APPROACHES TO ATTRIBUTION OF DETRIMENTAL HEALTH EFFECTS TO OCCUPATIONAL IONIZING RADIATION EXPOSURE AND THEIR APPLICATION IN COMPENSATION PROGRAMMES FOR CANCER

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Approaches to attribution of detrimental health effects to occupational ionizing radiation exposure and their application in compensation programmes for cancer
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Ionizing radiation is part of the human environment (e.g. cosmic rays and naturally occurring radioactive materials). It includes X-rays and gamma rays (i.e. electromagnetic radiation) as well as corpuscular radiation (i.e. subatomic particles: alpha, beta and neutron radiations). Radioactive sources are used throughout the world for a wide variety of beneficial purposes, e.g. in industry, medicine, research, agriculture and education. Worldwide, 6.5 million workers are monitored for their occupational exposure to ionizing radiation. Among them 800,000 are workers in the nuclear fuel cycle.

The use of these radioactive sources involves risks associated to radiation exposure. Ionizing radiation can induce acute effects cell killing extensive enough to imply functional impairment of tissues and/or organs. These effects, which are called “non-stochastic” or deterministic, are observable if the dose exceeds a certain level (threshold). Ionizing radiation can also induce non-lethal transformation of a cell, which may result in long-term effects called “stochastic” effects (e.g. cancer and hereditary effects).

In view of the growing number of work situations in which ionizing radiation is found, it is increasingly important that workers are adequately protected. Radiation protection is part of the fields of the ILO’s action on the protection of workers against sickness, disease and injury arising out of their employment, as mandated by the Organization’s Constitution. In June 1960, the International Labour Conference adopted the Convention concerning the Protection of Workers against Ionising Radiations (No. 115), and its accompanying Recommendation (No. 114).

When protection and safety are not adequately or properly implemented, workers can be injured or diseased due to exposure to ionizing radiation at the workplace. Diseases caused by work have to be identified and their victims properly compensated. The relationship between exposure and the severity of the impairment among workers and the number of workers exposed are important criteria for the determination of occupational diseases. In 1964, the International Labour Conference adopted the Employment Injury Benefits Convention (No. 121), and its accompanying Recommendation (No. 121). Convention No. 121 is appended with a separate schedule which contains a list of occupational diseases giving entitlement to benefit. Diseases caused by ionizing radiation at the workplace are included in the list.

With growing public awareness of the presumed health risks associated with the use of nuclear energy, there is an increasing number of claims for compensation by workers (or their relatives) in whom cancer may be attributable to exposure to ionizing radiation at work. The causal relationship between exposure to ionizing radiation and deterministic health effects is relatively easy to establish. Cancer is a frequent disease and many factors contribute to its development. In the absence of a radiation hallmark to identify exactly those individuals who have been principally affected by their occupational exposure to ionizing radiation, it will be difficult to distinguish those cancers attributable to
occupational radiation exposure from the background of cancers developed by other reasons and to compensate them accordingly.

This document provides guidance on procedures and methodology to assess attributability of cancer to occupational exposure to ionizing radiation and to assist decision making regarding compensation of workers occupationally exposed to ionizing radiation below the relevant dose limits who developed cancer. It is intended in particular for the use of competent authorities, employers and workers, and persons in charge of compensation programmes for occupational diseases, in order to assist governments and social partners to make strategic choices that effectively meld economic efficiency and social protection.

This document reflects the collective wisdoms and experts’ view of an international group of experts who have participated in the technical and consultant meetings in the drafting process. The contributions of all the experts and reviewers to the drafting and revision of this document are much appreciated. Dr S. Niu of the ILO, Mr P. Deboodt of IAEA and Dr H. Zeeb of WHO served as scientific co-secretaries in coordinating the technical and consultation meetings and preparing this publication.

Finally, I should note that the responsibility for conclusions and opinions presented in this publication rests solely with the experts who contributed to the drafting and review, and that publication of this document does not constitute an endorsement by the ILO, IAEA or WHO of the opinions expressed in it.

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Preface

The first International Conference on Occupational Radiation Protection, hosted by the Government of Switzerland, was held in Geneva from 26 to 30 August 2002. It was organized by the IAEA, which convened it jointly with the International Labour Office (ILO). It was co-sponsored by the European Commission (EC) and held in cooperation with the World Health Organization (WHO), the OECD Nuclear Energy Agency (NEA) and a number of other international organizations. One of the recommendations produced by the Conference was that “The international organizations should develop guidance on the formulation and application of probability of causation schemes for the compensation of workers for radiation-induced occupational diseases.”

As a follow-up to the Geneva conference, the IAEA and the ILO established an International Action Plan for Occupational Radiation Protection. The Action Plan was approved by the IAEA Board of Governors on 8 September 2003 and a Steering Committee was set up to advise on, monitor and assist in its practical implementation. This publication is the result of a decision taken by this Steering Committee at its first meeting, held in Vienna in February 2004, to produce internationally agreed protocols and procedures for assisting in the development of compensation schemes. It provides guidance on procedures and methodology for assessing the attributability of cancer to occupational exposure to radiation and for assisting decision-makers in establishing compensation schemes for workers who have contracted cancer following occupational exposure to ionizing radiation below the relevant dose limit. It is intended in particular for use by competent authorities, employers and workers, and persons in charge of compensation schemes for occupational diseases.

This publication does not imply new requirements at the national level or for radiation professionals. It provides, firstly, basic scientific information on the biological effects of ionizing radiation for consideration when developing compensation schemes. Secondly, it gives examples of schemes developed in various countries. These examples can be used for comparison when such a scheme already exists in a country or can help to ensure that countries wishing to develop such schemes are aware of the factors and parameters that need to be taken into account.

It is envisaged that this publication will be updated in future to reflect new scientific knowledge on the health effects of ionizing radiation or to incorporate additional examples of compensation schemes as they become available.
Abbreviations

ADS
Approved Dosimetry Service

AOR
Act on Occupational Risks (Argentina)

AS
assigned share

ASQRAD
Assessment System for the Quantification of Radiation Detriment

ATB
age at the time of bombing

BCSC
basal cell skin carcinoma

BEIR
Biological Effects of Ionizing Radiation

BGFE
Berufsgenossenschaft der Feinmechanik und Elektrotechnik (Germany)
(German Trade Association for Precision Mechanics and Electrical Engineering)

CDC
Centers for Disease Control (United States)

CEPN
Centre d’étude sur l’Evaluation de la Protection dans le domaine Nucléaire (France)

CLL
chronic lymphocytic leukaemia

CNAMS
Caisse Nationale de l’Assurance Maladie des Travailleurs Salariés (France)

CSRLD
Compensation Scheme for Radiation-Linked Diseases (United Kingdom)

DDREF
dose and dose rate effectiveness factor

DHHS
Department of Health and Human Services (United States)

DNA
deoxyribonucleic acid

DOE
Department of Energy (United States)

EAR
excess absolute risk

EC
European Commission

EEOICPA
Energy Employees Occupational Illness Compensation Program Act (United States)

EPA
Environmental Protection Agency (United States)

EPR
electron paramagnetic resonance

ERR
excess relative risk

ESC
Exposed in Special Circumstances

EU
European Union

FISH
fluorescence in situ hybridization

HMA
Health Management Allowance (Japan)
IONIZING RADIATION EXPOSURE AND COMPENSATION PROGRAMMES

HPA Health Protection Agency (United Kingdom)
IAEA International Atomic Energy Agency
IARC International Agency for Research on Cancer
ICD International Classification of Diseases
ICFTU International Confederation of Free Trade Unions
ICRP International Commission on Radiological Protection
ICRU International Commission on Radiation Units and Measurements
ILO International Labour Organization
IOE International Organisation of Employers
IREP Interactive RadioEpidemiological Program (United States)
LLE loss of life expectancy
LSS Life Span Study
MHLW Ministry of Health, Labour and Welfare (Japan)
MOD Ministry of Defence (United Kingdom)
NAS National Academy of Sciences (United States)
NCI National Cancer Institute
NEA/OECD Nuclear Energy Agency of the Organisation for Economic Co-operation and Development
NIH National Institutes of Health (United States)
NIOSH National Institute for Occupational Safety and Health (United States)
NRA Nuclear Regulatory Authority (Argentina)
NRC National Research Council (United States)
NRPB National Radiological Protection Board (United Kingdom)
ORIA Occupational Risk Insurance Agency (Argentina)
PC probability of causation
PCC premature chromosome condensation
RBE relative biological effectiveness
RECA Radiation Exposure Compensation Act (United States)
REF radiation effectiveness factor
RERF Radiation Effects Research Foundation (Japan)
RR relative risk
SEC Special Exposure Cohort
SEER Surveillance, Epidemiology and End Results
SMCA Special Medical Care Allowance (Japan)
SREC Specialized Regional Expert Council (Russian Federation)
UN United Nations
UNSCEAR United Nations Scientific Committee on the Effects of Atomic Radiation
WHO World Health Organization
WLM working level months
Introduction

1.1 Background

Ionizing radiation can cause adverse health effects in humans. These effects fall into two categories: deterministic and stochastic health events. Deterministic effects of ionizing radiation in humans are the result of whole-body or local exposures that cause sufficient cell damage or cell killing to impair function in the irradiated tissue or organ. Stochastic health effects involve the non-lethal modification of a cell rather than its death. This modification is conventionally considered to be due to mutation of the DNA of a cell nucleus that can lead to cancer in the exposed individual if it occurs in a somatic cell. If the affected cell is a germ cell, hereditary genetic anomalies in the descendants of the exposed individual are another, though extremely rare, possible outcome.

The system of dose limitation in the current framework for radiation protection is directed at ensuring that deterministic effects are prevented from occurring, while the occurrence of stochastic effects is kept to an acceptable level. This means, in effect, that where incurred doses are in compliance with the dose limits no deterministic effect is to be expected. Compensation of these claims of deterministic effects as radiation-related would not normally be considered at all.

Workers who have been exposed occupationally to ionizing radiation at some stage of their working life and develop cancer may claim for compensation. However, cancer is a common disease and many factors contribute to its development. Without being able to identify exactly those individuals who have been affected principally by their occupational exposure to ionizing radiation, it is difficult to recognize the cancers these workers have as occupational and to compensate them accordingly. The current document provides some guidance for these situations.

The issue of cancer induction from occupational radiation exposure cannot, however, be seen in isolation from occupational cancer in general and so it requires inter-agency cooperation. While the International Atomic Energy Agency (IAEA) has a leading function at the technical level, with the responsibility of providing for the application of international radiation safety standards and reporting necessary scientific evidence (which is collected by the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR)), the issue is, at a policy level, relevant to other United Nations (UN) agencies including the International Labour Organization (ILO) and the World Health Organization (WHO). For example, the ILO has adopted the Employment Injury Benefits Convention, 1964 (No. 121), which includes a list of occupational diseases. Diseases caused by ionizing radiations are included in this list. The WHO obviously has a great interest in this issue; as an example, the WHO’s International Agency for Research on Cancer (IARC) has reported on combined analyses of cancer mortality among nuclear workers.
The Institution of Professionals, Managers and Specialists (a UK trade union now known as “Prospect”) hosted an informal IAEA/ILO/WHO consultation meeting at their headquarters in London in December 2000. The meeting produced a report, *The potential for developing joint international guidance for aiding decision-making on attributing cases of detrimental health effects to occupational exposure to ionizing radiations*, which provided a basis for further planning and preparation of a collaborative project by the IAEA, ILO and WHO.

The first International Conference on Occupational Radiation Protection, hosted by the Swiss government, was held in Geneva from 26 to 30 August 2002. Organized by the IAEA, which convened it jointly with the ILO, it was co-sponsored by the European Commission (EC) and held in cooperation with the WHO, the Nuclear Energy Agency of the Organisation for Economic Co-operation and Development (NEA/OECD) and a number of other international organizations. The Conference produced a number of important findings and recommendations, one of which was that: “The international organizations should develop guidance on the formulation and application of probability of causation schemes for the compensation of workers for radiation-induced occupational diseases.” These were considered in September 2002 by the IAEA General Conference, which requested the IAEA’s Director General, in cooperation with the ILO and other relevant bodies, to formulate and implement an action plan.

The IAEA and ILO prepared a draft that was reviewed by the organizations and key participants involved in the Geneva conference as well as by the International Confederation of Free Trade Unions (ICFTU) and the International Organisation of Employers (IOE). The Action Plan was approved by the IAEA Board of Governors on 8 September 2003. In order to ensure the successful implementation of the Action Plan, the IAEA and the ILO agreed to establish a Steering Committee with the overall remit to advise on, monitor and assist in the practical implementation of the Action Plan.

The first meeting of the Steering Committee was held in Vienna in February 2004 and the scope of each action was clearly defined. In particular, it was agreed that the IAEA, in collaboration with ILO, WHO, NEA and other relevant bodies and drawing on the experience of other stakeholders, would “continue its work on developing international guidance for aiding decision-making on the attribution of cases of detrimental health effects to occupational exposure to ionizing radiation.” The desired outcome of this action, referred to as Action 14 of the Action Plan, was to produce internationally agreed protocols and procedures to help in the implementation of probability-of-causation agreements.

In the meantime, a consultancy meeting was organized in Vienna, Austria, from 13 to 17 October 2003. A group of eight experts worked on a preliminary document and the official draft of *Attributing Radiation-Linked Disease to Occupational Exposure* was made available in May 2004 for comment by the Steering Committee members.

During its second meeting, in Vienna in January 2006, the Steering Committee recognized the worth of the draft but also recommended further development, such as the addition of more examples of compensation schemes. It was also agreed that the WHO should lead Action 14 up to the publication of the final document.

A technical meeting was organized by the WHO in May 2006. Hosted by the BGFE (Berufsgenossenschaft der Feinmechanik und Elektrotechnik) in Bad Münstereifel, Germany, 17 experts proceeded to review the draft document and advised on information still to be added. The WHO then produced a new version, which was sent to the experts for comment in May 2007. The amended version was finalized in December 2007 and it was agreed that the ILO should publish the final document once it was approved by the publishing committees of both the WHO and the IAEA.
1.2 Objective

Cancer is a common disease. In economically developed countries over one-third of the population develops cancer at some time during their lives and between one-fifth and one-quarter of individuals eventually die of malignant disease. Also cancer is becoming more important as a major cause of disability and death in less developed countries. Therefore, in any population occupationally exposed to radiation, a significant proportion of exposed individuals will develop cancer for reasons other than their exposure at the workplace. The Employment Injury Benefits Convention, 1964 (No. 121), requires that those workers who have developed cancer as a result of occupational exposure to radiation should be compensated. Compensation is straightforward in cases where the cancer is included in lists of occupational diseases and exposure meets the criteria prescribed in the relevant country. Where no such list-based approaches are followed, the occupational origin of a given cancer needs to be established on an individual basis. In the absence of being able to identify exactly those individuals who have been so affected, a process to provide compensation in some fair and equitable fashion should be available. Workers who develop cancer and can show that they were exposed to radiation during the course of their employment have a right to ask for compensation. However, to compensate all workers who have experienced occupational exposure at some stage of their working life would almost invariably result in a large proportion of exposed workers receiving compensation for cancers that could be mainly induced by factors other than occupational exposure, and the burden on the compensation scheme would probably be financially onerous. Alternatively, if not all exposed workers who develop cancer are compensated, a process of compensation could be considered that is capable of distinguishing those cases of cancer most likely to have been caused by occupational exposure to radiation from the background of cancers that have developed for other reasons. This document discusses, with reference to IAEA-TECDOC-870 (IAEA, 1996b), how this might be done. It should be noted, however, that compensation is only the final step in dealing with the health hazards of ionizing radiation in the workplace. In particular, the principle of optimized prevention of potentially hazardous exposures and adverse health effects should be central to a comprehensive system of occupational health and safety.

This document provides guidance on procedures and methodology to assess attributability to cancer of occupational exposure to radiation and to assist decision-making in compensating workers with cancers when their occupational exposure to ionizing radiation is below the relevant dose limits. It is intended in particular for use by competent authorities, employers and workers, and persons in charge of compensation programmes for occupational diseases.

The purpose of this document is not to replace any existing national compensation schemes for occupational diseases including cancer, or to propose a universal model for countries that do not have a scheme to compensate occupational cancer due to exposure to ionizing radiation. The provisions in the document are not meant to be applied as they stand in all countries and regions, but to provide information that should be considered in line with the local situation, technical resources and scale of the scheme coverage, factors which will determine the potential for application. This document describes the scientific basis for the attribution of health events to occupational exposures. It outlines the main components of the compensation schemes for cancer currently existing in member States that include assessment of attributability of cancer to occupational exposure to radiation, to assist the decision-making process concerning the work-relatedness of the disease, compensation and strategies for risk management.
The general characteristics and specific features are discussed, as are the characteristic benefits and drawbacks in the actual implementation of the existing compensation schemes. The use of radioepidemiological data and models pertinent to assessments of assigned share or probability of causation contributed by occupational exposure to radiation forms a significant part of the document, which considers both stochastic (e.g. cancer) and deterministic (e.g. cataract) effects induced by occupational exposure to ionizing radiation. Examples of some of the existing compensation schemes in member States are provided in Appendix A.

The target audience for this document includes those countries that have already implemented compensation schemes as well as those that have yet to do so. For the first group, this document provides relevant information on approaches devised in several developed countries, which could allow comparison and fruitful exchanges.

Nevertheless, the most useful added value of this document will be the benefit to those countries that have not yet implemented compensation schemes for the detrimental effects of occupational ionizing radiation. Although the document does not propose to present an exhaustive list of compensation schemes, countries should find here the basic issues and the relevant factors to be taken into account when developing such a scheme. Moreover, the presentation of existing compensation schemes could be used to facilitate the development of national approaches, taking into account the technical, political and cultural background of the country.

This document should also foster future discussions and exchanges of information between experts in radiation protection on the one hand, and experts in social and legal matters on the other.

1.3 Scope

This document considers occupational exposure to radiation as defined in the *International Basic Safety Standards for Protection against Ionizing Radiation and for the Safety of Radiation Sources* (IAEA, 1996a): “All exposures of workers incurred in the course of their work, with the exception of exposures excluded from the Standards and exposures from practices or sources exempted by the Standards.” An overview of occupational exposure based on the continuing examination by UNSCEAR, and compared with worldwide exposure to natural radiation, is given in Appendix B.

This document specifically addresses cancer in workers whose exposure to ionizing radiation at work is below the relevant occupational dose limits. Therefore, health effects in the offspring of mothers who were occupationally exposed while pregnant, as well as the potential hereditary effects of ionizing radiation in general, are not considered, although the methodology described here could be applied to such cases.

1.4 Structure

The document consists of two parts. Part A describes the scientific basis of risk attribution in the context of occupational radiation exposure and disease. Part B deals with approaches to assessment in compensation and with the main features of risk attribution-based compensation programmes. Several national examples of risk attribution-based
compensation programmes for radiation-linked disease are described in detail in Appendix A. Appendices B and C provide details on worldwide occupational radiation doses and an overview of biological indicators of radiation exposure. Appendix D is a detailed discussion of an approach to quantifying uncertainties in calculating attributable risks as implemented in the US Interactive RadioEpidemiological Program (IREP), and Appendix E describes the calculation of assigned share as implemented in ASQRAD, a new EU software program.
Part A: The scientific basis of risk attribution

2

Approaches to attributing health effects to occupational radiation exposure

Workers in various occupational settings are exposed to ionizing radiation. Some workers may later develop health problems, and the question may arise as to whether or to what degree the occupational exposure to ionizing radiation has contributed to the occurrence of disease. Ionizing radiation can cause adverse health effects in humans. These effects fall into two categories: deterministic and stochastic health events.

2.1 Deterministic effects

2.1.1 Background

Deterministic effects of ionizing radiation in humans are the result of whole-body or local exposures that cause sufficient cell damage or cell killing to impair function in the irradiated tissue or organ. The damage is the result of collective injury to substantial numbers or proportions of cells. For any given deterministic effect, a given number or proportion of cells must be affected, so that there will be a threshold dose below which the number or proportion of cells affected is insufficient for the defined injury or clinical manifestation of the effect to occur (ICRP, 1984). With increasing radiation dose fewer cells survive intact, and therefore deterministic effects increase in severity and frequency with the dose (UNSCEAR, 1982). If the radiation exposure is severe enough, death may result as a consequence of the exposure: death is generally the result of severe cell depletion in one or more critical organ systems of the body.

Ionizing radiation can impair function in all tissues and organs in the body because of cell killing; however, tissues vary in their sensitivity to ionizing radiation (ICRP, 1984). The ovary, testis, bone marrow, lymphatic tissue and the lens of the eye are the most radiosensitive tissues. In general, the dose-response function for these tissues, i.e. the plot on linear axes of the probability of harm against dose, is sigmoid in shape. Above the appropriate threshold, the effect becomes more severe as the radiation dose increases, reflecting the number of cells damaged. The effect will usually also increase with dose rate, because a more protracted dose causes the cell damage to be spread out in time, allowing for more effective repair or repopulation (ICRP, 1991). This type of effect, which is characterized by a severity that increases with dose above some clinical threshold, was previously called “non-stochastic”. The initial changes on the cellular level occur essentially at random, but the large number of cells required to result in a clinically observ-
able, non-stochastic effect gives the effect a deterministic character. Until recently, such effects were therefore called “deterministic” effects. This terminology has changed, however, and these effects are now referred to as “tissue reactions”. The dose levels that result in the clinical appearance of pathological effects are generally of the order of a few gray to some tens of gray. This clinical threshold or critical dose is based on clinical examination and laboratory tests. The time of appearance of tissue damage ranges from a few hours to many years after the exposure, depending on the type of effect and the characteristics of the particular tissue.

UNSCEAR extensively reviewed the deterministic effects of radiation in Annex J of its 1982 report (UNSCEAR, 1982). The basic concepts of cell survival were reviewed, including the factors influencing tissue response to fractionated or continuous exposures to radiation. That review of effects was based mainly on results of animal experiments and clinical observations of adults who had received radiotherapy. Its main objective was to identify the nature of effects in various tissues and the doses and modalities of irradiation that cause the effects. Since that report a large amount of information has been collected, including the effects of the Chernobyl accident (ICRP, 2006; WHO, 2006).

### 2.1.2 Dependence on cell killing

The severity of a deterministic effect depends on dose because this determines the proportion of cells killed. If people of varying susceptibility are exposed to radiation, the threshold in a given tissue for deterministic effects of sufficient severity to be observable will be reached at lower doses in more sensitive individuals. As the dose increases, more individuals will incur the observable effect, up to a dose above which the whole group shows the effect.

Examples of deterministic effects are the induction of temporary and permanent sterility in the testes and ovaries; depression of the effectiveness of the blood-forming system, leading to a decrease in the number of blood cells; skin reddening, desquamation and blistering, possibly leading to a loss of skin surface; induction of opacities in the lens and visual impairment (cataract\(^1\)); and inflammation processes that may occur in any organ. Some effects are indirect in that they are the result of deterministic effects on other tissues. For example, radiation that leads to the inflammation and eventual fibrosis of blood vessels may result in damage to the tissues served by those blood vessels.

A special type of deterministic effect is the radiation syndrome resulting from acute, whole-body irradiation. If the dose is high enough, death may result from severe cell depletion and inflammation in one or more vital organs in the body (blood-forming organs, the gastrointestinal tract and the central nervous system, in decreasing order of sensitivity).

