDREF generally fall in the range of 2–4, and for the induction of cancer in animals and life shortening in animals, values of DDREF generally fall in the range of 2–3.

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

3.2.2. Risk of heritable effects

(74) There continues to be no direct evidence that exposure of parents to radiation leads to excess heritable disease in offspring. However, the Commission judges that there is compelling evidence that radiation causes heritable effects in experimental animals. Therefore, the Commission prudently continues to include the risk of heritable effects in its system of radiological protection.

(75) The Commission also notes reports (reviewed in UNSCEAR, 2001) which argue, on the basis of A-bomb survivor and mouse genetic data, that the risk of heritable diseases tended to be overestimated in the past. There are some post-1990 human and animal data on the quantitative aspects of radiation-induced germ cell mutation that impact on the Commission's judgement on the risk of induction of genetic disease expressing in future generations. There have also been substantial advances in the fundamental understanding of human genetic diseases and the process of germ line mutagenesis including that occurring after radiation. The Commission has reappraised the methodology used in *Publication 60* for the estimation of heritable risks including risks of multifactorial diseases (*Publication 83*, ICRP, 1999b).

(76) The Commission has now adopted a new framework for the estimation of heritable risks that employs data from human and mouse studies (UNSCEAR, 2001, NAS/NRC, 2006). Also, for the first time, a scientifically justified method for the estimation of risk of multifactorial disease has been included. Mouse studies continue to be used to estimate genetic risks because of the lack of clear evidence in humans that germline mutations caused by radiation result in demonstrable genetic effects in offspring.

(77) The new approach to heritable risks continues to be based on the concept of the doubling dose (DD) for disease-associated mutations used in *Publication 60*. However, the methodology differs in that recoverability of mutations in live births is allowed for in the estimation of DD. An additional difference is that direct data on spontaneous human mutation rates are used in conjunction with radiation-induced mutation rates derived from mouse studies. This new methodology (see Annex A, Box A.2) is based on the UNSCEAR 2001 report and has also been used recently by NAS/NRC (2006). In *Publication 60* genetic risks were expressed at a theoretical equilibrium between mutation and selection. In the light of further knowledge the Commission judges that many of the underlying assumptions in such calculations are no longer sustainable. The same view has been expressed by UNSCEAR (2001) and NAS/NRC (2006). Accordingly, the Commission now expresses genetic risks up to the second generation only.

(78) The Commission judges that this procedure will not lead to a significant underestimation of heritable effects. This issue is discussed in UNSCEAR (2001) and in detail in Annex A where it is argued that there are no substantial differences between genetic risks expressed at 2 and 10 generations.

(79) The Commission's present estimate of genetic risks up to the second generation of about 0.2% per Gy is essentially the same as that cited by UNSCEAR (2001) (see Annex A and UNSCEAR 2001, Table 46). However, given the major changes in methodology, the close similarity of the present second generation risk to that of *Publication 60* is coincidental. The present value relates to continuous low-dose-rate exposures over these two generations.

3.2.3. Detriment-adjusted nominal risk coefficients for cancer and heritable effects

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

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

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

(83) On the basis of these calculations the Commission proposes nominal probability coefficients for detriment-adjusted cancer risk as 5.5 10^{-2} Sv^{-1} for the whole population and 4.1 10^{-2} Sv^{-1} for adult workers. For heritable effects, the detriment-adjusted nominal risk in the whole population is estimated as 0.2 10^{-2} Sv^{-1} and in adult workers as 0.1 10^{-2} Sv^{-1}. The most significant change from *Publication 60* is the 6–8 fold reduction in the nominal risk coefficient for heritable effects. These estimates are shown in Table 1, where they are compared with the estimates of detriment used in the 1990 Recommendations in *Publication 60* (ICRP, 1991b). The revised estimate of genetic risk has reduced the judged value of the tissue weighting factor for the gonads considerably (see Chapter 4 and detailed arguments in Annex A). However, the Commission emphasises that this reduction in the gonadal tissue weighting factor provides no justification for allowing controllable gonadal exposures to increase in magnitude.

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

(85) Note that, although all coefficients in Table 1 are presented as fractional values, this presentation is used for the purposes of comparability to Annex A only and does not imply a level of precision (see paragraphs 81 and 82).

(86) In spite of changes in the cancer risk data and their treatment, the present nominal risk coefficients are wholly compatible with those presented by the Commission in *Publication 60* (ICRP, 1991b). Given the uncertainties discussed in Annex A, the Commission considers that the small difference in the estimate of nominal risk since 1990 is of no practical significance.

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

3.2.4. Genetic susceptibility to cancer

(88) The issue of individual genetic differences in susceptibility to radiation-induced cancer was noted in *Publication 60* and reviewed in *Publication 79* (ICRP, 1998a). Since 1990, there has been a remarkable expansion in knowledge of the various single gene human genetic disorders, where excess spontaneous cancer is expressed in a high proportion of gene carriers – the so-called high penetrance genes which can be strongly expressed as excess cancer. Studies with cultured human cells and genetically altered laboratory rodents have also contributed much to knowledge and, with more limited epidemiological and clinical data, suggest that most of the rare single gene, cancer prone disorders will show greater-than-normal sensitivity to the tumorigenic effects of radiation.

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

(90) On the basis of the data and judgements developed in *Publication 79* and further information reviewed in the UNSCEAR (2000, 2001) and NAS/NRC (2006) reports, the Commission believes that strongly expressing, high penetrance, cancer genes are too rare to cause significant distortion of population-based estimates of low-dose radiation cancer risk. Although the Commission recognises that variant cancer genes of low penetrance may, in principle, be sufficiently common to impact upon population-based estimates of radiation cancer risk, the information available is insufficient to provide a meaningful quantitative judgement on this issue.

3.3. The induction of diseases other than cancer

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

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

about 100 mSv. This agrees with the conclusion of UNSCEAR (2008), which found little evidence of any excess risk below 1 Gy.

3.4. Radiation effects in the embryo and fetus

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

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

(95) In respect of the induction of malformations, the new data strengthen the view that there are gestational age-dependent patterns of in-utero radiosensitivity with maximum sensitivity being expressed during the period of major organogenesis. On the basis of animal data it is judged that there is a true dose threshold of around 100 mGy for the induction of malformations; therefore, for practical purposes, the Commission judges that risks of malformation after in-utero exposure to doses well below 100 mGy are not expected.

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

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

3.5. Judgements and uncertainties

(98) Although the potential importance of synergistic effects between radiation and other agents is recognised by the Commission, at the present time there is no firm evidence for such interactions at low doses that would justify a modification of existing radiation risk estimates (UNSCEAR, 2000).

(99) Taking into account the information discussed in this Section, the practical system of radiological protection recommended by the Commission will continue to be based upon the assumption that, at doses below about 100 mSv, a given increment in dose will produce a directly proportionate increment in the probability of incurring cancer or heritable effects attributable to radiation. The Commission considers that the continued application of the LNT model combined with a judged value of DDREF provides a prudent basis for practical purposes of radiological protection, i.e., the management of risks from low-dose radiation exposure in prospective situations.

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4. QUANTITIES USED IN RADIOLOGICAL PROTECTION

4.1. Introduction

(100) Special *dosimetric quantities* have been developed for the assessment of doses from radiation exposures. The fundamental *protection quantities* adopted by the Commission are based on measures of the energy deposited in organs and tissues of the human body. In order to relate the radiation dose to radiation risk (detriment), it is also necessary to take into account variations in the biological effectiveness of radiations of different quality as well as the varying sensitivity of organs and tissues to ionising radiation.

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

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

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

4.2. Considerations of health effects

(104) Radiological protection in the low dose range is primarily concerned with protection against radiation-induced cancer and heritable disease. These effects are taken to be probabilistic in nature, with no threshold, and to increase in frequency in proportion to the radiation dose (see Chapter 3 and Annex A). In the definition and calculation of effective dose the recommended radiation weighting factors, w_R, allow for the differences in the effect of various radiations in causing stochastic effects while tissue weighting factors, w_T, allow for the variations in radiation sensitivity of different organs and tissues to the induction of stochastic effects (see Section 4.3.4 and Annex B). The radiation weighting factors for radiations characterised by a high linear energy transfer, so-called high-LET radiations (see Section 4.3.3), are derived for stochastic effects at low doses.

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

4.3. Dose quantities

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

4.3.1. Absorbed dose

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

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

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

The definition of absorbed dose has the scientific rigour required for a basic physical quantity (Annex B).

4.3.2. Averaging of dose

(109) When using the quantity absorbed dose in practical protection applications, doses are averaged over tissue volumes. It is assumed that, for low doses, the mean value of absorbed dose averaged over a specific organ or tissue can be correlated with radiation detriment for stochastic effects in that tissue with an accuracy sufficient for the purposes of radiological protection. The averaging of absorbed doses in tissues or organs and the summing of weighted mean doses in different organs and tissues of the human body comprise the basis for the definition of the protection quantities which are used for limiting stochastic effects at low doses. This approach is based on the LNT model and therefore allows the addition of doses resulting from external and internal exposure.

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

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

4.3.3. Equivalent dose and radiation weighting factors

(112) The protection quantities are used to specify exposure limits to ensure that the occurrence of stochastic health effects is kept below unacceptable levels and that tissue reactions are avoided. The definition of the protection quantities is based on

Table 2. Recommended radiation weighting factors.

Radiation type	**Radiation weighting factor, w_R**
Photons	1
Electrons[a] and muons	1
Protons and charged pions	2
Alpha particles, fission fragments, heavy ions	20
Neutrons	A continuous function of neutron energy (see Fig. 1 and Eq. 4.3)

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

[a] Note the special issue of Auger electrons discussed in paragraph 116 and in Section B.3.3 of Annex B.

the average absorbed dose, $D_{T,R}$ in the volume of a specified organ or tissue T (see Table 3), due to radiation of type R (see Table 2). The radiation R is given by the type and energy of radiation either incident on the body or emitted by radionuclides residing within it. The protection quantity *equivalent dose* in an organ or tissue, H_T, is then defined by

$$H_T = \sum_R w_R D_{T,R} \tag{4.2}$$

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

(113) In the early 1960s, radiation weighting in the definition of radiological protection quantities was related to the radiation quality factor, Q, as a function of LET and denoted as L in the $Q(L)$ function of *Publication 26* (ICRP, 1977). In *Publication 60* (ICRP, 1991b) the method of radiation weighting was changed in the calculation of the protection quantities equivalent dose and effective dose. The Commission selected a general set of radiation weighting factors (w_R) that were considered to be appropriate for application in radiological protection. The values of w_R were defined largely on the basis of the relative biological effectiveness (RBE) of the different radiations.

(114) A revised set of w_R values has been adopted in these Recommendations based upon a re-evaluation of the available data (see Annexes A and B). The values of w_R for neutrons and protons given in these Recommendations differ from those given in *Publication 60* (see below and Annex B). A w_R value for charged pions has been included. The value of w_R for photons is the same for x rays and gamma rays of all energies. The numerical values of w_R are specified in terms of type and, in the case of neutrons, in terms of energy of radiation either incident on the human body or emitted by radionuclides residing in the body (Table 2). The values of w_R are

Table 3. Recommended tissue weighting factors.

Tissue	w_T	$\sum w_T$
Bone-marrow (red), Colon, Lung, Stomach, Breast, Remainder tissues*	0.12	0.72
Gonads	0.08	0.08
Bladder, Oesophagus, Liver, Thyroid	0.04	0.16
Bone surface, Brain, Salivary glands, Skin	0.01	0.04
	Total	1.00

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

selected by judgement on the basis of a broad range of experimental RBE data which are relevant to stochastic effects. The RBE values increase to a maximum (RBE_M) with decreasing radiation dose (ICRP, 2003c). Values of RBE_M have been used for w_R selection, and fixed values are assigned to these w_R factors for radiological protection purposes.

(115) ***Reference radiation.*** Values of RBE obtained experimentally depend on the reference radiation chosen. Generally, low-LET photon radiation is taken as the reference, although no specific energy has been agreed upon for this purpose. When radiation weighting factors were selected for *Publication 60*, a broad range of experimental RBE data using either high energy x rays above about 200 kV or cobalt–60 or caesium–137 gamma radiation was considered (see Annex B). This approach is also used in these Recommendations, although it should be recognised that experimentally different RBE values can result depending upon the choice of the reference radiation between x rays and higher energy gamma radiation (e.g., cobalt–60). Such differences have been established mainly in studies on cells in vitro (see Annex B).

(116) ***Photons, electrons, and muons.*** Photons, electrons, and muons are radiations with LET values of less than 10 keV/μm. These radiations have always been given a radiation weighting of 1. There are good arguments (see Annex B) to continue to use a w_R of 1 for all low-LET radiations (Annex B, Table 3). This does not, however, imply that there are no differences in radiation quality of photons of different energies. The proposed simplification is sufficient only for the intended application of equivalent dose and effective dose, e.g., for dose limitation and assessment and control of doses in the low-dose range. In cases where individual retrospective risk assessments have to be made, more detailed information on the radiation field and appropriate RBE values may need to be considered if relevant data are available. Heterogeneity of the radiation dose within cells, as can occur with tritium or Auger emitters incorporated into DNA, may also require specific analysis (see Annex B). Equivalent dose and effective dose are not appropriate quantities for use in such assessments (see Section 4.4.6).

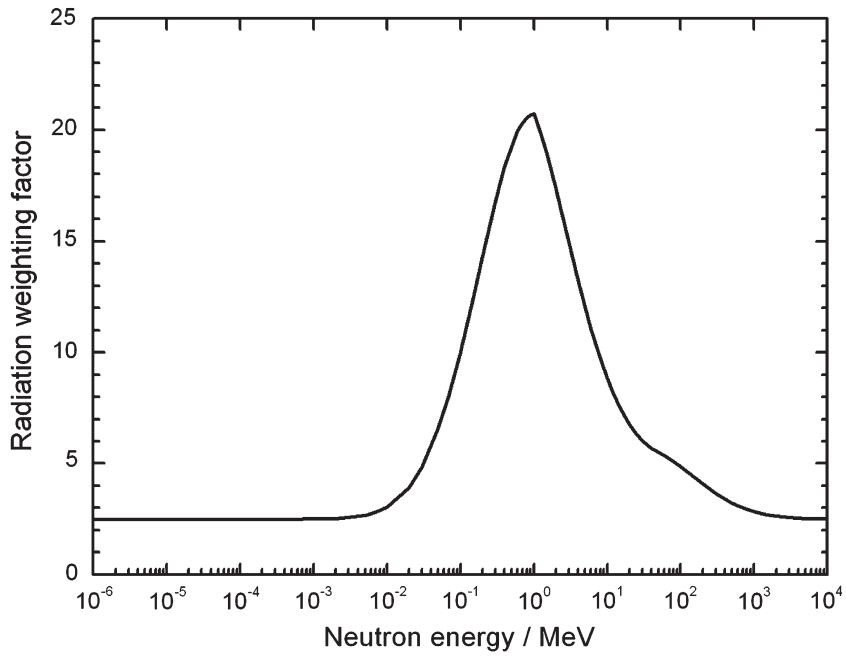

Fig. 1. Radiation weighting factor, w_R, for neutrons versus neutron energy.

(117) ***Neutrons.*** The radiation weighting factor for neutrons reflects their relative biological effectiveness following external exposure. The biological effectiveness of neutrons incident on the human body is strongly dependent on neutron energy (see Annex B).

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

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

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

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

(120) Pions are negatively or positively charged or neutral particles encountered in radiation fields resulting from interactions of primary cosmic rays with nuclei at high altitudes in the atmosphere. These particles contribute to exposures in aircraft. They are also found as part of the complex radiation fields behind shielding of high-energy particle accelerators and thus contribute to the occupational exposure of accelerator staff. Considering that the energy distribution of pions in radiation fields is very broad, the use of a single weighting factor of 2 is recommended for all charged pions.

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

(122) Despite substantial uncertainties in estimates of dose and risk from intakes of alpha-emitting radionuclides, the available human and animal data indicate that the RBE depends on the biological end-point under consideration. The limited human data that allow estimation of alpha particle RBE values suggest values of around 10–20 for lung and liver cancer and lower values for bone cancer and leukaemia. Judgements on the available data and the selection of a w_R value for alpha particles have been reviewed in *Publication 92* (ICRP, 2003c). As recent data do not provide compelling evidence for a change of the radiation weighting factor for alpha particles, the w_R value of 20 adopted in *Publication 60* (ICRP, 1991b) is retained.

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

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

4.3.4. Effective dose and tissue weighting factors

(125) The effective dose, E, introduced in *Publication 60* (ICRP, 1991b) is defined by a weighted sum of tissue equivalent doses as:

$$E = \sum_{T} w_T H_T = \sum_{T} w_T \sum_{R} w_R D_{T,R} \tag{4.4}$$

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

(126) The organs and tissues for which w_T values are specified are given in Table 3 (see also Annex A).

(127) On the basis of epidemiological studies on cancer induction in exposed populations, and risk assessments for heritable effects, a set of w_T values was chosen for these Recommendations (Table 3) based on the respective values of relative radiation detriment (see Table 5 in Annex A). They represent mean values for humans averaged over both sexes and all ages and thus do not relate to the characteristics of particular individuals.

(128) The w_T for the remainder tissues (0.12) applies to the arithmetic mean dose of the 13 organs and tissues for each sex listed in the footnote to Table 3. The so-called splitting rule in the treatment of the remainder in *Publication 60* (ICRP, 1991b) is no longer used and hence the effective dose is additive.

4.3.5. Determination of effective dose

Reference phantoms

(129) The quantities equivalent dose and effective dose are not measurable in practice. For occupational exposures, their values are determined by radiation monitoring using operational quantities (see Section 4.3.6). For the calculation of conversion coefficients for external exposure, computational phantoms are used for dose assessment in

various radiation fields. For the calculation of dose coefficients from intakes of radionuclides, biokinetic models for radionuclides, reference physiological data, and computational phantoms are used (see Annex B).

(130) The evaluation of equivalent doses for the Reference Male and Female and of effective dose for the Reference Person is based on the use of anthropomorphic models (phantoms). In the past, the Commission did not specify a particular phantom, and in fact various mathematical phantoms such as hermaphrodite MIRD-type phantoms (Snyder et al., 1969), the sex-specific models of Kramer et al. (1982), or the age-specific phantoms of Cristy and Eckerman (1987) have been used. The Commission now uses reference computational phantoms of the adult Reference Male and adult Reference Female for the calculation of equivalent doses for organs and tissues (Fig. 2). The phantoms are based on medical tomographic images (Zankl et al., 2005). They are made up of three-dimensional volume pixels (voxels). The voxels that make up defined organs have been adjusted to approximate the organ masses assigned to the Reference Male and Reference Female in *Publication 89* (ICRP, 2002). In order to provide a practicable approach for the assessment of equivalent doses and effective dose, conversion coefficients relating to physical quantities, e.g., particle fluence or air kerma for external exposure and activity intake for internal exposure, are calculated for standard exposure conditions for the reference phantoms.

(131) These models are computational representations of the Reference Male and Reference Female, and are used to compute the mean absorbed dose, D_T, in an organ or tissue T, from reference radiation fields external to the body and from decay of radionuclides after incorporation. They are used for calculations of dose conversion coefficients for external radiation fields and dose coefficients for the intake of radionuclides (see Annex B). These organ and tissue doses are multiplied with the radiation weighting factor to yield the equivalent doses in the tissues and organs of the Reference Male and the Reference Female (see Fig. 2). Reference computational phantoms will also be developed for children of different ages and for the pregnant woman and fetus.

Sex averaging for effective dose

(132) For the purposes of radiological protection, it is useful to apply a single value of effective dose for both sexes (see paragraph 33). The tissue weighting factors of Table 3 are sex- and age-averaged values for all organs and tissues, including the male and female breast, testis, and ovary (gonads: carcinogenic and heritable effects). This averaging implies that the application of this approach is restricted to the determination of effective dose in radiological protection and, in particular, cannot be used for the assessment of individual risk. The effective dose is then computed from the equivalent doses assessed for organ or tissue T of the Reference Male, H_T^M, and Reference Female, H_T^F, according to the following equation (see also Annex B):

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

(133) Analogous to the approach for other organs and tissues, the equivalent dose to the remainder is defined separately for the Reference Male and the Reference Female and these values are included in Eq. (4.5) – see Fig. 2. The equivalent dose

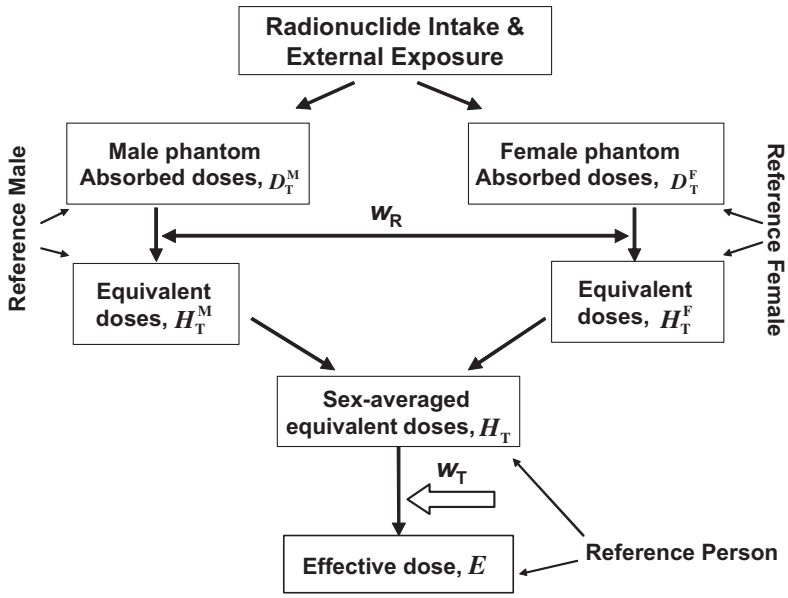

Fig. 2. Sex averaging to obtain the effective dose.

to the remainder tissues is computed as the arithmetic mean of the equivalent doses to the tissues listed in the footnote to Table 3. The equivalent doses to the remainder tissues of the Reference Male, $H^{\mathrm{M}}_{\mathrm{rmd}}$, and the Reference Female, $H^{\mathrm{F}}_{\mathrm{rmd}}$, are computed as

$$H^{\mathrm{M}}_{\mathrm{rmd}} = \frac{1}{13}\sum_{\mathrm{T}}^{13} H^{\mathrm{M}}_{\mathrm{T}} \quad \text{and} \quad H^{\mathrm{F}}_{\mathrm{rmd}} = \frac{1}{13}\sum_{\mathrm{T}}^{13} H^{\mathrm{F}}_{\mathrm{T}}. \tag{4.6}$$

where T is a remainder tissue from Table 3. The summation in Eqn. (4.5) extends over the equivalent dose to remainder tissues in the Reference Male and the Reference Female (Annex B).

(134) The effective dose for protection purposes is based on the mean doses in organs or tissues of the human body. It is defined and estimated in a Reference Person (see Fig. 2). This quantity provides a value which takes account of the given exposure conditions but not of the characteristics of a specific individual. In particular, the tissue weighting factors are mean values representing an average over many individuals of both sexes. The equivalent doses in the organs and tissues of the Reference Male and the Reference Female are averaged (Eqn. 4.5). The averaged dose is multiplied with the corresponding tissue weighting factor. The sum of these products yields the sex-averaged effective dose for the Reference Person (Fig. 2).

4.3.6. Operational quantities

(135) The body-related protection quantities, equivalent dose and effective dose, are not measurable in practice. Therefore, operational quantities are used for the

assessment of effective dose or mean equivalent doses in tissues or organs. These quantities aim to provide a conservative estimate for the value of the protection quantities related to an exposure, or potential exposure, of persons under most irradiation conditions. They are often used in practical regulations or guidance. Different types of operational quantities are used for internal and external exposures as summarised below. More details are given in Annex B.

(136) Operational quantities for area and individual monitoring of external exposures have been defined by ICRU (see Annex B). The operational quantities for area monitoring are the ambient dose equivalent, $H^*(10)$ and the directional dose equivalent, $H'(0.07, \Omega)$. The operational quantity for individual monitoring is the personal dose equivalent, $H_p(d)$, which is the dose equivalent in ICRU (soft) tissue at an appropriate depth, d, below a specified point on the human body. The specified point is normally taken to be where the individual dosimeter is worn. For the assessment of effective dose, $H_p(10)$ with a depth $d = 10$ mm is chosen, and for the assessment of the dose to the skin and to the hands and feet the personal dose equivalent, $H_p(0.07)$, with a depth $d = 0.07$ mm, is used. A depth $d = 3$ mm has been proposed for the rare case of monitoring the dose to the lens of the eye. In practice, however, $H_p(3)$ has rarely been monitored and $H_p(0.07)$ can be used for the same monitoring purpose. Operational quantities are measurable, and instruments for radiation monitoring are calibrated in terms of these quantities. In routine monitoring, the values of these operational quantities are taken as a sufficiently precise assessment of effective dose and skin dose, respectively, in particular, if their values are below the protection limits.

(137) No operational quantities have been defined which provide a direct assessment of equivalent or effective dose for internal dosimetry. In general, various measurements of incorporated radionuclides are performed and biokinetic models are used in order to estimate the intake of radionuclides. From the intake, equivalent or effective dose is calculated by using reference dose coefficients (doses per unit intake, Sv Bq^{-1}) recommended by the Commission (see Annex B).

4.4. Assessment of radiation exposure

4.4.1. External radiation exposure

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

4.4.2. Internal radiation exposure

(139) The system of dose assessment for intakes of radionuclides relies on the calculation of the intake of a radionuclide, which can be considered as an operational quantity for the dose assessment from internal exposure. The intake can be estimated either from direct measurements (e.g., external monitoring of the whole body or of specific organs and tissues) or indirect measurements (e.g., urine or faeces), or measurements on environmental samples, and the application of biokinetic models. The effective dose is then calculated from the intake using dose coefficients recommended by the Commission for a large number of radionuclides. Dose coefficients are given for members of the public of various ages and for adults who are occupationally exposed.

(140) Radionuclides incorporated in the human body irradiate the tissues over time periods determined by their physical half-life and their biological retention within the body. Thus they may give rise to doses to body tissues for many months or years after the intake. The need to regulate exposures to radionuclides and the accumulation of radiation dose over extended periods of time has led to the definition of committed dose quantities. The committed dose from an incorporated radionuclide is the total dose expected to be delivered within a specified time period. The *committed equivalent dose*, $H_{\mathrm{T}}(\tau)$, in a tissue or organ T is defined by:

$$H_{\mathrm{T}}(\tau) = \int_{t_0}^{t_0+\tau} \dot{H}_{\mathrm{T}}(t)\mathrm{d}t \tag{4.7}$$

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

$$E(\tau) = \sum_{\mathrm{T}} w_{\mathrm{T}} H_{\mathrm{T}}(\tau) \tag{4.8}$$

(141) For compliance with dose limits, the Commission continues to recommend that the committed dose is assigned to the year in which the intake occurred. For workers, the committed dose is normally evaluated over the 50-year period following the intake. The commitment period of 50 years is a rounded value considered by the Commission to be the working-life expectancy of a young person entering the workforce. The committed effective dose from intakes of radionuclides is also used in prospective dose estimates for members of the public. In these cases, a commitment period of 50 years is recommended for adults. For infants and children, the dose is evaluated to the age of 70 years.

(142) The effective dose from occupational intakes of radionuclides is assessed on the basis of the worker's intake and the reference dose coefficient. The calculations of dose coefficients for specified radionuclides (Sv Bq^{-1}) use defined biokinetic and dosimetric models. Models are used to describe the entry of various chemical forms of radionuclides into the body and their distribution and retention after entering the blood. The computational male and female phantoms are also used to compute, for a series of sources, the fraction of the energy emitted from a source region S that is

absorbed in target region T. These approximations are considered to be adequate for the main tasks in radiological protection.

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

$$e(\tau) = \sum_{\mathrm{T}} w_{\mathrm{T}} \left[\frac{h_{\mathrm{T}}^{\mathrm{M}}(\tau) + h_{\mathrm{T}}^{\mathrm{F}}(\tau)}{2} \right] \tag{4.9}$$

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

4.4.3. Occupational exposure

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

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

$$E \cong H_{\mathrm{p}}(10) + E(50) \tag{4.10}$$

where $H_{\mathrm{p}}(10)$ is the personal dose equivalent from external exposure and $E(50)$, the committed effective dose from internal exposure, which is assessed by:

$$E(50) = \sum_{\mathrm{j}} e_{\mathrm{j,inh}}(50) \cdot I_{\mathrm{j,inh}} + \sum_{\mathrm{j}} e_{\mathrm{j,ing}}(50) \cdot I_{\mathrm{j,ing}} \tag{4.11}$$

where $e_{\mathrm{j,inh}}(50)$ is the committed effective dose coefficient for activity intakes by inhalation of a radionuclide j, $I_{\mathrm{j,inh}}$ is the activity intake of a radionuclide j by inhalation, $e_{\mathrm{j,ing}}(50)$ is the committed effective dose coefficient for activity intakes of a radionuclide j by ingestion, and $I_{\mathrm{j,ing}}$ is the activity intake of a radionuclide j by ingestion. In the calculation of the effective dose from specific radionuclides, allowance may need to be made for the characteristics of the material taken into the body.

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

(146) The dose coefficients used in Eqn. (4.11) are those specified by the Commission with no departure from the anatomical, physiological, and biokinetic characteristics of the Reference Male and the Reference Female (ICRP, 2002). Account may be taken of the physical and chemical characteristics of the intake, including the activity median aerodynamic diameter (AMAD) of the inhaled aerosol and the chemical form of the particulate matter to which the specified radionuclide is attached. The effective dose assigned in the worker's dose record is that value which the Reference Person would experience owing to the radiation fields and activity intakes encountered by the worker. The commitment period of 50 years represents the period of possible dose accumulation over a working life (this is only relevant for radionuclides with long physical half-lives and long retention in body tissues).

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

(148) In the rare case of a significant contribution to external exposure of weakly-penetrating radiation, the contribution of the skin dose to the effective dose needs to be considered in addition to the terms given in Eq. (4.10) for the assessment of effective dose (see Annex B). The radiation dose from radon isotopes, primarily radon-222, and their decay products may also need to be taken into account in the overall dose assessment (ICRP 1993a).

(149) In certain situations where individual monitoring with personal dosimeters is not performed, such as exposure of aircrew, an assessment of effective dose may be obtained from values of the quantity ambient dose equivalent, $H^*(10)$. Effective dose in then calculated using appropriate factors derived from data on the radiation field, or by calculating effective dose directly from these data.

4.4.4. Public exposure

(150) The basic principles of estimation of effective doses are the same for members of the public as for workers. The annual effective dose to members of the public is the sum of the effective dose obtained within one year from external exposure and the committed effective dose from radionuclides incorporated within this year. The dose is not obtained by direct measurement of individual exposures as for occupational exposure but is mainly determined by effluent and environmental measurements, habit data, and modelling. The component due to discharges of radioactive effluents can be estimated by effluent monitoring for existing installations or prediction of effluents from the installation or source during the design period. Information on concentrations of radionuclides in effluents and the environment are used in conjunction with radioecological modelling (pathway analysis of environmental transport, through air, water, soil, sediments, plants, and animals to humans) to assess doses from external radiation exposure and intakes of radionuclides (see Annex B).

4.4.5. Medical exposure of patients

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

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

4.4.6. Application of the effective dose

(153) The main and primary uses of effective dose in radiological protection for both occupational workers and the general public are:

- prospective dose assessment for planning and optimisation of protection; and
- retrospective dose assessment for demonstrating compliance with dose limits, or for comparing with dose constraints or reference levels.

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

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

(156) In retrospective assessments of doses to specified individuals that may substantially exceed dose limits, effective dose can provide a first approximate measure of the overall detriment. If radiation dose and risk need to be assessed in a more

accurate way, further specific estimates of organ or tissue doses are necessary, especially if organ-specific risks for the specified individuals are needed.

(157) Effective dose is intended for use as a protection quantity on the basis of reference values and therefore is not recommended for epidemiological evaluations, nor should it be used for detailed specific retrospective investigations of individual exposure and risk. Rather, absorbed dose should be used with the most appropriate biokinetic biological effectiveness and risk factor data. Organ or tissue doses, not effective doses, are required for assessing the probability of cancer induction in exposed individuals.

(158) The use of effective dose is inappropriate for the assessment of tissue reactions. In such situations it is necessary to estimate absorbed dose and to take into account the appropriate RBE as the basis for any assessment of radiation effects (see Annex B).

4.4.7. Collective effective dose

(159) For the purpose of optimisation of radiological protection, predominantly in the context of occupational exposure, the Commission has introduced collective dose quantities (ICRP 1977, 1991b). These quantities take account of the exposure of all individuals in a group over a given time period or during a given operation executed by this group in designated radiation areas. In practice, the collective equivalent dose is used only in special circumstances. The Commission therefore discusses only the collective effective dose quantity in these Recommendations. The collective effective dose, S (ICRP, 1991b) is calculated as the sum of all individual effective doses over the time period or during the operation being considered. The special name used for the collective effective dose quantity is the 'man sievert'. In the optimisation process, different radiological protection measures and operational scenarios are compared in terms of assessments of expected individual and collective effective doses.

(160) The collective effective dose, S, is based on the assumption of a linear dose effect relationship for stochastic effects without a threshold (the LNT model). On this basis it is possible to regard effective doses as additive.

(161) Collective effective dose is an instrument for optimisation, for comparing radiological technologies and protection procedures. Collective effective dose is not intended as a tool for epidemiological studies, and it is inappropriate to use it in risk projections. This is because the assumptions implicit in the calculation of collective effective dose (e.g., when applying the LNT model) conceal large biological and statistical uncertainties. Specifically, the computation of cancer deaths based on collective effective doses involving trivial exposures to large populations is not reasonable and should be avoided. Such computations based on collective effective dose were never intended, are biologically and statistically very uncertain, presuppose a number of caveats that tend not to be repeated when estimates are quoted out of context, and are an incorrect use of this protection quantity.

(162) To avoid inappropriate aggregation of, e.g., very low individual doses over extended time periods and wide geographical regions, limiting conditions need to be

set. The dose range and the time period should be stated. The collective effective dose due to individual effective dose values between E_1 and E_2 is defined as:

$$S(E_1, E_2, \Delta T) = \int_{E_1}^{E_2} E\left(\frac{\mathrm{d}N}{\mathrm{d}E}\right)_{\Delta T} \mathrm{d}E \tag{4.12}$$

where $(\mathrm{d}N/\mathrm{d}E)\mathrm{d}E$ denotes the number of individuals who are exposed to an effective dose between E and $E + \mathrm{d}E$ within the time period ΔT (see Annex B). When the range of individual doses spans several orders of magnitude, the distribution should be characterised by dividing it into several ranges of individual dose, each covering no more than two or three orders of magnitude, with the population size, mean individual dose, and uncertainty being considered separately for each range. When the collective effective dose is smaller than the reciprocal of the relevant risk detriment, the risk assessment should note that the most likely number of excess health effects is zero (NCRP 1995).

4.5. Uncertainties and judgements

(163) In the evaluation of radiation doses, models are necessary to simulate the geometry of the external exposure, the biokinetics of the intake and retention of radionuclides in the human body, and the human anatomy. In many cases, these models and their parameter values have been developed from experimental investigations and human studies in order to derive 'best estimates' or 'central estimates' of model parameter values. Similar considerations apply to the choice of tissue and radiation weighting factors. It is recognised that there are appreciable uncertainties in the values of some of the parameters and in the formulation or structures of the models themselves. Judgement is needed on the best choice of the necessary models and parameter values for dose assessments (see Annex B).

(164) Uncertainty refers to the level of confidence that can be placed in a given parameter value or prediction of a model. It is an important factor in all extrapolation procedures. In this connection the variability of individual parameters and the accuracy of measurements are also of great importance. The accuracy of measurements and judgements will become less with decreasing doses and increasing complexity of the system. Variability refers to quantitative differences between individual members of the population in question. All these aspects are taken into account in model development in the judgements made (see Annex B).

(165) The lack of certainty or precision in radiation dose models varies for the various parameters and the circumstances in defined situations. Therefore it is not possible to give values for the uncertainties across the range of ICRP models, despite the fact that their assessment is an important part of model development. Uncertainties may need to be assessed, however, for special cases, and approaches to their use have been described in a number of publications, e.g., Goossens et al. (1997), CERRIE (2004), ICRP (1994b, 2005d), Bolch et al. (2003), and Farfan et al. (2005). In general, it can be said that uncertainties of assessments of radiation doses from internal exposures, including the biokinetics of radionuclides, are larger than those from external exposures. The degree of uncertainty differs between various radionuclides.

(166) The Commission is aware of the uncertainty or lack of precision in radiation dose models and efforts are undertaken to critically evaluate and to reduce them wherever possible. For regulatory purposes, the dosimetric models and parameter values that the Commission recommends are reference values. These are fixed by convention and are therefore not subject to uncertainty. Equally the Commission considers that the biokinetic and dosimetric models which are needed for the purpose of dose assessment are defined as reference data and, therefore, are also fixed and not applied with an uncertainty. These models and values are re-evaluated periodically and may be changed by ICRP on the basis of such evaluations when new scientific data and information are available.

(167) Regulatory compliance is determined using point estimates of effective dose that apply to a Reference Person, regarding these point estimates as subject to no uncertainties. In retrospective assessments of doses that may approach or exceed limits, it may be considered appropriate to make specific individual estimates of dose and risk, and also to consider uncertainties in these estimates.

(168) Despite changes in dosimetric modelling, as well as differences in the computation of effective dose, previous assessments of equivalent dose or effective dose should be considered adequate. In general, the Commission does not recommend re-computation of existing values with the new models and parameters.

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5. THE SYSTEM OF RADIOLOGICAL PROTECTION OF HUMANS

(169) Everybody is exposed to ionising radiation from natural and man-made sources. It is convenient to think of the processes causing these human exposures as a *network of events and situations*. Each part of the network starts from a source. Radiation or radioactive material then passes through environmental or other pathways leading to the exposure of individuals. Finally, the exposure of individuals to radiation or radioactive materials leads to doses to these individuals. Protection can be achieved by taking action at the source, or at points in the exposure pathways, and occasionally by modifying the location or characteristics of the exposed individuals. For convenience, the environmental pathway is usually taken to include the link between the source of exposure and the doses received by the individuals. The available points of action have a substantial effect on the system of protection.

(170) The assumed proportional relationship between an increment of dose and an increment of risk of stochastic effects makes it possible to deal separately with different parts of this network of events and situations leading to exposure, and to select those parts that are of relevance in a given situation. To make these selections, however, it is necessary to define, for each part of the network, the objectives, the organisations (and individuals) responsible for protection, the lines of responsibility, and the feasibility of obtaining the necessary information. This remains a complex procedure, and the Commission suggests two simplifications in managing radiological situations.

(171) The first simplification was used in the 1990 Recommendations and recognises that individuals are subject to several categories of exposure, which can be dealt with separately (ICRP, 1991b). For example, most workers who are exposed to radiation sources as part of their work are also exposed to environmental sources as members of the public, and to medical exposure as patients. The Commission's policy continues to be that the control of exposures due to work need not be influenced by the exposures from these other sources. This policy is still generally reflected in the present Recommendations by the separation of the exposure into three categories (see Section 5.3): occupational exposure, medical exposure of patients, and public exposure. The Commission continues to recommend that, for regulatory purposes, no attempt be made to add the exposures to the same individual from the different categories of exposure.

(172) The second simplification is that, in dealing with the network constituting a variety of exposure pathways, a distinction is drawn between source-related considerations and individual-related considerations (see Section 5.5). Although within each category of exposure individuals can be exposed to several sources, for the purposes of radiological protection each source, or group of sources, can be treated on its own (ICRP, 1991b). It is then necessary to consider the exposure of all the individuals who could be exposed by this source or group of sources. This procedure is called a 'source-related assessment'.

(173) For the practical control of exposures, in *Publication 60* the network of events and situations causing these exposures was divided into two broad classes

of situations: practices and interventions. Practices were defined as human actions increasing exposure either by introducing whole new blocks of sources, pathways, and individuals, or by modifying the network of pathways from existing sources to individuals and thus increasing the exposure of individuals or the number of individuals so exposed. Interventions were defined as human actions that 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. In the revised system of protection the Recommendations of the Commission have now evolved from a process-based approach to an approach based on the characteristics of three types of radiation exposure situation, i.e., planned, emergency, and existing exposure situations (see Section 5.2).

5.1. The definition of a source

(174) The Commission uses the term 'source' to indicate any physical entity or procedure that results in a potentially quantifiable radiation dose to a person or group of persons. It can be a physical source (e.g., radioactive material or an x-ray machine), an installation (e.g., a hospital or a nuclear power plant), or procedures or groups of physical sources having similar characteristics (e.g., nuclear medicine procedures or background or environmental radiation). If radioactive substances are released from an installation into the environment, the installation as a whole may be regarded as a source; if they are already dispersed in the environment, the portion of them to which people are exposed may be considered a source. Most situations will give rise to a predominant source of exposure for any single individual, making it possible to treat sources singly when considering actions.

(175) In general, the definition of a source will be linked to the selection of the relevant protection strategy, as appropriate, for optimisation. Difficulties will arise if the policy is distorted, e.g., by artificially subdividing a source in order to avoid the need for protective action, or by excessively aggregating sources to exaggerate the need for action. Provided that the regulatory authority and the user (where one can be defined) both apply the spirit of the Commission's broad policies, practical agreements can be reached on the definition of a source.

5.2. Types of exposure situations

(176) The Commission intends its Recommendations to be applied to all sources and to individuals exposed to radiation in the following three types of exposure situations which address all conceivable circumstances.

- *Planned exposure situations* are situations involving the deliberate introduction and operation of sources. Planned exposure situations may give rise both to exposures that are anticipated to occur (normal exposures) and to exposures that are not anticipated to occur (potential exposures; see Section 6.1.3).
- *Emergency exposure situations* are situations that may occur during the operation of a planned situation, or from a malicious act, or from any other unexpected

situation, and require urgent action in order to avoid or reduce undesirable consequences.

- *Existing exposure situations* are exposure situations that already exist when a decision on control has to be taken, including prolonged exposure situations after emergencies.

It follows that what the Commission has called 'practices' could be the origin of planned, emergency, and existing exposure situations. Medical exposures of patients are also planned exposure situations, but because of the characteristics of such exposures, they are discussed separately. The principles of protection for planned situations also apply to occupational exposure in connection with existing and emergency exposure situations.

5.3. Categories of exposure

(177) The Commission distinguishes between three categories of exposures: occupational exposures, public exposures, and medical exposures of patients. Exposures of comforters and carers, and exposures of volunteers in research, are discussed in Chapter 7.

5.3.1. Occupational exposure

(178) Occupational exposure is defined by the Commission as all radiation exposure of workers incurred as a result of their work. The Commission has noted the conventional definition of occupational exposure to any hazardous agent as including all exposures 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 'occupational exposures' to radiation exposures incurred at work as a result of situations that can reasonably be regarded as being the responsibility of the operating management (see also Section 6.3.1). Excluded exposures and exposures from exempt practices or exempt sources generally do not need to be accounted for in occupational protection.

(179) The employer has the main responsibility for the protection of workers. However, the licensee responsible for the source (if not identical to the employer) also has a responsibility for the radiological protection of workers. If workers are engaged in work that involves, or could involve, a source that is not under the control of their employer, the licensee and the employer should co-operate by the exchange of information and otherwise as necessary to facilitate proper radiological protection at the workplace.

5.3.2. Public exposure

(180) Public exposure encompasses all exposures of the public other than occupational exposures and medical exposures of patients (see Section 5.3.3). It is incurred

as a result of a range of radiation sources. 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 controllable, exposures to man-made sources. Exposures of the embryo and fetus of pregnant workers are considered and regulated as public exposures.

5.3.3. Medical exposure of patients

(181) Radiation exposures of patients occur in diagnostic, interventional, and therapeutic procedures. There are several features of radiological practices in medicine that require an approach that differs from the radiological protection in other planned exposure situations. The exposure is intentional and for the direct benefit of the patient. Particularly in radiotherapy, the biological effects of high-dose radiation, e.g., cell killing, are used for the benefit of the patient to treat cancer and other diseases. The application of these Recommendations to the medical uses of radiation therefore requires separate guidance (see Chapter 7, which also discusses the medical exposure of comforters and carers and of volunteers in research).

5.4. The identification of the exposed individuals

(182) It is necessary to deal separately with at least three categories of exposed individuals, namely workers, the public, and patients. They essentially correspond to individuals whose exposures fall into the three categories of exposure defined in Section 5.3. A given individual may be exposed as a worker, and/or as a member of the public, and/or as a patient.

5.4.1. Workers

(183) A worker is defined by the Commission as any person who is employed, whether full time, part time, or temporarily, by an employer and who has recognised rights and duties in relation to occupational radiological protection. A self-employed person is regarded as having the duties of both an employer and a worker. Workers in medical professions involving radiation are occupationally exposed.

(184) One important function of an employer and/or licensee is that of maintaining control over the sources of exposure and over the protection of workers who are occupationally exposed. In order to achieve this, the Commission continues to recommend the classification of areas of work rather than the classification of workers. Requiring that the areas of workplaces containing sources be formally designated helps their control. The Commission uses two such designations: *controlled areas* and *supervised areas*. A controlled area is a defined area in which specific protection measures and safety provisions are, or could be, required for controlling normal exposures or preventing the spread of contamination during normal working conditions, and preventing or limiting the extent of potential exposures. A supervised area is one in which the working conditions are kept under review but special procedures

are not normally needed. A controlled area is often within a supervised area, but need not be.

(185) Workers in 'controlled areas' of workplaces should be well informed and specially trained, and form a readily identifiable group. Such workers are most often monitored for radiation exposures incurred in the workplace, and occasionally may receive special medical surveillance.

The exposure of pregnant or breast-feeding workers

(186) In the 1990 Recommendations, the Commission concluded that, for the purpose of controlling occupational exposure, there was no reason to distinguish between the two sexes. The Commission maintains this policy with these Recommendations. However, if a female worker has declared (i.e., notified her employer) that she is pregnant, additional controls have to be considered to protect the embryo/fetus. It is the Commission's policy that the methods of protection at work for women who are pregnant should provide a level of protection for the embryo/fetus broadly similar to that provided for members of the public. The Commission considers that this policy will be adequately applied if the mother is exposed, prior to her declaration of pregnancy, under the system of protection recommended by the Commission. Once an employer has been notified of a pregnancy, additional protection of the embryo/fetus should be considered. The working conditions of a pregnant worker, after declaration of pregnancy, should be such as to ensure that the additional dose to the embryo/fetus would not exceed about 1 mSv during the remainder of the pregnancy. Additional guidance on protection of an embryo/fetus exposed to radiation is provided in Section 7.4.

(187) The restriction of the dose to the embryo/fetus does not mean that it is necessary for pregnant women to avoid work with radiation or radioactive materials completely, or that they must be prevented from entering or working in designated radiation areas (see paragraph 184). It does, however, imply that the employer should carefully review the exposure conditions of pregnant women. In particular, if required, their working conditions should be changed such that, during pregnancy, the probability of accidental doses and radionuclide intakes is extremely low. Specific recommendations on the control of exposures to pregnant workers are given in *Publications 84* and *88* (ICRP, 2000a, 2001a). The Commission has also published information in *Publication 95* (ICRP, 2004c) that enables doses to offspring following intakes to breast-feeding mothers to be calculated. The Commission strongly recommends that in order to protect the embryo/fetus or infant, females who have declared that they are pregnant or are nursing should not be involved in emergency actions involving high radiation doses (ICRP, 2005a).

(188) In *Publication 88* (ICRP, 2001a), the Commission gave dose coefficients for the embryo, fetus, and newborn child from intakes of radionuclides by the mother before or during pregnancy. In general, doses to the embryo, fetus, and newborn child are similar to or less than those to the Reference Female. In *Publication 95* (ICRP, 2004c) the Commission provided information on radiation doses to the breast-feeding infant due to intakes of radionuclides in maternal milk. For most of the radionuclides

considered, doses to the infant from radionuclides ingested in breast milk are estimated to be small in comparison with doses to the Reference Female.

Exposures in aviation and in space

(189) In *Publication 60* (ICRP, 1991b), the Commission recommended that exposures to cosmic radiation be part of occupational exposure in the operation of commercial jet aircraft and space flight. The Commission subsequently clarified its recommendation in *Publication 75* (ICRP, 1997a), indicating that it is not necessary to treat the exposure of frequent-flyer passengers as occupationally exposed for the purpose of control. Thus, essentially, only aircrew should be considered. At that time, the Commission had already noted that the only practical regulatory measures were controlling individual exposure through the control of flying time and route selection. The Commission maintains this view.

(190) Exceptional cases of cosmic radiation exposures, such as exposure in space travel, where doses may be significant and some type of control warranted, should be dealt with separately, taking into account the special type of situations that can give rise to this type of exposure.

5.4.2. Members of the public

(191) A member of the public is defined by the Commission as any individual who receives an exposure that is neither occupational nor medical (see also Section 5.4.3). A large range of different natural and man-made sources contribute to the exposure of members of the public.

(192) In general, especially for public exposure, each source will result in a distribution of doses over many individuals. For the purposes of protection of the public, the Commission has used the 'critical group' concept to characterise an individual receiving a dose that is representative of the more highly exposed individuals in the population (ICRP, 1977). Dose restrictions have been applied to the mean dose in the appropriate critical group. Over the past decades, a considerable body of experience has been gained in the application of the critical group concept. There have also been developments in the techniques used to assess doses to members of the public, notably the increasing use of probabilistic techniques. The adjective 'critical' has the connotation of a crisis, which was never intended by the Commission. Furthermore, the word 'group' may be confusing in the context where the assessed dose is to an individual.

(193) The Commission now recommends the use of the 'Representative Person' for the purpose of radiological protection of the public instead of the earlier critical group concept. The Commission provides guidance on characterising the Representative Person and assessing doses to the Representative Person in *Publication 101* (ICRP, 2006a).

(194) The Representative Person may be hypothetical, but it is important that the habits (e.g., consumption of foodstuffs, breathing rate, location, usage of local resources) used to characterise the Representative Person are typical habits of a small number of individuals representative of those most highly exposed and not the extreme habits of a single member of the population. Consideration may be given

to some extreme or unusual habits, but they should not dictate the characteristics of the Representative Persons considered.

5.4.3. Patients

(195) The Commission defines the patient as an individual who receives an exposure associated with a diagnostic, interventional, or therapeutic procedure. The Commission's dose limits and dose constraints are not recommended for individual patients because they may reduce the effectiveness of the patient's diagnosis or treatment, thereby doing more harm than good. The emphasis is therefore on the justification of the medical procedures and on the optimisation of protection and, for diagnostic procedures, the use of diagnostic reference levels (see Chapter 7).

(196) The exposure of patients who are pregnant is dealt with in Section 7.4.

5.5. Levels of radiological protection

(197) In the 1990 Recommendations it was noted that, provided that individual doses are well below the thresholds for harmful deterministic effects, the effect of a contribution to an individual dose from a source is independent of the effects of doses from other sources. For many purposes, each source or group of sources could usually be treated on its own. It is then necessary to consider the exposure of individuals exposed by this source or group of sources. This procedure is called a 'source-related' approach. The Commission now emphasises the primary importance of the source-related approach, because action can be taken on a source to assure the protection of a group of individuals from that source.

(198) For planned exposure situations, the source-related restriction to the dose that individuals may incur is the *dose constraint*. For potential exposures, the corresponding concept is the *risk constraint.* For emergency and existing exposure situations, the source-related restriction is the *reference level* (see Sections 5.9, 6.2, and 6.3). The concepts of a dose constraint and reference level are used in the process of optimisation of protection to assist in ensuring that all exposures are kept as low as reasonably achievable, societal and economic factors being taken into account. Constraints and reference levels can thus be described as key parts in the optimisation process that will ensure appropriate levels of protection under the prevailing circumstances.

(199) It could be argued that the source-related restriction would not provide sufficient protection where there are multiple sources. However, the Commission presumes that there will generally be a dominant source, and the selection of the appropriate reference level or constraint ensures an adequate level of protection. The Commission still considers that the source-related principle of optimisation below the constraint or reference level is the most effective tool for protection, whatever the situation.

(200) In the specific case of planned exposure situations, separate restrictions on the sums of the occupational doses and on the sums of the public doses are required. The Commission refers to such individual-related restrictions as dose limits (see Section 5.10) and the corresponding assessment of doses is called 'individual-related'.

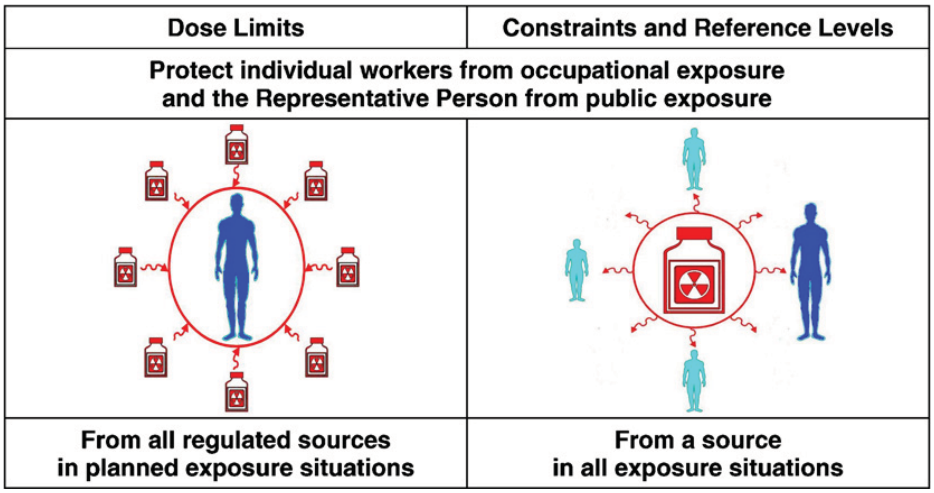

Fig. 3. Dose limits contrasted with dose constraints and reference levels for protecting workers and members of the public.

(201) It is rarely possible, however, to assess the total exposure of an individual from all such sources. It is therefore necessary to make approximations to the dose to be compared with the quantitative limit, especially in the case of public exposure. For occupational exposures, the approximations are more likely to be accurate because the operating management has access to the necessary information to identify and control the dose from all the relevant sources.

(202) Figure 3 illustrates the differences in concept between the use of individual dose limits in planned situations and constraints or reference levels for protection from a source in all situations.

5.6. The principles of radiological protection

(203) In the 1990 Recommendations, the Commission gave principles of protection for practices separately from intervention situations. The Commission continues to regard these principles as fundamental for the system of protection, and has now formulated a single set of principles that apply to planned, emergency, and existing exposure situations. In these Recommendations, the Commission also clarifies how the fundamental principles apply to radiation sources and to the individual, as well as how the source-related principles apply to all controllable situations.

Two principles are source-related and apply in all exposure situations

- **The principle of justification:** Any decision that alters the radiation exposure situation should do more good than harm.

This means that, by introducing a new radiation source, by reducing existing exposure, or by reducing the risk of potential exposure, one should achieve sufficient individual or societal benefit to offset the detriment it causes.

- **The principle of optimisation of protection:** the likelihood of incurring exposures, the number of people exposed, and the magnitude of their individual doses should all be kept as low as reasonably achievable, taking into account economic and societal factors.

This means that the level of protection should be the best under the prevailing circumstances, maximising the margin of benefit over harm. In order to avoid severely inequitable outcomes of this optimisation procedure, there should be restrictions on the doses or risks to individuals from a particular source (dose or risk constraints and reference levels).

One principle is individual-related and applies in planned exposure situations

- **The principle of application of dose limits:** The total dose to any individual from regulated sources in planned exposure situations other than medical exposure of patients should not exceed the appropriate limits recommended by the Commission.

(204) Regulatory dose limits are determined by the regulatory authority, taking account of international recommendations, and apply to workers and to members of the public in planned exposure situations.

5.7. Justification

(205) The Commission recommends that, when activities involving an increased or decreased level of radiation exposure, or a risk of potential exposure, are being considered, the expected change in radiation detriment should be explicitly included in the decision-making process. The consequences to be considered are not confined to those associated with the radiation – they include other risks and the costs and benefits of the activity. Sometimes, the radiation detriment will be a small part of the total. Justification thus goes far beyond the scope of radiological protection. It is for these reasons that the Commission only recommends that justification require that the net benefit be positive. To search for the best of all the available alternatives is a task beyond the responsibility of radiological protection authorities.

5.7.1. Application of the principle of justification

(206) There are two different approaches to applying the principle of justification in situations involving occupational and public exposure, which depend upon whether or not the source can be directly controlled. The first approach is used in the introduction of new activities where radiological protection is planned in advance and the necessary actions can be taken on the source. Application of the justification principle to these situations requires that no planned exposure situation should be introduced unless it produces sufficient net benefit to the exposed individuals or to society to offset the radiation detriment it causes. Judgements on whether it would be justifiable to introduce or continue particular types of planned situation

involving exposure to ionising radiation are important. The justification may need to be re-examined as new information or technology becomes available.

(207) The second approach is used where exposures can be controlled mainly by action to modify the pathways of exposure and not by acting directly on the source. The main examples are existing exposure situations and emergency exposure situations. In these circumstances, the principle of justification is applied in making the decision as to whether to take action to avert further exposure. Any decision taken to reduce doses, which always have some disadvantages, should be justified in the sense that they should do more good than harm.

(208) In both approaches, the responsibility for judging the justification usually falls on governments or national authorities to ensure an overall benefit in the broadest sense to society and thus not necessarily to each individual. However, input to the justification decision may include many aspects that could be informed by users or other organisations or persons outside of government. As such, justification decisions will often be informed by a process of public consultation, depending upon, among other things, the size of the source concerned. There are many aspects of justification, and different organisations may be involved and responsible. In this context, radiological protection considerations will serve as one input to the broader decision process.

(209) Medical exposure of patients calls for a different and more detailed approach to the process of justification. The medical use of radiation should be justified, as is any other planned exposure situation, although that justification lies more often with the profession than with government or the competent regulatory authority. The principal aim of medical exposures is to do more good than harm to the patient, due account being taken of the radiation detriment from the exposure of the radiological staff and of other individuals. The responsibility for the justification of the use of a particular procedure falls on the relevant medical practitioners, who need to have special training in radiological protection. Justification of medical procedures therefore remains part of the Commission's Recommendations (see Section 7.1).

5.7.2. Unjustified exposures

(210) The Commission considers that certain exposures should be deemed to be unjustified without further analysis, unless there are exceptional circumstances. These include the following:

- Increasing, by deliberate addition of radioactive substances or by activation, the activity of products such as food, beverages, cosmetics, toys, and personal jewellery or adornments.
- Radiological examination for occupational, health insurance, or legal purposes undertaken without reference to clinical indications, unless the examination is expected to provide useful information on the health of the individual examined or in support of important criminal investigations. This almost always means that a clinical evaluation of the image acquired must be carried out, otherwise the exposure is not justified.

- Medical screening of asymptomatic population groups involving radiation exposure, unless the expected advantages for the individuals examined or for the population as a whole are sufficient to compensate for the economic and societal costs, including the radiation detriment. Account should be taken of the potential of the screening procedure for detecting disease, the likelihood of effective treatment of cases detected, and, for certain diseases, the advantages to the community of control of the disease.

5.8. Optimisation of protection

(211) The process of optimisation of protection is intended for application to those situations that have been deemed to be justified. The principle of optimisation of protection, with restriction on the magnitude of individual dose or risk, is central to the system of protection and applies to all three exposure situations: planned exposure situations, emergency exposure situations, and existing exposure situations.

(212) The principle of optimisation is defined by the Commission as the source-related process to keep the likelihood of incurring exposures (where these are not certain to be received), the number of people exposed, and the magnitude of individual doses as low as reasonably achievable, taking economic and societal factors into account.

(213) The Recommendations of the Commission on how to apply the optimisation principle have been provided earlier (ICRP, 1983, 1989, 1991b, and 2006a), and these Recommendations remain valid and will not be repeated in detail here. The decision-aiding techniques are still essential to find the optimised radiological protection solution in an objective manner; these techniques include methods for quantitative optimisation such as cost-benefit analyses. The process of optimisation over the past decades has resulted in substantial reductions of occupational and public exposures.

(214) Optimisation is always aimed at achieving the best level of protection under the prevailing circumstances through an ongoing, iterative process that involves:

- evaluation of the exposure situation, including any potential exposures (the framing of the process);
- selection of an appropriate value for the constraint or reference level;
- identification of the possible protection options;
- selection of the best option under the prevailing circumstances; and
- implementation of the selected option.

(215) Experience has shown how optimisation of protection has improved radiological protection for planned situations. Constraints provide a desired upper bound for the optimisation process. Some sources and technologies are able to satisfy constraints that are set at a low level, while others are only able to meet constraints set at a higher level. This is normal and should be reflected in the freedom of regulatory authorities and others, as appropriate, to select values that are appropriate for particular circumstances.

(216) In all situations, the process of optimisation with the use of constraints or reference levels is applied in planning protective actions and in establishing the

appropriate level of protection under the prevailing circumstances. The doses to be compared with the dose constraint or reference levels are usually prospective doses, i.e., doses that may be received in the future, as it is only those doses that can be influenced by decisions on protective actions. They are not intended as a form of retrospective dose limit.

(217) The optimisation of protection is a forward-looking iterative process aimed at preventing or reducing future exposures. It takes into account both technical and socio-economic developments and requires both qualitative and quantitative judgements. The process should be systematic and carefully structured to ensure that all relevant aspects are taken into account. Optimisation is a frame of mind, always questioning whether the best has been done in the prevailing circumstances, and whether all that is reasonable has been done to reduce doses. It also requires commitment at all levels in all concerned organisations as well as adequate procedures and resources.

(218) The best option is always specific to the exposure situation and represents the best level of protection that can be achieved under the prevailing circumstances. Therefore it is not relevant to determine, a priori, a dose level below which the optimisation process should stop. Depending on the exposure situation, the best option could be close to or well below the appropriate source-related constraint or reference level.

(219) Optimisation of protection is not minimisation of dose. Optimised protection is the result of an evaluation, which carefully balances the detriment from the exposure and the resources available for the protection of individuals. Thus the best option is not necessarily the one with the lowest dose.

(220) In addition to the reduction of the magnitude of individual exposures, a reduction of the number of exposed individuals should also be considered. The collective effective dose has been and remains a key parameter for optimisation of protection for workers. The comparison of protection options for the purpose of optimisation must entail a careful consideration of the characteristics of the individual exposure distribution within an exposed population.

(221) When exposures occur over large populations, large geographical areas, or long time periods, the total collective effective dose is not a useful tool for making decisions because it may aggregate information inappropriately and could be misleading for selecting protective actions. To overcome the limitations associated with collective effective dose, each relevant exposure situation must be carefully analysed to identify the individual characteristics and exposure parameters that best describe the exposure distribution among the concerned population for the particular circumstance. Such an analysis – by asking when, where and by whom exposures are received – results in the identification of various population groups with homogeneous characteristics for which collective effective doses can be calculated within the optimisation process, and for which an optimised protection strategy can be defined (see Section 4.4). In practical optimisation assessments, collective doses may often be truncated, because the assessments use the difference between the integrals defining the collective doses assigned to the various alternative protective options under consideration, rather than the full integrals (ICRP, 1983).

(222) In *Publications 77* and *81* (ICRP, 1997d, 1998b), the Commission recognised that both the individual doses and the size of the exposed population become increasingly uncertain as time increases. The Commission is of the opinion that in the decision-making process, owing to the increasing uncertainties, giving less weight to very low doses and to doses received in the distant future could be considered (see also Section 4.4.7). The Commission does not intend to give detailed guidance on such weighting, but rather stresses the importance of demonstrating in a transparent manner how any weighting has been carried out.

(223) All aspects of optimisation cannot be codified; rather, there should be a commitment by all parties to the optimisation process. Where optimisation becomes a matter for the regulatory authority, the focus should not be on specific outcomes for a particular situation, but rather on processes, procedures, and judgements. An open dialogue should be established between the authority and the operating management, and the success of the optimisation process will depend strongly on the quality of this dialogue.

(224) Societal values usually influence the final decision on the level of radiological protection. Therefore, while this report should be seen as providing decision-aiding recommendations mainly based on scientific considerations on radiological protection, the Commission's advice will be expected to serve as an input to a final (usually wider) decision-making process, which may include other societal concerns and ethical aspects, as well as considerations of transparency (ICRP, 2006a). This decision-making process may often include the participation of relevant stakeholders rather than radiological protection specialists alone.

5.9. Dose constraints and reference levels

(225) The concepts of *dose constraint* and *reference level* are used in conjunction with the optimisation of protection to restrict individual doses. A level of individual dose, either as a dose constraint or a reference level, always needs to be defined. The initial intention would be to not exceed, or to remain at, these levels, and the ambition is to reduce all doses to levels that are as low as reasonably achievable, economic and societal factors being taken into account.

(226) For the sake of continuity with its earlier Recommendations (ICRP, 1991b), the Commission retains the term 'dose constraint' for this level of dose in planned exposure situations (with the exception of medical exposure of patients). For emergency exposure situations and existing exposure situations, the Commission proposes the term 'reference level' to describe this level of dose. The difference in terminology between planned and other exposure situations (emergency and existing) has been retained by the Commission to express the fact that, in planned situations, the restriction on individual doses can be applied at the planning stage, and the doses can be forecast so as to ensure that the constraint will not be exceeded. With the other situations a wider range of exposures may exist, and the optimisation process may apply to initial levels of individual doses above the reference level.

Table 4. The dose constraints and reference levels used in the Commission's system of protection.

Type of situation	Occupational exposure	Public exposure	Medical exposure
Planned exposure	Dose limit Dose constraint	Dose limit Dose constraint	Diagnostic reference level[d] (Dose constraint[e])
Emergency exposure	Reference level[a]	Reference level	N.A.[b]
Existing exposure	N.A.[c]	Reference level	N.A.[b]

[a] Long-term recovery operations should be treated as part of planned occupational exposure.
[b] Not applicable.
[c] Exposures resulting from long-term remediation operations or from protracted employment in affected areas should be treated as part of planned occupational exposure, even though the source of radiation is 'existing'.
[d] Patients.
[e] Comforters, carers, and volunteers in research only (see Sections 7.6 and 7.7).