### 2.1.3 Threshold dose values for deterministic effects

As discussed above, deterministic effects occur if a tissue or organ is exposed to a radiation dose high enough to cause impaired function because of cell damage or cell killing. The threshold for temporary sterility in the male for a single short exposure is about 0.15 Gy, while for prolonged exposures the threshold dose rate is about 0.4 Gy per year. The corresponding values for permanent sterility are in the range 3.5–6 Gy (acute exposures) and 2 Gy per year (chronic exposures). In women, the threshold dose rate for permanent sterility is in the range 2.5–6 Gy for an acute exposure, with women approaching the

---

\(^1\) The scientific discussion about cataract development as deterministic effect is noted.
menopause being more sensitive. For exposures continuing over many years, the threshold dose rate is about 0.2 Gy per year. These thresholds, like all thresholds for deterministic effects, apply to persons in a normal state of health. For individuals who are already close to exhibiting the effect from other causes, the threshold will be lower. Even in the extreme case where the effect is already present, there will still be a threshold representing the radiation dose needed to produce an observable change in the individual’s condition.

The threshold for lens opacities sufficient to result, after some delay, in vision impairment is 2–10 Gy for sparsely ionizing radiation (and about 1–2 Gy for densely ionizing radiation) in acute exposures. The threshold dose rate is not well known for long-term chronic exposures, but it is likely to exceed 0.15 Gy per year for sparsely ionizing radiation. These estimates are currently under review.

For acute exposures of whole bone marrow, the threshold dose for clinically significant depression of blood formation is about 0.5 Gy. The corresponding threshold dose rate for long-term exposure is somewhat above 0.4 Gy per year. Bone marrow failure is an important component of the radiation syndrome that follows whole-body exposures. An acute whole-body dose of between 3 and 5 Gy causes death in 50 per cent of the exposed population group in the absence of specific medical treatment.

In the case of skin exposures, the threshold for erythema and dry desquamation is in the range 3–5 Gy, with symptoms appearing about three weeks after exposure. Moist desquamation occurs after about 20 Gy, with blistering appearing about one month after the exposure. Tissue necrosis, appearing after three weeks, occurs after more than 50 Gy. Table 2.1 gives an overview of projected threshold estimates.

<table>
<thead>
<tr>
<th>Effect</th>
<th>Organ/tissue</th>
<th>Time to develop effect</th>
<th>Absorbed dose (Gy)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morbidity:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temporary sterility</td>
<td>Testes</td>
<td>3–9 weeks</td>
<td>~0.1a,b</td>
</tr>
<tr>
<td>Permanent sterility</td>
<td>Testes</td>
<td>3 weeks</td>
<td>~6a,b</td>
</tr>
<tr>
<td>Permanent sterility</td>
<td>Ovaries</td>
<td>&lt;1 week</td>
<td>~3a,b</td>
</tr>
<tr>
<td>Depression of blood-forming process</td>
<td>Bone marrow</td>
<td>3–7 days</td>
<td>~0.5a,b</td>
</tr>
<tr>
<td>Main phase of skin reddening</td>
<td>Skin (large areas)</td>
<td>1–4 weeks</td>
<td>&lt;3–6b</td>
</tr>
<tr>
<td>Skin burns</td>
<td>Skin (large areas)</td>
<td>2–3 weeks</td>
<td>5–10b</td>
</tr>
<tr>
<td>Temporary hair loss</td>
<td>Skin</td>
<td>2–3 weeks</td>
<td>~4b</td>
</tr>
<tr>
<td>Cataract (visual impairment)</td>
<td>Eye</td>
<td>Several years</td>
<td>~1.5a,c</td>
</tr>
<tr>
<td>Mortality:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bone marrow syndrome:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>without medical care</td>
<td>Bone marrow</td>
<td>30–60 days</td>
<td>~1b</td>
</tr>
<tr>
<td>with good medical care</td>
<td>Bone marrow</td>
<td>30–60 days</td>
<td>2–3a,b</td>
</tr>
<tr>
<td>Gastrointestinal syndrome:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>without medical care</td>
<td>Small intestine</td>
<td>6–9 days</td>
<td>~6a</td>
</tr>
<tr>
<td>with conventional medical care</td>
<td>Small intestine</td>
<td>6–9 days</td>
<td>&gt;6a,c,d</td>
</tr>
<tr>
<td>Pneumonitis</td>
<td>Lung</td>
<td>1–7 months</td>
<td>6b,c,d</td>
</tr>
</tbody>
</table>

*M: Most values rounded to nearest Gy; ranges indicate area dependence for skin and differing medical support for bone marrow.

Source: ICRP (2006); reproduced with kind permission of ICRP.
2.2 Stochastic effects

2.2.1 Background

Stochastic health effects involve the non-lethal modification of a cell rather than its death. This modification is conventionally considered to be due to mutation of the DNA of a cell nucleus, which can lead to cancer in the exposed individual if it occurs in a somatic cell. If the affected cell is a germ cell, hereditary genetic anomalies in the descendants of the exposed individual are another, though extremely rare, possible outcome. It should be noted that the scope of this document extends to the health of exposed workers only, and so does not cover diseases in the offspring of occupationally exposed workers, i.e. hereditary effects.

Stochastic events are thought to be no-threshold phenomena – any incremental dose of radiation, no matter how small, can theoretically produce an increase in the probability of a stochastic effect. Thus, even when standards for occupational radiation protection are met, there is a small likelihood of occurrence of stochastic effects. The probability of inducing cancer in a worker exposed to ionizing radiation increases with increasing dose of radiation (although at sufficiently high doses the probability will decrease due to the competing effects of cell killing). The severity of the radiation-induced effect does not depend on the dose, i.e. the stochastic health event depends solely upon the probability of the pertinent modification of a cell and the progression to cancer. A radiation-induced cancer, however, cannot be distinguished from equivalent diseases, either naturally (spontaneously) occurring or caused by other (e.g. chemical) exposures. Currently, the attribution of non-cancer somatic effects (in particular, diseases of the circulatory system) to radiation is equivocal because, according to scientific authorities, the evidence is insufficient to ascribe direct causation. More information about the existing evidence for these effects is provided by UNSCEAR (2000).

Given that radiation could play a role in the causation of a cancer in a worker who was occupationally exposed to radiation, and given that these radiation-induced cases of cancer currently cannot be individually identified by medical or biological means, a statistical approach to estimating the probability that a particular cancer may have been caused by prior occupational radiation exposure has been developed. This approach takes into account those relevant factors pertaining to an individual cancer case that influence the likelihood that the cancer is caused by occupational radiation exposure. These factors would certainly include the lifetime history of exposure to radiation and also the gender of the individual, the dose-response relationship, whether the exposure was acute or protracted, and the type of cancer under consideration. Other factors to be taken into account would be the latent period, the time since exposure and/or the attained age at diagnosis, the age at exposure, and the influence of other environmental, behavioural or social exposures (such as cigarette smoking in the case of lung cancer). Uncertainties in many of these factors can be dealt with by incorporating probability distributions in this approach. In some cases there are international standards (e.g. dose conversion factors) that should be used for the calculations. The concept of assigned share (AS) or probability of causation (PC) attempts to appropriately combine this individual-based information and is further described in Section 2.2.2.
2.2.2 Assigned share (probability of causation)

The theoretical possibility that a worker’s cancer has been induced by his or her past occupational exposures to ionizing radiation may be assessed by estimating the proportion of cancers in a notional large population of individuals with the characteristics of the case of cancer under consideration that may be ascribed to the exposure. The result of this calculation is usually termed the assigned share (AS) or probability of causation (PC). The use of the term “assigned share” implies that the AS/PC value only represents a mathematical expectation calculated over a population, rather than a strict probability as applied to the individual who clearly has developed a cancer. It must also be appreciated that the AS/PC methodology is applied to a case of cancer that has already occurred. Therefore, the AS/PC value should not be confused with the prospective probability of the cancer being induced in the future by a particular dose of radiation. For instance, an AS/PC value of, say, 40 per cent is the weight that can be attached to the specific prior radiation exposure having caused the particular cancer that has actually occurred in the individual; it is not a probability of 40 per cent that the particular cancer will develop in future as a result of the specific dose of radiation.

Epidemiological studies of relatively large populations exposed to significant levels of acute radiation exposure, in particular the studies of the cohort of Japanese survivors of the atomic bombings of Hiroshima and Nagasaki, have established that such exposure can cause most forms of cancer. They have also shown that the excess risk is modified by other factors (such as sex or age at exposure) and how this excess risk is expressed over time. For example, the studies show that the risk of radiation-induced acute leukaemia is significantly greater at younger ages at exposure and the excess risk is manifest as a “wave” over time since exposure, beginning at about two years after exposure, rising to a peak between five and ten years after exposure and then falling away to a low level some 20 years after exposure. Thus, if the convolution of size and dose of an exposed population is large enough, the epidemiological study of such a population allows the number of cases of cancer that may be attributed to irradiation to be determined and how this varies with other factors such as age and gender. Risk models have been developed to describe the expression of radiation-induced cancer risk in terms of important determinants of risk, for example the organ-specific cumulative equivalent dose, from which the excess relative risk (ERR = RR – 1, where RR is the relative risk) may be derived for a particular set of individual circumstances. Here, the excess relative risk is the proportional increase in the risk of the particular cancer that is due to exposure to radiation for the specific set of individual circumstances; this proportional increase is with respect to the background risk of the specific cancer in the absence of the additional dose of radiation. The AS/PC methodology attempts to take these risk models and work back from a given case of cancer to assess quantitatively the likelihood that the cancer was caused by a particular prior radiation exposure rather than some other factor.

The AS/PC value is expressed simply in terms of the ERR (and RR):

$$\text{AS/PC} = \frac{\text{ERR}}{\text{ERR} + 1} = \frac{(\text{RR} - 1)}{\text{RR}} = \frac{\text{ERR}}{\text{RR}}$$

It must be appreciated that what is being derived is a parameter applied to the circumstances of a specific individual using summary statistics obtained from exposed populations that are not necessarily directly related to that particular individual. Therefore elements of uncertainty, inaccuracy and imprecision are inevitably inherent in the calculation of the AS/PC values. The AS/PC methodology, however, generates a quantitative estimation of the role that an occupational exposure situation may have had in the development of a given cancer of an exposed worker. Thus, the AS/PC methodology provides
a filter through which cases must pass to extract those most likely to have been caused by occupational exposure to radiation from, in general, the much larger number of background cases; it allows the identification of those cases of cancer that are most deserving of consideration of attributability. It is clear that the objective should be to capture those cancers most likely to have been caused by occupational exposure without there being undue dilution by background cases.

In this type of methodological approach it is important that appropriate consideration is given to the selection of the technical basis, e.g. the specific radiation risk models used for each type of cancer and the management of uncertainty, inaccuracy and imprecision.

2.2.3 Uncertainties

A dose-specific risk estimate (e.g. ERR/Gy) obtained from epidemiological data has an explicit statistical uncertainty, estimated by the same statistical analysis used to obtain the estimate. When applied to a particular case and radiation dose, there is the additional uncertainty of the dose estimate. Inevitably, there are other uncertainties inherent in certain assumptions required to model risk as a function of dose, age and other factors, but which are not represented explicitly by model parameters and which may be best described as necessary but inherently subjective. An example is the latent period during which radiation-related risk is assumed to transition from between zero directly after exposure to an excess relative risk of (say) 0.5 per Sv at ten or more years after exposure. As more epidemiological data accrue, and as the understanding of biological mechanisms improves, these uncertainties will decrease to some extent or other, but it has to be accepted that uncertainties will remain within an AS/PC calculation. The level of uncertainty will vary between calculations, depending on the particular circumstances under consideration. For example, the statistical uncertainty associated with an uncommon cancer will, in general, be greater than that associated with a more common cancer with comparable dose-specific ERR values, because the epidemiological data are sparser, leading to a less precise estimate of risk. On the other hand, risk estimates for relatively rare cancers with high dose-specific ERR point-values can be relatively precise. It must be appreciated that for some cases of cancer, the statistical uncertainties could be considerable.

Even if a full treatment of statistical uncertainties in a radiation risk estimate is available, inaccuracies in the risk model itself must be considered as it is most unlikely that the model being used will be a perfect reflection of reality. For example, it is not reasonable to expect a risk model to exactly describe the expression of risk with time since exposure, or for the treatment of the interaction between cigarette smoke and radiation in the derivation of the overall risk of lung cancer to be without error. Nor should the uncertainties implicit within the application of a risk model derived from the study of a certain population to an individual from another population be ignored. So, for example, when baseline rates differ appreciably (say, female breast cancer or stomach cancer in Japanese and Western populations), whether the excess relative risk (ERR) or excess absolute risk (EAR) is calculated from the Japanese atomic bomb survivors and applied to an individual from a population of a different ethnicity will have a major effect on the calculated assigned share. For stomach cancer the transfer of the EAR value would lead to a higher assigned share than the transfer of the ERR value, and vice versa for female breast cancer.

What has been described thus far is the generic treatment of uncertainties that enter into AS calculations because of statistical imprecision and modelling inaccuracies. For individual calculations, errors arising from the consideration of the specific case must also be recognized. A particularly important aspect is the error associated with the relevant
dose estimate. Dosimetry records may be available. However, dose measurements will not be perfectly accurate, and the degree of inaccuracy will vary between various sets of circumstances. For example, radiation film badges will have differing sensitivities to photon energy spectra, and this sensitivity may well vary between film badge type and over time. Also, individual dosimeters will usually be estimating whole-body dose whereas, for a particular cancer under consideration, it will be the organ-specific dose that is required. It may be that certain components of dose (say, the dose due to fission neutrons) are missing and may have to be reconstructed if they form a significant fraction of the total dose. Inevitably, organ-specific doses due to internally deposited radionuclides will be uncertain and will need to be estimated from bioassay measurements, if these are available. Of course, it may be the case that dose records or other relevant information on exposure are not available at all so that doses must be reconstructed. This process carries with it a large degree of uncertainty.

Additional information to supplement the available dosimetry records may be available through biodosimetry techniques (see Appendix C). Use of these methods may be particularly informative when little occupational dose monitoring information is available. However, substantial uncertainties remain for these biodosimetry methods regarding the minimum detectable dose and inter-individual variability in their induction by radiation as compared to other risk factors.

In summary, the calculation of an AS value contains many uncertainties and it would be unrealistic to expect otherwise. Some appropriate approach to dealing with these uncertainties is an integral part of the AS process. Examples of additional uncertainty quantification and their incorporation into the AS calculation are given in Appendix D.

2.2.4 Estimation of assigned share for cancer

The large body of research on health effects of ionizing radiation has yielded models for estimating the risk of cancer, or some other health outcome like hereditary genetic disorders, as a mathematical function of radiation dose, sex, age at exposure, age at observation for risk or time following exposure, and (increasingly) lifestyle factors such as reproductive history and smoking history. Such estimates are of course uncertain, but the degree of uncertainty can also be quantified with some precision. The end result is that we can estimate, for a population with a given history of exposure to ionizing radiation, the population rate of diagnosis with a cancer of a given type at a given age, and the proportion of diagnosed cancers that would not have occurred in the absence of exposure. Both rate and proportion are characteristics of the exposed population rather than of any individual member, who can only have a cancer or not.

Assigned share can be defined in several, mathematically equivalent, ways. If $B(a)$ is the baseline cancer rate at attained age $a$ (i.e. that observed or expected in the absence of exposure), and if $EAR(a)$ is the excess rate (also called excess absolute risk) associated with exposure:

$$AS = \frac{EAR(a)}{B(a) + EAR(a)}$$

Generally, it is simpler to estimate excess relative risk:

$$ERR(a) = \frac{EAR(a)}{B(a)}$$

in which case,

$$AS = \frac{ERR(a)}{1 + ERR(a)}$$

i.e. AS is a monotonic, continuous function of ERR.

Assigned share pertains to inferences about attribution of a particular cancer case
to a particular exposure history. However, we estimate $\text{ERR}(a)$ by modelling $\text{ERR}$ globally, as a parametric function of radiation dose, exposure age, attained age, sex, and a number of other identifiable factors, including nationality or ethnicity, which may appear to influence risk, based on data from populations that are heterogeneous with respect to each of these factors. Information about radiation-related risk of cancers of sites other than the one of interest may also be relevant to a particular case. The mathematical form of the function, and the estimated values of the various parameters obtained by fitting the function to the data, allow observations on population members of different genders, with widely varying doses, exposure ages, and occurrences or non-occurrences of cancer at different ages, to contribute to an estimate of excess relative risk pertinent to a particular case. If we only considered data conforming to the specifics of a particular case, we generally would have so little data with which to work that we could not usefully estimate excess relative risk for the particular case.

A caveat pertinent to the necessity of working from the general to the particular is that the approach requires a number of assumptions to be made, any or all of which may introduce bias and/or uncertainty which could influence decisions about attributability and which therefore must be identified and taken into account as far as possible or, at least, practicable. The goal of the modelling process is to provide comprehensive information about estimated risk and its uncertainties. A quantitative approach to uncertainty estimation of radiation-related risk is presented in Appendix D, along with an approach that does not formally incorporate uncertainty (Appendix E).

### 2.2.5 Practical examples

As a starting point to describe the actual outcomes of assigned share calculations, reference is made to IAEA-TECDOC-870 (IAEA, 1996b) where a number of examples of simple assigned share calculations are presented for illustrative purposes. Some of these examples, with minor modifications, are given below. As noted previously, more complex and software-based approaches are described elsewhere in this document.

**Example A**

A male is diagnosed with leukaemia at the age of 68 years. He received a single uniform acute radiation dose to the red bone marrow of 100 mSv at the age of 43 years. Using the BEIR V model (NRC, 1990), what is the probability that this particular radiation dose was the cause of this leukaemia?

The leukaemia risk model relevant to these particular circumstances according to the BEIR V report is:

$$
\text{RR} = 1 + (0.243D + 0.271D^2) \exp(2.367)
$$

where:

- $\text{RR} =$ relative risk $= 1 + \text{ERR}$
- $D =$ dose
- $\exp =$ exponential function with $e$ (base of natural logarithm) raised to power $x$, here 2.367

The man received a single acute dose of 100 mSv ($= 0.1$ Sv) at the age of 43 years and was diagnosed with leukaemia 25 years later. Therefore

$$
\text{RR} = 1 + (0.243 \times 0.1 \times 0.271 \times 0.1^2) \times 10.665
$$
\[
RR = 1 + 0.02701 \times 10.665
\]
\[
RR = 1.2881; \ ERR = 0.2881
\]
and
\[
AS = \frac{ERR}{1 + ERR} = 22.37\%
\]

**Example B**

In this case a single acute dose of 100 mSv is received at the age of 20 years and leukaemia is diagnosed at the age of 33 years. Because the age at irradiation is less than 21 years, and the time since exposure is less than 16 years, other values from BEIR V apply:

\[
RR = 1 + 0.02701 \exp(4.885)
\]
\[
RR = 1 + 0.02701 \times 132.29
\]
\[
RR = 4.573; \ ERR = 3.573
\]
\[
AS = 78.13\%
\]

**Example C**

A male is diagnosed with leukaemia at age 68; he received a dose of 100 mSv spread out evenly over a period of ten years while he was aged 43 to 52 years. In this case, each annual dose of 10 mSv makes a contribution to the relative risk of leukaemia under the BEIR V model of:

\[
RR = 1 + (0.243 \times 0.01 + 0.271 \times 0.01^2) \exp(2.367)
\]
\[
RR = 1 + 0.026
\]
and the total ERR of the 100 mSv would be 0.26, giving

\[
AS = 20.6\%
\]

The examples illustrate how the excess relative risk (and hence the assigned share) varies with age at exposure and time since exposure under the BEIR V leukaemia model, as well as with other factors such as the spreading of total dose over time. Clearly, the choice of the underlying model influences the result of calculations to some extent. The model choice should be based on best available radioepidemiological evidence and considerations of appropriateness for the particular context.
Part B: Risk attribution-based compensation programmes

3

Approaches to assessment in risk attribution-based compensation programmes

This chapter describes approaches to attributing health effects such as cancer of occupational radiation exposure under the relevant occupational dose limits with respect to risk attribution-based compensation programmes. It should be noted that in all cases this refers to health effects that have already occurred. It does not deal with the probability that future health effects may occur.

3.1 Attributing deterministic effects

The system of dose limitation in the current framework for radiation protection is directed at ensuring that deterministic effects are prevented from occurring, while the occurrence of stochastic effects is kept to an acceptable level. This means, in effect, that the assigned share is effectively zero for deterministic effects in occupationally exposed workers where incurred doses are in compliance with the dose limits. Compensation of these claims of deterministic effects as radiation-related would not normally be considered at all.

On the other hand, exposure above the dose limit means that the system of radiation protection has been breached. The occurrence of such a situation (especially if the dose threshold for the effect is approached or exceeded), coupled with the actual development of a deterministic effect (appropriately related temporally to the exposure), may be regarded as a priori evidence that radiation exposure caused the deterministic effect. Full compensation would generally be warranted in such an event. In some cases injured workers with exposures close to the dose limits may still be eligible for inclusion in compensation schemes, depending on local regulations or provisions.

The combination of circumstances above will be rare. The majority of cases for adjudication will probably involve the development of an effect that might be related to a large radiation exposure, which the worker may allege took place but was not detected. The issue here is dose reconstruction to determine if any possibility exists that an unregistered exposure of such magnitude might have taken place. In order to justify not awarding compensation in such a case, it must be clearly demonstrated that there is a very low probability that any target organ or tissue, such as the lens of the eye in the case of development of cataract, could have incurred an unmeasured, inadvertent exposure of sufficient magnitude to give rise to the effect. To address this, the employee’s record of tasks, working
areas and radiation environment may be useful in investigating this point. It is unlikely that such a large exposure could have occurred without some related evidence as to the possibility. Biodosimetry (e.g. the rate of occurrence of chromosome aberrations in peripheral lymphocytes in a blood sample) may indicate a high exposure associated with an accidental exposure. Another factor to take into account in decision-making is the timing of the appearance of the effect in relation to the exposure allegedly causing the effect.

3.2 Attributing stochastic effects

Given the potential role of radiation in the causation of a cancer even at a level below the dose limits, and given that these cases of cancer cannot be specifically identified, a simple “blanket” approach to compensation would be to compensate any employee who experienced some exposure to radiation as a result of his or her employment and who later developed cancer. Because radiation is recognized as being capable of inducing most forms of cancer, such a scheme would entail ultimately compensating a substantial fraction of any exposed workforce. This would have the benefit of ensuring that all occupational radiation-induced cancers were captured by the scheme. However, because a significant proportion of any workforce is likely to have been exposed only to very low doses in an occupational setting (perhaps a few tens of μSv per year), such a process would also mean that compensation would be given to many more workers whose cancers are not attributable to occupational exposure to radiation. Large numbers of workers could show that they had been exposed to radiation as a result of their jobs, no matter how small that exposure, so such a scheme would inevitably lead to appreciable numbers of individuals receiving compensation when, on currently accepted risk estimates, the numbers of cancers caused by exposure to radiation at low doses in the workplace would be expected to be relatively small. As a consequence, the bulk of the compensation would be paid to workers who had not developed cancer as a result of occupational exposure. Also, this approach would not distinguish between a heavily exposed worker, whose cancer would be more likely to be attributable to radiation, and one with an exposure that was trivial. Such a “blanket” scheme could involve significant sums of money, much of which would not be spent on the intended purpose of compensating workers with cancers caused by exposure to radiation in the workplace.

To identify and properly compensate workers whose cancer is more likely to be occupational in origin, a method for assessing the chance that a cancer has been induced by a particular exposure to ionizing radiation that has been developed over the past two to three decades is the assigned share (AS) or probability of causation (PC) described in Section 2.2.1. The AS value calculated for a theoretically large population with the characteristics of interest in the specific cancer case under consideration is assigned to the affected person for the purpose of compensation.

A variety of practical approaches to dealing with the uncertainties inherent to the method of AS calculation is potentially available. We shall give some examples of these treatments by way of illustration:

- The simplest approach is to calculate the point estimate of the central value of the assigned share and if this exceeds 50 per cent (the conventional “balance of probabilities” criterion) then full compensation is awarded. The argument would run that the AS point estimate would be as likely to overestimate as underestimate the “true” AS value, and that therefore this method would be acceptable. It is unlikely, however, that
this approach would go unchallenged because a claimant with an associated AS estimate approaching 50 per cent may be able to point out that some other model that is equally acceptable scientifically produces an AS value in excess of 50 per cent. Frequent disputes under such an arrangement would seem inevitable. A natural extension to this approach is to incorporate, in an informal fashion, uncertainties into the point estimate of the assigned share so that the estimate acceptably reflects the inherent inaccuracies in the calculation. Usually, this would entail a certain degree of generosity towards the claimant, to a level that is thought appropriate under the circumstances. Under such a scheme, the factors adopted to deal with uncertainties need to be clearly stated.

• An alternative arrangement is to have a sliding scale of compensation under which the level of compensation varies from full compensation for an AS point estimate exceeding 50 per cent to a fraction of full compensation for AS values in some range below 50 per cent, with some cut-off (say, 10 or 20 per cent) below which compensation is not awarded. The fraction of full compensation would usually increase the closer the AS point estimate approaches 50 per cent, thus providing a system of proportional recovery of damages.

• A further approach is to attempt to quantify the uncertainty associated with an AS value through the calculation of a confidence interval. Usually, this would be a subjective confidence interval (or credibility interval), reflecting the judgements that will have to be made over the degree of uncertainty accompanying each component of the overall uncertainty. The upper confidence limit (typically the 90, 95 or 99 per cent limit) on the AS interval estimate may then be used to trigger compensation if it exceeds 50 per cent.

It should be noted that the above examples of treatments of uncertainty in AS calculations are not necessarily mutually exclusive in that two or more approaches could be combined in a particular scheme. For example, a sliding scale (proportional recovery) approach could be combined with the use of uncertainty quantification, with some quantified upper bound (e.g. the 75th percentile) of the AS estimate replacing the central values estimate.

The AS methodology is not uniformly accepted as an appropriate mechanism for determining compensation for radiation-induced personal injury. It has been argued that, in particular, the AS approach may not deal adequately with cases that have occurred earlier than they would have done in the absence of exposure to radiation. The practical significance of such objections has, however, been disputed. Nevertheless, these considerations have led to compensation schemes being suggested that are based on alternatives to the AS approach. One such alternative is the loss of life expectancy (LLE), where payment is proportional to the estimated average loss of life. A consensus on what is the most appropriate methodology to adopt for the purposes of compensating for radiation-induced cancer has not been reached and this highlights another aspect of uncertainty. A number of schemes based upon the AS approach have, however, been implemented or proposed, suggesting that such an approach is acceptable and practicable, providing uncertainties are treated with due attention.
Features of risk attribution-based compensation programmes

4.1 Background

In designing any arrangements for providing compensation to those exposed at levels below the occupational dose limits and who could have been harmed by ionizing radiations there are a number of issues to be addressed, and these are outlined below. Which options are considered and ultimately selected will depend on many different drivers specific to the country and/or organizations involved – these will be technical, political and cultural in nature. In the first place, there must be a will to establish compensation arrangements and with that must come reliable funding, either from government, business or some other appropriate party. In addition, attention should be paid to existing legal and social frameworks within the member State in order that the interests of other parties (e.g. those receiving benefits under existing arrangements) are not inadvertently compromised by any new programmes. However, whatever options are available, the features described below are likely to be common in the decision-making process leading to the creation of a new risk attribution-based compensation programme.

4.2 General features

4.2.1 Establishment of risk attribution-based compensation programmes

A competent institution or institutions should be designated, as appropriate, for the establishment, implementation and periodical review of risk attribution-based compensation programmes. This should be done through negotiations between the most representative organizations of employers and workers in consultation with radiation protection professional bodies, social security and with other bodies as appropriate. Risk attribution-based compensation programmes are normally developed in a process of voluntary negotiations and workers could resort to other means for compensation, including lawsuit and legal proceedings.

4.2.2 Population

After the establishment of a risk attribution-based compensation programme, the first point that must be addressed is to what population will the proposed compensation
arrangements apply? This may be as broadly or narrowly defined as necessary. Typically, populations covered will be defined by employer, site, location and/or profession.

4.2.3 Eligibility

Within the defined population, it may be necessary to further refine the covered population by use of eligibility criteria. For example, if the defined population is all persons who have worked for a particular employer, it is unlikely that every single person employed will have received some occupational radiation dose. Eligibility criteria are usually aimed at identifying those within the wider covered population who will have received occupational radiation exposures, either monitored or unmonitored. Typical criteria are the requirement that individuals have a dose record of some sort (or evidence which would suggest exposure) or have worked in specific plants at specific times. It is also likely to be a requirement that individuals must have died from or been diagnosed with a disease considered to be potentially related to radiation exposure.

4.2.4 Assessment criteria

Whilst the selection of assessment criteria is discussed in greater detail elsewhere it follows that, once the eligible population is identified, there must be some agreed method for assessing their suitability for compensation. It is also possible that differing types of criteria may be used within the same programme if circumstances dictate that this is necessary. Consideration should be given to uncertainties in the selected assessment criteria and to how the effect of these can be mitigated or counteracted when assessing individual cases.

An important feature in the selection of assessment criteria is the desire to use a methodology that is demonstrably consistent with the best scientific information in the field; consideration should also be given therefore to how the compensation programme will respond to changes in what is regarded as the best available scientific information and, if the assessment criteria may be modified in these circumstances, whether cases assessed under the previous criteria might be reviewed or reassessed in the light of new information.

4.2.5 Input data

Once the assessment criteria have been agreed, it will be necessary to evaluate the data required to facilitate the assessment. For arrangements based on assessment of the assigned share this will typically be:

- **Employment data.** It is normally a requirement that evidence of employment is provided.
- **Medical data.** There should be a requirement to prove cause of death or diagnosis of a disease that could potentially be related to radiation exposure. This will normally be done by reference to some system of classification (e.g. the International Classification of Diseases (ICD)). Cause of death may be taken from death certificates or autopsy data and confirmation of diagnosis can be sought from medical practitioners responsible for treatment of the claimant. Death certificates will usually specify the underlying cause of death as well as contributing causes.
- **Dose data.** Dose data may be taken from existing dose records or may be reconstructed from contemporaneous data or plant monitoring records, source terms, and so on. It
should also be noted that even where apparently complete dose records exist it will be necessary to evaluate the veracity of these, especially given technical limitations of dosimetry methods available historically. The aim should be to produce a dose history for the claimant that is either a true record of their radiation exposure or is a representation of their exposure history estimated from contemporaneous records (with reference to the claimant’s own work history) or, alternatively, is a reasonable upper bound estimate. Procedures should provide requirements for the decision-making process where some doubt concerning the results of the dose reconstruction remains.

When available, individual monitoring data should be preferentially used to evaluate exposures in a compensation programme. Without individual monitoring data, workplace monitoring records such as air sampling and external exposure measurements may be useful in estimating exposures. When using workplace measurements, it is important to assess the degree to which these might be representative of the individual dose being reconstructed. Although the radiation source-term can be used to evaluate doses, this method is subject to the highest amount of uncertainty and should be used with caution. For all evaluations the uncertainty (or the plausibility of the upper bound estimate) associated with the reconstructed dose should be addressed.

### 4.2.6 Compensability

Any compensation programme must set criteria for payment (and non-payment) based on the output from the chosen assessment methodology (or methodologies). In setting the criteria, it must be borne in mind that the guiding principle in the construction of a compensation programme should be to identify those individuals most deserving of compensation. The methodology of calculation to arrive at a causation probability must be designed so that there is confidence that all deserving cases are compensated.

### 4.2.7 Settlement options

The compensation programme must set out what benefits successful claimants will receive. This may be in terms of a fixed or negotiable sum of money, paid either as a lump sum or as an ongoing payment, or may be in terms of other benefits such as tax relief. The settlement method will need to be selected bearing in mind such factors as the expected value of personal injury claims in the country concerned and other methods of compensation employed in the country or culture. Where there is an element of negotiability of benefits, criteria should be set for that negotiation. For countries with little or no public health-care provision, the provision of health care should also be considered – if the compensation scheme indicates that there is a significant degree of causation attributable to radiation exposure, then it follows that there will be a responsibility for medical treatment of the affected individual.

### 4.2.8 Administration

Access to a risk attribution-based compensation scheme must be straightforward. Reasonable time limits must be applied to the process. This is important in morbidity cases where the claimant may be very ill. The claimant must be confident about the outcome. For this reason it is very important that those conducting the assessment be independent of the organization responsible for the worker’s exposure.
Workers may not understand the link between radiation and cancer and the assumption may be made that there is a direct causative link in all cases. Schemes should allow for claimants to receive independent advice and assistance on the progress of their case. Further, there should be an appeal process available to claimants who have been denied compensation. As it is possible that claims will fail, such cases must be dealt with sympathetically.

For claims where the exposed worker is deceased, the scheme must also allow for claims being made by relatives whose knowledge of the occupation in question may be limited. Accordingly, the basis for the assessment and the outcome must be capable of clear explanation so that the decision is understood, even if the result is unwelcome.

For cases where dose reconstruction is required, attention should be paid to the confidentiality of technical processes, as well as to avoid as far as possible any unnecessary dissemination of information.

### 4.2.9 Funding of compensation schemes

Compensation schemes need clear-cut funding and transparent funding procedures, and sufficient means of funding in order to secure an orderly assessment process as well as timely and appropriate claim payments once compensation is awarded. In general, employers – public or private – will be expected to fund compensation schemes, but other parties may contribute to the scheme.
Conclusions and recommendations

Cancer is a common disease in both developed and developing countries. A large number of causative factors are known or suspected.

Therefore, in any population occupationally exposed to radiation, a significant proportion of the exposed individuals will develop cancer for reasons other than their exposure at the workplace. ILO Convention No. 121 requires that workers who have developed cancer as a result of occupational exposure to radiation should be compensated. A process of compensating for the disease must be selected that is capable of distinguishing those cases of cancer most likely to have been caused by occupational exposure to radiation from the background of cancers that have developed for other reasons.

This document discusses how this might be done, describes the scientific basis for the attribution of health events to occupational exposures and outlines the main components of a risk attribution-based scheme, with the aim of assisting decision-making in occupational disease compensation.

Given that radiation could play a role in the causation of a cancer in a worker who has been occupationally exposed to radiation, and given that these radiation-induced cases of cancer currently cannot be specifically identified by medical or biological means, a statistical approach to estimating the probability that a particular cancer may have been caused by prior occupational radiation exposure has been developed. Different approaches to estimating the attributability of individual cases to occupational exposure are available and based on the concept of assigned share (AS) or probability of causation (PC).

There is a wide range of compensation programmes in practice. Examples from six countries are provided in Appendix A, which allows identification of the core features of compensation programmes. As described in Chapter 4, attention should be paid to the definition of the population concerned and, within this population, to the use of eligibility criteria such as the existence of dose records or the evidence of employment in specific plants.

The next step in the process is to identify the data to be collected. In some instances, accuracy level or uncertainties have to be addressed and efforts must be made to clarify the specific circumstances of exposure and disease for each case.

The compensation programme should be built in such a way as to identify those individuals most deserving of compensation. The settlement options have to be clearly defined and where there is an element of negotiability of benefits, criteria should be set for the negotiation.

Administrative procedures have to be accessible to and understandable by the claimants in order to promote confidence about the outcome of the compensation process.

As the last, but not least, requirement, funding procedures have to be transparent and means should allow an orderly assessment process as well as timely and appropriate payment when the claim is awarded.

In conclusion, whether a systematic approach based on probability of causation is recommended or not, it cannot replace societal negotiations about the actual structure of compensation programmes. At the international level, the exchange of experience built up in the development of such programmes at the national level should also be promoted.
Appendix A: Examples of compensation programmes

This appendix provides examples of current practice, describing arrangements in different countries with regard to compensation for cancers attributed to ionizing radiation exposure. While it is noted that the objective and scope of this document, as detailed in Sections 1.2 and 1.3, cover health effects of occupational exposures to workers, some of the examples cover, at least in part, the assessment of health effects in the general public following non-occupational exposures; although these examples are not provided to give specific guidance in this area, they do illustrate the approaches taken to such issues by some member States. Other programmes have been reviewed by Elliott (2003).

The section starts with a description of existing risk attribution-based compensation programmes using systematic approaches to calculating the probability of causation, followed by country examples using other approaches.

A.1 The UK Compensation Scheme for Radiation-Linked Diseases

A.1.1 Population

The UK Compensation Scheme for Radiation-Linked Diseases (CSRLD) is a private agreement between the majority of UK employers who operate nuclear licensed sites as defined by the Nuclear Installations Act 1965 (as amended) and their trade unions. As such, it applies to individuals who have been employed by one (or more) of the participating employers and who are (or have been) members of one of the participating trade unions (except for Ministry of Defence (MOD) employees where alternative arrangements are made because some MOD employees are not permitted to be members of a trade union).

A.1.2 Eligibility

In order to be considered eligible for assessment, claimants must fulfil the employment and trade union membership criteria described above, in addition to which they must have died from or been diagnosed with a disease considered by the Scheme to be potentially radiation-linked; they must also either have a dose record or there must be grounds for believing that they have been exposed to an unmonitored or unrecorded occupational dose.
Confirmation of employment is provided by the relevant employer’s personnel or human resources department. Trade union membership is confirmed by the trade union of which the claimant indicates he/she is or was a member. The Scheme trade unions do not limit their support only to current members; the general principle adopted is that, if the claimant was a member of the union at the time he/she received the occupational dose relevant to the claim, then the union will support that claim.

The Scheme usually takes confirmation from death certificates and/or written confirmation of diagnosis provided by the medical practitioner in charge of the claimant’s treatment (usually a hospital-based specialist, rather than a general practitioner or family doctor). In mortality cases, confirmation of the date of diagnosis by the treating practitioner is still preferred as this is generally beneficial to the claimant (assuming it places diagnosis at an earlier date than death). It is also helpful if the cause of death can be shown to be consistent with the disease diagnosed. Once diagnosis or cause of death is established, the disease(s) named in the relevant documentation is assigned a coding under the 8th edition of the International Classification of Diseases (ICD-8) (WHO, 1969). This then assigns the case to the appropriate Scheme schedule (see below) for assessment.

The UK CSRLD accepts all neoplasms as eligible except a small number known not to be, or to be only weakly related to radiation exposure, or where there are very strong links with other causative agents. Those diseases excluded are chronic lymphocytic leukaemia, hairy cell leukaemia, Hodgkin’s disease, malignant melanoma and malignant mesothelioma. The Scheme also accepts claims for cataract of the eye, although these will be rejected if the diagnosis attributes the development of the cataract to some other causative agent.

A.1.3 Assessment criteria

The technical basis of the UK Scheme is based on BEIR V (NRC, 1990), although some additional enhancements (which favour the claimant in all cases) have been included to overcome areas of uncertainty. The Scheme uses BEIR V to develop seven “schedules” (effectively dose-risk models), these being Leukaemia, Respiratory, Multiple Myeloma, Thyroid, Other Tissues, Skin, and Cataract. The Other Tissues schedule is also used to cover cancers of unknown origin.

The Leukaemia, Respiratory, Multiple Myeloma, Thyroid, and Other Tissues schedules use an AS methodology to calculate a probability of causation for a particular disease based on date of diagnosis or death and the claimant’s dose history.

The Skin schedule evaluates claims in a different way: for claimants with skin cancer, the relevant employer’s Chief Medical Officer will examine the claimant’s employment medical records to identify whether there is any evidence of the claimant having suffered a deterministic effect at the site of the cancer – the rationale used is that skin cancers require very high doses of ionizing radiation for initiation and such doses would inevitably lead to an erythema. Radiation workers in the United Kingdom are typically examined by a medical practitioner on an annual basis and would be expected to report to the employer’s medical department in the event of an erythema developing and thus any such incident should be recorded. Where there is no evidence of a deterministic effect, morbidity cases are deemed to have failed, whereas mortality cases that fail this criterion are subsequently assessed using the AS methodology offered under the Other Tissues schedule.

For cataracts, the Scheme assesses claims by compiling the lifetime dose to the lens of the eye. If it exceeds 5 Sv the case is awarded a full payment, if it exceeds 2 Sv but is less than 5 Sv a half-payment is awarded. Cases with less than 2 Sv are considered to
have failed. Given the current understanding of the doses required to cause cataract and the time period over which such doses need to be received, this method is considered (a) scientifically reasonable and (b) generous to the claimant.

A.1.4 Input data

All CSRLD cases (except morbidity skin cancer cases) require a compilation of the dose history, together with the ICD-8 coding of the disease in question.

Dose histories are provided to the Scheme by the relevant employer’s Approved Dosimetry Service (ADS) in accordance with agreed procedures (known within the Scheme as “dosimetry protocols”). These procedures are designed to give a compilation of the claimant’s dose history, which includes fair enhancements of records during periods where technical limitations may have affected the ability of the dosimeters used at the time to accurately record some doses or where our present understanding of the relationship between the dose measured by the dosimeter and the dose received by the wearer may differ from that current at the time the records were made. Thus the dose histories provided to the Scheme for purposes of claim assessment may not be identical to those held on the statutory dose record. Additionally, if there are identified omissions in the dose record, reasonable estimates (based on the upper values found in contemporaneous records) or upper bound estimates (based on locally or nationally enforced limitation regimes) will be used. For all malignant neoplasms except skin cancers, doses are reported as annual totals for “whole body dose” in mSv. For cataracts, doses are reported as annual totals for “whole body dose”, “surface dose” and “eye lens dose” (all in mSv).

Internal doses were only required to be assessed in the United Kingdom after 1986 (when the Ionising Radiations Regulations 1985 came into force). Before this time internal dose was usually limited by a system of airborne sampling in the workplace and bioassay (usually urine analysis and whole-body monitoring). Thus the Scheme requires that, where there are indications that a claimant has been monitored for potential internal dose, all relevant records are collated and an assessment made of internal dose. Such doses are reported to the Scheme as annual totals (in mSv), rather than committed doses (which would be how internal doses would be recorded for statutory purposes in the United Kingdom).

For skin cancer cases, the report of the relevant Chief Medical Officer is provided as a brief letter confirming the absence or presence of a deterministic effect. For skin mortality cases, the relevant employer’s ADS provides annual totals of “whole body dose” and “surface dose” (in mSv).

The relevant employer’s Chief Medical Officer also provides a summary of any data relating to claimants’ smoking habits for Respiratory schedule cases. This is required because the Scheme enhances (doubles) the calculated excess relative risk (ERR) for lifelong non-smokers, leaves it unmodified for pipe smokers and ex-smokers (those who are recorded as having been smokers but have not smoked for ten years or more) and reduces (halves) ERR for cigarette and cigar smokers.

One further generosity offered by the Scheme is that in cases where diagnosis is made at a relatively young age (i.e. below 50 years) the calculated risk is doubled before derivation of the probability of causation.

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2 The Ionising Radiations Regulations 1985 (S.I. 1985 No. 1333).
A.1.5 Compensability

For those Schedules where compensation is awarded directly from the assigned share calculated, payment is awarded on a sliding scale (known as “Proportional Recovery”) as follows.

For certain diseases where there may be other relevant factors, e.g. respiratory cancer where claimants have a history of smoking, or for cases where it is agreed special factors mean that the application of the Scheme schedules may be complicated or confounded, cases are referred to an Expert Panel for determination (the term used to describe the final decision made in respect of payment or rejection of a claim). The members of the Expert Panel are selected for their expertise in radiation or medical fields and the guidelines to which they operate are all agreed between the parties to the Scheme. In these cases, a Factual Report is prepared which gives the Expert Panel all relevant personal, dosimetry and medical data relevant to the case. The Expert Panel then meets to consider and discuss these cases and agrees the appropriate payment level, from “nil” to “full”, as in table A.1. Within these guidelines, the Expert Panel operates independently from the employers and unions; this is an important feature of the Expert Panel’s role within the Scheme.

<table>
<thead>
<tr>
<th>Assigned share</th>
<th>Payment band</th>
</tr>
</thead>
<tbody>
<tr>
<td>Less than 20%</td>
<td>Nil</td>
</tr>
<tr>
<td>20–29.9%</td>
<td>One-quarter</td>
</tr>
<tr>
<td>30–39.9%</td>
<td>One-half</td>
</tr>
<tr>
<td>40–49.9%</td>
<td>Three-quarters</td>
</tr>
<tr>
<td>50% and above</td>
<td>Full</td>
</tr>
</tbody>
</table>

A.1.6 The approach to uncertainty

It is recognized that there are uncertainties in the models presented by BEIR V that will automatically be transferred into the methodology used by the Scheme and also that there will be uncertainty inherent in the dosimetry data provided to the Scheme. The probability of causation values produced by the Scheme schedules are taken to be central values, although it is recognized that they may not be true central values as no evaluation of the uncertainties in the dosimetry data is made. However, the Scheme’s policy towards uncertainty in dosimetry data is to construct dose histories by retrospectively applying current assessment standards, as far as is possible, to historical dose records. Likewise, uncertainty in the calculation is overcome by the use of the proportional recovery payment system – if compensation were only paid to claimants achieving a PC value of 50 per cent or greater, it could be argued that, given the uncertainty inherent in the models used, even a small uncertainty distribution would mean some deserving claimants with PC values approaching but not reaching 50 per cent would be denied compensation. Thus it was considered more equitable to compensate claimants with PC values below 50 per cent in order to overcome questions of uncertainty in the PC result obtained – the fact that this also represents a generosity not allowed by the UK legal system was also an important factor in the adoption of this feature, as it serves to make the Scheme more attractive to claimants. The generosity of the Scheme in comparison to the UK legal system is reflected in the fact that almost three-quarters (78 of 108) of the Scheme’s successful claimants
have received payments for PC values lower than 50 per cent – the presumption being that, in a UK court, a claimant would need to prove his/her case “on balance of probabilities” (i.e. assuming the use of probability of causation, a PC value of 50 per cent would be needed for success).

A.1.7 Settlement value

Once it has been established that a case will qualify for a payment, the respective employer and union each appoint solicitors to agree the amount of compensation that is appropriate for a particular case. This “full value” is known in the Scheme as “Quantum”. In practice the procedure is exactly the same as if the case had been successful in a UK court. Once the value of Quantum has been agreed, the settlement figure is arrived at by applying the appropriate payment fraction awarded, i.e. if Quantum is agreed at £100,000 and the causation probability has been agreed as 35 per cent (thus qualifying for a half-payment), the settlement value will be £50,000. A number of case-specific factors such as loss of earnings, pain and suffering and number of dependent children are taken into account, hence there is a wide variation in awards made because individual circumstances differ. Throughout this process the union’s solicitor advises the claimant and seeks his/her agreement to the final amount payable.

The settlement figure and the legal costs associated with the negotiation of Quantum are paid by the claimant’s employer. In joint cases (i.e. where a claimant has been employed by more than one Scheme employer), the liability for compensation is shared between the relevant employers and is divided pro rata according to the excess relative risk calculated from the dose in each period of employment.

Further details on the UK Scheme can be found through the Scheme’s website at http://www.csrlrd.org.uk. Work is being done to update the Scheme schedules on the basis of BEIR VII.