(227) Diagnostic reference levels are already being used in medical diagnosis (i.e., planned exposure situations) to indicate whether, in routine conditions, the levels of patient dose or administered activity from a specified imaging procedure are unusually high or low for that procedure. If so, a local review should be initiated to determine whether protection has been adequately optimised or whether corrective action is required.

(228) The chosen value for a constraint or a reference level will depend upon the circumstances of the exposure under consideration. It must also be realised that neither dose and risk constraints nor reference levels represent a demarcation between 'safe' and 'dangerous' or reflect a step change in the associated health risk for individuals.

(229) In Table 4 the different types of dose restrictions used in the Commission's system of protection (limits, constraints, reference levels) are shown in relation to type of exposure situation and category of exposure. In planned exposure situations, there are also risk constraints in order to take account of potential exposures.

5.9.1. Dose constraints

(230) A dose constraint is a prospective and source-related restriction on the individual dose from a source in planned exposure situations (except in medical exposure of patients), which serves as an upper bound on the predicted dose in the optimisation of protection for that source. It is a level of dose above which it is unlikely that protection is optimised for a given source of exposure, and for which, therefore, action must almost always be taken. Dose constraints for planned situations represent a basic level of protection and will always be lower than the pertinent dose limit. During planning it must be ensured that the source concerned does not imply doses exceeding the constraint. Optimisation of protection will establish an acceptable level of dose below the constraint. This optimised level then becomes the expected outcome of the planned protective actions.

(231) The action necessary if a dose constraint is exceeded includes determining whether protection has been optimised, whether the appropriate dose constraint has been selected, and whether further steps to reduce doses to acceptable levels would be appropriate. For potential exposures, the corresponding source-related restriction is called a risk constraint (see Section 6.1.3). Treating a dose constraint as a target value is not sufficient, and optimisation of protection will be necessary to establish an acceptable level of dose below the constraint.

(232) The concept of dose constraints was introduced in *Publication 60* as a means of ensuring that the optimisation process did not create inequity, i.e., the possibility that some individuals in an optimised protection scheme may be subject to much more exposure than the average:

> '*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 optimization. The Commission calls these source-related restrictions dose constraints, previously called upper bounds. They form an integral part of the optimization of protection. For potential exposures, the corresponding concept is the risk constraint*.' (ICRP, 1991b)

This statement continues to represent the Commission's view.

(233) For occupational exposures, the dose constraint is a value of individual dose used to limit the range of options such that only options expected to cause doses below the constraint are considered in the process of optimisation. For public exposure, the dose constraint is an upper bound on the annual doses that members of the public could receive from the planned operation of a specified controlled source. The Commission wishes to emphasise that dose constraints are not to be used or understood as prescriptive regulatory limits.

5.9.2. Reference levels

(234) In emergency or existing controllable exposure situations, the reference levels represent the level of dose or risk, above which it is judged to be inappropriate to plan to allow exposures to occur (cf. Section 6.2), and for which therefore protective actions should be planned and optimised. The chosen value for a reference level will depend upon the prevailing circumstances of the exposure situation under consideration.

(235) When an emergency exposure situation has occurred, or an existing exposure situation has been identified, and protective actions have been implemented, doses to workers and members of the public can be measured or assessed. The reference level may then assume a different function as a benchmark against which protection options can be judged retrospectively. The distribution of doses that has resulted from

the implementation of a planned protective strategy may or may not include exposures above the reference level, depending on the success of the strategy. Efforts should, however, be aimed at reducing any exposures that are above the reference level to a level that is below, if possible.

5.9.3. Factors influencing the choice of source-related dose constraints and reference levels

(236) At doses higher than 100 mSv, there is an increased likelihood of deterministic effects and a significant risk of cancer. For these reasons, the Commission considers that the maximum value for a reference level is 100 mSv incurred either acutely or in a year. Exposures above 100 mSv incurred either acutely or in a year would be justified only under extreme circumstances, either because the exposure is unavoidable or in exceptional situations such as the saving of life or the prevention of a serious disaster. No other individual or societal benefit would compensate for such high exposures (see ICRP, 2005a).

(237) Many of the numerical criteria recommended by the Commission in *Publication 60* and subsequent publications can be, with the exception of the limits, regarded as constraints or reference levels. The values fall into three defined bands (see Table 5) with the attributes described in the following paragraphs. The Commission considers that it is useful to present these values in this manner as it enables selection of an appropriate value for a constraint or a reference level for a specific situation that has not been addressed explicitly by the Commission.

(238) The Commission's banding of constraints and reference levels (see Table 5) applies across all three exposure situations and refers to the projected dose over a time period that is appropriate for the situation under consideration. Constraints for planned exposures and reference levels in existing situations are conventionally expressed as an annual effective dose (mSv in a year). In emergency situations the reference level will be expressed as the total residual dose to an individual as a result of the emergency that the regulator would plan not to exceed, either acute (and not expected to be repeated) or, in case of protracted exposure, on an annual basis.

(239) The first band, 1 mSv or less, applies to exposure situations where individuals receive exposures – usually planned – that may be of no direct benefit to them but the exposure situation may be of benefit to society. The exposure of members of the public from the planned operation of practices is a prime example of this type of situation. Constraints and reference levels in this band would be selected for situations where there is general information and environmental surveillance or monitoring or assessment and where individuals may receive information but no training. The corresponding doses would represent a marginal increase above the natural background and are at least two orders of magnitude lower than the maximum value for a reference level, thus providing a rigorous level of protection.

(240) The second band, greater than 1 mSv but not more than 20 mSv, applies in circumstances where individuals receive direct benefits from an exposure situation. Constraints and reference levels in this band will often be set in circumstances where

Table 5. Framework for source-related dose constraints and reference levels with examples of constraints for workers and the public from single dominant sources for all exposure situations that can be controlled.

Bands of constraints and reference levels[a] (mSv)	**Characteristics of the exposure situation**	**Radiological protection requirements**	**Examples**
Greater than 20 to 100[b,c]	Individuals exposed by sources that are not controllable, or where actions to reduce doses would be disproportionately disruptive. Exposures are usually controlled by action on the exposure pathways.	Consideration should be given to reducing doses. Increasing efforts should be made to reduce doses as they approach 100 mSv. Individuals should receive information on radiation risk and on the actions to reduce doses. Assessment of individual doses should be undertaken.	Reference level set for the highest planned residual dose from a radiological emergency.
Greater than 1 to 20	Individuals will usually receive benefit from the exposure situation but not necessarily from the exposure itself. Exposures may be controlled at source or, alternatively, by action in the exposure pathways.	Where possible, general information should be made available to enable individuals to reduce their doses. For planned situations, individual assessment of exposure and training should take place.	Constraints set for occupational exposure in planned situations. Constraints set for comforters and carers of patients treated with radiopharmaceuticals. Reference level for the highest planned residual dose from radon in dwellings.
1 or less	Individuals are exposed to a source that gives them little or no individual benefit but benefits to society in general. Exposures are usually controlled by action taken directly on the source for which radiological protection requirements can be planned in advance.	General information on the level of exposure should be made available. Periodic checks should be made on the exposure pathways as to the level of exposure.	Constraints set for public exposure in planned situations.

[a] Acute or annual dose.

[b] In exceptional situations, informed volunteer workers may receive doses above this band to save lives, prevent severe radiation-induced health effects, or prevent the development of catastrophic conditions.

[c] Situations in which the dose threshold for deterministic effects in relevant organs or tissues could be exceeded should always require action.

there is individual surveillance or dose monitoring or assessment, and where individuals benefit from training or information. The constraints set for occupational exposure in planned exposure situations are examples. Exposure situations involving

abnormally high levels of natural background radiation, or stages in post-accident rehabilitation may also be in this band.

(241) The third band, greater than 20 mSv but not more than 100 mSv, applies in unusual, and often extreme, situations where actions taken to reduce exposures would be disruptive. Reference levels and, occasionally for 'one-off' exposures below 50 mSv, constraints could also be set in this range in circumstances where benefits from the exposure situation are commensurately high. Action taken to reduce exposures in a radiological emergency is the main example of this type of situation. The Commission considers that a dose rising towards 100 mSv will almost always justify protective action. In addition, situations in which the dose threshold for deterministic effects in relevant organs or tissues could be exceeded should always require action (see also paragraph 83 in ICRP, 1999a).

(242) A necessary stage in applying the principle of optimisation of protection is the selection of an appropriate value for the dose constraint or the reference level. The first step is to characterise the relevant exposure situation in terms of the nature of the exposure, the benefits from the exposure situation to individuals and society, as well as other societal criteria, and the practicability of reducing or preventing the exposures. Comparison of these attributes with the characteristics described in Table 5 should enable the selection of the appropriate band for the constraint or the reference level. The specific value for the constraint or reference level may then be established by a process of generic optimisation that takes account of national or regional attributes and preferences together, where appropriate, with a consideration of international guidance and good practice elsewhere.

5.10. Dose limits

(243) Dose limits apply only in planned exposure situations but not to medical exposures of patients. The Commission has concluded that the existing dose limits that it recommended in *Publication 60* (ICRP, 1991b) continue to provide an appropriate level of protection. The nominal detriment coefficients for both a workforce and the general public are consistent with, although numerically somewhat lower than, those given in 1990. These slight differences are of no practical significance (see Annex A). Within a category of exposure, occupational or public, dose limits apply to the sum of exposures from sources related to practices that are already justified. The recommended dose limits are summarised in Table 6.

(244) For occupational exposure in planned exposure situations, the Commission continues to recommend that the limit should be expressed as an effective dose of 20 mSv per year, averaged over defined 5 year periods (100 mSv in 5 years), with the further provision that the effective dose should not exceed 50 mSv in any single year.

(245) For public exposure in planned exposure situations, the Commission continues to recommend that the limit 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

Table 6. Recommended dose limits in planned exposure situations[a].

Type of limit	Occupational	Public
Effective dose	20 mSv per year, averaged over defined periods of 5 years[e]	1 mSv in a year[f]
Annual equivalent dose in:		
Lens of the eye[b]	150 mSv	15 mSv
Skin[c,d]	500 mSv	50 mSv
Hands and feet	500 mSv	–

[a] Limits on effective dose are for the sum of the relevant effective doses from external exposure in the specified time period and the committed effective dose from intakes of radionuclides in the same period. For adults, the committed effective dose is computed for a 50-year period after intake, whereas for children it is computed for the period up to age 70 years.

[b] This limit is currently being reviewed by an ICRP Task Group.

[c] The limitation on effective dose provides sufficient protection for the skin against stochastic effects.

[d] Averaged over 1 cm^2 area of skin regardless of the area exposed.

[e] 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.

[f] 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.

allowed in a single year, provided that the average over defined 5-year periods does not exceed 1 mSv per year.

(246) The limits on effective dose apply to the sum of doses due to external exposures and committed doses from internal exposures due to intakes of radionuclides. In *Publication 60* (ICRP, 1991b), the Commission stated that occupational intakes may be averaged over a period of 5 years to provide some flexibility. The Commission maintains this view. Similarly, averaging of public intakes over a period of 5 years would be acceptable in such special circumstances where averaging of the dose to members of the public could be allowed (see the previous paragraph).

(247) Dose limits do not apply in emergency exposure situations where an informed, exposed individual is engaged in volunteered life-saving actions or is attempting to prevent a catastrophic situation. For informed volunteers undertaking urgent rescue operations, the normal dose restriction may be relaxed. However, responders undertaking recovery and restoration operations in a later phase of emergency exposure situations should be considered as occupationally exposed workers and should be protected according to normal occupational radiological protection standards, and their exposures should not exceed the occupational dose limits recommended by the Commission. Since the Commission recommends specific protection measures for female workers who have declared that they are pregnant or are nursing an infant (see Section 5.4.1), and taking account of the unavoidable uncertainties surrounding early response measures in the event of an emergency exposure situation, female workers in those conditions should not be employed as first responders undertaking life-saving or other urgent actions.

(248) For informed individuals of the general public involved in caring and comforting patients released from a hospital following therapy with unsealed radionuclides, the normal dose restriction may be relaxed and such individuals should in general not be subject to the public dose limit (see Section 7.6).

(249) In addition to the limits on effective dose, limits were set in *Publication 60* for the lens of the eye and localised areas of skin because these tissues will not necessarily be protected against tissue reactions by the limit on effective dose. The relevant values were set out in terms of the equivalent dose. These dose limits remain unchanged (see Table 6). However, new data on the radiosensitivity of the eye with regard to visual impairment are expected. The Commission will consider these data and their possible significance for the equivalent dose limit for the lens of the eye when they become available. Because of the uncertainty concerning this risk, there should be particular emphasis on optimisation in situations of exposure of the eyes.

(250) The dose limits for tissues are given in equivalent dose. The reason for this is that the Commission assumes that the relevant RBE values for the deterministic effects are always lower than w_R values for stochastic effects. It is, thus, safely inferred that the dose limits provide at least as much protection against high-LET radiation as against low-LET radiation. The Commission, therefore, believes that it is sufficiently conservative to use w_R with regard to deterministic effects. In special situations where high-LET radiation is the critical factor and where it predominantly exposes a single tissue (such as the skin), it will be more appropriate to express the exposure in terms of the absorbed dose and to take into account the appropriate RBE (see Annex B). To avoid confusion, it is necessary to clearly mention whenever an RBE-weighted absorbed dose in Gy is used.

(251) The Commission's multi-attribute approach to the selection of dose limits necessarily includes societal 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 regulatory authorities and applied in the process of optimisation of protection.

5.11. References

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6. IMPLEMENTATION OF THE COMMISSION'S RECOMMENDATIONS

(252) The previous chapter describes the Commission's system of protection to be applied in all situations requiring a decision on the control of radiation exposures. This chapter addresses the implementation of the system in the three types of exposure situations: planned, emergency, and existing. Particular attention is focused on areas where implementation of the Recommendations may not be immediately straightforward. In a number of these areas, there is further guidance from the Commission as indicated in the text. A section comparing the radiological protection criteria in these Recommendations with those in the previous Recommendations, *Publication 60* (ICRP, 1991b) and derivative publications, is included. The last section of this chapter addresses common aspects of the implementation of the Commission's Recommendations, notably the responsibilities of the users and regulatory authorities.

6.1. Planned exposure situations

(253) Planned exposure situations are where radiological protection can be planned in advance, before exposures occur, and where the magnitude and extent of the exposures can be reasonably predicted. The term encompasses sources and situations that have been appropriately managed within the Commission's previous recommendations for practices. In introducing a planned exposure situation all aspects relevant to radiological protection should be considered. These aspects will include, as appropriate, design, construction, operation, decommissioning, waste management, and rehabilitation of previously occupied land and installations, and will take account of potential exposures as well as normal exposures. Planned exposure situations also include the medical exposure of patients, including their comforters and carers. The principles of protection for planned situations also apply to planned work in connection with existing and emergency exposure situations, once the emergency has been brought under control. Recommendations for planned situations are substantially unchanged from those provided in *Publication 60* (ICRP, 1991b) and subsequent publications for the normal operation of practices and protection in medicine. Because of its specific characteristics, medical exposure is discussed separately in Chapter 7.

(254) All categories of exposure can occur in planned exposure situations, i.e., occupational exposure (Section 6.1.1), public exposure (Section 6.1.2), and medical exposure of patients, including their comforters and carers (Chapter 7). The design and development of planned situations should have proper regard for potential exposures that may result from deviations from normal operating conditions. Due attention should be paid to the assessment of potential exposures and to the related issue of the safety and security of radiation sources (Section 6.1.3).

6.1.1. Occupational exposure

(255) The Commission has previously recommended general principles for the radiological protection of workers (*Publication 75*, ICRP, 1997a). These principles remain valid.

(256) The Commission continues to recommend that occupational exposure in planned exposure situations be controlled by the procedures of optimisation below a source-related constraint (see Section 5.9.1) and the use of prescriptive dose limits (see Section 5.10). A constraint should be defined at the design stage of a planned exposure situation for its operation. For many types of work in planned exposure situations, 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 work. This work should be specified in fairly broad terms, such as work in industrial radiography, the routine operation of nuclear power plants, or work in medical establishments. However, there may also be more specific situations where a constraint can be established to guide particular activities.

(257) It will usually be appropriate for such dose constraints to be set at the operational level. When using a dose constraint, a designer should specify the sources to which the constraint is linked so as to avoid confusion with other sources to which the workforce might be concurrently exposed. The source-related dose constraint for occupational exposure in planned situations should be set to ensure that the dose limit is not exceeded (see Section 5.10). Experience gained in managing workers exposed to radiation will inform the choice of a value for a constraint for occupational exposure. For this reason, large established organisations, having a comprehensive radiological protection infrastructure, will often set their own constraints for occupational exposure. Smaller organisations with less relevant experience may require further guidance on this topic from the appropriate expert bodies or regulatory authorities. Nevertheless, the overall responsibility for setting constraints lies with those who are responsible for worker exposure.

(258) Protection of transient or itinerant workers requires particular attention because of the potential shared responsibility of several employers and licensees. In addition, sometimes several regulatory authorities are involved. Such workers include contractors for maintenance operations in nuclear power plants and industrial radiographers, who are not on the staff of the operator. In order to provide for their protection, adequate consideration needs to be given to the previous exposures of these workers so as to ensure that dose limits are also respected, and their exposure should be followed up. Thus there should be an adequate degree of co-operation between the employer of the itinerant worker and the operators of the plants for whom contracts are being undertaken. Regulatory authorities should ensure that regulations are adequate in this respect.

6.1.2. Public exposure

(259) In planned exposure situations, the Commission continues to recommend that public exposure be controlled by the procedures of optimisation below the source-related constraint and by the use of dose limits. In general, especially for public exposure, each source will cause a distribution of doses over many individuals, so the concept of a *Representative Person* should be used to represent the more highly exposed individuals (ICRP, 2006a). Constraints for members of the public in

planned exposure situations should be smaller than public dose limits, and would typically be set by the national regulatory authorities.

(260) For the control of public exposure from waste disposal, the Commission has previously recommended that a value for the dose constraint for members of the public of no more than about 0.3 mSv in a year would be appropriate (ICRP, 1997d). These Recommendations were further elaborated for the planned disposal of long-lived radioactive waste in *Publication 81* (ICRP, 1998b).

(261) In *Publication 82* (ICRP, 1999a), the Commission issued guidance that in circumstances where there are planned discharges of long-lived radionuclides to the environment, planning assessments should consider whether build-up in the environment would result in the constraint being exceeded, taking account of any reasonable combination and build-up of exposures. Where such verification considerations are not possible or are too uncertain, it would be prudent to apply a dose constraint of the order of 0.1 mSv in a year to the prolonged component of the dose attributable to the long-lived artificial radionuclides. In planned exposure situations involving natural radioactive material, this limitation is not feasible and not required (ICRP, 1999a). These Recommendations remain valid. In order to ensure that the build-up of annual doses from continuing practices does not cause dose limits to be exceeded in the future, the dose commitment can be used (ICRP, 1991b, IAEA, 2000b). This is the total dose that would eventually result from an event, such as a year of a planned activity causing discharges. Some flexibility may be required for particular situations involving long-lived natural radionuclides, such as past mining and milling activities (see Sections 2.3 and 5.2.2 of *Publication 82*, ICRP, 1999a).

6.1.3. Potential exposures

(262) In planned exposure situations, a certain level of exposure is reasonably expected to occur. However, higher exposures may arise following deviations from planned operating procedures, accidents including the loss of control of radiation sources, and malevolent events. Such exposures are not planned to occur, although the situation is planned. These exposures are referred to by the Commission as *potential exposures*. Deviations from planned operating procedures and accidents can often be foreseen and their probability of occurrence estimated, but they cannot be predicted in detail. Loss of control of radiation sources and malevolent events are less predictable and call for a specific approach.

(263) There is usually an interaction between potential exposures and the exposures arising from planned operations in normal operation; for example, actions taken to reduce the exposure during normal operations may increase the probability of potential exposures. Thus, the storage of long-lived waste rather than its dispersal could reduce the exposures from discharges but would increase potential exposures. In order to control potential exposure, certain surveillance and maintenance activities will be undertaken. These activities may increase normal exposures.

(264) Potential exposures should be considered at the planning stage of the introduction of a planned exposure situation. It should be recognised that the potential

for exposures may lead to actions both to reduce the probability of the events occurring, and to limit and reduce the exposure (mitigation) if any event were to occur (ICRP, 1991b, 1997b). Due consideration should be afforded to potential exposures during application of the principles of justification and optimisation.

(265) Potential exposure broadly covers three types of events.

- Events where the potential exposures would primarily affect individuals who are also subject to planned exposures: The number of individuals is usually small, and the detriment involved is the health risk to the directly exposed persons. The processes by which such exposures occur are relatively simple, e.g., the potential unsafe entry into an irradiation room. The Commission has given specific guidance for the protection from potential exposures in such circumstances in *Publication 76* (ICRP, 1997b). This guidance remains valid. Some additional examples are discussed in Section 7.5 on accidents in medical contexts.
- Events where the potential exposures could affect a larger number of people and not only involve health risks but also other detriments, such as contaminated land and the need to control food consumption: The mechanisms involved are complicated and an example is the potential for a major accident in a nuclear reactor or the malicious use of radioactive material. The Commission has provided a conceptual framework for the protection from such type of events in *Publication 64* (ICRP, 1993a). This framework remains valid. In *Publication 96* (ICRP, 2005a), the Commission provides some additional advice concerning radiological protection after events involving malicious intent.
- Events in which the potential exposures could occur far in the future, and the doses be delivered over long time periods, e.g., in the case of solid waste disposal in deep repositories: Considerable uncertainties surround exposures taking place in the far future. Thus dose estimates should not be regarded as measures of health detriment beyond times of around several hundreds of years into the future. Rather, they represent indicators of the protection afforded by the disposal system. The Commission has given specific guidance for the disposal of long-lived solid radioactive waste in *Publication 81* (ICRP, 1998b). This guidance remains valid.

Assessment of potential exposures

(266) The evaluation of potential exposures, for the purpose of planning or judging protection measures, is usually based on: a) the construction of scenarios which are intended typically to represent the sequence of events leading to the exposures; b) the assessment of probabilities of each of these sequences; c) the assessment of the resulting dose; d) the evaluation of detriment associated with that dose; e) comparison of the results with some criterion of acceptability; and f) optimisation of protection which may require several iterations of the previous steps.

(267) The principles of constructing and analysing scenarios are well known and are often used in engineering. Their application was discussed in *Publication 76* (ICRP, 1997b). Decisions on the acceptability of potential exposures should take account of both the probability of occurrence of the exposure and its magnitude. In some circumstances, decisions can be made by separate consideration of these

two factors. In other circumstances, it is useful to consider the individual probability of radiation-related death, rather than the effective dose (ICRP, 1997b). For this purpose, the probability is defined as the product of the probability of incurring the dose in a year and the lifetime probability of radiation-related death from the dose conditional on the dose being incurred. The resulting probability can then be compared with a risk constraint. If the probability is lower than the risk constraint, it may be tolerated. Both of these approaches are discussed in the Commission's Recommendations for the disposal of long-lived solid radioactive waste in *Publication 81* (ICRP, 1998b).

(268) Risk constraints, like dose constraints, are source-related and in principle should equate to a similar health risk to that implied by the corresponding dose constraints for the same source. However, there can be large uncertainties in estimations of the probability of an unsafe situation and the resulting dose. Thus, it will often be sufficient to use a generic value for a risk constraint. In the case of workers, this could be based on generalisations about normal occupational exposures, rather than on a more specific study of the particular operation. Where the Commission's system of dose limitation has been applied and protection is optimised, annual occupational effective doses to an average individual may be as high as about 5 mSv in certain selected types of operation (UNSCEAR, 2000). For potential exposures of workers, the Commission therefore continues to recommend a generic risk constraint of $2\ 10^{-4}$ per year which is similar to the probability of fatal cancer associated with an average occupational annual dose of 5 mSv (ICRP, 1997b). For potential exposures of the public, the Commission continues to recommend a risk constraint of $1\ 10^{-5}$ per year.

(269) The use of probability assessment is limited by the extent that unlikely events can be forecast. In circumstances where accidents can occur as a result of a wide spectrum of initiating events, caution should be exercised over any estimate of overall probabilities because of the serious uncertainty of predicting the existence of all the unlikely initiating events. In many circumstances, more information can be obtained for decision-making purposes by considering the probability of occurrence and the resultant doses separately.

(270) In large nuclear installations, dose criteria as a design basis of accident prevention and mitigation may be prescribed by the regulatory agency for selected potential exposure scenarios. The dose criteria applied here for the potential exposure should be derived from the risk constraints by taking account of the probability of the accident.

Safety and security of radiation sources and malevolent events

(271) Potential exposures associated with planned exposure situations may result from the loss of control of radiation sources. This situation has received a growing attention over recent years and deserves a special consideration from the Commission. The Recommendations of the Commission presume that, as a precondition for adequate radiological protection, radiation sources are subject to proper security measures (ICRP, 1991b). The control of radiation exposure in all planned exposure situations is exercised by the application of controls at the source rather than in the

environment. The Commission's view is reflected in the International Basic Safety Standards (BSS), which require that the control of sources shall not be relinquished under any circumstances (IAEA, 1996). The BSS also requires that sources be kept secure so as to prevent theft or damage. In addition, the Code of Conduct on the Safety and Security of Radioactive Sources establishes basic principles applicable to the security of radioactive sources (IAEA, 2004). The Commission supports the global strengthening of the control of radiation sources.

(272) Security of radioactive sources is a necessary, but not sufficient, condition to ensure source safety. Radioactive sources can be secure, i.e., under proper control, for instance preventing malicious use of the sources, and still not safe, i.e., prone to accidents. Thus the Commission has historically included aspects of security in its system of protection (ICRP, 1991b). In the context of safety, security provisions are generally limited to general controls necessary to prevent loss, access, unauthorised possession or transfer and use of the material, devices or installations. Measures to ensure that the control of radioactive material and access to radiation devices and installations are not relinquished are also essential to maintain safety.

(273) The Commission's 1990 Recommendations did not give attention to measures specifically to protect against terrorism or other malicious acts. However, it has become evident that radiation safety must also include the potential for such scenarios. Past experience with unintentional breaches in source security, or where a discarded or orphan source was found by individuals unaware of the radiation hazard, indicates what might occur if radioactive materials are used intentionally to cause harm, e.g., by deliberate dispersion of radioactive material in a public area. Such events have the potential for exposing people to radiation and causing significant environmental contamination, which would require specific radiological protection measures (ICRP, 2005a).

6.2. Emergency exposure situations

(274) Even if all reasonable steps have been taken during the design stage to reduce the probability and consequences of potential exposures, such exposures may need to be considered in relation to emergency preparedness and response. Emergency exposure situations are unexpected situations that may require urgent protective actions, and perhaps also longer-term protective actions, to be implemented. Exposure of members of the public or of workers, as well as environmental contamination can occur in these situations. Exposures can be complex in the sense that they may result from several independent pathways, perhaps acting simultaneously. Furthermore, radiological hazards may be accompanied by other hazards (chemical, physical, etc.). Response actions should be planned because potential emergency exposure situations can be assessed in advance, to a greater or lesser accuracy depending upon the type of installation or situation being considered. However, because actual emergency exposure situations are inherently unpredictable, the exact nature of necessary protection measures cannot be known in advance but must flexibly evolve to meet actual circumstances. The complexity and variability of these situations give them

a unique character that merits their specific treatment by the Commission in its Recommendations.

(275) The Commission has set out general principles for planning intervention in the case of a radiation emergency in *Publications 60* and *63* (ICRP, 1991b, 1992). Additional relevant advice is given in *Publications 86, 96, 97,* and *98* (ICRP, 2000c, 2005a, 2005b, 2005c). While the general principles and additional advice remain valid, the Commission is now extending its guidance on the application of protective measures on the basis of recent developments in emergency preparedness and of experience since publication of its previous advice.

(276) The Commission now emphasises the importance of justifying and optimising protection strategies for application in emergency exposure situations, the optimisation process being guided by reference levels (see Section 5.9). The possibility of multiple, independent, simultaneous, and time-varying exposure pathways makes it important to focus on the overall exposures that may occur from all pathways when developing and implementing protective measures. As such, an overall protection strategy is necessary, generally including an assessment of the radiological situation and implementation of different protective measures. These measures may well vary with time, as the emergency exposure situation evolves, and with place, as the emergency exposure situation may affect distinct geographic areas differently. The overall exposure, which is projected to occur as a result of the emergency exposure situation, should no protective actions be employed, is called the *projected dose*. The dose that would result when a protection strategy is implemented is called the *residual dose*. In addition, each protective measure will avert a certain amount of exposure. This is referred to as *averted dose*, and is the concept for the optimisation of the individual protective measures as given in *Publication 63* (ICRP, 1992) that will make up the overall protection strategy. The Commission now recommends focusing on optimisation with respect to the overall strategy, rather than the individual measures. However, the levels of averted dose recommended in *Publication 63* for optimisation of protection in terms of individual protective measures may still be useful as inputs to the development of the overall response (see also *Publication 96*, ICRP, 2005a).

(277) In emergency exposure situations particular attention should be given to the prevention of severe deterministic health effects as doses could reach high levels in a short period of time. In case of major emergencies an assessment based on health effects would be insufficient and due considerations must be given to societal, economic and other consequences. Another important objective is to prepare, to the extent practicable, for the resumption of societal and economic activities considered as 'normal'.

(278) In planning for emergency situations, reference levels should be applied in the process of optimisation. Reference levels for the highest planned residual doses in emergency situations are typically in the 20 mSv to 100 mSv band of projected dose as presented in Section 5.9.3. Expected residual doses for the overall protection strategies are compared with the reference levels in initially assessing the suitability of the strategies. A protection strategy that would not reduce residual doses to below the reference levels should be rejected at the planning stage.

(279) Planning should result in a set of actions that would be implemented automatically once an emergency exposure situation has occurred, should the actual circumstances require such urgent actions. Following a decision on such immediate action, the projected residual dose distribution can be assessed, and the reference level acts as a benchmark for assessing the effectiveness of protection strategies and the need to modify or take additional actions. All exposures above or below the reference level should be subject to optimisation of protection, and particular attention should be given to exposures above the reference level.

(280) When preparing a protection strategy for a particular emergency exposure situation, a number of different populations, each needing specific protective measures, may be identified. For example, the distance from the origin of an emergency exposure situation (e.g., an installation, an emergency site) may be important in terms of identifying the magnitude of exposures to be considered, and thus the types and urgency of protective measures. With this diversity of exposed populations in mind, the planning of protective measures should be based on exposures to the Representative Persons, as described in *Publication 101* (ICRP, 2006a), from the various populations that have been identified. After an emergency situation has occurred, planned protection measures should evolve to best address the actual conditions of all exposed populations being considered. Particular attention should be given to pregnant women and children.

(281) Emergency plans should be developed (in more or less detail, as appropriate) to cope with all possible scenarios. The development of an emergency plan (national, local, or installation specific) is a multi-step iterative process that includes assessment, planning, resource allocation, training, exercises, audit, and revision. The radiation emergency response plans should be integrated into all-hazards emergency management programmes.

(282) In the event that an emergency exposure situation occurs, the first issue is to recognise its onset. The initial response should be to follow the emergency plan in a consistent but flexible way. The protection strategy initially implemented will be that described in the emergency plan for the relevant event scenario, based on the generic optimisation undertaken as part of the planning stage. Once the measures in the emergency plan have been initiated, emergency response can be characterised by an iterative cycle of review, planning, and execution.

(283) Emergency response is inevitably a process that develops in time from a situation of little information to one of potentially overwhelming information, with the expectations for protection and involvement by those affected similarly increasing rapidly with time. As discussed in *Publication 96* (ICRP, 2005a), three phases of an emergency exposure situation are considered: the early phase (which may be divided into a warning and possible release phase), the intermediate phase (which starts with the cessation of any release and regaining control of the source of releases), and the late phase. At any stage, decision-makers will necessarily have an incomplete understanding of the situation regarding the future impact, the effectiveness of protective measures, and the concerns of those directly and indirectly affected, amongst other factors. An effective response must therefore be developed flexibly with regular review of its impact. The reference level provides an important

input to this review, providing a benchmark against which what is known about the situation and the protection afforded by implemented measures can be compared. The management of long-term contamination resulting from an emergency situation is treated as an existing exposure situation (see Section 6.3).

6.3. Existing exposure situations

(284) Existing exposure situations are those that already exist when a decision on control has to be taken. There are many types of existing exposure situations that may cause exposures high enough to warrant radiological protective actions, or at least their consideration. Radon in dwellings or the workplace, and naturally occurring radioactive material (NORM) are well-known examples. It may also be necessary to take radiological protection decisions concerning existing man-made exposure situations such as residues in the environment resulting from radiological emissions from operations that were not conducted within the Commission's system of protection, or contaminated land resulting from an accident or a radiological event. There are also existing exposure situations for which it will be obvious that action to reduce exposures is not warranted. The decision as to what components of existing exposure are not amenable to control requires a judgement by the regulatory authority that will depend on the controllability of the source or exposure, and also on the prevailing economic, societal, and cultural circumstances. Principles for exclusion and exemption of radiation sources are presented and discussed in Section 2.3.

(285) Existing exposure situations can be complex in that they may involve several exposure pathways and they generally give rise to wide distributions of annual individual doses ranging from the very low to, in rare cases, several tens of millisieverts. Such situations often involve dwellings, for example in the case of radon, and in many cases the behaviour of the exposed individuals determines the level of exposure. Another example is the distribution of individual exposures in a long-term contaminated territory, which directly reflects differences in the dietary habits of the affected inhabitants. The multiplicity of exposure pathways and the importance of individual behaviour may result in exposure situations that are difficult to control.

(286) The Commission recommends that reference levels, set in terms of individual dose, should be used in conjunction with the implementation of the optimisation process for exposures in existing exposure situations. The objective is to implement optimised protection strategies, or a progressive range of such strategies, which will reduce individual doses to below the reference level. However, exposures below the reference level should not be ignored; these exposure circumstances should also be assessed to ascertain whether protection is optimised, or whether further protective measures are needed. An endpoint for the optimisation process must not be fixed a priori and the optimised level of protection will depend on the situation. It is the responsibility of regulatory authorities to decide on the legal status of the reference level, which is implemented to control a given situation. Retrospectively, when protective actions have been implemented, reference levels may also be used as benchmarks for assessing the effectiveness of the protection strategies. The use of

reference levels in an existing situation is illustrated in Fig. 4, which shows the evolution of the distribution of individual doses with time as a result of the optimisation process.

(287) Reference levels for existing exposure situations should be set typically in the 1 mSv to 20 mSv band of projected dose as presented in Sections 5.9.2 and 5.9.3 and Table 5. The individuals concerned should receive general information on the exposure situation and the means of reducing their doses. In situations where individual life-styles are key drivers of the exposures, individual monitoring or assessment as well as education and training may be important requirements. Living on contaminated land after a nuclear accident or a radiological event is a typical situation of that sort.

(288) The main factors to be considered for setting the reference levels for existing exposure situations are the feasibility of controlling the situation, and the past experience with the management of similar situations. In most existing exposure situations, there is a desire from the exposed individual, as well as from the authorities, to reduce exposures to levels that are close to or similar to situations considered as 'normal'. This applies particularly in situations of exposures from material resulting from human actions, e.g., NORM residues and contamination from accidents.

6.3.1. Indoor radon in dwellings and workplaces

(289) Exposure to radon in dwellings and workplaces may arise from existing exposure situations or from practices, e.g., storage or processing of monazite sands. The Commission has previously made specific recommendations in relation to radon exposure (ICRP, 1993b). Since then, several epidemiological studies have confirmed the risk of radon-222 exposure even at relatively moderate concentrations (UNSCEAR, 2008). European, North American, and Chinese residential case-control studies also demonstrate a significant association between the risk of lung cancer and exposure to residential radon-222 (Darby et al., 2006, Krewski et al., 2006, Lubin et al., 2004). These studies have generally provided support for the Commission's Recommendations on protection against radon.

(290) There is now a remarkable coherence between the risk estimates developed from epidemiological studies of miners and residential case-control radon studies. While the miner studies provide a strong basis for evaluating risks from radon exposure and for investigating the effects of modifiers to the dose–response relation, the results of the recent pooled residential studies now provide a direct method of estimating risks to people at home without the need for extrapolation from miner studies (UNSCEAR, 2008).

(291) The Commission's view on radon risk assessment has, until now, been that it should incorporate epidemiological studies of miners. Given the wealth of data now available on domestic exposure to radon, the Commission recommends that the estimation of risk from domestic radon exposure should include the results of pooled residential case control radon-222 studies. However, there is still great value in the miner epidemiology studies for investigating dose response relationships and confounding effects of smoking and exposure to other agents. The currently available

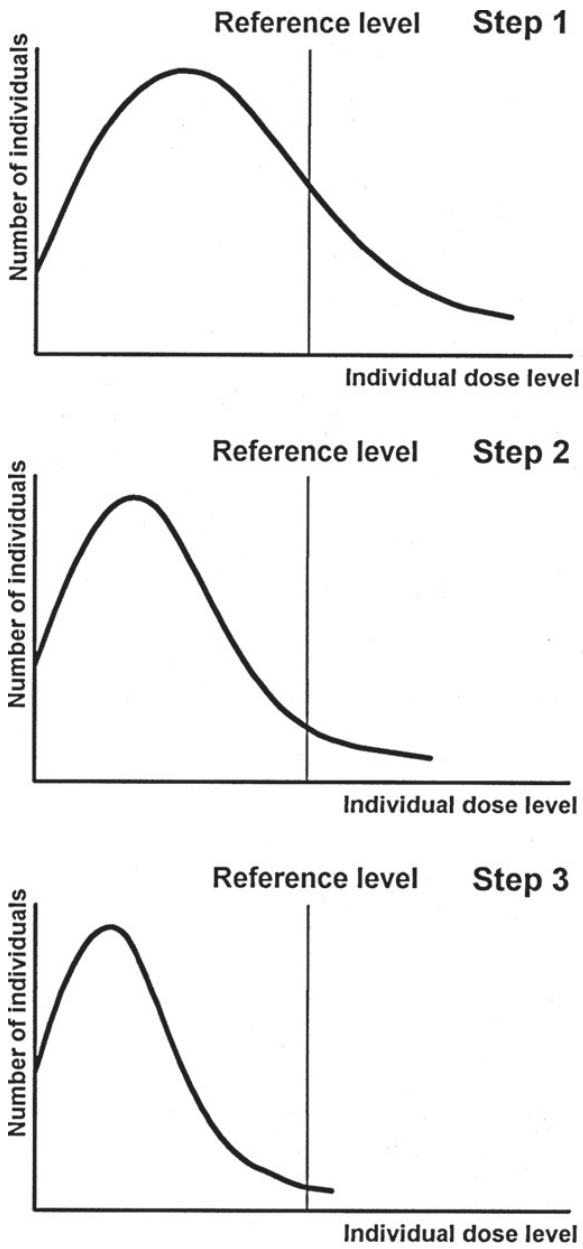

Fig. 4. The use of a reference level in an existing exposure situation and the evolution of the distribution of individual doses with time as a result of the optimisation process.

epidemiological evidence indicates that risks other than lung cancer from exposure to radon-222 (and decay products) are likely to be small.

(292) The underlying theme of the Commission's Recommendations on radon is the controllability of exposure. The ability to control exposure distinguishes the circumstances under which exposure to radon in workplaces, including underground mines, may need to be subject to the Commission's system of protection and where the need for action to limit radon exposure in dwellings should be considered. There

are several reasons for treating radon-222 in this separate manner. The exposure route differs from that of other natural sources, and there are dosimetric and epidemiological issues specific to radon-222. For many individuals radon-222 is an important source of exposure which, in principle, can be controlled. The Commission issued the current recommendations for protection against radon-222 at home and at work in *Publication 65* (ICRP, 1993b). The policy has found wide acceptance and the present Recommendations broadly continue the same policy, with an adaptation to the new approach based on exposure situations and where a central role is given to the optimisation principle and the use of reference levels.

(293) In *Publication 65* (ICRP, 1993b), the policy was based upon first setting a level to an effective dose of 10 mSv per year from radon-222 where action would almost certainly be warranted to reduce the exposure. Regulatory authorities were expected to apply the optimisation of protection in a generic way to find a lower level at which to act, in the range from 3 to 10 mSv. The effective dose was converted by a dose conversion convention into a value of radon-222 concentration, which was different between homes and workplaces largely because of the different number of hours spent at each. For dwellings this range was a radon concentration of 200–600 Bq m^{-3}, while the corresponding range for workplaces was 500–1500 Bq m^{-3}. The result of the optimisation was to set action levels, i.e., levels above which action was required to reduce the dose.

(294) The Commission now recommends applying the source-related principles of radiological protection for controlling radon exposure. This means that national authorities need to set national reference levels to aid the optimisation of protection. Even though the nominal risk per Sv has changed slightly, the Commission, for the sake of continuity and practicality, retains the upper value of 10 mSv for the individual dose reference level, and the corresponding activity concentrations as given in *Publication 65* (ICRP, 1993b). Thus, the upper values for the reference level expressed in activity concentrations remain at 1500 Bq m^{-3} for workplaces and 600 Bq m^{-3} for homes (Table 7).

(295) It is the responsibility of the appropriate national authorities, as with other sources, to establish their own national reference levels, taking into account the prevailing economic and societal circumstances and then to apply the process of optimisation of protection in their country. All reasonable efforts should be made to reduce radon-222 exposures in homes and at work places to below the reference levels that are set at the national level and to a level where protection can be considered optimised. The actions taken should be intended to produce substantial reduction in radon exposures. It is not sufficient to adopt marginal improvements aimed only

Table 7. Reference levels for radon-222[†].

Situation	Upper value of reference level: Activity concentration
Domestic dwellings	600 Bq m^{-3}
Workplaces	1500 Bq m^{-3}

[†] Head or initial radionuclide of the decay chain activity level.

at reducing the radon concentrations to a value just below the national reference level.

(296) The implementation of the optimisation process should result in concentration activities below the national reference levels. In general, no further action will be required apart from perhaps monitoring activity concentration sporadically to ensure that levels remain low. National authorities should, however, periodically review the values of the national reference levels for radon exposure to ensure that they remain appropriate.

(297) Responsibility for taking action against radon in houses and other premises will often fall on the individual owners, who cannot be expected to carry out a detailed optimisation exercise for each property. Therefore, in addition to reference levels, regulatory authorities may also wish to specify levels at which protection against radon-222 can be considered optimised, i.e., where no further action is needed. The Commission's view continues to be that there is merit in defining radon-prone areas in which the concentration of radon in buildings is likely to be higher than is typical of the country as a whole. This allows attention to be focused on radon where it is most exigent and action to be concentrated where it is most likely to be effective (ICRP, 1993b).

(298) Radon exposure at work at levels above the national reference level should be considered part of occupational exposure whereas exposures at levels below should not. In the interest of international harmonisation of occupational safety standards, a single action level value of 1000 Bq m^{-3} was established in the BSS (IAEA, 1996). For the same reasons, the Commission considers that this internationally established value, which is a reference value in current terminology, might be used globally to define the entry point for occupational protection requirements for exposure situations to radon. In fact, this international level serves as a much needed globally harmonised monitoring and record-keeping system. This is relevant for determining when the occupational radiological protection requirements apply – i.e., what is actually included within the system of regulatory control. On this basis the BSS establishes limits on intake and exposures for radon and thoron progeny (see Table II.1 in IAEA, 1996).

6.4. Protection of the embryo/fetus in emergency and existing exposure situation

(299) In *Publication 82* (ICRP, 1999a), the Commission concluded that prenatal exposure would not be a specific protection case, i.e., would not require protective actions other than those aimed at the general population. The protection of the embryo/fetus and infants is discussed in Section 5.4.1. In *Publication 82* (ICRP, 1999a), the Commission provided practical recommendations concerning in-utero exposures. Dose coefficients for the embryo/fetus due to intakes of radionuclides by the mother were provided in *Publication 88* (ICRP, 2001a). The Commission's conclusion in *Publication 90* (ICRP, 2003a) was that newly available information on in-utero risk at low doses (up to a few tens of mSv) supported the advice developed in *Publications 60*, *82*, *84*, and *88* (ICRP 1991b, 1999a, 2000a, 2001a). The Commission position on these issues remains unchanged.

6.5. Comparison of radiological protection criteria

(300) The current recommended values for protection criteria are compared in Table 8 with those provided by the previous Recommendations in *Publication 60* (ICRP, 1991b) and the derivative publications. The comparison shows that the current Recommendations are essentially the same as the previous Recommendations for planned exposure situations. In the case of existing and emergency exposure situations, the current Recommendations generally encompass the previous values but are wider in their scope of application. It should be noted that in some cases the values cited are in different quantities; for example, in emergency exposure situ-

Table 8. Comparison of protection criteria between the 1990 and the 2007 Recommendations (numbers in brackets refer to ICRP Publication numbers; ICRP, 1991b,c, 1992, 1993b, 1994b, 1997a,d, 1998b, 1999a, 2004b, 2005a,c).

Categories of exposure (Publications)	**1990 Recommendations and subsequent publications**	**Present Recommendations**
Planned exposure situations		
	Individual dose limits[a]	
Occupational exposure (60, 68, 75) including recovery operations (96)	20 mSv/year average over defined periods of 5 years[c]	20 mSv/year average over defined periods of 5 years[c]
– lens of the eye	150 mSv/year[b]	150 mSv/year[b]
– skin	500 mSv/year[b]	500 mSv/year[b]
– hands and feet	500 mSv/year[b]	500 mSv/year[b]
– pregnant women, remainder of pregnancy	2 mSv to the surface of abdomen or 1 mSv from intake of radionuclides	1 mSv to the embryo/fetus
Public exposure (60)	1 mSv in a year	1 mSv in a year
– lens of the eye	15 mSv/year[b]	15 mSv/year[b]
– skin	50 mSv/year[b]	50 mSv/year[b]
	Dose constraints[a]	
Occupational exposure (60)	⩽20 mSv/year	⩽20 mSv/year
Public exposure (77, 81, 82)		To be selected below 1 mSv/year
– general	–	according to the situation
– radioactive waste disposal	⩽0.3 mSv/year	⩽0.3 mSv/year
– long-lived radioactive waste disposal	⩽0.3 mSv/year	⩽0.3 mSv/year
– prolonged exposure	< ∼ 1 & ∼0.3 mSv/year[f]	<∼1 & ∼0.3 mSv/year[f]
– prolonged component from long-lived nuclides	⩽0.1 mSv/year[h]	⩽0.1 mSv/year[h]
Medical exposure (62, 94, 98)		
– volunteers for biomedical research, if benefit to society is:		
– minor	< 0.1 mSv	< 0.1 mSv
– intermediate	0.1–1 mSv	0.1–1 mSv
– moderate	1–10 mSv	1–10 mSv
– substantial	> 10 mSv	> 10 mSv
– comforters and carers	5 mSv per episode	5 mSv per episode

Table 8 (*continued*)

Categories of exposure (Publications)	1990 Recommendations and subsequent publications	Present Recommendations
Emergency exposure situations		
	Intervention levels[a,d,g]	**Reference levels**[a,g]
Occupational exposure (60, 96)		
– life-saving (informed volunteers)	No dose restrictions[i]	No dose restrictions if benefit to others outweighs rescuer's risk[k]
– other urgent rescue operations	~500 mSv; ~5 Sv (skin)[i]	1000 or 500 mSv[k]
– other rescue operations	. . .	⩽100 mSv[k]
Public exposure (63, 96) ***Public exposure:***		
– foodstuffs	10 mSv/year[l]	
– distribution of stable iodine	50–500 mSv (thyroid) [b,l]	
– sheltering	5–50 mSv in 2 days[l]	
– temporary evacuation	50–500 mSv in 1 week[l]	
– permanent relocation	100 mSv first year or 1000 mSv[l]	
– all countermeasures combined in an overall protection strategy	. . .	In planning, typically between 20 and 100 mSv/year according to the situation[e]
Existing exposure situations		
	Action levels[a]	**Reference levels**[a,m]
Radon (65)		
– at home	3–10 mSv/year (200–600 Bq m^{-3})	<10 mSv/year (<600 Bq m^{-3})
– at work	3–10 mSv/year (500–1500 Bq m^{-3})	<10 mSv/year (<1500 Bq m^{-3})
	Generic reference levels[e]	**Reference levels**[c,m]
NORM, natural background radiation, radioactive residues in human habitat (82)		
Interventions:		
– unlikely to be justifiable	< ~ 10 mSv/year	Between 1 and 20 mSv/year according to the situation (See Section 5.9.2)
– may be justifiable	> ~ 10 mSv/year	
– almost always justifiable	towards 100 mSv/year	

[a] Effective dose unless otherwise specified.

[b] Equivalent dose.

[c] With the further provision that the effective dose should not exceed 50 mSv in any one year. Additional restrictions apply to the occupational exposure of pregnant women. When applied to the intake of radionuclides, the dose quantity is committed effective dose.

[d] Averted dose.

[e] See Sections 5.9 and 6.2.

[f] The dose constraint should be less than 1 mSv and a value of no more than about 0.3 mSv would be appropriate.

[g] Intervention Levels refer to averted dose for specific countermeasures. Intervention Levels remain valuable for optimisation of individual countermeasures when planning a protection strategy, as a supplement to Reference Levels for evaluation of protection strategies; these refer to residual dose.

[h] To be considered if dose assessment methodologies to ensure compliance under any conceivable situation of combination of doses is not available.

[i] *Publication 60* (ICRP, 1991b).

[k] *Publication 96* (ICRP, 2005a). Effective doses below 1000 mSv should avoid serious deterministic effects; below 500 mSv should avoid other deterministic effects.

[l] *Publication 63* (ICRP, 1992).

[m] Reference Levels refer to residual dose and are used to evaluate protection strategies, as opposed to the previously recommended Intervention Levels which referred to averted doses from individual protective actions.

ations the criteria in *Publication 60* (ICRP, 1991b) are specified in terms of averted dose (intervention levels) whereas the criteria in the current Recommendations are specified in terms of incremental dose (reference levels). These differences are noted in Table 8.

6.6. Practical implementation

(301) This section addresses the general implementation of the Commission's Recommendations, dealing with factors which are common to the three types of exposure situations. It focuses on organisational features that may help in the implementation of the Commission's Recommendations. Since the organisational structures will differ from country to country, the chapter is illustrative rather than exhaustive. The International Atomic Energy Agency and the Nuclear Energy Agency of OECD issue further advice on the infrastructure required for radiological protection in various circumstances to their member states (e.g., see IAEA, 1996, 2000a, 2002, and NEA, 2005). Generic advice on organisation for health and safety at work is provided by the International Labour Organization, the World Health Organization and the Pan-American Health Organization.

6.6.1. The infrastructure for radiological protection and safety

(302) An infrastructure is required to ensure that an appropriate standard of protection is maintained. This infrastructure includes at least a legal framework, a regulatory authority, the operating management of any undertaking involving ionising radiation (including the design, operation, and decommissioning of equipment and installations as well as adventitious enhancement of natural radiation including aviation and space flight), and the employees at such undertakings. It may include additional organisations and persons responsible for protection and safety.

(303) The legal framework must provide for the regulation, as required, of undertakings involving ionising radiation and for the clear assignment of responsibilities for protection and safety. A regulatory authority must be responsible for the regulatory control, whenever required, of undertakings involving radiation and for the enforcement of the regulations. This regulatory authority must be clearly separate from organisations that conduct or promote activities causing radiation exposure.

(304) The nature of radiological hazards necessitates a number of special features in the legal framework and the provision of expertise within the regulatory authority. The important issues are that radiological questions are addressed properly, that the appropriate expertise is available, and that decisions concerning radiation safety cannot be unduly influenced by economic or other non-radiological considerations.

(305) The primary responsibility for achieving and maintaining a satisfactory control of radiation exposures rests 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 national authorities, 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.

(306) There are various reasons why there may not be a relevant operating management available. For instance, the radiation may not have been caused by any human actions, or an activity may have been abandoned and the proprietors could have disappeared. In such cases, the national regulatory authority, or some other designated body, will have to accept some of the responsibilities usually carried by the operating management.

(307) 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. However, the management of the organisation remains accountable for the provision of adequate radiological protection, and the delegation of tasks and responsibilities does not detract from that accountability. There should be a clear line of accountability running right to the top of each organisation. There is also an interaction between the various kinds of organisation. Advisory and regulatory authorities should be held accountable for the advice they give and any requirements they impose.

(308) 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 activity, from the individual workers and their representatives to the senior management, should regard protection and emergency prevention as integral parts of their everyday functions. Success and failure in these areas are at least as important as they are in the primary function of the activity.

(309) 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 in principle of prescriptive requirements, where the regulatory authority prescribes in detail how protection standards are to be maintained. However, prescriptive requirements concerning the conduct of operations result in some de facto transfer of responsibility and accountability from the user to the regulatory authority. In the long run, they also reduce the user's incentive for self-improvement. Therefore, it is usually better to adopt a regulatory regime that places a more explicit responsibility on the user, and forces the user to convince the regulatory authority that adequate protection methods and standards are used and maintained.

(310) Therefore, the use of prescriptive requirements should always be carefully justified. 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.

6.6.2. External expertise and advice; delegation of authority

(311) The prime responsibility for radiological protection and radiation safety in an undertaking involving ionising radiation rests with the operating organisation. In order to assume this responsibility, the organisation needs expertise in radiological protection. It is not always necessary or reasonable to demand that this expertise is available within the operating organisation. As an alternative, it may be acceptable and recommendable for the operating organisation to use consultants and advisory organisations, particularly if the operating organisation is small and the complexity of the radiological protection issues is limited.

(312) Such an arrangement will not in any way relieve the operating organisation of its responsibility. The role of a consultant or an advisory organisation will be to provide information and advice as necessary. It still remains the responsibility of the operating management to take decisions and actions on the basis of such advice, and individual employees still need to adhere to a 'safety culture', constantly asking themselves whether they have done all that they reasonably can to achieve a safe operation.

(313) Similarly, the use of consultants or advisory bodies will not in any way diminish or change the responsibility of the regulatory authority. Furthermore, it will be particularly important when the regulatory authority uses consultants that these are free from any conflicts of interest and are able to provide impartial advice. The need for transparency in decision making should also be kept in mind.

6.6.3. Incident reporting

(314) An accident and incident reporting routine with feedback to users is indispensable in the prevention of emergencies. In order for such a system to work and achieve its goals, mutual trust is required. Licensing constitutes the formal confirmation of a regulatory authority's trust in a user. However, operating organisations also need to be able to trust the regulatory authority. A primary requirement is that all users are treated in a fair and equal manner. Honest reporting of a problem combined with immediate action to rectify the situation should be encouraged, not punished.

6.6.4. Management requirements

(315) 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.

(316) The explicit commitment of an organisation to safety should be made manifest by written policy statements from the highest level of management, by the establishment of formal management structures for dealing with radiological protection,

by issuing clear operating instructions, and by clear and demonstrable support for those persons with direct responsibility for radiological protection in the workplace and the environment (*Publication 75,* ICRP, 1997a). To translate this commitment into effective action, senior management should identify appropriate design and operational criteria, determine organisational arrangements, assign clear responsibilities to put these policies into effect, and establish a culture within which all those in the organisation recognise the importance of restricting both normal and potential exposures to ionising radiation.

(317) There should be plans for dealing with accidents and emergencies. These plans should be subject to periodic review and exercise, and result in written management requirements. Planning for 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. Requirements to, and mechanisms for, implementing lessons learned should be established.

(318) The organisational approach should include involvement and participation of all workers. It is sustained by effective communications and the promotion of competence that enables all employees to make a responsible and informed contribution to the health and safety effort. The visible and active leadership of senior managers is necessary to develop and maintain a culture supportive of health and safety management. The aim is not simply to avoid accidents, but to motivate and empower people to work safely. It is important that management ensures that mechanisms are in place by which workers may provide feedback on radiological protection issues, and workers should be fully involved in developing methods to ensure that doses are as low as reasonably achievable.

(319) Another common responsibility of the operating management is to provide access to occupational services dealing with protection and health. The protection service should provide specialist advice and arrange any necessary monitoring provisions commensurate with the complexity of the operation and its potential hazards. The head of the protection service should have direct access to the senior operating management. The principal role of the occupational health service is the same as it is in any occupation.

6.6.5. Compliance with the intended standard of protection

(320) The measurement or assessment of radiation 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 and discussion should be made of the uncertainties inherent in these results (see Section 4.4).

(321) 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. National authorities 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.

6.7. References

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7. MEDICAL EXPOSURE OF PATIENTS, COMFORTERS AND CARERS, AND VOLUNTEERS IN BIOMEDICAL RESEARCH

(322) Medical exposures are predominantly delivered to individuals (patients) undergoing diagnostic examinations, interventional procedures, or radiation therapy. Other individuals caring for and comforting patients are also exposed to radiation. These individuals include parents and others, normally family or close friends, who hold children during diagnostic procedures or may come close to patients following the administration of radiopharmaceuticals or during brachytherapy. Exposure to members of the general public from released patients also occurs, but this exposure is almost always very small. In addition, volunteers in biomedical research often undergo medical procedures involving radiation exposure that are similar to procedures performed on patients. Medical exposure refers to all these types of exposures, and the present chapter, in particular, covers the following:

- The exposure of individuals for diagnostic, interventional, and therapeutic purposes, including exposure of the embryo/fetus or infant during medical exposure of patients who are pregnant or breast-feeding;
- Exposures (other than occupational) incurred knowingly and willingly by individuals such as family and close friends helping either in hospital or at home in the support and comfort of patients undergoing diagnosis or treatment;
- Exposures incurred by volunteers as part of a programme of biomedical research that provides no direct benefit to the volunteers.

(323) Radiation exposures of patients in medicine require an approach that differs from the radiological protection in other planned exposure situations. The exposure is intentional and for the direct benefit of the patient. In radiation therapy, the biological effects of high-dose radiation (e.g., cell killing) are used for the benefit of the patient to treat cancer and other diseases. The application of the Commission's Recommendations to the medical uses of radiation therefore requires separate guidance, and medical exposure of patients is therefore dealt with in the present chapter.

(324) In diagnostic and interventional procedures, this means avoiding unnecessary exposures, while in radiation therapy it requires delivery of the required dose to the volume to be treated, avoiding unnecessary exposure of healthy tissues.

(325) The objectives are the justification of medical procedures and the optimisation of protection commensurate with the medical purposes. The Commission's Recommendations for radiological protection and safety in medicine are given in *Publication 73* (ICRP, 1996a), and remain valid. These Recommendations note important differences between the implementation of the system of protection in medicine and its implementation in the other two categories of exposure (occupational and public). These differences include the following.

- The principle of justification applies at three levels in medicine as described in Section 7.1.1.

- In applying the principle of optimisation of protection of the patient, the benefits and detriments are received by the same individual, the patient, and the dose to the patient is determined principally by the medical needs. Dose constraints for patients are therefore inappropriate, in contrast to their importance in occupational and public exposure. Nevertheless, some management of patient exposure is needed and the use of diagnostic reference levels is recommended in *Publication 73* (ICRP, 1996a) with further guidance in *Supporting Guidance 2* (ICRP, 2001b).
- The limitation of the dose to the individual patient is not recommended because it may, by reducing the effectiveness of the patient's diagnosis or treatment, do more harm than good. The emphasis is then on the justification of the medical procedures and on the optimisation of protection.

(326) The basic framework for protection established in *Publication 60* (ICRP, 1991b) has been further elaborated in a series of publications described below. The recommendations, guidance, and advice in these publications remain valid, forming part of an increasing library of medical exposure information provided by the Commission [see also *Publication 105* (ICRP, 2007b)].

(327) The exposure of patients is deliberate. Except in radiation therapy, it is not the aim to deliver a radiation dose, but rather to use the radiation to provide diagnostic information or to conduct an interventional procedure. Nevertheless, the dose is given deliberately and cannot be reduced indefinitely without prejudicing the intended outcome. Medical uses of radiation are also voluntary in nature, combined with the expectation of direct individual health benefit to the patient. The patient, or legal guardian, agrees or consents to a medical procedure using radiation. This decision is made with varying degrees of informed consent that includes not only the expected benefit but also the potential risks (including radiation). The amount of information provided in order to obtain informed consent varies based on the exposure level (e.g., whether diagnostic, interventional, or therapeutic) and on the possible emergent medical complications that may be attributable to radiation exposure.

(328) The physicians and other health professionals involved in the procedures that irradiate patients should always be trained in the principles of radiological protection, including the basic principles of physics and biology. The final responsibility for the medical exposure of patients lies with the physician, who therefore should be aware of the risks and benefits of the procedures involved.

(329) Medical exposures of patients to external radiation are commonly concerned with limited parts of the body only, and it is important that medical staff are fully aware of the doses to normal tissue in the irradiated fields. Care has to be taken in such situations so that no undesirable tissue reactions occur.

7.1. Justification for medical procedures

(330) Medical exposure of patients calls for a different and more detailed approach to the process of justification. The medical use of radiation should be justified, as is any other planned exposure situation, although that justification lies usually with the medical profession rather than with government or regulatory authorities. The prin-

cipal aim of medical exposures is to do more good than harm to the patient, subsidiary account being taken of the radiation detriment from the exposure of the radiological staff and of other individuals. The responsibility for the justification of the use of a particular procedure falls on the relevant medical practitioners. Justification of medical procedures therefore remains a principal part of the Commission's Recommendations.

(331) The principle of justification applies at three levels in the use of radiation in medicine.

- At the first level, the use of radiation in medicine is accepted as doing more good than harm to the patient. This level of justification can now be taken for granted and is not discussed further below.
- At the second level, a specified procedure with a specified objective is defined and justified (e.g., chest radiographs for patients showing relevant symptoms, or a group of individuals at risk to a condition that can be detected and treated). The aim of the second level of justification is to judge whether the radiological procedure will usually improve the diagnosis or treatment or will provide necessary information about the exposed individuals.
- At the third level, the application of the procedure to an individual patient should be justified (i.e., the particular application should be judged to do more good than harm to the individual patient). Hence all individual medical exposures should be justified in advance, taking into account the specific objectives of the exposure and the characteristics of the individual involved.

The second and third levels of justification are discussed below.

7.1.1. The justification of a defined radiological procedure (level 2)

(332) The justification of the radiological procedure is a matter for national and international professional bodies, in conjunction with national health and radiological protection authorities and the corresponding international organisations. The possibility of accidental or unintended exposures should also be considered. The decisions should be reviewed from time to time, as more information becomes available about the risks and effectiveness of the existing procedure and about new procedures.

7.1.2. The justification of a procedure for an individual patient (level 3)

(333) Justification of individual exposures should include checking that the required information is not already available and that the proposed examination is the most suitable method of providing the clinical information required. For high-dose examinations, such as complex diagnostic and interventional procedures, individual justification is particularly important and should take account of all available information. This includes the details of the proposed procedure and of alternative procedures, the characteristics of the individual patient, the expected dose to the patient, and the availability of information on previous or expected examinations or

treatment. It will often be possible to speed up the justification process by defining referral criteria and patient categories in advance.

7.2. Optimisation of protection in medical exposures

(334) The Commission now uses the same conceptual approach in source-related protection, irrespective of the type of source. In the case of exposure from diagnostic and interventional medical procedures, the *diagnostic reference level* has as its objective the optimisation of protection, but it is not implemented by constraints on individual patient doses. It is a mechanism to manage patient dose to be commensurate with the medical purpose (see Section 7.2.1).

7.2.1. Diagnostic reference levels

(335) Diagnostic reference levels apply to radiation exposure of patients resulting from procedures performed for medical imaging purposes. They do not apply to radiation therapy. Diagnostic reference levels have no direct linkage to the numerical values of the Commission's dose limits or dose constraints. In practice, the values are selected on the basis of a percentile point on the observed distribution of doses to patients or to a reference patient. The values should be selected by professional medical bodies in conjunction with national health and radiological protection authorities and reviewed at intervals that represent a compromise between the necessary stability and the long-term changes in the observed dose distributions. The selected values could be specific to a country or region.

(336) Diagnostic reference levels are used in medical imaging to indicate whether, in routine conditions, the levels of patient dose from, or administered activity (amount of radioactive material) for, a specified imaging procedure are unusually high or low for that procedure. If so, a local review should be initiated to determine whether protection has been adequately optimised or whether corrective action is required (ICRP, 1996a). The diagnostic reference level should be expressed as a readily measurable patient-dose-related quantity for the specified procedure. Screening programmes, such as mammography of asymptomatic women in the general population, may require different diagnostic reference levels from the clinical use of similar diagnostic methods. Additional guidance is given in *Publication 105* (ICRP, 2007b) and in *Supporting Guidance 2* (ICRP, 2001b).

(337) In principle, it might be possible to choose a lower diagnostic reference level below which the doses would be too low to provide a sufficiently good image quality. However, such diagnostic reference levels are difficult to set, because factors other than dose also influence image quality. Nevertheless, if the observed doses or administered activities are consistently far below the diagnostic reference level, there should be a local review of the quality of the images obtained.

(338) Extensive information on the management of patient dose in fluoroscopically guided interventional procedures, computed tomography and digital radiology is provided in *Publications 85*, *87*, and *93*, respectively (ICRP, 2000b, 2000d, 2004a).

7.2.2. Radiation therapy

(339) In radiation therapy, optimisation involves not only delivering the prescribed dose to the tumour, but also planning the protection of healthy tissues outside the target volume. These radiation therapy issues are considered in *Publication 44* (ICRP, 1985a).

7.3. Effective dose in medical exposure

(340) The age distributions for workers and the general population (for which the effective dose is derived) can be quite different from the overall age distribution for the patients undergoing medical procedures using ionising radiation. The age distribution also differs from one type of medical procedure to another, depending on the prevalence of the individuals for the medical condition being evaluated. For these reasons, risk assessment for medical diagnosis and treatment using ionising radiation is best evaluated using appropriate risk values for the individual tissues at risk and for the age and sex distribution of the individuals undergoing the medical procedures. Effective dose can be of value for comparing the relative doses from different diagnostic procedures and for comparing the use of similar technologies and procedures in different hospitals and countries as well as the use of different technologies for the same medical examination, provided that the reference patient or patient populations are similar with regard to age and sex.

(341) The assessment and interpretation of effective dose from medical exposure of patients is problematic when organs and tissues receive only partial exposure or a very heterogeneous exposure, which is the case especially with diagnostic and interventional procedures.