A.1.8 Summary of important features

- **Flexibility.** The UK CSRLD is a private agreement between the Scheme employers and the trade unions. This means that, in effect, if the parties agree that any proposed change is of mutual benefit in the operation of the Scheme, then it can be varied as desired. However, the fact that the Scheme is a private agreement does mean that the Scheme employers may still be taken to court under the Nuclear Installations Act. Claimants do not have to forgo their right to legal action by using the Scheme (unless they are paid compensation through the Scheme) but, until now, no claimant failing under the Scheme has got as far as taking the employer to court as an alternative.

- **Defensibility.** The Scheme uses an internationally recognized publication (BEIR V) to underpin its technical basis. Modifications to BEIR V (NRC, 1990) are agreed between the employers and unions (and thus are supported by both) as are the underlying Scheme procedures (e.g. dose protocols). Thus outcomes for individual cases are supported by all parties to the Scheme. Furthermore, all decisions made by Scheme bodies are required to be made by consensus and so represent the agreed position of all parties to the Scheme; thus claimants can be assured that their interests are safeguarded.

- **Currency.** The use of BEIR V and the commitment by the Scheme to monitor relevant developments in related fields means the Scheme can claim that it is founded on
the best available scientific understanding of the relationship between radiation exposure and health effects. If future developments indicate that the Scheme’s technical basis requires modification the Scheme is able to do this (see “Flexibility” above) and, in fact, changed its technical basis in 1991 when the emergence of BEIR V superseded the methodology based on ICRP Publication 26 (1977), originally used by the Scheme (Mummery and Alderson, 1989). At the time of writing, the Scheme is in the process of reviewing the BEIR VII report to evaluate any implications for the technical basis.

• **Reduction of legal claims.** The fact that the Scheme was conceived as an alternative to lengthy and expensive court action means that there is a significant reduction in the possibility that Scheme employers will be taken to court under the Nuclear Installations Act. As participants in the Scheme, the unions are effectively committing to use the Scheme in all cases where potential claims are eligible under Scheme criteria.

• **Speed of evaluation.** The target for all cases is that the assessment which determines whether or not a claimant will receive a payment is issued within six months of the claim form being received. This is achieved in 75–80 per cent of cases. For successful cases, the aim is that the claimant will receive payment within 18 months of the case being filed; the practice of employers giving interim payments whilst Quantum negotiations are in progress is used to alleviate potential hardship during this time.

• **Generosity of assessment.** Cases are assessed using agreed criteria, which are designed to favour the claimant both in terms of the methodology used to assess causation probability (which is more generous than that presented by BEIR V itself) and in the use of proportional recovery (i.e. that cases with causation probabilities as low as 20 per cent will receive some compensation) whereas for a claim to be successful in the United Kingdom in a legal case, the court would have to find for the claimant “on balance of probabilities”, i.e. a causation probability of 50 per cent or more would have to be established. Thus it is highly unlikely that any case failing the criteria for payment under the Scheme could be successful through the UK legal system.

• **Cost savings.** One benefit of the Scheme is that it provides a much cheaper method of resolving such claims and also allows claims that might be rejected at an early stage under the legal system to be assessed in full.

• **Stakeholder involvement.** The Scheme is operated jointly between the UK nuclear employers and their trade unions and all decisions made by Scheme bodies are taken jointly by consensus. This creates common ground between employers and their unions. The fact that the Scheme employers and unions work closely together can be seen by disgruntled claimants as acting against their interests. However, such complaints are rare, especially when considered against the total number of claims received.

• **Issues of transparency.** Because the Scheme is a private agreement and because it uses a technical basis which is modified to be more generous than that already available in the public domain (i.e. BEIR V), the Scheme schedules cannot be put into the public domain in case they are then used as the basis of court action. This does mean, however, that the Scheme can be criticized for not being totally transparent, although the Scheme unions are party to the schedules.

• **Unquantified uncertainties.** Whilst the Scheme overcomes the question of uncertainty by modifying the dosimetry data for claimants, by enhancing the calculation methodology and by adopting a sliding scale for payments below a PC value of 50 per cent, the Scheme does not quantitatively analyse uncertainty for each case. However,
the technical representatives appointed by the unions have satisfied themselves that
the overall effect of the generosities used sufficiently overcomes the uncertainties
inherent in the models used and ensures that no claimant who may be deserving of
compensation is denied.

A.2 The US Department of Energy Employees
Occupational Illness Compensation Program

A.2.1 Population

In October 2000, the United States nuclear weapons production workforce was
provided with a compensation programme to cover employees suffering from certain
designated illnesses. This programme, which was enacted under the Energy Employees
Occupational Illness Compensation Program Act of 2000, specifically compensates work-
ers for cancer incurred as a result of exposure to ionizing radiation at US Department of
Energy (DOE) and other specified contractor facilities. A copy of the EEOICPA, as well
as a number of documents related to implementation of the Act, can be viewed at http://
www.cdc.gov/niosh/ocas. It is estimated that approximately 650,000 nuclear weapons
production workers have been employed by the DOE and its principal contractors since the
inception of these programmes in the 1940s. In addition, as many as 100,000 workers may
have been employed in production in the first decades of these programmes by short-term
contractors of the DOE, referred to under the EEOICPA as Atomic Weapons Employers.

A.2.2 Eligibility

To be eligible for compensation under the provisions of the EEOICPA, the
employee must have contracted cancer after beginning employment at a covered facility,
as defined in the Act. For purposes of adjudication of claims all primary cancers are
considered, with chronic lymphocytic leukaemia being the only cancer specifically
excluded from consideration. With the exception of researchers in residence at DOE facil-
ities, there is no minimum duration of employment to apply under the general provisions
of the programme; however, the EEOICPA does define a Special Exposure Cohort (SEC)
that includes workers with specified cancers at four DOE facilities. Specified cancers that
develop in these workers are presumed to be related to exposure at their facility, as long
as criteria for time period of employment, duration of employment, and monitoring status
are met. Claims are filed by the covered employee; however, spouses and children are
eligible to apply for compensation in the event the covered employee is deceased. The
criteria governing eligibility for spouses and children are described in the EEOICPA.

A.2.3 Assessment method

For a worker who does not have a specified cancer as part of the SEC, the basis
for assessment of the merit of a compensation claim, as required by EEOICPA, is use of
the probability of causation (or assigned share, used interchangeably throughout this

3 The Energy Employees Occupational Illness Compensation Program Act (EEOICPA), Public Law
106-398, 114 Stat. 1654, 1654A-1231 (30 October 2000), enacted as Title XXXVI of the Floyd D. Spence
discussion). Namely, use of the radioepidemiological tables developed by the US National Institutes of Health (NIH), as they are periodically updated, was cited as a legal mandate; however, the US National Institute for Occupational Safety and Health (NIOSH) was charged by executive order to develop a set of guidelines to use these methods to determine the probability that an eligible worker’s cancer was caused by occupational exposure to ionizing radiation (Schubauer-Berigan et al., 2003). The radioepidemiological tables were developed initially in 1985 and updated in 2003 by the NIH (Land et al., 2003; see Appendix D). These tables are based upon the concept of assigned share, and have been used in the United States in modified form for veterans exposed to radiation in the line of duty (see below).

The 1985 tables contain models to estimate assigned share from external radiation exposure for 12 cancers (leukaemia, malignant neoplasms of salivary glands, oesophagus, stomach, colon, liver, pancreas, lung, female breast, kidney, bladder and thyroid), from bone cancer due to exposure to radium-224, and radon-induced lung cancer. The EEOICPA, however, did not limit the cancer types for which probability of causation should be estimated or the types of radiation exposure that should be used in its estimation. Coincidentally, the US National Cancer Institute (NCI) was in the process of updating the tables when the US DOE compensation programme was established in late 2000, and many additional cancer and exposure types were explicitly modelled. Staff at the incipient NIOSH programme worked with the NCI to ensure that adequate models and exposure types were considered in the updated NCI radioepidemiological tables (National Institute for Occupational Safety and Health, 2002). The use of these methods in the Energy Employees Occupational Illness Compensation Program was formalized in a regulation published in the US Federal Register (DHHS, 2002a).

The revised radioepidemiological tables (Land et al., 2003) are based on more recent epidemiological data (primarily, dose-induced risk of cancer incidence in atomic bomb survivors through 1987 with DS-86 dose estimates). The NCI updated methods account for factors that modify the carcinogenicity of radiation, including the dose level, variations by cancer type, the timing of dose relative to the occurrence of cancer, and (for respiratory cancers only) smoking history. The methods incorporate uncertainty analysis in a computationally intensive, transparent manner, including such factors as bias in the method for assignment of dose to atomic bomb survivors, the method by which radiation risk is transferred from the Japanese atomic bomb survivors to the US population, a dose and dose rate effectiveness factor (DDREF), and radiation effectiveness factors (analogous to radiation weighting factors) for different types of radiation exposure (Kocher et al., 2005). Each of these factors is applied to the radiation risk coefficients using a detailed uncertainty distribution, which allows the full range of scientific knowledge about the factor to be considered. The updated NCI approach also replaces look-up tables with a computer program, the Interactive RadioEpidemiological Program (IREP), which incorporates these sources of uncertainty into an estimate of assigned share with its own uncertainty distribution (Land et al., 2003). A slightly adapted version of the IREP computer program, the NIOSH-IREP, was adopted for use by the US Department of Labor in estimating the probability of causation for claimants in the US nuclear workers’ programme. The adaptations include the addition of AS calculation for malignant melanoma and male breast cancer, and the use of different interaction assumptions for lung cancer and smoking history (for non-radon exposures). A feature of the Energy Employees Occupational Illness Compensation Program is that the models in NIOSH-IREP may be modified to reflect new scientific information, such as the incorporation of risk estimates from epidemiological studies of nuclear workers. The NIOSH-IREP may be accessed at http://www.niosh-irep.com/irep_niosh.
The EEOICPA specifies that methods be established to reconstruct doses incurred in the DOE workplace for eligible claimants.\(^4\) These dose reconstruction methods are discussed in detail below. However, an important and unique component of the methods is their incorporation into IREP. The NIOSH-IREP contains 12 types of radiation dose encountered in the DOE workforce, including radon, electrons of two energy ranges, photons of three energy ranges, neutrons of five energy ranges, and alpha radiation (NIOSH, 2002, p. 43). The dose reconstruction methods prescribe the estimation of dose for the organ in which the cancer occurred, and also include several assumptions about the rate of delivery (acute or chronic) of the external dose at the unit of badge reading, which weigh in favour of the claimant. The NIOSH-IREP uses each type of dose, along with its uncertainty distribution, associated radiation quality factor distribution and in some cases unique DDREF distribution, to produce estimates of risk that incorporate uncertainty from the relevant components of radiation dose, including level, effectiveness by type of exposure in inducing cancer, and dose-rate effects.

The EEOICPA also specifies that the compensability of a claim shall “be based on the radiation dose received by the employee … and the upper 99th per cent confidence interval of the probability of causation in the radioepidemiological tables…”.\(^5\) Therefore, in the US DOE worker compensation programme, the uncertainty distribution of the probability of causation calculation, which can include uncertainty from dose received by the claimant, is estimated. A hypothetical example of such a distribution, for a man diagnosed with leukaemia at age 50 and exposed to 110 mSv of high-energy photon radiation at age 40, is shown in figure A.1.

**Figure A.1** Uncertainty distribution in the probability of causation (PC) estimate for a male worker with leukaemia diagnosed at age 50 who was exposed to 110 mSv of high-energy photon exposure at age 40, calculated using NIOSH-IREP


A.2.4 Input data

Claimants are required to provide medical evidence that they have been diagnosed with a covered cancer. Cancers that have acceptable, documented evidence are reviewed and the date of diagnosis is ascertained. To be compatible with the probability of causation analysis, the medical file is reviewed and all primary cancers are assigned an ICD-9 code (DHHS, 1991). There are provisions for determining the likely primary cancers in the event that information for only a secondary cancer is available. The start and end date(s) of employment provided by the claimant are also verified using several methods.

The EEOICPA requires that radiation doses be reconstructed for all workers who were inadequately monitored. For claims that are not part of the SEC, the dose received by the organ/tissue that developed cancer is reconstructed in accordance with the methods specified in the dose reconstruction rule published in the US Federal Register (DHHS, 2002b). To provide appropriate input to the probability of causation calculation, the annual internal and external dose to the organ or tissue that developed cancer is reconstructed from the date of the covered employee’s first employment to the date of diagnosis. If the worker’s individual monitoring data are available, these are used as the starting point for the dose reconstruction. All data are evaluated for quality and used in the dose reconstruction only if they are found to reflect accurately the exposure conditions in the worker’s environment. If individual monitoring data are unavailable, the dose reconstruction is performed using workplace and/or source-term data. If insufficient data are available, a determination can be made that a dose reconstruction is not possible. In these cases, the claimant’s recourse would be to file a petition requesting that he/she be considered in a class of workers to be added to the SEC.

To expedite claims, an efficiency process has been adopted where an energy employee’s dose is evaluated only as far as necessary so that an unambiguous determination of compensability can be made. At any point during the dose reconstruction process it may be determined that the dose reconstruction process is complete if:

1. the dose estimated thus far would qualify the energy employee for compensation, or
2. the dose is determined from worst-case assumptions and the energy employee fails to qualify for compensation.

For claims processed under one of the conditions described above, it is often not necessary to include uncertainty in the dose estimates. For claims where a more complete dose reconstruction is required, the uncertainty for each dose is estimated and included as part of the input file for the probability of causation calculation.

An important part of the dose reconstruction process is the claimant interview. As part of the dose reconstruction rule, all claimants are interviewed using a standard interview script that attempts to capture as much as possible the characteristics of the worker’s exposure environment. If the energy employee is deceased, interviews are conducted with surviving family members who have filed a claim under the programme.

To account for the needs of a compensation programme, corrections are applied to convert the energy employee’s dose that was recorded for regulatory monitoring purposes to the actual dose received by the organ. For example, the external dose from exposure to photons is converted from the personal dose equivalent at 10 mm depth (Hp(10)) to the dose to the cancer site. For internal exposures, doses delivered in each tissue in each year of exposure are calculated, rather than the regulation-prescribed 50-year dose commitments. For claimants with incomplete or inadequate monitoring records, the dose that could have been received, but not detected, is also estimated. This includes an
evaluation of external dosimetry data that have been censored because of recording practices that omitted measurement data below administrative control or reporting levels. Internal exposures are also evaluated to determine the minimum detectable dose that could have been received, but undetected by the facilities’ bioassay monitoring programme.

Several other types of exposure not typically included in a regulatory monitoring programme are also evaluated. This includes the dose received by diagnostic X-ray examinations that were required as a condition of employment and the dose received from environmental sources of internal and external exposure that might have been unmonitored by the facilities’ monitoring programmes.

A.2.5 Compensability criteria

Any claimant whose cancer has been determined to have a probability of causation of greater than or equal to 50 per cent at the 99 per cent subjective confidence level is judged to have developed cancer in the performance of duty at a DOE or DOE contractor facility. As previously discussed, members of the SEC with certain specified cancers are presumed to have developed that cancer in the performance of duty. Although those claims with a probability of causation of less than 50 per cent are not awarded compensation, the claim may be submitted for reconsideration any time there is a change in status that may affect the compensability determination. This includes the development of additional primary cancers or the discovery of new information that might change the results of the dose reconstruction.

A.2.6 Nature of compensation

The EEOICPA mandates lump sum Federal compensation of US$150,000 for those claims where the cancer was judged to be at least as likely as not to have been incurred as the result of exposure at a covered facility. In addition to lump sum compensation, medical expenses are reimbursed for cancer treatment from the date of application to the compensation programme.

A.2.7 Summary of important features

- **Generosity.** The legislatively mandated use of the upper 99 per cent subjective confidence interval of the probability of causation estimate reduces the chance that a meritorious claim would be rejected. The use of the upper 99 per cent subjective confidence interval necessarily means that non-meritorious claims are awarded in many cases. The decision to compensate at such a level is usually determined by social factors, including compensation practices within a country and the number of potentially compensable cases. It has been pointed out by epidemiologists (Greenland, 2000) that the use of a single decision criterion, such as the 50 per cent probability of causation level, may result in non-payment of some meritorious claims. This limitation is minimized, however, by the use of the upper tail of the uncertainty distribution.

- **Incorporating uncertainty.** The explicit incorporation of uncertainty distributions rather than the simple propagation of an increasing number of conservative assumptions permits scientific knowledge to be used to the greatest possible extent. The dose reconstruction methods are also designed to incorporate the full range of scientific uncertainty, and are relatively complete with respect to the types of radiation encountered in the US DOE workforce.
• **Technical aspects.** From a technical standpoint, the use of IREP in its current form requires knowledge of country-specific rates of cancer incidence, which may not be available if cancer registry data are limited. The use of highly detailed methods for dose reconstruction may not be technically feasible or cost-effective in some programmes. The dose reconstruction methods, although they may be tailored within a programme to incorporate efficiencies such as the use of screening techniques, are time-consuming, which may be a prohibitive limitation in some settings.

### A.2.8 Other US programmes

The US Department of Veterans Affairs administers a compensation programme covering approximately 400,000 service members who participated in the post-Second World War occupation of Hiroshima and Nagasaki, were prisoners of war there, or who took part in atmospheric nuclear tests between 1945 and 1962 in the United States and elsewhere. Like the US DOE worker programme, the Veterans programme has a presumptive component, which covers 21 types of cancer, and a non-presumptive component, which covers any other potentially radiogenic disease (such as all cancers, cataracts, thyroid nodular disease, and central nervous system tumours) experienced by any veteran exposed to radiation during military service. Award of claims for these non-presumptive diseases is based on other factors including radiation dose, duration of exposure and timing of dose. The IREP and other resources as required are consulted to provide information on probability of causation for a non-presumptive claim. The nature of the compensation includes special health-care services and may include other benefits such as disability compensation and payment of survivor benefits. Since the establishment of the EEOICPA programme, military employees with presumptive cancers who worked at one of the US gaseous diffusion plants at Amchitka are covered by the Veterans Affairs compensation programme, as are military personnel with certain diseases who were exposed to radiation as part of their official duties. More information about this programme is available at http://www.va.gov/irad

The US Department of Justice administers a legislatively mandated programme for uranium miners, millers, ore transporters and non-military participants in atomic weapons testing in the USA as part of the Radiation Exposure Compensation Act of 1990 (RECA), as amended in 2000 (42 USC §2210 note (1994) and PL 106-245), and as supplemented by EEOICPA in 2000. The eligibility criteria for uranium miners include having worked in a uranium mine in one of 11 US states between 1942 and 1971, and having contracted primary lung cancer or certain non-malignant respiratory diseases. The compensation criterion is having been exposed to at least 40 working level months of radiation exposure (or employed at least one year) in a uranium mine. The compensation award consists of a lump sum “compassionate payment” of US$150,000. Other workers with primary lung cancer, certain non-malignant respiratory diseases, renal cancer or non-malignant chronic renal disease receive the same compensation award provided they worked for a period of at least one year at a uranium mill or as an ore transporter in one of these 11 states between 1942 and 1971. For the non-military participants in atomic weapons tests conducted by the United States, eligibility and compensability criteria include having been present “onsite” (above or on the ground) during a period of atmospheric testing, having participated in the atmospheric detonation of a nuclear device, and having contracted one of several specific types of cancer. The compensation award is US$75,000. More information about the RECA programme is available at http://www.usdoj.gov/civil/torts/const/reca/index.htm
A.3 The Japanese compensation programme for atomic bomb survivors

A.3.1 Population

Currently, there are nearly 300,000 atomic bomb (A-bomb) survivors in Japan and officially 259,556 survivors have been approved as of 31 March 2006. Officially approved A-bomb survivors means those in possession of an “A-bomb survivor’s certificate” issued by local governments. The conditions for obtaining one of these certificates are as follows:

Category 1 those who were present in the cities of Hiroshima or Nagasaki or officially designated vicinities at the time of the bombings.

Category 2 those who entered the designated areas within two weeks of the bombings, i.e. by August 20 in Hiroshima and August 23 in Nagasaki.

Category 3 those who were in other situations that might have caused radiation exposure due to the A-bombings.

Category 4 those who were unborn babies of pregnant mothers applicable to any of the above.

A.3.2 Eligibility

Although there are various allowances established for A-bomb survivors in Japan, this section mainly describes issues concerning the Special Medical Care Allowance (SMCA), because a probability of causation (PC) approach is used to authorize the allowance.

Only authorized survivors (see above) are eligible for SMCA, as established on the basis of the A-bomb survivor disease list developed by the Health Care Commission of A-bomb Survivors under the Ministry of Health, Labour and Welfare (MHLW) of Japan. If the disease is judged as possibly caused by radiation exposure of the A-bomb, the survivor can be authorized for SMCA. In the MHLW, requirements for SMCA are decided as follows:

(1) Disease or injury is claimed to be caused by A-bomb radiation or residual radioactivity.

(2) The survivor has a condition which needs medical treatment.

(3) Although condition (1) or (2) is not applicable, the curability of the disease or injury seems to be in question and the treatment is delayed due to A-bomb radiation.

A PC approach is used as a criterion of decision-making for cases under (1).

A.3.3 Assessment criteria and methods

To establish criteria, PC values for cancers (all solid cancers including leukaemia) of A-bomb survivors have been prepared. For this purpose, two epidemiological references about A-bomb survivors reported from the Radiation Effects Research Foundation (RERF) were used. One is the mortality (life span) study from 1950 through 1990 by Pierce et al. (1996) and was used for PC calculation of leukaemia and solid cancers except female breast and thyroid cancers. The other paper is the cancer incidence study spanning
the years 1958 to 1987 and published by Thompson et al. (1994), which was used for female breast and thyroid cancer.

Concerning leukaemia and cancers of the stomach, colon, lung, breast (female) and thyroid, there was a statistically significant dose-response for each cancer. PC values were calculated for each disease. On the other hand, concerning cancers of the liver, skin (malignant melanoma excluded), ovary, urinary tract (bladder excluded) and oesophagus, the effect of A-bomb radiation was suggested, however, a confidence interval of dose-response relation was large and there was no definite statistical significance. Therefore, for those cancers, the epidemiological data were collected and a grouped PC was estimated.

A.3.4 Input data

PC is calculated according to the expression:

$$PC(\%) = \frac{A \cdot \text{dose} \cdot \exp(B(\text{ATB} - 30))}{1 + A \cdot \text{dose}/100 \cdot \exp(B(\text{ATB} - 30))}$$

Constants A and B were defined according to the epidemiological study of A-bomb survivors, and vary according to cancer site and sex. Therefore input data (parameters) which are required for PC calculation are:

- **Dose**: radiation dose to individual A-bomb survivor. Dose can be estimated using DS86 (Dosimetry System 1986), which is the dosimetric method developed for the epidemiological study of A-bomb survivors. In addition, dose of the induced radioactivity and the radioactive fallout are also included.
- **ATB** (age at the time of bombing). Generally, the lower the ATB, the higher the PC value.
- **Cancer site**: as described above, for leukaemia, cancers of the stomach, colon, thyroid, breast and lung, PC is calculated separately. That is, constants A and B were defined for each. On the other hand, for PC calculation of cancers of the liver, skin, ovary, urinary tract and oesophagus, the sites are grouped and constants A and B are unified.
- **Sex**: PC is calculated separately for males and females. Therefore, constants A and B were defined independently.

Table A.2 is an example of PC values for male colon cancer. It demonstrates that PC varies according to ATB and dose. Table A.3 shows that PC varies according to cancer site and sex.
Table A.2 Male colon cancer: Example of probability of causation (%)

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<td>6.3</td>
<td>7.3</td>
<td>8.3</td>
<td>9.2</td>
<td>10.1</td>
<td>15.8</td>
<td>27.3</td>
</tr>
<tr>
<td>24</td>
<td>1.0</td>
<td>2.0</td>
<td>3.0</td>
<td>4.0</td>
<td>4.9</td>
<td>5.8</td>
<td>6.7</td>
<td>7.6</td>
<td>8.5</td>
<td>9.3</td>
<td>14.6</td>
<td>25.6</td>
</tr>
<tr>
<td>25</td>
<td>0.9</td>
<td>1.8</td>
<td>2.7</td>
<td>3.6</td>
<td>4.5</td>
<td>5.3</td>
<td>6.2</td>
<td>7.0</td>
<td>7.8</td>
<td>8.6</td>
<td>13.5</td>
<td>23.9</td>
</tr>
<tr>
<td>26</td>
<td>0.9</td>
<td>1.7</td>
<td>2.5</td>
<td>3.3</td>
<td>4.1</td>
<td>4.9</td>
<td>5.7</td>
<td>6.4</td>
<td>7.2</td>
<td>7.9</td>
<td>12.5</td>
<td>22.2</td>
</tr>
<tr>
<td>27</td>
<td>0.8</td>
<td>1.5</td>
<td>2.3</td>
<td>3.0</td>
<td>3.8</td>
<td>4.5</td>
<td>5.2</td>
<td>5.9</td>
<td>6.6</td>
<td>7.3</td>
<td>11.5</td>
<td>20.7</td>
</tr>
<tr>
<td>28</td>
<td>0.7</td>
<td>1.4</td>
<td>2.1</td>
<td>2.8</td>
<td>3.4</td>
<td>4.1</td>
<td>4.8</td>
<td>5.4</td>
<td>6.0</td>
<td>6.7</td>
<td>10.6</td>
<td>19.2</td>
</tr>
<tr>
<td>29</td>
<td>0.6</td>
<td>1.3</td>
<td>1.9</td>
<td>2.5</td>
<td>3.2</td>
<td>3.8</td>
<td>4.4</td>
<td>5.0</td>
<td>5.5</td>
<td>6.1</td>
<td>9.8</td>
<td>17.9</td>
</tr>
<tr>
<td>30</td>
<td>0.6</td>
<td>1.2</td>
<td>1.8</td>
<td>2.3</td>
<td>2.9</td>
<td>3.4</td>
<td>4.0</td>
<td>4.5</td>
<td>5.1</td>
<td>5.6</td>
<td>9.0</td>
<td>16.5</td>
</tr>
</tbody>
</table>

ATB = age at the time of bombing.

Table A.3 PC values for various cancers (dose 50 cGy, ATB 12 years)

<table>
<thead>
<tr>
<th>Cancer</th>
<th>Leukaemia</th>
<th>Stomach</th>
<th>Colon</th>
<th>Thyroid</th>
<th>Breast</th>
<th>Lung</th>
<th>Liver, skin, ovary, urinary, oesophagus</th>
<th>Others</th>
</tr>
</thead>
<tbody>
<tr>
<td>PC (%)</td>
<td>65.7</td>
<td>83.8</td>
<td>3.6</td>
<td>38.0</td>
<td>34.0</td>
<td>58.2</td>
<td>69.0</td>
<td>55.1</td>
</tr>
<tr>
<td>ATB = age at the time of bombing; PC = probability of causation.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
A.3.5 Compensability

The officially approved A-bomb survivor with an “A-bomb survivor’s certificate” is basically supplied with the following provisions:

1. Annual health check-up for general, cancer and other specific medical examinations.
2. Medical provision: necessary medical treatment at home or in a hospital is supplied without charge under the national health insurance system.
3. If a certified A-bomb survivor is diagnosed with a designated disease, he/she will be provided with a Health Management Allowance (HMA) of 33,900 yen (about US$300)/month (in 2006; annually changing).

Moreover, if the survivor is authorized for SMCA, he/she will be provided with 137,840 yen (about US$1,260)/month. Thus, A-bomb survivors with SMCA are sufficiently supported; even A-bomb survivors without SMCA are well supported. Therefore it is thought that A-bomb survivors are thoroughly compensated.

A.3.6 The approach to uncertainty

The PC values used in our system were based on the epidemiological study of A-bomb survivors in RERF. The uncertainties in our system are thought to be derived from the epidemiological data.

One uncertainty is that the attributable risk of a population is used as PC for individuals. In our case, however, the risk of A-bomb survivor population is applied for an individual A-bomb survivor. Accordingly, such an uncertainty does not seem to be much of a problem.

Another uncertainty relates to the fact that there are some cancers, for example prostate cancer, for which appropriate PC values could not be calculated because they do not show statistical significance in dose-response relationships in the epidemiological study of RERF. However, even if there is no statistical significance, according to a general radiobiological understanding, it cannot be denied that any cancer might occur as a result of stochastic effects. Therefore, in practice, the lowest PC, that of male stomach cancer, is substituted for such cancers (see table A.3, “Others”).

A.3.7 Nature of compensation/settlement values

In the Health Care Commission of A-bomb Survivors, the judgment according to PC is described as follows: if PC is over 50 per cent, it is estimated that the applied disease condition is attributable to A-bomb radiation with certain likelihood and if PC is less than 10 per cent, it is estimated to be unlikely that the disease is caused by A-bomb radiation. If PC is more than 10 per cent and less than 50 per cent, it is judged independently (on a case-by-case basis). In the actual situation, however, if PC is more than 10 per cent, most cases are approved and eligible for SMCA. Besides, there is currently no procedure in Japan to vary the allowance in proportion to the PC value.

Thus, there is a boundary or PC limit which separates compensated cases and others in the Japanese system. Some survivors have complaints about the judgment, although most survivors appear to be compensated.
A.4 The Russian Federation Compensation Scheme for Radiation-Linked Diseases

A.4.1 Population

The Russian Federation Compensation Scheme for Radiation-Linked Diseases is part of the National System for Compensation of Occupational Diseases. The contemporary legislative base for the compensation of occupational diseases in the Russian Federation was started in 1996 and is guaranteed by a number of Federal Laws of the Russian Federation:

(1) Labour Code of the Russian Federation;\(^6\)
(2) Part II of the Civil Code of the Russian Federation;\(^7\)
(3) “Bases of Labour Safety in the Russian Federation”;\(^8\)
(4) “Bases of Obligatory Social Insurance”;\(^9\)
(5) “Obligatory Social Insurance Accidents for Work and Occupational Diseases”.\(^{10}\)

In the case of occupational diseases potentially linked to irradiation, the following two categories of workers may apply for compensation.

A: Workers Exposed in Special Circumstances

This category includes selected groups of Workers (including soldiers, policemen and others) Exposed in Special Circumstances (ESC Workers) during wide-scale radiation events in the course of their duties. “Wide-scale radiation events” are defined as:

- Radiation accidents and remediation actions connected with:
  - the accident at Chernobyl Nuclear Power Plant (NPP) in 1986;
  - the accident at Production Association “Mayak” in 1957 (Kyshtym accident);
  - radiation accidents with nuclear submarines, nuclear ships and nuclear weapons.
- Radiation exposure due to radionuclide discharges into the Techa River basin (past practice of the Production Association “Mayak”) and during the relevant remediation actions.
- Nuclear weapons tests and military exercises with nuclear weapons.

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B: Radiation Workers

This category includes Radiation Workers who were exposed in the course of routine operations with man-made sources.

A.4.2 Eligibility

Applicants for compensation related to occupational radiation-linked disease must be:

- Workers Exposed in Special Circumstances (ESC Workers); or
- Radiation Workers

and suffer from the disease that is defined as radiation-linked or linked to a wide-scale radiation event.

In order to qualify for consideration as an ESC Worker, individuals must have been present for a specified period in the area defined as an area of wide-scale radiation event. The status of each group of ESC Workers is determined by specific Federal Laws:

1. Federal Law concerning social protection of the citizens affected by radiation due to accident at the Chernobyl NPP" (Chernobyl Law);\(^{11}\)
2. Decree of the Supreme Council of the Russian Federation “About distribution of action of the Federal Law concerning social protection of the citizens affected by radiation due to the accident at the Chernobyl NPP” on citizens from divisions of special risk (Decree on Divisions of special risk);\(^{12}\)
3. Federal Law concerning social protection of the citizens affected by radiation due to nuclear tests at the Semipalatinsk test site (Semipalatinsk Law);\(^{13}\)
4. Federal Law concerning social protection of citizens of the Russian Federation affected by radiation due to the accident in 1957 on a production association “Mayak” and dumps of radioactive waste products into the Techa River (Mayak Law).\(^{14}\)

In order to qualify for consideration as Radiation Workers, individuals must have worked permanently with man-made sources of ionizing radiation (for more details see Kutkov et al., 2003). The Radiation Safety Standards of the Russian Federation issued in 1999\(^{15}\) define a *man-made source* as any source of radiation that is:

- specially manufactured for its useful application as a source of ionizing radiation;
- specially manufactured for the useful application of its properties of radioactivity; or

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\(^{13}\) Social protection of the citizens affected by radiation due to nuclear tests at the Semipalatinsk test site, No. 149-FZ, Federal Law of the Russian Federation, 1995.


• a by-product (radioactive waste) of practices involved in the foregoing sources.

The application of this definition means that underground workers from uranium mines are classified as Radiation Workers, but workers from other mines are not.

A.4.3 Assessment criteria

The main assessment criterion for assessing the suitability of the eligible population for compensation is that claimants must have developed a disease that is defined by the State as:

• a disease linked to a radiation event for ESC Workers; or
• a radiation-linked occupational disease for Radiation Workers.

The list of diseases defined as occupational diseases that may be radiation-linked or disease-linked with the wide-scale radiation event is approved by the appointed governmental body, the Ministry of Health and Social Development.

A.4.4 Workers Exposed in Special Circumstances

The current list of diseases considered to be linked to the Chernobyl accident, the accident at the production association Mayak (1957) and dumps of radioactive waste products into the Techa River was approved by Executive Order on 4 November 2004. The classification is presented in table A.4.

Table A.4 List of diseases defined as linked to the Chernobyl accident, the accident at the production association Mayak (1957) and dumps of radioactive waste products into the Techa River in the Russian Federation

<table>
<thead>
<tr>
<th>No.</th>
<th>Diseases</th>
<th>ICD-10</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Acute and chronic radiation syndrome</td>
<td>T66</td>
</tr>
<tr>
<td>2</td>
<td>Radiation cataract</td>
<td>H26.8</td>
</tr>
<tr>
<td>3</td>
<td>Radiation hypothyroidism</td>
<td>E03.8</td>
</tr>
<tr>
<td>4</td>
<td>Local radiation injuries (radiation burns)</td>
<td>L58</td>
</tr>
<tr>
<td>5</td>
<td>Neoplasms</td>
<td>C00–D48</td>
</tr>
</tbody>
</table>

The classification presented in table A.4 is the fourth edition of the List of Occupational Diseases Defined as Linked to the Chernobyl Accident. The classification known as the “1999 Chernobyl List” is presented in table A.5. This classification is also used for other radiation events mentioned in the previous subsection.

The classification presented in table A.5 is the third edition of the List of Occupational Diseases Defined as Linked to the Chernobyl Accident. The classifications in force in different periods after this accident are presented in tables A.6 and A.7.

In the former Soviet Union medical care for persons exposed during radiation events and for overexposed Radiation Workers was concentrated in the Clinical Department of the Institute of Biophysics (Moscow), better known as Clinical Hospital No. 6. For a long time the only Specialized Expert Council for causation of occupational diseases was based in this Clinical Department. In the early 1990s a number of additional Specialized Regional Expert Councils (SRECs) were created by the Ministry of Public Health in

different regions of the Russian Federation in order to implement the provisions of the Chernobyl Law and of other Laws on compensation for diseases developed by ESC Workers.\textsuperscript{17} Now these SRECs are based in Moscow, St Petersburg, Novosibirsk, Chelyabinsk and Rostov-on-Don.

The decision-making process for compensation of diseases of ESC Workers is as follows:

(1) If he/she develops a disease listed in table A.1, he/she shall be compensated in any case without any consideration of his/her dose related to the radiation event, the occupational or health history prior to the development of the disease.

(2) If he/she develops a disease not listed in table A.1, he/she will be compensated unless the SREC has proof to deny the causation between the disease developed and the radiation event under consideration. In this case consideration of his/her dose related to the radiation event, together with the occupational and health history prior to the development of the disease is obligatory.

\textsuperscript{17} For details see footnotes 11-14.
### Table A.6 Occupational diseases defined as linked to the Chernobyl accident in the Russian Federation in 1992–97 (1992 Chernobyl List)

<table>
<thead>
<tr>
<th>No.</th>
<th>Diseases</th>
<th>ICD-9*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Acute and chronic radiation syndrome</td>
<td>909.2, 990</td>
</tr>
<tr>
<td>2</td>
<td>Radiation cataract</td>
<td>366.46</td>
</tr>
<tr>
<td>3</td>
<td>Local radiation injuries (radiation burns)</td>
<td>990</td>
</tr>
<tr>
<td>4</td>
<td>Disorders of thyroid gland:</td>
<td></td>
</tr>
<tr>
<td></td>
<td>radiation hypothyroidism</td>
<td>244 (244.8)</td>
</tr>
<tr>
<td></td>
<td>radiation autoimmune thyroiditis</td>
<td>245 (245.2)</td>
</tr>
<tr>
<td></td>
<td>nodular goitre and benign nodules of the thyroid gland (benign thyroid nodules)</td>
<td>241, 240</td>
</tr>
<tr>
<td>5</td>
<td>Malignant neoplasms:</td>
<td></td>
</tr>
<tr>
<td></td>
<td>of thyroid</td>
<td>193</td>
</tr>
<tr>
<td></td>
<td>of breast</td>
<td>174, 175</td>
</tr>
<tr>
<td></td>
<td>of ovary</td>
<td>183</td>
</tr>
<tr>
<td></td>
<td>of lung and the respiratory tract</td>
<td>163</td>
</tr>
<tr>
<td></td>
<td>of stomach</td>
<td>151</td>
</tr>
<tr>
<td>6</td>
<td>Hemoblastoses:</td>
<td></td>
</tr>
<tr>
<td></td>
<td>acute (myeloid) leukaemia</td>
<td>205.0</td>
</tr>
<tr>
<td></td>
<td>chronic myeloid leukaemia</td>
<td>205.1</td>
</tr>
<tr>
<td></td>
<td>myeloma</td>
<td>203</td>
</tr>
<tr>
<td></td>
<td>lymphosarcoma</td>
<td>200</td>
</tr>
<tr>
<td>7</td>
<td>Myeloid dysplasia and aplastic anaemia</td>
<td>284</td>
</tr>
<tr>
<td>8</td>
<td>Mental retardation and microcephaly developed by a child if the period of his/her prenatal development took place when the mother was in the zone of the Chernobyl accident in 1986 or in the first half of 1987</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Defects of development which were formed in the main period of organogenesis during the pregnancy taking place in the second part of 1986 or in the first half of 1987</td>
<td></td>
</tr>
</tbody>
</table>

*References on the ICD-9 were absent in the text of the table in the original publication (see source). These numbers were included for the purposes of this document.


In many cases, even when the SREC declared the absence of causation between the disease and event, this causation was nevertheless recognized through a decision by the court. The courts, in their decision-making, are too often led by social or political considerations rather than scientific expertise.

### A.4.5 Radiation Workers

The list of occupational diseases currently in force was approved by Executive Order No. 90 of the Ministry of Public Health of 14 March 1996 as amended on 11 September 2000 and 6 February 2001. For selected diseases the Executive Order defines the hazards of the working environment (such as hazardous chemicals, physical factors, biological factors) and practices where the levels of these factors may be significant. The radiation-linked diseases included in this list are presented in table A.8.

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18 Executive Order of the Ministry of Public Health No. 90, Realization of preliminary and periodic medical surveys of workers and medical rules of the admission to work, 1996.
Table A.7 Occupational diseases defined as linked to the Chernobyl accident in the Russian Federation in 1997–99 (1997 Chernobyl List)

<table>
<thead>
<tr>
<th>No.</th>
<th>Diseases</th>
<th>Time of development</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Protracted neurotic status and depression with expressed vegetative dysfunction, decrease of sexual potency, pathological (hypochondriacal) development of personality, symptoms of decreased memory, attention or intellectual ability</td>
<td>During work in the 30-km zone of accident or within 2 years after leaving the work here</td>
</tr>
<tr>
<td>2</td>
<td>Neuro-circulation dystonia with expressed fluctuation of blood pressure, frequent vegetative crises of vessels and stable astenic (cerebro-astenic) failures</td>
<td>During work in the 30-km zone of accident or within 2 years after leaving the work here</td>
</tr>
<tr>
<td>3</td>
<td>Chronic progressive failures of brain blood circulation with neurological symptoms and mental dysfunctions</td>
<td>Within 5 years after leaving the work in the 30-km zone of accident</td>
</tr>
<tr>
<td>4</td>
<td>Hypertension with progressive development, frequent acute conditions and other complications</td>
<td>Within 5 years after leaving the work in the 30-km zone of accident</td>
</tr>
<tr>
<td>5</td>
<td>Progressive ischaemic heart disease with frequent attacks of stenocardia in tension and at rest complicated by acute disorders of coronary blood circulation, grave failures of heart rhythm or acute or progressive chronic coronary deficiency</td>
<td>Within 5 years after leaving the work in the 30-km zone of accident</td>
</tr>
<tr>
<td>6</td>
<td>Chronic non-specific diseases of the respiratory organs with frequent acute conditions and progressive breathing deficiency</td>
<td>Within 5 years after leaving the work in the 30-km zone of accident</td>
</tr>
<tr>
<td>7</td>
<td>Stomach ulcer and duodenal ulcer with frequent acute conditions and complications</td>
<td>Within 5 years after leaving the work in the 30-km zone of accident</td>
</tr>
<tr>
<td>8</td>
<td>Diffuse toxic goitre, nodular goitre and autoimmune thyroiditis</td>
<td>Within 5 years after leaving the work in the 30-km zone of accident</td>
</tr>
<tr>
<td>9</td>
<td>Acute and chronic radiation syndrome, radiation cataract, local radiation injuries (radiation burns)</td>
<td>No time discrimination</td>
</tr>
<tr>
<td>10</td>
<td>Erythro-myeloid dysplasia, aplastic anaemia, acute and chronic myeloid leukaemia</td>
<td>No time discrimination</td>
</tr>
<tr>
<td>11</td>
<td>Solid malignant neoplasms developed by witnesses of the accident and emergency workers of the years 1986–87</td>
<td>No time discrimination</td>
</tr>
</tbody>
</table>


The decision-making process for compensation of diseases of Radiation Workers requires the consideration of his/her dose related to work, and the occupational and health history prior to the development of the disease. The decision-maker is, in this case, one of two centers for occupational diseases, either that based at Clinical Hospital No. 6 (Moscow) or the Medical Sanitary Department No. 71 (Ozersk). The assessment is made by reference to the medical and hygienic occupational history of the worker. All Radiation Workers are covered by a special medical care scheme on the basis of specialized hospitals. As a rule, all the workers working in one branch of any facility are treated by the same therapist (“workplace doctor”), who has an additional qualification in the field of occupational health and in treating occupational diseases. At least once every two years all Radiation Workers must be examined by a workplace doctor, with assistance from another medical specialist as defined by Executive Order No. 83 of the Ministry of Health and Social Development of 16 August 2004. Every five years a number of Radiation Workers from separate workplaces are examined in hospitals with special clinics competent in radiation diseases.

As shown in table A.8, occupational diseases are treated as multifactorial and for their causation all aspects of employment should be examined, including:

- retrospective analysis of the working environment;
- sanitary hygienic history of the relevant working places;
- retrospective analysis of the health status of the workers from a corresponding workplace;
- the habit status (including smoking history).

SREC\$ do not formally (in terms of probability theory) assess the probability of causation as a tool in the decision-making process. The main difficulty in the implementation of these procedures is the quality of dosimetry information available for analysis. The problems are uncertainties in the radiation measurements, significant gaps in dosimetry records for Radiation and ESC Workers, inadequacy of existing radiation monitoring data and the definition of individual dose. All these problems could be solved by the reconstruction of the personal dose for each applicant, but this approach is not widely used because of a lack of resources.

*ICD-9 includes “Cataract associated with radiation and other physical influences” as disease with code 366.46.

**This includes not only the liver, but all digestive organs and peritoneum.

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**Table A.8 Occupational diseases defined as radiation-linked in the Russian Federation**

<table>
<thead>
<tr>
<th>Occupational diseases</th>
<th>ICD-9 code</th>
<th>Risk factors</th>
<th>Activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>3. Caused by physical factors</td>
<td>90</td>
<td>Acute exposure to external radiation or acute intake of a large amount of radioactive material</td>
<td>All types of work with radioactive material and radiation sources</td>
</tr>
<tr>
<td>3.1. Caused by ionizing radiation</td>
<td>990</td>
<td>Local exposure to external penetrating radiation or radioactive material</td>
<td>All types of work with radioactive material and radiation sources</td>
</tr>
<tr>
<td>3.8 Cataract</td>
<td>366.2, 366.9*</td>
<td>Exposure to radiation (non-ionizing, X-ray, photon, neutron and proton radiation)</td>
<td>Welding, production of glass, work with sources of non-ionizing and ionizing radiation</td>
</tr>
<tr>
<td>7. Tumours</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(a) Skin cancers</td>
<td>172, 173</td>
<td>Exposure to products of exploitation of coal, oil, etc.; exposure to ionizing radiation</td>
<td>Work with products mentioned above, work with radiation sources</td>
</tr>
<tr>
<td>(b) Malignant neoplasm of lip, oral cavity and pharynx</td>
<td>140–149</td>
<td>Exposure to compounds of Ni, Cr, As, coal tar, asbestos, asphalt, inhalation of radioactive minerals and dust with adsorbed particles of carbon plastics</td>
<td>Work with products mentioned above, work with radiation sources including survey, mining and milling of radioactive minerals</td>
</tr>
<tr>
<td>(c) Malignant neoplasm of liver</td>
<td>150–159**</td>
<td>Exposure to vinyl chloride and chronic exposure to liverotrophic radionuclides (Po, Th, Pu)</td>
<td>Work with products mentioned above, work with radioactive material mentioned above</td>
</tr>
<tr>
<td>(d) Malignant neoplasm of lymphatic and haematopoietic tissue</td>
<td>200–208</td>
<td>Exposure to benzol, exposure to ionizing radiation</td>
<td>Work with benzol and work with radiation sources</td>
</tr>
<tr>
<td>(e) Malignant neoplasm of bone and articular cartilage</td>
<td>170</td>
<td>Chronic exposure to osteotrophic radionuclides (Ra, Sr, Pu)</td>
<td>Work with radioactive material mentioned above</td>
</tr>
</tbody>
</table>
A.4.6 Input data
The input data for the cases considered by the SREC consists of:

1. Exposure history of the applicant:
   • related to a radiation event in the case of an ESC Worker;
   • related to the full occupational history in the case of a Radiation Worker.

2. Employment history of the applicant, including not only the records of his/her work with sources of ionizing radiation, but also of work with hazardous materials (chemical, biological, etc.).

3. Health history of the applicant, including the results of his/her medical examinations in the course of occupational health surveys.