7.4. Exposure of patients who are pregnant

(342) Before any procedure using ionising radiation, it is important to determine whether a female patient is pregnant. The feasibility and performance of medical exposures during pregnancy require specific consideration owing to the radiation sensitivity of the developing embryo/fetus.

(343) Prenatal doses from most correctly performed diagnostic procedures present no measurably increased risk of prenatal or postnatal death, developmental damage including malformation, or impairment of mental development over the background incidence of these entities. Life-time cancer risk following in-utero exposure is assumed to be similar to that following irradiation in early childhood. Higher doses such as those involved in therapeutic procedures have the potential to result in developmental harm (see Section 3.4).

(344) The pregnant patient has a right to know the magnitude and type of potential radiation effects that might result from in-utero exposure. Almost always, if a diagnostic radiology examination is medically indicated, the risk to the mother of not doing the procedure is greater than the risk of potential harm to the embryo/fetus. However, some procedures and some radiopharmaceuticals that are used in

nuclear medicine (e.g., radioiodides) can pose increased risks to the embryo/fetus. The Commission has given detailed guidance in *Publication 84* (ICRP, 2000a).

(345) It is essential to ascertain whether a female patient is pregnant prior to radiation therapy and some abdominal interventional procedures. In pregnant patients, cancers that are remote from the pelvis can usually be treated with radiation therapy. This however requires particular attention in treatment planning. The expected radiation dose to the embryo/fetus, including the scattering component, must be estimated. Cancers in the pelvis can rarely be adequately treated with radiation therapy during pregnancy without severe or lethal consequences for the embryo/fetus.

(346) Termination of pregnancy owing to radiation exposure is an individual decision affected by many factors. Absorbed doses below 100 mGy to the embryo/fetus should not be considered a reason for terminating a pregnancy. At embryonic/fetal doses above this level, the pregnant patient should receive sufficient information to be able to make informed decisions based upon individual circumstances, including the magnitude of the estimated embryonic/fetal dose and the consequent risks of serious harm to the developing embryo/fetus and risks of cancer in later life.

(347) Radiation risks after prenatal radiation exposure are discussed in detail in *Publication 90* (ICRP, 2003a). The exposure of patients who are pregnant is dealt with in detail in *Publication 84* (ICRP, 2000a) and in *Publication 105* (ICRP, 2007b), which also discuss the considerations to be taken into account regarding termination of pregnancy after radiation exposure. Radiation exposure of pregnant females in biomedical research is discussed in Section 7.7.

7.5. Accident prevention in external beam therapy and brachytherapy

(348) Accident prevention in external beam therapy and brachytherapy should be an integral part of the design of equipment and premises and of the working procedures. A key focus of accident prevention has long been the use of multiple defences against the consequences of failures. This approach, called 'defence in depth', is aimed at preventing equipment failures and human errors and mitigating their consequences, should they happen. The Commission has given extensive advice on reducing the probability of potential exposure and preventing accidents in *Publications 76, 86, 97* and *98* (ICRP, 1997b, 2000c, 2005b, 2005c).

7.6. Protection of carers and comforters of patients treated with radionuclides

(349) Unsealed radionuclides are used in the diagnosis and treatment of various diseases in the form of radiopharmaceuticals that are given to the patient by injection, ingestion, or inhalation. These radiopharmaceuticals may localise in body tissues until they decay or they may be eliminated through various pathways (e.g., urine). Sealed sources are implanted in the patient's body.

(350) Precautions for the public are rarely required after diagnostic nuclear medicine procedures but some therapeutic nuclear medicine procedures, particularly those involving iodine–131, can result in significant exposure to other people, espe-

cially those involved in the care and support of patients. Hence, members of the public caring for such patients in hospital or at home require individual consideration.

(351) *Publication 94* (ICRP, 2004b) provides recommendations for the release of patients after therapy with unsealed radionuclides. These recommendations include that young children and infants, as well as visitors not engaged in direct care or comforting, should be treated as members of the public for radiological protection purposes (i.e., be subject to the public dose limit of 1 mSv/year). For individuals directly involved in comforting and caring, other than young children and infants, a dose constraint of 5 mSv per episode (i.e., for the duration of a given release after therapy) is reasonable. The constraint needs to be used flexibly. For example, higher doses may well be appropriate for parents of very sick children.

(352) The thyroid gland of persons under the age of 15 is more radiosensitive than that of adults, so that particular care should be taken to avoid the contamination of infants, children, and pregnant women from patients treated with radioiodine.

(353) The decision to hospitalise or release a patient after therapy should be made on an individual basis considering several factors including residual activity in the patient, patient's wishes, family consideration (particularly the presence of children), environmental factors, and existing guidance and regulations. *Publication 94* (ICRP, 2004b) comments on the use of holding tanks for the storage of urine, implying that their use is unnecessary.

(354) The unintentional exposure of members of the public in waiting rooms and on public transport usually is not high enough to require special restrictions on nuclear medicine patients, except for those being treated with radioiodine (*Publications 73* and *94*, ICRP, 1996a, 2004b).

(355) In principle, similar reasoning applies when patients are treated with permanently implanted sealed sources. However, the available data show that, in the vast majority of cases, the dose to comforters and carers remains well below a value of 1 mSv/year except for the rare case where the patient's partner is pregnant at the time of implantation and the anticipated dose to the pregnant partner could exceed 1 mSv in a year (*Publication 98*, ICRP, 2005c).

(356) If the patient dies in the first few months after implantation of a sealed source, cremation of the corpse (frequent in some countries) raises several issues related to: 1) the radioactive material that remains in the patient's ashes; and 2) the radioactive material that is released into the air, potentially inhaled by crematorium staff or members of the public. Available data shows that cremation can be allowed if 12 months have elapsed since implantation with iodine–125 (3 months for palladium–103). If the patient dies before this delay has elapsed, specific measures should be undertaken (ICRP, 2005c).

7.7. Volunteers for biomedical research

(357) Volunteers make a substantial contribution to biomedical research. Some of the research studies are of direct value in the investigation of disease; others provide information on the metabolism of pharmaceuticals and of radionuclides that may be absorbed from contamination of the workplace or the environment. Not all these

studies take place in medical institutions, but the Commission includes the exposure of all volunteers in biomedical research under the category of medical exposure.

(358) The ethical and procedural aspects of the participation of volunteers in biomedical research and its justification have been addressed by the Commission in *Publication 62* (ICRP, 1991c). That report also discusses dose constraints for volunteers under different conditions, as briefly summarised in Table 8 (Chapter 6).

(359) In many countries, radiation exposure of pregnant females as subjects in biomedical research is not specifically prohibited. However, their involvement in such research is very rare and should be discouraged unless pregnancy is an integral part of the research. For the protection of the embryo/fetus, strict controls should be placed on the use of radiation in these cases.

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8. PROTECTION OF THE ENVIRONMENT

(360) Interest in the protection of the environment has greatly increased in recent years, in relation to all aspects of human activity. Such interest has been accompanied by the development and application of various means of assessing and managing the many forms of human impact upon it. The Commission is thus aware of the growing need for advice and guidance on such matters in relation to radiological protection even though such needs have not arisen from any new or specific concerns about the effects of radiation on the environment. The Commission also recognises that there is a current lack of consistency at international level with respect to addressing such issues in relation to radioactivity, and therefore believes that a more proactive approach is now necessary.

8.1. The objectives of radiological protection of the environment

(361) The Commission acknowledges that, in contrast to human radiological protection, the objectives of environmental protection are both complex and difficult to articulate. The Commission does however subscribe to the global needs and efforts required to maintain biological diversity, to ensure the conservation of species, and to protect the health and status of natural habitats, communities, and ecosystems. It also recognises that these objectives may be met in different ways, that ionising radiation may be only a minor consideration – depending on the environmental exposure situation – and that a sense of proportion is necessary in trying to achieve them.

(362) The Commission has previously concerned itself with mankind's environment only with regard to the transfer of radionuclides through it, primarily in relation to planned exposure situations, because this directly affects the radiological protection of human beings. In such situations, it has been considered that the standards of environmental control needed to protect the general public would ensure that other species are not put at risk, and the Commission continues to believe that this is likely to be the case.

(363) However, the Commission considers that it is now necessary to provide advice with regard to all exposure situations. It also believes that it is necessary to consider a wider range of environmental situations, irrespective of any human connection with them. The Commission is also aware of the needs of some national authorities to demonstrate, directly and explicitly, that the environment is being protected, even under planned situations.

(364) The Commission therefore believes that the development of a clearer framework is required in order to assess the relationships between exposure and dose, and between dose and effect, and the consequences of such effects, for non-human species, on a common scientific basis. This issue was first discussed in *Publication 91* (ICRP, 2003b), and it was concluded that it was necessary to draw upon the lessons learned from the development of the systematic framework for the protection of human beings. This framework is based on an enormous range of knowledge that the Commission attempts to convert into pragmatic advice that will be of value in

managing different exposure situations, bearing in mind the wide range of errors, uncertainties, and knowledge gaps of the various databases.

(365) The advantage of such a comprehensive and systematic approach is that, as the needs for change to any component of the system arise (as in the acquisition of new scientific data, or changes in societal attitudes, or simply from experience gained in its practical application) it is then possible to consider what the consequences of such a change may be elsewhere within the system, and upon the system as a whole. Such an approach would not work unless it was based on a numerical framework that contained some key points of reference.

8.2. Reference Animals and Plants

(366) In the case of human radiological protection, the Commission's approach to such issues has been greatly assisted by the development of anatomical and physiological reference models (ICRP, 2002). It has concluded that a similar approach would be of value as a basis for developing further advice and guidance for the protection of other species. The Commission is therefore developing a small set of Reference Animals and Plants (Pentreath, 2005), plus their relevant databases, for a few types of organisms that are typical of the major environments. Such entities will form the basis of a more structured approach to understanding the relationships between exposures and dose, dose and effects, and the potential consequences of such effects.

(367) The Reference Animals and Plants can be considered as hypothetical entities with certain assumed basic biological characteristics of a particular type of animal or plant, as described to the generality of the taxonomic level of Family, with defined anatomical, physiological, and life-history properties. They are not, therefore, necessarily the *direct* objects of protection themselves but, by serving as points of reference, they should provide a basis upon which some management decisions could be made. Simple dosimetric models, plus relevant data sets, are currently being developed for different stages of the life cycle of each type. Available data on radiation effects for each type are also being reviewed.

(368) Some form of practical means is obviously required in order to make judgements, based on our current level of knowledge of the effects of radiation on different types of animals and plants, in order to meet the Commission's objectives. With the exception of mammals, however, there is a general paucity of information upon which dose-response relationships can be established that would enable sensible conclusions to be drawn, particularly with respect to the relatively low dose rates likely to obtain in most exposure situations. Indeed, in general, the databases on radiation effects for the majority of animals and plants are not dissimilar to those relating to 'chemical toxicity' studies, where the levels required to produce a given effect are many orders of magnitude greater than those expected in the majority of environmental situations.

(369) With radiation there is another source of reference, and that is the natural background radiation to which such animals and plants are continuously and 'typically' exposed. Thus additional radiation doses to animals and plants can be

compared with those dose rates known or expected to have certain biological effects in those types of animals and plants, and with the dose rates normally experienced by them in their natural environments.

(370) The Commission does not therefore propose to set any form of 'dose limits' with respect to environmental protection. By setting out data for some Reference Animals and Plants, in a transparently derived way, and upon which further action may be considered, the Commission intends to offer more practical advice than in the past. The Commission will use this framework to gather and interpret data in order to provide more comprehensive advice in the future, particularly with regard to those aspects or features of different environments that are likely to be of concern under different radiation exposure situations.

8.3. References

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ANNEX A. BIOLOGICAL AND EPIDEMIOLOGICAL INFORMATION ON HEALTH RISKS ATTRIBUTABLE TO IONISING RADIATION: A SUMMARY OF JUDGEMENTS FOR THE PURPOSES OF RADIOLOGICAL PROTECTION OF HUMANS

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Preface to Annex A

When the Commission initiated its project to review and update its 1990 Recommendations, at the Main Commission meeting in Cape Town, South Africa, in 1998, it was clear from the outset that the main text of the new Recommendations would need to be supported by scientific Annexes and reports in much the same manner as the 1990 Recommendations.

Therefore, ICRP Committees 1 (on radiation effects) and 2 (on doses from radiation exposure) were asked to outline and begin to draft Annexes on the health effects of radiation and on dosimetric considerations. (Committees 3 on protection in medicine and 4 on the application of ICRP recommendations were similarly asked to produce supporting documents which were and are being published as separate reports: *Publication 105*, ICRP 2007b on protection in medicine and *Publication 101*, ICRP 2006a, on assessing dose to the representative person and on optimisation).

After initial plenary work, Committee 1 formed a Task Group in 2001 to advise the Main Commission and draft the present Annex to the Recommendations.

The membership of the Task Group was as follows:

R. Cox, Chairman	J. Hendry	A. Kellerer
C. Land	C. Muirhead	D. Preston
J. Preston	E. Ron	K. Sankaranarayanan
R. Shore	R. Ullrich	

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A. Akleyev	M. Blettner	R. Clarke
J.D. Harrison	R. Haylock	J. Little
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The membership of ICRP Committee 1 during the preparation of this Annex was:
(2001-2005)

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E. Ron	W. Rühm	R. Shore
F. Stewart	M. Tirmarche	R. Ullrich, Vice-Chairman
P.-K. Zhou		

Principal conclusions and recommendations

The following summary statements relate largely to health effects attributable to radiation in the dose range up to around 100 mSv (as single or annual doses) for the purposes of radiological protection.

- **For the induction of cancer and heritable disease at low doses/low dose rates the use of a simple proportionate relationship between increments of dose and increased risk is a scientifically plausible assumption; uncertainties on this judgement are recognised.**
- **A dose and dose-rate effectiveness factor (DDREF) of 2 recommended in *Publication 60* (ICRP, 1991b) should be retained for radiological protection purposes; the effect of introducing the possibility of a low-dose threshold for cancer risk is judged to be equivalent to that of an uncertain increase in the value of DDREF.**
- **Proposed changes in radiation weighting factors for protons and neutrons are noted; these judgements are discussed in Annex B to the present Recommendations: 'Quantities used in radiological protection'.**
- **New radiation detriment values and tissue weighting factors (w_T) have been proposed; the most significant changes from *Publication 60* relate to breast, gonads, and the treatment of remainder tissues. The w_T changes in question are: breast (0.12 from 0.05); gonads (0.08 from 0.20); remainder tissues (0.12 from 0.05 using a new additive system).**
- **Based upon cancer incidence data, detriment adjusted nominal risk coefficients for cancer are 5.5 10^{-2} Sv^{-1} for the whole population and 4.1 10^{-2} Sv^{-1} for adult workers; the respective *Publication 60* values are 6.0 10^{-2} Sv^{-1} and 4.8 10^{-2} Sv^{-1}.**
- **Detriment adjusted probability coefficients for heritable disease up to the second generation are 0.2 10^{-2} Sv^{-1} for the whole population and 0.1 10^{-2} Sv^{-1} for adult workers; the respective *Publication 60* values are 1.3 10^{-2} Sv^{-1} and 0.8 10^{-2} Sv^{-1} but these relate to risks at a theoretical equilibrium and no longer seem justified.**
- **Cancer risk following in-utero exposure is judged to be no greater than that following exposure in early childhood.**
- **Knowledge of the roles of induced genomic instability, bystander cell signalling and adaptive response in the genesis of radiation–induced health effects is insufficiently well developed for radiological protection purposes; in many circumstances these cellular processes will be incorporated in epidemiological measures of risk.**
- **Genetic susceptibility to radiation-induced cancer involving strongly expressed genes is judged to be too rare to appreciably distort estimates of population risk; the potential impact of common but weakly expressing genes remains uncertain.**
- **Dose responses for radiation-induced tissue reactions (deterministic effects) in adults and children are, in general, judged to have true dose thresholds which result in the absence of risk at low doses; further consideration of the extent of the dose threshold for cataract induction (visual impairment) is recommended.**
- **Dose responses for in-utero radiation-induced tissue reactions, malformations and neurological effects are also judged to show dose thresholds above around 100**

mGy; uncertainty remains on the induction of IQ deficits but at low doses the risk is judged to be of no practical significance.

- **Risks of non-cancer disease at low doses remain most uncertain and no specific judgement is possible.**

A.1. Introduction

(A 1) Since the publication of the 1990 Recommendations of the ICRP (*Publication 60*, ICRP 1991b), ICRP Committee 1 has continued to maintain broad surveillance on scientific developments regarding the quantification of health effects attributable to radiation exposure and the biological mechanisms that underlie these effects. Much of the output of Committee 1 is represented in the reports of ICRP Task Groups, and Committee 1 Working Parties have reviewed data in other relevant areas.

(A 2) The purpose of the present Annex is to summarise all post–1990 Committee 1 judgements relating to the health effects of radiation in order to support the development by the Commission of its new Recommendations. In many of the areas considered in the present Annex, Committee 1 had already provided specific judgements, e.g., on the risk of multifactorial diseases (*Publication 83*, ICRP 1999b), on radiation weighting factors (*Publication 92*, ICRP 2003c) and on cancer risk at low doses (*Publication 99*, ICRP 2005d). However, the revision of a) judgements on the induction of tissue reactions; b) nominal risk coefficients for risks of cancer and heritable disease; c) the transport of cancer risk between different populations; and d) the choice of tissue weighting factors required much additional work. For this reason the above topics are covered in detail in this Annex.

(A 3) An additional feature of the present Annex is the extent to which the accumulation of epidemiological and biological knowledge since 1990 has served to strengthen some of the judgements made in *Publication 60* or, in some cases, has led to a revision in procedures for risk estimation. In spite of the detailed nature of these gains in knowledge, the principal objective of this Annex is the provision of broad judgements for practical purposes of radiological protection. Accordingly, much of the work presented here centres on the continuing use of effective dose as a radiological protection quantity for prospectively estimating risks in the population and to demonstrate compliance with dose limits. The application of the concept of effective dose is discussed in Annex B.

(A 4) The Annex is structured in the following way. Section A.2 provides a brief summary of the gains in knowledge since 1990 on the biological processes that underlie the health effects of radiation exposure. Section A.3 provides updated judgements on the mechanisms and risks of radiation-induced tissue reactions. Section A.4 considers the mechanisms and genetics of cancer induction, summarises previous judgements on radiation weighting factors and details new epidemiologically based judgements on nominal risk coefficients, transport of risk, radiation detriment and tissue weighting factors; Section A.4 also summarises an earlier judgement on cancer risk in utero. Section A.5 briefly considers non-cancer diseases after radiation. In Section A.6, the Annex details a newly developed approach to the estimation of risks of heritable disease and provides a revised estimate of this risk. Finally, in Section A.7, a simple tabular format is used to summarise the principal recommendations of the Annex and to map these judgements to the appropriate sections of the Annex.

A.1.1. References, Preface and Section A.1

ICRP, 1991b. The 1990 Recommendations of the International Commission on Radiological Protection. ICRP Publication 60. Ann. ICRP 21 (1–3).

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ICRP, 2005d. Low-dose extrapolation of radiation-related cancer risk. ICRP Publication 99. Ann. ICRP 35 (4).

ICRP, 2006a. Assessing dose of the representative person for the purpose of radiation protection of the public *and* The optimisation of radiological protection: Broadening the process. ICRP Publication 101. Ann. ICRP 36 (3).

ICRP, 2007b. Radiological protection in medicine. ICRP Publication 105. Ann. ICRP 37 (5).

A.2. Interactions of radiation with cells and tissues

(A 5) The purpose of this Section is to summarise knowledge on the interactions of radiation with cells and tissues in the body with emphasis on the information and concepts that have developed since 1990. The intention is to provide a biological framework for the judgements to be developed in subsequent sections of the Annex. Although some of these biological data and concepts are complex, much of this Annex is intended for the non-specialist reader. Consequently the Annex will not enter into the detail of many of the biological and biophysical debates but rather seeks clarity and simplicity on the judgements made. Details of these debates may be found in earlier ICRP publications and other reviews.

A.2.1. Biophysical aspects of radiation action on cells

(A 6) ICRP has not specifically reviewed the broad topics of radiation biophysics and microdosimetry since 1990 but important advances and judgements are given in *Publication 92* (ICRP, 2003c) and in an ICRP Task Group report on Low-dose risks (*Publication 99*, ICRP, 2005d). The understanding of the early post-irradiation biophysical processes in cells and tissues has advanced substantially and the following paragraphs briefly highlight some major points of development. Further information is available in *Publication 92* (ICRP, 2003c), *Publication 99* (ICRP, 2005d), Goodhead et al. (1996) and NAS/NRC (2006).

(A 7) Knowledge of the fine structure of energy deposition from radiation tracks in DNA dimensions has grown, largely through the further development of Monte-Carlo track structure codes. Coupled with radiobiological information, track structure data have impacted greatly on thinking in respect of the nature of biologically critical damage to DNA.

(A 8) In particular, it has been recognised that a high proportion of radiation-induced damage in DNA is represented in complex clusters of chemical alterations. Such clustered damage can arise via a combination of damages induced by the main tracks, secondary electrons and secondary reactive radical species. Double- and single-strand breaks (DSB and SSB) in the DNA sugar-phosphate backbone plus a variety of damaged DNA bases can combine together in clusters, with a substantial fraction of total damage being closely spaced. There is also evidence that both the frequency and the complexity of complex clustered damage depends upon the linear energy transfer (LET) of the radiation.

(A 9) When DSB, SSB and base damages are considered together, complex clustered damage may constitute as much as 60% and 90% of total DNA damage after low and high LET radiations respectively. These data highlight a major difference between DNA lesions induced by radiation and those arising spontaneously via oxidative attack by reactive chemical radicals. Whereas the former are predominantly complex and clustered, the latter are randomly distributed and simple in their chemical structure.

(A 10) As described in ICRP *Publication 99* and noted in Section A.4.1, the different repair characteristics of simple and complex DNA lesions are an important

factor in the development of judgements on health effects after low doses of radiation.

(A 11) In addition to improvements in our understanding of the induction of complex DNA damage by radiation there have been other advances in radiation biophysics. For example, radiation-induced damage has been investigated at the level of chromosome structure, and this work has been paralleled by the biophysical modelling of the induction of gene/chromosomal mutations. There has also been valuable technical innovation including the development of single particle irradiation systems (microbeams) and of imaging methods for the cellular visualisation of DNA-protein interactions during DNA damage response (see *Publication 99,* ICRP, 2005d; Cherubini et al., 2002).

A.2.2. Chromosomal DNA as the principal target for radiation

(A 12) In addition to the biophysical information outlined in Section A.2.1, there is more direct evidence that implicates chromosomal DNA as the principal cellular target for biological effects. Much of the early evidence on this issue concerned the greater radiobiological effectiveness of radionuclides incorporated into DNA in the cell nucleus as compared with cellular proteins in general (UNSCEAR 1993). More recently the use of microbeam irradiation facilities capable of delivering a defined dose to different parts of the cell has fully confirmed the radiosensitivity of the cell nucleus. However, as noted in Section A.2.5, these microbeam techniques have also provided evidence of the potential complexity of cellular radiation response.

(A 13) In addition, since 1990 the critical importance of DNA damage for radiobiological effects, including cancer induction, has been emphasised by a large number of studies with cells and animals that are genetically deficient in DNA damage response – many of these specific genetic deficiencies increase the frequency of radiobiological effects (UNSCEAR 1993, 2000; *Publication 79*, ICRP 1998a; NAS/NRC 2006). Finally the rapidly developing concordance noted in Section A.2.1 between biophysical predictions on radiation action, the biological importance of complex DNA damage and the characteristics of radiation-induced gene and chromosomal mutations add weight to the conclusion that certain forms of DNA damage are critically important to radiobiological effects.

A.2.3. DNA damage response and repair

DNA repair, apoptosis, and cellular signalling

(A 14) Advances in knowledge of the mechanisms and consequences of post-irradiation processes in cells arguably represent the most profound change in our understanding of radiobiology. Much of this advance can be ascribed to the greatly improved technology and knowledge base that is now characteristic of modern cell/molecular biology and genetics. The UNSCEAR 2000, NCRP 2001, NAS/NRC 2006 and ICRP 2005d (*Publication 99*) reports deal with these issues in detail and only a few key conclusions are given here.

- The isolation and characterisation of critical DNA damage response genes, e.g., for ATM, NBS and DNA PK_{cs} proteins, have provided insights into the structure and function of the most important biochemical pathways that operate to recognise and signal the presence of DNA damage.
- There is now good understanding of many of these pathways and this leads to the view that error-prone repair of chemically complex DNA double-strand lesions best explains the cellular radiobiological responses known for many years, viz., the induction of chromosome aberrations, gene mutation, and cell killing.
- The potential for error-free, recombinational repair of radiation-induced DNA double-strand lesions is recognised but, since it is thought to be restricted to the later phases of the cell cycle, its impact on radiation risk overall is not likely to be great.
- Coupled with earlier cellular studies, molecular and biochemical data add weight to the view that the activity of DNA damage response and repair processes are major determinants of dose/dose rate and radiation quality effects in cells.
- Post-irradiation programmed cell death (apoptosis) and delaying effects on the passage of cells through their reproductive cycles are now much better understood at the molecular and biochemical levels.
- In terms of protective effects, apoptotic elimination of radiation damaged cells may be viewed as an alternative to repair, i.e., apoptotic death reduces the frequency of viable cells carrying mutations.
- The imposition of cell cycle checkpoints in irradiated cells has been biochemically linked with the complex network of DNA damage signalling and may serve to maximise opportunities for repair or as points where the cell decides its fate (life or death) on the basis of biochemical balance. The evidence for this is, however, limited.
- New highly sensitive techniques for studying the induction of DNA double-strand breaks in single cells and post-irradiated cellular signalling show great promise for gaining knowledge of DNA damage response at low doses.

(A 15) A critical element in the advances that underpin the above judgements is the now compelling evidence that perturbation of DNA damage response/repair and apoptotic/cell cycle control are often closely associated with tumorigenic development. This concept gives increased confidence that these cellular activities are integral to the cellular defences mounted against post-irradiation tumour development. This in turn means that the characteristics of these cellular processes are important elements in the development of judgements in radiological protection.

Adaptive responses

(A 16) The relatively high level of knowledge gained on post-irradiation DNA repair, apoptosis and cellular signalling may be contrasted with the continuing uncertainty on the mechanisms and significance of so-called adaptive responses. Typically, in some experimental systems, adaptive responses are seen in cells conditioned by a priming dose of radiation. In some way this conditioning dose allows cells to develop increased resistance to a second radiation challenge.

(A 17) Data relating to adaptive responses of various types have been reviewed extensively (UNSCEAR 1994, 2000, NCRP 2001, NAS/NRC 2006, ICRP 2005d). The principal conclusions from these reviews may be summarised as follows:

- Adaptive responses are not a universal feature of cells in vitro or in vivo.
- Even in the most well-studied cellular system (cytogenetic response in human lymphocytes) there is a) no evidence that adaptive responses may be triggered by doses of a few tens of milligray and b) there is considerable donor variation in the expression of the response.
- Although some studies support an association with more general stress response mechanisms, chemical radical scavenging and/or more efficient DNA repair, mechanistic knowledge of adaptive responses remains fragmentary.
- Although there are some positive results, animal studies on tumour induction (and immune response) do not provide consistent evidence of adaptive responses that reduce adverse health effects.

A.2.4. The induction of gene and chromosomal mutations

(A 18) As noted earlier there are now strong links between the biophysical processes that determine the induction of complex DNA double-strand lesions, error-prone DNA damage response/repair processes and the forms of gene and chromosomal mutations (DNA sequence loss or rearrangement) characteristic of ionising radiation exposure. Much of the available quantitative dose-response data for cells pre-date *Publication 60*, and the specific forms of mutational dose response recorded depend upon the biological system, the mutational endpoint, the radiation quality (LET) and the dose rate (Thacker et al., 1992, UNSCEAR, 1993, 2000).

(A 19) In general, however, mutational dose-responses are linear-quadratic for low LET, and tend towards linearity as LET increases. For low LET radiations, reduction in dose rate usually reduces the frequency of induced gene/chromosomal mutations in mammalian somatic and germ cells. The maximum dose-rate reduction factor is usually 3–4 but it can be somewhat higher for chromosome aberration induction in human lymphocytes. A reasonably consistent relationship between RBE and LET for mutation induction has also been recorded with maximum values for RBE of around 10–20 usually being seen in the LET range 70–200 keV μm^{-1}.

(A 20) A novel feature of recent studies involving 'chromosome painting' techniques is that complex chromosome exchanges involving the interaction of more than two breakpoints are infrequent at low doses of low LET radiation but can be a significant fraction of high LET induced events at all doses. Advances in the understanding of radiation action on cellular DNA has included modelling of the formation of chromosomal exchanges but contention remains as to whether these exchanges demand the interaction of two damaged sites or whether a significant fraction derives from the interaction of damaged and undamaged sites (UNSCEAR 2000). Since 1990 considerable effort has been made to investigate the induction of gene and chromosomal mutations at low doses. There are many

technical factors that limit the resolution of such low-dose effects but two studies are notable.

(A 21) First, a large-scale investigation of chromosome aberration induction by x rays in human lymphocytes provided evidence of a linear dose-response at low doses with a limit of resolution of around 20 mGy. Second, the use of a highly sensitive in-vivo mutation system relating to pigment-producing cells in mouse skin showed linearity of mutational dose response down to the lowest x-ray doses of around 50 mGy (see UNSCEAR 2000, ICRP 2005d).

(A 22) There have also been valuable developments in the use of chromosomal aberration not only as a biomarker of radiation exposure but also for the purposes of establishing relationships between in-vivo cellular response, dose/dose-rate effects and potential health outcomes (Tucker et al., 1997, Tawn et al., 2004).

A.2.5. Epigenetic responses to radiation

(A 23) A major feature of radiobiological research since 1990 has been a range of studies that provide evidence of post-irradiation cellular responses that appear to result in genomic change and/or cellular effect without an obvious requirement for directly induced DNA damage (see Cherubini et al. 2002, NAS/NRC 2006, ICRP 2005d). In a broad sense these processes may be termed epigenetic and they contrast with the well-established radiobiological concept of direct DNA targeting by ionising radiation tracks which has underpinned much of the post-1990 developments in biophysics and DNA damage response. Although there are elements of overlap, these epigenetic effects may be placed in two categories: a) radiation-induced genomic instability; b) post-irradiation bystander signalling between cells.

Radiation-induced genomic instability

(A 24) Whereas conventional DNA damage response is known to result in the expression of genomic damage within the first or second post-irradiation cell cycles, the term 'induced genomic instability' broadly describes a set of phenomena whereby genomic damage and its cellular consequences are expressed persistently over many post-irradiation cell cycles (Little 2003, Morgan 2003). This instability, as expressed in cultured cells, can take the form of increased frequencies of chromosome aberrations, gene mutations and apoptosis/cell death; other manifestations have also been recorded. *Publication 99* (ICRP 2005d) and the NAS/NRC (2006) report have reviewed the recent evidence concerning induced genomic instability including the examples noted below.

(A 25) Much of the in-vitro cellular work on induced genomic instability has been performed using chromosomal endpoints. Although persistent chromosomal instability has been reproducibly demonstrated in mass cultures of established cell lines, there have been fewer studies of clonal cell populations and normal diploid cells. In this context a recent cytogenetic study with human diploid fibroblasts using mass culture and clonal techniques was particularly revealing in that it found no evidence of instability phenomena.

(A 26) This negative result raises the possibility that induced genomic instability is preferentially expressed in abnormal or genetically altered cells, and this would be consistent with the difficulties experienced in clearly demonstrating the phenomenon in vivo. After in-vivo exposure of humans and mice to high and low LET radiations, cytogenetic results have been negative or showed inconsistent evidence of persistent instability in haemopoietic cells. Nevertheless there are some positive results in certain mouse strains and normal cells, and further work is called for. In addition, there are indications that, in mice, the expression of induced genomic instability varies with genetic background and, in some cases, it may associate with deficiency in DNA damage response.

(A 27) The biological basis for induced genomic instability in its various forms is not well understood. Some biochemical data suggest the involvement of cellular stress and oxidative processes; other cytogenetic studies implicate potentially unstable DNA segments encoding DNA repeat sequences.

Post-irradiation bystander signalling

(A 28) The so-called bystander effect relates to the expression of cell death/apoptosis, gene/chromosomal mutation, genomic instability and/or changing patterns of protein abundance in cells not directly intersected by radiation tracks (see Little, 2003, Morgan, 2003, Mothersill and Seymour, 2001). These bystander cells are believed to be responding to signals from their irradiated neighbours via intercellular communication mediated by molecules passing through gap junctions in adjoining cell membranes or via diffusion of these signalling molecules through the cell culture medium. Data relating to the bystander effects of radiation are reviewed in *Publication 99* (ICRP, 2005d) and the NAS/NRC (2006) report and only a few points are noted here.

(A 29) Experimental studies on the bystander effect in cultured cells have been greatly facilitated by the development of microbeam irradiation facilities which allow the delivery of defined numbers of radiation tracks to cells or their nuclei. In this way, cellular effects arising in unirradiated cells may be specifically determined. Alternatively cells may be irradiated in mass culture with a fluence of particles that allows for only a fraction of cells/cell nuclei to be intersected. The expression of bystander signalling is then evidenced by a frequency of cellular effects that exceeds the number of track intersections.

(A 30) The majority of bystander studies relate to cellular irradiation with high LET alpha particles and protons although some low LET studies, particularly on signalling through the growth medium, are available. The biological mechanisms involved in bystander signalling are probably diverse and remain to be adequately elucidated. Some data point towards induction of oxidative stress and modulation of DNA damage-response pathways. In the case of effects mediated through the culture medium, there is some evidence for the release of chromosome-damaging (clastogenic) factors from irradiated cells and the mobilisation of intracellular calcium together with increased reactive oxygen species in recipient cells.

(A 31) Thus, the phenomena of induced genomic instability and bystander effects when expressed in vitro may show some common stress-related mechanisms. There are, however, few data and some controversies on the relative contribution of bystander signalling to cellular effects overall and the extent to which this is dose-dependent. Studies on bystander effects in vivo are in their infancy although there are some positive data relating to clastogenic factors.

A.2.6. Tissue reactions (deterministic effects)

(A 32) There have been no profound changes in scientific views on the quantitative aspects of harmful radiation-induced tissue reactions (deterministic effects) since 1990. However, there have been some developments concerning the mechanisms through which these reactions may be modified (see also Section A.3).

(A 33) An increasing number of studies on early tissue reactions have shown the ability to modify these using various cytokines and growth factors, primarily to stimulate regeneration of progenitor cells. Other biological response modifiers can be used for late reactions, in particular vascular modifying agents that delay the expression of organ damage induced in experimental animal systems. This ability to modify the response of tissues and organs means that the term 'deterministic effects' is not entirely accurate because, quantitatively, the effects are not necessarily pre-determined. Nevertheless, this term has become widely and firmly established, and the Commission continues to use the expression 'deterministic effects' to denote tissue and organ reactions.

(A 34) It has been recognised more since the 1990 Recommendations that the structure of tissues and organs plays a major role in their response to irradiation. Paired organs, or organs where the functional subunits (FSU) are arranged in parallel, rather than in series, can sustain inactivation of many FSU without clinical signs of injury, because of a substantial reserve capacity and compensation by the remainder of the FSU. This is one of the major reasons for the presence of a threshold dose for overt injury, and in particular for a high tolerance to partial-body irradiation, where a critical part of such organs may be spared.

(A 35) Late tissue reactions not only have a long and dose-dependent latency period before expression, but also they have a long progression period, with the incidence in many cases still rising well past 10 years after irradiation. Late reactions can be 'generic', which means that they arise directly in the responsible target tissue. Alternatively, late reactions can be 'consequential', meaning that they arise as a later consequence of a severe early reaction affecting the target tissue.

(A 36) There has been a consolidation of the use of the linear-quadratic formalism for describing the changes in iso-effective dose resulting from changes in the pattern of dose delivery, i.e., acute single doses, multifractionated doses, or continuous exposures. In general, the ratio of the linear and quadratic constants is higher for early reactions and consequential late reactions, and lower for generic late reactions.

A.2.7. Mechanisms of radiation tumorigenesis

(A 37) The technical and academic developments in biology since 1990 have also had a major impact on our understanding of the complex process of multistage tumorigenic development (e.g., UNSCEAR 1993, 2000, NCRP 2001, NAS/NRC 2006, ICRP 2005d). In simple terms the complex multistage process may be subdivided in the following way: a) Tumour initiation – the entry of a normal cell into an aberrant cellular pathway (pre-neoplastic state) that can lead to cancer; b) Tumour promotion – enhancement of the growth and development of a pre-neoplastic clone of initiated cells; c) Malignant conversion – the change from a pre-neoplastic state to one where cancer development is likely; and d) Tumour progression – the later phases of tumorigenesis where cells gain properties that allow more rapid development and the acquisition of invasive characteristics.

(A 38) In brief both lympho-haemopoietic and solid tumours are believed to originate from single stem-like cells in their respective tissues. Certain gene and chromosomal mutations, which are often tissue-specific, can confer cellular properties which allow these target stem cells to partially escape from their normal constraints of growth and development. In some cases these cells acquire novel properties via gain of function mutations in so-called oncogenes; in others, it is loss of function of so-called tumour-suppressor genes that applies. On current hypotheses, the full potential for malignancy in these tumour-initiated cell clones is then developed in a step-wise fashion via the appearance of other gene/chromosomal mutations or in some cases the non-mutational silencing of key genes. In this way, over time, tumours develop increasing malignant potential by growth selection and the bypass of cell senescence. In some cases the rate of tumour development may be increased following the acquisition of mutations that result in the de-stabilisation of DNA and chromosomes. This process of accelerated mutation rate can be a major drive for tumorigenesis in many tissues but, given its clear mutational basis, tumour-associated genomic instability is distinct from the phenomenon of radiation-induced genomic instability noted in Section A.2.5.

(A 39) Tumour development is, however, far more complex than the stepwise accumulation of clonal mutations. There is good evidence that the micro environmental interaction of tumorigenic and normal cells is a critical element in cancer development, and the recruitment of a blood supply to an evolving solid tumour is one important example of this.

(A 40) Since 1990 there has been good progress in understanding the mechanistic basis of radiation tumorigenesis using animal models and by undertaking genetic analysis of certain radiation-associated human tumours (see UNSCEAR 1993, 2000, NCRP 2001, NAS/NRC 2006, ICRP 2005d).

Animal models of radiation tumorigenesis

(A 41) A combination of cellular, cytogenetic, molecular and histopathological techniques has been employed to investigate experimentally multistage radiation tumorigenesis. Much of the most informative work has been undertaken in rodent models with some of these models having a genetic basis which has been informed

by studies with human counterpart tumours. In brief, for leukaemia and solid tumours of the skin, bone, brain, lung, breast and gastro-intestinal tract there is evidence on the process of multistage tumorigenesis after radiation and the identity of some of the critical mutations involved. Many of these mutations are present in the human counterpart tumours and also in the same rodent tumours arising spontaneously or after exposure to other carcinogens. Overall a key message from these studies is that radiation tumorigenesis appears to proceed in an unremarkable multistage manner with no obvious features that distinguish radiation as an unusual carcinogen. In particular, although data remain sparse, there are as yet no indications that the epigenetic process of induced genomic instability makes a consistent and major contribution to radiation tumorigenesis.

(A 42) Animal models have also been used to investigate the point of action of radiation in multistage tumour development (UNSCEAR, 1993, 2000, NCRP, 2001, ICRP, 2005d, NAS/NRC, 2006). These data provide evidence that radiation is only a weak promoter of tumour development and a role in the earliest (initiation) phase of tumorigenesis seems more likely. More direct evidence of such initiation properties has been obtained in a recent study of post-irradiation intestinal tumorigenesis in *Apc*-deficient mice (Ellender et al., 2005). This study showed that the principal effect of radiation was to increase the number of microscopic pre-neoplastic intestinal lesions rather than to enhance tumour development, and also that direct single-gene mutational events could account for radiation-induced intestinal adenoma yields. Molecular and cytogenetic studies using animal models add further weight to the argument that radiation acts early in the tumorigenic process via a gene-loss mechanism.

(A 43) In principle, its mutagenic properties should allow radiation to contribute throughout multistage tumorigenesis. However, the very high spontaneous rate of genome instability and damage that frequently characterises the post-initiation phases would tend to make these later phases less dependent on radiation-induced mutations (UNSCEAR 2000).

(A 44) Data from quantitative animal studies on radiation tumorigenesis are important for the development of some critical judgements in radiological protection. The implications of such data in respect of the effects of dose, dose-rate and radiation quality are noted later in this Annex.

Radiation-associated human tumours

(A 45) There are limited opportunities for mechanistic investigations with human tumours which have a high probability of radiation causation. The cytogenetic and molecular studies undertaken with radiation-associated tumours of lung, liver, thyroid, skin and bone marrow have tended to focus on particular gene or chromosomal mutations, and the relationship between these mutations and initial radiation damage remains unclear (UNSCEAR, 2000). However, in general accord with the results of animal studies, the human data developed since 1990 do not suggest that radiation tumorigenesis proceeds in an unusual fashion; evidence for the presence of specific mutational signatures of radiation is currently lacking. The involvement of induced

genomic instability in radiation tumorigenesis has been found to be lacking or is viewed as controversial (Nakanishi et al., 2001, Cox and Edwards, 2002, Lohrer et al., 2001).

Genetic susceptibility to cancer

(A 46) The issue of inter-individual genetic differences in susceptibility to radiation-induced cancer was noted in *Publication 60* and reviewed in *Publication 79* (ICRP 1998a), UNSCEAR (2000, 2001) and the BEIR VII report (NAS/NRC 2006). Since 1990 there has been a remarkable expansion in knowledge of the various single-gene human genetic disorders where excess spontaneous cancer is expressed in a high proportion of gene carriers – the so-called high penetrance genes. There is also a growing recognition and some data on variant genes of lower penetrance where gene–gene and gene–environment interactions determine a far more variable expression of cancer.

(A 47) Studies with cultured human cells and genetically altered laboratory rodents have also contributed much to knowledge and, with more limited epidemiological/clinical data, suggest that a high proportion of single-gene, cancer-prone disorders will show increased sensitivity to the tumorigenic effects of radiation.

(A 48) Recently, good progress has been made in demonstrating experimentally the complex interactions that may underlie the expression of cancer-predisposing genes of lower penetrance (NAS/NRC 2006); this work is however in its infancy.

A.2.8. Heritable diseases

(A 49) Views on the risks of induction of heritable diseases by radiation exposure of the gonads were developed in *Publication 60* (ICRP, 1991b) by extrapolating quantitative data on dose response for germ cell mutations in experimental animals (predominantly mice) to humans. Although extended follow-ups of mortality and cancer incidence in the offspring of the Japanese A-bomb survivors have been published (Izumi et al., 2003a, 2003b) these data do not alter the conclusions of previous analyses. In addition, few new quantitative data on mutation induction in mice have become available. However, since 1990 there have been significant developments in our understanding of the mutational process and new concepts for genetic risk estimation in human populations (UNSCEAR 2001, NAS/NRC, 2006). Although it remains the case that no human studies provide direct evidence of a radiation-associated excess of heritable disease, the data from experimental animals provide a compelling reason for ICRP to continue to make best use of advances in genetics in order to improve its estimate of these risks.

(A 50) The application of molecular genetic techniques has provided detailed knowledge of the molecular basis of naturally occurring mutations that cause heritable diseases in humans; also of radiation-induced gene (specific locus) mutations in mouse germ cells. There is now strong evidence that large multilocus deletions of the genome constitute the predominant class of radiation-induced mutation. It is judged that only a proportion of such multigene loss events will be compatible with

embryonic/fetal developmental and live birth. These findings have led to the concept that the principal adverse genetic effect in humans is likely to take the form of multisystem developmental abnormalities rather than single gene diseases.

(A 51) Another conceptual change based upon new human genetic information is the development of methods to assess the responsiveness of the frequency of chronic multifactorial diseases (e.g., coronary heart disease and diabetes) to an increase in mutation rate. This has allowed an improved estimate to be made of the risks associated with this large and complex class of disease where expression requires the interaction of genetic and environmental factors.

(A 52) These human genetic, experimental and conceptual advances have been integrated to form a new and more robust framework for the estimation of genetic risks (UNSCEAR 2001).

(A 53) There have also been developments on the estimation of radiation-induced mutation rates in mice and humans using expanded simple tandem DNA repeat (ESTR) loci in mice and minisatellite loci in humans. These DNA repeats are highly mutable with the mutations manifesting as changes in the number of tandem repeats. This increased mutability is expressed spontaneously and after radiation, and attention has been given to the mutational mechanisms involved, including the untargeted and transgenerational effects of radiation (UNSCEAR, 2000, 2001, CERRIE, 2004). However, since on current knowledge mutations at these DNA repeat sequences are only rarely associated with genetic disorders, the Commission judges that there is no good reason to include quantitative mutational data for these loci in the estimates of genetic risk given in Section A.6 of this report.

A.2.9. References, Section A.2

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A.3. Risks of tissue reactions (deterministic effects)

A.3.1. Revision of judgements given in ICRP *Publication 60*

Definition of stochastic effects and tissue reactions

(A 54) The deposition of energy by ionising radiation is a random process. Even at very low doses it is possible that sufficient energy may be deposited into a critical volume within a cell to result in cellular changes or cell death. The killing of one or a small number of cells will, in most cases, have no consequences in tissues, but modifications in single cells, such as genetic changes or transformations leading ultimately to malignancy, may have serious consequences. These effects resulting from damage in a single cell are termed stochastic effects. There is a finite probability of the occurrence of such stochastic events even at very low doses, so there will be no threshold dose unless all such events can be repaired up to some level of dose. As the dose is increased the frequency of such events increases, but in the absence of other modifying factors, the severity of the resultant effects is not expected to increase, in contrast to the case for tissue reactions (see below).

(A 55) With larger doses there may be a substantial amount of cell killing, sufficient to result in detectable tissue reactions. These reactions may occur early or late after irradiation. The depletion of renewing parenchymal cell populations, modified by stromal influences, plays a crucial role in the pathogenesis of early tissue reactions. In order to reach the level of detection, a given proportion of cells must be depleted. This constitutes a threshold, which depends on the specified level of injury. These reactions are distinct from the stochastic effects in single cells, which are the induction of cancers from irradiated somatic cells and genetic diseases in offspring following parental germ cell irradiation.

(A 56) When the term stochastic was introduced regarding single-cell effects, effects caused by injury in populations of cells were called non-stochastic (*Publication 41*, ICRP 1984). This was later considered an unsuitable term, and in *Publication 60* (ICRP 1991b) it was replaced by the term deterministic, meaning 'causally determined by preceding events'. Now it is recognised that both early and late tissue reactions are not necessarily predetermined, and they can be modified after irradiation by the use of various biological response modifiers. Hence it is considered more accurate to refer to these effects as early or late tissue or organ reactions. However, the Commission recognises that the generic terms, deterministic and stochastic effects, have a firmly embedded use in its system of protection and will use the generic and directly descriptive terms synonymously, according to context.

Tissue and organ reactions

(A 57) Early tissue reactions (on a timescale of hours to a few weeks) can be inflammatory-type reactions as a result of cell permeability changes and histamine release, e.g., erythema, and subsequent reactions as a consequence of cell loss, e.g., mucositis, and desquamatory reactions in epithelial tissues.

(A 58) Late tissue reactions (on a timescale of months to years) are called 'generic' if they occur as a result of injury directly in the target tissue, e.g., vascular occlusions

leading to deep tissue necrosis after protracted irradiations, or 'consequential' if they occur as a result of early reactions, e.g., dermal necrosis as a result of severe epidermal denudation and chronic infection, and intestinal strictures caused by severe mucosal ulceration (Dörr and Hendry 2001).

Cell survival curves

(A 59) Cell depletion plays a major role in the early desquamatory reactions in epithelial tissues after irradiation. In a few cell types and tissues, rapid cell loss after irradiation is mediated by apoptosis, as exemplified by lymphocytes and salivary glands. In other tissues, cell death is caused by reproductive failure of regenerative stem cells, which may undergo apoptosis before or after attempted mitoses, or of proliferating transit (differentiating) cells. The majority of non-proliferating mature cells do not die from irradiation, but from natural senescence. For a given level of tissue damage, it has been shown that dose modifying factors for different irradiation conditions are the same for survival of tissue target cells and for a given level of early tissue reactions, demonstrating the importance of target cell survival for these types of reaction (Hendry and Thames 1987).

(A 60) The survival of cells as a function of dose (Fig. A.3.1) is commonly described using the linear-quadratic equation:

$$S = \exp -(\alpha D + \beta D^2)$$

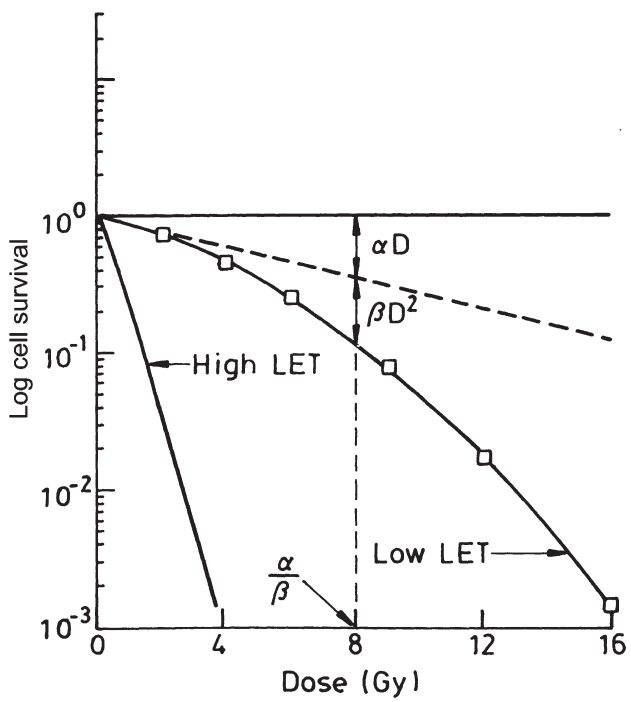

Fig. A.3.1. Dose response for cell survival (S) on a semi-log plot described by the linear-quadratic equation $S = \exp - (\alpha D + \beta D^2)$. From ICRP (1991b).

(A 61) The constant α describes the linear component of cell sensitivity to killing on a semi-log plot of survival (log) versus dose (linear), and β describes the increasing sensitivity of cells to higher radiation doses. The ratio α/β is the dose at which the linear and quadratic components of cell killing are equal. This ratio is a measure of the curvature of the survival curve. The α/β ratio is lower and the curve on a semi-log plot is more pronounced for homogeneous, slowly proliferating cell populations, such as in slow-renewing organ systems such as kidney and spinal cord. The α/β ratio is higher and the survival curve is straighter for heterogeneous, rapidly proliferating cell populations, such as the regenerative target cell populations in oral mucosa and intestine. One possible contributor to this straightening is the presence of sub-populations with different sensitivities as a function of cell-cycle phase. The α/β ratio is generally in the range 7–20 Gy for early reactions in tissues (10 Gy is commonly used) and 0.5–6 Gy for late reactions (3 Gy is commonly used).

(A 62) When dose rates are lower than around 0.1 Gy/hour there is repair of cellular radiation injury during the irradiation. This causes the β component to decrease and to reach zero at very low dose rates. The α component is not modifiable by changing dose rate. A special feature for some cell types is hypersensitivity to doses less than 0.5 Gy, typically at 0.2–0.3 Gy (Joiner et al. 2001), but not at higher doses. This causes a deviation from the smooth linear-quadratic cell survival curve. It is considered by some to be due to stimulation of repair processes at doses above 0.2–0.3 Gy. The deviation has been detected for early skin reactions in humans, and for skin reactions and kidney injury in experimental animal systems. The relevance of this hypersensitivity phenomenon for tissue injury thresholds is not yet clear.

(A 63) With high LET irradiations, there is less repairable injury and hence the β component and dose rate effects are small or absent. There is also no hypersensitivity component to the survival curve.

Early and late reactions in tissues and organs

(A 64) Early desquamatory reactions in epithelia, and depression of the haemopoietic system, are caused by the sterilisation of stem and progenitor cells in the tissues, resulting in a transitory or permanent lack of mature cells depending on the level of dose. Such reactions are characteristic of the radiation response of renewing cell lineages, such as those of the epidermis, mucosa, haemopoiesis and spermatogenesis. The time course of expression and restoration of tissue components generally depends on their normal rate of renewal, and is dose dependent at low doses but not at high doses. Complete denudation of such tissues after high doses occurs at a time equivalent to the lifetime of new mature cells plus those produced by any radioresistant progenitor cells. The stroma produces a variety of growth factors that induce the repopulation and differentiation needed to restore particular tissue components. The time course can be advanced and the restoration made more complete by the application of exogenous growth factors that further stimulate the reparative processes.

(A 65) Late reactions in tissues are due in part to the slow rate of renewal and death of component cell populations, where the cells are functional as well as capable

of division (Michalowski 1981, Wheldon et al. 1982). Late reactions are also due to dysfunction of a complex system of inter-cellular signalling pathways which normally regulate the various tissue and organ functions (Rubin et al. 1998). In some tissues it has been shown that different types of damage appear after different latency periods. For example, in spinal cord, there is an early demyelination effect within a few months, then a second phase of demyelination and necrosis of the white matter after 6–18 months, and a later phase after 1–4 years that is mostly a vasculopathy (van der Kogel 2002).

(A 66) In most tissues, responses are greater when irradiated volumes are larger. With early skin reactions, the volume effect is due largely to the decreasing ability to heal large areas mainly because of limited cell migration from the margins. With late reactions the volume effect relates to organ architecture. In spinal cord the critical elements are arranged in series, so that when more elements are irradiated, there is a greater chance of inactivating one of them to cause paralysis. There is also less benefit from cellular migration from the edges of the radiation field when irradiated volumes are larger. By contrast, in for example kidney and lung, the tissue functional subunits (FSU, respectively nephrons and alveoli) are arranged in parallel (Withers et al. 1988). In these cases, there can be inactivation of some FSU without causing a decrease in organ function, until a critical number of FSU is reached. Late tissue injury is progressive and strongly dose dependent, and it has been shown that the incidence of late morbidity after radiotherapy in humans continues to increase gradually to 10 years and beyond (Jung et al. 2001). There are various procedures that have been shown in experimental animal systems to delay the onset and development of late radiation morbidity (see below).

(A 67) Tissues vary not only in their temporal responsiveness, but also in their radiosensitivity. Among the most radiosensitive tissues are the ovary and testes, bone marrow, and the lens of the eye. In general, the dose-incidence relationship for these tissues will be sigmoid in shape when plotted on linear axes, the effect becoming more frequent as the dose increases (Fig. A.3.2a). Tissue and organ reactions vary with the dose, in severity as well as in incidence. The upper panel in Fig. A.3.3 illustrates how the incidence of a particular reaction, defined as a clinically recognisable pathological condition, increases as a function of dose in a population of individuals of

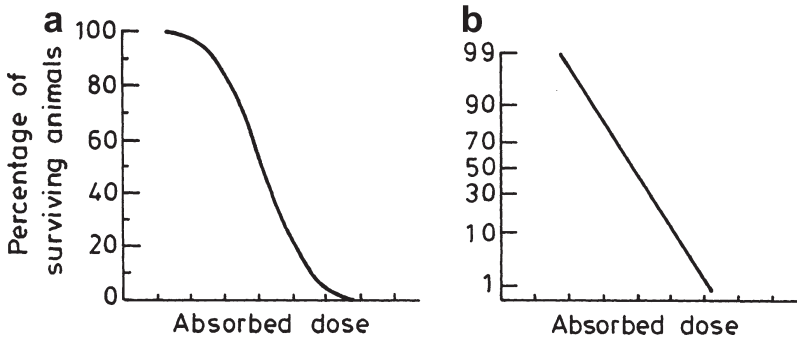

Fig. A.3.2. Relationship between mortality and dose: (a) sigmoid relationship on a linear–linear plot, (b) linear relationship on a transformed-probability–linear plot. From ICRP (1991b).

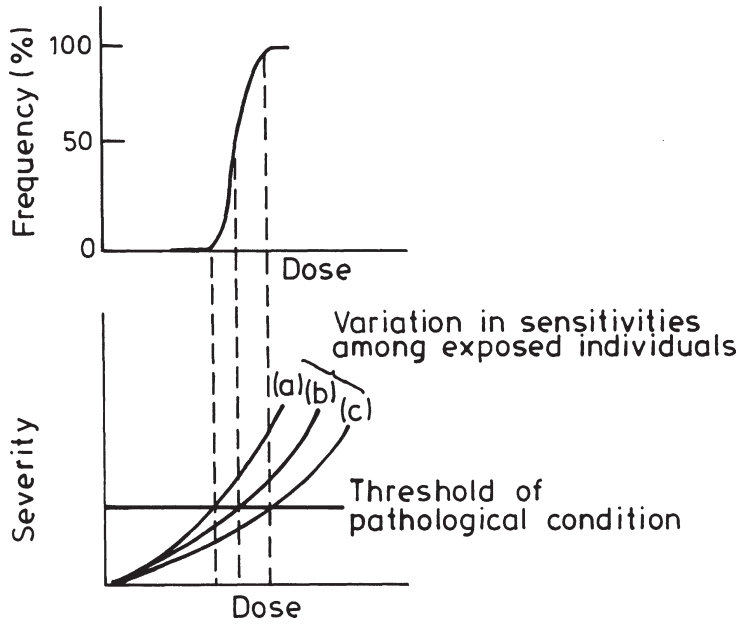

Fig. A.3.3. Relationships between dose and the frequency and severity of tissue reactions (deterministic effects). Upper panel: expected sigmoidal increase in frequency in a population of individuals with varying sensitivities. Lower panel: expected dose-severity relationships for three individuals with different sensitivities. From ICRP (1991b).

varying sensitivities. The lower panel in Fig. A.3.3 represents the dose-severity relationship for a population of individuals with various sensitivities. The severity of the pathological condition increases most markedly in those individuals in a subgroup who are most sensitive (curve a), reaching the threshold of detectability at a lower dose than in the less sensitive groups (curves b and c). The range of dose over which the different subgroups cross the same threshold of severity is reflected in the upper panel of Fig. A.3.3, which shows the frequency of the pathological condition in the total population, and which reaches 100% only at that dose which is sufficient to exceed the defined threshold of severity in all members of the population.

(A 68) In reality, substantially less than 1% of a general population is very radiosensitive because of inherited mutations in important DNA damage-sensing or repair genes. The remainder has a spectrum of sensitivities, and this has a flattening influence on the slope of the dose-incidence curve. This modification of the slope is in addition to primary contributions from inherent target-cell sensitivity and from features of tissue architecture discussed above. It is not yet possible to determine accurately the sensitivity of individuals within this spectrum of radiosensitivities, using cellular or molecular tests.

(A 69) Threshold doses for some tissue and organ reactions in the more radiosensitive tissues in the body are shown in Table A.3.1. These have been deduced from various radiotherapeutic experiences and accidental exposure incidents. In general, fractionated doses or protracted doses at low dose rate are less damaging than are acute doses.

Table A.3.1. Estimates of the thresholds for tissue effects in the adult human testes, ovaries, lens, and bone marrow (from ICRP 1984, *Publication 41*[1]).

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

See Table A.3.4 and Section A.3.1.7 for revised judgements.

[1] For further details consult *Publication 41* (ICRP 1984).

[2] NA denotes Not Applicable, since the threshold is dependent on dose rate rather than on total dose.

[3] See UNSCEAR (1988).

[4] See also Otake and Schull (1990).

[5] Given as 2–10 Sv (NCRP 1989) for acute dose threshold.

Mortality after whole body exposure

(A 70) Mortality after irradiation is generally the result of severe cell depletion in tissues of, or other major dysfunction of, one or more vital organs of the body. After partial body irradiation, or inhomogeneous whole body irradiation, the probability of death of an individual will depend on the particular organs exposed, the volume irradiated, and the dosage level. After whole body irradiation which is fairly homogeneous, for example with penetrating photon beams above about 1 MeV energy, death may occur from one of several distinct syndromes which are characteristic of particular dose ranges, and which are due to injury in specific organ systems.

(A 71) For a specific syndrome potentially leading to death, the relationship between the percentage of survivors and the dose is sigmoid in shape on a linear plot, whereas for a transformed probability-linear plot the shape is approximately linear (Fig. A.3.2b). The survival–dose relationship is often described by its midpoint, the LD_{50}, i.e., the dose that is lethal for half of the individuals, and the slope of the curve. The slope can be characterised by the probit width, which is the standard deviation of the distribution, or by other parameters in other transformations of the data. Values of $LD_{5\text{-}10}$ and $LD_{90\text{-}95}$ are helpful in assessments of the dose that will result in the death of only a few or of many.

(A 72) For a normal healthy adult human, the $LD_{50/60}$, i.e., within 60 days, is around 4 Gy midline dose, but there are estimates in the literature ranging from 3 to 5 Gy. Estimates of LD_{10} are around 1–2 Gy, and around 5–7 Gy for LD_{90} (UNSCEAR, 1988 Annex G, NUREG, 1997). The cause of death is haemopoietic failure, resulting primarily from a lack of progenitor cells that produce functional short-lived granulocytes, as well as from haemorrhages without the replacement of radioresistant red cells. It is possible to improve the chances of survival of individuals exposed to doses around or even above the $LD_{50/60}$ by appropriate medical care such as fluid replacement, antibiotics, antifungal drugs, and barrier nursing (UNSCEAR, 1988 Annex G), by infusing platelets and concentrates of isologous blood stem cells, and by injecting growth factors such as granulocyte-macrophage colony-stimulating factor. Some experts have considered that supportive medical treatment may increase the $LD_{50/60}$ to around 5 Gy, and possibly to around 6 Gy if growth factors are also employed (NUREG, 1997). In experimental animal systems these procedures have been shown to significantly increase the LD_{50} values (Table A.3.2). Growth factors have been used for many years in the treatment of humans following whole body irradiation for haematological diseases. However, in the few cases of accidental radiation exposures where they have been used, they did not save the individuals who were considered at risk of death, possibly because of the delay in starting treatment. Although the growth factors were considered to be of some benefit in the early post-exposure period, treated individuals died from organ reactions such as pneumonitis.

(A 73) 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 haemopoietic damage, causes death in 1–2 weeks. There are few human data to assess accurately the LD_{50} for this syndrome, but it may be approaching 10 Gy acute dose (UNSCEAR, 1988 Annex G, NUREG, 1997), and supportive medical treatment and growth factors are expected to increase this approximate value. If some marrow and most of the gut have been spared because of inhomogeneous irradiation, then at acute doses above 10 Gy to the lungs, acute inflammation (pneumonitis) may occur leading to death. Renal damage also occurs in the same dose range, if the kidneys have been irradiated. All these effects potentially can be alleviated to some extent, as evidenced by the success of growth factors and other molecules in reducing tissue and organ injury in animal systems after irradiation (Table A.3.2). At even higher doses towards 50 Gy and above, there is acute damage in the nervous and cardiovascular systems and the individual dies of shock after a few days (NCRP, 1974). Approximate doses for death at different times are given in Table A.3.3. These are for high dose, low LET radiation given over a few minutes.

(A 74) If the dose is given over a period of hours or more it requires a greater whole body dose for these effects to occur. For example, if the dose rate is about 0.2 Gy per hour, LD_{50} values may be increased by around 50% (NUREG, 1997). If the dose is delivered over a month, the $LD_{50/60}$ may be doubled (UNSCEAR, 1988 Annex G). At low (chronic) radiation dose rates, there is evidence of a chronic radiation syndrome affecting in particular the haemopoietic, immune and neural systems (Guskova et al., 2002, AFRRI, 1994, 1998, Akleyev and Kisselyov, 2002). The threshold doses for depression of the immune system is about 0.3–0.5 Gy per year

Table A.3.2. Dose-modifying factors (DMF) reported in mice or other species where stated. Updated from Hendry (1994).

Organ	Agent	DMF[a]
Bone marrow:		
Early reactions	Antibiotics Granulocyte-macrophage Colony-stimulating-factor	1.2–1.8 (rodents and monkeys)
Intestine:		
Early reactions	Antibiotics	1.1–1.4 (rats)
	Interleukin–1	1.1
	Angiogenic growth factors	1.1 (mice)[b]
	Interleukin–11, Transforming growth factor-β3	>1.0
Late reactions	Low molecular weight diet	>1.0 (rats)
	Antiplatelet Clopidogrel	>1.0 (rats)[c]
Skin:		
Alopecia	Prostaglandin E2	1.2–1.5
Early reactions	γ-linolenic acid	1.1–1.2 (pigs)
Late reactions	γ-linolenic acid	1.1–1.2 (pigs)
	Blood-cell modifiers	1.4
	Cu/Zn/Mn-SOD	>1.0 (pigs)[d]
Oral mucosa:		
Early reactions	Keratinocyte growth factor	about 2.0
Lung:		
Pneumonitis	Interleukin–1,	>1.0
	Tumour necrosis factor-α	>1.0
Spinal cord:		
Late reactions	Vasoactive agents	1.1 (rats)
Kidney:		
Late reactions	Captopril, angiotensin II blockers	>1.0 (rats)

[a] DMF = ratio of radiation doses with or without the protective agent, causing the same level of effect.

>1.0 indicates that the observed protection could not be quantified in terms of a DMF value, because dose-response relationships were not available. Reactions were assessed as less severe for combined radiation and agent.

[b] Okunieff et al. (1998).

[c] Wang et al. (2002).

[d] Lefaix et al. (1996).

(Akleyev et al., 1999), and estimated threshold doses for effects in other organs are given in Table A.3.1. Severe reactions do not occur in most body tissues of adults or children after annual doses below 0.1 Gy over many years. Red bone marrow, reproductive cells, and the lens of the eye show the greatest sensitivity.

Table A.3.3. 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[a] (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	7–20
5–15	Damage to the lungs and kidney	60–150
>15	Damage to nervous system	<5, dose-dependent

[a] Some dose range data include judgements from outcomes of partial body irradiations.

(A 75) Tissue and organ reactions resulting from exposure to high LET irradiation are similar to those from low LET exposure, but their frequency and severity are greater per unit absorbed dose of high LET irradiation. These differences are 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 absorbed doses of the reference low LET radiation and the high LET radiation that result in the same level of biological effect.