A.4.7 Compensability
In order to determine the level of compensation awarded, the Medical Social Expert Commissions of the Ministry of Health and Social Development determine the degree of disability of the worker due to the occupational disease developed. The value of compensation depends on the degree of disability of the worker due to his/her occupational disease, as follows:

- III. degree of disability (loss of 25 per cent of working capacity);
- II. degree of disability (loss of 50 per cent of working capacity);
- I. degree of disability (loss of 75 per cent of working capacity).

A.4.8 Settlement value
When awarded, compensation under the Russian Federation system includes:

- special free-of-charge medical assurance;
- direct payments and social guarantees for employees;
- non-direct payments in the form of reduced taxes or free-of-charge services (municipal transport, municipal housing, electricity, etc.).

The total value of the award depends on the degree of disability suffered and the previous salary of workers. In case of death of the worker due to his/her occupational disease the special payment to his/her heirs is assumed in some cases which depend on family financial status.

A.4.9 Summary of important features: Workers Exposed in Special Circumstances

- Comprehensive approach. The compensation scheme is based on medical and social approaches to solving the problem of causation of occupational diseases. It includes the compensation of harm related to stochastic and deterministic effects of radiation developed by an ESC Worker. The compensation scheme was developed to solve a specific social problem and its implementation has helped to address compensation issues for a huge number of ESC Workers in a short period of time. This was particularly important in the early 1990s because of the dramatic decline in the standard of living for most Russian citizens.
• **Scientific basis and generosity.** The compensation scheme is not optimal from a scientific point of view, because the most significant factor in the development of the diseases compensated was not radiation exposure but rather the outcome of the social changes that took place in the Russian Federation in the 1990s. No risk consideration of dose is made; therefore, some cancers unrelated to occupational radiation exposure will be compensated. The line “Neoplasm” of the current list of diseases given in table A.1 and defined as linked to the Chernobyl accident, the accident at the production association Mayak (1957) and dumps of radioactive waste products into the Techa River needs correcting by removing “Neoplasm” and including the more specific notation “Malignant neoplasm and myeloid leukaemia”.

**A.4.10 Summary of important features:**

**Radiation Workers**

• **Taking account of multifactorial causation.** The compensation scheme is based on a medical approach to solving the problem of causation of occupational diseases. It includes the compensation of harm related to stochastic and deterministic effects of radiation developed by Radiation Workers. It was developed to solve the problem of causation of occupational diseases so that these are treated as multifactorial diseases that may be caused not only by radiation, but also by other factors in the working environment. The scheme also jointly considers chemical and physical agents, which maximizes the opportunities for compensation for workplace exposure.

    The compensation scheme is based on a medical approach to solving the problem of causation of occupational diseases, so human factors and subjectivity may play too great a role in decision-making.
A.5 The French compensation programme

A.5.1 Origin of the compensation system and its principles

In France, the compensation system set up for occupational injuries and diseases is largely influenced by its history. In fact, in response to the difficulties encountered at the end of the nineteenth century for the compensation of occupational injuries, a law was adopted (9 April 1898) involving significant social progress.

Before this law, the injured worker was obliged to establish proof of his/her employer’s fault and the link between this fault and the damage in order to receive compensation. The process was long and the worker was disadvantaged in obtaining the proof because of lack of means to do it.

In this context, the promulgation of this law reflects a social compromise where:

• automatic compensation is given to the injured worker (even if there is no fault on the part of his/her employer); there is no need to establish proof of the fault on the part of the employer (this is called the “presumption of attributability” of the damage according to the working conditions);
• in some circumstances employers avoid fully compensating the damage (there is a lump sum for the compensation) and benefit from an immunity with regard to their “civil responsibility” because the injured worker has no other possibility to get a compensation;
• the injured worker may benefit from additional compensation if they can prove an unforgivable fault on the part of his/her employer.

On the same principles, a new law was adopted on 25 October 1919 concerning the compensation of occupational diseases.

Initially based on an agreement between employers and employees, this system was transferred to the general social security system in 1946. Employers now have to pay a premium to the social security system. Calculation of this premium is related to the number of injuries and diseases associated with the firm.

In order to allow an automatic compensation for occupational disease, the following principles have been established: “all diseases mentioned in a ‘table’ and contracted in the conditions mentioned in this table are automatically considered as occupationally related”. Therefore, workers do not have to prove the link between the work and the occurrence of their diseases as long as they respect the conditions mentioned in the “table”: i.e. exposure period of the disease, characteristics of the disease, list of eligible working activities, and in some cases the duration of exposure.

In fact, all these elements explain the importance of the social negotiation associated with the elaboration of the compensation system and its current evolution.

A.5.2 Eligibility for diseases associated with ionizing radiation

A dedicated table of diseases induced by ionizing radiations was created on 4 January 1931. Modifications were regularly introduced in order to expand the charac-
teristics of the diseases eligible for compensation as well as the list of working activities, and the last modification was adopted on 26 June 1984.\textsuperscript{20}

Currently, the eligible diseases and the associated exposure period are presented in table A.9.

**Table A.9 List of diseases related to ionizing exposure eligible for recognition in the compensation system**

<table>
<thead>
<tr>
<th>Designation of the disease</th>
<th>Exposure period</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anaemia, leucopenia, thrombopenia, or haemorrhagic syndrome due to acute exposure</td>
<td>30 days</td>
</tr>
<tr>
<td>Anaemia, leucopenia, thrombopenia, or haemorrhagic syndrome due to chronic exposure</td>
<td>1 year</td>
</tr>
<tr>
<td>Blepharitis or conjunctivitis</td>
<td>7 days</td>
</tr>
<tr>
<td>Keratitis</td>
<td>1 year</td>
</tr>
<tr>
<td>Cataract</td>
<td>10 years</td>
</tr>
<tr>
<td>Acute radiodermatitis</td>
<td>60 days</td>
</tr>
<tr>
<td>Chronic radiodermatitis</td>
<td>10 years</td>
</tr>
<tr>
<td>Acute radio-epithelitis of the mucous membrane</td>
<td>60 days</td>
</tr>
<tr>
<td>Chronic radiolesions of the mucous membrane</td>
<td>5 years</td>
</tr>
<tr>
<td>Bone radionecrosis</td>
<td>30 years</td>
</tr>
<tr>
<td>Leukaemia</td>
<td>30 years</td>
</tr>
<tr>
<td>Primary lung cancer due to inhalation</td>
<td>30 years</td>
</tr>
<tr>
<td>Bone sarcoma</td>
<td>50 years</td>
</tr>
</tbody>
</table>

In addition, the list of working activities considered as potentially for inducing the occupational diseases associated with ionizing radiation is indicative and concerns all activities with exposure to X-rays or radioactive substances (naturally occurring or artificial), or to any other emitted sources, notably:

- Mining and milling activities with radioactive materials.
- Preparation of radioactive substances.
- Preparation of radioactive chemical or pharmaceutical products.
- Preparation and application of radiferous luminescent products.
- Research activities or measurements on radioactive substances and X-rays in laboratories.
- Activities inducing occupational exposures to ionizing radiation in hospitals, sanatoriums, private clinics, dispensaries, doctors’ surgeries, dentists’ surgeries, radiology units, private hospitals, cancer treatment centres.
- Activities in all industries and trade using X-rays, radioactive substances, substances or processes emitting ionizing radiation.

According to the severity of the disease, the compensation obtained by the worker is as follows:

- Reimbursement of medical care.
- Temporary indemnities associated with work-days lost.
- Permanent compensation for disability (lump sum or annual premium defined according to the degree of severity of the permanent disability).

\textsuperscript{20} Décret No. 96-445 du 22 mai 1996 modifiant et complétant les tableaux de maladies professionnelles annexes au livre IV du code de la Sécurité sociale, J.O.
A.5.3 Population concerned

Although the specific number of people exposed to ionizing radiation and covered by the compensation system is not defined precisely, one can refer to the number of persons included in the surveillance system for occupational external exposure to ionizing radiation. In 2004, this surveillance system covered 255,321 persons (Rannou and Couasnon, 2005), broken down as follows:

- Nuclear industry 57,781
- Non-nuclear industry 29,174
- Research activities 17,747
- Medical sector 140,092
- Other activities 10,527

A.5.4 Compensation statistics

Table A.10 presents the evolution of the number of compensated occupational diseases due to ionizing radiation from 1994 to 2003 for the “General Compensation System” (CNAMTS, 2005). Table A.11 sets out, for the year 2003, the distribution of compensated diseases due to ionizing radiation according to the characteristics of the disease.

Table A.10 Evolution of compensated diseases related to ionizing radiation

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of compensated diseases</td>
<td>21</td>
<td>23</td>
<td>18</td>
<td>9</td>
<td>13</td>
<td>17</td>
<td>20</td>
<td>23</td>
<td>22</td>
<td>28</td>
</tr>
</tbody>
</table>

Table A.11 Type of radiation-induced diseases compensated in 2003

<table>
<thead>
<tr>
<th>Type of disease</th>
<th>Number of compensated cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary lung cancer due to inhalation</td>
<td>13</td>
</tr>
<tr>
<td>Leukaemia</td>
<td>8</td>
</tr>
<tr>
<td>Bone sarcoma</td>
<td>1</td>
</tr>
<tr>
<td>Anaemia due to acute exposure</td>
<td>1</td>
</tr>
<tr>
<td>Cataract</td>
<td>2</td>
</tr>
<tr>
<td>Chronic radiodermatitis</td>
<td>1</td>
</tr>
<tr>
<td>Anaemia due to chronic exposure</td>
<td>2</td>
</tr>
</tbody>
</table>

A.5.5 Extension towards attributability

In order to introduce flexibility into the compensation system for occupational disease in France, a complementary system was created in 1993, allowing compensation of diseases when the disease or the activity is not listed in the table or when the limit or the duration of exposure is not respected. For this purpose, a dedicated commission is mandated to examine the requests.

In this perspective, it is possible to get compensation for a disease not listed in the table only if the occurrence is mainly due to occupational exposure and if the degree of disability associated with the disease is above 25 per cent.
Currently, only 3.5 per cent of the total number of compensated occupational diseases is recognized through this complementary system, and, as far as we know, no compensation for diseases induced by ionizing radiation has been established through this mechanism. Nevertheless, the issue of assigned share for radiation-induced cancers, except for those currently in the list, could be discussed in the future.

A.6 Legal provisions applicable to workers affected by occupational exposure to ionizing radiation in the Argentine Republic

A.6.1 Introduction

Regulatory legislation in Argentina\(^{21}\) and the relevant implementing regulations comprise the body of norms that set forth safety requirements for workers exposed to ionizing radiation.

Labour laws in Argentina\(^{22}\) and the relevant implementing and supplementary norms set forth the conditions for claims and compensation in respect of occupational accidents and diseases. Accordingly, in its article 1, the Act on Occupational Risks enumerates the following objectives:

- to reduce occupational accidents by preventing occupational risks;
- to make good the harm caused by occupational accidents and diseases, and to provide rehabilitation for injured workers;
- to foster collective bargaining to improve preventive measures and the reparation provided.

Ionizing radiation is listed in the implementing regulations of the Act on Occupational Risks\(^{23}\) as one risk factor which may cause the occupational diseases restrictively enumerated in the said norm for purposes of compensation. The list of occupational diseases identifies the following elements:

- risk factors
- clinical picture
- exposure
- measures to determine the occupational disease.

Accordingly, the disorders listed in the norm shall be considered as occupational only if they are caused by certain “risk factors” and in specific activities. The legislation in question covers cases in which workers exposed to ionizing radiation claim damages or compensation on the grounds that their disorders may be attributed to exposure to ionizing radiation.

As a general rule, the legislation sets forth a schedule of damages for occupational disorders caused by ionizing radiation, in accordance with the degree of disability combined with the worker’s age and the wage received.

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\(^{21}\) National Act on Activities in the Nuclear Sphere, Act 24.804, articles 7, 8 and 16 (paragraph L).

\(^{22}\) Act 24.557, “Act on Occupational Risks” (AOR).

A.6.2 Legal system of compensation: 
Normative framework 

Act 24.557, “Act on Occupational Risks” (AOR), was adopted in 1995 and came into force on 1 July 1996; it is made up of 15 chapters, five supplementary provisions and three final provisions and is enforceable in respect of all industrial relations, both public and private. It is almost impossible within the scope of this report to refer to and analyse all the norms that derive from AOR; accordingly, we shall examine those aspects which in our opinion are most relevant to this report.

A.6.3 Objectives of the Act on Occupational Risks 

The Act starts with a statement of its aims, which are (a) prevention of occupational risks, and (b) redress for occupational injury. 

The initial purpose of AOR is prevention, in other words the adoption of deliberate health and safety measures to ensure that work does not cause accidents that cause injury to workers.

Accordingly, the Act itself and the extensive enabling norms develop a preventive system to ensure that entrepreneurs comply with their duties in respect of occupational hygiene and safety, based on the duty to protect those under their responsibility from harm; the Occupational Risk Insurance Agencies (ORIA) are responsible for monitoring the system.

In its article 4, AOR lays down the obligation to adopt such measures as are provided for by law to ensure effective protection against occupational risks. The legal hygiene and safety regulations are derived from Act 19.587,\(^{24}\) which is itself the subject of detailed regulations in each province.

In addition, specific hygiene and safety regimes, specific regulations and specific hygiene and safety norms, and so on, may be required by the collective agreements of each enterprise.

In requiring that risks be effectively addressed, the law lays down the obligation to produce results; it is thus not sufficient for those liable for any injury to claim that they have taken appropriate measures if, despite such measures, the risk actually did cause harm.

A second objective, in case of failure to prevent the risk, and when an event that causes injury occurs, is redress for the occupational injury. Such redress is to be provided in the form of compensation in kind or financial compensation such as is provided for by the system to make up for any physical and/or financial injury to the victims resulting from the occupational accident.

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\(^{24}\) Regulatory Decree N° 351/1979, relating to the Occupational Hygiene and Safety Act N° 19.587, in its article 62, paragraph 2 states as follows: “2. The National Atomic Energy Commission is the competent authority for the implementation of Act 19.587 in respect of the use or application of radioactive materials, nuclear materials and particle accelerators whose main purpose is not specifically to generate X-rays, of ionizing radiation originating therefrom and of nuclear reactions or transmutations; it shall be authorized to process and issue, to persons responsible for such practices or operations, licences and specific permits governing their siting, building, bringing into operation, running and final closure.” With the adoption of Decree 1540/94 and subsequently Act 24.804, the Nuclear Regulatory Authority took over responsibility for regulating nuclear activities which were previously the responsibility of the National Atomic Energy Commission.
A.6.4 Compulsory and self-insurance

AOR requires employers to take out insurance with an ORIA – in which it vests responsibility for the management of worker health, safety and prevention policies together with the provision of welfare measures. Thus, the law requires anyone who contracts workers who come within its scope to join an ORIA, although it also allows them to take out self-insurance, i.e. it relieves them of the obligation to take out such insurance with an ORIA, while requiring them to provide it themselves; however, in order to avail themselves of this possibility, they must satisfy certain requirements which are laid down by the Act.

A.6.5 Occupational illnesses and accidents covered

Occupational illnesses and accidents constitute those contingencies covered by article 6 AOR, to which the system will respond by providing financial and/or welfare benefits to workers who have suffered injury not caused by themselves and which result in any pathological disorder (pain, illness, disability), whether the causative factor is a typical accident (sudden and violent) or the effect of a risk factor identified in the list of disorders.

Table A.12, which is contained in Annex I of Decree 658/96, regulating AOR, specifically includes ionizing radiation among the possible causes of occupational diseases that may give rise to compensation.

<table>
<thead>
<tr>
<th>Diseases</th>
<th>Occupational activity potentially responsible for exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anaemia, leucopenia, thrombocytopenia, or haemorrhagic syndrome following acute radiation</td>
<td>List of activities in which exposure may occur: All activities involving exposure to X-rays or to natural or artificial radioactive substances and to any source of corpuscular emission or of radiation in particular</td>
</tr>
<tr>
<td>Anaemia, leucopenia, thrombocytopenia, or haemorrhagic syndrome following chronic radiation</td>
<td>Extraction and processing of radioactive minerals</td>
</tr>
<tr>
<td>Blepharitis or conjunctivitis</td>
<td>Preparation of radioactive compounds including radioactive chemicals and drugs</td>
</tr>
<tr>
<td>Chronic keratitis</td>
<td>Preparation and application of phosphorescent radioactive products</td>
</tr>
<tr>
<td>Cataract</td>
<td>Manufature and use of radiotherapy and X-ray equipment</td>
</tr>
<tr>
<td>Acute radiodermatitis</td>
<td>All activities in hospitals, sanatoriums, policlinics, clinics and dental clinics in which health workers are exposed to the effects of X-rays</td>
</tr>
<tr>
<td>Chronic radiodermatitis</td>
<td>Industrial radiography using X-ray equipment or other sources of gamma radiation emission</td>
</tr>
<tr>
<td>Acute radiation lesions of the mucous</td>
<td>Plants producing radioactive isotopes</td>
</tr>
<tr>
<td>Chronic radiation lesions of the mucous</td>
<td>Nuclear power stations</td>
</tr>
<tr>
<td>Necrosis of the bones caused by radiation</td>
<td></td>
</tr>
<tr>
<td>Leukaemia</td>
<td></td>
</tr>
<tr>
<td>Incipient cancer of the bronchi or lungs caused by inhalation</td>
<td></td>
</tr>
<tr>
<td>Bone sarcoma</td>
<td></td>
</tr>
<tr>
<td>Skin cancer</td>
<td></td>
</tr>
<tr>
<td>Reproductive disorders: oligo- or azoospermia, miscarriage</td>
<td></td>
</tr>
</tbody>
</table>

The risk factor or harmful agent – ionizing radiation – if so confirmed, is the factor that harms workers’ health and may make them ill (through their occupation, given its nature) not suddenly and violently, but slowly and gradually and, as a rule, not visibly but invisibly and silently.

The same legislation also provides for the possibility of treating as occupational illnesses other disorders not included in those listed in Decree 658/96, in which case an ad hoc Medical Board is convened to determine whether the condition is directly and immediately the result of the work performed, to the exclusion of factors that may be
ascribed to the worker, such as predisposition or susceptibility to contracting a particular illness, and of factors alien to the work.

We should point out that occupational accidents and illnesses are exempt from coverage by ORIA or by self-insured employers if:

• the occupational accident or illness is the result of the worker’s own misconduct or of a factor alien to the work environment;
• the disability affecting the worker was present before he or she was recruited and was identified at the recruitment examination. In this case, ORIA shall not be liable for any disabilities present at the outset of the employment contract, but only proportionally; in other words, if the condition is worsened by a risk factor and in an activity listed in Decree 658/96, ORIA will bear liability for the new, additional injury, but not for full injury.

A.6.6 Civil liability

AOR does not allow the possibility of resorting to the courts of law via a civil liability case against employers. In this respect, article 39, paragraph 1 AOR stipulates that the system’s benefits exonerate employers from any civil liability, unless the injury has been caused by their neglect. The legislator’s decision not to extend to workers the same general right as is given to all Argentina’s citizens, and even to temporary residents, has given rise to numerous decisions by the courts declaring the article to be unconstitutional.

Article 39, paragraph 1 AOR having been declared unconstitutional, it is thus possible under the Civil Code to file a suit for full damages against an employer, whereas AOR sets a scale of damages with ceilings. Acceptance by the courts of a civil suit for damages does not exempt ORIA from the financial compensation and compensation in kind for which the Act makes it liable.

This system does not exclude the possibility of a parallel claim under the Vienna Convention on Civil Liability for Nuclear Damage, which Argentina signed in 1966, and to which the National Act on Activities in the Nuclear Sphere – Act 24.804 – explicitly refers.

A.6.7 Procedural aspects

The system introduced by AOR\(^{25}\) derives from a special procedure whereby it represents the sole administrative mechanism for resolving factual and legal conflicts, subject to review by the courts.

The procedure is set in motion by a “complaint” which is admissible if it is based on an event that may give rise to benefits under the system, i.e. a contingency covered by the Act (an occupational accident or illness).

Acceptance of the complaint, whether expressly or tacitly, implies acceptance, i.e. acknowledgement of legal liability and recognition that:

• the accident took place and is an occupational one;
• none of the exemptions provided for by the law are applicable;
• the occupational illness concerned actually exists, is of an occupational nature and is among those in the relevant list;
• the action is not a violation of the legal provisions.

\(^{25}\) Articles 21, 22 and 46. Regulatory Decree 717/96.
Acceptance covers only these aspects; where secondary aspects such as the existence or not of any disability, the type and degree thereof and the appropriate benefits are concerned, these are not the responsibility of the complainant, as they come within the competence of ORIA or of the self-insured employer, who must provide details of:

- the medical diagnosis;
- the type of occupational disability (if appropriate, as the contingency may be recognized, even in the absence of any disability)
- the nature (temporary or permanent, provisional or definitive) of the occupational disability and its degree;
- the content and scope of the benefits in kind to be awarded (which should apply as from the time of the complaint regarding the contingency, in conformity with the provisions of Decree 717/96, article 5).

This system makes it possible for victims to refuse to accept the decision by requesting the intervention of the Medical Boards, whose remit is laid down in article 21 AOR.

### A.6.8 Applicable nuclear regulatory norms

In developing its system of protection for workers subject to occupational exposure, Argentina based its regulatory norms on the recommendations of various international agencies, and in particular those of the International Commission on Radiological Protection (ICRP).

These international recommendations include in particular the Basic Safety Standards for Radiation Protection and for the Safety of Radiation Sources, which are co-sponsored by the UN Food and Agriculture Organization (FAO), IAEA, ILO, OECD/NEA, Pan American Health Organization (PAHO) and WHO, and the recommendations of the ICRP. One of the principles the Argentine norms adhere to is that radiation protection requirements must not be replaced by privileges or compensation for workers.

By virtue of the remit assigned to it by the National Act on Activities in the Nuclear Sphere, Act 24.804, the Nuclear Regulatory Authority (NRA) is the State’s national technical agency responsible for controlling and regulating all areas of nuclear activity in respect of radiological and nuclear safety.

Argentine legislation stipulates that NRA is the implementing authority in respect of hygiene and safety in the use and application of radioactive material, nuclear material and particle accelerators whose fundamental purpose is not specifically the production of X-rays and of ionizing radiation originating therefrom, and of nuclear reactions or transformations.

### A.6.9 Legal precedent

The National Atomic Energy Commission and a number of its subsidiaries have been defendants in numerous lawsuits in different provinces in Argentina for damage and compensation brought by workers on grounds of cancers caused by exposure to ionizing radiation, as well as legal action by members of the public alleging a causal relationship between their illness and the nuclear activities carried out by the defendant.

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These cases have established no causal effect – with regard to ionizing radiation received within the limits laid down by regulatory legislation on nuclear activities – between harm and exposure to such radiation; consequently, no proven causality has been determined by law between the nuclear activity carried out and the disorders contracted and denounced by the plaintiffs.

This comes within the theory of causality known as “sufficient causal relationship” which, even in those cases where Argentine legislation adopts the theory of objective liability, forms an unavoidable background that has to be taken into account.

Before the promulgation of AOR, the verdicts of the court formed part of the case law on exposure to ionizing radiation from nuclear activities. Since the adoption of the Act, the legal departments of the government agencies responsible for the application of regulations on ionizing radiation from nuclear activities have not heard of any further claims of this kind.

A.6.10 Summary of important features

Argentine regulatory legislation is based on the recommendations of ICRP, as are its norms, and it has determined that since it ensures compliance with the dose thresholds laid down by the radiological protection system for workers, there is no need for any special administrative regulations in respect of the radiation risks concerned.

In conformity with the provisions of Argentine legislation, we may conclude that there have been no cases of compensation associated with the development of illnesses simply caused by work in facilities using ionizing radiation; in other words, application of the common system of Argentine legislation requires proof that the threshold doses for the activity have been exceeded.
Appendix B: Global average occupational exposure and average radiation dose from natural sources

The global average annual effective doses from occupational exposure are the subject of continuing examination by UNSCEAR, and were most recently tabulated in its 2000 report (UNSCEAR, 2000). Occupational radiation exposures were assessed from data submitted to the Committee by national authorities in response to questionnaires. Exposures from man-made sources were given the most attention; countries usually record such data for regulatory purposes. Where average exposures over a workforce were needed, the number of workers was taken to be the number of workers monitored. The estimates of occupational radiation exposure in UNSCEAR’s 2000 report benefited from a much more extensive and complete database than was previously available; this led to improved estimates of occupational doses. The current estimates are summarized in table B.1. Data are summarized for five categories of occupational exposure to man-made sources and five categories of exposure to enhanced natural sources. Data shown are averaged over five-year periods where available (1975–79; 1980–84; 1985–89; 1990–94). More recent periods are covered in a subsequent UNSCEAR report, but the data were not available when this document was produced.

The comparison of five-year average data for various occupations exposed to man-made sources reported for 1975–94 allows examination of trends in exposure.

The Committee’s current estimate of the worldwide collective effective dose to workers from man-made sources for the early 1990s, 2,700 man Sv, is lower by a factor of about 2 than that made by the Committee for the late 1970s. A significant part of the reduction came in the nuclear power fuel cycle, in particular in uranium mining. However, reductions were seen in all the main categories: industrial uses, medical uses, defence activities and education. This trend is also reflected in the worldwide average annual effective dose, which has fallen from about 1.9 mSv to 0.6 mSv.

No attempt was made to deduce any trend in the estimates of dose from occupational exposure to enhanced natural sources of radiation, as the supporting data were somewhat limited. From that source, UNSCEAR made a crude estimate of about 20,000 man Sv in its 1988 report, which was subsequently revised downward to 8,600 man Sv in its 1993 report (UNSCEAR, 1988, 1993). The comparable figure for 1990–94 is 5,700 man Sv; however, an important new element has been added for this period, namely, occupational exposure to elevated levels of radon and its progeny. This brought the overall estimate of collective occupational dose from enhanced natural sources to 11,700 man Sv. This is still considered at this stage to be a crude estimate. The data suggest, however, that 80 per cent of occupational exposure may be from enhanced natural as opposed to man-made sources. Caution should be used in interpreting this percentage in the context of compensation, as organ dose can vary substantially from effective dose.

For comparison, the worldwide annual average effective dose from natural sources is 2.4 mSv. The annual worldwide per caput effective dose is determined by
adding the various components, as summarized in table B.2. While the annual global per caput effective dose due to natural radiation sources is 2.4 mSv, the extent of variation in the individual doses that comprise this is wide. In any large population about 65 per cent would be expected to have annual effective doses between 1 mSv and 3 mSv, about 25 per cent of the population would have annual effective doses less than 1 mSv and 10 per cent would have annual effective doses greater than 3 mSv.

Table B.1 Occupational radiation exposures

<table>
<thead>
<tr>
<th>Source/practice</th>
<th>Number of monitored workers</th>
<th>Average annual effective dose (mSv)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Man-made sources</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nuclear fuel cycle (including mining)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1975–79</td>
<td>560</td>
<td>4.1</td>
</tr>
<tr>
<td>1980–84</td>
<td>800</td>
<td>3.7</td>
</tr>
<tr>
<td>1985–89</td>
<td>880</td>
<td>2.9</td>
</tr>
<tr>
<td>1990–94</td>
<td>800</td>
<td>1.8</td>
</tr>
<tr>
<td>Industrial uses of radiation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1975–79</td>
<td>530</td>
<td>1.6</td>
</tr>
<tr>
<td>1980–84</td>
<td>690</td>
<td>1.4</td>
</tr>
<tr>
<td>1985–89</td>
<td>560</td>
<td>0.9</td>
</tr>
<tr>
<td>1990–94</td>
<td>700</td>
<td>0.5</td>
</tr>
<tr>
<td>Defence activities*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1975–79</td>
<td>100</td>
<td>1.3</td>
</tr>
<tr>
<td>1980–84</td>
<td>120</td>
<td>0.7</td>
</tr>
<tr>
<td>1985–89</td>
<td>130</td>
<td>0.7</td>
</tr>
<tr>
<td>1990–94</td>
<td>140</td>
<td>0.2</td>
</tr>
<tr>
<td>Medical uses of radiation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1975–79</td>
<td>1,300</td>
<td>0.8</td>
</tr>
<tr>
<td>1980–84</td>
<td>1,900</td>
<td>0.6</td>
</tr>
<tr>
<td>1985–89</td>
<td>2,200</td>
<td>0.5</td>
</tr>
<tr>
<td>1990–94</td>
<td>2,320</td>
<td>0.3</td>
</tr>
<tr>
<td>Education/veterinary</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1990–1994</td>
<td>360</td>
<td>0.1</td>
</tr>
<tr>
<td><strong>Total from man-made sources</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1975–79</td>
<td>2,490</td>
<td>1.9</td>
</tr>
<tr>
<td>1980–84</td>
<td>3,510</td>
<td>1.4</td>
</tr>
<tr>
<td>1985–89</td>
<td>3,770</td>
<td>1.1</td>
</tr>
<tr>
<td>1990–94</td>
<td>4,320</td>
<td>0.6</td>
</tr>
<tr>
<td><strong>Enhanced natural sources (all data for 1990–1994)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Air travel (crew)</td>
<td>250</td>
<td>3.0</td>
</tr>
<tr>
<td>Mining (other than coal)</td>
<td>760</td>
<td>2.7</td>
</tr>
<tr>
<td>Coal mining</td>
<td>3,910</td>
<td>0.7</td>
</tr>
<tr>
<td>Mineral processing</td>
<td>300</td>
<td>1.0</td>
</tr>
<tr>
<td>Above ground workplaces (radon)</td>
<td>1,250</td>
<td>4.8</td>
</tr>
<tr>
<td><strong>Total from natural sources</strong></td>
<td>6,500</td>
<td>1.8</td>
</tr>
</tbody>
</table>

*Data from table 38, p. 631 of UNSCEAR (2000, Vol. 1).
Table B.2. Average radiation dose from natural sources

<table>
<thead>
<tr>
<th>Source</th>
<th>Worldwide average annual effective dose (mSv)</th>
<th>Typical range (mSv)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>External exposure</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cosmic rays</td>
<td>0.4</td>
<td>0.3–1.0</td>
</tr>
<tr>
<td>Terrestrial gamma rays²</td>
<td>0.5</td>
<td>0.3–0.6</td>
</tr>
<tr>
<td><strong>Internal exposure</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inhalation (mainly radon)</td>
<td>1.2</td>
<td>0.2–10⁵</td>
</tr>
<tr>
<td>Ingestion</td>
<td>0.3</td>
<td>0.2–0.8⁴</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>2.4</td>
<td>1–10</td>
</tr>
</tbody>
</table>

¹ Range from sea level to high ground elevation. ² Depending on radionuclide composition of soil and building materials. ³ Depending on indoor accumulation of radon gas. ⁴ Depending on radionuclide composition of foods and drinking water.
Appendix C: Biological indicators
(“biological dosimetry”)

C.1 Introduction

There are several situations in which the use of biological indicators is helpful in compensation cases after occupational exposure to ionizing radiation:

1. Confirmation or rejection of dubious physical dose measurements.
2. Missing physical dosimetry.
3. Unrecognized radiation accidents in the past.
4. Consideration or assessment, respectively, of individual radiosensitivity.

One of the crucial advantages of biological indicators over physical dosimetry is the fact that biological indicators include individual radiosensitivity. Thus, they do not reflect dose in the physical sense, but they show the response of the body to a specific dose. For this reason scientists working in this field refrain from using the term “biological dosimeters” and prefer the term “biological indicators” instead.

It is common to all biological indicators that they are very useful immediately after an external, high dose rate, acute, whole-body exposure. In all other cases, problems occur that have only partly been overcome by specific modifications of the various techniques.

The following overview is not exhaustive but it does highlight some important aspects of biological indicators, in particular with regard to compensation cases. More detailed reviews are available (ICRU, 2002; IAEA, 2001; Müller and Streffer, 1991). Also, in order to keep this outline concise, the methods will not be described in detail, but reference will be made to relevant publications.

C.2 Techniques

C.2.1 Dicentric chromosomes

These chromosomal aberrations originate from the fusion of two chromosomes, thus presenting a structure with two centromeres instead of one. Usually, a dicentric is accompanied by a fragment.
IONIZING RADIATION EXPOSURE AND COMPENSATION PROGRAMMES

Method
In most cases lymphocytes are used due to the advantage that almost all of the peripheral lymphocytes are in the $G_0$-phase of the cell cycle. Many methods for the preparation of dicentrics have been published. In particular, several organizations have made huge efforts to standardize the procedures used in various laboratories (ICRU, 2002; IAEA, 2001).

Advantages
The dicentrics system is the best studied system of all biological indicators.

- The spontaneous frequency is comparatively low (0.5 to 1 per 1,000 lymphocytes).
- There are only a few chemicals that induce dicentrics; thus the observation of dicentrics is a strong indicator that ionizing radiation is involved.
- Partial body exposures can be taken into account (IAEA, 2001, p. 51).

Disadvantages
- The damage is unstable and gets lost exponentially at a half-life of about three years with marked individual variation.
- Protracted and fractionated exposures are difficult to handle, in particular when no additional information is available (e.g. with respect to the duration of a protracted exposure and the time since exposure); if additional information is available, a rough estimate is possible (IAEA, 2001, p. 56).

Limits
- In individual cases without knowledge of the individual background frequency of dicentrics, the lower level of a significant increase is about 0.1 to 0.2 Gy; if populations are studied, an enhancement after about 0.05 Gy may be detectable.
- The dose-response curve levels off at doses exceeding about 5 Gy low LET radiation.

C.2.2 Micronuclei
Micronuclei originate from the main nucleus during mitosis due to acentric chromosomal fragments (most frequent mechanism after radiation exposure), kinetochore defects, spindle failure or multicentric chromosomes that are fragmented during mitosis. They show up as “small nuclei” in the cytoplasm. All mechanisms need a mitosis in order to express a micronucleus. Thus, it is necessary to prove that a mitosis has actually happened, because otherwise all non-dividing cells will erroneously be counted as cells without a micronucleus. Therefore micronucleus studies should be carried out using cytochalasin B, a fungal toxin that allows division of the cell nucleus, but prevents cell division, resulting in binucleated cells.

Method
The following publications outline the method in detail (IAEA, 2001; Müller and Streffer, 1994; Fenech, 1993).

Advantages
- Scoring of micronuclei is much faster than scoring of dicentrics.
• Training of technicians doing the scoring and automatization of scoring is easier than for dicentrics.
• Because of these advantages, micronuclei are the best choice when rapid screening of many casualties is required.

**Disadvantages**

• The background frequency is clearly higher and more variable than that for dicentrics (for adults about 5 to 40 micronuclei per 1000 binucleated cells).
• Exposures that occurred a long time ago, partial body, protracted and fractionated exposures are clearly more difficult to handle than in the case of dicentrics.

**Limits**

• With the conventional cytochalasin B-based micronucleus assay, the lowest individual dose that can be detected is about 0.3 Gy. This limit is markedly lower when one restricts the analysis to sensitive lymphocyte subpopulations (Wuttke et al., 1993) and/or eliminates the problem of the variability of spontaneous background frequency by using centromere-specific probes (Kryscio et al., 2001).
• Similar to the dicentrics, micronuclei start to level off at doses exceeding about 5 to 6 Gy; however, if one takes into consideration cell proliferation parameters, doses up to about 15 Gy can be detected (Müller and Rode, 2002).

**C.2.3 PCC (premature chromosome condensation)**

A non-mitotic cell can be forced to condense its chromosomes after fusion with a mitotic cell. In particular, cells in the G1-phase of the cell cycle are valuable in this context, because the number of induced chromosomal breaks can be determined and reflects the response to a radiation exposure.

**Method**

A description of the method can be found in IAEA (2001) and Pantelias and Maillie (1984).

**Advantages**

• Results can be obtained quickly (within about 3–4 hours) after an assumed radiation accident.
• There is no problem with regard to mitotic delay or cells not reaching mitosis at all. (Both aspects clearly limit the dicentric and micronucleus assays, in particular after high doses.)
• Partial body exposure is easier to handle using PCC compared with dicentrics.

**Disadvantages**

• As not all cells can be forced into mitosis, there might be a selection bias.
Limits

- It is difficult to find definite information on the lowest dose detectable in an individual. The PCC assay has been reported to be somewhat more sensitive than the dicentric assay and there are techniques using PCC plus FISH to increase sensitivity.
- The assay can be used up to 20 Gy; beyond that, levelling off is observed.

C.2.4 Reciprocal translocations

These result from the mutual exchange of parts of two or more chromosomes. The genomic information is almost unchanged, but the location of the genes is rearranged, sometimes leading to severe diseases (e.g. leukaemia). Reciprocal translocations are stable, meaning that the affected cells can survive and be detected long after radiation exposure.

Method

A detailed description of the method is given in IAEA (2001).

Advantages

- Reciprocal translocations are stable and so comparatively persistent aberrations. Nevertheless they also decline with time, although at a much slower rate than unstable aberrations.

Disadvantages

- Discrimination of low dose levels is inferior to that of dicentrics because of a higher and more variable spontaneous frequency of reciprocal translocations.
- One problem is “clones” of a specific type of reciprocal translocation caused by damaged stem cells that produce daughter cells all showing an aberration.

Limits

- Lifetime doses above about 0.5 Gy in excess of background radiation are detectable for individuals.

C.2.5 EPR (electron paramagnetic resonance; = ESR, electron spin resonance)

In biological material containing no or almost no water (e.g. hair, bones, tooth enamel) very long-lived radicals are induced by ionizing radiation, which can be detected by EPR even years after radiation exposure. One must keep in mind that this method is not a biological indicator in its strict sense, because individual radiosensitivity does not play a role.

Method

A detailed description is given in ICRU (2002).
Advantages

- The assay can be used over a huge dose range (see “Limits”).
- The technique can be applied immediately after radiation exposure.

Disadvantages

- The assay does not take into account individual radiosensitivity (although this may be seen as an advantage by a physicist).
- The assay requires a biopsy; great care must be taken not to induce radicals by the technique used to obtain the material.
- The necessary equipment is expensive and needs experienced personnel.

Limits

- Routine measurements are possible in the dose range of 0.5 to 100 Gy; using specific techniques 0.1 Gy is detectable.

C.2.6 γ-H2AX foci

When a double strand break occurs in a cell, specific positions in the neighbouring histones are phosphorylated. These positions can be detected using antibodies tagged with a fluorescent molecule, then showing up as “foci” under a fluorescence microscope.

Method

Information on the various techniques available can be obtained from Nakamura et al. (2006).

Advantages

- This assay seems to be very sensitive in the low dose range (see “Limits”).

Disadvantages

- There is little experience of using this assay to date.
- In particular, nothing is known about the time dependence of the detectability of the effect or about possible interfering factors.

Limits

- Should the preliminary results hold up after further scrutiny, this assay may be useful to detect effects after very low radiation doses (in the range of about 3 mGy) (Lobrich et al., 2005).

C.2.7 Comet assay

In most cases, this assay is not meant for dose assessment, but for appraisal of individual radiosensitivity. The name “comet” refers to the appearance of the nuclear material after removal of all non-DNA material of a cell and running an electrophoresis. As a result of this procedure, “comet tails” are formed that reflect the amount of damage
in the DNA (strand breaks, conformational changes) after radiation exposure. If one gives the cells the opportunity to repair before lysis takes place, information can be obtained on the repair capacity of the cells of an individual.

**Method**

Many different techniques are described in the literature (Müller et al., 2004; Tice et al., 2000). Some of these methods are designed in a way to detect predominantly specific types of lesions (e.g. single or double strand breaks).

**Advantages**

- One can determine initial DNA damage (i.e. DNA damage without interference by repair enzymes) and DNA repair.
- The comet assay does not measure averages of cell populations (like most other assays detecting DNA damage), but informs about DNA damage in individual cells. Thus, an idea of the distribution of damage and repair and not just averages can be obtained.

**Disadvantages**

- There is still a lot of controversy on the exact type(s) of damage detected with the various methods used.
- Because of repair processes taking place in vivo, initial DNA damage rapidly disappears over time. Thus, the assay is not very useful as a biological indicator of radiation exposure; after several hours, only non-repaired lesions can be detected.

**Limits**

- The assay in its original form easily detects DNA damage down to 0.1 Gy; the limits of detection, however, strongly depend on the specific technique used.

**C.3 Conclusions**

The “universally applicable” biological indicator does not exist. The choice strongly depends on the specific exposure conditions. Quite often, the occupational exposures in compensation cases occurred several years before the expert is asked for an opinion. Thus, reciprocal translocations and EPR are the assays one should primarily take into consideration. If there is the suspicion that a pronounced radiosensitivity might play a role, one should apply the comet assay. Specific exposure conditions might even require methods not mentioned here. For example, if the exposure was very localized, methods using hair from the exposed area can be helpful. In any case, as a result of the drastically reduced radiation research, there are nowadays only very few people worldwide experienced enough to use biological indicators properly.
Appendix D: A quantitative uncertainty analysis approach to estimation of radiation-related risk

The following discussion is based on the report by the NCI/CDC Working Group to revise the 1985 NIH radioepidemiological tables (Land et al., 2003). This report is based mainly, but not solely, on the 1958–1987 Life Span Study (LSS) tumour registry (Thompson et al., 1994) and 1950–1987 LSS leukaemia registry (Preston et al., 1994) data pertaining to cancer risk among atomic bomb survivors, and it is expected that it will be revised as new data from the A-bomb survivors and other exposed populations become available and are reviewed by expert committees such as UNSCEAR and the US National Academy of Sciences (NAS) BEIR committees. The focus of this discussion, therefore, is on the approach followed, rather than on the estimates themselves.

Briefly, models for risk estimation were obtained by statistical curve-fitting procedures applied to pertinent data sets. If we were concerned only with compensation claims by (for example) members of the A-bomb survivor population based on their A-bomb exposures, assigned share, and its uncertainty, could be computed from the fitted models with no modification except (possibly) for extrapolation to low doses. The A-bomb survivor experience is certainly relevant to the radiation-related risks of other exposed populations, but applying the information gained from studies of the A-bomb survivors (or any other exposed population) to a different exposed population is not straightforward.

For example, the A-bomb exposures were mainly to high-energy photons, with a small admixture of fast neutrons, notably in Hiroshima. Qualitatively different radiations, such as medical X-ray, may result in higher (or lower) levels of dose-specific risk and adjustment may therefore be required. A-bomb dose reconstruction is an uncertain and possibly biased process, and account must be taken of this in applying dose-specific risk estimates to other populations. Chronic exposures may result in different levels of risk than acute exposures like those from the A-bombs. Baseline cancer risks differ among populations, possibly related to differential exposure to risk factors other than radiation, and to lifestyle factors that may modify risks from radiation and other carcinogens. Concerning the mathematical relationship among radiation-related excess relative risk, excess rate, and baseline rate:

\[
\text{ERR}(a) = \frac{\text{EAR}(a)}{B(a)}
\]

it is clear that if the baseline rate \(B(a)\) differs between two populations, then either \(\text{ERR}(a)\) or \(\text{EAR}(a)\), or both, must also differ between populations, and this problem must be addressed.

Finally, there is a limited, but growing, amount of information about interactions between radiation and lifestyle factors (smoking, reproductive history) as cancer risk factors, and such information is relevant to adjudication of compensation claims for possibly radiation-related cancer.

It is worth emphasizing here that decisions must be made about each of these adjustments. The practical effect of ignoring the problem is (by default) to choose one of
a range of possible choices, without examining the consequences for risk estimation and public policy. It is also important to consider that estimates of radiation-related risk, and adjustments needed to apply such estimates to different populations, are uncertain, and that uncertainty is highly relevant to adjudication of compensation claims for possibly radiation-related disease. In the case of compensation rules currently in effect in the United States under EEOICPA, the connection is obvious because the mandated procedure is to base decisions on upper probability bounds for assigned share. Even the mean or median of the uncertainty distribution of an estimate, however, is affected by the uncertainties of different factors that make up the estimate.

D.1 Modelling of statistical risk estimates

Site-specific excess relative risk (ERR) for solid cancers was modelled as a function of radiation dose, exposure age, attained age or time since exposure, gender and population as represented by age-standardized (ASR World) cancer rates. As recommended by an expert review committee (NRC, 2000), site-specific estimates based on A-bomb survivor data were limited to sites (or groups of sites) for which the data contained at least 50 cases among LSS members exposed to 10 mSv or more. In the models described in this section, thyroid cancer and non-melanoma skin cancers are excluded, and the term “all solid cancers” is used throughout to indicate solid cancers (ICD-9 codes 140-199 (DHHS, 1991)) without these two cancers. Site-specific baseline risks were modelled by stratifying on gender, city of exposure (Hiroshima or Nagasaki), calendar time, and attained age or calendar year, using the general approach described by Pierce et al. (1996). The following linear dose-response function was used to model the excess relative risk:

\[
\text{ERR}(D, s, e, a) = \alpha D \exp[\beta I_s(\text{sex}) + \gamma f(e) + \delta g(a)]
\]

where \(D\) is dose in Sv, \(I_s(\text{sex})\) is an indicator function for the opposite sex (i.e. \(I_s(\text{sex}) = 1\) for females and 0 for males if \(s\) corresponds to “male”, and conversely if \(s\) corresponds to “female”), \(e\) is age at exposure in years, \(a\) is attained age in years, \(f\) and \(g\) are specified functions of \(e\) and \(a\), respectively, and \(\alpha, \beta, \gamma\) and \(\delta\) are unknown parameters. The term \(\beta I_s(\text{sex})\) in expression (1) is a computational convenience that allows the ratio between gender-specific estimates to be determined using site-non-specific data, as discussed later. Functions \(f(e)\) and \(g(a)\) were specified as:

\[
\begin{align*}
\text{f}(e) &= -15 \text{ for } e \leq 15, \text{f}(e) = e - 30 \text{ for } e \text{ between } 15 \text{ and } 30, \text{ and } f(e) = 0 \text{ for } e > 30; \\
\text{g}(a) &= \log(a/50) \text{ for } 0 < a < 50, \text{ and } = 0 \text{ for } a \leq 50.
\end{align*}
\]

This general form was chosen because the estimates were not intended to apply to childhood exposure and because evidence for a decline in excess relative risk with exposure age over 30 and attained age over 50 is slight; in the event, the model gave a better overall fit to the combined solid cancer data than the usual model, \(f(e) = e - 30\) and \(g(a) = \log(a/50)\) (e.g. see Thompson et al., 1994).

The approach used to model parameters for site-specific solid cancers is based on the “joint analysis” approach of Pierce and Preston (1993). As applied here, the approach involves an analysis with three replicates of the data, with a “case” defined as the cancer of interest in the first set, as all other non-gender-specific cancers combined in the second replicate, and as all other gender-specific cancers combined in the third. The first replicate provides information about parameters \(\alpha, \beta, \gamma\) and \(\delta\), the second about parameters \(\beta, \gamma\) and \(\delta\), and the third about \(\gamma\) and \(\delta\). Letting parameters \(\beta, \gamma\) and \(\delta\) differ between the first replicate and the other two provides a test of homogeneity, and the site-
specific parameter estimates are used if they are statistically significantly different from the common parameter values. For most sites, there was no significant difference and the common values were used.