(A 76) RBE values for tissue and organ reactions are higher at lower doses and also when low doses per fraction are given repeatedly to accumulate the total dose (*Publication 58*, ICRP 1989b). RBE values tend to be smaller for early effects in haemopoietic and reproductive tissue, larger for gastrointestinal tract and skin, and even larger for late reactions in, for example, lung and kidney.

(A 77) The effective maximum RBE will be that value which applies at the threshold dose for the particular effect under consideration. This will be less than the value RBE_M, which is defined as the ratio of such doses at very low doses. This is the ratio of the linear components of the linear-quadratic fittings to data at higher doses. Hence it represents an extrapolation to dose levels below the threshold dose, which is of theoretical but not of practical interest. It also ignores the possibility of occult hypersensitivity at very low doses (see Section 3.1, paragraphs (A 59)–(A 63). RBE_M values for neutrons are 2–5 times lower, and effective maximum RBE values are even lower than values of RBE_M for stochastic effects in corresponding tissues. Thus the use of Q or w_R values in cases where tissue effects are over-riding would result in an overestimate of the contribution to the risk from high LET radiation.

Summary of projected estimates of dose thresholds for morbidity and mortality

(A 78) For the purposes of developing judgements for the present ICRP Recommendations, the Commission decided to update and summarise threshold estimates of the acute absorbed doses for 1% incidences of morbidity and mortality involving adult human organs and tissues after whole body gamma ray exposures. These 1% incidence estimates, derived from publications which utilise mathematical

Table A.3.4. Projected threshold estimates of the acute absorbed doses for 1% incidences of morbidity and mortality involving adult human organs and tissues after whole body gamma ray exposures.

Effect	Organ/tissue	Time to develop effect	Absorbed dose (Gy)[e]
Morbidity:			*1% Incidence*
Temporary sterility	Testes	3–9 weeks	~0.1[a,b]
Permanent sterility	Testes	3 weeks	~6[a,b]
Permanent sterility	Ovaries	< 1week	~3[a,b]
Depression of blood-forming process	Bone marrow	3–7 days	~0.5[a,b]
Main phase of skin reddening	Skin (large areas)	1–4 weeks	<3–6[b]
Skin burns	Skin (large areas)	2–3 weeks	5–10[b]
Temporary hair loss	Skin	2–3 weeks	~4[b]
Cataract (visual impairment)	Eye	Several years	~1.5[a,c]
Mortality:			
Bone marrow syndrome:			
– without medical care	Bone marrow	30–60 days	~1[b]
– with good medical care	Bone marrow	30–60 days	2–3[b,d]
Gastro-intestinal syndrome:			
– without medical care	Small intestine	6–9 days	~6[d]
– with good medical care	Small intestine	6–9 days	>6[b,c,d]
Pneumonitis	Lung	1–7 months	6[b,c,d]

[a] ICRP (1984).
[b] UNSCEAR (1988).
[c] Edwards and Lloyd (1996).
[d] Scott and Hahn (1989), Scott (1993).
[e] Most values rounded to the nearest Gy; ranges indicate area dependence for skin and differing medical support for bone marrow.

projections of dose-response data, are given in Table A.3.4, together with estimates of development times for the effects in question.

Dose limits for specific tissues

(A 79) *Publication 60* (ICRP 1991b, paragraph 194 and Table 6) describes the need to provide dose limits for exposure of the eye and localised areas of the skin because these tissues are not necessarily protected against radiation-induced reaction/injury by the limit on effective dose which, in these circumstances, protects against cancer development.

(A 80) Information available since 1990 has not provided evidence necessitating a change of view of the tumorigenic radiosensitivity of the skin or relevant subcutaneous tissues. It is judged therefore that the occupational and public dose limits for the skin and hands/feet given in Table 6 of *Publication 60* remain applicable. However, recent studies have suggested that the lens of the eye may be more radiosensitive than previously considered. In particular, among both A-bomb survivors (Minamoto et al., 2004) and a group of children treated for skin haemangioma (Hall et al., 1999), there is evidence of excesses of both cortical and posterior subcapsular cataract at doses somewhat lower than expected. In the assignment of a dose threshold for cataract, uncertainties are recognised on the mechanisms of cataract development, and

also on the relationship between the detection of lens opacity and the expression of visual impairment. The recent data and mechanistic uncertainties noted above highlight the need for a detailed reappraisal of the radiosensitivity of the lens of the eye and a newly formed Task Group of ICRP Committee 1 will address this issue.

A.3.2. Effects in the embryo and fetus

(A 81) The risks of tissue injury and developmental changes (including malformations) in the irradiated embryo and fetus have been reviewed recently in *Publication 90* (ICRP, 2003a). In the main, this review reinforced the judgements on in-utero risks given in *Publication 60* (ICRP, 1991b) although, on some issues, new data allow for clarification of views. On the basis of *Publication 90*, the following conclusions can be summarised on the in-utero risks of tissue injury and malformation at doses up to a few tens of mGy low LET.

(A 82) The new data from animal studies confirm embryonic sensitivity to the lethal effects of irradiation in the pre-implantation period of embryonic developments. At doses of a few tens of mGy such lethal effects will be very infrequent, and the data reviewed provide no reason to believe that there will be significant risks to health expressed after birth.

(A 83) In respect of the induction of malformations, the animal data strengthen the view that there are gestation age-dependent patterns of in-utero radiosensitivity with maximum sensitivity being expressed during the period of major organogenesis. On the basis of these animal data it is judged that there is a dose threshold of around 100 mGy for the induction of malformations; therefore, for practical purposes, risks of malformation after low-dose in-utero exposure may be discounted. *Publication 90* (ICRP 2003a) reviews the experimental data on neurodevelopment following in-utero irradiation for which dose thresholds generally apply; it also considers human epidemiological data as summarised below.

(A 84) The review of human A-bomb data on the induction of severe mental retardation after irradiation in the most sensitive prenatal period (8–15 weeks post conception) now more clearly supports a dose threshold of at least 300 mGy for this effect and therefore the absence of risk at low doses. The associated data on IQ losses estimated at around 25 points per Gy are more difficult to interpret and their significance is unclear. Although a non-threshold dose response cannot be excluded, even in the absence of a true dose threshold, any effects on IQ following in-utero doses of a few tens of mGy would be of no practical significance for the vast majority of individuals. This judgement accords with that developed in *Publication 60* (ICRP, 1991b).

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A.4. Risks of radiation-induced cancer

(A 85) In the development of judgements on the risk of radiation-induced cancer in the dose range up to around 100 mSv, the Commission has given attention to: a) the implications of fundamental data on radiation response; b) quantitative aspects of animal tumorigenesis; and c) direct epidemiological observation of cancer risk in humans, albeit at doses generally greater than 100 mSv. The conclusions reached by the Commission on the implications of fundamental and animal data are used: i) to guide the projection of higher dose epidemiological data for the purposes of estimating cancer risk in the low dose region of interest; and ii) to consider the application of a dose and dose rate effectiveness factor (DDREF) that would apply to human exposures at low doses and low dose rates. Judgements developed in Section A.6 on heritable effects are brought forward in order to provide new estimates of detriment and the nominal risk coefficients for risk in a single section of the Annex.

A.4.1. Fundamental data on radiation response

(A 86) In formulating Recommendations for protecting humans against the tumorigenic effects of radiation, the Commission is required to consider a very broad span of biological data and concepts; many of these are subject to ongoing debate and, in some cases, contention. There is, however, general agreement that epidemiological methods used for the estimation of cancer risk do not have the power to directly reveal cancer risks in the dose range up to around 100 mSv. Accordingly there is a growing role for biological data in the development of ICRP Recommendations and, where there is uncertainty and/or contention, there is a need to arrive at a scientifically balanced judgement based upon peer-reviewed data.

(A 87) The principal criteria used by the Commission in seeking a balanced view of biological data are captured in the questions given below.

- How relevant to in-vivo human tumorigenesis are the radiobiological end-points in question?
- Is the design, methodology and statistical strength of a given study sufficient to support the published conclusions?
- Do these published conclusions accord with those of similar studies and take adequate account of other relevant experimental data?

Where there are conflicting data and concepts:

- Which of the conflicting elements show greatest coherence with fundamental knowledge of the cancer process in general and, where possible, with epidemiological data?
- How critical is the issue for the broad purposes of radiological protection?

(A 88) These questions have been applied to a large set of published cancer-related fundamental data considered by ICRP Committee 1 and by other committees with interests in radiation cancer risk (e.g., UNSCEAR 2000, NCRP 2001, NAS/NRC

2006, ICRP 2005d). From this evaluation the following judgements have been developed by the Commission.

Dose-response relationships for gene and chromosomal mutations

(A 89) On the basis that the induction, by radiation, of gene and chromosomal mutations is of direct importance to the cancer process, the majority of relevant data from cellular studies is compatible with a simple relationship between dose and effect. A linear-quadratic form generally describes the full dose response for low LET radiations. The most informative data, although sparse, suggest linearity down to doses of a few tens of mGy, and there is no good reason to suggest a departure from this simple proportionality in the dose range down to a few mGy. At low LET radiation doses of a few mGy and below, linearity of response for targeted events in cells is expected because the fluence of tracks becomes equal to or less than the number of cells in the radiation field (see Section A.2.1). If, however, bystander effects were to be shown to contribute substantially to low-dose cellular effects in general then this expectation might not be met.

DNA damage response in cells

(A 90) There is much data to support the view that the activity of DNA damage response processes in cells is closely coupled with both cellular radiobiological effects and cancer development. On this basis the fidelity of post-irradiation DNA repair is expected to be a critical determinant of low-dose response. Current data point towards the predominance of an inherently error-prone repair process for the chemically complex DNA double-strand lesions that are characteristic of radiation action. Error-prone DNA repair at doses down to a few tens of mGy is consistent with the approximate linearity of cellular dose response for gene/chromosomal mutations and implies a simple proportionality between dose and the cancer risk associated with such mutations. The possibility of biochemical changes in DNA repair fidelity at doses below a few tens of mGy cannot be excluded but there are no specific reasons to predict such changes.

(A 91) A challenge to this conventional scientific view has come from proposals based upon the capacity of cells to sustain and repair a relatively high flux of spontaneously arising oxidative damage to DNA (see UNSCEAR 2000, NAS/NRC 2006, ICRP 2005d). The issue raised is that if cells can deal adequately with this relatively high level of spontaneous DNA damage then a small number of additional DNA lesions resulting from exposure to a few tens of mGy ($\sim$ 2 DNA double-strand lesions or $\sim$1 complex cluster per cell at $\sim$ 50 mGy low LET) would be of little or no consequence for cancer risk.

(A 92) This challenge might have some strength if spontaneously arising and radiation-induced DNA lesions were of the same type. However, as noted in Sections A.2.1 and A.2.3, there is good reason to believe that the clustered and chemically complex DNA lesions characteristic of radiation action arise very infrequently from spontaneous oxidative processes in cells; these oxidative processes tend to result in simple and readily repairable damage to the single strands of DNA. Since complex

DNA lesions are inherently difficult to repair correctly, the challenging argument loses much of its scientific strength.

(A 93) These issues have been addressed in detail by UNSCEAR (2000), NAS/NRC (2006) and ICRP (2005d) and, for the reasons summarised above, the Commission concludes that the balance of evidence weighs against challenges to simple proportionality in low-dose response that is based upon the relative abundances of spontaneous and radiation-induced DNA damage.

(A 94) It has also been proposed that simple proportionality between dose and radiobiological effect may not apply in all circumstances because of the activity of the adaptive DNA damage-response processes noted in Section A.2.3. The Commission recognises that the data on adaptive responses in human lymphocytes is reasonably reproducible but even these data show that this form of response is not consistently expressed in cell strains and has a poorly understood mechanistic basis. Other forms of adaptive response, e.g., immunological stimulation, considered by UNSCEAR (1994, 2000), and that were seen in some recent animal studies on tumorigenesis (Mitchel et al., 1999, 2003), are also judged to have most uncertain biological bases.

(A 95) Similar conclusions have been drawn by the BEIR VII Committee (NAS/NRC, 2006). The Commission does, however, recognise that the dose-dependence of post-irradiation cellular signalling and its potential implications for DNA damage response and cancer risk is an area where more information is needed. A report from the French Academies (2005) emphasises the potential importance of such cellular signalling and cites other data to support arguments in favour of a practical threshold for low-dose cancer risk (see also Section A.4.4, paragraphs A 178–A 187).

(A 96) Overall, the Commission concludes that the concept of adaptive responses to radiation lacks adequate biological support, and the available data fail to provide good evidence of robust adaptive and protective effects for cancer. The integration of the concept of adaptive response into a biological framework for radiological protection is therefore judged to be unjustified at this time.

Epigenetic responses to radiation

(A 97) Although the Commission is well aware that research is proceeding at a good pace, the available data do not provide good evidence of a robust causal association between cancer risk and the epigenetic phenomena of induced genomic instability and bystander signalling. It seems likely that diverse stress-related cellular processes underlie the expression of both types of response, but there is much uncertainty on dose-response characteristics, the extent to which in-vivo expression occurs, and how this might influence cancer risk. On this basis, the Commission suggests that, at present, it is not possible to meaningfully integrate data on these processes into the low-dose judgements necessary for radiological protection. Indeed, since direct human epidemiological data at low LET doses of above around 100 mGy provide the principal means for estimating nominal cancer risk coefficients, at these doses cancer risk estimates will incorporate all relevant biological processes including the epigenetic factors noted in this Annex. The critical issue of uncertainty

is therefore not simply whether such epigenetic factors influence cancer risk per se but rather whether the in-vivo dose response characteristics might provide for differential contributions to risk at, say, 200 mSv compared with, say, 10 mSv. The BEIR VII (NAS/NRC, 2006) and CERRIE (2004) Committees have also commented on the uncertain contribution of these epigenetic processes to radiation tumour risk.

A.4.2. Animal data on tumour induction and life shortening

(A 98) Animal data, largely from rodent studies, were included in consideration of relative biological effectiveness (RBE) in *Publication 92* (ICRP, 2003c) and have been reviewed in *Publication 99* (ICRP, 2005d) in respect of dose response and judgements on the dose and dose-rate effectiveness factor (DDREF). The relationship between RBE and radiation weighting (w_R) is adequately summarised in *Publication 92* and further developed in *Publication 99.*

(A 99) In respect of dose response, the most reliable animal data are generally compatible with a simple proportionate relationship between dose and risk but there are examples of highly curvilinear threshold-like responses for the induction of thymic lymphoma and ovarian cancer in mice. The processes that underlie the induction of these tumour types have a high degree of dependence upon cell killing and, for this reason, these responses are judged by the Commission to be atypical (see ICRP 2005d).

(A 100) When mouse data for thymic lymphoma and ovarian cancers are excluded from analyses the values for DDREF from animal studies are generally compatible and, at doses at or below around 2 Gy, a DDREF value of about 2 is implied.

A.4.3. Relative biological effectiveness (RBE) and radiation weighting (w_R)

(A 101) The relationships between RBE and w_R were reviewed in *Publication 92* (ICRP 2003c). The outcome of this review, which involved input from ICRP Committees 1 and 2, was a recommendation that although the w_R values for protons and neutrons required revision, w_R values for other radiations given in *Publication 60* (ICRP 1991b) remained appropriate.

(A 102) For protons of energy >2 MeV it was judged in *Publication 92* that the w_R value of 5 given in *Publication 60* is a significant overestimate of their biological effectiveness and for incident protons of practical importance (> 10 MeV) a w_R of 2 was proposed. For neutrons, *Publication 92* proposed that ICRP continues the use of w_R values that depend upon the energy of the incident neutrons. However, the continuous function given in *Publication 92* (Fig. 1 on page 3) was recommended rather than the step function given in *Publication 60*. *Publication 92* noted that, for practical purposes, this procedure will reduce problems of computation of effective dose but should not be taken to imply precise knowledge of the underlying biological effectiveness. The issues of w_R for neutrons and photons/electrons have been considered further by ICRP Committee 2, and detailed judgements are given in Annex B of these Recommendations.

(A 103) Those Auger-emitting radionuclides and compounds that have the potential to localise to the cell nucleus and bind to DNA were recognised in *Publication 60* as a special case for low-LET radiation. The Commission supports the view given in *Publication 92* that Auger emitters will continue to need special attention in radiological protection and that specific physiological and biophysical data would be needed in order to consider Auger-emitting compounds on a case-by-case basis.

A.4.4. Estimation of cancer risk from epidemiological data

(A 104) The Task Group that drafted this Annex was specifically charged by the Commission with developing nominal risk coefficients for cancer risk and providing recommendations on the transport of risk between populations, the estimation of radiation detriment and the derivation of tissue weighting factors. This was a major new element of work for ICRP Committee 1 and required input from Committee 2 and the Commission. The outcome of this work is summarised below.

Nominal risk coefficients, radiation detriment, and tissue weighting factors

(A 105) Nominal risk coefficients are derived by averaging sex and age-at-exposure lifetime risk estimates in representative populations. In general, cohort studies were preferred for risk assessment, because in retrospective case-control studies, selection biases may be a problem and the dose estimates can be highly uncertain when exposure data come from personal recall without documentation. The lifetime risk estimates are computed using risk estimates specific to various cancer sites. Radiation risk estimates are derived for incidence data for specific tumour sites when adequate dose response data are available from the Japanese Life Span Study (LSS), pooled analyses of multiple studies, or other sources. Incidence data tend to have less diagnostic misclassification than mortality data and provide better estimates for sites that have relatively low lethality. To simplify risk calculations by users of the ICRP system, estimates are derived for males and females combined. Because of the uncertainty in applying risk models generated from one population to another population with different cancer patterns, population-specific nominal risks are averages of risk estimates from alternative models; these are discussed in paragraphs A 110 – A 124. These nominal risks are computed for each site of interest and summed to give the population total nominal risk. The overall site-specific and total nominal risks are computed by averaging the population-specific average risks.

(A 106) Radiation detriment is a concept used to quantify the harmful effects of radiation exposure in different parts of the body. It is determined from nominal risk coefficients, taking into account the severity of the disease in terms of lethality and years of life lost. Total detriment is the sum of the detriment for each part of the body (tissues and/or organs).

(A 107) The concept of 'effective dose' associated with a given exposure involves weighting individual organs and tissues of interest by the relative detriments for these parts of the body. In such a system, the weighted sum of the tissue-specific dose equivalents, called the effective dose, should be proportional to the total estimated

detriment from the exposure, whatever the distribution of equivalent dose within the body. The components of detriment are essentially the same for cancer and heritable disease and, if desired, these detriments may be combined.

(A 108) In general, the risk estimates summarised here are derived as averages across Asian and Euro-American populations. An attempt was made to choose an appropriate model to use for transferring risks across various populations whenever there is sufficient evidence to favour one model over another. The risk modelling was conducted principally with the data from the Japanese Life Span Study of A-bomb survivors (LSS), but the broader radiation epidemiology literature was examined for compatibility with the LSS-derived estimates. For several tissues it was possible to use a group of data sets to estimate cancer risk.

(A 109) The following text briefly outlines the general models of risk and the sources of data used, methodological aspects of the risk estimates, and the detriments associated with a range of tissues. Estimated numerical values and recommendations that derive from this work are summarised in Tables A.4.1, A.4.3, and A.4.4.

(A 110) ***Risk modelling.*** Within a given exposed population, comparable descriptions of the radiation-associated risk can be made using either excess relative risk (ERR) or excess absolute risk (EAR) models, so long as the models allow for variation in the excess risk with factors such as sex, attained age, and age-at-exposure. While suitably data-rich multiplicative (ERR) or additive (EAR) models lead to virtually identical descriptions of the excess risk in the population used to develop the risk estimates, they can lead to markedly different excess risk estimates when applied to populations with different baseline rates.

(A 111) Both ERR and EAR models were developed for oesophagus, stomach, colon, liver, lung, breast, ovary, bladder, thyroid and leukaemia (bone marrow). As noted below, *Publication 60* nominal risks were used for bone and skin cancers (ICRP, 1991b). Because the data for other human tissues and organs are insufficient to individually judge the magnitude of their radiation risk, they were consigned to a 'remainder' category (called 'other solid'). ERR and EAR models were also developed for this group.

(A 112) In general, the parameters in these risk models were estimated using incidence data from the studies of the Japanese atomic bomb survivors with follow-up from 1958 through to 1998 for solid cancers (Preston et al., 2007). For solid cancers these models involved a linear dose response allowing for modifying effects of sex, exposure age, and attained age. These effects were constrained to equal the values seen for all solid cancers as a group unless there were indications that these constraints resulted in a marked reduction in the goodness of fit when modelling cause-specific cancer types. Leukaemia risk estimates were based on an EAR model with a linear-quadratic dose response that allows for effect modification by sex, exposure age, and time following exposure (Preston et al., 1994). Model parameters are given in Section A.4.5.

(A 113) While the LSS studies do provide some information on skin cancer risks (Ron et al., 1998), it was judged that they may not be adequate for a general population because of differences in risk related to skin pigmentation. Therefore, the

Table A.4.1. Summary of sex-averaged nominal risks and detriment.

Tissue	Nominal Risk Coefficient (cases per 10,000 persons per Sv)	Lethality fraction	Nominal risk adjusted for lethality and quality of life*	Relative cancer-free life lost	Detriment (relating to column 1)	Relative detriment+
a) Whole population						
Oesophagus	15	0.93	15.1	0.87	13.1	0.023
Stomach	79	0.83	77.0	0.88	67.7	0.118
Colon	65	0.48	49.4	0.97	47.9	0.083
Liver	30	0.95	30.2	0.88	26.6	0.046
Lung	114	0.89	112.9	0.80	90.3	0.157
Bone	7	0.45	5.1	1.00	5.1	0.009
Skin	1000	0.002	4.0	1.00	4.0	0.007
Breast	112	0.29	61.9	1.29	79.8	0.139
Ovary	11	0.57	8.8	1.12	9.9	0.017
Bladder	43	0.29	23.5	0.71	16.7	0.029
Thyroid	33	0.07	9.8	1.29	12.7	0.022
Bone Marrow	42	0.67	37.7	1.63	61.5	0.107
Other Solid	144	0.49	110.2	1.03	113.5	0.198
Gonads (Heritable)	20	0.80	19.3	1.32	25.4	0.044
Total	***1715***		***565***		***574***	***1.000***
b) Working age population (18–64 years)						
Oesophagus	16	0.93	16	0.91	14.2	0.034
Stomach	60	0.83	58	0.89	51.8	0.123
Colon	50	0.48	38	1.13	43.0	0.102
Liver	21	0.95	21	0.93	19.7	0.047
Lung	127	0.89	126	0.96	120.7	0.286
Bone	5	0.45	3	1.00	3.4	0.008
Skin	670	0.002	3	1.00	2.7	0.006
Breast	49	0.29	27	1.20	32.6	0.077
Ovary	7	0.57	6	1.16	6.6	0.016
Bladder	42	0.29	23	0.85	19.3	0.046
Thyroid	9	0.07	3	1.19	3.4	0.008
Bone Marrow	23	0.67	20	1.17	23.9	0.057
Other Solid	88	0.49	67	0.97	65.4	0.155
Gonads (Heritable)	12	0.80	12	1.32	15.3	0.036
Total	***1179***		***423***		***422***	***1.000***

* Defined as $R*q + R*(1 - q)*((1 - q_{min})\ q + q_{min})$, where R is the nominal risk coefficient, q is the lethality, and $(1 - q_{min})\ q + q_{min}$ is the weight given to non-fatal cancers. Here q_{min} is the minimum weight for non-fatal cancers. The q_{min} correction was not applied to skin cancer (see text).

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

Commission used the nominal skin cancer risk estimate of 0.1 per Gy from *Publication 59* (ICRP, 1991a). This estimate was also used in *Publication 60* (ICRP, 1991b). The nominal risk estimate for bone was also taken from *Publication 60* because the LSS atomic bomb studies provide no data, and other data sources were extremely limited. The low-LET estimate used in *Publication 60* was 0.00065 per Gy. It should

Table A.4.2. Comparison of sex-averaged nominal risks and detriment in whole population based on different methods of calculation.

Tissue	Method of calculation	Nominal risk (cases per 10,000 persons per Sv)			Nominal risk adjusted for lethality and quality of life*	Detriment	Relative detriment+
		Total	Fatal	Non-fatal			
Oesophagus	Current Incidence	15.1	14.0	1.1	15.1	13.1	0.023
	Current Mortality	29.1	27.0	2.1	29.0	25.2	0.037
	BEIR VII	14.1	13.1	1.0	14.1	12.2	0.019
	Current ICRP 60	26.7	24.8	1.9	26.6	23.2	0.032
	ICRP 60 actual	31.6	30.0	1.6	31.5	24.3	0.033
Stomach	Current Incidence	79.1	65.5	13.5	77.0	67.7	0.118
	Current Mortality	72.0	59.7	12.3	70.1	61.7	0.091
	BEIR VII	96.3	79.8	16.5	93.8	82.5	0.129
	Current ICRP 60	56.2	46.6	9.6	54.7	48.1	0.067
	ICRP 60 actual	122.2	110.0	12.2	121.0	100.8	0.139
Colon	Current Incidence	65.4	31.3	34.2	49.4	47.9	0.083
	Current Mortality	71.8	34.3	37.5	54.2	52.6	0.078
	BEIR VII	74.5	35.6	38.9	56.2	54.5	0.085
	Current ICRP 60	245.3	117.2	128.1	185.1	179.5	0.249
	ICRP 60 actual	154.5	85.0	69.5	123.3	102.7	0.142
Liver	Current Incidence	30.3	28.9	1.4	30.2	26.6	0.046
	Current Mortality	67.5	64.4	3.1	67.4	59.3	0.088
	BEIR VII	40.0	38.2	1.8	39.9	35.1	0.055
	Current ICRP 60	15.8	15.0	0.8	15.7	13.8	0.019
	ICRP 60 actual	15.8	15.0	0.8	15.8	15.8	0.022
Lung	Current Incidence	114.2	101.5	12.6	112.9	90.3	0.157
	Current Mortality	110.8	98.6	12.2	109.6	87.7	0.130
	BEIR VII	136.9	121.8	15.1	135.4	108.3	0.169
	Current ICRP 60	70.3	62.5	7.8	69.5	55.6	0.077
	ICRP 60 actual	89.5	85.0	4.5	89.3	80.3	0.111
Bone	Current Incidence	7.0	3.2	3.9	5.1	5.1	0.009
	Current Mortality	7.0	3.2	3.9	5.1	5.1	0.008
	BEIR VII	7.0	3.2	3.9	5.1	5.1	0.008
	Current ICRP 60	7.0	3.2	3.9	5.1	5.1	0.007
	ICRP 60 actual	6.9	5.0	1.9	6.4	6.4	0.009
Skin	Current Incidence	1000.0	2.0	998.0	4.0	4.0	0.007
	Current Mortality	1000.0	2.0	998.0	4.0	4.0	0.006
	BEIR VII	1000.0	2.0	998.0	4.0	4.0	0.006
	Current ICRP 60	1000.0	2.0	998.0	4.0	4.0	0.006
	ICRP 60 actual	1000.0	2.0	998.0	4.0	4.0	0.006
Breast	Current Incidence	112.1	33.0	79.1	61.9	79.8	0.139
	Current Mortality	56.5	16.6	39.8	31.2	40.2	0.059
	BEIR VII	111.9	32.9	78.9	61.8	79.7	0.124
	Current ICRP 60	47.5	14.0	33.5	26.2	33.9	0.047
	ICRP 60 actual	40.0	20.0	20.0	30.0	36.3	0.050

Table A.4.2. (*continued*)

Tissue	Method of calculation	Nominal risk (cases per 10,000 persons per Sv)			Nominal risk adjusted for lethality and quality of life*	Detriment	Relative detriment+
		Total	Fatal	Non-fatal			
Ovary	Current Incidence	10.6	6.0	4.6	8.8	9.9	0.017
	Current Mortality	21.2	12.0	9.2	17.6	19.7	0.029
	BEIR VII	11.5	6.5	5.0	9.6	10.7	0.017
	Current ICRP 60	23.4	13.3	10.2	19.4	21.8	0.030
	ICRP 60 actual	14.3	10.0	4.3	13.0	14.6	0.020
Bladder	Current Incidence	43.4	12	31	23.5	16.7	0.029
	Current Mortality	71.7	20	51	38.7	27.5	0.041
	BEIR VII	51.9	15	37	28.0	19.9	0.031
	Current ICRP 60	100.4	29	72	54.2	38.5	0.053
	ICRP 60 actual	60.0	30	30	45.0	29.3	0.040
Thyroid	Current Incidence	32.5	2.2	30.3	9.8	12.7	0.022
	Current Mortality	23.3	1.6	21.8	7.1	9.1	0.013
	BEIR VII	32.0	2.1	29.9	9.7	12.5	0.020
	Current ICRP 60	120.3	8.0	112.3	36.4	47.0	0.065
	ICRP 60 actual	80.0	8.0	72.0	15.2	15.2	0.021
Bone Marrow	Current Incidence	41.9	28.0	13.9	37.7	61.5	0.107
	Current Mortality	54.2	36.3	18.0	48.9	79.6	0.118
	BEIR VII	41.9	28.0	13.9	37.7	61.5	0.096
	Current ICRP 60	46.9	31.4	15.6	42.3	68.9	0.096
	ICRP 60 actual	50.5	50.0	0.5	50.5	104.0	0.143
Other Solid	Current Incidence	143.8	70.5	73.3	110.2	113.5	0.198
	Current Mortality	226.3	111.0	115.3	173.4	178.6	0.264
	BEIR VII	163.3	80.1	83.2	125.1	128.9	0.201
	Current ICRP 60	196.4	96.3	100.0	150.5	155.0	0.215
	ICRP 60 actual	70.4	50.0	20.4	64.5	58.7	0.081
Gonads (Heritable)	Current Incidence	20.0	16	4	19.3	25.4	0.044
	Current Mortality	20.0	16	4	19.3	25.4	0.038
	BEIR VII	20.0	16	4	19.3	25.4	0.040
	Current ICRP 60	20.0	16	4	19.3	25.4	0.035
	ICRP 60 actual	100.0	100	0	100.0	133.0	0.183
Total	Current Incidence	1715.4	414	1301	564.8	574.3	1
	Current Mortality	1831.4	503	1328	675.4	675.8	1
	BEIR VII	1801.2	474	1327	639.6	640.4	1
	Current ICRP 60	1976.3	479	1497	709.2	719.9	1
	ICRP 60 actual	1835.8	600	1236	709.3	725.3	1

Footnote and numerical values as per Table A.4.1.

Additional notes: The BEIR VII estimates are based on application of the BEIR VII risk models to the combined Euro-American and Asian populations with an assumed DDREF of 2. Nominal risks and detriment values would be increased by 4/3 if the BEIR VII DDREF of 1.5 were used. BEIR VII risks for skin, bone surface, and gonads are taken as the same as the ICRP values since risk estimates for these outcomes were not considered in the BEIR VII lifetime risk estimates. The 'Current ICRP 60' estimates are based on application of the *Publication 60* risk models to the Euro-American and Asian populations used here with an assumed DDREF of 2. The 'ICRP 60 actual' estimates were determined from the data in *Publication 60*.

Table A.4.3. Proposed tissue weighting factors.

Tissue	w_T	$\sum w_T$
Bone-marrow (red), Colon, Lung, Stomach, Breast, Remainder Tissues* (Nominal w_T applied to the average dose to 14 tissues)	**0.12**	0.72
Gonads	**0.08**	0.08
Bladder, Oesophagus, Liver, Thyroid	**0.04**	0.16
Bone surface, Brain, Salivary glands, Skin	**0.01**	0.04

* Remainder Tissues (14 in total): Adrenals, Extrathoracic (ET) region, Gall bladder, Heart, Kidneys, Lymphatic nodes, Muscle, Oral mucosa, Pancreas, Prostate, Small intestine, Spleen, Thymus, Uterus/cervix.

Table A.4.4. Detriment adjusted nominal risk coefficients for cancer and heritable effects (10^{-2} Sv^{-1})[1].

Exposed population	Cancer		Heritable effects		Total	
	Present	ICRP 60	Present	ICRP 60	Present	ICRP 60
Whole	5.5	6.0	0.2	1.3	5.7	7.3
Adult	4.1	4.8	0.1	0.8	4.2	5.6

[1] Values from Tables A.4.1a, A.4.1b, and *Publication 60*.

be noted that the ICRP risk estimate for bone cancer was based on average bone dose from radium–244 while current dosimetric models estimate doses to bone surfaces. As discussed by Puskin et al. (1992), the risk estimate would be a factor of 9 lower if calculated on the basis of dose to the bone surface. However, proposed changes in bone dosimetry will reduce this difference. For the purposes of the present report, the risk estimate based on average bone dose is used while recognising its possible conservatism.

(A 114) The risk models described above were used to compute sex-specific lifetime risk estimates for a range of ages at exposure (0 to 85 years in 5-year intervals) in Asian and Euro-American composite populations as described subsequently. Lifetime risks for exposure ages were then averaged using weights reflecting the age distribution of the full population or for a working age (18–64 year old) population.

(A 115) In *Publication 60*, nominal cancer risks were computed based on mortality data but, in the current report, risk estimates are based principally on incidence data. The reason for the change is that incidence data provide a more complete description of the cancer burden than do mortality data, particularly for cancers that have a high survival rate. In addition, cancer registry (incidence) diagnoses are more accurate and the time of diagnosis is more precise. It is recognised, however, that incomplete coverage of the A-bomb population because of migration from Hiroshima or Nagasaki introduces a factor of uncertainty on risk estimates based on these cancer incidence data. At the time of *Publication 60*, comprehensive incidence data were not available. Since then, a thorough evaluation of cancer incidence in the Life Span Study (LSS) of Japanese atomic bomb survivors has been published (Thompson et al., 1994; Preston et al., 1994). Site-specific risk estimates were taken from the most recent solid cancer incidence analyses of the atomic bomb survivor LSS (Preston

et al., 2007), with follow-up from 1958 through 1998, and adjusted to reduce the bias in risk estimates that is due to uncertainty in individual dose estimates (Pierce et al., 1990). The newly implemented atomic bomb dosimetry system, DS02, is a considerable improvement over DS86. On average, the DS02 dose estimates are slightly greater than the DS86 estimates. Risk estimates using the two systems differ by less than 10% (Preston et al., 2004).

(A 116) Although the primary estimates are based on models derived from the LSS data, information from other radiation-exposed populations was also considered. Such information is available from studies of:

- Patients with therapeutic or diagnostic exposures to radiation;
- Workers exposed to radiation in the course of their job, e.g., uranium miners;
- Persons with environmental exposures, e.g., from fallout or from natural radiation.

(A 117) These studies have been reviewed in detail by UNSCEAR (2000) and the International Agency for Research on Cancer (IARC 2000, 2001). Some of these studies are more informative than others about radiation risks. The LSS is particularly valuable in estimating radiation risks for a general population, because of the very long, mainly prospective follow-up, the large size of the cohort, and the inclusion of persons of all ages and both sexes who received a wide range of doses. In contrast, many studies of medical exposures lack the sample size and dosimetry quality for precise estimation of risk as a function of dose (NAS/NRC 2006). Also, studies of therapeutic exposures often involve doses in excess of 5 Gy, where cell killing may lead to an under-estimation of the cancer risk per unit dose.

(A 118) However, studies other than the LSS can provide information on the effects of exposure received under different circumstances, such as exposure to high-LET rather than low-LET radiation, exposures received in a chronic or fractionated manner rather than acutely, or risks in countries other than Japan. For example, because the baseline rates of breast cancer are very low in Japan, data from seven cohorts in North America and Western Europe were used in addition to the LSS for determining the site-specific risk estimate (Preston et al. 2002). Also, for thyroid cancer, data from four populations exposed to radiation for medical reasons in various countries were considered in addition to the LSS (Ron et al. 1995). As mentioned earlier, the nominal risk estimates for bone and skin are those used in *Publication 60* (ICRP 1991b). These estimates are largely based on studies of groups with medical exposures (e.g., intakes of radium–224 in the case of bone).

(A 119) For cancers at some sites there is reasonable compatibility between the data from the LSS and those from other sources. However, it is recognised by the Commission that there are indications of differences in radiation risks for a number of sites, e.g., lung when compared with radon-exposed miners (UNSCEAR 2000), although here the differences – within a factor of 2–3 – are not large relative to the uncertainties in these estimates. More direct information on the effects of low level radon exposures comes from recent combined analyses of case-control studies that show raised risks of lung cancer from radon exposure in homes (Darby et al.

2005, Krewski et al. 2005, Lubin et al. 2004). Precise comparison with estimates based on the LSS and the miner studies is difficult but, bearing in mind the various uncertainties, the findings appear to be broadly compatible. In *Publication 60*, the liver cancer risk estimate was derived from studies of patients injected with the radioactive contrast medium Thorotrast, whilst in the current report the LSS liver cancer risk estimate was preferred. The LSS estimate is higher than that of other groups exposed to x or gamma radiation (UNSCEAR 2000), probably because of a reported strong interaction between hepatitis virus and radiation in the LSS (Sharp et al. 2003). However, as indicated below, the estimate derived here based on the LSS is similar to that in *Publication 60*. More generally, when the LSS cancer incidence risks were compared to those from medically or occupationally irradiated populations exposed to low-LET external radiation, the risk estimates were broadly compatible (NAS/NRC 2006).

(A 120) *Cancer risk in different tissues.* Nominal cancer risks and tissue weights were developed for 12 tissues and organs (oesophagus, stomach, colon, liver, lung, bone, skin, breast, ovary, bladder, thyroid, and red bone marrow) with the remaining tissues and organs grouped into one 'remainder' category. These individual tissues and organs were selected because it was deemed that there was sufficient epidemiological information on the tumorigenic effects of radiation to make the judgements that were necessary for estimating cancer risks. Leukaemia, excluding chronic lymphocytic leukaemia (CLL), and multiple myeloma were included in the bone marrow category. The remainder category also includes other tissues not explicitly evaluated as individual cancer sites.

(A 121) *Composite populations.* Composite baseline rates were computed using incidence rates averaged across six populations for cancers of the oesophagus, stomach, colon, liver, lung, female breast, ovary, bladder, thyroid, leukaemia (excluding CLL) and solid cancers combined. The aim was to compile rates for representative populations in different parts of the world. Population-based cancer incidence rates were obtained from the 8th edition of Cancer Incidence In Five Continents (Parkin et al. 2002) and population size data were obtained from the WHO international mortality statistics database. In Annex B of *Publication 60* (ICRP 1991b), risks were calculated separately for five different populations. The approach taken here is slightly different, in that cancer rates were compiled for selected Asian (Shanghai, Osaka, Hiroshima and Nagasaki) and Euro-American (Sweden, United Kingdom, US SEER) populations with long-running cancer registries. These rates are shown in Section A.4.5. An unweighted average of the Asian and the Euro-American data was calculated to form a composite population.

(A 122) Sex-specific, all-stage relative survival statistics from the US SEER programme for 1994–1999 (5-year survival) and 1979–1999 (20-year survival) were averaged to compute overall relative survival rates for different cancer sites. Although the SEER relative survival rates are higher than those found for many other European and Asian countries, reducing the survival rates did not change estimates of relative detriment appreciably.

(A 123) *Heritable risks.* The estimate of genetic (heritable) risk from radiation has been substantially revised since the *Publication 60* report as a result of both new

information that has become available and the work of ICRP during the interim. These revised estimates and their derivation are given in Section A.6. Several factors have led to this revision of genetic risk estimates; in brief:

- Most radiation-induced mutations are large multigene deletions, which are more likely to cause multisystem developmental abnormalities rather than single-gene (i.e., Mendelian) diseases. Importantly, only a fraction of these are likely to be compatible with live births.
- Nearly all chronic diseases have a genetic component, but because most of these are multigenic and multifactorial, the mutation component (i.e., the responsiveness of these diseases to an alteration in mutation rate) is small, so that chronic diseases respond only minimally to a radiation-induced increase in mutation rate.
- *Publication 60* (ICRP 1991b) made the implicit assumption that all genetic diseases should be treated as lethal. In view of the range of severity and lethality for the various types of genetic disease, the lethality fraction for genetic diseases has now been explicitly designated as 80%.
- New genetic risk coefficients recommended by ICRP consider exposure and genetic risk for two generations only; the equilibrium value used in *Publication 60* is judged to be of limited scientific validity because of the unsupported assumptions necessary on selection coefficients, mutation component and population changes over hundreds of years.

(A 124) As a result, the risk of heritable effects in the whole population associated with gonadal dose is now estimated to be around 20 cases per 10,000 people per Sv, rather than around 100 cases per 10,000 per Sv in *Publication 60* (see Section 6, Table 6.6). As in *Publication 60*, the risk of heritable effects in the working population is taken to be 60% of that for the whole population. The corresponding relative contribution of the gonadal dose to the total detriment is now estimated as 3–4%, versus the former ~18%.

(A 125) ***Methodological aspects.*** *Uncertainty and sensitivity analyses.* There are uncertainties in radiation risk estimates which stem from several sources. The most familiar is statistical uncertainty, represented by confidence limits or statistical likelihood distributions. For a chronic or low-dose exposure, the estimate and its statistical uncertainty are divided by an uncertain dose and dose-rate effectiveness factor (DDREF), a process that both reduces the estimate and further increases its uncertainty (see below).

(A 126) When an estimate based on a particular exposed population is applied to other populations or to other radiation sources, further uncertainty is introduced. Differences between radiation sources can produce uncertainty owing to random or systematic error in dose estimates in either the original or the secondary population.

(A 127) Risk-based radiological protection depends heavily on the assumption that estimates based on studies of informative exposed populations, such as the Life Span Study cohort of atomic bomb survivors, can be applied to other exposed populations. Combined analyses of dose-response data from different populations (e.g., Preston et al. 2002) provide valuable information relevant to that assumption.

Unfortunately, such information is available for very few site-specific cancers. Transfers of risk estimates between populations pose a particularly difficult problem for cancer sites for which baseline rates differ widely between the two populations. This problem is discussed in more detail below.

(A 128) Other major sources of uncertainty include possible interaction of radiation exposure with other cancer risk factors, notably including smoking history in the case of lung cancer, and reproductive history in the case of female breast cancer. This problem is similar to that of transfer of risk estimates between populations, in that the interaction can be represented as an uncertain linear combination of an additive and a multiplicative model. However, there is epidemiological evidence favouring an additive or submultiplicative interaction in the case of lung cancer and smoking (Pierce et al. 2003, Travis et al. 2002, Lubin et al. 1995), and a multiplicative interaction in the case of breast cancer and reproductive history (Land et al. 1994).

(A 129) Another source of uncertainty is the relative biological effectiveness, relative to high-energy photons, of radiations of different qualities including medical x rays in the 30–200 keV range, electrons, neutrons, protons, and alpha particles. Quantification of such uncertainties has been discussed in detail elsewhere, e.g., NCI/CDC (2003). The use of central values is preferred by the Commission for radiological protection purposes, but it should be kept in mind that RBE values for specific radiations are intrinsically uncertain. Other aspects of uncertainty associated with the possible existence of a low-dose threshold for cancer risk are summarised in Section A.4.4, paragraphs A 173 – A 187. Uncertainties associated with dose estimates for internal radionuclides (e.g., CERRIE, 2004) are noted in *Publication 99* (ICRP, 2005d).

(A 130) *Dose and dose-rate effectiveness factor.* Because it is difficult to detect small risks in epidemiological studies, the dose-specific estimates of radiation-related risk in this report are largely determined by people exposed to acute doses of 200 mSv or greater. However, many of the more contentious issues in radiation protection involve risks from continuous exposures, or fractionated exposures with acute fractions of a few mSv or less. Experimental investigations tend to show that fractionation or protraction of dose is associated with reduced risk, suggesting that dose-specific estimates based on high-dose, acute exposure data should be divided by a dose and dose-rate effectiveness factor (DDREF) for applications to low-dose, continuous, or fractionated exposures.

(A 131) As already noted, direct estimation from epidemiological studies of cancer risks from doses below a few hundred mSv is difficult, largely for reasons of statistical power. Combined analyses of datasets can help increase statistical power, although precise estimation of risks is currently not possible. A recent example concerns a combined analysis of data on cancer mortality among nuclear workers in 15 countries (Cardis et al. 2005). Notwithstanding the large population (around 400,000 workers in the main analysis), the cohort is still relatively young and only 6% of the workers had died by the end of follow-up. Consequently, the confidence intervals for the estimated trends in cancer risk with dose were wide. In particular, the findings were consistent with risks extrapolated from high-dose, acute exposure data using

a DDREF of 2, as well as with a range of other values. Furthermore, part of the increased risk observed for cancers other than leukaemia appeared to be due to confounding caused by smoking. This highlights the impact that relatively small biases might have on studies at low doses.

(A 132) The magnitude of DDREF is uncertain, and has been treated as such in a number of recent reports based on quantitative uncertainty analysis; for example, NCRP (1997), EPA (1999), and NCI/CDC (2003). However, the mean of the probabilistic uncertainty distribution for DDREF employed in those analyses differs little from the value of 2 recommended in *Publication 60* (ICRP, 1991b) and UNSCEAR (1993). A DDREF of 2 is also generally compatible with the animal data noted in Section A.4.2. Recognising uncertainties, the Commission recommends that a DDREF of 2 continues to be used for radiological protection purposes.

(A 133) The Commission notes that the BEIR VII Committee (NAS/NRC, 2006) employed a Bayesian statistical approach to the choice of DDREF based upon a combination of human data from the LSS and results from appropriately selected animal studies. This analysis indicated that DDREF values in the range 1.1–2.3 were consistent with these data, and BEIR VII chose a DDREF value of 1.5 for the purposes of cancer risk estimation. BEIR VII discusses the elements of subjectivity that are inherent in choices of DDREF, and the Commission stresses that its recommendation to retain an ICRP DDREF summary value of 2 for radiological protection purposes is a broad judgement which embodies elements of both subjectivity and probabilistic uncertainty.

(A 134) *Sex averaging.* Some radiation-related cancers are sex-specific and, for many others, sex is a major modifier of radiation-related risk. In accordance with current ICRP procedures, intermediate and final numerical risk estimates presented here are sex-averaged. Radiation risks were also calculated by retaining sex specificity of intermediate results and sex-averaging only at the final stage. The final results were similar, within acceptable limits, for the two methods of calculation, and sex-specific data are not recommended for the general purposes of radiological protection.

(A 135) *Transfer of risk between populations.* If two populations differ with respect to prevalence of known modifiers of radiation-related risk, their responses to radiation exposure might be expected to differ. However, even in the absence of such information, it is problematic to transfer site-specific estimates of radiation-related risk from one population to the other if the corresponding baseline rates differ. For (an extreme) example, the LSS population provides by far the most usable estimates available of radiation-related gastric cancer risk, but age-specific baseline rates differ by a factor of 12 between Japan and the United States. There is rough equivalence between dose-specific excess absolute risk (EAR_{LSS}) and the product of excess relative risk (ERR_{LSS}) and baseline rates for the population of Japan, but the relationship

$$EAR_{LSS} = ERR_{LSS} \times baseline_{Japan}$$

corresponds approximately to

$$EAR_{LSS} = 12 \times ERR_{LSS} \times baseline_{US}$$

(A 136) Thus, a multiplicative model estimate of excess risk for stomach cancer in the US population based on an ERR model, i.e.,

$$\mathrm{ERR}_{\mathrm{mult}} = \mathrm{ERR}_{\mathrm{LSS}}$$

is about one twelfth as high as the estimate based on directly transferring the $\mathrm{EAR}_{\mathrm{LSS}}$:

$$\mathrm{ERR}_{\mathrm{add}} = \mathrm{EAR}_{\mathrm{LSS}}/\mathrm{baseline}_{\mathrm{US}} = \mathrm{ERR}_{\mathrm{LSS}} \times (\mathrm{baseline}_{\mathrm{Japan}}/\mathrm{baseline}_{\mathrm{US}})$$

(A 137) Assuming that ionising radiation exposure acts primarily as a cancer initiator, multiplicative transfer would be plausible if the difference in population rates were associated with differential exposure to cancer promoters, and additive transfer would be plausible if the rate difference could be ascribed to differential exposure to competing cancer initiators. Given little information about radiation-related stomach cancer risk in the US population, or about modification of radiation-related risk by whatever factors are responsible for the 12-fold difference between gastric cancer rates in the two countries, it would not be unreasonable to consider all estimates of the form

$$\mathrm{ERR}_{\mathrm{US}}(p) = p \times \mathrm{ERR}_{\mathrm{add}} + (1 - p) \times \mathrm{ERR}_{\mathrm{mult}}$$

for $0 \leqslant p \leqslant 1$, as being equally likely. With this approach, the overall uncertainty is high, and the mean value, $\mathrm{ERR}_{\mathrm{US}}(1/2)$, does not really represent the range of (presumably) equally likely transfer estimates.

(A 138) For most sites, the difference between Japanese and US rates is considerably less than 12-fold, which means that inability to discriminate between the additive and multiplicative transfer models is less consequential. However, among the sites considered for the present report, only for lung, breast, and thyroid was it considered that there was sufficient information to justify a representative value other than $\mathrm{ERR}_{\mathrm{US}}(1/2)$.

(A 139) Because a pooled analysis of radiation effects on breast cancer risk (Preston et al., 2002) provides strong evidence against the use of common ERR models, breast cancer risks were based solely on an EAR model, namely that based on the A-bomb data. However, the use of EAR models for predicting thyroid cancer risks is problematic because variation in screening intensity will have a marked effect on the rate of radiation-associated thyroid cancers. Therefore, thyroid cancer risks were based solely on the ERR model developed from the pooled analysis of radiation-associated thyroid cancer risks (Ron et al., 1995).

(A 140) Therefore, the population risks were defined as weighted averages of the additive (absolute) and multiplicative excess risk estimates with weights based on judgements concerning the relative applicability of the two risk estimates. Weights of 0.5 were used for all tissues except breast and bone marrow for which only an EAR model was used, thyroid and skin for which only an ERR model was used, and lung for which the ERR model was given a weight of 0.3 because of suggestions in the atomic bomb survivor data that the EAR is more comparable across sexes than the ERR, and also that radiation dose and smoking history interact additively as lung cancer risk factors (Pierce et al., 2003).

(A 141) *Computation of radiation detriment.* As in *Publication 60*, the detriment for a tissue, T, is defined as

$$D_T = (R_{F,T} + q_T R_{NF,T}) l_T$$

where R_F is the nominal risk of fatal disease, R_{NF} is the nominal risk of non-fatal disease, q is a non-fatal weight (between 0 and 1) reflecting the reduced quality of life associated with living with a serious illness, and l is the average life lost due to the disease relative to normal life expectancy, expressed relative to the average over all cancers. As discussed below, the quality of life factor is a function of the lethality (k) of the disease and a subjective judgement accounting for pain, suffering, and adverse effects of treatment. Box 1 summarises the steps by which radiation detriment was calculated for the purposes of developing a system of tissue weighting.

(A 142) Since incidence data are being used here, the nominal risk coefficients are $R_I = R_F + R_{NF}$ and the detriment is computed as

$$(k_T R_{I,T} + q_T(1 - k_T) R_{I,T}) l_T = R_{I,T}(k_T + q_T(1 - k_T)) l_T$$

(A 143) The computations in *Publication 60* were based on nominal mortality risk coefficients, R_F, and q was taken to be equal to the lethality fraction k. Thus, the ICRP *Publication 60* cause-specific detriment is $(R_F + k\,(1 - k)R_F\,/k)l$ which is equal to R_F (2-k)l (cf pages 134–136 and Table B20 in *Publication 60*), where

$$R_{NF} = (1 - k)R_F/k.$$

(A 144) *Quality of life detriment.* Cancer survivors generally experience adverse effects on their quality of life. Thus, the Commission judges that cancers should be weighted not only by lethality but also for pain, suffering, and any adverse effects of cancer treatment. To achieve this, a factor termed q_{min} is applied to the non-lethal fractions of cancers to produce an adjusted lethality fraction termed q_T. The formula used to calculate q_T with an adjustment for non-lethal detriment is:

$$q_T = q_{min} + k_T(1 - q_{min})$$

where k_T is the lethality fraction and q_{min} is the minimum weight for non-lethal cancers.

(A 145) The value of q_{min} was set equal to 0.1 (in most instances the result is not highly sensitive to the value chosen). In effect, the q_{min} adjustment has an impact upon detriment calculations in proportion to the fraction of cancers that are non-lethal. Accordingly, highly lethal cancers such as lung and stomach cancer are little affected by q_{min} whereas relatively non-lethal cancers such as breast or thyroid are. For example, if the lethality of a cancer type was 0.30, the adjusted q_T would be 0.37. However, the q_{min} adjustment was not used for skin cancer because radiogenic skin cancer is almost exclusively of the basal cell type which is usually associated with very little pain, suffering or treatment sequelae.

(A 146) *Lethality adjustment of nominal risk.* The nominal risk coefficients are adjusted to reflect the relative lethality for the cancers (or heritable effects) that occur. Highly lethal cancers receive a relatively greater weight than those that seldom cause death. The lethality adjustment is given by $(R \times q)$, where R is the nominal risk

coefficient for a tumour site and q is its lethality fraction, derived from national cancer survival data.

(A 147) *Relative life lost.* Relative years of life lost is an important component of the detriment computation. Average years of life lost for a given cause was computed for each sex in each composite population as the average over ages at exposure and subsequent attained ages of the residual lifetime. The weights were equal to the number of deaths from the cause of interest in each age group. These were converted to relative values by division by the average years of life lost for all cancers.

(A 148) Table A.4.5 in Section A.4.5 presents the lethality factors, non-fatal case weights, and relative life lost values used in the current computations. *Publication 60* values are shown for comparison.

(A 149) ***Principal features of new estimates of cancer risk.*** In *Publication 60* the ERR and EAR models were given equal weights for various tissues, except for bone marrow. In the present assessment, the relative weights assigned to the ERR and EAR models were allowed to depart from 50:50 when warranted by the available data. This made a more realistic model for the inter-country transfer of radiogenic breast cancer risks, and largely prevented the potential problem of thyroid cancer or skin cancer risk estimates being affected by differing degrees of cancer screening.

(A 150) The present relative detriments (Table A.4.1) are similar to the values calculated in *Publication 60* except for four tissue groups: breast, bone marrow, remainder tissues and gonads. There appear to be several reasons why the relative detriment for breast cancer has increased from 0.05 to 0.139. Those exposed as juveniles in the LSS cohort now make a larger contribution to the overall breast cancer risk, whereas the mortality data used for the *Publication 60* analysis only partially reflected this contribution. Furthermore, in the current incidence analyses (Preston et al. 2007), the ERR estimates for women exposed over age 40 years are higher than those used in *Publication 60*. In the 1958–1987 LSS Tumour Registry report on radiation and solid cancer incidence (Thompson et al. 1994), breast cancers contributed about 11% of the total excess solid cancers as averaged over males and females. In the current analyses, breast cancers account for about 18% of the radiation-associated solid cancers. Studies of other exposed populations have confirmed the substantial breast cancer risk from radiation (Preston et al. 2002). On the other hand, the lethality fraction for breast cancer has decreased in the past 15 years, probably reflecting increased early detection and improved treatments, but this appears to have little impact on the relative detriment estimates.

(A 151) Improved description of the temporal diminution of leukaemia risk has contributed to a reduction in the relative detriment for bone marrow from 0.143 to 0.101. The reduction of gonadal risk has already been explained above and pertains to new information and a revised approach for assessing risks of heritable disease.

(A 152) The further accumulation of LSS data in the period following *Publication 60* has significantly influenced the 'remainder tissues' category. There is now evidence for excess radiation risk, in the aggregate, among a variety of other tissues, although the degree of risk for any single tissue is unclear. Since the risk in the remainder

category is spread over a large number of tissues and organs, the judgement of the Commission is that any given tissue should receive a small weight. This judgement

Box A.1. Steps in the development of the tissue weighting system.

The development of the tissue weighting system was based upon relative radiation detriment, primarily for cancer. The sequential steps used were as follows:

a) Determine lifetime cancer incidence risk estimates for radiation-associated cancers: For 14 organs or tissues, male and female lifetime excess cancer risks were estimated using both the excess relative risk (ERR) and excess absolute risk (EAR) models and were then averaged across sexes.
b) Apply a dose and dose-rate effectiveness factor (DDREF): The lifetime risk estimates were adjusted downward by a factor of two to account for a DDREF (except for leukaemia, where the linear-quadratic model for risk already accounts for the DDREF).
c) Transfer risk estimates across populations: To estimate radiation risk for each cancer site, a weighting of the ERR and EAR lifetime risk estimates was established that provided a reasonable basis for generalising across populations with different baseline risks. (ERR:EAR weights of 0:100% were assigned for breast and bone marrow, 100:0% for thyroid and skin, 30:70% for lung, and 50:50% for all others).
d) Nominal risk coefficients: These weighted risk estimates, when applied to and averaged across seven western and Asian populations, provided the nominal risk coefficients given in Tables A.4.1 and A.4.2.
e) Adjustment for lethality: The lifetime risks for respective cancer sites, which were based on excess incident cancers, were converted to fatal cancer risks by multiplying by their lethality fractions, as derived from representative national cancer survival data.
f) Adjustment for quality of life: A further adjustment was applied to account for the morbidity and suffering associated with non-fatal cancers.
g) Adjustment for years of life lost: Since the age distributions of types of cancers differ, the average ages of the several types of cancer were estimated from national cancer data and converted to average years of life lost when a cancer occurs. An adjustment for years of life lost was then applied to the result of the previous steps.
h) Radiation detriment: The results of the calculations above yielded an estimate of the radiation detriment associated with each type of cancer. These, when normalised to sum to unity, constitute the relative radiation detriments in Table A.4.1.
i) Tissue weighting factors: Since the detailed relative radiation detriments in Table A.4.1 are imprecise because of uncertainties associated with their estimation, they were grouped into four categories broadly reflecting the relative detriments. A group of residual 'remainder tissues' was also added to account for radiation risks to organs or tissues for which detailed radiation-risk calculations were uninformative.

is consistent with the LSS and/or other evidence suggesting the risk is probably very small or that evidence is lacking.

(A 153) In order to provide additional supporting information on factors that influence detriment estimates, the Commission computed site-specific, lethality adjusted nominal risks and detriment values using various methods. The methods used were: 1) the current incidence-based estimates; 2) mortality-based computations using risk models based on the most recent LSS mortality data (Preston et al. 2003) applied to the current composite populations together with the current lethality and life lost factors (i.e., the same as (1), but using risk models derived from current mortality rather than incidence data); 3) mortality-based computations using *Publication 60* ERR models (Table 1, Land and Sinclair 1991) applied to the current composite populations together with the current lethality and life lost factors (i.e., the same as (1), but using the *Publication 60* relative risk models for mortality in place of the models based on current incidence data); and 4) the actual *Publication 60* values.

(A 154) Results of these computations are shown in Table A.4.2. Table A.4.2 also includes computations of relative detriment based upon application of the BEIR VII models (NAS/NRC, 2006) to the combined Euro-American and Asian populations with an assumed DDREF of 2 (see Table A.4.2 footnote). Parameter estimates for the risk models used in method 2 are given in Section A.4.5. It can be seen that the values of relative detriment using incidence- and mortality-based risk models (i.e., approaches (1) and (2) above) are generally similar. There are, however, greater differences for some tissues in respect of the application of *Publication 60* methodology to current data ('Current ICRP 60') and the specific published *Publication 60* values ('ICRP 60 actual'). The application of BEIR VII models (NAS/NRC, 2006) shows similar degrees of difference in relative detriment for some tissues. However, in only a few instances were these differences greater than a factor of 3, and total detriment differed by a factor of less than 2.

(A 155) Overall, these comparative calculations suggest that LSS-based central estimates of radiation cancer risk are reasonably robust and not highly sensitive to choices of risk models.

(A 156) During the computation of sex-averaged values for detriment based on cancer incidence and mortality data the Commission was required to compute male- and female-specific data. These data (Tables A.4.18 and A.4.19 of Section A.4.6) do not contribute specifically to the formulation of the ICRP tissue weighting scheme as summarised in Box A.1, but can act to inform other related judgements. It is emphasised that these sex-specific data have limited utility because the Commission's estimates of nominal risk relate to a nominal population of females and males with typical age distributions and are computed by averaging over age groups and sex; the dosimetric quantity, effective dose, is also computed by age- and sex-averaging.

(A 157) ***The use of relative detriment from incidence data for a tissue weighting system.*** The Commission has made a policy decision that there should be a single set of w_T values that are averaged over both sexes and all ages.

(A 158) However, whilst adhering to this policy, the Commission fully recognises that there are significant differences in risk between males and females (particularly for the breast) and in respect of age at exposure.

(A 159) A set of w_T values could be proposed that closely follows the respective values of relative detriment based on incidence data given in Table A.4.1 together with the supporting comparative data of Table A.4.2. However, the Commission feels that additional judgements need to be exercised to include subjective factors, not reflected in the mathematical formulation of detriment. In particular, the following judgements were applied:

- The detriments for heritable effects and cancer following gonadal irradiation were aggregated to give a w_T of 0.08.
- The detriment of thyroid cancer was set at 0.04 to take account of the concentration of cancer risk in childhood, i.e., young children are considered to be a particularly sensitive subgroup.
- Cancer risk in salivary glands and brain, whilst not specifically quantifiable, is judged to be greater than that of other tissues in the remainder fraction and, for this reason, each is ascribed a w_T of 0.01.

(A 160) Re-ordering of w_T values using the above judgements was made ensuring that these values did not diverge from the relative detriments of Table A.4.1 by more than around two-fold. This reassignment gives a w_T value for the remainder tissues of 0.12. The Commission presents a new proposal on the way in which the weighting of remainder tissues is treated.

(A 161) According to this proposal the w_T for remainder tissues (0.12) is divided equally between the 14 tissues given in the footnote to Table A.4.3, 0.0086 each, which is lower than the w_T for the lowest of the named tissues (0.01). The low cancer risk in connective tissues is taken into account through its contribution to cancer in the named organs specified in Table A.4.3. Cancer risk in adipose tissue is judged to be insignificant and, for this reason, it has not been included in remainder tissues. The number of tissues included in remainder could be increased if necessary. The system preserves additivity in effective doses. This is judged to be an appropriate simplification of the scheme of *Publication 60* in which the w_T for the remainder is divided among the five remainder tissues which receive the highest dose, i.e., a non-additive system. Mass weighting of tissues in the remainder fraction was explored but rejected. The principal reason for this rejection was that the very large disparities in tissue masses caused unacceptable distortions of effective dose for certain radionuclides.

(A 162) On the basis of the detriment data of Tables A.4.1 and A.4.2, plus the judgements summarised above, the Commission proposes the tissue weighting scheme given in Table A.4.3. This scheme which seeks to generally represent tissue-specific radiation detriment is, of necessity, imprecise. In particular, for the remainder tissues there is little or no epidemiological evidence of radiation-associated cancer for individual tissues, and their inclusion is largely a prudent measure. The Commission also emphasises that w_T is solely a radiation protection quantity and is not intended for other purposes, e.g., in judging radiation causation of cancers.

Nominal risk coefficients for cancer and heritable effects

(A 163) New data on the risks of radiation-induced cancer and heritable effects have been used by the Commission in risk modelling and disease detriment calculations in order to estimate nominal risk coefficients.

(A 164) On the basis of these calculations (Table A.4.1) the Commission proposes nominal risk coefficients for lethality-adjusted cancer risk as 5.5 10^{-2} Sv^{-1} for the whole population and 4.1 10^{-2} Sv^{-1} for adult workers aged 18–64. For heritable effects, the lethality-adjusted nominal risk in the whole population is estimated as 0.2 10^{-2} Sv^{-1} and in adult workers as 0.1 10^{-2} Sv^{-1}. These estimates are shown in Table A.4.4, where they are compared with the estimates of detriment used in the 1990 Recommendations of ICRP *Publication 60*. These estimates are intended to apply only to populations and are not recommended for use in estimating risks in individuals or subgroups.

(A 165) In respect of Table A.4.4 it is important to note that the detriment-adjusted nominal risk coefficient for cancer estimated here has been computed in a different manner from that of *Publication 60*. The present estimate is based upon lethality/life-impairment-weighted data on cancer incidence with adjustment for relative life lost, whereas in *Publication 60* detriment was based upon fatal cancer risk weighted for non-fatal cancer, relative life lost for fatal cancers and life impairment for non-fatal cancer. In this respect it is also notable that the detriment-unadjusted nominal risk coefficient for fatal cancer in the whole population that may be projected from the cancer incidence-based data of Table A.4.1a is around 4% per Sv as compared with the *Publication 60* value of 5% per Sv. The corresponding value using cancer mortality-based models is essentially unchanged at around 5% per Sv.

(A 166) An additional point relating to the present detriment-adjusted cancer coefficients of Table A.4.4 is that, during the period that the present ICRP Recommendations are likely to apply, the survival rates for many cancers are expected to rise. In this respect the nominal risk coefficient proposed here will tend to be an overestimate of risks in the future.

(A 167) The differences in the estimates of detriment-adjusted heritable effects between the present report and *Publication 60* are explained and discussed in Section A.6.5.

Cancer risk following prenatal (in-utero) irradiation

(A 168) Studies on cancer risk following irradiation of the unborn child were reviewed in *Publication 90* (ICRP 2003a).

(A 169) The largest case-control study of cancer after in-utero irradiation, the Oxford Study of Childhood Cancers (OSCC), found that radiation increased all types of childhood cancer by approximately the same degree. The second largest study showed a larger relative risk of leukaemia than for solid tumours, while several cohorts studies of in-utero radiation found no clear evidence of radiation-induced childhood cancer. The limited data from the atomic bomb survivors suggest that the lifetime cancer risk from in-utero exposure may be similar to that from exposure in early childhood.

(A 170) The OSCC data suggest that cancer induction is at least as likely following exposure in the first trimester as in later trimesters. From the data published to date,

it is not possible to determine tissue-weighting factors in order to define cancer risk in different tissues and organs. Adequate human in-utero exposure data are not available to define the dose and dose-rate effectiveness factor (DDREF) for low-LET radiation or the RBE values for neutron or other high-LET radiations.

(A 171) Given the limitations of the available data, the Commission has not attempted to derive a specific value for the nominal coefficient for life-time cancer risk after prenatal exposure, and supports the *Publication 90* (ICRP, 2003a) judgement that it is reasonable to assume that this risk is, at most, a few times that of the population as a whole. This in-utero risk is judged to be no greater than that following exposure in early childhood.

Genetic susceptibility to radiation-induced cancer

(A 172) On the basis of the data analyses and judgements developed in *Publication 79* (ICRP, 1998a) and further information reviewed by UNSCEAR (2000), UNSCEAR (2001), and the BEIR VII Committee (NAS/NRC, 2006), the Commission believes that strongly expressing, high penetrance, cancer genes are too rare to cause significant distortion of the population-based estimates of low-dose radiation cancer risk made in this Section of the report. However, as noted in *Publication 79*, there are likely to be implications for individual cancer risks, particularly for second cancers in gene carriers receiving radiotherapy for a first neoplasm. Although the Commission recognises that weakly expressing variant cancer genes may, in principle, be sufficiently common to impact upon population-based estimates of radiation cancer risk, the information available is not sufficient to provide a meaningful quantitative judgement on this issue.

The possibility of non-linear low-dose responses for cancer risk

(A 173) The emergence of new data and hypotheses frequently poses questions on the validity of scientific hypotheses and their practical applications. This is certainly the case in radiological protection and particularly for the so-called linear-non-threshold (LNT) model and the derived LNT model used for projecting cancer risk to low doses and low dose rates (UNSCEAR, 2000, CERRIE, 2004, NAS/NRC, 2006, French Academies Report 2005). As given below, there are two principal categories of challenge, both of which hypothesise non-linear low-dose responses.

(A 174) ***Supra-linear low-dose responses.*** It has been proposed by some that the radiation dose response for cancer induction has a supra-linear component at low doses (i.e., a bimodal dose-response relationship) and therefore the projection of low-dose risk from observations made at higher doses will lead to a substantial underestimate of the true risk (CERRIE 2004 and references therein). Such hypotheses are frequently cited in association with reports on unusual epidemiological and experimental observations.

(A 175) The UK CERRIE Committee (CERRIE 2004) considered the scientific validity of claims of such underestimation of cancer risk, particularly in respect of internal radiations. The claims considered were largely based upon; a) the interpretation of selected epidemiological datasets; b) biophysical proposals on the mode of action of certain internal radiations; c) the role of induced genomic instability/

bystander signalling in cancer development; and d) the fitting of bimodal or polymodal dose-responses to epidemiological and experimental data.

(A 176) The Commission agrees with the general view expressed by the majority of CERRIE members that none of the proposals on the gross underestimation of risk that were considered have a sound scientific basis and that some are demonstrably flawed. The following points illustrate the views of the Commission:

- The epidemiological evidence cited did not provide consistent evidence that risk of childhood leukaemia from nuclear test fallout was seriously underestimated by established radiation risk models.
- The so-called Second Event Theory cited in support of higher than expected cancer risk from ^{90}Sr and particulate forms of alpha-emitters was inadequately formulated and inconsistent with a well-established body of biological data.
- The association between induced genomic instability/bystander signalling and cancer risk has yet to be established adequately (see Section A.4.1, paragraph A 97).
- The data relating to bimodal/polymodal dose responses were generally weak, the statistical analyses were inadequate, and the phenomena, if real, had no obvious mechanistic basis.

(A 177) While recognising considerable uncertainty on estimates of cancer risk at low doses, the Commission judges that the data and theories concerning supra-linear dose response do not provide evidence that the application of current cancer risk models based upon the linear-non-threshold (LNT) model and application of the concept of effective dose leads to a gross underestimate of cancer risk.

Table A.4.5. Values for lethality factors, non-fatal case weights, and relative life lost values used in the current computations, together with the corresponding values in *Publication 60*.

Site	Current			ICRP 60	
	Lethality (k)	Non-fatal case weight (q)	Relative life lost	Lethality ($k = q$)	Relative life lost
Oesophagus	0.93	0.935	0.87	0.95	0.65
Stomach	0.83	0.846	0.88	0.90	0.83
Colon	0.48	0.530	0.97	0.55	0.83
Liver	0.95	0.959	0.88	0.95	1.00
Lung	0.89	0.901	0.80	0.87	0.90
Bone	0.45	0.505	1.00	0.72	1.00
Skin	0.002	0.002	1.00	–	1.00
Breast	0.29	0.365	1.29	0.50	1.21
Ovary	0.57	0.609	1.12	0.70	1.12
Bladder	0.29	0.357	0.71	0.50	0.65
Thyroid	0.07	0.253	1.29	0.10	1.00
Bone Marrow	0.67	0.702	1.63	0.99	2.06
Other Solid	0.49	0.541	1.03	0.71	0.91
Gonads	0.80	0.820	1.32	–	1.33

k, q and the relative life lost are defined in Section A.4, paragraphs A 141 – A 148. In particular, q is taken as $q_{min} + (1 - q_{min})*k$ in the current calculations, where q_{min} is 0 for skin, 0.2 for thyroid and 0.1 for all other sites.

(A 178) ***Dose thresholds.*** In the preceding discussion and computations it has been assumed that, at low doses and at low dose rates, site-specific cancer risk from low-LET radiation is proportional to radiation dose, consistent with the LNT model. Thus, the possibility that there might be a threshold dose, below which there would be no radiation-related cancer risk, has been ignored. The LNT model is not universally accepted as biological truth, but rather, because we do not actually know what level of risk is associated with very-low-dose exposure, it is considered to be a prudent judgement for public policy aimed at avoiding unnecessary risk from exposure.

(A 179) As discussed at length in *Publication 99* (ICRP, 2005d), the LNT model receives considerable, although not decisive, support from epidemiological studies of radiation-related cancer risk, in the sense that the risk of mortality and morbidity from all solid cancers combined in the LSS is proportional to radiation dose down to about 100 mGy, below which statistical variation in baseline risk, as well as small and uncontrollable biases, increasingly tend to obscure evidence concerning any radiation-related risk. This uncertainty is the main reason why it is generally impossible to determine, on epidemiological grounds alone, that there is, or is not, an increased risk of cancer associated with radiation exposures of the order of a few tens

Table A.4.6. Coefficients in the current cancer incidence-based ERR models.

Site	Sex	ERR per Gy at age 70 for exposure at age 30	Age at exposure: % change in ERR per decade increase	Power of attained age by which the ERR varies	F:M ratio	$P^{*}_{Consistency}$
All solid	M	0.35	–17%	–1.65	1.65	
	F	0.58				
Oesophagus	M	0.40	–17%	–1.65	1.65	>0.5
	F	0.65				
Stomach	M	0.23	–17%	–1.65	1.65	>0.5
	F	0.38				
Colon	M	0.68	–17%	–1.65	0.48	0.006
	F	0.33				
Liver	M	0.25	–17%	–1.65	1.65	>0.5
	F	0.40				
Lung	M	0.29	+17%	–1.65	4.77	0.09
	F	1.36				
Breast	F	0.87	0%	–2.26	–	0.37
Ovary	F	0.32	–17%	–1.65	–	>0.5
Bladder	M	0.67	–17%	–1.65	1.65	0.27
	F	1.10				
Thyroid	M	0.53	–56%	0.00	2.00	0.04
	F	1.05				
Other	M	0.22	–34%	–1.65	0.78	0.50
	F	0.17				

* P-values are for tests of the hypothesis that the age, age-at-exposure, and (where relevant) sex effects on the ERR describe the LSS data better than do those from a tissue-specific analysis. An exception arises for thyroid cancer in which case the P-value is for a test of the hypothesis that the model used in BEIR VII (NAS/NRC, 2006), which was based on results of the pooled analysis (Ron et al. 1995), adequately describes the current LSS data.

of mSv and below. Risk estimates for such exposures are obtained through the use of mathematical models that assume a simple relationship, e.g., linear, linear-quadratic, or linear with a dose and dose rate effectiveness factor (DDREF) between risk at higher doses, where epidemiological data tend to be informative, and at doses so low that direct epidemiological observation is uninformative.

(A 180) In spite of the biological evidence supporting the LNT model with respect to the induction by ionising radiation of complex DNA damage, for which repair mechanisms in mammalian species tend to be error-prone, the possibility of a threshold for cancer induction at some unknown low dose cannot be ruled out (see Section A.4.1).

(A 181) At the molecular level, the generation of multiple DNA lesions within close spatial proximity, creating complex damage for which mammalian repair mechanisms tend to be error-prone, is believed to be the primary mechanism by which ionising radiation contributes to the induction of mutations and chromosome aberrations and hence to the pathogenesis of cancer. Such clustered damage in DNA

Table A.4.7. Coefficients in the current cancer incidence-based EAR models.

Site	Sex	Excess deaths per 10000 persons per year per Gy at age 70 for exposure at age 30	Age at exposure: % change in EAR per decade increase	Power of attained age by which the EAR varies	F:M ratio	$P^{a}_{Consistency}$
All Solid	M	43.20	−24%	2.38	1.38	
	F	59.83				
Oesophagus	M	0.48	64%	2.38	1.38	0.08
	F	0.66				
Stomach	M	6.63	−24%	2.38	1.38	>0.5
	F	9.18				
Colon	M	5.76	−24%	2.38	0.42	0.02
	F	2.40				
Liver	M	4.18	−24%	2.38	0.31	0.06
	F	1.30				
Lung	M	6.47	1%	4.25	1.38	<0.001
	F	8.97				
Breast	F	10.9	−39%	3.5*	—	0.06
				1.0		
Ovary	F	1.47	−24%	2.38	—	>0.5
Bladder	M	2.00	−11%	6.39	1.38	0.01
	F	2.77				
Thyroid	M	0.69	−24%	0.01	3.36	<0.001
	F	2.33				
Other	M	7.55	−24%	2.38	1.38	0.12
	F	10.45				

[a] P-values are for tests of the hypothesis that the age, age-at-exposure, and (where relevant) sex effects on the EAR describe the LSS data better than do those from a tissue-specific analysis. An exception arises for breast cancer, in which case the P-value is for a test of the hypothesis that the model based on the pooled analysis described by Preston et al. (2002) adequately describes the current LSS data.

[*] The upper term is the age effect before age 50 and the lower term is the effect for age greater than 50.

can, in principle, be induced even by a single radiation track through a cell. Also, while many of the viable cells containing such radiation-induced damage may be eliminated by damage response pathways involving cell cycle checkpoint control and apoptotic cell death, it is clear from analysis of cytogenetic and mutation data that damaged or altered cells are capable of evading these protective measures and propagating.

(A 182) Recent studies using newly developed animal models of radiation tumorigenesis support the view that the essential radiation-associated events in the tumorigenic process are predominantly early events involving DNA losses targeting specific genomic regions harbouring critical genes (see Section A.2.7, paragraphs A 41 – A 44). As such, the response for early initiating events is likely to correspond to that for the induction of cytogenetic and mutagenic damage. On this basis, mechanistic arguments support a linear response in the low-dose region, i.e., the process should be independent of dose rate because interactions between different electron tracks should be rare. Quantitative analyses of dose responses for tumorigenesis and for life shortening in laboratory animals also tend to support this prediction albeit with considerable quantitative uncertainty.

(A 183) There are also long-standing arguments on whether some form of low-dose stimulation of anti-tumorigenic components of the immune system might serve to reduce cancer risk. Such proposals have been considered in depth by UNSCEAR (UNSCEAR 1993, 1994), and the Commission shares the doubts of UNSCEAR that

Table A.4.8. Coefficients in the current mortality-based ERR models.

Site	Sex	ERR per Gy at age 70 for exposure at age 30	Age at exposure: % change in ERR per decade increase	Power of attained age by which the ERR varies	F:M ratio	$P_{Consistency}$
Solid	M	0.35	−31%	−0.74	1.68	
	F	0.58				
Oesophagus	M	0.76	−31%	−0.74	1.68	0.47
	F	1.27				
Stomach	M	0.26	−31%	−0.74	1.68	0.48
	F	0.43				
Colon	M	0.25	−31%	−4.46	1.00	0.43
	F	0.25				
Liver	M	0.21	−31%	−0.74	1.68	0.94
	F	0.34				
Lung	M	0.55	−4%	−0.74	1.68	0.76
	F	0.92				
Breast	F	0.96	−31%	−0.74		0.70
Ovary	F	0.67	−31%	−0.74		0.67
Bladder	M	0.74	12%	−0.74	1.68	0.75
	F	1.24				
Other	M	0.13	−56%	−0.74	1.68	0.40
	F	0.22				

the immune system plays a significant role in any cancer-related adaptive processes at low doses (UNSCEAR 2000).

(A 184) As discussed in *Publication 99*, the statistical uncertainty highlighted earlier in this section is accompanied by other uncertainties, on the model assumptions needed to estimate the risk of radiation-related cancer at low radiation doses. These latter uncertainties are usually subject to only subjective quantification. Such uncertain assumptions include, among others, the DDREF to be applied at low doses and low dose rates, the relationship between excess and baseline cancer rates when transferring estimates from one population to another, and the relationship between estimated and true radiation dose in the exposed population from which the risk estimate was derived (see paragraphs A 125–A 148). All of these assumptions can profoundly affect the estimated risk and its probabilistic uncertainty limits. If one also allows for the uncertain possibility of a universal threshold dose at some known level, or a threshold the value of which is highly uncertain or which varies widely among members of the exposed population, this also affects the risk estimate and its uncertainty limits. In an analysis described in*Publication 99* it was found that, unless the existence of a threshold was assumed to be virtually certain, and its possible values restricted well beyond that which can be justified on current knowledge, the effect of introducing the uncertain possibility of a threshold was equivalent to that

Table A.4.9. Coefficients in the current mortality-based EAR models.

Site	Sex	Excess deaths 10000 persons per year per Gy at age 70 for exposure at age 30	Age at exposure: % change in EAR per decade increase	Power of attained age by which the EAR varies	F:M ratio	$P_{Consistency}$
All Solid	M	28.91	−24%	3.63	1.04	
	F	29.99				
Oesophagus	M	0.98	−24%	3.63	1.00	0.42
	F	0.98				
Stomach	M	5.79	−24%	3.63	1.00	0.45
	F	5.79				
Colon	M	2.24	−24%	3.63	1.00	0.66
	F	2.24				
Liver	M	6.46	−24%	5.56	0.37	0.42
	F	2.36				
Lung	M	6.72	−24%	6.56	1.00	0.70
	F	6.72				
Breast	F	15.73	−44%	5.78[a]		0.01[b]
				−2.83		
Ovary	F	1.40	−24%	3.63		0.90
Bladder	M	0.83	0%	8.04	1.00	0.23
	F	0.83				
Other	M	3.68	−52%	3.63	1.00	0.29
	F	3.68				

[a] Test of hypothesis that a spline in attained age is unnecessary.
[b] The upper term is the age effect before age 50 and the lower term is the effect for age greater than 50.

Table A.4.10. Female Euro-American cancer incidence rates by age and site.

Number of cases per 100,000 persons per year

Age	All cancer	All solid	Oesophagus	Stomach	Colon	Liver	Lung	Breast	Ovary	Bladder	Thyroid	Leukaemia	Non-CLL leukaemia	CLL
0–4	18.37	10.95	0.00	0.01	0.01	0.32	0.01	0.02	0.05	0.06	0.01	6.95	6.92	0.03
5–9	9.03	5.28	0.00	0.01	0.03	0.03	0.04	0.00	0.23	0.00	0.08	3.07	3.05	0.02
10–14	10.20	6.57	0.00	0.04	0.11	0.04	0.02	0.01	0.69	0.00	0.54	2.15	2.15	0.00
15–19	17.49	11.03	0.01	0.08	0.25	0.07	0.04	0.12	1.77	0.07	1.80	2.20	2.19	0.00
20–24	29.46	21.96	0.02	0.09	0.36	0.09	0.19	1.19	2.89	0.19	3.87	1.63	1.59	0.04
25–29	51.15	43.58	0.04	0.27	0.83	0.17	0.39	7.17	4.03	0.31	5.60	1.66	1.61	0.04
30–34	83.77	76.06	0.10	0.75	1.27	0.24	1.04	23.53	5.82	0.50	6.38	1.90	1.86	0.04
35–39	137.56	129.33	0.13	1.17	3.27	0.39	3.20	54.12	9.00	0.98	7.00	2.41	2.27	0.14
40–44	227.67	215.47	0.50	2.28	6.00	0.64	8.29	107.57	13.73	1.85	7.20	3.72	3.41	0.31
45–49	372.68	355.20	1.07	3.31	11.90	1.42	20.20	183.33	24.54	4.05	8.48	4.52	3.72	0.80
50–54	540.14	512.41	2.42	5.02	21.92	2.43	40.44	243.57	34.33	7.90	8.07	7.61	5.28	2.34
55–59	703.34	663.31	5.27	8.76	41.98	4.07	67.32	263.17	41.39	13.25	7.97	9.99	6.59	3.40
60–64	907.16	851.75	7.92	14.26	63.80	6.73	106.00	298.07	49.35	22.38	7.16	15.15	9.82	5.33
65–69	1127.22	1048.58	11.24	21.99	94.46	9.82	154.72	305.57	55.60	33.45	7.79	21.91	12.96	8.94
70–74	1385.31	1279.59	16.96	33.48	138.10	14.11	190.74	328.61	62.04	47.83	8.53	30.29	17.72	12.57
75–79	1557.27	1427.72	21.52	47.53	177.76	17.32	191.05	339.09	61.42	56.59	8.13	37.99	21.96	16.03
80–84	1707.07	1565.32	26.77	65.22	234.14	22.02	166.82	365.99	56.31	68.67	8.73	43.94	26.88	17.05
85–89	1660.82	1667.88	34.82	76.14	241.25	21.66	127.96	335.97	49.39	83.68	8.73	43.98	26.91	17.07
90+	1720.81	1706.61	23.34	73.73	266.50	16.94	76.51	382.23	38.63	54.69	8.73	73.39	44.90	28.48

Table A.4.11. Male Euro-American cancer incidence rates by age and site.

Number of cases per 100,000 persons per year

Age	All cancer	All solid	Oesophagus	Stomach	Colon	Liver	Lung	Breast	Ovary	Bladder	Thyroid	Leukaemia	Non-CLL leukaemia	CLL
0–4	21.64	12.70	0.00	0.01	0.00	0.62	0.01			0.12	0.00	7.78	7.77	0.01
5–9	11.66	6.18	0.00	0.00	0.00	0.10	0.00			0.01	0.05	3.80	3.80	0.00
10–14	12.26	6.18	0.00	0.00	0.06	0.05	0.03			0.02	0.13	3.07	3.07	0.00
15–19	18.72	11.10	0.00	0.06	0.13	0.10	0.11			0.10	0.43	2.73	2.73	0.00
20–24	29.00	20.81	0.02	0.10	0.33	0.15	0.19			0.39	0.77	1.98	1.98	0.00
25–29	43.12	32.54	0.09	0.27	0.92	0.22	0.36			0.60	1.54	2.36	2.33	0.03
30–34	58.48	45.37	0.21	0.82	1.75	0.32	0.99			1.27	1.47	2.87	2.80	0.07
35–39	77.82	61.65	0.64	1.45	3.15	0.72	3.19			2.52	1.78	3.61	3.20	0.41
40–44	115.96	95.95	1.94	3.27	6.71	2.06	9.41			5.70	2.15	4.65	3.81	0.84
45–49	198.61	170.47	4.26	6.02	12.42	3.12	23.28			12.63	2.83	6.67	4.85	1.82
50–54	380.05	337.58	9.47	11.72	25.26	5.53	56.22			25.29	3.34	11.59	7.20	4.38
55–59	676.04	617.96	15.68	21.64	47.90	9.60	108.53			46.07	3.81	16.47	9.56	6.91
60–64	1136.55	1053.31	24.79	36.02	84.67	15.00	189.00			79.67	4.16	25.34	14.06	11.28
65–69	1767.07	1651.87	33.72	58.28	129.65	22.80	304.06			132.28	5.24	37.75	20.92	16.83
70–74	2415.76	2255.06	46.59	87.72	185.35	30.88	400.78			184.53	5.69	56.29	30.97	25.33
75–79	2882.34	2680.83	49.57	114.49	248.89	36.70	456.24			229.94	5.98	68.43	39.48	28.95
80–84	3225.05	2983.09	55.88	145.00	310.36	36.96	459.96			275.56	6.26	86.36	50.15	36.21
85–89	3033.46	3166.00	59.36	165.76	316.71	37.73	404.07			266.44	6.26	91.89	38.53	53.36
90+	3676.73	3290.99	49.36	137.84	335.18	39.21	337.79			376.32	6.26	102.86	43.13	59.73

Table A.4.12. Female Euro-American cancer mortality rates by age and site.

Number of deaths per 100,000 persons per year

Age	All cause	All cancer	All solid	Oesophagus	Stomach	Colon	Liver	Lung	Breast	Ovary	Bladder	Leukaemia	Non-CLL leukaemia	CLL
0–4	114.61	2.22	1.46	0.00	0.00	0.00	0.06	0.02	0.00	0.01	0.00	0.76	0.76	0.00
5–9	11.35	2.01	1.42	0.00	0.00	0.00	0.02	0.01	0.00	0.01	0.01	0.59	0.59	0.00
10–14	13.28	2.05	1.34	0.00	0.02	0.01	0.02	0.01	0.00	0.03	0.00	0.71	0.71	0.00
15–19	28.51	2.76	1.74	0.00	0.03	0.04	0.05	0.02	0.00	0.10	0.00	1.02	1.02	0.00
20–24	33.03	3.40	2.46	0.01	0.05	0.06	0.10	0.04	0.09	0.21	0.00	0.94	0.94	0.00
25–29	40.17	5.97	5.10	0.02	0.14	0.21	0.11	0.10	0.96	0.31	0.01	0.87	0.87	0.00
30–34	55.43	12.77	11.86	0.04	0.41	0.35	0.15	0.53	3.85	0.74	0.06	0.91	0.91	0.00
35–39	81.36	26.07	24.79	0.10	0.69	1.11	0.28	1.90	9.49	1.41	0.09	1.27	1.27	0.00
40–44	122.96	48.98	47.14	0.30	1.23	2.02	0.58	5.45	18.24	3.34	0.19	1.84	1.84	0.00
45–49	193.21	88.79	86.48	0.87	1.76	4.59	1.07	13.34	31.03	7.13	0.49	2.31	2.31	0.00
50–54	309.20	150.52	147.17	1.87	2.98	8.82	1.82	28.25	45.67	13.39	1.00	3.34	3.34	0.00
55–59	489.59	232.48	227.46	3.93	5.16	16.19	3.28	48.94	57.28	21.10	1.82	5.15	5.02	0.13
60–64	801.25	343.06	335.47	6.24	8.47	25.88	5.31	81.35	68.26	27.83	3.70	7.59	7.59	0.00
65–69	1283.49	487.75	476.42	9.10	14.54	39.32	8.87	123.13	82.37	34.97	6.63	12.06	11.33	0.73
70–74	2098.33	654.11	636.96	13.79	21.54	58.94	12.40	158.51	97.91	42.39	11.95	17.97	17.15	0.83
75–79	3406.46	801.53	778.31	20.07	32.16	81.11	16.83	167.46	117.85	45.48	17.98	25.36	23.22	2.15
80–84	5934.90	988.90	956.69	26.37	47.48	118.84	21.81	159.62	146.37	47.35	29.09	35.14	32.21	2.94
85–89	9876.82	1178.13	1146.03	35.87	64.84	165.46	26.79	137.93	188.77	46.61	48.53	38.97	35.71	3.25
90+	19441.90	1220.69	1172.64	24.05	62.78	182.78	20.95	82.47	214.76	36.46	31.72	65.02	59.59	5.43

Table A.4.13. Male Euro-American cancer mortality rates by age and site.

Number of deaths per 100,000 persons per year

Age	All cause	All cancer	All solid	Oesophagus	Stomach	Colon	Liver	Lung	Breast	Ovary	Bladder	Leukaemia	Non-CLL leukaemia	CLL
0–4	143.02	2.75	1.97	0.00	0.00	0.00	0.11	0.00			0.00	0.78	0.78	0.00
5–9	15.39	2.74	1.70	0.00	0.00	0.00	0.05	0.01			0.01	1.04	1.04	0.00
10–14	19.43	2.52	1.39	0.00	0.00	0.01	0.02	0.01			0.01	1.12	1.12	0.00
15–19	66.78	3.50	2.10	0.00	0.01	0.04	0.05	0.02			0.00	1.41	1.41	0.00
20–24	94.71	4.50	3.27	0.02	0.06	0.13	0.09	0.12			0.01	1.23	1.23	0.00
25–29	99.79	5.87	4.56	0.05	0.14	0.28	0.12	0.20			0.01	1.31	1.31	0.00
30–34	124.33	9.09	7.75	0.18	0.36	0.55	0.21	0.64			0.05	1.34	1.34	0.00
35–39	160.80	16.28	14.65	0.48	0.83	1.12	0.50	2.23			0.14	1.63	1.63	0.00
40–44	224.83	34.98	32.89	1.66	1.78	2.46	1.33	7.19			0.46	2.08	2.08	0.00
45–49	321.50	69.83	67.16	3.62	3.33	5.22	2.38	18.84			1.00	3.09	2.67	0.42
50–54	505.70	143.81	139.31	7.94	6.11	10.74	3.90	45.14			2.87	4.79	4.50	0.30
55–59	821.44	262.09	254.99	13.88	11.61	20.26	7.03	89.61			6.09	7.64	7.11	0.54
60–64	1378.11	457.53	446.19	21.98	21.78	35.75	11.69	162.02			12.33	12.85	11.34	1.51
65–69	2241.12	734.15	714.15	30.93	34.77	56.32	17.62	260.63			23.18	20.56	20.00	0.56
70–74	3590.14	1065.72	1036.77	41.20	53.11	85.62	24.51	354.10			39.44	32.65	28.94	3.70
75–79	5634.15	1427.76	1387.32	49.19	75.51	116.26	31.46	421.65			61.53	45.15	40.44	4.71
80–84	9122.79	1880.96	1826.90	55.21	103.50	165.63	36.27	464.57			96.92	64.25	54.06	10.19
85–89	13879.10	2208.86	2287.11	63.41	132.47	221.43	37.50	445.09			135.96	82.03	69.02	13.01
90+	24029.19	2677.26	2377.40	52.73	110.15	234.35	38.98	372.08			192.04	91.82	77.26	14.57

Table A.4.14. Female Asian cancer incidence rates by age and site.

Number of cases per 100,000 persons per year

Age	All cancer	All solid	Oesophagus	Stomach	Colon	Liver	Lung	Breast	Ovary	Bladder	Thyroid	Leukaemia	Non-CLL leukaemia	CLL
0–4	16.18	10.16	0.00	0.00	0.00	0.41	0.00	0.00	0.017	0.23	0.00	4.63	4.63	0.00
5–9	7.47	4.04	0.00	0.00	0.00	0.15	0.00	0.00	0.248	0.00	0.18	2.44	2.44	0.00
10–14	10.32	6.13	0.00	0.00	0.00	0.15	0.05	0.00	1.170	0.00	0.55	3.25	3.25	0.00
15–19	9.62	7.27	0.00	0.20	0.30	0.11	0.12	0.00	1.485	0.00	1.54	1.62	1.62	0.00
20–24	16.76	13.77	0.00	0.95	0.26	0.22	0.14	0.51	2.075	0.06	3.26	1.58	1.58	0.00
25–29	29.87	26.73	0.11	2.41	1.52	0.32	0.86	3.62	2.492	0.15	3.84	1.76	1.76	0.00
30–34	61.04	56.94	0.05	8.54	2.40	0.92	1.26	14.77	3.452	0.13	5.74	2.02	2.02	0.00
35–39	113.76	107.71	0.20	15.25	5.53	2.25	2.97	38.85	5.848	0.43	6.78	3.29	3.27	0.01
40–44	184.71	177.61	0.65	24.58	9.34	3.69	7.70	67.94	9.592	0.75	10.45	3.93	3.92	0.01
45–49	242.53	233.01	1.15	27.18	16.76	5.89	12.55	86.55	13.050	0.94	13.31	4.26	4.18	0.08
50–54	302.19	290.49	2.17	34.98	28.27	11.12	19.96	81.36	15.142	2.80	12.54	6.02	5.89	0.13
55–59	401.39	386.17	6.38	52.62	44.43	21.21	34.36	76.81	16.122	4.62	11.59	5.96	5.60	0.36
60–64	592.40	565.68	12.35	75.78	71.50	46.70	63.49	88.33	19.615	7.49	12.86	9.70	9.19	0.51
65–69	776.54	744.60	17.66	113.21	89.08	75.39	89.27	86.57	19.888	10.82	12.59	11.11	10.75	0.36
70–74	1017.79	974.89	28.42	159.53	126.39	84.23	145.22	84.42	20.507	18.15	13.96	15.34	14.84	0.49
75–79	1177.00	1127.05	34.69	195.44	138.59	96.89	171.64	82.73	20.268	25.43	13.00	14.35	13.56	0.79
80–84	1338.05	1279.76	38.69	260.54	152.09	111.69	176.17	82.34	15.482	35.23	11.16	19.49	18.58	0.92
85–89	1470.65	1400.73	28.65	284.69	174.60	114.47	184.59	52.17	21.20	50.41	11.16	21.61	19.69	1.91
90+	1733.18	1653.38	27.96	354.64	244.83	113.01	193.15	65.36	23.17	34.96	11.16	22.70	20.69	2.01

Table A.4.15. Male Asian cancer incidence rates by age and site.

Number of cases per 100,000 persons per year

Age	All cancer	All solid	Oesophagus	Stomach	Colon	Liver	Lung	Breast	Ovary	Bladder	Thyroid	Leukaemia	Non-CLL leukaemia	CLL
0–4	16.69	10.30	0.00	0.08	0.00	0.74	0.03			0.03	0.00	5.17	5.09	0.08
5–9	10.73	4.54	0.00	0.05	0.00	0.24	0.05			0.00	0.02	4.73	4.73	0.00
10–14	10.72	5.48	0.00	0.06	0.06	0.33	0.07			0.00	0.23	3.31	3.31	0.00
15–19	12.15	7.20	0.00	0.33	0.10	0.13	0.14			0.06	0.59	3.51	3.51	0.00
20–24	13.97	9.68	0.00	0.81	0.50	0.70	0.41			0.31	0.74	2.30	2.30	0.00
25–29	21.59	16.88	0.10	2.29	0.91	1.67	0.51			0.59	0.99	2.94	2.89	0.05
30–34	37.04	31.17	0.13	5.05	3.54	3.60	2.30			0.81	1.16	3.55	3.49	0.06
35–39	72.78	65.58	0.80	14.96	5.45	11.41	5.09			2.20	1.67	3.03	2.93	0.10
40–44	140.70	131.55	2.94	29.51	12.43	21.68	14.83			3.59	2.15	3.90	3.71	0.19
45–49	227.28	213.75	7.05	47.43	24.55	36.58	23.27			5.14	3.17	5.45	5.30	0.15
50–54	357.46	339.23	14.35	76.73	39.96	54.82	44.64			10.69	2.82	7.01	6.67	0.34
55–59	588.80	564.44	25.49	127.25	72.34	95.29	80.55			17.08	2.86	9.51	9.07	0.43
60–64	1059.95	1019.71	44.55	217.15	119.83	170.87	176.67			33.03	3.84	13.36	12.55	0.81
65–69	1523.88	1468.59	58.10	316.67	162.08	195.63	317.21			55.42	5.13	20.21	18.61	1.60
70–74	1948.97	1878.15	82.63	412.58	186.30	192.09	439.32			73.66	5.16	27.13	25.46	1.67
75–79	2267.27	2180.80	92.66	488.08	214.56	183.31	509.83			108.13	4.68	30.62	28.83	1.79
80–84	2470.31	2375.91	94.17	520.98	222.27	187.30	540.57			120.05	4.35	31.68	28.87	2.81
85–89	3372.14	3223.64	69.75	716.89	326.54	232.57	682.18			158.97	4.35	49.11	44.17	4.94
90+	3907.81	3742.07	68.97	863.48	422.02	215.09	608.83			264.33	4.35	49.86	44.84	5.02

Table A.4.16. Female Asian cancer mortality rates by age and site.

Number of deaths per 100,000 persons per year

Age	All cause	All cancer	All solid	Oesophagus	Stomach	Colon	Liver	Lung	Breast	Ovary	Bladder	Leukaemia	Non-CLL leukaemia	CLL
0–4	127.18	3.38	1.70	0.00	0.01	0.00	0.10	0.02	0.00	0.01	0.01	1.34	1.34	0.00
5–9	16.67	3.08	1.33	0.00	0.00	0.00	0.03	0.00	0.00	0.01	0.00	1.33	1.33	0.00
10–14	15.15	3.52	1.42	0.01	0.00	0.01	0.05	0.00	0.01	0.04	0.00	1.66	1.66	0.00
15–19	18.31	3.39	1.46	0.02	0.07	0.04	0.08	0.04	0.01	0.13	0.01	1.24	1.24	0.00
20–24	27.75	3.97	2.31	0.01	0.28	0.17	0.20	0.16	0.08	0.19	0.00	1.16	1.16	0.00
25–29	33.29	6.37	4.66	0.04	0.89	0.39	0.40	0.38	0.36	0.20	0.01	1.15	1.15	0.00
30–34	44.91	13.20	11.14	0.06	2.28	1.02	0.98	1.06	1.67	0.52	0.04	1.43	1.43	0.00
35–39	62.83	23.88	21.06	0.15	4.13	1.95	1.79	2.27	4.58	1.24	0.06	1.79	1.79	0.00
40–44	107.45	45.04	41.40	0.46	7.14	3.39	3.74	5.45	8.89	2.26	0.09	2.32	2.32	0.00
45–49	162.17	66.72	62.51	1.26	9.31	5.26	6.20	9.08	12.01	4.36	0.16	2.65	2.65	0.00
50–54	237.87	94.83	90.12	2.16	12.01	7.43	9.43	15.19	14.91	6.52	0.38	2.71	2.57	0.14
55–59	399.63	151.41	144.12	4.31	19.77	12.43	15.91	29.64	17.01	6.21	0.81	3.65	3.57	0.08
60–64	740.16	245.00	234.08	8.43	30.60	20.91	28.82	54.90	17.67	9.05	1.45	5.44	5.26	0.18
65–69	1239.84	357.21	342.78	15.26	47.37	30.14	41.39	83.63	18.97	9.55	3.27	6.05	5.32	0.72
70–74	2184.11	508.02	488.66	25.09	73.47	46.13	57.19	115.76	20.60	10.22	6.20	8.56	7.23	1.33
75–79	3682.84	653.04	630.76	34.41	101.60	64.40	67.38	138.34	24.32	11.85	10.27	8.60	7.58	1.02
80–84	6509.31	780.83	755.96	37.66	134.47	82.36	73.27	148.97	31.19	9.55	15.88	9.19	8.56	0.63
85–89	8923.98	712.91	693.30	39.96	126.81	75.93	63.03	119.29	29.99	8.63	21.78	6.95	6.71	0.23
90+	17750.63	840.17	818.35	39.00	157.96	106.46	62.23	124.82	37.57	9.43	15.10	7.30	7.05	0.25

Table A.4.17. Male Asian cancer mortality rates by age and site.

Number of deaths per 100,000 persons per year

Age	All cause	All cancer	All solid	Oesophagus	Stomach	Colon	Liver	Lung	Breast	Ovary	Bladder	Leukaemia	Non-CLL leukaemia	CLL
0–4	149.24	3.79	1.75	0.00	0.00	0.01	0.15	0.02			0.02	1.60	1.60	0.00
5–9	24.88	3.96	1.62	0.00	0.00	0.01	0.08	0.01			0.00	1.77	1.77	0.00
10–14	23.65	4.78	2.00	0.00	0.01	0.01	0.10	0.01			0.00	1.98	1.98	0.00
15–19	35.16	4.81	2.20	0.00	0.09	0.05	0.18	0.09			0.01	1.66	1.66	0.00
20–24	50.43	5.06	2.87	0.02	0.25	0.19	0.47	0.22			0.02	1.44	1.44	0.00
25–29	59.21	7.79	5.40	0.06	0.62	0.37	1.36	0.59			0.03	1.46	1.46	0.00
30–34	80.39	14.60	11.97	0.17	1.67	0.91	3.75	1.70			0.04	1.74	1.74	0.00
35–39	114.64	29.41	25.77	0.48	3.83	1.99	8.34	4.17			0.14	2.13	2.12	0.00
40–44	188.22	58.32	53.62	2.13	8.05	3.58	17.40	9.85			0.25	2.61	2.55	0.06
45–49	276.69	95.90	90.33	5.09	14.22	5.43	26.64	18.17			0.57	3.03	2.59	0.44
50–54	399.85	149.26	141.77	9.83	23.38	8.45	36.85	31.35			1.04	3.48	2.97	0.51
55–59	646.43	252.16	242.34	17.39	42.54	14.49	55.24	58.84			2.09	4.85	4.73	0.12
60–64	1257.04	482.58	466.03	34.20	80.47	28.65	95.25	130.56			5.07	6.98	6.33	0.65
65–69	2107.53	755.18	732.35	54.58	130.26	43.47	118.07	230.26			11.07	10.31	9.74	0.57
70–74	3550.26	1065.73	1035.03	82.96	194.71	65.39	131.80	335.02			19.49	13.49	12.52	0.97
75–79	5749.87	1365.66	1325.91	102.71	259.01	90.86	142.09	409.23			37.80	16.55	15.52	1.02
80–84	9661.98	1661.07	1614.41	121.87	328.69	122.29	155.29	446.43			62.69	18.78	16.66	2.12
85–89	12799.94	1586.63	1542.42	121.60	307.77	128.12	137.19	397.35			73.45	19.76	18.03	1.74
90+	22367.18	1838.67	1790.47	120.24	370.70	165.59	126.88	354.63			122.13	20.06	18.30	1.76

of an uncertain increase in the value of DDREF, i.e., merely a variation on the result obtained by ignoring the possibility of a threshold.

(A 185) The existence of a low-dose threshold for cancer induction in certain tissues is not implausible. Indeed, as noted in *Publication 99* there is no clear evidence for a radiation-associated excess of cancers for a number of human tissues, e.g., chronic lymphocytic leukaemia, testicular cancer, and melanoma skin cancer.

Table A.4.18. Estimates of sex-specific population detriments for ages 0–85 years at exposure.

Tissue	Nominal risk coefficient (cases per 10,000 persons per Sv)	Lethality fraction	Lethality-adjusted nominal risk* (relating to column 1)	Relative cancer-free life lost	Detriment (relating to column 1)	Relative detriment[a]
Male						
Oesophagus	15	0.93	14	0.87	12.6	0.026
Stomach	68	0.83	66	0.88	57.9	0.120
Colon	91	0.48	69	0.97	66.8	0.138
Liver	41	0.95	41	0.88	36.1	0.075
Lung	76	0.89	75	0.80	59.9	0.124
Bone	7	0.45	5	1.00	5.1	0.011
Skin	1000	0.002	4	1.00	4.0	0.008
Breast	0	0.29	0	1.29	0.0	0.000
Ovary	0	0.57	0	1.12	0.0	0.000
Bladder	46	0.29	25	0.71	17.5	0.036
Thyroid	12	0.07	4	1.29	4.8	0.010
Bone marrow	48	0.67	43	1.63	69.8	0.144
Other solid	157	0.49	120	1.03	123.9	0.256
Gonads (heritable)	20	0.80	19	1.32	25.4	0.053
Total	***1580***		***485***		***483.9***	***1.00***
Female						
Oesophagus	16	0.93	16	0.87	13.6	0.021
Stomach	91	0.83	88	0.88	77.5	0.117
Colon	40	0.48	30	0.97	29.0	0.044
Liver	19	0.95	19	0.88	17.0	0.026
Lung	153	0.89	151	0.80	120.7	0.182
Bone	7	0.45	5	1.00	5.1	0.008
Skin	1000	0.00	4	1.00	4.0	0.006
Breast	224	0.29	124	1.29	159.7	0.240
Ovary	21	0.57	18	1.12	19.8	0.030
Bladder	41	0.29	22	0.71	15.8	0.024
Thyroid	53	0.07	16	1.29	20.6	0.031
Bone marrow	36	0.67	33	1.63	53.2	0.080
Other solid	131	0.49	100	1.03	103.1	0.155
Gonads (heritable)	20	0.80	19	1.32	25.4	0.038
Total	***1851***		***645***		***664.6***	***1.00***

[a] Estimates based on cancer incidence data. These sex-specific values for detriment do not have specific functions in the Commission's system of radiological protection (see paragraph A 156).

Table A.4.19. Estimates of sex-specific population detriments for ages 18–64 years at exposure.

Tissue	Nominal risk coefficient (cases per 10,000 persons per Sv)	Lethality fraction	Lethality-adjusted nominal risk* (relating to column 1)	Relative cancer-free life lost	Detriment (relating to column 1)	Relative detriment[a]
Male						
Oesophagus	14	0.93	14	0.91	12.8	0.035
Stomach	51	0.83	50	0.89	44.5	0.122
Colon	73	0.48	55	1.13	62.0	0.170
Liver	31	0.95	31	0.93	28.5	0.078
Lung	84	0.89	83	0.96	80.0	0.219
Bone	5	0.45	3	1.00	3.4	0.009
Skin	670	0.002	3	1.00	2.7	0.007
Breast	0	0.29	0	1.20	0.0	0.000
Ovary	0	0.57	0	1.16	0.0	0.000
Bladder	40	0.29	22	0.85	18.6	0.051
Thyroid	4	0.07	1	1.19	1.6	0.004
Bone Marrow	24	0.67	22	1.17	25.2	0.069
Other Solid	94	0.49	72	0.97	70.1	0.192
Gonads (Heritable)	12	0.80	12	1.32	15.3	0.042
Total	***1103***		***368***		***365***	***1.00***
Female						
Oesophagus	16	0.93	16	0.91	14.4	0.028
Stomach	70	0.83	68	0.89	60.7	0.119
Colon	33	0.48	25	1.13	27.7	0.054
Liver	16	0.95	16	0.93	14.7	0.029
Lung	174	0.89	172	0.96	165.4	0.325
Bone surface	5	0.45	3	1.00	3.4	0.007
Skin	670	0.002	3	1.00	2.7	0.005
Breast	116	0.29	64	1.20	76.6	0.150
Ovary	16	0.57	14	1.16	15.7	0.031
Bladder	39	0.29	21	0.85	17.7	0.035
Thyroid	20	0.07	6	1.19	7.0	0.014
Bone Marrow	22	0.67	20	1.17	22.9	0.045
Other Solid	88	0.49	67	0.97	65.1	0.128
Gonads (Heritable)	12	0.80	12	1.32	15.3	0.030
Total	***1242***		***505***		***509***	***1.00***

These sex-specific values for detriment do not have specific functions in the Commission's system of radiological protection (see paragraph A 156).

[a] Estimates based on cancer incidence data.

(A 186) Although the available data do not exclude the existence of a universal low-dose threshold, the evidence as a whole, as interpreted and summarised in this Annex, does not favour this proposition. The BEIR VII Committee (NAS/NRC, 2006) has recently published a report on low-dose risk that essentially arrives at the same conclusion based on epidemiological and biological data. However, an

equally recent low-dose report from the French Academies (2005) emphasises evidence on the potential dose-dependence of post-irradiation cellular signalling, DNA repair, apoptosis and other adaptive anti-tumorigenic processes in order to argue for the existence of a practical low-dose threshold for radiation cancer risk. Overall, the long standing question on the true validity of the LNT model may well prove to be beyond definitive scientific resolution and that 'weight of evidence' arguments and practical judgements are likely to continue to apply in the foreseeable future.

(A 187) In summary, the Commission judges that there are at present no good scientific reasons to include the possibilities of supra-linear dose responses or of a low-dose threshold in cancer risk calculations for the purposes of radiological protection. On this basis it is recommended that the LNT model, combined with a judged value of DDREF for extrapolation from high doses, remains a prudent basis for the practical purposes of radiological protection at low doses and low dose rates.

A.4.5. Further details of the detriment calculations

(A 188) In this Section, the model parameters used for the Commission's risk model are provided in detail. Table A.4.5 lists the lethality factors, non-fatal case weights, and relative life lost for the various sites considered. Tables A.4.6 and A.4.7 show coefficients in the current cancer incidence-based ERR and EAR models, respectively, while Tables A.4.8 and A.4.9 show coefficients in the current cancer mortality-based ERR and EAR models. Female and male Euro-American cancer incidence rates by age and site are given in Tables A.4.10 and A.4.11, and female and male Euro-American cancer mortality rates are given in Tables A.4.12 and A.4.13. Tables A.4.14 and A.4.15 show Asian female and male cancer incidence rates, and Tables A.4.16 and A.4.17 provide Asian female and male cancer mortality rates.

A.4.6. Estimates of sex-specific population detriments

(A 189) This Section provides estimates of sex-specific detriments, based on cancer incidence data, for ages 0–85 years at exposure in Table A.4.18 and for ages 18–64 years at exposure in Table A.4.19. The Commission emphasises that these sex-specific values for detriment have no specific function in its system of radiological protection (see paragraph A 156).

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A.5. Non-cancer diseases after radiation exposure

(A 190) Since 1990 evidence has accumulated that the frequency of non-cancer diseases is increased in irradiated populations. The strongest evidence for the induction of these non-cancer effects at doses of the order of 1 Sv derives from the A-bomb LSS, and the most recent mortality analysis (Preston et al., 2003) has strengthened the statistical evidence for an association with dose – particularly for heart disease, stroke, digestive disorders and respiratory disease. However, the Commission notes current uncertainties on the shape of the dose response at low doses and that the LSS data are consistent both with there being no dose threshold for risks of disease mortality and with a threshold of around 0.5 Sv. It is unclear what forms of cellular/tissue mechanisms might underlie such a diverse set of non-cancer disorders reported among the LSS data although some association with subclinical inflammation (e.g., Hayashi et al., 2003) is possible.

(A 191) Additional evidence of the non-cancer effects of radiation, albeit at high doses, comes from studies of cancer patients receiving radiotherapy. Studies of patients treated for Hodgkin's disease (e.g., Hancock et al. 1993, Aleman et al. 2003) and for breast cancer (e.g., Early Breast Cancer Trialists Collaborative Group 2000) have shown raised risks of mortality from cardiovascular disease, associated with doses of several tens of Gy. The situation at lower doses is less clear. A review of published epidemiological data of groups with medical or occupational exposures, which compared the rates of circulatory disease in irradiated and non-irradiated individuals drawn from the same population, concluded that there was no clear evidence of an increased risk in the majority of studies over the dose range 0 to 4 Sv (McGale and Darby 2005). Interpretation of many studies was, however, complicated by the very limited dose response data available and by a lack of information on possible confounding factors such as smoking.

(A 192) Whilst recognising the potential importance of these observations on non-cancer diseases, the Commission judges that the data available do not allow for their inclusion in the estimation of detriment following radiation doses in the range up to around 100 mSv. This agrees with the conclusion of UNSCEAR (2008), which found little evidence of any excess risk below 0.5 Sv.

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A.6. Risks of heritable diseases

A.6.1. Introduction

(A 193) The term 'genetic risks' as used in this document denotes the probability of harmful genetic effects manifest in the descendants of a population that has sustained radiation exposures. These effects are expressed as increases over the baseline frequencies of genetic diseases in the population per unit dose of low-LET, low-dose/chronic irradiation.

(A 194) Since the publication of the 1990 Recommendations of the ICRP (ICRP, 1991b), the 1990 BEIR report (NRC, 1990), and the UNSCEAR (1993) report, several important advances have been made in the prediction of genetic risks of exposure of human populations to ionising radiation. On the basis of these, UNSCEAR (2001) revised its earlier risk estimates. The aim of this section of the report is to provide a brief background on the available information and the methods that are used for risk estimation, to summarise the recent advances, to present the revised risk estimates, and to indicate how the new estimates can be used to derive a risk coefficient for genetic effects.

A.6.2. Background information

Naturally occurring genetic diseases

(A 195) The genetic diseases of interest in the present context are those due to mutations in single genes (Mendelian diseases) and those which are due to multiple genetic and environmental factors (multifactorial diseases). Historically, UNSCEAR, the BEIR Committees, and ICRP had also considered an additional class of genetic diseases, namely, chromosomal diseases which are due to gross structural and numerical abnormalities of chromosomes.

(A 196) *Mendelian diseases* are further subdivided into autosomal dominant, autosomal recessive, and X-linked recessive categories depending on the chromosomal location (autosomes or the X-chromosome) of the mutant genes and their transmission patterns. In the case of an autosomal dominant disease, a single mutant gene inherited from either parent (i.e., in a heterozygous state) is sufficient to cause disease (e.g., achondroplasia, neurofibromatosis, Marfan syndrome, etc.). The somewhat unusual genetics of dominantly inherited cancer predisposition are discussed in *Publication 79* (ICRP, 1998a). Autosomal recessive diseases, however, require two mutant genes, one from each parent, at the same locus (i.e., homozygosity) for disease manifestation (e.g., cystic fibrosis, haemochromatosis, Bloom syndrome, ataxia telangiectasia, etc.). In the case of X-linked recessive diseases, since males have only one X-chromosome, usually only males are affected (e.g., haemophilia, Duchenne muscular dystrophy, Fabry disease, etc.). However, some X-linked dominant diseases are also known (e.g., Rett syndrome), but, for the purpose of the present document, they are included under X-linked recessive diseases. The important general point with respect to Mendelian diseases is that the relationship between mutation and disease is simple and predictable.

(A 197) *Multifactorial diseases* are aetiologically complex and consequently the relationship between mutation and disease is also complex, i.e., these do not show Mendelian patterns of inheritance. The two subgroups that constitute multifactorial diseases are the common congenital abnormalities (e.g., neural tube defects, cleft lip with or without cleft palate, congenital heart defects, etc.) and chronic diseases of adults (e.g., coronary heart disease, essential hypertension, diabetes mellitus, etc.). Evidence for a genetic component in their aetiology comes from family and twin studies which show that the first-degree relatives of affected individuals have a higher risk of disease than matched controls. For most of them, knowledge of the genes involved, the types of mutational alterations, and the nature of environmental factors still remains limited. Among the models used to explain the inheritance patterns of multifactorial diseases and estimate recurrence risks in relatives is the multifactorial threshold model (MTM) of disease liability. This is considered in a later section.

(A 198) Chromosomal diseases arise as a result of gross numerical (e.g., Down syndrome due to trisomy for chromosome 21) or structural abnormalities of chromosomes (e.g., Cri du chat syndrome due to deletion of part or whole short arm of chromosome 5) generally detectable in cytological preparations of cells. This is really not an aetiological category and, further, deletions (microscopically detectable or not) are now known to contribute to a number of genetic diseases grouped under autosomal dominant, autosomal recessive and X-linked diseases.

The doubling dose method

(A 199) In the absence of human data on radiation-induced genetic diseases, all the methods that have been developed and used since the mid 1950s up to the present are indirect; their aim is to make the best use of mutation data obtained in radiation studies with mice, data on baseline frequencies of genetic diseases in the population, and population genetic theory, to predict the radiation risk of genetic diseases in humans. One such method that has been used from the early 1970s onwards until now (e.g., UNSCEAR 2001) is the doubling dose method. This method enables one to express the expected increase in the frequencies of genetic diseases in terms of their baseline frequencies using the following equation:

$$\text{Risk per unit dose} = \text{P} \times [1/\text{DD}] \times \text{MC} \qquad \text{(A.6.1)}$$

where P is the baseline frequency of the genetic disease class under study, DD is the doubling dose (and [1/DD] is the relative mutation risk per unit dose), and MC is the disease-class-specific mutation component.

(A 200) The genetic theory that underlies the use of the DD method for risk estimation is what is referred to as the equilibrium theory which population geneticists use to explain the dynamics of mutant genes in populations. The theory postulates that the stability of mutant gene frequencies (and thus of disease frequencies) in a population is the result of the existence of a balance between the rate at which spontaneous mutations enter the gene pool of the population in every generation and the rate at which they are eliminated by natural selection, i.e., through failure of survival or reproduction. Under normal conditions (i.e., in the absence of radiation expo-

sures), the population is assumed to be in equilibrium between mutation and selection.

(A 201) When the mutation rate is increased as a result of radiation, say, in every generation, the balance between mutation and selection is disturbed by the influx of induced mutations, but the prediction is that the population will eventually attain a new equilibrium (over a number of generations) between mutation and selection. The amount of increase in mutation frequency, the time it takes for the population to reach the new equilibrium, and the rate of approach to it are all dependent on induced mutation rates, the intensity of selection, the type of genetic disease, and whether radiation exposure occurs in one generation only or generation after generation. Worth mentioning here is that, since the starting population (before radiation exposure) is assumed to be in equilibrium between mutation and selection, the quantity P in Eqn. (A.6.1) represents the equilibrium incidence.

(A 202) *Doubling dose*. The doubling dose (DD) is the amount of radiation that is required to produce as many mutations as those that arise spontaneously in a generation. Ideally, it is estimated as a ratio of the average rates of spontaneous and induced mutations in a given set of genes.

(A 203) The reciprocal of the DD (i.e., [1/DD]) is the relative mutation risk (RMR) per unit dose. Since RMR is a fraction, the smaller the DD, the higher is the RMR and vice versa.

(A 204) *Mutation component*. Formally defined, mutation component (MC) is the relative increase in disease frequency per unit relative increase in mutation rate:

$$\mathrm{MC} = [\Delta P/P]/[\Delta m/m] \tag{A.6.2}$$

where P is the baseline disease frequency, ΔP its change due to Δm change in mutation rate, and m the spontaneous mutation rate. The procedures used for estimating MC are relatively straightforward for autosomal dominant and X-linked diseases, slightly complicated for autosomal recessives (since an induced recessive mutation does not precipitate a recessive disease in the immediate post-radiation generations) and more complex for multifactorial diseases, and depends on the model that is used to explain their stable frequencies in the population.

A.6.3. Recent advances in understanding

(A 205) The advances that have been made during the past few years include: a) an upward revision of the estimates of the baseline frequencies of Mendelian diseases; b) the introduction of a conceptual change in the calculation of the DD; c) the elaboration of methods for estimating MC for Mendelian and chronic diseases; d) the introduction of an additional factor called the 'potential recoverability correction factor' (PRCF) in the risk equation to bridge the gap between the rates of radiation-induced mutations in mice and the risk of radiation-inducible genetic disease in human live births; and e) the introduction of the concept that the adverse effects of radiation-induced genetic damage in humans are likely to be manifest predominantly as multisystem developmental abnormalities in the progeny. All these have been discussed in detail in a series of recent publications (Chakraborty et al. 1998,

Denniston et al. 1998, Sankaranarayanan 1998, 1999, Sankaranarayanan and Chakraborty 2000a, 2000b, 2000c, Sankaranarayanan et al. 1994, 1999, NAS/NRC 2006). Box 2 summarises the procedures used by the Commission to estimate radiation risk for heritable disease which take account of these advances in understanding.

Baseline frequencies of genetic diseases

(A 206) Until the 1993 UNSCEAR report, the baseline frequencies used in risk estimation were based on those compiled by Carter (1977) for Mendelian diseases, by UNSCEAR (1977) for chromosomal diseases, by Czeizel and Sankaranarayanan (1984) for congenital abnormalities, and by Czeizel et al. (1988) for chronic diseases. While the estimates for the last three groups of diseases have remained unchanged, those for Mendelian diseases have now been revised upwards (Sankaranarayanan, 1998). Both the earlier and the current estimates (the latter used in UNSCEAR, 2001) are presented in Table A.6.1.

Box A.2. Steps in estimating radiation risk for heritable diseases.

a) Establish the baseline frequencies of human genetic diseases of all classes (a set of values of P).
b) Estimate average spontaneous mutation rate per generation for human genes.
c) No human data available, so estimate average rate of radiation-induced mutations in mice – assume that mouse rates are similar to those of humans.
d) From b) and c) above, estimate the genetic doubling dose (DD). DD is the radiation dose required to produce as many mutations as those that arise spontaneously in one generation.
e) Estimate the mutation component (MC) for different classes of genetic diseases. MC is a relative measure of the relationship between change in mutation rate and increase in disease frequency.
f) Estimate the potential recoverability correction factor (PRCF) for different classes of mutation. PRCF allows for differing degrees of recoverability of mutations in live births, i.e., the fraction of mutations that is compatible with embryonic/fetal development.
g) For each class of human genetic disease complete the following equation using the estimates from a) to f) above.

$$\textbf{Risk per unit dose} = \mathbf{P} \times [\mathbf{1/DD}] \times \mathbf{MC} \times \mathbf{PCRF}$$

The doubling dose

(A 207) ***A re-examination of the assumptions involved in using the DD based on mouse data for risk estimation.*** The DD used until the 1993 UNSCEAR report was 1 Gy (for chronic, low-LET radiation conditions) and was based entirely on mouse data on spontaneous and induced rates of recessive mutations in seven genes.

Table A.6.1. Baseline frequencies of genetic diseases in human populations.

Disease class	Baseline frequencies (per cent of live births)	
	UNSCEAR (1993)	UNSCEAR (2001)
Mendelian		
Autosomal dominant	0.95	1.50
X-linked	0.05	0.15
Autosomal recessive	0.25	0.75
Chromosomal	0.40	0.40
Multifactorial		
Chronic diseases	65.00[a]	65.00[a]
Congenital abnormalities	6.00	6.00

[a] Population frequency.

One of the assumptions underlying the use of mouse-data-based DD for risk estimation is that both the spontaneous and induced mutation rates in mice and humans are the same. The assumption regarding induced rates of mutations, while unavoidable, is defensible on the grounds of generally similar gene organisation, 70–90% homology in DNA sequence of genes, and substantial conservation of synteny for many (although not all) chromosomal regions in both the species. However, the situation is different with respect to spontaneous mutation rates.

(A 208) Arguments supporting the view that the spontaneous mutation rates in mice and humans are unlikely to be similar have been discussed (Sankaranarayanan, 1998, Sankaranarayanan and Chakraborty, 2000a, UNSCEAR, 2001). Briefly, unlike in the mouse, in humans there are pronounced sex-differences in spontaneous mutation rates (being higher in males than in females), and the mutation rate increases with the age of the father (paternal age effect). These differences when considered along with the fact that the human lifespan is longer than that of the mouse, suggest that extrapolating from the short-lived mouse to humans is unlikely to provide a reliable average spontaneous rate in a heterogeneous human population of all ages. Additionally, recent analyses of mouse data on mutations that arise as germinal mosaics (which result in clusters of identical mutations in the following generation) have introduced considerable uncertainty about the spontaneous mutation rate in the mouse (Selby, 1998).

(A 209) ***The use of human data on spontaneous mutation rates and mouse data for induced mutation rates for DD calculations.*** In view of the reasons stated in the preceding paragraphs, UNSCEAR (2001) considered it prudent to base DD calculations on human data on spontaneous mutation rates, and mouse data on induced mutation rates, as was first done in the 1972 BEIR report (NRC 1972). The advantages of using human data in DD calculations are: a) they pertain to human disease-causing genes; b) the mutation rate estimates in humans, because they are averaged over the sexes, automatically include paternal age effects; and c) in estimating mutation rates, human geneticists count all mutations irrespective of whether they are part of a cluster or not; consequently, had clusters occurred, they would have been included.

(A 210) ***Average spontaneous mutation rate for human genes.*** For calculating an average spontaneous mutation rate for human genes, UNSCEAR (2001) focused on published data on those genes for which estimates of selection coefficient(*s*) were also available, the reason being that selection coefficients are relevant for estimating MC (to be discussed in the next section). Further, only autosomal dominant diseases, but not X-linked ones, were included in the analysis, the rationale being that: a) among Mendelian diseases, autosomal dominants constitute the most important group from the standpoint of genetic risks; b) while X-linked diseases are also expected to respond directly to an increase in mutation rate, their incidence in the population is an order of magnitude lower than that of autosomal dominants (0.15% versus 1.50%); and, consequently, c) the assumption of similar average mutation rates for these two classes of disease in the context of risk estimation is unlikely to result in an underestimate of the risk.

(A 211) The average (unweighted) spontaneous mutation rate based on a total of 26 autosomal dominant disease phenotypes (which, on current knowledge, relate to mutations in an estimated 135 genes) was $(2.95 \pm 0.64)\ 10^{-6}$ gene^{-1} generation^{-1} (Sankaranarayanan and Chakraborty 2000a). This estimate is well within the range of $0.5\ 10^{-5}$ to $0.5\ 10^{-6}$ per gene assumed in the 1972 BEIR report (NRC 1972). The data used for spontaneous mutation rate calculations also permit an estimate of 0.294 for average selection coefficient(s) associated with these diseases.

(A 212) ***Average rate of induced mutations in mice.*** As mentioned earlier, until the 1993 UNSCEAR report, the average rate of induced mutations used in DD calculations was based on data from studies of recessive specific locus mutations in seven genes. In the 2001 report, however, UNSCEAR expanded the database to include not only the above, but also data from studies of enzyme activity mutations, as well as dominant mutations at four loci (*Sl*, *W*, *Sp* and *T*). All these data come from studies of males in which the irradiated germ cell stages were stem-cell spermatogonia (the relevant germ cell stages in males from the standpoint of risks). The data from studies with female mice were not used since, as discussed in the 1988 UNSCEAR report, there is uncertainty as to whether the mouse immature oocytes (with nearly zero sensitivity to mutation induction after acute as well as chronic irradiation) would provide a good model for assessing the mutational radiosensitivity of human immature oocytes that are the relevant germ cell stages in the females. For the purpose of risk estimation, to err on the side of caution, it was assumed that the induced rates in females will be the same as those in males.

(A 213) Details of the data used are discussed in the UNSCEAR, 2001 report and by Sankaranarayanan and Chakraborty (2000a). The average induced mutation rate, based on mutations recovered at a total of 34 mouse genes, is $(1.08 \pm 0.30)\ 10^{-5}$ gene^{-1} Gy^{-1} for acute X or γ irradiation. With a dose-rate reduction factor of 3 traditionally used, the rate for chronic irradiation conditions becomes (0.36 ± 0.10) 10^{-5} gene^{-1} Gy^{-1}.

(A 214) ***The doubling dose.*** With the revised estimates for average spontaneous mutation rate $(2.95 \pm 0.64)\ 10^{-6}$ gene^{-1} generation^{-1} for human genes and for average rate of induced mutations $(0.36 \pm 0.10)\ 10^{-5}$ gene^{-1} Gy^{-1} for mouse genes, the

new DD becomes (0.82 ± 0.29) Gy. This estimate, however, is not very different from 1 Gy that has been used thus far but which was based entirely on mouse data.

(A 215) UNSCEAR (2001) has suggested the continued use of the 1 Gy estimate in order to avoid the impression of undue precision, but noting that a conceptual change has now been made (i.e., use of human data on spontaneous and mouse data on induced mutation rates) and that the present estimate is supported by more extensive data than had been the case thus far. The Commission supports the UNSCEAR judgement and therefore ICRP retains a DD value of 1 Gy.

Mutation component

(A 216) As noted in Section A.6.2, the quantity 'mutation component' (MC) used in Eqn. (A.6.1) provides a measure of the relative change in disease frequency per unit relative change in mutation rate for the different classes of genetic diseases. The elements of the basic MC concept were introduced already in the 1972 BEIR report (NRC, 1972) and were subsequently considered in the papers of Crow and Denniston (1981, 1985). Within the framework of an ICRP Task Group, set up in 1993, the problem was studied in detail, and the concept, theory, methods for estimation, and algebraic formulations were fully elaborated for both Mendelian and multifactorial diseases. The Task Group report has since been published (*Publication 83*, ICRP, 1999b). The methods developed in that document now enable the evaluation of the magnitude of the MC for any post-radiation generation of interest, after either a one-time or a permanent increase in mutation rate, i.e., radiation exposure in every generation. In what follows, a brief summary of the main findings is presented.

(A 217) ***Mutation component for autosomal dominant diseases.*** For autosomal dominant diseases (for which the relationship between mutation and disease is straightforward) the estimation procedure is relatively simple. For a one-generation radiation exposure which produces a one-time increase in mutation rate ('burst', indicated by the subscript 'b' in MC_b below), the change with time 't' (in generations) is given by the equation:

$$MC_b(t) = s(1 - s)^{t-1} \tag{A.6.3}$$

For radiation exposure to many successive generations producing a permanent increase in mutation rate (indicated by the subscript 'p'),

$$MC_p(t) = [1 - (1 - s)^t] \tag{A.6.4}$$

(A 218) Equations (A.6.3) and (A.6.4) show that $MC_b = MC_p = s$ for the first post-radiation generation following either a one-time or a permanent increase in mutation rate. With no further irradiation in subsequent generations, the value of MC will decay back to zero at a rate of $(1 - s)$ per generation. With a permanent increase in mutation rate, however, the MC value will slowly increase to 1 at the new equilibrium. Consistent with these changes in MC, for a one-time irradiation scenario, the disease frequency will show a transitory increase in the first generation, but over time, reach the earlier or 'old' equilibrium value; for a permanent increase in mutation rate, the disease frequency will continue to increase until the new equilibrium

value of MC = 1 is reached. At the new equilibrium, an x% increase in mutation rate will result in an x% increase in disease frequency.

(A 219) ***Mutation component for X-linked and autosomal recessive diseases.*** For X-linked diseases, for a one-time increase in mutation rate, the first generation MC = s, as in the case of autosomal dominants, but the s value needs to be adjusted to take into account the fact that only one-third of the total X-chromosome complement is in males. The dynamics of change in MC in subsequent generations is similar to that for autosomal dominants. For autosomal recessives, MC in the first generation is close to zero (consistent with the fact that an autosomal recessive mutation does not result in disease in the first generation).

(A 220) With a permanent increase in mutation rate, for both kinds of diseases, MC progressively increases to reach a value of 1 at the new equilibrium, but the rates of approach to the new equilibrium are different and are dictated by s values and time (in generations) following irradiation. In particular, for autosomal recessive diseases, the rate of approach to the new equilibrium is very slow and much slower than that for autosomal dominants and X-linked diseases.

(A 221) The important point that emerges from the above discussion is that MC is related to s and therefore, given s, one can estimate the dynamics of increase in MC and in disease frequencies for any post-radiation generation of interest. As mentioned in paragraph (A 211), the average selection coefficient estimated from data on naturally occurring autosomal dominant diseases is 0.294. This value, rounded to 0.30, is the one used as the best estimate for MC for autosomal dominant and X-linked diseases.

(A 222) ***Mutation component for chronic diseases.*** As mentioned earlier, multifactorial diseases have a high population frequency, but, unlike in the case of Mendelian diseases, the lack of adequate models to explain their stable frequencies in the population precluded any meaningful assessment of the radiation risk of these diseases. Descriptive models such as the multifactorial threshold model (MTM) of disease liability to explain the observed transmission patterns of these diseases and to estimate risks to relatives of affected individuals from data on population frequencies have existed for a long time but, as such, they are not suitable for assessing the impact of an increase in mutation rate on disease frequency. Similarly, although there was a wealth of literature on mechanistic models (that invoke mutation and selection as opposing forces in the evolution and maintenance of variability of polygenic/quantitative traits in populations), none of these models was geared towards assessing the impact of an increase in mutation rate on the frequency of multifactorial diseases.

(A 223) The ICRP Task Group drafting *Publication 83* (ICRP 1999b) took the first step in addressing the above issue by formulating a 'hybrid model' which included some elements of the MTM and some of the mechanistic models mentioned above. The hybrid model is henceforth referred to as the 'finite locus threshold model' (FLTM). Although the original intention was to use the model to estimate MC for both congenital abnormalities and chronic diseases, it soon became clear that its use for congenital abnormalities is not biologically meaningful and consequently, the 1999 Task Group decided to limit its use to chronic diseases only. As discussed

later in this Annex, this does not pose any problem for estimating the risk of congenital abnormalities since this can now be done without recourse to the DD method. To provide a background, the assumptions and use of the MTM are first discussed below.

(A 224) ***Multifactorial threshold model (MTM) of disease liability.*** In the absence of information on the genetic or environmental factors that underlie multifactorial diseases, in the early 1960s the MTM used in quantitative genetics for threshold characters was extended to these diseases to explain their transmission patterns and estimate risks to relatives. Since multifactorial diseases are 'all-or-none' traits (unlike quantitative traits such as height or weight), in order to use the MTM for these diseases, it was necessary to postulate a hypothetical variable called 'liability' that underlies multifactorial diseases and a 'threshold' of liability which, when exceeded, would result in disease (Carter, 1961, Falconer, 1965). Worthy of note here is the fact that the MTM has been (and remains) useful for our understanding of familial aggregations and recurrence risks in families, and makes good predictions even when there is uncertainty about the underlying mechanisms. Details of the MTM for disease liability have been discussed in a number of publications (see ICRP, 1999b for a listing of the references).

(A 225) Briefly, the assumptions of the standard version of MTM are the following:

- all environmental and genetic causes can be combined into a single continuous variable called 'liability' which cannot, as such, be measured;
- liability is determined by a combination of numerous (essentially an infinite number of) genetic and environmental factors, that act additively, without dominance or epistasis, each contributing a small amount of liability and therefore show a Gaussian (normal) distribution; and
- the affected individuals are those whose liability exceeds a certain threshold value.

(A 226) The MTM enables the conversion of information on the incidence of a given multifactorial disease in the population (P) and in the relatives of those affected (q) into an estimate of correlation in liability between relatives from which a quantity called heritability (h^2), which provides a measure of the relative importance of genetic factors in disease causation, can be estimated.

(A 227) ***Heritability.*** Heritability, a common statistic used in quantitative genetics, provides a measure of the relative importance of transmissible genetic variation to the overall phenotypic variation. Since the phenotype owes its origin to genetic and environmental factors, in the analysis of variance, the total phenotypic variance (V_P) is usually partitioned into two components, genetic (V_G) and environmental (V_E), assuming that these are independent of each other (i.e., they are not correlated). The ratio V_G/V_P is called the 'broad-sense heritability', or degree of genetic determination, symbolised by h^2 (strictly, h^2_B). Estimates of the heritability of liability for many multifactorial diseases have been published in the literature and are in the range from about 0.30 to 0.80 although for most types of cancer the heritability coefficient is judged to be less than 0.30.

(A 228) The genotypic variance, V_G, can be subdivided into an additive component (V_A) and a component due to deviations from additivity. Additive genetic variance is the component that is attributable to the average effects of genes considered singly, as transmitted in the gametes. The ratio, V_A/V_G, called 'narrow-sense heritability' h^2_N, determines the magnitude of correlation between relatives (Falconer, 1960).

(A 229) ***The finite-locus-threshold model used for estimating MC for chronic diseases.*** The FLTM incorporates the assumptions of liability threshold from the MTM (but suitably redefined to take into account mutations at a finite number of genes) and the concepts of mutation and selection from models on the maintenance and evolution of polygenic variability underlying quantitative traits. The choice of the FLTM was dictated by two main considerations: a) current knowledge of the genetic basis of well-studied chronic diseases, such as coronary heart disease (CHD), supports the view that a large proportion of the variability of intermediate quantitative traits (such as serum cholesterol levels, a risk factor for CHD) in the population is due to mutations at a limited number of gene loci (ICRP, 1999b, Sankaranarayanan et al., 1999) and b) in the absence of precise information on the genetic basis of most multifactorial diseases, the FLTM provides a useful starting point because, with such a model, the meaning of parameters reflecting mutation rates and selection can be quantitatively assessed in terms of those for single gene effects.

(A 230) Briefly, the FLTM assumes that the liability to disease, made up of genetic and environmental factors, is a continuous variable. The genetic component of liability is discrete, i.e., it is determined by the total number of mutant genes (defined as a random variable, g, the number of mutant genes in a genotype at n loci) and the environmental effect, e, which is a random variable which has a Gaussian (normal) distribution with mean = 0 and variance = V_e. The total liability therefore has two components: a) a function [$f(g)$] of the number of mutant genes in the n-locus genotype of an individual and b) a normally distributed environmental effect, e. The threshold characteristic of the model is described by assuming that individuals with liability exceeding a threshold value T are phenotypically affected and have a fitness of $(1 - s)$ and those below it are normal with fitness equal to 1.

(A 231) Although the mathematical formulations of the FLTM cannot be expressed in the form of a single equation, the predictions of the model can be iteratively evaluated from the computer program that was developed for this purpose. The steps include the following: first, with a defined set of parameter values (mutation rate, selection coefficients, threshold, etc.), the program is run until the population reaches equilibrium between mutation and selection. When this is achieved, the mutation rate is increased once or permanently and the computer run is resumed with the new mutation rate (with the other parameters remaining the same). The changes in the magnitude of MC and its relationship to heritability of liability (h^2) are examined in desired generations and at the new equilibrium. The h^2 estimates are not inputs, but outputs of the program, obtained with different combinations of parameter values (for the numbers of gene loci from 3 to 6, mutation rate, selection coefficients, environmental variance, and threshold). The conclusions discussed

below are for the 5-locus model, but they remain qualitatively unaltered for other values of the number of gene loci.

(A 232) ***Main conclusions of the computer simulation studies.*** In these studies, a 5-locus model was used and the relationship between h^2 and changes in MC were assessed for two scenarios: a) the population sustains an increase in mutation rate every generation, and b) the population sustains an increase in mutation rate in one generation only. The initial (spontaneous) mutation rate assumed in the calculations was 10^{-6} per gene and the effects were examined for a 15% increase in mutation rate (i.e., 1.0 10^{-6}/gene to 1.15 10^{-6}/gene) with selection coefficients, $s = 0.2$ to 0.8. The conclusions are the following:

- Under conditions of a permanent increase in mutation rate, the MC at the new equilibrium is close to 1 over a wide range of h^2 values from about 0.3 to 0.8 that are of importance in the present context; stated differently, an x% increase in mutation rate will cause an x% increase in disease frequency at the new equilibrium.
- Again, under the same conditions and over the same range of h^2 values, the MC in the first several generations is very small, in the range 0.01–0.02, often closer to 0.01 than to 0.02. In other words, the predicted relative increase in disease frequency is very small.
- If the population sustains radiation exposure in one generation only, the MC in the first generation is as indicated in the previous conclusion, and its value gradually decays back to zero.
- The above three conclusions are valid when there is no sporadic component of disease, i.e., non-occurrence of individuals with disease that is unrelated to the genotype; when sporadics occur, the effect is to reduce the MC both in early generations and at the new equilibrium.

(A 233) The conclusions discussed above hold for so many different combinations of parameter values (i.e., threshold, selection coefficient, number of loci, environmental variance, spontaneous mutation rate, increases in mutation rate, etc.) that they can be considered robust. Additionally, it was found that, for mutation rates of the order known for Mendelian genes, the FLTM with a few loci and weak selection provides a good approximation to study the possible increases in the frequencies of chronic diseases in populations exposed to radiation.

(A 234) In its 2001 report UNSCEAR used MC = 0.02 as the best estimate in the risk equation for estimating the risk of chronic diseases.

The concept of potential recoverability correction factor

(A 235) The use of Eqn. (A.6.1) (i.e., risk = P × [1/DD] × MC) for risk estimation implies that the genes at which spontaneous mutations are known to cause disease (included under P) will also respond to induced mutations, that such mutations will be compatible with viability and therefore recoverable in live born progeny of irradiated individuals. This assumption gained support from studies of induced mutations in specific genes in several model systems. However, no radiation-induced

germ-cell gene mutations, let alone induced genetic diseases, have thus far been identified in human studies.

(A 236) Advances in human molecular biology and in radiobiology have now shown that: a) spontaneous disease-causing mutations and radiation-induced mutations in experimental systems differ in several respects, both in their nature and in the mechanisms by which they arise (or are induced); b) there are both structural and functional constraints that preclude the recoverability of induced mutations in all genomic regions, i.e., only a small proportion of human genes of relevance from the disease point of view are likely to be responsive to radiation-induced mutations that are recoverable in live born progeny; and c) genes that have hitherto been used in studies on induced mutations are those that are non-essential for viability and also happen to be located in genomic regions, also non-essential for viability (reviewed in Sankaranarayanan 1999). So the crux of the argument is that the induced mutation rates from mouse studies that are used in risk estimation are likely to be overestimates of the rate at which induced mutations in humans will precipitate disease.

(A 237) Since there is no alternative to the use of mouse data on induced mutations for risk estimation, methods need to be devised to bridge the gap between empirically determined rates of induced mutations in mice and the rates at which disease-causing mutations may be recovered in human live births. One such method that has been developed involves the incorporation of a correction factor termed 'potential recoverability correction factor' (PRCF) into risk Eqn. (A.6.1) so that the risk now becomes a product of four quantities instead of the original three:

$$\text{Risk per unit dose} = \text{P} \times [1/\text{DD}] \times \text{MC} \times \text{PRCF} \qquad \text{(A.6.5)}$$

where the first three are as defined earlier and PRCF is the disease-class specific potential recoverability correction factor. Since PRCF is a fraction, the estimate of risk will now be lower.

(A 238) In order to estimate *potential recoverability* of induced mutations, a set of criteria was first defined using molecular information on recovered mutations in experimental systems. The operative words are the italicised ones, since a) knowledge of the structural and functional genomics of the human genome is not yet complete; b) so far, no radiation-induced human germ cell mutations have been recovered to provide a frame of reference; and c) the criteria may change with advances in knowledge in the coming years. The criteria that could be developed were then applied to human genes of relevance from the disease point of view, taking into account gene size, organisation, function, genomic context (i.e., whether the gene is located in a 'gene-rich' or 'gene-poor' region), spectra of spontaneous mutations in the gene, whether deletions, including contiguous genes, are known in the region, and the known mutational mechanisms. The question asked was: if a deletion (the predominant type of radiation-induced change) were to be induced in this gene/gene region, would it be potentially recoverable in a live birth?

(A 239) Details of the criteria used and the classification of the genes into three groups, viz. group 1, 'induced deletion is unlikely to be recovered', group 2, 'uncertain recoverability', and group 3, 'potentially recoverable', are discussed in detail by Sankaranarayanan and Chakraborty (2000b) and in the UNSCEAR (2001) report.

Table A.6.2. Summary of assessments of potential recoverability of radiation-induced mutations in autosomal and X-linked genes.

Groups	No. of genes	Unweighted[a] PRCF	Incidence ($\times 10^4$)[b]	Weighted[c] PRCF
Autosomal dominants				
1 (unlikely to be recovered)	42	–	46.45	–
2 & 3 (uncertain + potentially recoverable)	17	0.29	55.90	0.157
Subtotal	59		102.35	
Autosomal dominants + X-linked				
1 (unlikely to be recovered)	43	–	48.95	–
2 & 3 (uncertain + potentially recoverable)	24	0.36	60.90	0.199
Total	67		109.85	

[a] Unweighted PRCF: aut. dominants: 17/59 = 0.29; aut. dominants + X-linked = 24/67 = 0.36.

[b] Estimates from Sankaranarayanan (1998) and Sankaranarayanan and Chakraborty (2000b).

[c] Weighted PCRF: aut. dominants: $(55.9 \times 17)/(102.35 \times 59) = 0.157$; aut. dominants + X-linked: $(60.9 \times 24)/(109.85 \times 67) = 0.199$.

Since the assignment to group 1 is less subjective (and therefore relatively more reliable), to err on the side of caution, potential recoverability was calculated as follows: if a total of N genes are analysed and if n among them could be excluded as 'unlikely to be recovered', the remainder (made up of groups 2 and 3) constitute $(N - n)$ and the fraction $(N - n)/N$ provides a crude measure of genes at which induced mutations may be recoverable. This fraction is called the 'unweighted' PRCF.

(A 240) The PRCF as estimated above, however, does not take into account differences in incidence of the different diseases. For example, if a disease with high incidence belongs to group 1, societal concern will be far less than when it belongs to the other groups. Consequently, a weighted PRCF was also calculated. If P is the total incidence of diseases due to mutations in N genes, and p is the incidence of diseases due to mutations in $(N - n)$ genes, then $[p(N - n)/PN]$ represents the 'weighted PRCF'.

(A 241) The results of analysis of a total of 67 autosomal and X-linked genes are summarised in Table A.6.2.

(A 242) ***PRCF for autosomal dominant and X-linked diseases.*** In view of the fact that autosomal dominants have an order-of-magnitude higher overall incidence than X-linked ones (1.5% versus 0.15%), the PRCFs for the former are more relevant. UNSCEAR therefore suggested the use of the PRCF range of 0.15 to 0.30 in the risk equation for estimating the risk of both autosomal dominant and X-linked diseases.

(A 243) ***PRCF for autosomal recessives.*** While the recoverability of induced recessive mutations is also subject to structural and functional constraints, in view of the fact that these mutations are first present in heterozygotes (and 50% of the gene products are generally sufficient for normal function), one can assume that even large deletions may be recoverable in the heterozygotes. Additionally, as discussed earlier, induced recessive mutations do not, at least in the first several generations, result in recessive diseases. Consequently, no attempt was made to estimate PRCF for

recessive diseases. However, it should be noted that ignoring PRCF in the risk equation is equivalent to assuming PRCF = 1, but in reality this does not affect the estimate of risk – since MC is nearly zero in the first several generations, the product of P and MC is already zero.

(A 244) ***PRCF for chronic diseases.*** As may be recalled, in the FLTM used to estimate MC for chronic diseases, one of the assumptions is that of simultaneous increase in mutation rate in all the underlying genes which, in turn, causes the liability to exceed the threshold. A crude approximation of the PRCF for each multifactorial phenotype is the xth power of that for mutations at a single locus, where x is the number of gene loci, assumed to be independent of each other, that underlie the disease. Since the PRCF for single gene mutations is in the range from 0.15 to 0.30, for chronic diseases, the figures become 0.15^x to 0.30^x. With the assumption of just two loci, the estimates become 0.02 to 0.09 and, with more loci, substantially smaller. Intuitively, these conclusions are not unexpected when one considers that here one is estimating the probability of simultaneous recoverability of induced mutations at more than one independent gene.

(A 245) UNSCEAR adopted the PRCF range of 0.02 to 0.09 with the view that the use of this range will not underestimate risk.

(A 246) ***PRCF for congenital abnormalities.*** The available data do not permit PRCF estimation for congenital abnormalities. However, since risk estimation for this class of diseases is now done without using the DD method (see the next section), our inability to estimate PRCF is not a problem.

The concept that multisystem developmental abnormalities are likely to be the major manifestations of radiation-induced genetic damage in humans

(A 247) As discussed in the preceding paragraphs, in genetic risk estimation, the emphasis has been on expressing risks in terms of inducible genetic diseases, the expectation being that their phenotypes will be similar to those known from studies of naturally occurring genetic diseases. However, when one considers the following facts it is clear that the emphasis on genetic diseases gives only a partial answer to the question of genetic risks. The facts and observations are:

- radiation induces genetic damage through random deposition of energy;
- the whole genome is the target;
- most radiation-induced mutations studied in experimental systems are DNA deletions, often encompassing more than one gene;
- the recoverability of induced deletions is subject to structural and functional constraints so that only a small proportion of them are compatible with live births; and
- the phenotype of viability-compatible deletions will reflect the gene functions that are lost because of the deletion and we do not as yet have 'windows' for all genomic regions.

It follows therefore, that the problem in genetic risk estimation is one of delineating the phenotypes of viability-compatible deletions that may be induced in different genomic regions which may or may not have counterparts in naturally occurring genetic diseases.

(A 248) ***Microdeletion syndromes in humans.*** Some inferences are now possible on the potential phenotypes of radiation-induced deletions from studies of naturally occurring microdeletion syndromes in humans. These result from deletions of multiple, physically contiguous, often functionally unrelated, genes that are compatible with viability in heterozygous condition and are identified clinically through a characteristic association of unusual appearance and defective organ development. Many examples of microdeletions have been (and continue to be) reported in the human genetics literature. They have been found in nearly all the chromosomes, but their occurrence in different chromosomal regions is non-random (e.g., Brewer et al., 1998). This is not unexpected in the light of differences in gene density in different chromosomes/chromosomal regions. The important point here is that, despite their occurrence in different chromosomes, the common denominators of the phenotype of many of these deletions are: mental retardation, a specific pattern of dysmorphic features, serious malformations, and growth retardation. These findings in humans are supported, among others, by studies of Cattanach et al. (1993, 1996) showing that, in the mouse, radiation-induced multilocus deletions constitute the genetic basis for a significant proportion of growth-retarded animals recovered in their work.

(A 249) It was therefore suggested that the predominant adverse effects of gonadal irradiation in humans are likely to be manifest as multisystem developmental abnormalities which are formally called 'congenital abnormalities' (Sankaranarayanan, 1999). However, unlike naturally occurring congenital abnormalities which are interpreted as being multifactorial, radiation-induced congenital abnormalities, because

Table A.6.3. Current estimates of genetic risks from continuing exposure to low-LET, low-dose or chronic irradiation (UNSCEAR, 2001) with assumed doubling dose of 1 Gy.

Disease class	Baseline frequency (per million live births)	Risk per Gy per million progeny:	
		1st generation	2nd generation
Mendelian			
Autosomal dominant & X-linked	16,500	~750 to 1500[a]	~1300 to 2500
Autosomal recessive	7500	0	0
Chromosomal	4000	[b]	[b]
Multifactorial			
Chronic	650,000[c]	~250 to 1,200	~250 to 1,200
Congenital abnormalities	60,000	~ 2,000[d]	~ 2400 to 3000[e]
Total	738,000	~3000 to 4700	~3950 to 6700
Total per Gy expressed as per cent of baseline		~0.41 to 0.64	~0.53 to 0.91

[a] The ranges reflect biological and not statistical uncertainties.

[b] Assumed to be subsumed in part under autosomal dominant and X-linked diseases and in part under congenital abnormalities.

[c] Frequency in the population.

[d] Estimated from mouse data without using the DD method.

[e] Newly induced damage of pre-existing damage (It is assumed that 20–50% of the progeny affected in the first generation will transmit the damage to the next generation resulting in 400 to 1000 cases.)

they are multilocus deletions, are predicted to show, by and large, autosomal dominant patterns of inheritance. This prediction has been fulfilled in mouse radiation studies on skeletal abnormalities (Ehling, 1965, 1966, Selby and Selby, 1977), cataracts (Favor, 1989), growth retardation (Searle and Beechey, 1986), and congenital anomalies (Kirk and Lyon, 1984, Lyon and Renshaw, 1988, Nomura, 1982, 1988, 1994). No transmission tests could be carried out, however, for congenital abnormalities because they were ascertained in utero.

(A 250) ***Risk of developmental abnormalities.*** UNSCEAR (2001) used the mouse data on skeletal abnormalities, cataracts and congenital abnormalities (appropriately adjusting the rates for chronic low-LET radiation conditions) to obtain an overall estimate of the risk of developmental abnormalities about 20 10^{-4} Gy^{-1} (given in Table A.6.3 in this document under the heading 'congenital abnormalities' as 2000 per Gy per million for the first generation). All the data used in these calculations come from studies of irradiation of males, and the rate so estimated was assumed to be applicable to both sexes.

A.6.4. The 2001 UNSCEAR risk estimates

Estimates of genetic risk for a population sustaining radiation exposure generation after generation

(A 251) Table A.6.3 summarises the risk estimates presented in the 2001 UNSCEAR report. The risks given below and in the tables are expressed as the predicted number of additional cases (i.e., over the baseline) of different classes of genetic disease per million live births per Gy for a population exposed to low-LET, low-dose or chronic irradiation, generation after generation. For all classes except congenital abnormalities, the estimates are based on a DD of 1 Gy and the respective values of P, MC and PRCF for the different classes. For congenital abnormalities, the risk estimate comes from mouse data (discussed in the preceding paragraph) and is not based on the DD method.

(A 252) As can be noted from Table A.6.3, the first generation risk (i.e., the risk to the children of an exposed population) is estimated to be of the order of 750 to 1500 cases per million live births per Gy for autosomal dominant and X-linked diseases, zero for autosomal recessive diseases, 250 to 1200 cases for chronic diseases and 2000 cases of congenital abnormalities. The total risk is of the order of about 3000 to 4700 cases which represent about 0.4 to 0.6% of the baseline risk.

(A 253) The risk to the second generation (i.e., to the grandchildren) becomes slightly higher for all classes except for chronic diseases in view of the fact that the mutation component for these diseases does not increase over the first several generations.

Estimates of genetic risks for a population that sustains radiation exposure in one generation only

(A 254) The estimates of genetic risk under conditions when the population sustains radiation exposure in one generation only (and no further radiation in subsequent generations) are presented in Table A.6.4. Again, all estimates are expressed

Table A.6.4. Current estimates of genetic risks from one-generation exposure to low-LET, low-dose or chronic irradiation (UNSCEAR, 2001) with assumed doubling dose of 1 Gy.

Disease class	Baseline frequency (per million live births)	Risk per Gy per million progeny:	
		1st generation	2nd generation
Mendelian			
Autosomal dominant & X-linked	16,500	~750 to 1,500[a]	~500 to 1,000
Autosomal recessive	7,500	0	0
Chromosomal	4,000	[b]	[b]
Multifactorial			
Chronic	650,000[c]	~250 to 1,200	~250 to 1,200
Congenital abnormalities	60,000	~ 2,000[d]	~ 400 to 1,000[e]
Total	738,000	~3,000 to 4,700	~1,150 to 3,200
Total per Gy expressed as per cent of baseline		~0.41 to 0.64	~0.16 to 0.43

[a] Risk to second generation is lower than that in the first because of the assumption that the radiation exposure occurs in one generation only; the risk will progressively decrease with time (in generations).

[b] Assumed to be subsumed in part under the risk of autosomal dominant and X-linked diseases and in part under that of congenital abnormalities.

[c] Frequency in the population.

[d] Estimate obtained using mouse data on developmental abnormalities and not with the doubling dose method.

[e] Under the assumption that about 20 to 50% of those affected in the first generation transmit the damage to the next generation.

per Gy per million progeny. As expected, the first generation risks (i.e., risks to the children of those exposed) are the same as those given in Table A.6.3. With no further radiation, the risk of autosomal dominant and X-linked diseases to the second generation (i.e., to the grandchildren) declines as a result of selection. For chronic multifactorial diseases, since the mutation component remains low for several generations, the risk to the second generation remains about the same as that in the first generation. The risk of congenital abnormalities is predicted to be of the order of 400 to 1000 cases (under the assumption that about 20 to 50% of those affected in the first generation transmit the damage to the next generation).

Strengths and limitations of the risk estimates

(A 255) On the basis of UNSCEAR (2001) the Commission has, for the first time, been able to provide ICRP estimates of risks for all classes of genetic diseases. While these estimates reflect the current state of knowledge in this area, the strengths and limitations of these estimates need to be borne in mind, in view of various assumptions that have been used.

(A 256) ***Equal mutational sensitivity of human males and females.*** The prevalent view that the mouse immature oocytes may not be an adequate model for assessing the mutational radiosensitivity of human immature oocytes necessitated the assumption that human females and males have the same mutational radiosensitivity which in turn is equal to that of mouse males. If, however, human females have a lower

sensitivity in this regard, the average rate of induced mutations would be expected to be lower than the one used. In turn, this implies that the DD will be higher (and 1/DD will be smaller than 0.01 that has been used). At present it is not possible to address this issue.

(A 257) ***Average spontaneous and induced mutation rates used in DD calculations.*** As may be recalled, the average estimate of 2.95 10^{-6} per human gene was based on an estimated 135 genes underlying some 26 autosomal dominant disease phenotypes which constitute a subset of such diseases included in the estimate of baseline frequencies. Bearing in mind the fact that the human genome contains about 30,000 genes, one can only speculate whether the above average spontaneous mutation rate estimate is an over- or underestimate of the true average rate.

(A 258) Similarly, although the estimate of induced mutation rate for mouse genes is based on more data than was the case until now, the total number of genes included in the present analysis is still only 34 and, in a sizeable proportion of them, induced mutations were rare. Therefore, while the possibility remains that the presently estimated induced rate may be biased upwards, its extent is difficult to determine at present.

(A 259) ***Mutation components.*** The estimate MC = 0.3 for autosomal dominant and X-linked diseases is based on the average s value for the autosomal dominant diseases (since MC = s in the first generation), the data of which provided the basis for spontaneous mutation rate calculations. However, it should be realised that, for a substantial proportion of diseases, onset is in middle and later ages (i.e., beyond the age of reproduction) which means that s is smaller and therefore the MC value used may be an overestimate.

(A 260) ***Potential recoverability correction factors.*** For autosomal dominant and X-linked diseases, a range of PRCF from 0.15 to 0.30 was used, the lower limit being a weighted estimate and the upper limit, an unweighted one. However, the criteria developed for potential recoverability of induced deletions do not include breakpoint specificities which are undoubtedly important in the case of deletion-associated naturally occurring Mendelian diseases. It seems unlikely that radiation-induced deletions would share these specificities, and certainly not in all genomic regions. If these specificities are indeed relevant for recovering induced deletions, even the weighted PRCF may be an overestimate.

(A 261) For chronic diseases, it has been assumed that the PRCF may simply be the xth power of that for a single-gene disease, with x = the number of genes which have to be simultaneously mutated to cause disease; the values of 0.02 to 0.09 have assumed $x = 2$ (the minimum number). Although, statistically, such a calculation can be defended, the implicit biological assumption that, at low doses of radiation, two independent mutations underlying a chronic disease may be simultaneously induced and recovered seems unrealistic.

(A 262) There is an additional issue here, namely that the PRCF for chronic diseases is very sensitive to x (e.g., even if $x = 3$, the PRCF range becomes 0.003 to 0.03). The essence of the argument then is that the PRCFs used for chronic diseases may overestimate the risk.

(A 263) ***Overlap in estimates of risk.*** It should be recalled that: a) the estimates for autosomal dominant and X-linked diseases have been obtained using the DD method; b) the risk of induced congenital abnormalities which are also adverse dominant effects have been estimated independently using mouse data without recourse to the DD method; and c) the risk of 'chromosomal diseases' has been assumed to be subsumed under the risk of autosomal dominant and X-linked diseases. The important point here is that, since all these represent dominant effects (and mutations in many developmental genes are known to cause Mendelian diseases), there must be overlap between the classes of risk grouped under the headings of 'autosomal dominant + X-linked' and 'congenital abnormalities' although it is difficult to assess its magnitude. The consequence is that the sum may overestimate the actual risk of dominant effects.

A.6.5. Earlier and present assessments of risk estimates by ICRP for deriving risk coefficients for genetic effects

ICRP Publication 60

(A 264) In *Publication 60* (ICRP, 1991b), the commission used the genetic risk estimates then available (UNSCEAR, 1988, NRC, 1990) as a starting point for deriving risk coefficients for 'severe heritable effects'. It is important to mention here that in the commission's calculations then, while the DD assumed (1 Gy) was the same as that used now, the baseline frequency of Mendelian diseases was only about one-half of that currently used (1.25% then versus 2.4% now). Additionally, for multifactorial diseases as a whole (estimated baseline frequency of 71%; same as now), the Commission assumed that MC = 0.05 for all post-radiation generations (this assumption is incorrect in the light of current calculations; see paragraphs (A 216) to (A 234)) and also incorporated an additional arbitrary correction factor (called 'severity correction factor') of 1/3 to estimate the proportion of inducible multifactorial diseases that may be deemed 'severe' (no such correction is used in the present assessments).

(A 265) For a population exposed to low-dose-rate, low-LET irradiation, the risk coefficients estimated by ICRP (1991b) are summarised in Table A.6.5 (see also Table 3 of Sankaranarayanan 1991).

(A 266) The estimates for the 'reproductive population' apply when the radiation doses received by all individuals in the population are genetically significant. However, when the total population of all ages is considered, the genetically significant dose will be markedly lower than the total dose received over a lifetime. Genetic damage sustained by germ cells of individuals who are beyond the reproductive period, or who are not procreating for any reason, poses no genetic risks. On the assumption that the average life expectancy at birth is of the order of 75 years, the dose received by 30 years of age (i.e., the mean reproductive age) is 40% (i.e., 30/75 = 0.4) of the total dose. The risk coefficients for the total population, therefore, are estimated to be 40% of the above values.

(A 267) Although ICRP (1991b) presented risk coefficients for the first two generations as well as for the new equilibrium, it used the equilibrium estimate of 1.0 10^{-2} Gy^{-1} for the total population (with an additional weighting factor for years of life

Table A.6.5. Estimates of risk coefficients in *Publication 60* for a population sustaining continuous radiation exposure, generation after generation (ICRP, 1991b, Sankaranarayanan, 1991).

Time span	Disease category	Risk coefficient in % per Gy for	
		Reproductive population	Total population
Up to two generations	Mendelian & chromosomal	0.3	0.1
	Multifactorial	0.23	0.09
	Total	0.53	0.19
New equilibrium	Mendelian & chromosomal	1.2	0.5
	Multifactorial	1.2	0.5
	Total	2.4	1.0[a]

[a] The estimate used by ICRP (1991b) in its summary of 'nominal probability coefficients for stochastic effects' (Table 3, ICRP, 1991b); the figure given in that Table of 1.3 10^{-2} Gy^{-1} takes into account a weighting factor for years of life lost (ICRP, 1991b).

lost to arrive at a figure of 1.3 10^{-2} Gy^{-1} for 'severe heritable effects' in its summary table of 'nominal probability coefficients' (Table 3, ICRP 1991b).

Current assessments

(A 268) In its current assessments, the Commission used the estimates of risk presented in Table A.6.3 as starting points. The upper and lower limits of each of the estimated ranges were first used to obtain average estimates, and the latter were then combined to generate a single estimate of risk coefficient for all genetic effects. Details of calculations are given in the next section.

(A 269) ***Risk coefficients up to two generations for a population sustaining radiation exposure in every generation.***

- risk of Mendelian diseases = 1300 to 2500 cases per 10^6 progeny per Gy (= 0.13 10^{-2} to 0.25 10^{-2} Gy^{-1}; average: 0.19 10^{-2} Gy^{-1});
- risk of chronic multifactorial diseases = 250 to 1200 cases per 10^6 progeny per Gy (= 0.03 10^{-2} Gy^{-1} to 0.12 10^{-2} Gy^{-1}; average: 0.08 10^{-2} Gy^{-1});
- risk of congenital abnormalities = 2400 to 3000 cases per 10^6 progeny per Gy (0.24 10^{-2} to 0.30 10^{-2} Gy^{-1}; average: 0.27 10^{-2} Gy^{-1}); and,
- risk of all classes (i.e., the above three risks combined) = 3950 to 6700 cases per 10^6 progeny per Gy or 0.40 10^{-2} to 0.67 10^{-2} Gy^{-1}; average: 0.54 10^{-2} Gy^{-1}.

The above estimates are for a reproductive population. For the total population, the estimates are multiplied by 0.4. All the estimates are summarised in Table A.6.6.

(A 270) It is evident that, despite different baseline frequencies for Mendelian diseases, MCs and differences in risk estimates for comparable classes of diseases, the present estimates for the reproductive (0.54) as well as for the total population (0.22) are remarkably similar to those arrived at in ICRP *Publication 60* (1991b); respectively, 0.53 and 0.19; see Table 5. It should be stressed that this similarity is a matter of pure coincidence!

Table A.6.6. Risk coefficients for the reproductive and the total population obtained up to two generations when the population sustains radiation exposure generation after generation (all values expressed in percent per Gy).

Disease class	Reproductive population		Total population
	Range	Average[a]	Average[b]
(a) Mendelian diseases	0.13 to 0.25	0.19	0.08
(b) Chronic diseases	0.03 to 0.12	0.08	0.03
(c) Congenital abnormalities	0.24 to 0.30	0.27	0.11
Total for all classes		0.54	0.22

[a] Average of the limits of the indicated ranges.
[b] 40% of that for the reproductive population.

(A 271) As may be recalled, the ranges in the estimates of risk coefficients for Mendelian and chronic diseases are a reflection of the ranges of PRCFs (0.15 to 0.30 for autosomal dominant and X-linked diseases and 0.02 to 0.09 for chronic diseases). Arguments to suggest that the upper limits of these ranges may represent overestimates and that the actual values may be closer to the lower limits were presented in Section A.6.3. If this reasoning is accepted, then it is meaningful to use the lower limit of the ranges for the above two classes of diseases and the average of the range for congenital abnormalities. When this is done, the risk coefficients become smaller than those presented in Table A.6.6 as noted below:

- *Reproductive population:* Mendelian diseases, 0.13; chronic diseases, 0.03; congenital abnormalities, 0.27; Total: 0.43 10^{-2} Gy^{-1}
- *Total population:* Mendelian diseases, 0.05; chronic diseases, 0.01; congenital abnormalities, 0.11; Total: 0.17 10^{-2} Gy^{-1}

(A 272) ***Risk coefficients for the first post-radiation generation only.*** The risk coefficients for the first post-radiation generation are summarised in Table A.6.7. Again as expected, the values are smaller than those up to the first two generations.

(A 273) If, however, the lower limits of the ranges for Mendelian and chronic diseases are used, then the estimates are 0.30 10^{-2} Gy^{-1} for the reproductive population

Table A.6.7. Risk coefficients for the reproductive population and the total population for the first post-irradiation generation (all values are expressed as per cent per Gy).

Disease class	Reproductive population		Total population
	Range	Average[a]	Average[b]
(a) Mendelian diseases	0.075 to 0.150	0.11	0.05
(b) Chronic diseases	0.025 to 0.120	0.07	0.03
(c) Congenital abnormalities	–	0.20	0.08
Total for all classes		0.38	0.16

[a] Average of the limits of the indicated ranges.
[b] 40% of that for the reproductive population.

(i.e., 0.075 + 0.025 + 0.20 = 0.30) and 0.12 10^{-2} Gy^{-1} for the total population (i.e., $[0.075 \times 0.4] + [0.025 \times 0.4] + [0.20 \times 0.4] = 0.12$).

Justification for using risk estimates up to generation two versus for calculating risk coefficients

(A 274) There are some problems in comparing genetic risk coefficients with those for cancers. This is because of the fact that cancer risk coefficients quantify the probability of harmful effects of radiation to the exposed individuals themselves, and genetic risk coefficients quantify the probability of harmful effects to the descendants of those exposed resulting from the induction of germline mutations and their transmission over generations. Following consideration of the available data and the recent analyses of UNSCEAR (2001) and NAS/NRC (2006), the Commission position is to express genetic risks up to the second generation (Table A.6.6). As given below, there are important scientific arguments that favour this approach.

(A 275) The population genetic theory of equilibrium between mutation and selection that underlies the use of the doubling dose method and the available mathematical formulations permit, in principle, the prediction of genetic risks at the new equilibrium (under conditions of continuous radiation in every generation). As noted earlier, in the absence of informative analyses and in order not to underestimate genetic risks, *Publication 60* (ICRP, 1991b) used the equilibrium estimates as a basis for calculating risk coefficients for genetic effects. The current arguments against such an equilibrium calculation centre on the very unrealistic and untestable assumptions that a) the estimates of selection coefficients, mutation components, and the other quantities used in the risk equation, will remain valid for tens or hundreds of human generations; b) the population structure, demography and health care facilities will remain constant over many hundreds of years.

(A 276) In the view of the Commission these assumptions can no longer be sustained and therefore, for the practical purposes of radiological protection, the Commission recommends a genetic risk estimate based upon risks up to the second generation. UNSCEAR (2001) and NAS/NRC (2006) have made the same judgement on this matter.

(A 277) The concepts that a) radiation-induced genetic changes are predominantly deletions, often encompassing more than one gene, and that only a small proportion of such induced deletions is compatible with live births, and b) radiation-induced heritable effects in humans are more likely to be manifest as multisystem developmental abnormalities in the progeny rather than as diseases due to mutations in single genes, are particularly relevant to this issue. Because reproductive fitness of the affected progeny will be reduced, many radiation-induced genetic changes affecting development are expected to be strongly selected against at the first and second generations. It is judged therefore that expressing genetic risks up to the second generation will not lead to any substantial underestimate of the heritable effects of radiation.

(A 278) Nevertheless a degree of caution is used in the derivation of a tissue weighting factor for the gonads. In respect of whole populations, Table A.4.1a gives relative detriment values of 0.044 for heritable effects and 0.017 for ovarian cancer.

The sum of these computed values, 0.061, is less than the judged tissue weighting factor of 0.08 (Table A.4.3).

(A 279) In addition, the Commission notes that because of the different ways used to calculate the risk of autosomal dominant plus X-linked disease (the DD method) and congenital abnormalities (directly from mouse data), there must be a considerable element of 'double counting' of risk. Therefore, the summing of these risk categories, as used conventionally by UNSCEAR and ICRP, must represent a significant overestimate of genetic risk overall.

(A 280) Finally the Commission has considered whether an estimate of genetic risks at say 5 or 10 generations might be more appropriate. This judgement can be informed by some of the model predictions provided by UNSCEAR (UNSCEAR 2001).

(A 281) With the parameters specified, the model used by UNSCEAR and by the Commission predicts that, for a permanent increase in mutation rate, the responsiveness of disease incidence (mutation component, MC) is most pronounced for autosomal dominant diseases, less so for X-linked diseases, and far less pronounced for autosomal recessives. In this respect, for autosomal dominants, disease frequency in the population at generations 5 and 10 is predicted to be less than a factor 1.5 greater than that at generation 2 (Fig. V, UNSCEAR, 2001).

(A 282) The position regarding the responsiveness of multifactorial diseases is illustrated in Fig. VII of UNSCEAR 2001 which gives the relationship between mutation component and heritability of liability. These relationships are not significantly different at generations 1, 5 and 10. Further to this, for the dose rate of interest the model predicts minimal responsiveness (MC_{TU}) of these disorders at generation 10 to a permanent increase in mutation rate.

(A 283) It is notable that the above modelling predictions are wholly consistent with several animal genetic studies (largely with mice) that provide no evidence of the accumulation of a mutational load following x-irradiation at each generation up to more than 30 generations (reviewed by Green 1968 and UNSCEAR 1972).

(A 284) Overall, the Commission concludes that expressing the heritable risks of radiation at generations 5 or 10 rather than 2 would not materially affect judgements on the risk coefficient.

(A 285) In conclusion, the Commission, whilst fully recognising uncertainties, agrees with the UNSCEAR 2001 judgement (paragraph 531) that 'the risk estimates presented for the first two generations adequately reflect the current state of knowledge in this evolving area'. ICRP will maintain surveillance on scientific developments in the area and, if judged to be appropriate, will revise its estimates of these heritable risks.

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A.7. Summary of principal conclusions and proposals

(A 286) Although additional work was required, many of the conclusions and proposals from the Task Group that drafted this Annex are based upon ICRP Committee 1 judgements developed over the past 10 years or so. Accordingly many sections of the Annex are themselves summaries of these pre-existing judgements. For this reason a simple tabular format (Table A.7.1) has been used to provide an overall summary of the principal conclusions that have now been adopted by the Commission. The inclusion in Table A.7.1 of identifiers for the relevant sections and tables for each topic serves to map the document and guide readers to the topic of interest. These sections often detail methodologies, uncertainties and caveats not fully reflected in Table A.7.1. Accordingly Table A.7.1 cannot be taken as being fully informative of Commission views and judgements.

(A 287) The Commission also wishes to emphasise an important issue discussed in Annex B (drafted by a Task Group of ICRP Committee 2) of these Recommendations. The conclusions and proposals summarised in Table A.7.1 are principally for the broad purposes of prospective planning in radiological protection. For other purposes many of the proposed judgements may well be insufficient, and in these circumstances, specific, well-justified judgements on radiation effects and their health risks will need to be made.

Table A.7.1. Summary of principal conclusions and proposals specifically intended for radiological protection purposes.

	Topic	Data source/methodology	Conclusions/numerical judgements
1	Dose response at low doses/dose rates for cancer and heritable effects (*Sections A.2.1–A.2.5, A.2.7–A.2.8, A.4.1 paragraphs A89-A96*)	Judgements based on studies reviewed in *Publication 99* (ICRP, 2005d), UNSCEAR 2000, 2001, NCRP 2001, NAS/NRC 2006	Uncertainties are considerable but the balance of evidence weighs in favour of the use of a simple proportionate relationship between increments of dose and risk
2	Role of induced genomic instability, bystander signalling and adaptive responses in the risk of induced health effects (*Sections A.2.3, A.2.5, A.4.1 paragraphs A90-A97*)	Judgements based on studies reviewed in ICRP *Publication 99*, NCRP 2001, UNSCEAR 2000, UNSCEAR 1994, NAS/NRC 2006	Knowledge of these biological effects is growing but is currently insufficient for radiological protection purposes
3	Relative biological effectiveness and radiation weighting factors (w_R) (*Section A.4.3*)	Judgements based upon recommend-ations included in *Publication 92* (ICRP, 2003c)	Judgements are fully developed in Annex B
4	Dose and dose-rate effectiveness factor (DDREF) and the impact of a possible dose threshold (*Sections A.2.4, A.4.2, A.4.4 paragraphs A125-A148, A.4.4 paragraphs A173-A187*)	Judgements largely based upon studies reviewed in *Publication 99,* UNSCEAR 2000 and NAS/NRC 2006	A DDREF value of 2 should be retained for use by ICRP; the uncertain possibility of a low-dose threshold for cancer risk is equivalent to an uncertain increase in the value of DDREF.
5	Radiation detriment and tissue weighting factors (w_T) (*Section A.4.4 paragraphs A105-A162*)	New judgements developed largely from cancer incidence in the A-bomb Life Span Study (LSS), international cancer mortality databases and new estimates of heritable effects (see 7 below); judgements supported by additional consideration of cancer mortality data	Revised w_T scheme proposed; significant w_T changes for breast and gonads (see Table A.4.3), revised method for treatment of remainder tissues (see Table A.4.3)
6	Detriment adjusted nominal risk coefficients for cancer (*Section A.4.4 paragraphs A105-A162*)	New risk estimates developed based upon lethality/life impairment weighted data on cancer incidence (see 5 above)	Detriment adjusted nominal risk coefficients of 5.5 10^{-2} Sv^{-1} for the whole population and 4.1 10^{-2} Sv^{-1} for adult workers are proposed (see Table A.4.4.)

7	Detriment adjusted nominal risk coefficients for heritable effects (*Section A.6*)	New risk estimates based upon UNSCEAR 2001 judgements using risks for all classes of heritable effects up to the second post-irradiation generation (see Tables A.6.4 and A.6.6)	Second generation, detriment adjusted nominal risk coefficients of 0.2 10^{-2} Sv^{-1} for the whole population and 0.1 10^{-2} Sv^{-1} for adult workers are proposed (see Table A.4.4); *Publication 60* (ICRP, 1991b) used population genetic risks at a theoretical equilibrium so the present estimates are markedly lower
8	Cancer risk following in-utero exposures (*Section A.4.4 paragraphs A168-A171*)	Judgements based upon the studies reviewed in *Publication 90* (ICRP, 2003a)	Life-time cancer risk judged to be no greater than that following exposure in early childhood
9	Genetic susceptibility to radiation-induced cancer (*Sections 2.7 paragraphs A46-A48, A.4.4 paragraph 172*)	Judgements based upon studies reviewed and analyses made in *Publication 79* (ICRP 1998a), UNSCEAR 2000, 2001 and NAS/NRC 2006	Strongly expressing cancer-predisposing disorders are too rare to appreciably distort risk estimates for the whole population; the impact of potentially common but weak genetic determinants remains uncertain
10	Radiation-induced tissue reactions in adults (*Sections A.2.6 and A.3*)	Mechanisms have been re-evaluated and dose thresholds for morbidity/mortality revised on the basis of various data	Tables A.3.1, A.3.2, and A.3.4 provide revised judgements but with few changes from other ICRP publications. The dose threshold for cataract induction and judgements on dose limits for the eye require further attention.
11	In-utero risks of tissue reactions, malformations and neurological effects (*Section A.3.2*)	Judgements based upon studies reviewed in *Publication 90*	Strengthened judgement on the existence of a dose-threshold for tissue reactions, malformation and severe mental retardation – therefore, absence of risk at low doses. Greater uncertainty for IQ deficits but low-dose risk judged to have no practical significance
12	Risks of non-cancer diseases (Section A.5)	Judgements based upon LSS data and studies on post-radiotherapy outcomes particularly for cardiovascular disease	Great uncertainty on the form of the dose-response below 1 Sv – no specific judgement on low-dose risk is possible

A.7.1. References, Section A.7

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ANNEX B. QUANTITIES USED IN RADIOLOGICAL PROTECTION

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Preface to Annex B

When the Commission initiated its project to review and update its 1990 Recommendations, at the Main Commission meeting in Cape Town, South Africa, in 1998, it was clear from the outset that the main text of the new Recommendations would need to be supported by scientific Annexes and reports in much the same manner as the 1990 Recommendations.

Therefore, ICRP Committees 1 (on radiation effects) and 2 (on doses from radiation exposure) were asked to outline and begin to draft Annexes on the health effects of radiation and on dosimetric considerations. (Committees 3 on protection in medicine and 4 on application of ICRP recommendations were similarly asked to produce supporting documents which were and are being published as separate reports: *Publication 105*, ICRP (2007b) on protection in medicine and *Publication 101*, ICRP (2006a), on assessing dose to the representative person and on optimisation).

After initial plenary work, Committee 2 formed a Task Group in 2001 to advise the Main Commission and draft the present Annex to the Recommendations.

The membership of the Task Group was as follows:

C. Streffer, Chair	G. Dietze	K. Eckerman
J. Harrison	H. Menzel	J. Stather

The corresponding members were:

W. Alberts	M. Balonov	V. Berkovski
A. Bouville	A. Edwards	J. Lipsztein
M. Pelliccioni	A. Phipps	A. Pradhan

The membership of ICRP Committee 2 during the preparation of this Annex was: (2001–2005)

C. Streffer, Chair	B. Boecker	A. Bouville
G. Dietze	K. Eckerman	J. Inaba
I. Likhtarev	J. Lipsztein	H. Menzel
H. Métivier	H. Paretzke	A. Pradhan
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(2005–2009)

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Executive summary

(B a) Dosimetric quantities are needed to assess radiation exposures to humans and other organisms in a quantitative way. This is necessary in order to describe dose–response relationships for radiation effects which provide the basis for risk estimation in radiological protection.

(B b) Absorbed dose, D, is the basic physical quantity for radiological protection. It is defined as the mean of the distribution of energy deposited in a tissue volume. It is well defined at any point in matter. It is measurable. In the low dose range, important for radiological protection, the distribution of energy deposition is heterogeneous, particularly in the case of exposure to high-LET radiation. In practical applications, averaging of absorbed dose over organ or tissue volumes is performed. It is assumed that the mean value of absorbed dose in an organ or tissue is correlated with radiation detriment from stochastic effects in the low dose range. The averaging of absorbed doses in tissues and organs of the human body and their weighted sum are the basis for the definition of protection quantities. Dose distributions that are highly heterogeneous (e.g., DNA precursors labelled with tritium or Auger emitters) may need special treatment.

(B c) The definition of protection quantities is based on the mean absorbed dose, $D_{\mathrm{T,R}}$, in an organ or tissue T, due to radiation of type R. The protection quantity equivalent dose, H_{T}, is defined by

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

where w_{R} is the radiation weighting factor for radiation R. These w_{R} values are based on experimental data for the relative biological effectiveness (RBE) of various types of radiations at low doses, on biophysical considerations and on judgements. A set of w_{R} values was given in *Publication 60* (ICRP, 1991b). The general concept of these values remains unchanged. Some modifications are recommended: The w_{R} value for protons is reduced to a factor of 2 and a continuous function is used for neutrons with a reduction of the w_{R} value to 2.5 at energies below 10 KeV and above 1 GeV. The principal definition of effective dose,

$$E = \sum_{\mathrm{T}} w_{\mathrm{T}} H_{\mathrm{T}}$$

remains unchanged from *Publication 60* (ICRP, 1991b). However, some of the tissue weighting factors, w_{T}, have been changed on the basis of new epidemiological data for cancer induction (see Annex A).

(B d) The w_{T} values are age- and sex-averaged. Therefore E is not calculated for an individual but for a Reference Person. The Commission has now defined sex-specific computational phantoms for a Reference Male and a Reference Female. These phantom models will be used for calculations of dose conversion coefficients for external exposures and dose coefficients for internal exposures. The new sex-specific computational models allow the calculation of male and female organ doses separately, from which the averaged equivalent organ doses are calculated. These are

used for the calculation of E. Computational phantoms for chinldren of various ages and the fetus will also be defined. With the assumption of the linear-non-threshold dose response for stochastic radiation effects (LNT model) in the low dose range (< 100 mSv) and, under the conditions of the described concept of calculation, E is an additive quantity. At higher radiation doses, when tissue reactions (deterministic effects) can occur, the absorbed doses in organs and tissues have to be used for risk evaluation. In the case of high-LET radiation exposures, appropriate RBE values relating to deterministic effects should be used.

(B e) The body related protection quantities (equivalent dose and effective dose) cannot be applied directly in radiation monitoring as they are not directly measurable. Operational quantities are measured instead for the assessment of E and H_T. For external exposures, operational dose equivalent quantities have been defined for area and individual monitoring. Measurements with an area monitor are preferably performed free in air, and personal dosimeters are worn on the body. The radiation fields 'seen' by these dosimeters differ and therefore different operational dose quantities have been defined. Dose equivalent quantities, based on doses to the depth of 10 mm and 0.07 mm of the ICRU sphere or in the human body respectively, have been recommended. E and organ doses are calculated by dose conversion coefficients for external exposure. For dose assessment from internal exposures, the intake of radionuclides and the resultant equivalent dose as well as the effective dose are calculated on the basis of direct (e.g., measuring radioactivity of the whole body) or indirect (e.g., measuring the radioactivity in excreta) measurement using biokinetic models describing the behaviour of the radionuclide in the body.

(B f) Dose quantities in radiological protection for workers and the general public are needed mainly for prospective dose assessment in planned exposure situations and optimisation as well as for retrospective dose assessments for testing compliance with dose limits. The intake of a radionuclide during a year is assigned a committed effective dose. A commitment period of 50 years is considered for adults, and to age 70 years for children. The annual effective doses of workers and of members of the public are the sum of the effective dose obtained within one year from external exposure and the committed effective dose from radionuclide intake during this year.

(B g) For external exposures at workplaces usually the effective dose is assigned by measuring personal dose equivalent, Hp(10), as an acceptable assessment, assuming uniform whole body exposure. The committed effective dose from intakes of radionuclides is assessed by considering the ingestion and inhalation of radioactive materials. Public exposures can occur from natural radiation sources and from technical installations. Doses are mainly determined by environmental measurements, habit data and modelling. The use of E for medical exposures of patients has important limitations, as often only parts of an organ or the human body are exposed, and the age distribution of patients differs from that of the general public; other factors may also need to be considered.

(B h) The primary use of E is for demonstrating compliance with dose limits. In that sense it serves to limit and to regulate the occurrence of stochastic effects in the low dose range, and it is used for regulatory purposes worldwide. E is calculated on the basis of reference values for a Reference Person. The weighting factors are

selected from a range of experimental and epidemiological data by judgement, and they apply to a population of all ages and both sexes. For retrospective dose and especially risk assessments in individual cases individual parameters such as sex, age and organ doses would need to be taken into account. *E* should not be used for epidemiological studies. In the case of accidents that could give rise to deterministic effects it is necessary to estimate absorbed dose and dose rates to organs and tissues. Considerations of threshold doses are then important and, for exposures to high-LET radiation, appropriate RBE values have to be chosen.

(B i) Collective effective dose is retained as an important and useful instrument for optimisation especially for occupational exposures. In the past, collective effective dose was frequently computed as the sum of radiation exposures over a wide range of doses, over long time periods and over large geographical regions. On this basis radiation-related detriments have been calculated. Such calculations are not meaningful because large uncertainties are included with respect to the dose assessments and extrapolation procedures from high and medium radiation doses to very low doses. To avoid the aggregation of low individual doses over extended time periods, limiting conditions have to be set. The following aspects could be considered: number of exposed individuals, age and sex of exposed individuals, range of individual doses, dose distribution in time, and geographical distribution of exposed individuals.

(B j) For the dose assessments in radiological protection a number of models and parameter values are necessary. These have been developed from experimental investigations and human studies in order to derive 'best estimates' of model parameter values. It is recognised that in some cases there may be large uncertainties in these values. Besides these uncertainties, the biological variability is high for many parameters and therefore reference values have to be selected from a wide range of values. These reference values and models have been fixed by convention and thus are point values without uncertainty. They are periodically re-evaluated and may be updated when new scientific data become available. The reference systems are mainly developed for prospective dose assessments in regulatory processes. For dose assessments, and especially for risk estimates in dose ranges above the dose limits, and in individual cases, uncertainties in models and parameter values may need to be taken into consideration.

B.1. Introduction

(B 1) For establishing principles and systems of radiological protection, dosimetric quantities are needed to assess the radiation exposures of humans and other organisms in a quantitative way. The quantification of radiation doses for exposed human populations or experimental animals is also important for developing dose–response relationships for radiation effects. Such relationships are used over wider dose ranges than those for which data are available, particularly in the low dose range, which is important for radiological protection.

(B 2) The development of health effects caused by ionising radiation starts with the physical processes of energy absorption in biological tissues, resulting in ionisations which cause molecular changes and which may occur in clusters, e.g., in the genetic information of cells, the DNA in the cell nucleus. This damage manifests itself as radiation damage to the organs and tissues of the body which can result in both short-term and long-term health effects. At high doses acute damage to organs and tissues mainly arises as a result of loss of function involving cell killing and, in extreme cases, can cause death of the exposed individual. These types of damage are termed *deterministic effects* (*Publication 60*, ICRP, 1991b) or *tissue reactions* (see Annex A, paragraph A 56), having previously been called *non-stochastic* effects in *Publication 26* (ICRP, 1977). At lower doses and at low dose rates these tissue reactions are not seen, but damage to the genetic material may occur that can result in an increase in the risk of cancer observed years later, or heritable disease in future generations. Such damage continues to be termed *stochastic* as the probability of the effect, but not its severity, is assumed to increase with dose.

(B 3) Other interactions with cells, organs and tissues may also be important in understanding the response of the body to radiation exposure (for example damage to membranes), as described in Annex A. However, it is concluded that the information on the implications of other responses in terms of the observed tissue effects is unclear at present, and that such effects cannot at present be taken into account in dose and risk assessments for protection purposes.

(B 4) Radiological protection is concerned with controlling exposures to ionising radiation so that tissue reactions are prevented and the risk of stochastic effects is limited to acceptable levels. For assessing doses from radiation exposures special *dosimetric quantities* have been developed by ICRP and by the International Commission on Radiation Units and Measurements (ICRU). The fundamental *protection quantities* adopted by ICRP are based on measures of the energy imparted to organs and tissues of the human body. These quantities allow quantification of the extent of exposure to ionising radiation from both whole and partial body irradiation from external radiation sources and from intakes of radionuclides. The estimated doses can then be compared with recommended dose limits for people who are occupationally exposed and for members of the public.

(B 5) This scheme of quantities was first adopted by the Commission in its Recommendations in *Publication 26* (ICRP, 1977). The quantities were modified in the 1990 Recommendations in *Publication 60* (ICRP, 1991b) and have been developed further in the 2007 Recommendations.

(B 6) For demonstrating compliance with dose limits, it is useful to have a single protection quantity specifying the 'amount' of whole or partial body exposure which is quantitatively related to the probability of an effect for all types of radiations, regardless of whether the radiation is incident on the body or emitted by radionuclides within it. Achieving this ideal is complicated by variations in the response of organs and tissues to radiations of different quality and by the varying radiosensitivity of the organs and tissues of the body. These effects generally influence the radiation response of all members of the population in a similar way. Therefore they were taken into account in the protection quantities recommended in *Publication 26* using quality factors and tissue weighting factors and in *Publication 60* using radiation and tissue weighting factors. Individually related factors including sex, age and individual sensitivity will also influence the risk but such biological effects are not taken into account in the definition of the protection quantities, which are applied for all members of the population.

(B 7) In *Publication 26* the different qualities of ionising radiation were considered with the quantity dose equivalent. The dose equivalent, H, was defined by

$$H = DQN \tag{B.1.1}$$

where D is the absorbed dose at a point in the specified tissue and Q is the *quality factor* for the specific radiation at this point. N was included to cover any other factor that could modify the risk from a radiation dose. However, in *Publication 26* no such modifying factors were specified. Hence the definition of H was later changed to

$$H = DQ \tag{B.1.2}$$

(see ICRP, 1991b, ICRU, 1993b).

(B 8) The Commission first introduced the protection quantity, *effective dose equivalent*, in *Publication 26* (ICRP, 1977) as proposed by Jacobi (1975). It was intended to be used for exposure limitation and risk management at low doses and was developed principally for use in relation to occupational exposure, although it has also been used more broadly for members of the public. The Commission updated this concept in *Publication 60* (ICRP, 1991b) with the quantity *effective dose*. The underlying principle was to use the *absorbed dose* as the fundamental physical quantity, to average it over specified organs and tissues and then to apply suitably chosen weighting factors to take account of differences in biological effectiveness of different radiations and the differences in radiation sensitivities of organs and tissues to stochastic health effects.

(B 9) The development of effective dose equivalent and subsequently effective dose has made a very significant contribution to radiological protection as it has enabled doses to be summed from whole and partial body exposure from external radiation and from intakes of radionuclides.

(B 10) Effective dose, as defined in *Publication 60*, has been implemented into legislation and regulations in many countries worldwide. It has been shown to provide a practicable approach to the management and limitation of radiation risk in relation to both occupational exposures and exposures of the general public. The general acceptance of effective dose as well as the demonstration of its practicability are

important reasons for maintaining it as the central quantity in radiological protection.

(B 11) Effective dose cannot be measured directly in the body. The protection system therefore includes *operational quantities* that can be measured (Fig. B.1) and used to assess effective dose. ICRU has developed a set of operational dose quantities for exposure to external radiation which were evaluated by a joint Task Group of ICRP and ICRU (*Publication 74*, ICRP, 1996b). The analysis in *Publication 74* indicated that the operational dose quantities recommended by ICRU generally achieve the objective of providing 'measurable quantities that adequately represent the protection quantities'. For internal exposures following intakes of radionuclides, activity quantities in combination with dose coefficients developed by ICRP are also used as operational quantities.

(B 12) There are a number of aspects to the dosimetry system given in *Publication 60* that needed to be addressed and further clarified. This Annex considers the dosimetric quantities developed by ICRP for radiological protection purposes and provides a detailed description of the Commission's dosimetry system adopted in these Recommendations. The health effects resulting from exposures to ionising radiation are briefly summarised in Section B.2, and their place in setting and applying protection standards are described. The basis for the development of the tissue weighting factors, w_T, is summarised although this is considered in more detail in Annex A. Section B.3 considers the development of the dosimetric quantities and those adopted in these Recommendations. It also examines tissue and radiation weighting factors in more detail, with emphasis on the latter. Section B.4 describes the

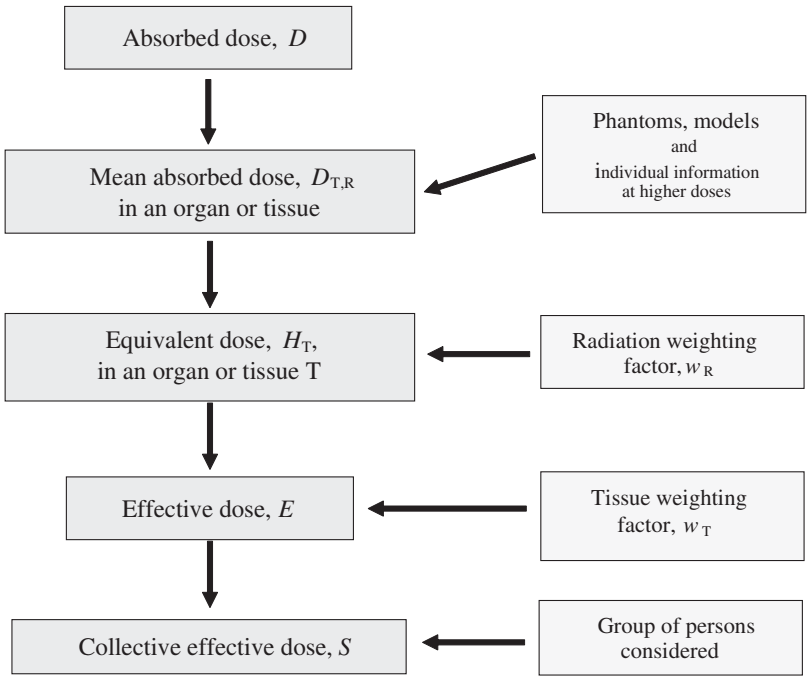

Fig. B.1. System of dose quantities for use in radiological protection.

operational quantities developed in conjunction with ICRU. The practical application of these dosimetric quantities in radiological protection, together with a discussion of situations in which the use of effective dose is, or is not, appropriate is covered in Section B.5. Finally, Section B.6 examines uncertainties and judgements that may need to be considered in using these quantities.

B.1.1. References, Section B.1

ICRP, 1977. Recommendations of the International Commission on Radiological Protection. ICRP Publication 26. Ann. ICRP 1(3).

ICRP, 1991b. 1990 Recommendations of the ICRP. ICRP Publication 60. Ann. ICRP 21 (1–3).

ICRP, 1996b. Conversion coefficients for use in radiological protection against external radiation. ICRP Publication 74. Ann. ICRP 26 (3/4).

ICRU, 1993b. Quantities and units in radiation protection dosimetry. ICRU Report 51. ICRU Publications: Bethesda, MD.

Jacobi, W., 1975. The concept of effective dose – A proposal for the combination of organ doses. Radiat. Environ. Biophys. 12, 101–109.

B.2. Health effects

(B 13) Radiological protection in the low dose range is primarily concerned with protection against radiation-induced cancer and heritable diseases. These diseases are termed stochastic effects, as they are probabilistic in nature. It is assumed that any exposure is capable of causing an effect, with no threshold (Annex A). As a consequence it is not possible to prevent stochastic risks and dose limits are set to limit their occurrence and thus to prevent unacceptable levels of risk. As indicated above, ICRP has developed the quantity effective dose to allow doses from external and internal exposure to be assessed on a common basis by using the above mentioned weighting factors.

(B 14) At exposures giving an absorbed dose above about 0.5–1 Gy (for low-LET radiation; LET: linear energy transfer, see Section B.3.5.1), associated mainly with accident situations, tissue reactions may occur if exposures exceed threshold doses for such health effects (Annex A). These thresholds vary with dose rate and with radiation quality, and the extent as well as the severity of the effect increases with increasing dose and dose rate. Tissue reactions must be considered separately from stochastic effects and cannot be addressed within the framework of effective dose and its parameters, w_R and w_T.

B.2.1. Stochastic effects

(B 15) Exposure to ionising radiation, even at low doses, may cause damage to the nuclear (genetic) material in cells that may result in the development of radiation-induced cancer many years later, heritable disease in future generations and some developmental effects under certain conditions (ICRP, 2003a). The induction of cancer by low-LET radiation has been firmly demonstrated in the dose range of about 100 mGy and higher, and it was concluded by UNSCEAR that 'studies on DNA repair and the cellular/molecular processes of radiation tumorigenesis provide no good reason to assume that there will be a low-dose threshold for the induction of tumours in general' (UNSCEAR, 2000). Radiation-induced heritable disease has not been demonstrated in human populations but there is substantial evidence from animal studies of heritable damage to germ cells (ova and spermatozoa as well as their precursor cells). For both radiation-induced cancer and heritable disease it is the probability of the occurrence of the effect, not its severity, that depends upon the dose. The general assumption for radiological protection is that the risk of these stochastic effects increases in the low dose range linearly with dose, with no threshold (LNT model) (UNSCEAR, 2000, Streffer et al., 2004, Annex A).

(B 16) Annex A gives detailed information on the risk of radiation-induced cancer in organs and tissues of the body and on dose–response relationships as well as of heritable disease. It is notable that there are significant differences in sensitivity to cancer induction among the organs and tissues of the body. Thus, for example, the thyroid in children, the female breast and the bone marrow have a relatively high sensitivity for the induction of solid cancer and leukaemia whereas the muscle and connective tissue have a relatively low sensitivity.

(B 17) Annex A also gives information on other stochastic effects that may occur following radiation exposure. This includes damage to the vascular tissue of the circulatory system of blood. At present, however, insufficient data are available to determine any dose–response relationships in the dose range below about 0.5 to 1 Gy or to use them as a basis for setting dose limits.

(B 18) A central position of the Recommendations in *Publication 26* (ICRP, 1977) was that the overall risk of stochastic effects at exposures corresponding to the Commission's dose limits are approximately equal, regardless of the manner of irradiation – whether the body is uniformly or heterogeneously irradiated from external radiation or from intakes of radionuclides if the sensitivity to the different types of radiation are correctly considered. This principle resulted in the inclusion of two types of weighting factors in the definition of effective dose equivalent for use in radiological protection.

(B 19) The quality factors, first used in *Publication 6* (ICRP, 1964), allowed for the relative effectiveness of different radiations in causing biological effects and could be thought of as the factor representing the relative biological effectiveness (RBE) of the radiation. Experimental measurements of RBE in cellular studies in vitro, and in animal studies, show that high-LET radiations, including neutrons and alpha particles, cause more damage per unit of absorbed dose than low-LET radiations. The weighting factors, w_T (later termed tissue weighting factors in *Publication 60*) accounted for the varying radiation sensitivity of tissues to the induction of stochastic effects.

(B 20) The w_T values recommended by the Commission in *Publication 26* were based on the risk of fatal cancer and of serious heritable disease in the first two generations (Table B.1). *Publication 60* (ICRP, 1991b) developed this concept further with an extended set of tissue weighting factors based upon more information on stochastic radiation effects on tissues and a broader concept of radiation detriment. In addition to assessing the risk of radiation-induced fatal cancer and heritable disease in all future generations it also took into account the severity of the disease and the years of life lost in determining total radiation detriment. Radiation detriment then provided the basis for setting revised values of tissue weighting factors, w_T, in *Publication 60* (Table 1). In addition, radiation weighting factors, w_R, replaced quality factors, Q, in the definition of the protection quantities. The assumption was made that, for protection purposes, the weighting factors are independent of dose and dose rate in the low dose range. Values of w_R are taken to be independent of the organ or tissue irradiated and w_T values to be independent of the type and energy of radiation.

(B 21) In the 2007 Recommendations, the Commission has further developed the concept of tissue weighting factors, and now bases values of w_T to a large extent on the incidence of radiation-induced cancer rather than on mortality as well as on the risk of heritable disease over the first two generations (Annex A). This is considered to be a more appropriate basis for the assessment of radiation detriment. The risk of cancer is again adjusted for severity and for years of life lost. The tissue weighting factors given in the 2007 Recommendations are presented in Table B.2 and discussed further in Section B.3.5, paragraphs B 132 – B 145.

Table B.1. ICRP Recommendations for tissue weighting factors in *Publication 26* (1977) and *Publication 60* (1991b).

Tissue	Tissue weighting factor, w_T	
	1977 *Publication 26*	1991 *Publication 60*[2,3]
Bone surfaces	0.03	0.01
Bladder		0.05
Breast	0.15	0.05
Colon		0.12
Gonads	0.25	0.20
Liver		0.05
Lungs	0.12	0.12
Oesophagus		0.05
Red bone marrow	0.12	0.12
Skin		0.01
Stomach		0.12
Thyroid	0.03	0.05
Remainder	0.30[1]	0.05
TOTAL	1.0	1.0

[1] The five most highly irradiated other organs and tissues are included in remainder, each with a $w_T = 0.06$.

[2] 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.

[3] Further footnotes in *Publication 60*. Table 5.2, page 68.

Table B.2. Tissue weighting factors, w_T, in the 2007 Recommendations.

Organ/Tissue	Number of tissues	w_T	Total Contribution
Lung, stomach, colon, bone marrow, breast, remainder	6	0.12	0.72
Gonads	1	0.08	0.08
Thyroid, oesophagus, bladder, liver	4	0.04	0.16
Bone surface, skin, brain, salivary glands	4	0.01	0.04

1. The w_T for gonads is applied to the mean of the doses to testes and ovaries.
2. The dose to the colon is taken to be the mass-weighted mean of ULI and LLI doses, as in the *Publication 60* formulation.
The specified remainder tissues (14 in total, 13 in each sex) are: adrenals, extrathoracic tissue (ET), gall bladder, heart, kidneys, lymphatic nodes, muscle, oral mucosa, pancreas, prostate (♂), small intestine (SI), spleen, thymus, uterus/cervix (♀).

B.2.2. Tissue reactions (deterministic effects)

(B 22) At doses much higher than the dose limits recommended in the protection system, and especially in accident situations, radiation exposures may cause *deterministic effects* (tissue reactions). These effects result from the impairment of the integrity and function of organs and tissues: clinically observable damage then occurs above a threshold dose, although the extent of any damage depends upon the absorbed dose and dose rate as well as radiation quality. The expression of injury varies from one tissue or organ to another depending upon cellular radiosensitivity, the function of differentiated cells, cellular composition, and cell renewal capacity. Loss of reproductive capacity of cells, the development of fibrotic processes and cell death play a central role in the pathogenesis of most tissue reactions. Some of the most sensitive tissues, with respect to early tissue reactions, are those with rapidly proliferating cell systems including haematopoietic tissue, the cells lining the gastrointestinal tract, the basal cell layer in the skin, and the male germ cells. Late tissue reactions may also depend in part on damage to blood vessels or connective tissue elements that are essential for the functioning of all organs and tissues as well as of the lens of the eye. Such damage can be expressed many months or even years after radiation exposure.

(B 23) High-LET radiations, such as neutrons and alpha particles, cause more damage per unit of absorbed dose than low-LET radiation. Values of RBE for tissue reactions were given in *Publication 58* (ICRP, 1989b). In general the RBE values were found to be lower for tissue reactions than those for stochastic effects at low doses and to vary with the tissue damage described.

(B 24) The radiation weighting factors, w_R, for high-LET radiation are derived for stochastic effects at low doses. The application of these w_R values to assess the exposure and damage at high doses, when compared with photon irradiation, would result in an over-estimate of the occurrence and severity of any tissue reaction. When assessing radiation exposure for determining the potential for tissue reactions, the mean absorbed dose to the organ or tissue, weighted by an appropriate value of RBE for the biological end point of concern, should therefore be used. These RBE values may differ for different biological endpoints and different tissues or organs. Guidance on appropriate values of the RBE can be obtained in *Publication 58* (ICRP 1989b), NCRP Report No. 104 (1990) and *Publication 92* (ICRP, 2003c).

(B 25) As a consequence, the quantities, equivalent dose and effective dose, with their unit with the special name sievert (Sv), should not be used in the quantification of radiation doses or in determining the need for any treatment in situations where tissue reactions are caused. In general, in such cases doses should be given in terms of absorbed dose in gray (Gy), and if high-LET radiations (e.g., neutrons or alpha particles) are involved, an RBE-weighted dose, RBE·D (Gy), may be used. The RBE value to be considered depends, however, not only on the type and energy of the particles involved but also may depend on dose and dose rate in the specific situation and on the tissue as well as organs. In such cases it is necessary to clearly state which RBE value has been applied.

B.2.3. References, Section B.2

ICRP, 1964. Recommendations of the International Commission on Radiological Protection. ICRP Publication 6. Pergamon Press, Oxford, UK.

ICRP, 1977. Recommendations of the International Commission on Radiological Protection. ICRP Publication 26. Ann. ICRP 1 (3).

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ICRP, 2003a. Biological effects after prenatal irradiation (embryo and fetus). ICRP Publication 90. Ann. ICRP 33 (1/2).

ICRP, 2003c. Relative biological effectiveness (RBE), quality factor (Q), and radiation weighting factor (w_R). ICRP Publication 92. Ann. ICRP 33 (4).

NCRP, 1990. The Relative Biological Effectiveness of Radiations of Different Quality. NCRP Report No. 104. National Council on Radiation Protection and Measurements, Bethesda, MD.

Streffer, C., Bolt, H., Follesdal, D., et al., 2004. Low Dose Exposures in the Environment – Dose-Effect Relations and Risk Evaluation. Springer, Berlin-Heidelberg-New York-Hong Kong-London-Milan-Paris-Tokyo.

UNSCEAR, 2000. Sources and Effects of Ionizing Radiation. Report of the United Nations Scientific Committee on the Effects of Atomic Radiation. Volume II: Effects. United Nations, New York.

B.3. Quantities in radiological protection

(B 26) Radiological protection has the general aim of protecting humans and the environment from harm caused by ionising radiation after external as well as internal exposures. This requires a quantitative description of the radiation fields external to and internal within the human body. Similar considerations apply to protection of other biological organisms. This latter aspect will not be considered further in this Annex.

(B 27) While radiation fields external to the body can be well described by physical quantities such as particle fluence or air kerma free in air, the internal radiation fields following the intake of radionuclides depend upon their biokinetics and on anatomical and physiological parameters of the human body.

(B 28) Fluence is a quantity used to describe external radiation fields. It is not, however, practicable for general use in radiological protection and the definition of limits. Fluence always needs the additional specification of the particle and particle energy as well as direction distributions. Its correlation with detriment is complex.

(B 29) As mentioned in the Introduction, in radiological protection practice, a single quantity has been developed for specifying the 'amount' of exposure which is quantitatively related to the probability of stochastic effects in human bodies for all types of radiations regardless of which type of ionising radiation is considered or whether the radiation is incident on the body or emitted by radionuclides within the body. It needs to be stressed that this is a practical protection quantity that involves parameter values that are based on judgement.

(B 30) The initial step in the interaction of ionising radiation with biological material is energy transfer that leads to ionisations. It might appear reasonable to use the amount of absorbed energy per unit of mass (absorbed dose) as the only term for quantifying the radiation exposure in radiological protection in order to estimate the risk caused by a given exposure. This is not sufficient, however, as radiation effects depend not only on the absorbed dose but also on the type of radiation, on the distribution of energy absorption in time and space within the human body, and on the radiosensitivity of the exposed tissues or organs.

(B 31) The basic procedure of dose assessment adopted by the Commission is to use *absorbed dose* as the fundamental physical quantity, to average it over specified organs and tissues, and to apply suitably chosen weighting factors to take account of differences in biological effectiveness of different radiations and of differences in sensitivities of organs and tissues to stochastic health effects. *Effective dose* is therefore a quantity based on the internal and external radiation fields and the primary physical interactions in human tissues as well as on judgements about the biological reactions resulting in stochastic health effects.

B.3.1. Fluence and kerma

(B 32) A radiation field of a specific type is fully described by the number N of particles, their distributions in energy and direction, and their spatial and temporal

distribution. This requires the definition of scalar and vector quantities. Definitions of radiation field quantities are given in detail in ICRU Report 60 (1998). While vector quantities providing information on direction distributions are mainly applied in radiation transport theory and calculations, scalar quantities such as particle fluence or kerma are often used in dosimetry applications.

(B 33) Radiation field quantities are defined at any point in a radiation field. There are two classes of radiation field quantities referring either to the number of particles, such as fluence and fluence rate, or to the energy transported by them, such as energy fluence. Radiation fields may consist of various types of radiation, and those field quantities which are based on particle numbers are always related to a specific type. This is often expressed by adding the particle name to the quantity, e.g., neutron fluence.

(B 34) The quantity fluence is based on the concept of counting the number of particles incident or passing a small sphere.

(B 35) The *fluence*, Φ, is the quotient of $\mathrm{d}N$ by $\mathrm{d}a$, where $\mathrm{d}N$ is the number of particles incident upon a small sphere of cross-sectional area $\mathrm{d}a$, thus

$$\Phi = \frac{\mathrm{d}N}{\mathrm{d}a} \tag{B.3.1}$$

The fluence is independent of the direction distribution of the particles entering the sphere. In calculations, fluence is often alternatively expressed in terms of the length of trajectories of particles passing through a small volume $\mathrm{d}V$. The fluence, Φ, is then given by

$$\Phi = \frac{\mathrm{d}l}{\mathrm{d}V} \tag{B.3.2}$$

where $\mathrm{d}l$ is the sum of the lengths of trajectories through this volume $\mathrm{d}V$.

(B 36) In radiation fields the number of particles traversing a small sphere is always subject to random fluctuations. However, fluence – as well as related quantities – is defined as a non-stochastic quantity and hence has a single value at a given point and time with no inherent fluctuations. Its value should be considered as an expectation value.

(B 37) The transfer of energy from uncharged particles (indirectly ionising particles, e.g., photons or neutrons) to matter is performed by the liberation and slowing down of secondary charged particles in this matter. This led to the definition of the quantity kerma. The *kerma*, K, is the quotient of $\mathrm{d}E_{\mathrm{tr}}$ by $\mathrm{d}m$, where $\mathrm{d}E_{\mathrm{tr}}$ is the sum of the kinetic energies of all charged particles liberated by uncharged particles in a mass $\mathrm{d}m$ of material. It is given by:

$$K = \frac{\mathrm{d}E_{\mathrm{tr}}}{\mathrm{d}m} \tag{B.3.3}$$

(B 38) The SI unit of kerma is J kg^{-1} and its special name is gray (Gy). Kerma is a non-stochastic quantity in which $\mathrm{d}E_{\mathrm{tr}}$ is seen to be the expectation value of the sum of energies of liberated charged particles.

B.3.2. Absorbed dose

(B 39) In radiation biology, clinical radiology and radiological protection the absorbed dose, D, is the basic physical dose quantity. It is used for all types of ionising radiation and any irradiation geometry.

(B 40) Absorbed dose, D, is defined as the quotient of $\mathrm{d}\bar{\varepsilon}$, by $\mathrm{d}m$, where $\mathrm{d}\bar{\varepsilon}$ is the mean energy imparted to matter of mass $\mathrm{d}m$ by ionising radiation, that is

$$D = \frac{\mathrm{d}\bar{\varepsilon}}{\mathrm{d}m} \tag{B.3.4}$$

The SI unit is J kg^{-1} and its special name is gray (Gy). While the value of kerma depends only on interactions in the material of mass element $\mathrm{d}m$, the value of absorbed dose also depends on the secondary charged particles which are released in the surroundings of the mass element $\mathrm{d}m$ and which enter this element. Absorbed dose is derived from the mean value of the stochastic quantity of energy imparted, ε, and does not reflect the random fluctuations of the interaction events in tissue. While it is defined at any point in matter, its value is obtained as an average over $\mathrm{d}m$ and hence over many atoms or molecules of matter.

(B 41) The definition of absorbed dose has the scientific rigour required for a basic physical quantity. It implicitly takes account of the radiation field as well as of all of its interactions with matter inside and outside the specified volume. It does not, however, take account of the atomic structure of matter and the stochastic nature of the interactions. Absorbed dose is a measurable quantity and primary standards exist to allow its determination by measurement.

(B 42) A particular feature of ionising radiations is their discontinuous interaction with matter and the related stochastic (probabilistic) nature of energy deposition. Energy is transferred to the tissue by charged particles in interactions with individual atoms and molecules. The human body is made up of organs and tissues, which consist of cells, sub-cellular structures and macromolecules such as DNA. Absorbed dose is defined as the mean of the stochastic distribution of energy deposited in a volume element. The fluctuations of energy deposited in individual cells and sub-cellular structures and the microscopic tracks of charged particles are the subject of *microdosimetry*.

(B 43) The magnitude of the fluctuations of energy deposited in different small tissue volumes depends on the value of the absorbed dose and on the size of the volume considered. At a given dose, these fluctuations increase with increasing ionisation density in charged particle tracks (characterised by the linear energy transfer, LET, see Section B.3.5, paragraphs B 73 – B 131) of the radiation. At the low absorbed doses generally of concern in radiological protection, the statistical fluctuation of energy deposited can be substantial between individual cells and within a single hit cell. This is the case particularly for densely ionising radiations (high-LET radiation) such as alpha particles and secondary charged particles from neutron interactions.

(B 44) At a given absorbed dose, the actual value of energy imparted, ε, in a small tissue volume, e.g., in a single cell, is given by the sum of energies deposited in that

volume by all individual events. In any volume, fluctuations of ε are caused by variation in the number of events and by variation in the energy deposited in each event. For low-LET radiations (e.g., photons and electrons) the energy imparted in each event (hit) is relatively low, and at low doses more cells experience energy deposition events than in the case of exposure to high-LET radiation at the same dose. As a consequence, the fluctuation in the energy deposited among cells is smaller for low-LET than for high-LET radiation.

(B 45) (B 45) For low mean doses of high-LET radiation (e.g., charged particles from neutron interactions or alpha particles), the frequency of hits in most cells is zero, in a few it is one and exceptionally it can be more than one. The value of energy deposited in most individual cells is then zero but in the 'hit' cells it can exceed the mean value (i.e., absorbed dose) in the tissue by orders of magnitude. Even among the hit cells the distribution of these events is very heterogeneous. These large differences in the energy deposition distribution in microscopic regions for different types (and energies) of radiation have been correlated to observed differences in biological effectiveness or radiation quality (Goodhead, 1994). Further information is given, for example, in the UNSCEAR 1993 and 2000 reports (UNSCEAR, 1993; 2000).

(B 46) Auger electrons emitted from radionuclides in the body need special attention if such emitters are in or near to the DNA. Often a radionuclide, which decays via internal conversion, emits many Auger electrons. These emissions can result in a very localised energy deposition, and the biological effect may, therefore, be similar to that of a high-LET radiation. This has already been considered in *Publication 60* (ICRP, 1991b); see Section B.3.5., paragraphs B 86 – B 99).

(B 47) In the definition of radiological protection quantities no attempts are made to specify the stochastic distribution of physical processes at a microscopic level. Instead of explicitly considering such distribution functions, a pragmatic and empirical approach has been adopted to take account of radiation quality differences. Radiation weighting factors take into account the effects due to differences in distribution of energy deposited in microscopic regions through judgements based on the results of radiobiological experiments. This is discussed in more detail in Section B.3.5, paragraphs B 73 – B 131.

B.3.3. Averaging of absorbed dose

(B 48) As described above, the quantity absorbed dose is defined to give a specific value at any point in matter. However, in practical applications absorbed doses are often averaged over larger tissue volumes. It is thus assumed that, for low doses, the mean value of absorbed dose in a specific organ or tissue can be correlated with radiation detriment from stochastic effects in all parts of that organ or tissue with sufficient accuracy for the purposes of radiological protection.

(B 49) The mean absorbed dose in the region of an organ or tissue T, $\overline{D}_{\mathrm{T}}$, is defined by

$$\overline{D}_{\mathrm{T}} = \frac{\int_{\mathrm{T}} D(x,y,z)\rho(x,y,z)\mathrm{d}V}{\int_{\mathrm{T}} \rho(x,y,z)\mathrm{d}V} \tag{B.3.5}$$

where V is the volume of the tissue region T, D the absorbed dose at a point (x,y,z) in that region and ρ the mass density at this point. In practice, the mean absorbed dose in an organ or tissue T, $\overline{D}_T$, is usually written D_T.

(B 50) The averaging of absorbed doses in different tissues or organs of the human body and their weighted sum are the basis for the definition of the protection quantities which are used for limiting stochastic effects at low doses. This approach is based upon the assumption of a linear-non-threshold, dose–response relationship (LNT model) and allows the addition of doses from external and internal exposure. This concept is considered to be an acceptable approximation for radiological protection purposes and was first adopted by the Commission in *Publication 9* (ICRP, 1966). It was subsequently reaffirmed in later Recommendations including *Publications 26* and *60* (ICRP, 1977, 1991b) and is further supported in Annex A of the present Recommendations. The definitions of all the protection quantities rely on this fundamental assumption of the LNT model in the low-dose region.

(B 51) The averaging of absorbed dose is carried out over the volume of a specified organ (e.g., liver) or tissue (e.g., muscle) or a region of a tissue (e.g., endosteal surfaces of the skeleton, skin). The extent to which the mean absorbed dose (Eqn. B.3.5) is representative of the local absorbed dose throughout organs, tissues, or tissue regions depends on a number of factors. For external radiation exposure, this depends mainly on the homogeneity of the exposure and on the penetrability or range of the incident radiation in the body. For penetrating radiation (photons, neutrons) the absorbed dose distribution within most organs may be sufficiently homogeneous, and thus the mean absorbed dose is a suitable measure of the dose throughout the organ or tissue.

(B 52) The absorbed dose distribution within the specified organ or tissue may be very heterogeneous for radiation with low penetration or limited range (low-energy photons, charged particles) as well as for widely distributed tissues and organs (e.g., active [red] bone marrow or lymphatic nodes) in non-homogeneous radiation fields. In cases of extreme partial body exposure tissue damage may occur even if the mean tissue or organ dose or the effective dose is below the dose limit. For example, this may occur in the case of exposure of the skin to low-penetrating radiation. A special limit is applied to local skin dose to avoid tissue reactions (see Section B.5.5).

(B 53) For radiations emitted by radionuclides retained within body organs or tissues, so-called internal emitters, the absorbed dose distribution in the organs depends on the distribution of the radionuclides and the penetration and range of the radiations emitted. It also depends on the structure of the organ or tissue (e.g., 'walled' organs such as the urinary bladder, airways of the respiratory tract, and the highly heterogeneous mixture of bone mineral, inactive and active bone marrow). The absorbed dose distribution for radionuclides emitting alpha particles, soft beta particles, low-energy photons, or Auger electrons may be highly heterogeneous.

(B 54) This heterogeneity occurs in particular in the case of radionuclides deposited in the respiratory tract (e.g., radon decay products on the bronchial mucosa), passing through the alimentary tract, or deposited on bone surfaces (e.g., plutonium and related elements) or on skin. In such situations the mean absorbed dose averaged over the entire organ or tissue is not considered to be an appropriate dose quantity

for estimating the probability of stochastic damage. The Commission has addressed this issue and has developed dosimetric models for the respiratory system (ICRP, 1994a), the alimentary tract (ICRP, 2006c), and the skeleton (ICRP, 1979) that take account of the distribution of radionuclides and the location of sensitive cells in the calculation of mean absorbed dose to these tissues. In these cases the dose determined in the identified tissue region considered to be the target for the development of radiation-induced cancer is treated as the average dose.

(B 55) As discussed above, the heterogeneous distribution of energy deposition is of concern with respect to the averaging procedure in the low dose range and especially with radionuclides which are heterogeneously distributed in an organ or tissues and which emit particles with short ranges. However, no established approaches are presently available for use in radiological protection practice which take into account microdosimetric considerations or the three-dimensional track structure in tissues and the related energy deposition. Considering the stochastic nature of the induction of cancer and of heritable disease and the assumptions that one single track of ionising particles may be sufficient for the initiation process, it appears that the present approach is pragmatic for radiological protection purposes with a justified scientific basis. The uncertainty associated with such an approach should be kept in mind.

(B 56) In the case of deposition of 'hot particles' in the lung or other tissues, (e.g., aerosols deposited in the lung with low solubility and high specific activity) the Commission continues to consider that the associated hazard of malignant disease induction is similar to or lower than that from homogenous distribution of equal activity in the lungs (Lafuma et al., 1974, ICRP, 1980, Charles et al., 2003).

(B 57) Dose distributions that are highly heterogeneous can result from the incorporation of DNA precursors labelled with tritium (e.g., thymidine, deoxycytidine) or Auger emitters incorporated into DNA in cell nuclei. Owing to the specific location of the emitter and the very short range of tritium beta radiation and Auger electrons, cell nuclei can be exposed to doses which are much higher than the mean dose to the cell or the organ or tissue. Tritiated DNA precursors may therefore be more radiotoxic than tritiated compounds, such as tritiated water, which are not specifically located in the cell nucleus (Streffer et al., 1978). In such cases, risks might be estimated on the basis of dose to cell nuclei. Another approach is to take account of experimental mammalian data on the relative biological effectiveness of heterogeneously distributed radionuclides (e.g., tritiated thymidine) compared with the same nuclides distributed more uniformly (e.g., tritiated water) (Streffer et al., 1978) or with external irradiation. The Commission is not proposing a specific scheme for the treatment of doses and risks from such localised nuclear irradiation (see Section B.3.5, paragraphs B 86 – B 99).

B.3.4. Equivalent dose and effective dose

(B 58) The protection quantities are used to specify dose limits to ensure that the occurrence of stochastic health effects is kept below unacceptable levels and tissue reactions are avoided. The system of protection quantities is shown in Figs B.1

and B.2. Their definition is based on the mean absorbed dose, $D_{T,R}$, in the volume of a specified organ or tissue, T, due to the radiation of type R – or in another specified target region of the body – (see Eqn. B.3.5). The radiation R is given by the type and energy of radiation either incident on the body or emitted by radionuclides residing within the body. The protection quantity *equivalent dose* in an organ or tissue, H_T, is then defined by

$$H_T = \sum_R w_R D_{T,R} \tag{B.3.6}$$

where w_R is the radiation weighting factor for radiation R (see Section B.3.5, paragraphs B 73 – B 131, and Table B.4). The sum is performed over all types of radiations involved. The unit of equivalent dose is J kg^{-1} and has the special name sievert (Sv).

(B 59) Values of w_R are mainly based upon experimental data of the relative biological effectiveness (RBE) for various types of radiations at low doses (see Section B.3.5, paragraphs B 73 – B 131). A set of w_R values for various radiations was given in *Publication 60* (ICRP, 1991b); cf. Table B.3. The general concept of these radiation weighting factors remains unchanged. Some modifications to the values of w_R adopted in *Publication 60* (ICRP, 1991b) are provided and discussed in Section B.3.5, paragraphs B 73 – B 131 (see Table B.4).

(B 60) The effective dose, E, introduced in *Publication 60* was defined as:

$$E = \sum_T w_T \sum_R w_R D_{T,R} = \sum_T w_T H_T \tag{B.3.7}$$

where w_T is the tissue weighting factor for tissue T (see Section B.3.5, paragraphs B 132 – B 145, and Table B.2) and $\Sigma w_T = 1$. The sum is performed over all organs and tissues of the human body considered in the definition of E and for which w_T

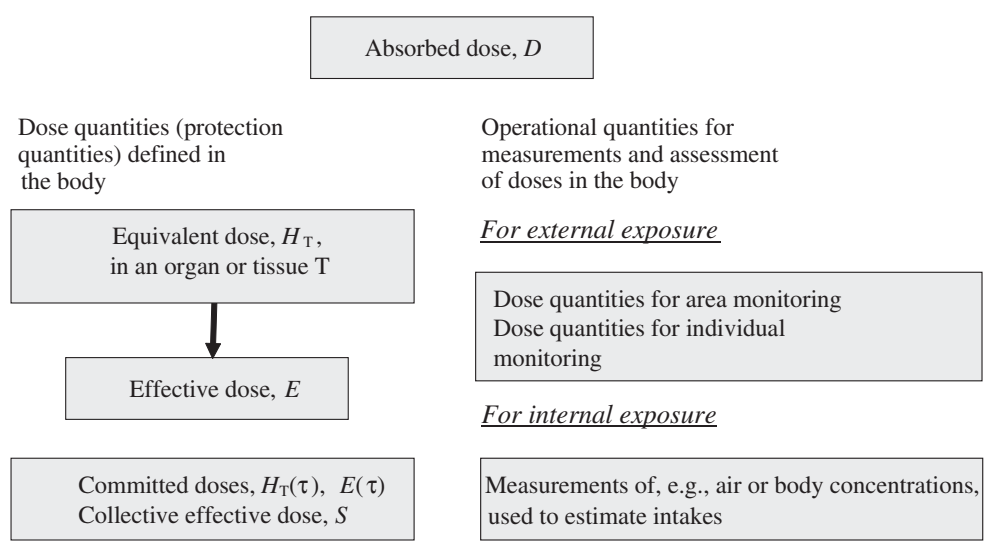

Fig. B.2. System of protection and operational quantities for use in radiological protection.

Table B.3. Radiation weighting factors[1] (ICRP 1991b).

Type and energy range[2]	Radiation weighting factors, w_R
Photons, all energies	1
Electrons and muons, all energies[3]	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
Protons, other than recoil protons, energy>2 MeV	5
Alpha particles, fission fragments, heavy nuclei	20

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

[2] The choice of values for other radiations is discussed in paragraph A14 in ICRP (1991b).

[3] Excluding Auger electrons emitted from nuclei bound to DNA (see paragraph A13 in ICRP 1991b).

Table B.4. Radiation weighting factors[1] in the 2007 Recommendations.

Radiation type	Radiation weighting factor, w_R
Photons	1
Electrons and muons	1
Protons and charged pions	2
Alpha particles, fission fragments, heavy ions	20
Neutrons	A continuous curve as a function of neutron energy (see Fig. B.4 and Eqn. B.3.16)

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

values are given in Table B.2. The unit of effective dose is J kg^{-1} with the special name sievert (Sv). The same unit used for equivalent dose and effective dose is also used for the operational dose quantities (see Section B.4.1, paragraphs B 159 – B 170). Care must be taken in ensuring that the quantities being used are clearly stated.

(B 61) While absorbed dose in a specified tissue is a physical quantity, the equivalent dose and effective dose include weighting factors which are based on radio-

biological and epidemiological findings. These weighting factors are selected for application in radiological protection by judgement and include acceptable simplifications (see Section B.3.5). Therefore the definition and the value of effective dose are not based on physical properties only. For example, the tissue weighting factors, w_T, are based on epidemiological studies of cancer induction as well as on experimental genetic data after radiation exposure, and on judgements. Furthermore they represent mean values for humans, averaged over both sexes and all ages.

(B 62) The definition of the effective dose is based on the mean doses in organs or tissues of the human body. The quantity provides a value which takes account of the given exposure situation but not, however, the characteristics of a specific individual. For internal exposure of humans, for example, the organ doses are often determined by assessing the intake of incorporated radionuclides and applying dose coefficients which relate the intake of activity to the corresponding mean organ doses. These coefficients are calculated using general biokinetic models and reference phantoms. Hence this means that, for a given incorporated activity of a specific radionuclide, the corresponding effective dose is estimated. This approximation of the dose is judged to be acceptable for radiological protection purposes.

(B 63) The use of effective dose allows exposures in very different situations (e.g., internal and external exposure by different types of radiation) to be combined in a single value. As a consequence, the primary exposure limits can be expressed in terms of a single quantity. This facilitates the system of dose limitation and record-keeping.

(B 64) In order to provide a practicable approach for the assessment of effective dose, coefficients relating it to physical quantities, e.g., particle fluence or air kerma for external exposure or activity intake for internal exposure, are calculated for standard conditions (e.g., mono-energetic radiations, standard irradiation geometries, selected chemical compounds labelled with radionuclides, models for the transfer of radionuclides in the body) in anthropomorphic phantoms with clearly defined geometries. These phantoms include most organs and tissues in the body, especially those listed in the table of the tissue weighting factors (Table B.2).

(B 65) In the Commission's publications since *Publication 26* (ICRP, 1977), the calculation of effective dose (or effective dose equivalent) from external radiation and from radionuclides incorporated into the body has been based on the equivalent dose to organs and tissues derived from sex-invariant anatomical and biokinetic models weighted by the sex-averaged tissue weighting factors (ICRP, 1994b). The scheme of calculation has now changed further with the development of male and female phantoms (Section B.5.2).

(B 66) For the calculation of conversion coefficients relating effective dose to radiation field quantities (for external radiation exposure situations), e.g., air kerma or particle fluence, ICRP departed from this approach in *Publication 74* (ICRP, 1996b), as sex-specific anatomical models were used. The following formula with sex-specific equivalent doses in organs and tissues for the calculation of effective dose was applied in *Publication 74*:

$$E = w_{\text{breast}} H_{\text{breast,female}} + \sum_{\text{T} \neq \text{breast}} w_{\text{T}} \left[\frac{H_{\text{T,male}} + H_{\text{T,female}}}{2} \right] \tag{B.3.8}$$

where the summation includes the dose to the gonads (ovaries in the female, testes in the male). The different procedures (using sex specific or hermaphrodite models), however, yield values of effective dose which are not very different and are sufficiently precise for applications in radiological protection.

(B 67) The Commission has defined adult male and female computational phantoms (see Section B.5.2). These models will be used for calculations of dose conversion coefficients for external and of dose coefficients for internal radiation exposures. The use of sex-specific computational models allows the calculation of the male and female organ doses from which the averaged equivalent dose is calculated and used for the calculation of the effective dose. This can be done for the doses to the breast and the gonads in the same way as with the other organs and tissues.

(B 68) The procedure adopted to determine the tissue weighting factors is to first assess the risks of radiation-induced stochastic effects in males and females separately, then calculate sex specific radiation detriment and from these values give sex-averaged w_{T} values (Annex A). The sex-averaged w_{T} values, as well as the sex-averaged organ and tissue doses, are then used for the calculation of the effective dose (Fig. B.3). Under these conditions it is not reasonable to treat the contribution of the male and female doses separately in the calculation of effective dose. All tissues can be treated according to Eqn. (B.3.9).

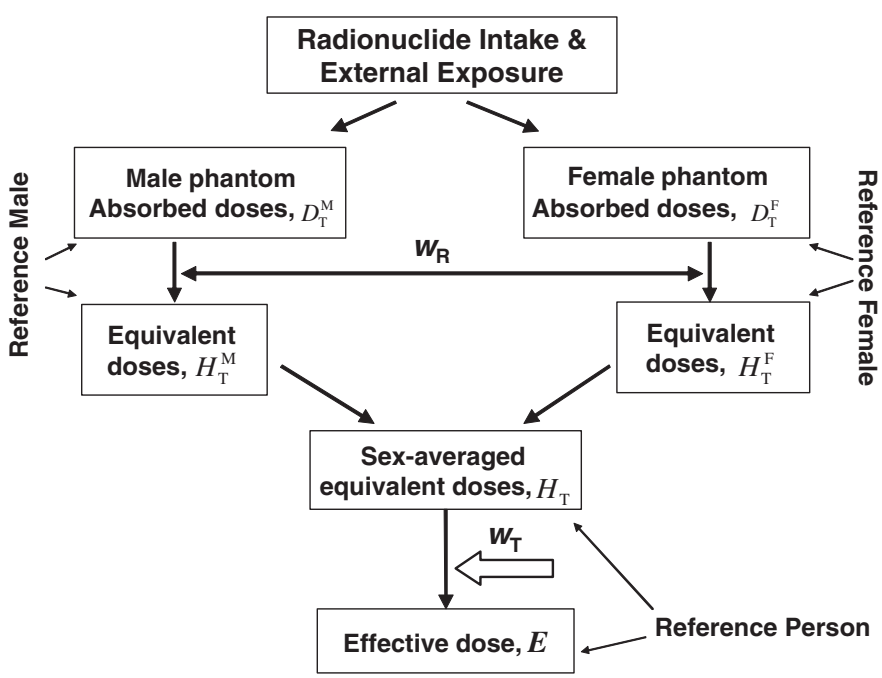

Fig. B.3. Sex – averaging in the calculation of effective dose (E).

(B 69) The effective dose E is then computed from the equivalent dose assessed for organ or tissue T of the Reference Male, H_T^M, and Reference Female, H_T^F, including the remainder tissues (see Section B.3.5, paragraphs B 132 – B 145, and Eqn. B.3.17), as in the following equation:

$$E = \sum w_T \left[\frac{H_T^M + H_T^F}{2} \right] \tag{B.3.9}$$

This equation together with the new female and male reference phantoms (see Section B.5.2 and Figs B.2 and B.3) will be used for the future calculation of conversion coefficients and dose coefficients.

(B 70) For practical use, the calculation of organ doses or conversion coefficients in cases of external exposures and of dose coefficients (dose per intake, Sv Bq^{-1}) in cases of internal exposures is not based on data from individual persons but on reference values for the human body given in *Publication 89* (ICRP, 2002). In addition, age-specific data, e.g., food consumption etc., may need to be considered for assessment of exposures for members of the public. The use of reference values and the averaging over both sexes in the calculation of effective dose indicates that the reference dose coefficients are not aimed at providing a dose for a specific individual but for a Reference Person. Reference computational phantoms for children of various ages will also be developed for use in the calculation of dose coefficients for members of the public.

B.3.5. Weighting factors

(B 71) As noted previously, mean absorbed dose is insufficient, on its own, for assessing detriment caused by ionising radiation exposure. In order to establish a correlation between dose quantities applied in radiological protection and stochastic effects (radiation-induced cancer and heritable diseases), two types of weighting factors have been introduced, a radiation weighting factor, w_R, and a tissue weighting factor, w_T.

(B 72) The weighting factors are intended to take account of different types of radiation and of stochastic effects in different organs and tissues of the body. They are therefore broadly based on a wide range of experimental data and epidemiological studies and they are taken to be independent of age and sex. In *Publication 60* (ICRP, 1991b) the Commission selected a general set of these weighting factors that were considered to be appropriate for the needs of radiological protection (Tables 1 and 3). This procedure is maintained in these 2007 Recommendations.

Radiation weighting factors

(B 73) The method of radiation weighting in the definition of radiological protection quantities has been used since the early 1960s. Before 1991, this was achieved by applying the quality factor concept using a specified $Q(L)$ function (ICRP, 1977). In *Publication 60* (ICRP, 1991b), radiation weighting was defined differently for the

protection quantities and for the operational dose quantities used in measurements of external exposure.

(B 74) Radiation weighting is based mainly on an evaluation of the relative biological effectiveness (RBE) of the different radiations with respect to stochastic effects. The RBE is used in radiobiology for characterising the different biological effectiveness of radiations. RBE values are given as the ratio of the absorbed doses of two types of radiation producing the same specified biological effect in identical irradiation conditions (dose value of a reference radiation divided by the corresponding dose value of the considered radiation which causes the same level of effect).

(B 75) RBE values for a specific radiation depend upon the conditions of exposure including the biological effect investigated, the tissue or cell type involved, the dose and the dose rate, and the dose fractionation scheme; therefore, for a given type and energy of radiation, there will be a range of RBE values. The RBEs reach maximum values (RBE_M) at low doses and low dose rates. RBE_M is therefore of particular interest for defining radiation weighting factors for use in radiological protection. The weighting factors are taken to be independent of the dose and dose rate in the low-dose region.

(B 76) The concepts of the quality factor and radiation weighting are based on differences in the biological effectiveness of the various types of radiation which have their origin in the differences of their energy deposition properties along the tracks of charged particles. For applications in radiological protection, the complex structure of the charged particle tracks in tissue is characterised by a single parameter only, the unrestricted linear energy transfer, L_∞, (often denoted linear energy transfer, *LET* or L), and the quality factor Q is defined by a function of L as given in various publications of ICRP and ICRU (ICRP, 1963, 1977, 1991b, ICRU, 1970, 1986) – for more details, see Section B.4.2.

(B 77) Another feature of the energy transfer of low- and high-LET particles is the difference in the event distribution as has already been mentioned and discussed in Section B.3.2. This effect influences their biological effectiveness.

(B 78) Radiation weighting factors, w_R, have been specified in the definition of the protection quantities since *Publication 60* (ICRP, 1991b). They are factors by which the mean absorbed dose in any tissue or organ is multiplied to account for the detriment caused by the different types of radiation relative to photon radiation. Numerical values of w_R are specified in terms of type and energy of radiations either incident on the human body or emitted by radionuclides residing within it. Values of w_R adopted in *Publication 60* (ICRP, 1991b) are given in Table B.3.

(B 79) The same values of the radiation weighting factors, w_R, are applied to all tissues and organs of the body, independent of the fact that the actual radiation field varies owing to attenuation and degradation of the primary radiation and the production of secondary radiations of different radiation quality. The value of w_R may therefore be seen as a factor representing radiation quality averaged over the different tissues and organs of the body.

(B 80) The averaging procedure implied in the determination of w_R has raised some concern, especially in the case of external low-energy neutron radiation exposure where secondary photons (low-LET radiation) contribute significantly to tissue

and organ doses (Dietze and Alberts, 2004). Therefore the mean radiation quality in a tissue or organ exposed to low-energy neutrons depends on its position in the body and varies with the direction of incidence on the body.

(B 81) This problem of bi-locality of specifying radiation quality and absorbed dose is discussed in detail in *Publication 92* (ICRP, 2003c). That report proposes how to achieve an improved radiation weighting factor for high-LET particles, and a modified function is given. A fixed relationship is proposed between the radiation weighting factor and a mean quality factor averaged over the human body and calculated for isotropic exposure. The present 2007 Recommendations do not, however, fully follow the procedure proposed in *Publication 92*. Details are given in Section B.3.5, paragraphs B 100 – B 115.

(B 82) Ideally the determination of w_R values would be predominantly based on RBE data from in-vivo investigations related to stochastic effects. Often cancer and leukaemia induction or life shortening after whole-body exposure has been determined. While in-vitro investigations with cells can provide important contributions to the understanding of basic mechanisms regarding carcinogenesis, the RBE values obtained in such studies may not be well correlated with carcinogenesis in humans. In many cases, however, there are only limited data available from in-vivo investigations on animals for the range of radiation qualities of interest in radiological protection. Therefore the $Q(L)$ function which is mainly based on data from in-vitro experiments (NCRP, 1990) is used where necessary as the basis of the calculation of a mean Q-value for the human body which in turn is then used for estimating radiation weighting factor values. This is especially the case for protons and heavy ions, and to some extent for neutrons (ICRP, 2003c).

(B 83) Generally, a broad range of RBE values has been obtained in investigations of various biological effects which do not exhibit a direct relationship to the effects for which radiation weighting factors are required. Experimentally determined RBE values are often associated with large uncertainties due, for example, to the small numbers of animals used and many other influencing factors. The weighting factors are selected to give a representative value for the known data and to be sufficiently accurate for application in radiological protection. The values of w_R are selected by judgement for use in the determination of protection quantities; as such they have fixed values and are not associated with any uncertainty (see Section B.6).

(B 84) ***Reference radiation.*** Values of RBE obtained experimentally depend on the reference radiation chosen. Generally, low-LET radiation is taken as the reference, and mostly ^{60}Co- or ^{137}Cs-gamma rays or high-energy x rays, > 200 kV, have been used in experimental investigations. There exists, however, no international agreement on the choice of a specific photon type or energy as a general reference radiation. Therefore, for all RBE-related studies, information on the reference radiation used is needed.

(B 85) In *Publication 60* (ICRP, 1991b) the Commission adopted a radiation weighting factor of 1 for all photons (Table B.3). This is also proposed in *Publication 92* (ICRP, 2003c) and is consistent with the fact that no specific photon energy has been fixed as a reference. An average of RBE data related to photons of different energies is judged to be most appropriate for establishing w_R values for radiation

protection. This approach does not, however, imply that no differences exist with respect to the biological effectiveness of photons of different energy (see Section B.3.5, paragraphs B 86 – B 99).

(B 86) ***Radiation weighting factors for photons, electrons, and muons.*** Photons, electrons, and muons are low-LET radiations with LET-values of less than 10 keV/μm. Low-LET radiations have always been given a radiation weighting of one. Before 1991 this was achieved by setting $Q(L) = 1$ for $L < 3.5$ keV/μm. *Publication 60* (ICRP, 1991b) defined $w_R = 1$ for these radiations, and $Q(L) = 1$ for $L < 10$ keV/μm for operational dose quantities (see Eqn. B.4.2). This has been decided mainly for practical reasons but also in consideration of the large uncertainties in estimating radiation risk factors which did not justify a more detailed description.

(B 87) Details on published RBE values for low-LET radiation are presented in *Publication 92* (ICRP, 2003c), and the consequences with respect to the weighting of photon radiations of different energies are discussed. Other publications also deal with this subject (e.g., SSK, 2005, Harder et al., 2004).

(B 88) In-vitro investigations of dicentric chromosome aberrations in human lymphocytes (Sasaki, 1991, Schmid et al., 2002, Guerrero-Carbajal et al., 2003), and for mutations and transformations in other cell lines, e.g., in human and human–hamster hybrid cells by Frankenberg et al. (2002), have shown that low energy x rays have a significantly larger RBE than ^{60}Co-gamma rays. In such experiments with cells, 20 kV x rays may be about 2 to 3 times as effective as conventional 200 kV x rays and these are about twice as effective as ^{60}Co gamma rays. In animal experiments, much lower ratios have been observed while epidemiological data are not sufficiently precise to see any differences.

(B 89) While photons of 1 to 5 MeV are less effective than x rays, as demonstrated by cellular effects in vitro, the situation may be different for very high energy photons, e.g., near high energy accelerators, or in radiation fields of cosmic rays. Such photons are able to produce secondary particles in nuclear interactions, e.g., neutrons or other high-LET particles. It can, therefore, not be excluded that the RBE value for these photons is higher than that of photons of about 1 to 5 MeV.

(B 90) The Commission stated in *Publication 60* (ICRP, 1991b) that 'simplicity is important to reflect our lack of precise information in man and an appreciation of the practical aspects of radiological protection. For example, the Commission does not believe it is helpful to adopt different quality-factor values for different photon energies.' More data is now available from investigations on cells showing significant differences in radiation quality of photons of different energies. However, there are additional practical arguments for keeping a single w_R value for all photons and electrons for the calculation of effective dose (Dietze and Alberts, 2004).

(B 91) In the case of external exposure to photons with energies from 30 keV to 5 MeV a proportion of the dose delivered to the organs is due to Compton-scattered photons in the body with an average energy significantly lower than that of the incident photons (Harder et al., 2004). Therefore, the variation of the mean RBE averaged over the human body for external photon radiations with different energies is expected to be smaller than the corresponding differences observed in investigations with thin cell layers in vitro (frequently mono-layers). Chen et al. (2005) have

calculated the microdosimetric quantity, dose mean lineal energy, y_D, in small and large receptors, and have shown that the above-mentioned effect is not as large as assumed by Harder et al. (2004).

(B 92) Furthermore, external low-energy photon radiation (less than about 30 kV x rays) is strongly attenuated in tissue close to the surface of the body and its contribution to effective dose is generally small. An exception to this statement is the use of low-energy photons in radiodiagnostic procedures such as mammography. In this case of external exposure, however, the operational dose quantities $H^*(10)$ and $H_p(10)$ (see Sections B.4.3 and B.4.4) are used for radiation protection monitoring and for assessing effective dose. For photons with energies between 10 keV and 40 keV and frontal irradiation (AP) of the body, $H^*(10)$, is up to a factor 6 higher than E and, for other directions of radiation incidence (PA, LAT, ROT, ISO), this conservatism is even greater (ICRP, 1996b).

(B 93) In internal dosimetry, a single w_R value for all photons and electrons is a major simplification, but the arguments in support of this approach are the same as for external exposures. The special case of the probable greater effectiveness of short-range emissions from tritium and Auger emitters when the radionuclides are incorporated into DNA or otherwise localised in cell nuclei is discussed in Section B.3.3.

(B 94) However, the use of $w_R = 1$ for low-energy beta emissions from tritium is still the subject of scientific debate (CERRIE, 2004). Straume and Carsten (1993) provided a thorough review of experimental data on the carcinogenic, genetic, developmental and reproductive effects of exposure to tritiated water (HTO) and organically bound forms of tritium (OBT) in animals and in in-vitro cell systems. The spectrum of observed effects is indistinguishable from the effects of whole body external irradiation with x rays or gamma rays. Although the observed effects of tritium are very largely attributable to damage from ionising radiation, the transmutation of tritium to helium also has the potential to cause damage to DNA. The observed effects of tritium will include any contribution from such transmutation damage. Considering all observed effects of HTO exposure, RBE values were in the range 1–3.5. For comparisons with gamma rays, most values were between 1 and 3 while for x rays most were from 1 to 2, with values of 1–1.5 predominating. These measured RBEs for tritium beta irradiation are reasonably consistent with estimates based on microdosimetric considerations (Bigildeev et al., 1992, Morstin et al., 1993, Moiseenko et al., 1997).

(B 95) For the purposes of assessing risk at low chronic doses, studies of carcinogenesis are the most appropriate. These include studies of the acceleration of the appearance of mammary tumours in rats (Gragtmans et al., 1984) and the induction of acute myeloid leukaemia in mice (Johnson et al., 1995). Both these studies compared chronic exposure to HTO or to x rays (250 kVp) and gave RBE values of 1–1.3. In-vitro studies of transformation in 10T1/2 cells gave RBE values of up to about 2 compared to gamma rays.

(B 96) The RBE values obtained for beta emissions from tritium as HTO are within the range of values observed generally for low-LET radiations and therefore the simplified approach of using a single w_R value of 1 is applicable to tritium. The

limited RBE data for OBT (organically bound tritium) show similar values to those for HTO in most cases (e.g., labelled amino acids) but higher values for tritiated DNA precursors. For example, Ueno et al. (1989) compared RBE values for HTO, ^{3}H-thymidine (^{3}HTdR) and ^{3}H-amino acids, measuring cell killing and mutation rates in mouse cells cultured in vitro. Doses were estimated on the basis of measurements of the ^{3}H content of cells and on the assumption that ^{3}HTdR was concentrated in the nucleus and that HTO and ^{3}H-amino acids had a uniform cellular distribution. On this basis for ^{3}HTdR a greater effect by a factor of 2 was obtained than for HTO and for ^{3}H-amino acids.

(B 97) The biological effects of Auger emitters have been extensively studied in a variety of in-vitro and in-vivo experimental systems (Bingham et al., 2000, Goddu et al., 1996). In vivo, rodent spermatogenesis has been utilised as a model system to evaluate the cytotoxicity of a range of Auger emitters including ^{55}Fe, ^{99m}Tc, ^{111}In, ^{114m}In, ^{123}I, ^{125}I, and ^{210}Tl. In vitro, the cytotoxic effects of ^{35}S, ^{75}Se, ^{51}Cr, ^{67}Ga, ^{77}Br, and a range of compounds labelled with ^{123}I and ^{125}I, have been studied in a variety of human and rodent cell lines and model culture systems. Representative of various results reported are an increase of biological effectiveness by a factor of 7–9 for ^{125}I when the radionuclide is incorporated into DNA following administration as ^{125}I-iododeoyuridine (^{125}IUdR), RBE values of around 4 for ^{125}I localised in the nucleus, but not directly bound to DNA, and RBE values of around 1 when ^{125}I is localised in the cytoplasm (Hofer et al., 1975; Howell et al., 1993; Kassis et al., 1989; Rao et al., 1990; Warters et al., 1978).

(B 98) Various dosimetric schemes have been proposed for Auger emitters, including the use of a w_R of 20 for the proportion of emitters bound to DNA where this is known (Howell et al., 1993). It is clear that assessment of doses and risks will require information on the distribution of radionuclides within tissues and cells, which will depend on the chemical form involved. It is only when the Auger emitter is concentrated in the nucleus that a significantly enhanced effect would be anticipated, compared with that evaluated on the basis of average tissue dose. The Commission recognises these uncertainties and has stated that Auger emitters will need analysis on a case-by-case basis.

(B 99) In summary, there are good arguments for the continued use of a w_R of 1 for all low-LET radiations for general radiological protection purposes. It is, however, important to note that this simplification is sufficient only for the intended application of assessing effective dose, e.g., for dose limitation, assessment and controlling of doses. It is not intended for retrospective assessment of individual risks of stochastic effects from radiation exposures. In such cases of individual retrospective dose assessment more detailed information on the radiation field (including the type of low-LET radiation) and appropriate RBE values may need to be considered if they are available (see Section B.5.8). Heterogeneity of dose within cells, as can occur with tritium or Auger emitters incorporated into DNA, may also require specific analysis.

(B 100) ***Radiation weighting factors for neutrons.*** The biological effectiveness of neutrons incident on the human body is strongly dependent on the neutron energy because of the variation of the secondary radiation with energy. Qualitatively, the following effects are important:

- the production of secondary photons by neutron absorption in tissue which increases with decreasing neutron energy;
- the increase of the energy of recoil protons with increasing neutron energy;
- the release of heavier charged particles at higher neutron energies; and
- nuclear spallation processes at very high neutron energies.

(B 101) In *Publication 60* (ICRP, 1991b) the radiation weighting factor for neutrons has been given in two ways, by a step function defining five neutron energy ranges with w_R values of 5, 10, 20, 10, and 5 respectively (Table B.3, Fig. B.4), and by a continuous function for use in calculations. The tabulated values of w_R have generally not been used in practice; the continuous function has usually been applied. In radiation fields containing neutrons with a broad energy spectrum very often calculations using energy-dependent conversion coefficients are performed for estimating doses. All internationally recommended conversion coefficients, including those given in *Publication 74* (ICRP, 1996b) are based on the continuous function. Therefore, a continuous function is given here for defining radiation weighting factors for neutrons. It should be noted, however, that the use of a continuous function is based only on practical and computational considerations and does not imply the availability of more precise data.

(B 102) In *Publication 60* (ICRP, 1991b) a maximum value of 20 was fixed for w_R. In *Publication 92* (ICRP, 2003c) it is stated that, in the neutron energy region near 1 MeV, the maximum value of w_R of about 20 is still an acceptable approximation. This judgement is not based on a specific experimental value but rather reflects a representative value considering the broad range of RBE values from experimental animal data for carcinogenesis and life shortening obtained from investigations using

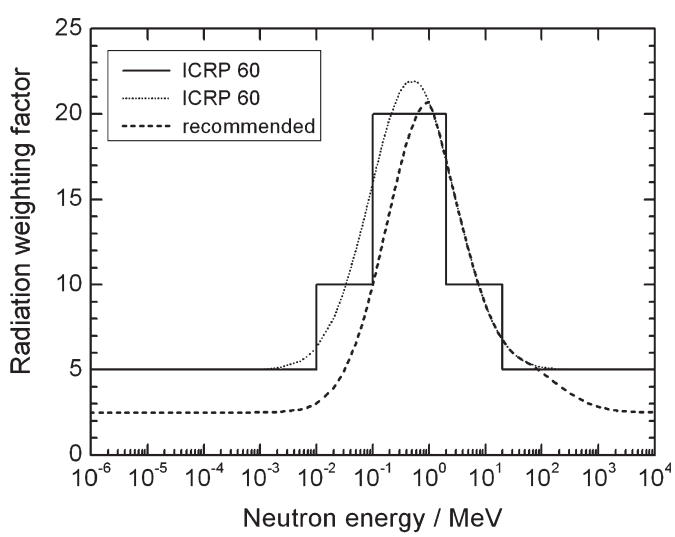

Fig. B.4. Radiation weighting factor, w_R, for neutrons versus neutron energy. Step function and continuous function given in *Publication 60* (ICRP 1991b) and function adopted in the 2007 Recommendations.

fission neutrons from reactors (ICRP, 2003c). This value of 20 is, therefore, retained for neutron energies at about 1 MeV.

(B 103) When the human body is exposed to neutrons with energies below 1 MeV, a significant fraction of the absorbed dose is deposited by secondary photons mainly from the H(n, γ) reaction, which reduces the biological effectiveness. In this energy range this effect on RBE is much larger than the influence of the change in the LET-distribution of the neutron-produced secondary charged particles, mainly protons.

(B 104) When RBE data for fission neutrons or low-energy neutrons obtained from investigations with small animals are used as the basis for the evaluation of w_R values for human exposure, it should be recognised that the dose contribution from secondary photons in the human body is higher than in small animals such as mice (Dietze and Siebert, 1994). The photons are mainly produced by the capture of degraded neutrons, predominantly in hydrogen, and their contribution to the total equivalent dose in an organ is strongly dependent on the body size and on the position of the organ in the body. At the time of *Publication 60* (ICRP, 1991b) data from calculations with neutrons in anthropomorphic phantoms were not available, and data calculated for the ICRU sphere were used instead. It has been shown (ICRP, 2003c, SSK, 2005) that for neutrons below about 1 MeV, the consideration of the secondary photons in an anthropomorphic phantom results in considerably lower values for mean quality factors, and thus of w_R, than those given in *Publication 60*.

(B 105) In *Publication 92* (ICRP, 2003c), it is suggested that the dependence of the radiation weighting factor on neutron energy should be based on the $Q(L)$ function defined in *Publication 60* (ICRP, 1991b) and the calculation of a human body averaged mean quality factor q_E (see Eqn. B.3.10). The relationship between q_E and the weighting factor w_R is given there by a function

$$w_R = 1.6(q_E - 1) + 1 \tag{B.3.10}$$

This equation preserves a value of w_R of about 20 at neutron energies near 1 MeV. Calculations of q_E have been performed taking the dose distribution in the human body into account and using the tissue weighting factors w_T of the different organs and tissues by the equation

$$q_E = \sum_T w_T Q_T D_T / \sum_T w_T D_T \tag{B.3.11}$$

where Q_T is the mean quality factor in the tissue or organ T, and D_T the corresponding mean absorbed dose. Owing to the different w_T values of the organs and tissues not being symmetrically distributed in the human body, the value of q_E depends on the directional incidence of the radiation on the body. The calculations have shown that, for thermal neutrons, the deduced w_R (Eqn. B.3.10) may vary from 2.5 (for ISO and ROT incidence) to 3.2 (for AP incidence) for the various exposure conditions, and that there are also differences depending on the sex of the selected model (Kellerer et al., 2004). In general, the value of q_E depends also on the model of the human body, e.g., if the calculations are performed with a MIRD-type phantom or a voxel type phantom (see Section B.5.2).

(B 106) In principle, the proposal of defining a general relationship between w_R and a mean quality factor q_E for all types and energies of particles as given in Eqn. (B.3.10) is attractive, because it points more clearly to the common scientific basis of the concept of radiation weighting and quality factor used in the definition of the operational quantities. In practice, however, Eqn. (B.3.10) can only be applied to strongly penetrating external high-LET radiation, e.g., neutrons, high-energy protons and heavy ions with very high energies. A factor of 1.6 has been introduced in Eqn. (B.3.10) in order to fit the calculated w_R-value for 1 MeV neutrons to experimental data. It is questionable whether it is justified to extend this factor to other particles and energies with different secondary charged particle spectra. Another shortcoming of defining this general relationship is the fact that q_E depends on many parameters, such as the phantom selected, the w_T values, the exposure situation chosen and even the computer code used. Many parameters may give rise to changes in future while w_R should remain stable. Equation (B.3.10) is therefore to be used only as a guide in establishing values of w_R for neutrons.

(B 107) For neutron energies of less than 1 MeV a similar energy dependence of the radiation weighting has been obtained also by other considerations (SSK, 2005, Dietze and Harder, 2004) without using a fixed relationship between Q and w_R. The relationship is based on the assumption that, with neutron energies below 1 MeV, the energy dependence of the neutron weighting for the human body depends mainly on the dose contribution of secondary photons and that, for a small tissue probe, the mean RBE value for the neutron-induced high-LET component ($RBE_{high\text{-}LET}$, mainly determined by recoil protons, protons from N(n,p) and heavier ions) is approximately independent of neutron energy (Edwards, 1997, Sasaki, 1991, Schmid et al., 2003).

(B 108) For anterior-posterior radiation incidence the mean absorbed dose contribution from secondary photons $f_{low\text{-}LET}$ (low-LET component relative to the total dose) in the human body and the contribution from secondary charged particles (high-LET component) have been calculated by

$$f_{low\text{-}LET} = (\Sigma w_T D_T f_{low\text{-}LET,T})/(\Sigma w_T D_T) \text{ and} \tag{B.3.12}$$

$$f_{high\text{-}LET} = 1 - f_{low\text{-}LET} \tag{B.3.13}$$

where $f_{low\text{-}LET,T}$ is the relative absorbed dose contribution in the tissue or organ T from secondary low-LET radiation. For the calculation of a body-averaged relative biological effectiveness the following equation has been applied:

$$RBE_{av} = RBE_{high\text{-}LET}(1 - f_{low\text{-}LET}) + RBE_{low\text{-}LET} f_{low\text{-}LET} \tag{B.3.14}$$

where RBE_{av} is the resulting RBE properly averaged over the human body. This 'mixing rule' is applied in the neutron energy range from thermal up to 1 MeV. For the photon contribution a value of $RBE_{low\text{-}LET} = 1$ is taken, and for the high-LET component a mean value of $RBE_{high\text{-}LET} = 25$ is chosen which is consistent with experimental data on the induction of dicentric chromosomal aberrations in cells (Schmid et al., 2003) and animal data for tumour induction and life shortening (SSK, 2005). These selected RBE-values result in an RBE_{av} value of about 20 in

the human body for neutrons of 1 MeV which is consistent with the value mentioned above. Depending on the exposure conditions chosen, the energy dependence of RBE_{av} is similar to that of w_R calculated by Eqn. (B.3.10) in the energy range from thermal up to 1 MeV neutrons.

(B 109) In view of all these considerations the following function is given for the definition of the radiation weighting factor in the energy range below 1 MeV:

$$w_R = 2.5 + 18.2 \exp[-(\ln E_n)^2/6] \text{ for } E_n < 1 \text{ MeV} (E_n \text{in MeV}) \tag{B.3.15}$$

(B 110) Figure B.4 shows that, in the neutron energy range below 1 MeV, the values of w_R are lower than those given in *Publication 60* (1991b). The function fully reflects the effect of secondary photons in the body and is well related to the mean quality factor q_E as given in *Publication 92* (ICRP, 2003c).

(B 111) The energy range above 1 MeV needs different considerations. In this energy range, almost no new experimental data is available from investigations of animals. All existing experimental data either on animals or on cells, however, show a clear decrease of RBE with increasing neutron energy. This is consistent with calculations based on the $Q(L)$ function (ICRP, 2003c). If, however, the relationship between q_E and w_R as defined in Eqn. (B.3.10) were applied, this would result in an increase of w_R for neutrons of about 30% in the energy range between 5 MeV and 100 MeV relative to the data of the continuous function as defined in *Publication 60* (ICRP, 1991b). This difference is much less than the uncertainty of RBE values in this energy range. Therefore, from a practical point of view, it seems more appropriate not to apply minor changes to the existing function in this energy range, but to stay with the values defined in *Publication 60*.

(B 112) There is no published experimental data with animals for neutron energies above about 50 MeV. Some RBE data on the induction of dicentric chromosomes in human lymphocytes was published recently (Nolte et al., 2005). These data together with the calculations of Pelliccioni (1998, 2004), Yoshizawa et al. (1998), and Sato et al. (2003) have shown that the mean quality factor averaged over the human body decreases with increasing neutron energy to values of less than 5, and reaches values near to those of protons at very high energies above 1 GeV. While this topic may need more detailed considerations in the future, a continuous weighting factor function for neutrons is also used for energies above 50 MeV. Its value decreases with increasing energy from about 5.5 at 50 MeV to about 2.5 at 10 GeV. This function is fitted to the function for lower neutron energies at 50 MeV. The neutron energy dependence of the data published by Pelliccioni (1998, 2004), Yoshizawa et al. (1998), and Sato et al. (2003) has been used as a guideline for the higher energies.

(B 113) In summary, the following continuous functions are used for the calculation of radiation weighting factors for neutrons:

$$w_R = \begin{cases} 2.5 + 18.2\, e^{-[\ln(E_n)]^2/6}, & E_n < 1 \text{ MeV} \\ 5.0 + 17.0\, e^{-[\ln(2E_n)]^2/6}, & 1 \text{ MeV} \leqslant E_n \leqslant 50 \text{ MeV} \\ 2.5 + 3.25\, e^{-[\ln(0.04E_n)]^2/6}, & E_n > 50 \text{ MeV} \end{cases} \tag{B.3.16}$$

Obviously these functions are complex. They have been chosen as an empirical approach describing the weighting of neutrons over more than 10 decades of neutron energy. The detailed functions, however, should not be misinterpreted to reflect precisely the biological data which, in fact, show a broad range of RBE values depending on neutron dose, neutron dose rate and biological endpoint considered.

(B 114) The preceding extensive discussion of this important matter of energy dependence of w_R for neutrons can be summarised as follows:

- The new Recommendations adopt a w_R function for neutrons which is based upon that given in *Publication 92* (ICRP, 2003c) but takes into account additional data.

The function for w_R for neutrons is derived using the following criteria:

- A continuous w_R function is chosen instead of a step function (ICRP, 1991b) for practical reasons. This decision, however, is not the result of a higher precision of the available radiobiological data but is based on practical considerations.
- For neutrons of about 1 MeV a maximum w_R value of about 20 is retained as given in *Publication 60* (ICRP, 1991b) and in *Publication 92* (ICRP, 2003c).
- For neutron energies below about 1 MeV the shape of the curve for the energy dependence of w_R is generally based on that related to the mean quality factor q_E, as well as on the mean RBE_{av} expressed in Eqn. (B.3.14). The recommended w_R values are similar to those proposed in *Publication 92* (ICRP, 2003c).
- At energies above 50 MeV, for physical reasons w_R should asymptotically approach a value close to that of protons (for which some radiobiological data exist). Based on calculations by Pelliccioni (1998, 2004), Yoshizawa et al. (1998) and Sato et al. (2003) an asymptotic value of 2.5 at neutron energies above 1 GeV is chosen.

(B 115) The resulting function (Fig. B.4) is consistent with existing relevant physical and biological knowledge. The function does not establish a strict relationship between the mean quality factor and the radiation weighting factor for all neutron energies as proposed in *Publication 92* (ICRP, 2003c), and therefore there is not a fully common approach for protection and operational quantities, for the reasons given above. For radiological protection it appears, however, to be more important that the operational dose quantities for use in external exposure monitoring provide a good and conservative estimate of effective dose under most exposure conditions. This is achieved when applying the radiation weighting factors for neutrons as given in Eqn. (B.3.16).

(B 116) ***Radiation weighting factor for protons and pions.*** Only external radiation sources have to be considered for exposure to protons in practical radiological protection. In *Publication 60* (ICRP, 1991b) a radiation weighting factor of 5 was recommended for all protons with energies above 2 MeV except recoil protons (Table B.3).

(B 117) In recent years, proton radiation has received more attention owing to an increased interest in dose assessment for exposure to aircrew and to astronauts in space vehicles. In these cases the external proton radiation exposure is from solar and cosmic radiation. In the primary radiation fields, high energy protons strongly

dominate, and protons with energy of a few MeV are of minor significance, even when considering the increasing biological effectiveness at low energies. The range of low-energy protons in tissue is small (range of protons in tissue: 4 MeV protons: 0.25 mm; 10 MeV protons: 1.2 mm) and they will mostly be absorbed in the skin.

(B 118) It is, therefore, judged to be sufficiently accurate for applications in radiological protection to adopt a single w_R value for protons of all energies. It is appropriate to rely on data for high energy protons as these are the most relevant in cosmic radiation fields.

(B 119) There are very few investigations using animals that give information on the RBE for high energy protons. Most measured RBE values are between 1 and 2. With respect to the ionisation density in tissue, high energy protons can be regarded as low-LET radiation (with a mean LET value much less than 10 keV/μm) and, applying the $Q(L)$ function from *Publication 60* (ICRP, 1991b), the mean quality factor of 100 MeV protons stopping in tissue is calculated to be less than 1.2 (ICRP, 2003c). At very high proton energies near 1 GeV, secondary charged particles from nuclear reactions become more important, and the mean quality factor increases up to about 1.8.

(B 120) Taking all considerations and available data into account, the radiation weighting factor adopted for protons in the new Recommendations is 2 (Table B.4).

(B 121) Pions are negatively or positively charged or neutral particles encountered in radiation fields at altitude in the atmosphere, resulting from interactions of the primary cosmic rays (predominantly high-energy protons) with nuclei in the atmosphere, thus contributing to the exposure of aircraft crew and passengers (approximately 0.1% of $H^*(10)$). They are also found as part of the complex radiation fields behind shielding of high-energy particle accelerators and thus may contribute to the occupational exposure of accelerator staff (up to 4% of $H^*(10)$). The mass of pions is equivalent to 273 electron masses and approximately 1/7 of a proton mass. Charged pions lose their energy mainly through Coulomb interactions. When negative pions come to rest they are usually captured by nuclei which then disintegrate emitting a variety of high-LET particles ('star fragmentation').

(B 122) Pelliccioni (1998) has carried out Monte Carlo calculations for evaluating mean quality factors averaged over the human body (see Eqn. B.3.12) for pions as a function of their energy. The results show that there is a moderate energy dependence of the mean quality factor for positive pions and for negative pions above 50 MeV (values between 1 and 2). Below this energy, the star fragmentation leads to an increase of q_E of negative pions.

(B 123) Considering that the energy distribution of pionsa in real radiation field is very broad and in view of their small contribution to total exposure in complex high-energy fields, it is recommended to use a weighting factor of 2 for all charged pions.

(B 124) ***Radiation weighting factor for alpha particles.*** Exposure of humans to alpha particles is predominantly from internal emitters, e.g., from inhaled radon progeny or ingested alpha-emitting radionuclides such as isotopes of plutonium, polonium, radium, thorium, and uranium. There are a number of epidemiological studies that provide information on the risk from inhaled or intravenously injected alpha emitters. The distribution of radionuclides and the estimation of dose and its

distribution in tissues and organs are very complex and dependent on the models used. The dose distribution is generally very heterogeneous and the calculated doses are, therefore, associated with substantial uncertainties. For this reason epidemiological as well as experimental studies, although they can provide valuable guidance, cannot be used as the only basis for an assessment of the RBE for alpha emitters. From calculations using stopping power data for alpha particles in tissue and the $Q(L)$ function, the mean quality factor of 6 MeV alpha particles slowing down in tissues is estimated to be about 20.

(B 125) Reviews of available human and animal data on RBE for alpha-emitting radionuclides indicate that RBE depends on the biological end-point under consideration (UNSCEAR, 2000, Harrison and Muirhead, 2003). Variations between radionuclides in RBE values for the same end-point can be attributed mainly to differences in location of the emitter in tissue. The limited human data that allow estimation of alpha particle RBE values suggest values of around 10–20 for lung and liver cancer and lower values for bone cancer and leukaemia.

(B 126) There is good evidence, from animal and in-vitro studies, of RBE values for alpha emitters of around 10 or greater, compared with external low-LET radiations, for cancer-related effects. Studies of bone cancer induction in dogs suggest different RBE values for this endpoint for different bone-seeking alpha-emitting radionuclides, with high values for ^{239}Pu and low values for Ra isotopes (UNSCEAR, 2000). However, these comparisons are based on average skeletal doses, and the differences are most likely to be attributable to the different locations of the radionuclides in bone, with greater doses to target cells near to bone surfaces from ^{239}Pu and related actinide isotopes, which concentrate at bone surfaces, compared to isotopes of Ra which (as alkaline earth elements chemically similar to Ca) tend to be distributed more uniformly through the calcified bone matrix (ICRP, 1993c, Harrison and Muirhead, 2003). Human and animal data suggest that the RBE for the risk of leukaemia from alpha emitters deposited in bone is less than 20 (WHO, 2001, Harrison and Muirhead, 2003). The use of a w_R of 20 for alpha particles may thus lead to an overestimation of risk to target cells within active (red) bone marrow.

(B 127) Judgements on the available data and the selection of a w_R value for alpha particles have been reviewed in *Publication 92* (ICRP, 2003c). As recent data do not strongly support the need for a change of the radiation weighting factor for alpha particles, the w_R value of 20 is retained for these Recommendations (see Table B.4).

(B 128) ***Radiation weighting factor for heavy ions and fission fragments.*** Doses from fission fragments are of importance for radiological protection mainly in internal dosimetry, and the situation regarding radiation weighting factors may be seen as similar to that for alpha particles. Owing to their short ranges, the biokinetics and distribution of the actinides in the organs and tissues are very important and have a strong influence on their biological effectiveness. A radiation weighting factor of 20, as given in Tables B.3 and B.4, equal to that for alpha particles, may be regarded as a rough conservative estimate. The short range of the fission fragments in tissue and the high energy transferred, therefore, to a small volume of tissue results in a very high local dose at this point, which may reduce their RBE. As has been

discussed in Section B.3.2, care must be taken when applying the concept of mean organ or tissue doses in such cases, and specific considerations are necessary.

(B 129) In external exposure heavy ions are mainly encountered in radiation fields near high energy accelerators, at aviation heights, and in space. Few RBE data for heavy ions are available and most are from in-vitro experiments. *Publication 92* (ICRP, 2003c) provides an overview on radiobiological data from which RBE values have been derived of relevance for defining radiation weighting factor values.

(B 130) RBE_M values of about 30 have been reported for induction of Harderian gland tumours in mice by the heavy ions ^{40}Ar and ^{56}Fe and lower values with radiation beams of lower LET (Fry et al., 1985, Alpen et al., 1993). The results indicate that RBE values reach a peak at about 100–200 keV μm^{-1} and remain at this level at higher LETs. RBE values for fission neutrons in the same system were shown to correspond to the maximum observed RBE value for heavy ions. In vitro studies of chromosome aberrations, cell transformation, and mutations also provide evidence of increasing RBE of heavy ions with increasing LET up to around 100–200 keV μm^{-1}, but suggest a decrease at very high LETs.

(B 131) Mean quality factors have been calculated by Sato et al. (2004). The radiation quality of the particle changes strongly along the track for heavy ions incident on a human body and stopped in the body. An averaged value may be chosen to derive a w_R. The selection of a single w_R value of 20 for all types and energies of heavy ions is judged to be appropriate for general application in radiological protection. For applications in space, where these particles contribute significantly to the total dose in the human body, a more realistic approach may be chosen based on the calculation of a mean quality factor in the human body as mentioned in Section B.3.5, paragraphs B 100 – B 115.

Tissue weighting factors

(B 132) The definition of effective dose takes into account the different relative radiosensitivities of the various organs and tissues in the human body with respect to radiation detriment from stochastic effects. For this purpose, weighting factors, w_T, were introduced in *Publication 26* (ICRP, 1977) for six identified tissues and for a remaining group of tissues (collectively referred to as the 'remainder'). In *Publication 60* (ICRP, 1991b) tissue weighting factors were specified for twelve tissues and organs and the 'remainder' (Table B.1). The tissue weighting factors are relative values, and their sum is equal to one so that a uniform dose distribution in the whole body gives an effective dose numerically equal to the equivalent dose in each organ and tissue of the body.

(B 133) The tissue weighting factors determined for these 2007 Recommendations are based on detriment-adjusted nominal risk coefficients for stochastic effects (Annex A). The unadjusted nominal risk coefficients are calculated by averaging estimates of the radiation-associated lifetime risk for cancer incidence for a composite population of equal numbers of males and females. The detriment is modelled as a function of life lost, lethality and loss of quality of life. With a few exceptions, the parameters in the risk models are estimated using cancer incidence data from

the studies of the Japanese atomic bomb survivors. Both excess relative risk and excess absolute risk models are developed for most cancer sites.

(B 134) For heritable disease, the risk in the first two generations is taken into account as discussed and described in Annex A. The relative radiation detriments differ from those given in *Publication 60*, and this has resulted in changes to the w_T values. The main changes are for breast (from 0.05 to 0.12), gonads (from 0.20 to 0.08) and remainder tissues (from 0.05 to 0.12). In addition, specific w_T values of 0.01 are now given for the brain and salivary glands. The tissue weighting factors proposed by the Commission for the present Recommendations are given in Table B.2.

(B 135) The tissue weighting factors, w_T, are sex-averaged and are for the assessment of effective dose for workers as well as members of the public, including children. Recently the w_T values were also applied to the developing fetus in *Publication 88* (ICRP, 2001), although it was recognised that 'these w_T values have been developed for exposure of individuals after birth and that the apportionment of radiation detriment that these values imply may not be appropriate for doses received in utero'. The approach was, however, adopted in the absence of comprehensive data on the relative risks to organs and tissues from exposures in utero. It was concluded in *Publication 90* (ICRP, 2003a) and by Streffer (2005) that there are at present insufficient data to be able to make recommendations of specific w_T values for prenatal radiation exposures.

(B 136) In the case of sex-specific differences in relative detriment based on cancer incidence for the ovaries of females (Annex A, Section A.4.6) the sex-averaged w_T of 0.08 assigned to the gonads (cancer plus heritable effects) is similar to that of the female ovaries (0.036) plus heritable effects (0.039). In this way the ovary of females is judged to be sufficiently protected.

(B 137) In the case of the thyroid, the values of the relative detriment based on cancer incidence for females (0.021) and males (0.008) (Annex A, Section A.4.6) differ by a factor of almost 3. However, since the w_T assigned to the thyroid is given as 0.04 to allow for the high susceptibility of young children, the difference in detriment between the sexes is considered in a conservative manner.

(B 138) A particular issue in the calculation of effective dose is the assessment of the dose to 'remainder' tissues. In *Publication 26* (ICRP, 1977), the remainder tissue was assigned a weighting factor of 0.30. The dose equivalent to the remainder tissues was taken to be the arithmetic average of the dose to the five most highly irradiated tissues of the remainder by allocating a w_T value of 0.06 to each of these tissues. This procedure resulted in a lack of additivity of the effective dose equivalent quantity, since the five tissues could vary for different exposures, either external or internal.

(B 139) In *Publication 60*, the remainder tissue was given a weighting factor of 0.05. However, additivity was still lacking although reduced in magnitude owing to the splitting rule given in Note 3 of Table A-3 in *Publication 60* (see below). The equivalent dose for the remainder was given as the mean value for ten specified tissues and organs (Table B.1). The upper large intestine, formerly included in the remainder (ICRP, 1991b), was taken together with the lower large intestine, to define the colon (ICRP, 1995a). *Publication 66* (ICRP, 1994a) dealing with doses to the

respiratory tract and dose coefficients for inhaled radionuclides specified that the extrathoracic airways be considered as part of the remainder.

(B 140) While not detailed in *Publication 60* (ICRP, 1991b), the treatment of remainder was described in *Publications 68* and *72* (ICRP, 1994b, 1996c). The remainder dose was defined by the mass-weighted average of the equivalent dose to organs and tissues of the remainder (Note 2 of Table A-3 in *Publication 60*). Owing to the very different masses the contribution of the specified tissues and organs to the remainder dose was very different. Because of its large mass, muscle received an effective weighting factor of nearly 0.05 which is not justified because its radiation sensitivity is judged to be low. For external exposure, however, the dose to the various tissues are similar (differ little from that of muscle) and hence in *Publication 74* (ICRP, 1996b) a simple arithmetic dose averaging with no further weighting was used as an approximation (see Section B.3.4).

(B 141) The method for calculating effective dose recommended in *Publication 60* (ICRP, 1991b) includes provision for cases when a remainder tissue which does not have an explicit weighting factor (w_T) receives the highest dose of all tissues. In these cases the w_T for remainder (0.05) is divided equally between the mass-weighted average dose to remainder tissues (i.e., the default remainder dose, see above) and the particular tissue. This is often referred to as the 'splitting rule' and cases where the rule applies are known as 'split remainder' cases.

(B 142) Implications of this rule were explored by Nelson et al. (1997). The intention of the splitting rule was to provide protection, through the effective dose and its related limits, to potentially highly exposed tissues (such as extrathoracic region, ET_1 or the kidney after incorporation of certain radionuclides) which had not been assigned a specific weighting factor. One of the drawbacks of this approach, however, is that, since the formulation of the effective dose can differ for different radionuclides or for different external photon beam energies, it is not strictly an additive quantity.

(B 143) It is now recommended that the equivalent doses to the specified tissues of the remainder given in Table B.2 are added with no further mass weighting. This means that the weighting factor given to each of the remainder tissues is smaller than the least value assigned to any of the named tissues (0.01). For remainder tissues the w_T is 0.12.

(B 144) In its computations, the Commission assigns a dose to the remainder which represents the arithmetic average of the doses to the remainder tissues of both sexes. Analogous to the approach for other organs and tissues, the equivalent dose to the remainder is defined separately for males and females, and these values are included into Eqn. (B.3.9). The equivalent dose to the remainder tissues is computed as the arithmetic mean of the equivalent doses to the tissues listed in the footnotes to Table B.2. The current remainder formulation specifies 12 tissues common to both sexes and one sex-specific tissue in each sex (prostate in the male and uterus/cervix in the female) for a total of 13 tissues. The equivalent dose to the tissues of remainder of the male, H^{M}_{rem}, and female, H^{F}_{rem}, are computed as:

$$H_{\mathrm{rem}}^{\mathrm{M}} = \frac{1}{13}\sum_{\mathrm{T}}^{13} H_{\mathrm{T}}^{\mathrm{M}} \text{ and } H_{\mathrm{rem}}^{\mathrm{F}} = \frac{1}{13}\sum_{\mathrm{T}}^{13} H_{\mathrm{T}}^{\mathrm{F}}. \tag{B.3.17}$$

(B 145) The summation in Eqn. (B.3.9) extends over the equivalent dose to remainder tissues in the male and female.

B.3.6. References, Section B.3

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B.4. Operational quantities

(B 146) The body-related protection quantities (equivalent dose and effective dose) are not measurable in practice and therefore cannot be used directly as quantities in radiation monitoring. Operational quantities are, therefore, used for the assessment of effective dose or equivalent doses in tissues or organs (Figs B.1 and B.2).

(B 147) Operational quantities are aimed at providing an estimate or upper limit for the value of the protection quantities related to an exposure, or potential exposure of persons under most irradiation conditions. They are often used in practical regulations or guidance. As shown in Fig. B.2, different types of operational quantities are used for internal and external exposures. For monitoring of external radiation exposures operational dose quantities have been defined by ICRU (ICRU, 1985, 1988), see Section B.4.2, and, during the 1990s, introduced into radiological protection practice in many countries. Their further use is recommended, and only small changes are proposed. In internal dosimetry, no operational dose quantities have been defined which directly provide an assessment of equivalent or effective dose. Different methods are applied to assess the equivalent or effective dose due to radionuclides in the human body. They are mostly based on various activity measurements and the application of biokinetic models (computational models) (see Section B.4.2).

B.4.1. External exposure

(B 148) Specific operational dose equivalent quantities are defined for radiation monitoring in situations of external exposure (area or individual monitoring). In routine monitoring, the values of these dose-equivalent quantities are taken as sufficiently precise assessments of effective dose or skin dose, respectively, especially if their values are below the protection limits.

(B 149) Operational dose quantities are used for monitoring external exposures because:

- point quantities are needed for area monitoring;
- in area monitoring, the value of the dose quantity should not depend on the directional distribution of the incident radiation;
- instruments for radiation monitoring need to be calibrated in terms of a physical quantity for which calibration standards exist.

(B 150) Different operational dose equivalent quantities have been defined for area and individual monitoring.

(B 151) The basic concept of the operational dose quantities for external exposure is described in ICRU Reports 39 and 43 (ICRU, 1985, 1988). The definitions adopted for the 2007 Recommendations are given in ICRU Report 51 (ICRU, 1993b) and in ICRU Report 66 (ICRU, 2001b).

(B 152) As described in Section B.1, the quantity dose equivalent, H, is defined by

$$H = Q \cdot D \tag{B.4.1}$$

where D is the absorbed dose at the point of interest in tissue and Q the corresponding quality factor at this point, the value of which is determined by the type and energy of charged particles passing a small volume element at this point. It is well known that the biological effectiveness of a radiation is correlated with the ionisation density along the track of charged particles in tissue. Therefore, Q is defined as a function of the unrestricted linear energy transfer, L_∞ (often denoted as L or LET), of charged particles in water:

$$Q(L) = \begin{cases} 1 & L < 10\ \text{keV}/\mu\text{m} \\ 0.32\,L - 2.2 & 10 \leqslant L \leqslant 100\ \text{keV}/\mu\text{m} \\ 300/\sqrt{L} & L > 100\ \text{keV}/\mu\text{m} \end{cases} \tag{B.4.2}$$

(B 153) The quality factor function $Q(L)$ was given in *Publication 60* (ICRP, 1991b). The function is the outcome of judgements taking account of results of radiobiological investigations on cellular and molecular systems as well as on the results of animal experiments. The radiobiological database for the assessment of this function is largely unchanged since 1990 (see ICRP, 2003c) and no changes are proposed.

(B 154) The quality factor Q at a point in tissue is then given by:

$$Q = \frac{1}{D}\int_{L=0}^{\infty} Q(L) D_L \mathrm{d}L \tag{B.4.3}$$

where $D_L = \frac{\mathrm{d}D}{\mathrm{d}L}$ is the distribution of D in L for the charged particles contributing to absorbed dose at the point of interest. This function is particularly important for neutrons because various types of secondary charged particles are produced in tissue by neutron interactions.

(B 155) Different operational dose quantities are required for different tasks in radiological protection. These include area monitoring for controlling the radiation in workplaces and for defining controlled or restricted areas, and individual monitoring for the control and limitation of individual exposures. While measurements with an area monitor are preferably performed free in air, personal dosimeters are worn on the body. As a consequence, in a given situation, the radiation field 'seen' by an area monitor free in air differs from that 'seen' by a personal dosimeter worn on the body where the radiation field is strongly influenced by the backscatter and absorption of radiation in the body. The use of different operational dose quantities reflects these differences.

(B 156) Table B.5 can be used in order to describe the application of the different operational dose quantities for the different tasks of monitoring of external exposures.

(B 157) Using the scheme of Table B.5, it is not necessary to use the terms 'strongly penetrating radiation' (also called 'penetrating radiation') and 'low-penetrating radiation' (also called 'weakly penetrating radiation') in specifying the range of application of the operational quantities. ICRU (1993b) stated that $H^*(10)$ and $H_\mathrm{p}(10)$ are designed for monitoring strongly penetrating radiation, e.g., photons (above about 12 keV) and neutrons, while $H'(0.07, \Omega)$ and $H_\mathrm{p}(0.07)$ are applied for monitoring low-penetrating radiation, e.g., beta particles. Furthermore, $H_\mathrm{p}(0.07)$ is also used

Table B.5. Application of operational dose quantities for monitoring of external exposures.

Task	Operational dose quantities for	
	area monitoring	individual monitoring
Control of effective dose	ambient dose equivalent, $H^*(10)$	personal dose equivalent, $H_p(10)$
Control of doses to the skin, the hands and feet and the lens of the eye	directional dose equivalent, $H'(0.07,\Omega)$	personal dose equivalent, $H_p(0.07)$

for monitoring the doses to the hands and feet from all ionising radiation. The rarely used quantities $H'(3, \Omega)$ and $H_p(3)$ for monitoring the exposure of the lens of the eye are not included in this scheme. Monitoring of $H_p(0.07)$ can be used for the same monitoring purpose (see also this Section, paragraphs B 165 – B 167).

(B 158) There are situations in which individual monitoring is not used and where area monitoring or computational methods are applied to assess individual exposures. These situations include the assessment of doses to aircrew, prospective dose assessments and assessment of doses in workplaces and the natural environment.

Operational quantities for area monitoring

(B 159) For all types of external radiation, the operational quantities for area monitoring are defined on the basis of a dose equivalent value at a point in a simple phantom, the ICRU sphere. It is a sphere of tissue-equivalent material (30 cm in diameter, ICRU (soft) tissue with density: 1 g cm^{-3}, and mass composition: 76.2% oxygen, 11.1% carbon, 10.1% hydrogen, and 2.6% nitrogen). For radiation monitoring, in most cases it adequately approximates the human body as regards the scattering and attenuation of the radiation fields under consideration.

(B 160) The operational quantities for area monitoring defined in the ICRU sphere should retain their character of a point quantity and the property of additivity. This is achieved by introducing the terms 'expanded' and 'aligned' radiation field in the definition of these quantities.

(B 161) An *expanded* radiation field, defined as a hypothetical field, is a radiation field in which the spectral and the angular fluence have the same value in all points of a sufficiently large volume equal to the value in the actual field at the point of interest. The expansion of the radiation field ensures that the whole ICRU sphere is thought to be exposed to a homogeneous radiation field with the same fluence, energy distribution and direction distribution as in the point of interest of the real radiation field.

(B 162) If all radiation is aligned in the expanded radiation field so that it is opposed to a radius vector Ω specified for the ICRU sphere, the *aligned and expanded* radiation field is obtained. In this hypothetical radiation field, the ICRU sphere is homogeneously irradiated from one direction, and the fluence of the field is the integral of the angular differential fluence at the point of interest in the real radiation field over all directions. In the expanded and aligned radiation field, the value of

the dose equivalent at any point in the ICRU sphere is independent of the direction distribution of the radiation in the real radiation field. Conversion coefficients relating radiation field quantities to the operational quantities are usually calculated assuming a vacuum outside of the phantom considered.

(B 163) ***Ambient dose equivalent, H^*(10)***. For area monitoring the operational quantity for assessing effective dose is the ambient dose equivalent, $H^*(10)$, defined by (ICRU, 2001b):

- The *ambient dose equivalent*, $H^*(10)$, at a point in a radiation field, is the dose equivalent that would be produced by the corresponding expanded and aligned field in the ICRU sphere at a depth of 10 mm on the radius vector opposing the direction of the aligned field.

(B 164) In most practical situations of external radiation exposure, the ambient dose equivalent fulfils the aim of providing a conservative estimate or upper limit for the value of the limiting quantities. This is not always the case for persons in high energy radiation fields such as in the vicinity of high energy accelerators and in cosmic ray fields (Pelliccioni, 1998). The depth at which secondary charged particle equilibrium is achieved is very important in these cases. For very high energy particles a depth of 10 mm in ICRU tissue, as defined with the operational quantities, is not sufficient to complete the charged particle build-up in front of that point and hence the operational quantities will underestimate effective dose. In radiation fields relevant for aircrew exposure, however, $H^*(10)$ appears to be an appropriate operational quantity if the recommended radiation weighting factors for neutrons and protons (see Section 3.5, paragraphs B 100 – B 123) are considered (Pelliccioni, personal communication).

(B 165) ***Directional dose equivalent, $H'(d,\Omega)$***. For area monitoring of low-penetrating radiation, the operational quantity is the directional dose equivalent, $H'(0.07, \Omega)$ or, in rare cases, $H'(3, \Omega)$ defined as follows:

- The *directional dose equivalent*, $H'(d, \Omega)$, 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 a depth, d, on a radius in a specified direction Ω.
- For low-penetrating radiation it is $d = 0.07$ mm, and $H'(d, \Omega)$ is then written $H'(0.07, \Omega)$.

(B 166) In the case of monitoring the dose to the lens of the eye, $H'(3, \Omega)$ with $d = 3$ mm was recommended by ICRU. The quantities directional dose equivalent, $H'(3, \Omega)$, and personal dose equivalent $H_p(3)$, however, have rarely been used in practice and very few instruments exist for measuring these quantities. It is suggested that their use is discontinued because the monitoring of the exposure to the eye lens is also sufficiently achieved if the dose to the eye lens is assessed in terms of the other operational quantities. $H_p(0.07)$ is normally used for this special purpose (ICRU 1998).

(B 167) For area monitoring of low-penetrating radiation, $H'(0.07, \Omega)$ is almost exclusively used. With unidirectional radiation incidence, mainly occurring in calibration procedures, the quantity may be written $H'(0.07,\alpha)$, where α is the angle

between the direction Ω and the direction opposite to radiation incidence. In radiological protection practice the direction Ω is often not specified, because it is mostly the maximum value of $H'(0.07, \Omega)$ at the point of interest which is of importance. It is usually obtained by rotating the dose rate meter during the measurement and looking for the maximum reading.

Operational quantities for individual monitoring

(B 168) Individual monitoring of external exposure is usually performed with personal dosimeters worn on the body, and the operational quantity defined for this application takes this situation into account. The true value of the operational quantity is determined by the irradiation situation near the point where the dosimeter is worn. The operational quantity for individual monitoring is the personal dose equivalent, $H_p(d)$.

(B 169) The *personal dose equivalent*, $H_p(d)$, is the dose equivalent in ICRU (soft) tissue at an appropriate depth, d, below a specified point on the human body. The specified point is usually given by the position where the individual dosimeter is worn. For the assessment of effective dose a depth $d = 10$ mm is recommended, and for assessing equivalent dose to the skin, and to the hands and feet, a depth $d = 0.07$ mm. In special cases of monitoring the dose to the lens of the eye, it has been proposed that a depth $d = 3$ mm would be appropriate (cf. paragraph B 166).

(B 170) An operational quantity for individual monitoring should allow the effective dose to be assessed or should provide a conservative estimate under nearly all irradiation conditions. This, however, requires that the personal dosimeter be worn at a position on the body which is representative with respect to the exposure. For a dosimeter position in front of the trunk, the quantity $H_p(10)$ mostly furnishes a conservative estimate of E even in cases of lateral or isotropic radiation incidence on the body. In cases of exposure from the back only, however, a dosimeter worn at the front side and correctly measuring $H_p(10)$, will not appropriately assess E. Also in cases of partial body exposures the reading of a personal dosimeter may not provide a representative value for the assessment of effective dose.

B.4.2. Internal exposure

(B 171) The system of dose assessment for intakes of radionuclides that is generally applied relies first on the calculation of the intake of a radionuclide either from direct measurements (e.g., measuring the radioactivity of the whole body by whole body counter or of specific organs and tissues by external counting devices) or indirect measurements (e.g., measuring the radioactivity in urine, faeces, air or other environmental samples). Biokinetic models have to be applied and the effective dose is calculated from the intake using reference dose coefficients (doses per unit intake, Sv Bq^{-1}) recommended by the Commission, and also reproduced in the EU Basic Safety Standards Directive (EU, 1996) and in the International Basic Safety Standards (IAEA, 1996). The Commission has provided dose coefficients for intakes by inhalation and ingestion for a large number of radionuclides, relating the intake of a specific radionuclide to the corresponding organ and effective dose committed

within a specified period (ICRP, 1994b, 1996c). Dose coefficients have been given for members of the public and for adults who are occupationally exposed.

(B 172) A paper by Berkovski et al. (2003) indicated that an alternative approach may be more useful in some circumstances. There can be advantages in calculating the committed effective dose *directly* from the measurements using functions that relate them to the time of the intake. The measurements could be the whole body or organ content, urine or faecal sample, or even an environmental measurement. This approach would require that the Commission provides additional tables of 'dose per unit content' as a function of time after the intake for interpreting the measurement data, but this approach should facilitate the interpretation of monitoring data in many circumstances. It aids the analysis by ensuring that current models are used in the dose assessment and limits the opportunity to make errors by reading data from tables.

B.4.3. References, Section B.4

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B.5. Practical application of dose quantities in radiological protection

(B 173) The main areas of application of dose quantities in radiological protection of both occupational workers and the general public to exposures from controlled sources are as follows:

- prospective dose assessment for planning and optimisation of protection; and
- retrospective dose assessment for demonstrating compliance with dose limits.

(B 174) In practice, limits, constraints, reference values, and action levels are defined in terms of dose quantities in order to restrict the risks from radiation exposure for both occupational workers and the public. The primary dose limits in radiological protection are given in terms of equivalent dose or effective dose. Since neither quantity can be directly measured, as has been explained above, they are assessed using other measurable quantities, models and computations (Figs. B.1 and B.2). Depending on the situation considered (occupational or public exposure), different procedures are applied.

B.5.1. Radioactivity and committed dose

(B 175) Calculations of the radiation dose from internal or external exposure to radiation emitted from radionuclides require information on their half-life and the type, energies, and intensities of the nuclear and atomic radiations emitted by the radionuclide. The data of *Publication 38* (ICRP, 1983b) are the same as have been used in ICRP publications since 1980. The strategy for preparing a database of nuclear decay data to replace *Publication 38* has been outlined by Endo et al. (2003, 2005). This database will be used in future calculations of dose coefficients.

(B 176) The activity A of an amount of a radionuclide in a particular energy state at a given time is the quotient of $\mathrm{d}N$ by $\mathrm{d}t$, where $\mathrm{d}N$ is the expectation value of the number of spontaneous nuclear transitions from that energy state in the time interval $\mathrm{d}t$, that is:

$$A = -\frac{\mathrm{d}N}{\mathrm{d}t} \tag{B.5.1}$$

The SI unit of activity is s^{-1} with the special name becquerel (Bq), 1 Bq = 1 s^{-1}

(B 177) Radionuclides are frequently included in or absorbed onto other solid, liquid, or gaseous material as well as being accompanied by stable isotopes of the same element, and the amount is defined by further quantities.

(B 178) The *specific activity* a_m (also called *massic activity* or *activity divided by mass* or *activity per mass*) of a specified radionuclide in a sample is the activity A of the radionuclide in the sample divided by the total mass m of the sample.

(B 179) The *activity concentration* a_v (also called *volumic activity* or *activity divided by volume* or *activity per volume*) of a specified radionuclide in a volume is the activity A of the radionuclide in the volume divided by the volume V.

(B 180) The *surface activity concentration* a_F (also called *areal activity concentration* or *aeric activity*) of a specified radionuclide on a surface is the activity A of the radionuclide on the surface area F divided by the area.

(B 181) The names and symbols of these three quantities have not been consistently standardised and there are some differences between the definitions used by different international bodies, including ICRU (ICRU, 2001b), ISO (ISO, 1992), IEC (IEC 2005), and ICRP. Harmonisation would be very helpful to avoid errors and inconsistencies.

(B 182) The activity intake, I, is the amount of the specified radionuclide entering the human body by ingestion, inhalation, or absorption through the skin. This intake is often used as an operational quantity for the assessment of effective dose. In general, it cannot be measured directly, and must be determined from other data such as whole or partial body measurements, assessments of activity in excreta or environmental measurements such as air samples (Fig. B.1). In the case of accidents, activity may also enter the body through wounds. A model to describe entry into the body through wounds, and subsequent uptake to blood, has been described by NCRP (2006).

(B 183) Radionuclides incorporated in the human body irradiate tissues over time periods determined both by their physical half-life and their biological retention within the body. Thus they may give rise to doses to body tissues over very short periods or throughout life. For example, in the case of intakes of tritiated water, because of its short biological half-time of retention (10 days; physical half-life of 12.3 yr), essentially all the dose is delivered within 2–3 months after intake. For ^{239}Pu, however, both biological retention times and the physical half-life (24,000 yr) are very long, and dose will be accumulated over the remaining lifespan of the individual. Thus, for inhalation of ^{239}Pu as plutonium nitrate (a Type M form in the Human Respiratory Tract Model, HRTM, ICRP, 1994a) models predict that only about 10% of the committed effective dose is received within the first year and about 30% by the end of 10 years. These and other examples are shown in Fig. B.5. The figure also shows the different rates of accumulation of committed equivalent doses to different tissues after inhalation of insoluble thorium–232 (Type S).

(B 184) The need to regulate exposures to radionuclides and the accumulation of radiation dose over extended periods of time led to the definition of committed dose quantities. The committed dose from an incorporated radionuclide is the total dose expected to be delivered within a specified time period. The *committed equivalent dose*, $H_T(\tau)$, in a tissue or organ T is defined by

$$H_T(\tau) = \int_{t_0}^{t_0+\tau} \dot{H}_T(t) \tag{B.5.2}$$

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

$$E(\tau) = \sum_T w_T H_T(\tau) \tag{B.5.3}$$

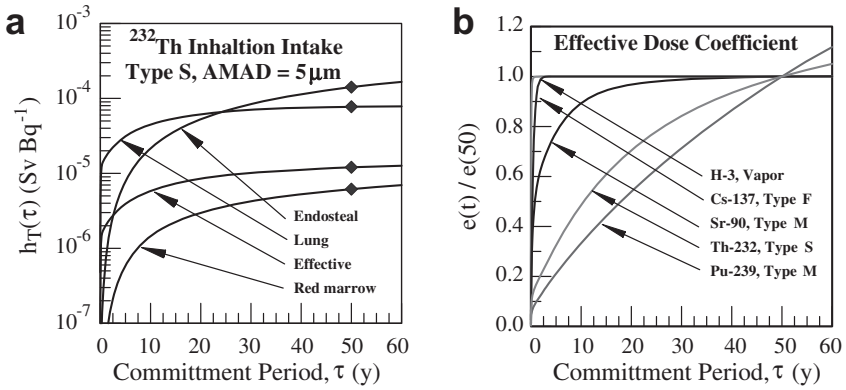

Fig. B.5. The committed dose coefficients as a function of integration period. (a) Committed equivalent dose coefficients in endosteal tissue, lung, and active (red) bone marrow, and committed effective dose coefficient as a function of integration time (τ) following inhalation intake of ^{232}Th. Points mark the 50 year period. (b) Committed effective dose coefficient for selected radionuclides normalised to their value at 50 y.

The Commission continues to recommend that, for compliance with dose limits and management of staff, the committed dose is assigned to the year in which the intake occurred.

(B 185) For workers, the committed dose is normally evaluated over the 50-year period following the intake. The commitment period of 50 years is a rounded value considered by the Commission to be the life expectancy of a young person entering the workforce. The committed effective dose from intakes is also used in prospective dose estimates for members of the public. In these cases a commitment period of 50 years is considered for adults. For infants and children the dose is evaluated to age 70 years (ICRP, 1996c).

B.5.2. Reference phantoms

(B 186) Effective dose is defined for the sex-averaged Reference Person (Section B.3.4). To determine effective dose, first the equivalent doses in the organs and tissues of the reference male and the reference female have to be evaluated and then averaged in order to obtain the equivalent doses of the Reference Person. Effective dose is obtained by multiplying these with sex-averaged tissue weighting factors and summing over all tissue weighted equivalent doses of the Reference Person (Eqn. B.3.7; Fig. B.3).

(B 187) The evaluation of equivalent doses for the Reference Male and Female, and of effective dose for the Reference Person, is based on the use of anthropomorphic models. In the past, the Commission did not specify a particular phantom and, in fact, various mathematical phantoms such as hermaphrodite MIRD-type phantoms, the sex-specific models of Kramer et al. (1982) or the age-specific phantoms of Cristy and Eckerman (1987) have been used.

(B 188) The Commission has now adopted male and female reference phantoms for the calculation of equivalent doses for organs and tissues. In order to provide

a practicable approach for the assessment of equivalent doses and effective dose, conversion coefficients relating to physical quantities, e.g., particle fluence or air kerma for external exposure, and activity intake for internal exposure, are calculated for standard exposure conditions of the reference phantoms (mono-energetic radiations, standard geometries for external irradiations, standard biokinetics of radionuclides in the human body, etc.).

(B 189) Voxel (voxel: volume element) models, constructed from medical image data of real persons, give a more realistic description of the human body than the mathematical, stylised phantoms. Thus, the Commission decided to use voxel models to define its reference phantoms to be used for the update of organ dose conversion coefficients. These models (or computational phantoms) represent the Reference Male and Female, and have organ masses in compliance to the reference values, compiled in *Publication 89* (ICRP, 2002).

(B 190) Two voxel reference models of an adult male and an adult female have been developed (Zankl et al., 2005, Zankl et al., 2007), based on voxel models of two individuals whose body height and weight were close to those of the Reference Male and Female. They were developed from computed tomographic images obtained from high resolution continuous scans of a single individual and consist of millions of voxels, providing a three-dimensional representation of the human body and the spatial form of its constituent organs and structures. Approximately 140 organs and tissues were defined, including different skeletal tissues, cartilage, muscle and the main blood vessels. The organ masses of both models were adjusted to approximate those assigned to the Reference Adult Male and Female in *Publication 89* (ICRP, 2002) without distorting the realistic anatomy.

(B 191) The voxel reference models are thus computational representations of Reference Male and Female and can be used, together with codes simulating the radiation transport and energy deposition, for the computation of dose coefficients for workers and adult members of the public for internal exposure. The models can be used to compute the fraction of the energy of radiation emitted within source region S_i that is absorbed in target region T_j. Similarly the models will be used to compute the mean absorbed dose, D_T, in an organ or tissue T, from radiation fields external to the body, and the relationship of the effective dose to the quantities specific to the radiation field. Reference computational phantoms for children of different ages will also be developed for use in the calculation of dose coefficients for members of the public.

B.5.3. Committed effective dose coefficients for internal exposure

(B 192) In the occupational setting, each intake of a radionuclide during a year is assigned a committed effective dose, $E(\tau)$, where a commitment period τ of 50 years is considered for workers. The same period is chosen for adult members of the public, while for infants and children the dose rate is integrated up to the age of 70 years (ICRP, 1996c).

(B 193) Committed effective dose coefficients, $e(\tau)$, are conversion coefficients for a Reference Person which provide numerical links between $E(\tau)$ and measurable quan-

tities, in this case between $E(\tau)$ and the intake of radionuclide(s) by either inhalation (e_{inh}) or ingestion (e_{ing}) of radionuclides. The dose coefficients for the female and male are based on the sex-specific physiological, anatomical, and biokinetic parameters of the reference adult females and males. In addition, the dosimetric parameters in the evaluation of the mean absorbed dose in tissue T are derived for sex-specific computational phantoms (see Section B.5.2).

(B 194) The contribution of the remainder tissue to the effective dose is derived by applying the tissue weighting factor for this group of tissues to the arithmetic average equivalent dose among tissues not assigned an explicit tissue weighting but listed as remainder tissues (see Section B.3.5, paragraphs B 132 – B 145). The dose to remainder tissues is evaluated in a manner that provides for additivity of the effective dose.

(B 195) Thus, committed effective dose coefficients based on the sex and population-averaged tissue weighting factors given in Table B.2, should be computed as

$$e(\tau) = \sum_{\mathrm{T}} w_{\mathrm{T}} \left[\frac{h_{\mathrm{T}}^{\mathrm{M}}(\tau) + h_{\mathrm{T}}^{\mathrm{F}}(\tau)}{2} \right] \tag{B.5.4}$$

where $h_{\mathrm{T}}^{\mathrm{M}}(\tau)$ and $h_{\mathrm{T}}^{\mathrm{F}}(\tau)$ are the committed equivalent dose coefficients for tissue T of the Reference Male and Reference Female, respectively (Fig. B.3). An analogous equation is applicable to external exposures.

B.5.4. Conversion coefficients for external exposure

(B 196) As described in Section B.4 the protection quantities, equivalent dose and effective dose, are not measurable, and their values are assessed using their relationship to either physical radiation field quantities, e.g., air kerma free in air, K_{a}, or particle fluence, ϕ, or operational dose quantities. Conversion coefficients defined for a reference person provide numerical links between these quantities, and it is very important that an internationally agreed set of conversion coefficients is available for general use in radiological protection practice for occupational exposures and exposures of the public.

(B 197) Based on the work of a joint ICRU/ICRP task group, the Commissions published reports (ICRP, 1996b, ICRU, 1997) on 'Conversion coefficients for use in radiological protection against external radiation' which recommended a set of evaluated data of conversion coefficients for protection, and operational quantities for external exposure to mono-energetic photon, neutron, and electron radiation under specific irradiation conditions. Most of the data for protection quantities used for the evaluation were calculated on the basis of MIRD-like models of the anatomy. In all cases, whole body exposure was assumed. For photons the mean absorbed dose in an organ or tissue per air kerma free in air and the effective dose per air kerma free in air are given, while for neutrons and electrons the doses are related to the particle fluence. Furthermore, *Publication 74* (ICRP, 1996b) explored in detail the relationship between the protection quantity effective dose and the operational dose quantities for specific idealised irradiation exposure geometries. Partial body

exposures were not discussed in that publication, and recommended conversion coefficients are not available for these cases.

(B 198) The definition of new reference phantoms for the human body (male and female voxel phantoms based on medical imaging data) requires the calculation of a new set of conversion coefficients for all types of radiation and irradiation geometries considered. For most organs, however, the differences from the existing data of $D_{T,R}$ (ICRP, 1996b) are likely to be moderate. The values of the coefficients for effective dose are also dependent on w_R and w_T values, and their changes may have a larger influence on the changes in conversion coefficients, especially for neutrons and protons.

(B 199) The adoption of voxel-based reference phantoms (ICRP, 2002) requires new calculations of conversion coefficients for all radiations and irradiation geometries of interest which will replace the existing data sets (ICRP, 1996b). Calculations for photons have shown that the changes in the values of effective dose for photon radiation are generally small (Zankl et al., 2002). At low photon energy, however, the change in exterior shape of the body and hence the depth of an organ in the reference phantoms can influence the absorbed dose, e.g., for the thyroid. The resultant change in the effective dose coefficients is expected to be rather modest (Schlattl et al., 2007).

B.5.5. Occupational exposure

(B 200) In cases of occupational exposure, doses may arise from external and internal radiation sources. For external exposure individual dose monitoring is usually performed by measuring the personal dose equivalent $H_p(10)$ using a personal dosimeter and taking this measured value as an acceptable assessment of the value of effective dose under the assumption of a uniform whole-body exposure. For internal exposure committed effective doses are determined based on assessment of intakes of radionuclides from bioassay measurements or other quantities (e.g., activity retained in the body or in daily excreta – in exceptional cases the airborne activity concentration can be used) and the application of appropriate dose coefficients.

(B 201) For practical purposes the values from both kinds of quantities should be combined in the assessment of the value of total effective dose for demonstrating compliance with dose limits and constraints.

(B 202) In most situations of occupational exposure the effective dose, E, can be derived from operational quantities using the following formula:

$$E \cong H_p(10) + E(50) \qquad \text{(B.5.5)}$$

where $H_p(10)$ is the personal dose equivalent from external exposure (see Section B.4.4) and $E(50)$ the committed effective dose from internal exposure.

(B 203) For the assessment of effective dose from external exposure, according to Eqn. (B.5.5) by monitoring the exposure with a personal dosimeter measuring $H_p(10)$ it is necessary that the personal dosimeter is worn at a position on the body which is representative of the exposure of the body. If the measured dose value is well below the annual dose limit, the value of $H_p(10)$ is usually taken as a sufficient

estimate of effective dose. For high personal doses approaching or exceeding the annual dose limit, or in strongly inhomogeneous radiation fields, however, this procedure might not be sufficient and it would then be necessary to carefully consider the actual situation of exposure in the human body in assessing the effective dose. The use of personal protective equipment (PPE) and other protection measures may also need to be taken into account.

(B 204) In the special case of exposure of aircrew to cosmic radiation, individual monitoring with personal dosimeters, measuring $H_p(10)$, is usually not performed for the assessment of effective dose. There may be other working environments in which personal dosimeters are not used. In these cases the effective dose from external exposure can be assessed from monitoring ambient dose equivalent, $H^*(10)$, or by calculation using radiation field properties.

(B 205) In cases of external exposure to low-penetrating radiation, e.g., β-rays, $H_p(10)$ will not sufficiently assess effective dose. In such cases $H_p(0.07)$ may be used to assess the equivalent dose to the skin and its contribution to effective dose by applying the tissue weighting factor of 0.01 for the skin.

(B 206) The new computational phantoms will be used to compute the equivalent dose in tissue T, H_T, from radiation fields external to the body and the relationship of the effective dose to the operational quantities specific to the radiation field. Conversion coefficients representing the effective dose per unit fluence or air kerma as a function of radiation energy need to be calculated for various irradiation geometries and will be applicable to external exposures at the workplace. The same reference computational phantoms will also be used to derive dose coefficients for equivalent dose, H_T in relevant target regions as well as for effective dose.

(B 207) In cases of external exposure to beta particles, very inhomogeneous irradiation of the body will occur. Even at effective doses below the limits high local skin doses could occur where tissue reactions are possible. For this reason the annual limit on skin dose (500 mSv for occupational exposure) corresponds to the local skin dose defined by the mean equivalent dose in 0.07mm depth averaged over any 1 cm^2 of the skin.

(B 208) The committed effective dose, $E(50)$, from intakes of radionuclides is assessed by:

$$E(50) = \sum_{j} e_{j,inh}(50) \cdot I_{j,inh} + \sum_{j} e_{j,ing}(50) \cdot I_{j,ing} \qquad \text{(B.5.6)}$$

where $e_{j,inh}(50)$ is the committed effective dose coefficient for activity intakes by inhalation of a radionuclide j, $I_{j,inh}$ is the activity intake of a radionuclide j by inhalation, $e_{j,ing}(50)$ is the committed effective dose coefficient for activity intakes of a radionuclide j by ingestion, and $I_{j,ing}$ is the activity intake of a radionuclide j by ingestion. In the calculation of the effective dose from specific radionuclides, allowance will need to be made for the characteristics of the material taken into the body.

(B 209) The dose coefficients used in Eqn. (B.5.6) are those specified by the Commission with no departure in anatomical, physiological, and biokinetic characteristics from those of the Reference Male and Reference Female (ICRP, 2002). Account may, however, be taken of the activity medium aerodynamic diameter

(AMAD) of the inhaled aerosol, and the chemical form of the particulate matter to which the specified radionuclide is attached. The effective dose assigned in the worker's dose record, the 'dose of record', is that value of effective dose which the Reference Person would experience owing to the radiation fields and activity intakes encountered by the worker (see Section 5.8). The commitment period of 50 years relates to the life expectancy of a person entering the workforce, as noted in Section B.5.1.

(B 210) The radiation dose from radon isotopes and their decay products may also need to be taken into account in the overall dose assessment (ICRP, 1993b). If incorporation of radionuclides through the skin occurs, an additional term for the associated effective dose would have to be included in Eqn. (B.5.6). The incorporation of radionuclides through uncontrolled events involving wounds has implications beyond compliance with work practices, and thus these events are not included in Eqn. (B.5.6). The significance of these events must be evaluated and recorded, appropriate medical treatment provided, and further restriction of the worker's exposure considered if warranted.

(B 211) Exposure to airborne noble gas radionuclides in the workplace may need to be assessed beyond that indicated by $H_p(10)$. In such cases it is necessary to include in Eqn. (B.5.6) a term representing the product of the time-integrated airborne concentration of the noble gas and an effective dose coefficient for so-called submersion exposure. Such dose coefficients are specified by the Commission for both prospective and retrospective applications.

(B 212) In the assessment of committed effective doses for workers from operational data related to an actual intake of specific radionuclide(s) or of radionuclide concentration(s) in the air at a workplace it is often useful to refer these data to the Annual Limit on Intake (ALI) and the Derived Air Concentration (DAC).

(B 213) The ALI was defined in *Publication 60* (ICRP, 1991b, paragraph S30) as the activity intake (Bq) of a radionuclide which would lead to an effective dose corresponding to the annual limit $E_{\mathrm{limit,w}}$, under the expectation that the worker is exposed to only this radionuclide. The ALI of radionuclide j is:

$$\mathrm{ALI_j} = \frac{E_{\mathrm{limit,w}}}{e(50)} \tag{B.5.7}$$

where $e(50)$ is the corresponding reference committed effective dose coefficient in (Sv Bq^{-1}). The Commission recommended in *Publication 60* that the ALI should be based on the dose limit of 0.020 Sv in a year, with no time averaging.

(B 214) The DAC is the activity concentration in air in Bq m^{-3} of the radionuclide considered, which would lead to an intake of an ALI (Bq) assuming a gender-averaged breathing rate of 1.1 $\mathrm{m}^3\,\mathrm{h}^{-1}$ and an annual working time of 2000 h (an annual air intake of 2200 m^3). Then the DAC of radionuclide j is given by:

$$\mathrm{DAC_j} = \frac{\mathrm{ALI_j}}{2200} \tag{B.5.8}$$

(B 215) The Commission does not now give ALI values, because it considers that, for compliance with dose limits, it is the total dose from external radiation as well as from intakes of radionuclides that must be taken into account as indicated above. It is, however, noted that the ALI concept can be useful in various practical situations, e.g., in characterising the relative hazard of radiation sources to ensure that appropriate administrative controls are in place.

(B 216) The DAC for inert gases which are not incorporated is limited by the effective dose arising from radiations incident on the body from the airborne activity. Thus the DAC is given by

$$\mathrm{DAC} = \frac{E_{\mathrm{limit,w}}}{2000\,\dot{e}_{\mathrm{sub}}} \tag{B.5.9}$$

where $\dot{e}_{\mathrm{sub}}$ is the effective dose rate coefficient [mSv m^3(Bq h)$^{-1}$] for submersion in an airborne cloud containing the noble gas radionuclide and 2000 h is the annual working time. For some radionuclides the DAC is limited by the dose to the skin.

B.5.6. Public exposure

(B 217) Public exposures can occur from natural radiation sources, which may be modified by human activities, from technical installations, or from combinations of such sources. The annual effective dose to members of the public is the sum of the effective dose obtained within one year from external exposure and the committed effective dose from incorporated radionuclides within this year. The dose is usually not obtained by individual monitoring as for occupational exposure but is mainly determined by environmental measurements, habit data, and modelling. It can be estimated from:

- Simulation and prediction of radionuclide levels in effluents from the technical installation or source during the design period;
- Effluent and stray radiation monitoring during the operational period; and
- Radioecological modelling (pathway analysis of environmental transport, e.g., from the release of radionuclides and transport through soil to plants to animals to humans).

(B 218) External exposures of individuals may occur from radionuclides released from installations and which are present in the air, soil, or water. Doses can be calculated from activity concentrations in the environment by modelling and computation.

(B 219) Internal exposures can occur by inhalation of airborne radionuclides from a cloud, inhalation of resuspended radionuclides, and by ingestion of contaminated food or water.

B.5.7. Medical exposures of patients

(B 220) The use of effective dose for assessing the exposure of patients has severe limitations that must be taken into account by medical professionals. Effective dose

can be of value for comparing doses from different diagnostic procedures – and in a few special cases from therapeutic procedures – and for comparing the use of similar technologies and procedures in different hospitals and countries as well as using different technologies for the same medical examination. Such data have been reviewed by UNSCEAR (1988, 2000). For planning the exposure of patients and risk-benefit assessments, however, the equivalent dose or preferably the absorbed dose to irradiated tissues is the more relevant quantity. This is especially the case when risk estimates are intended.

(B 221) Medical exposures of patients to external radiation are commonly concerned with only limited parts of the body and it is important that medical professionals are fully aware of the doses to normal tissue in the irradiated fields. With low tissue weighting factors for skin and relatively low values for a number of other body tissues partial body exposure can result in appreciable equivalent doses to local tissues even though the corresponding effective dose may be small. Similar considerations apply to doses from intakes of radionuclides.

B.5.8. Application of effective dose

(B 222) The main and primary use of effective dose is to provide a means of demonstrating compliance with dose limits. In this sense effective dose is used for regulatory purposes worldwide.

(B 223) Effective dose is used to limit the occurrence of stochastic effects (cancer and heritable effects) and is not applicable to the assessment of the possibility of tissue reactions. In the dose range below the annual effective dose limit tissue reactions should not occur. Only in a few cases (e.g., an acute localised exposure of a single organ with a low tissue weighting factor such as the skin) could the use of the annual limit on effective dose be insufficient to avoid tissue reactions. In such cases local tissue doses will also need to be assessed.

(B 224) The calculation of reference dose coefficients for intakes of radionuclides and dose conversion factors for external exposures is based on reference anatomical data for the organs and tissues of the human body together with defined biokinetic and dosimetric models. The general approach is to monitor individuals or the environment and from these measurement data to assess the external exposure or radionuclide intake. The dose coefficients and dose conversion factors published by the Commission are then used to assess the effective dose from the exposure or the intake. The weighting factors used in the calculation of reference dose coefficients and conversion factors apply to a population of both sexes and all ages. Thus dose coefficients, and the reference models and weighting factors used in their calculation, are not individual specific but apply to a Reference Person for the purposes of regulatory control. Conversion coefficients or dose coefficients are calculated for a reference adult worker or a reference member of the public of a defined age group.

(B 225) The effective dose of a worker assessed by the sum of the measured personal dose equivalent, $H_P(10)$, and the committed effective dose estimated from results of individual monitoring of the worker, and ICRP reference biokinetic and dosimetric computational models, is called *dose of record*. Dose of record is assigned

to the worker for purposes of recording, reporting, and retrospective demonstration of compliance with regulatory dose limits.

(B 226) Particularly in retrospective dose assessments for occupational exposures, information may be available that differs from the reference parameter values used in the calculation of dose conversion factors and dose coefficients. In such situations it may be appropriate, depending on the level of exposure, to use specific data in the assessment of exposure or the intake and calculation of doses. It is, therefore, important to distinguish between those parameter values that might be altered in the calculation of effective dose under the particular circumstances of an exposure and those values that cannot be changed under the definition of effective dose.

(B 227) In the assessment of effective dose in occupational situations of exposure to radionuclides, changes may reasonably be made to the physical and chemical characteristics of inhaled or ingested radionuclides to better assess intakes and exposures. These changes need to be notified. Examples of the use of material-specific data in the calculation of doses from inhaled radionuclides have been given in *Supporting Guidance 3* (ICRP, 2002).

(B 228) For retrospective assessments of occupational doses to specific individuals in situations where the radiation dose could exceed a limit or constraint, it may be considered appropriate to make specific individual estimates of dose and risk. Consideration might then be given to changes in dosimetric assumptions used to calculate absorbed doses, and organ-specific risk estimates relating to the age and sex of the individual and the radiation exposure. Such changes from reference parameter values are not consistent with the definition or intended use of effective dose. They should only be performed by radiation protection specialists, with the level of effort determined by the level of exposure. In such situations the changes of parameter values must be described.

(B 229) In cases of incidents and accidents that could give rise to tissue reactions (deterministic effects), it is necessary to estimate absorbed dose and dose rates to organs and tissues and to take into account dose–response relationships to assess the potential for radiation effects that are likely to occur above dose thresholds (NCRP, 1990; ICRP, 1989b). It should also be noted that, in cases of accidents involving high-LET radiations (neutrons and alpha particles), radiation weighting factors (w_R) applicable to stochastic effects do not apply to tissue reactions; values of relative biological effectiveness (RBE) relevant to tissue reactions should be used.

(B 230) Effective dose is a risk-related quantity based upon the consequences of whole body exposure. The w_T values are selected values that are chosen to take account of the contribution of individual organs and tissues to total radiation detriment from stochastic effects, in terms of cancer and heritable effects, on the basis of current epidemiological (or, for heritable effects, experimental) evidence. Furthermore, w_T values are averages applying to both sexes and all ages. While effective dose is sometimes used for pilot studies aimed at generation of hypotheses of effects of radiation on human health, it is not an appropriate quantity for use in epidemiological studies of radiation risks. Epidemiological analyses instead require estimates of absorbed doses to tissues and organs, taking full account, to the extent possible, of the circumstances of exposure and the characteristics of the exposed individuals in

the study population. Similarly, absorbed doses, not effective doses, are required for calculations of probability of causation of cancer in exposed individuals.

(B 231) In summary, effective dose should be used for assessing exposure and controlling stochastic effects for regulatory purposes. It can be used to demonstrate compliance with dose limits and for dose records. Effective dose provides a convenient quantity for the assessment of overall radiation exposure, taking account of all exposure pathways, internal and external, for dose record keeping and regulatory purposes. Used in this way effective dose is a valuable quantity for practical radiological protection purposes although it is not individual-specific but applies to a Reference Person. In retrospective situations the assessment of effective dose gives an insight into the quality of radiological protection and gives information on whether the dose limits could have been exceeded.

(B 232) However, there are situations in which the use of effective dose is not appropriate, and individual organ and tissue absorbed doses should be used instead. These include epidemiological studies, assessment of the probability of causation of cancer, assessments of the possibility of tissue reactions, or assessments of doses when treatment or medical surveillance are needed.

B.5.9. Collective dose

(B 233) The dosimetric quantities for radiological protection discussed above refer to a Reference Person. The task of radiological protection includes optimisation and the reduction of radiation exposure of groups of occupationally exposed persons or of the public. For this purpose ICRP has introduced the collective dose quantities (ICRP, 1977, 1991b) which should be used and understood as instruments for optimisation. These quantities take account of the group of persons exposed to radiation from a source and of a specified time period of exposure. The quantities have been defined as the collective equivalent dose, S_T, which relates to a tissue or an organ T, and the collective effective dose, S (ICRP, 1991b). The special name of the unit of these collective dose quantities is the man sievert (man Sv).

(B 234) Collective effective dose is defined in *Publication 60* (ICRP, 1991b) as the integral over effective doses received by the population (paragraph A34). The Commission introduced both the collective equivalent dose and the collective effective dose. Since the intent of the collective quantities is to serve as an instrument in optimisation of radiological protection especially for occupational exposures, and the collective equivalent dose is used only in special circumstances, only the collective effective dose is discussed in the present Recommendations.

(B 235) In occupational exposure, the quantity collective effective dose is used for optimisation of planned exposure situations of a group of workers. The collective effective dose, and the distribution of individual doses, is assessed prospectively for different operational scenarios before starting the planned work. Collective effective dose is then used as a relevant parameter in the decision process for the choice of the operational scenario. Comparison of the prospectively assessed collective effective dose, and the sum of all individual effective doses obtained from monitoring data

after the completion of the work, can provide relevant information for future optimisation procedures and radiation protection measures. Collective effective dose can also be used as an instrument for comparing radiological technologies in medical practices and comparing the same radiological technologies at different locations (e.g., different hospitals, different countries).

(B 236) The definition of collective quantities, as described above, has led people in some cases to use collective effective dose incorrectly for summing up radiation exposures over a wide range of doses, over very long time periods and over large geographical regions, and to calculate on this basis radiation-related detriments. However, such a use of collective effective dose would only be meaningful if there were sufficient knowledge of the risk coefficients for the detrimental radiation effects in all dose ranges which contribute to the collective dose (Kaul et al., 1987). Owing to the large uncertainties, such a knowledge of risk coefficients is not available in the very low dose range.

(B 237) In this context it has to be realised that the risk factors, e.g., for carcinogenesis at low doses, are obtained from the extrapolation of epidemiological data observed in dose ranges of medium and high radiation doses. As described in Section B.2, the extrapolation is based on the assumption of a linear dose effect relationship without a threshold (LNT model). The Commission considers that, in the low dose range, the risk factors have a high degree of uncertainty. This is particularly the case for very low individual doses which are only small fractions of the radiation dose received from natural sources. The use of collective effective dose under such conditions for detailed risk estimates is not a valid procedure.

(B 238) To avoid aggregation of low individual doses over extended time periods and wide geographical regions the range in effective dose and the time period should be limited and specified. The collective effective dose due to individual effective dose values between E_1andE_2 for the time period ΔT is defined as:

$$S(E_1, E_2, \Delta T) = \int_{E_1}^{E_2} E \left[\frac{\mathrm{d}N}{\mathrm{d}E}\right]_{\Delta T} \mathrm{d}E \tag{B.5.10}$$

The number of individuals experiencing an effective dose in the range E_1 to E_2, $N(E_1, E_2, \Delta T)$ is:

$$N(E_1, E_2, \Delta T) = \int_{E_1}^{E_2} \left[\frac{\mathrm{d}N}{\mathrm{d}E}\right]_{\Delta T} \mathrm{d}E \tag{B.5.11}$$

and the average value of effective dose $\bar{E}(E_1, E_2, \Delta T)$ in the interval of individual doses between E_1 and E_2 for the time period ΔT is:

$$\bar{E}(E_1, E_2, \Delta T) = \frac{1}{N(E_1, E_2, \Delta T)} \int_{E_1}^{E_2} E \left[\frac{\mathrm{d}N}{\mathrm{d}E}\right]_{\Delta T} \mathrm{d}E \tag{B.5.12}$$

(B 239) For a group of individuals, the collective effective dose S could also be calculated by:

$$S = \sum_{\mathrm{i}} E_{\mathrm{i}} N_{\mathrm{i}} \tag{B.5.13}$$

where E_i is the average effective dose in the subgroup i, and N_i is the number of individuals in this subgroup (ICRP, 1991b).

(B 240) In the calculation and interpretation of collective effective dose, the following aspects should be considered and critically reviewed in order to avoid a misuse of collective effective dose:

- Number of exposed individuals;
- Age and sex of exposed persons;
- Range of individual doses;
- Dose distribution in time; and
- Geographical distribution of exposed individuals.

B.5.10. References, Section B.5

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B.6. Uncertainties and judgements in radiological protection

(B 241) In *Publication 60* (ICRP, 1991b) the Commission stressed, as has been done in this document, that the assessment of radiation dose is fundamental to radiological protection, although neither the equivalent dose in an organ or tissue nor the effective dose can be measured directly. In the evaluation of these doses, models are necessary to simulate the geometry of the external exposure, the biokinetics of the intake and retention of radionuclides in the human body, and the human anatomy. Dosimetric considerations with respect to methodology and practical use are also of great importance.

(B 242) These models and their parameter values have been developed in many cases from experimental investigations and human studies in order to derive 'best estimates' of model parameter values. It is recognised that there may be large uncertainties in the values of some of the parameters and in the formulation or structures of the models themselves. Some of these uncertainties have been addressed in various publications (Leggett et al., 1998, ICRP, 2002, Harrison et al. 2001, Likhtarev et al., 2003) and estimates of the illustrated variability of parameter values, e.g., for physiological and anatomical characteristics have been demonstrated (ICRP, 2002). Such variations of parameter values are of particular significance with respect to the models necessary for dose assessments from internal exposure. From situations with a broad range of values the necessary parameters are selected by judgements in order to evaluate weighting factors and other parameters for the dose assessment.

(B 243) It is important to distinguish between uncertainty and variability. Uncertainty refers to the level of confidence that can be placed in a given parameter value or prediction of a model or estimate of the central value of dose for a population. Uncertainties of the measurements in the low ranges of the determined parameters are included. It is an important factor in all extrapolation procedures and particularly in assessing radiation doses and their effects in the low dose range.

(B 244) Variability (strictly, biological variability) refers to quantitative differences between different members of the population in question, e.g., with respect to their physiological and metabolic parameters. For example, two healthy persons of the same age and sex and having identical diets may exhibit substantially different rates of transit of material through the colon. Similarly individual members of a population will show substantial variation in the uptake of radioiodine by the thyroid for the same initial intake. Variability will be an important source of uncertainty in the estimate of a central value when the estimate is based on a few, highly variable observations.

(B 245) Risk factors for stochastic effects, from which w_R and w_T values are derived, have been obtained from epidemiological and experimental radiobiological data in the medium and higher dose ranges. The risk factors for the lower dose ranges that are important for radiological protection as well as the concept of effective dose, are based on extrapolation from the measured data in the higher dose ranges using the linear-non-threshold model (LNT model).

(B 246) This model is an assumption which has not been scientifically validated. It is considered to be the most appropriate interpretation of current experimental and

epidemiological data and is consistent with current understanding of stochastic radiation effects. However, its use also introduces a high degree of uncertainty, especially in relation to exposures at low doses and low dose rates (UNSCEAR, 2000). The assumed linearity of dose–response, and the additivity of doses are necessary conditions for the concepts used in radiological protection in the low dose ranges, especially for the use of effective dose, as described in previous sections.

(B 247) The uncertainties which are associated with the assessment of radiation doses and health detriments have been discussed in various sections of this document. Some of the more important factors considered are:

- The heterogeneity of energy deposition within tissues has been described in the low dose ranges of external as well as of internal exposures (Section B.3.2).
- The heterogeneous distribution of radionuclides has been described in the body and in tissues which is especially significant when considering ionising particles with short ranges such as alpha particles (Sections B.3.2, B.3.3).
- For dose assessments from internal exposures, the biokinetic models and their parameter values are variable and dependent on the specific conditions of exposure. Frequently, animal data have to be used and to be extrapolated to humans.
- Human populations vary worldwide on ethnic grounds with respect to physiological and other parameters (ICRP, 2002). Variability can become large when radioecological models are used to assess concentrations of radionuclides in food, and hence intakes from habit data as the parameters are frequently very uncertain, biological variability is large, and measured activity values are frequently low.
- The RBE values which are important for the choice of w_R values vary with the end point considered and the experimental design. Frequently the values rely on animal and in-vitro data (Section B.3.5, paragraphs B 73 – B 131).
- The target cells for the induction of cancer and their location in tissues are unclear. The dose response in the low dose range for stochastic effects, the mode of extrapolation and the LNT model are uncertain (Annex A).
- For the estimation of parameters connected to the assessment of health detriments, sex averaging is performed which causes uncertainty (Section B.3.4).

(B 248) The degree of uncertainty varies for the various parameters and different circumstances in defined exposure situations. Therefore, it is not possible to give general values of uncertainties, but considerations of this kind should be and have been made for special cases and should be included in comprehensive evaluations (e.g., CERRIE, 2004, ICRP, 2006c). In general it can be said that uncertainties in the assessment of radiation doses from internal exposures including the biokinetics of radionuclides are larger than those from external exposures. The degree of uncertainty differs between various radionuclides.

(B 249) The Commission is aware of these uncertainties, and efforts are being undertaken to critically evaluate and to reduce them wherever possible. However, for prospective dose assessments in regulatory processes the Commission takes the position that the dosimetric models, as well as the parameter values that it recommends for determining doses from quantitative information about radiation fields at working places and in the environment or from intakes of radionuclides, should

be taken as reference models. These values have been fixed by convention and are not subject to uncertainty.

(B 250) Equally the Commission considers that the dosimetric models and parameter values which are needed for the purpose of recommending dose limits or constraints are defined as reference data and, therefore, are not uncertain. Nevertheless, these models and values are re-evaluated periodically and may be updated by ICRP on the basis of such evaluations when new scientific data and information become available.

(B 251) It should be noted that the dosimetric models, conversion coefficients, and other parameters recommended by the Commission have been developed principally and primarily for planning and assessing normal occupational exposures, for planning for discharges into the environment and for generic assessments of doses. They are needed to demonstrate compliance with dose limits. These are circumstances in which doses are low (Section B.5.5). At higher doses, for example following accidental exposures, or for epidemiological studies, more specific information on the individual and the exposure conditions are needed. In such situations all sources of uncertainty should be taken into consideration including the variability of individual anatomical and physiological data, specific information on radionuclide source-term, biokinetics, and the direction of radiation incidence in cases of external exposure.

(B 252) In conclusion, the reference models and their parameter values have been developed for use in prospective radiological protection. These models and parameter values are also used for demonstrating compliance with dose limits when exposures are low but in general should not be used for individual risk estimates or for epidemiological studies. In cases where this is done the uncertainty must be critically reviewed. If such individual data are not available the reference parameters may be used but this must be clearly documented. This limitation of usage applies particularly to effective dose. For the assessment and judgement of individual cases absorbed doses to organs or tissues should be used together with the most appropriate biokinetic parameters, data on biological effectiveness of the ionising radiation and risk coefficients. In these cases uncertainties should be taken into consideration.

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