The means, variances and covariances of the uncertainty distributions for the parameter estimates are shown in table D.1. Statistical likelihood profile distributions for $\alpha$ are given in table D.2 for most sites for which approach 2 was used. Figure D.1 shows an example of an uncertainty distribution for stomach cancer.

Table D.1 Computation of the uncertainty distribution for ERR at 1 Sv. Approach 1 as applied to specific solid cancer sites.

<table>
<thead>
<tr>
<th>Cancer site</th>
<th>$\log(\alpha)$</th>
<th>$\gamma$</th>
<th>$\delta$</th>
<th>$\text{Var}(\log(\alpha))$</th>
<th>$\text{Cov}(\log(\alpha), \gamma)$</th>
<th>$\text{Var}(\gamma)$</th>
<th>$\text{Cov}(\gamma, \delta)$</th>
<th>$\text{Var}(\delta)$</th>
</tr>
</thead>
<tbody>
<tr>
<td>All digestive</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>–1.590</td>
<td>–0.0477</td>
<td>–1.622</td>
<td>0.10621</td>
<td>0.001868 (0.314)</td>
<td>–0.020011 (–0.082)</td>
<td>0.0003332</td>
<td>0.56236</td>
</tr>
<tr>
<td>All digestive</td>
<td>–0.8614</td>
<td>–0.0477</td>
<td>–1.622</td>
<td>0.05018</td>
<td>0.001403 (0.343)</td>
<td>–0.001882 (–0.011)</td>
<td>0.0003332</td>
<td>0.56236</td>
</tr>
<tr>
<td>Females</td>
<td>–0.7998</td>
<td>–0.04723</td>
<td>–1.781</td>
<td>0.07512</td>
<td>0.001380 (0.279)</td>
<td>0.006263 (0.031)</td>
<td>0.0003252</td>
<td>0.54764</td>
</tr>
<tr>
<td>Stomach Females</td>
<td>–1.049</td>
<td>–0.05204</td>
<td>–1.579</td>
<td>0.17108</td>
<td>0.002291 (0.307)</td>
<td>–0.03610 (–0.115)</td>
<td>0.0003255</td>
<td>0.57368</td>
</tr>
<tr>
<td>Liver, both genders</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>–1.049</td>
<td>–0.05204</td>
<td>–1.579</td>
<td>0.17108</td>
<td>0.002291 (0.307)</td>
<td>–0.03610 (–0.115)</td>
<td>0.0003255</td>
<td>0.57368</td>
</tr>
<tr>
<td>Breasts</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Females</td>
<td>0.02109</td>
<td>–0.03722</td>
<td>–2.006</td>
<td>0.05456</td>
<td>0.002586 (0.589)</td>
<td>–0.01907 (–0.107)</td>
<td>0.0003530</td>
<td>0.58018</td>
</tr>
</tbody>
</table>

Note:(ERR/Sv is assumed to be lognormally distributed with geometric mean (GM) and geometric standard deviation (GSD). GM = $\alpha \times \exp\{\gamma f(e) + \delta g(a)\}$

\[
\text{GSD} = \exp\{\text{Var}(\log(\alpha)) + 2\text{Cov}(\log(\alpha), \gamma) f(e) + \text{Var}(\gamma) f(e)^2 + 2\text{Cov}(\gamma, \delta) g(a) + \text{Var}(\delta) g(a)^2\}\]

where $f(e) = \min(\max(–15, e – 30), 0)$ and $g(a) = \min(\ln(a/50), 0)$ for exposure age $e$ and attained age $a$.

Figure D.1 Example: Gastric cancer risk at age 60 for a woman exposed to gamma radiation at age 32

Note: According to the coefficients presented in table D.1, the statistical estimate of ERR(60) per Sv is an uncertain number, lognormally distributed with geometric mean (GM) 0.45 and geometric standard deviation (GSD) 1.32

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APPENDIX D
Table D.2  Computation of the uncertainty distribution for ERR at 1 Sv. Likelihood profile distributions for $\alpha$, obtained by approach 2 treatment of specific cancer for exposure age $e \geq 30$ and attained age $a \geq 50$: sites for which a lognormal approximation was not appropriate, and for which default values of $\gamma$ and $\delta$ were used.

<table>
<thead>
<tr>
<th>Profile quantiles</th>
<th>Oral cavity and pharynx</th>
<th>Oesophagus</th>
<th>Stomach</th>
<th>Colon</th>
<th>Rectum</th>
<th>Gall bladder</th>
<th>Pancreas</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Males</td>
<td>Females</td>
<td>Males</td>
<td>Females</td>
<td>Males</td>
<td>Females</td>
<td>Males</td>
</tr>
<tr>
<td>0.9975</td>
<td>0.8004</td>
<td>1.765</td>
<td>1.216</td>
<td>2.523</td>
<td>0.3802</td>
<td>1.531</td>
<td>1.671</td>
</tr>
<tr>
<td>0.995</td>
<td>0.7321</td>
<td>1.619</td>
<td>1.117</td>
<td>2.519</td>
<td>0.3516</td>
<td>1.429</td>
<td>1.567</td>
</tr>
<tr>
<td>0.9875</td>
<td>0.6404</td>
<td>1.423</td>
<td>0.9820</td>
<td>2.492</td>
<td>0.3137</td>
<td>1.289</td>
<td>1.423</td>
</tr>
<tr>
<td>0.975</td>
<td>0.5694</td>
<td>1.271</td>
<td>0.8755</td>
<td>2.179</td>
<td>0.2846</td>
<td>1.177</td>
<td>1.308</td>
</tr>
<tr>
<td>0.95</td>
<td>0.4962</td>
<td>1.113</td>
<td>0.7634</td>
<td>1.869</td>
<td>0.2545</td>
<td>1.058</td>
<td>1.185</td>
</tr>
<tr>
<td>0.875</td>
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<td>0.6025</td>
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<td>0.2112</td>
<td>0.8852</td>
<td>1.005</td>
</tr>
<tr>
<td>0.8413</td>
<td>0.3651</td>
<td>0.8288</td>
<td>0.5563</td>
<td>1.324</td>
<td>0.1967</td>
<td>0.8357</td>
<td>0.9537</td>
</tr>
<tr>
<td>0.5</td>
<td>0.2055</td>
<td>0.4755</td>
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<td>0.5405</td>
<td>0.6430</td>
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<tr>
<td>0.1587</td>
<td>0.0907</td>
<td>0.2136</td>
<td>0.0784</td>
<td>0.1779</td>
<td>0.0497</td>
<td>0.3020</td>
<td>0.3857</td>
</tr>
<tr>
<td>0.125</td>
<td>0.0739</td>
<td>0.1736</td>
<td>0.0545</td>
<td>0.1229</td>
<td>0.0369</td>
<td>0.2672</td>
<td>0.3523</td>
</tr>
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<td>0.0724</td>
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<td>0.0190</td>
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<td>&lt;0</td>
<td>&lt;0</td>
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</tr>
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<td>&lt;0</td>
<td>&lt;0</td>
<td>0.0049</td>
<td>&lt;0</td>
</tr>
<tr>
<td>Profile quantiles</td>
<td>Respiratory, non-lung</td>
<td>Urinary tract</td>
<td>Bladder</td>
<td>Ovary</td>
<td>Male genital</td>
<td>Central nervous system</td>
<td>Residual solid cancers</td>
</tr>
<tr>
<td>-------------------</td>
<td>-----------------------</td>
<td>---------------</td>
<td>---------</td>
<td>-------</td>
<td>--------------</td>
<td>-----------------------</td>
<td>------------------------</td>
</tr>
<tr>
<td>0.9975</td>
<td>0.7400 0.7009</td>
<td>1.716</td>
<td>1.480</td>
<td>3.561</td>
<td>1.561</td>
<td>3.887</td>
<td>2.02</td>
</tr>
<tr>
<td>0.995</td>
<td>0.5725 0.4810</td>
<td>1.619</td>
<td>1.396</td>
<td>3.354</td>
<td>1.474</td>
<td>3.577</td>
<td>1.86</td>
</tr>
<tr>
<td>0.9875</td>
<td>0.3930 0.2755</td>
<td>1.319</td>
<td>1.281</td>
<td>3.071</td>
<td>1.312</td>
<td>3.172</td>
<td>1.65</td>
</tr>
<tr>
<td>0.975</td>
<td>0.2344</td>
<td>1.105</td>
<td>1.189</td>
<td>2.848</td>
<td>1.188</td>
<td>2.864</td>
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</tr>
<tr>
<td>0.95</td>
<td>0.0606</td>
<td>0.9008</td>
<td>1.092</td>
<td>2.613</td>
<td>1.062</td>
<td>2.551</td>
<td>1.30</td>
</tr>
<tr>
<td>0.875</td>
<td>&lt; 0</td>
<td>0.6291</td>
<td>0.9489</td>
<td>2.273</td>
<td>0.8843</td>
<td>2.115</td>
<td>1.05</td>
</tr>
<tr>
<td>0.8413</td>
<td>&lt; 0</td>
<td>0.5366</td>
<td>0.9080</td>
<td>2.176</td>
<td>0.8311</td>
<td>0.987</td>
<td>0.982</td>
</tr>
<tr>
<td>0.5</td>
<td>&lt; 0</td>
<td>0.1377</td>
<td>0.6635</td>
<td>1.601</td>
<td>0.5388</td>
<td>1.282</td>
<td>0.576</td>
</tr>
<tr>
<td>0.1587</td>
<td>&lt; 0</td>
<td>&lt; 0</td>
<td>0.4650</td>
<td>1.137</td>
<td>0.3091</td>
<td>0.7337</td>
<td>0.267</td>
</tr>
<tr>
<td>0.125</td>
<td>&lt; 0</td>
<td>&lt; 0</td>
<td>0.4380</td>
<td>1.073</td>
<td>0.2778</td>
<td>0.6587</td>
<td>0.230</td>
</tr>
<tr>
<td>0.05</td>
<td>&lt; 0</td>
<td>&lt; 0</td>
<td>0.3571</td>
<td>0.8820</td>
<td>0.1869</td>
<td>0.4414</td>
<td>0.117</td>
</tr>
<tr>
<td>0.025</td>
<td>&lt; 0</td>
<td>&lt; 0</td>
<td>0.3102</td>
<td>0.7698</td>
<td>0.1352</td>
<td>0.3176</td>
<td>0.0569</td>
</tr>
<tr>
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<td>&lt; 0</td>
<td>0.2712</td>
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<td>0.0925</td>
<td>0.2159</td>
<td>&lt; 0</td>
</tr>
<tr>
<td>0.005</td>
<td>&lt; 0</td>
<td>&lt; 0</td>
<td>0.2285</td>
<td>0.5716</td>
<td>0.0457</td>
<td>0.1057</td>
<td>&lt; 0</td>
</tr>
<tr>
<td>0.0025</td>
<td>&lt; 0</td>
<td>&lt; 0</td>
<td>0.2011</td>
<td>0.5038</td>
<td>0.0173</td>
<td>0.0393</td>
<td>&lt; 0</td>
</tr>
</tbody>
</table>

Notes: Likelihood profile distributions for $\alpha$, for exposure age $e \geq 30$ and attained age $a \geq 50$: sites for which a lognormal approximation was not appropriate, and for which default values of $\gamma$ and $\delta$ were used. For exposure age $e < 30$ and/or attained age $a < 50$, ERR at 1 Sv = $\alpha \times h(e, a, \gamma, \delta)$, where $h(e, a, \gamma, \delta)$ is assumed to be statistically independent of and lognormally distributed with geometric mean (GM) and geometric standard deviation (GSD) as follows:

$$GM = \exp(-0.05255f(e) - 1.626g(a))$$

$$GSD = \exp\left[0.0003261 \times h(e, a, \gamma, \delta) + 0.9648 \times g(a)\right]^{\frac{1}{2}}$$

where $f(e)$ and $g(a)$ are defined in the accompanying table D.1.
For leukaemia, site-specific baseline incidence was modelled as a function of gender, city of exposure (Hiroshima or Nagasaki), year of birth, calendar time (where indicated), and age at observation for risk (attained age), as discussed in Preston et al. (1994). Default dose-response models were linear (proportional to dose equivalent \( D \) in Sv, henceforth called “dose” for brevity) for leukaemia associated with exposure to high-LET radiation or low-LET radiation delivered at low dose rates (chronic exposure), and linear-quadratic for leukaemia associated with acute exposure to low-LET radiation. The linear-quadratic model was set to have equal contributions of the dose and dose-squared terms at 1 Sv (proportional to \( D + D^2 \)). Fitting a general linear-quadratic model (proportional to \( D + \xi D^2 \)) for all types of leukaemia except chronic lymphocytic (CLL), considered as a group, and for acute myelogenous, acute lymphocytic, and chronic myelocytic leukaemia separately, various estimates of the unknown parameter were obtained, depending on the type of leukaemia, that were greater than zero. However, because all these estimates were statistically consistent with the default value \( \xi = 1 \), the final models for leukaemia and its subtypes were based on \( \xi = 1 \).

In terms of potential modifying factors such as sex (\( s \)), age at exposure (\( e \)), attained age (\( a \)), and time since exposure (\( t \)), the fitted model was

\[
\text{ERR}(D,e,a) = \alpha(D + D^2)\exp\{\beta e + \gamma t + \delta e t\}
\]

(2)

where \( \alpha, \beta, \gamma \) and \( \delta \) are unknown parameters which may be gender-specific. Parameter \( \alpha \) was estimated from the data, as were parameters \( \beta, \gamma \) and \( \delta \) unless they made no significant contribution to improvement of the fit of the model to the data, in which case they were set to zero; similarly, individual parameters were made gender-specific only if doing so led to significant improvement in fit. Following Preston et al. (1994), the leukaemia dose response was modelled in terms of \( e \) and \( t = a - e \) instead of \( e \) and \( a \).

The statistical uncertainty distribution of the resulting estimate is described by the profile likelihood distribution of the fitted parameter \( \alpha \) in table D.3.

Table D.3 Computation of the uncertainty distribution for ERR at 1 Sv; leukaemia other than chronic lymphocytic, combined genders. Likelihood profile distributions, by representative values for age at exposure and time since exposure

<table>
<thead>
<tr>
<th>Profile quantiles</th>
<th>Exposure age 20</th>
<th>Exposure age 30</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>5 yr 10 yr 15 yr 25 yr 35 yr 45 yr</td>
<td>5 yr 10 yr 15 yr 25 yr 35 yr 45 yr</td>
</tr>
<tr>
<td>0.9975</td>
<td>72.69 29.87 13.54 3.967 1.671 0.8029</td>
<td>37.55 18.19 9.412 3.361 1.672 0.9342</td>
</tr>
<tr>
<td>0.995</td>
<td>65.99 27.68 12.71 3.744 1.538 0.7102</td>
<td>34.69 17.09 8.944 3.206 1.556 0.8387</td>
</tr>
<tr>
<td>0.9875</td>
<td>57.46 24.83 11.62 3.438 1.358 0.5913</td>
<td>30.97 15.62 8.311 2.991 1.400 0.7154</td>
</tr>
<tr>
<td>0.975</td>
<td>51.20 22.68 10.78 3.194 1.217 0.5038</td>
<td>28.16 14.49 7.816 2.818 1.277 0.6239</td>
</tr>
<tr>
<td>0.95</td>
<td>45.05 20.51 9.922 2.934 1.071 0.4180</td>
<td>25.33 13.33 7.299 2.633 1.149 0.5334</td>
</tr>
<tr>
<td>0.875</td>
<td>36.94 17.57 8.719 2.554 0.8658 0.3065</td>
<td>21.47 11.70 6.559 2.358 0.9676 0.4137</td>
</tr>
<tr>
<td>0.8413</td>
<td>34.80 16.76 8.385 2.445 0.8091 0.2778</td>
<td>20.42 11.25 6.350 2.278 0.9168 0.3820</td>
</tr>
<tr>
<td>0.5</td>
<td>23.55 12.35 6.481 1.784 0.4911 0.1352</td>
<td>14.65 8.662 5.121 1.789 0.6253 0.2185</td>
</tr>
<tr>
<td>0.1587</td>
<td>16.10 9.173 5.015 1.239 0.2730 0.0585</td>
<td>10.52 6.674 4.124 1.366 0.4606 0.1173</td>
</tr>
<tr>
<td>0.125</td>
<td>15.21 8.776 4.824 1.168 0.2480 0.0511</td>
<td>10.01 6.416 3.991 1.308 0.3786 0.1062</td>
</tr>
<tr>
<td>0.05</td>
<td>12.65 7.592 4.244 0.9509 0.1783 0.0320</td>
<td>8.481 5.633 3.580 1.127 0.2979 0.0755</td>
</tr>
<tr>
<td>0.025</td>
<td>11.25 6.925 3.907 0.8277 0.1428 0.0234</td>
<td>7.627 5.180 3.338 1.019 0.2535 0.0601</td>
</tr>
<tr>
<td>0.0125</td>
<td>10.14 6.380 3.627 0.7271 0.1161 0.0175</td>
<td>6.933 4.804 3.134 0.9281 0.2181 0.0486</td>
</tr>
<tr>
<td>0.005</td>
<td>8.959 5.788 3.315 0.6185 0.0898 0.0123</td>
<td>6.184 4.389 2.905 0.8259 0.1809 0.0374</td>
</tr>
<tr>
<td>0.0025</td>
<td>8.227 5.412 3.113 0.5503 0.0745 0.0095</td>
<td>5.709 4.120 2.754 0.7591 0.1581 0.0310</td>
</tr>
</tbody>
</table>
**APPENDIX D**

Thyroid cancer risk was estimated from a combined analysis of six different data sets. Thyroid is the only cancer site in Land et al. (2003) for which most of the dose-response data were from populations exposed to medical X-ray. In the analysis, it was assumed that medical X-ray dose and gamma-ray dose from the atomic bombs were equivalent in effectiveness, as in the original analysis of Ron et al. (1995). Elsewhere in the report, arguments are presented in support of an RBE (relative biological effectiveness) of around 2 for 30–250 keV (e.g. medical X-ray) compared to higher-energy photons (e.g. gamma ray from atomic bomb explosions). However, because the atomic bomb exposures considered by Ron et al. (1995) were acute and the medical X-ray exposures were fractionated, we considered that no correction was required because, at moderate to high doses, the fractionation and the RBE factor appropriate to medical X-ray should have had opposite and approximately equal effects on risk.

The statistical uncertainty distribution is shown in table D.4.

<table>
<thead>
<tr>
<th>Age at exposure</th>
<th>Geometric mean</th>
<th>Geometric standard deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>9.463</td>
<td>2.183</td>
</tr>
<tr>
<td>5</td>
<td>6.262</td>
<td>1.924</td>
</tr>
<tr>
<td>10</td>
<td>4.136</td>
<td>1.976</td>
</tr>
<tr>
<td>15</td>
<td>2.732</td>
<td>2.160</td>
</tr>
<tr>
<td>20</td>
<td>1.804</td>
<td>2.301</td>
</tr>
<tr>
<td>25</td>
<td>1.192</td>
<td>2.367</td>
</tr>
<tr>
<td>30</td>
<td>0.788</td>
<td>2.365</td>
</tr>
<tr>
<td>35</td>
<td>0.521</td>
<td>2.379</td>
</tr>
<tr>
<td>40</td>
<td>0.345</td>
<td>2.732</td>
</tr>
<tr>
<td>45</td>
<td>0.228</td>
<td>3.140</td>
</tr>
<tr>
<td>50</td>
<td>0.151</td>
<td>3.611</td>
</tr>
</tbody>
</table>

**Skin cancer.** The Working Group was initially reluctant to include skin cancers in the present report, because of a high level of uncertainty about how to transfer estimates of ERR/Sv between the Japanese A-bomb survivors and populations in the United States. Non-melanoma skin cancer is not a reportable disease in the United States (although it is in Japan), and baseline rates are not readily available, e.g. from NCI’s SEER programme (Ries et al., 1997). However, the NRC (2000) report pointed out that estimated rates were available for white and African-American US residents (Scotto et al., 1983), and recommended that the Working Group seriously consider including skin among the cancer sites covered by the present report. Also, both the Department of Veterans Affairs and NIOSH expressed interest in having skin cancer estimates.

Our data source was the data set of Thompson et al. (1994), located at the RERF in Hiroshima. Dale Preston, RERF Chief of Statistics, kindly offered to run analyses for the Working Group. We initially asked for analyses similar to those for other solid tumours, i.e. using the general model used in Ron et al. (1998), and the model specified in equation (1) of the present report.

For basal cell skin carcinoma, the only subtype for which a significant dose response was obtained by Ron et al. (1998), there was a steep decline in ERR/Sv by exposure age, which extended beyond age 30 and was otherwise different from the common trend assumed for other sites, and there was no dependence on attained age. We therefore replaced the age function \( f(e) \) as specified above by:

\[
 f(e) = \begin{cases} 
 -30 & \text{for } e \leq 10, \\
 e - 40 & \text{for } 10 < e < 40, \\
 0 & \text{for } e \geq 40 
\end{cases}
\]
Thus, there was no dependence upon attained age, and constant values of $\text{ERR/Sv}$, at different levels, for exposure ages less than 10 and for ages 40 or older, with a linear transition in the logarithmic scale between $e = 10$ and $e = 40$. Likelihood profile distributions for $\text{ERR/Sv}$ were computed for $e = 10$, 20, 30 and 40, and interpolated for $e$ between 10 and 40 (see Land et al. (2003) and table D.5).

For non-melanoma skin cancers other than basal cell carcinoma, which is dominated by squamous cell carcinoma, the unmodified point estimate of $\text{ERR/Sv}$ was negative and no convergent estimate could be obtained if an age-dependent modifying term was introduced with either a free or fixed parameter value. We therefore computed a single profile for $\text{ERR/Sv}$, with no modification by age.

The data set in Ron et al. (1998) had only ten cases of malignant melanoma, far below our inclusion criterion of 50 cases at doses greater than 10 mSv, and we therefore did not include that cancer type.

Table D.5 Computation of the uncertainty distribution for $\text{ERR}$ at 1 Sv. Likelihood profile distributions for non-melanoma skin cancer, both genders combined, and for basal cell carcinoma: exposure ages 0–10, 20, 30, and 40 or older.

<table>
<thead>
<tr>
<th>Profile quantiles</th>
<th>Basal cell skin cancer, by age at exposure</th>
<th>Other non-melanoma skin cancer</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0–10</td>
<td>20</td>
</tr>
<tr>
<td>0.9975</td>
<td>149.7</td>
<td>23.79</td>
</tr>
<tr>
<td>0.995</td>
<td>129.1</td>
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</tr>
<tr>
<td>0.9875</td>
<td>104.3</td>
<td>18.26</td>
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<td>0.975</td>
<td>87.30</td>
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<td>0.95</td>
<td>71.53</td>
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<td>47.61</td>
<td>10.27</td>
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<tr>
<td>0.5</td>
<td>25.22</td>
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</tr>
<tr>
<td>0.1587</td>
<td>13.14</td>
<td>3.970</td>
</tr>
<tr>
<td>0.125</td>
<td>11.88</td>
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<tr>
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<td>2.837</td>
</tr>
<tr>
<td>0.025</td>
<td>6.778</td>
<td>2.376</td>
</tr>
<tr>
<td>0.0125</td>
<td>5.524</td>
<td>1.998</td>
</tr>
<tr>
<td>0.005</td>
<td>4.295</td>
<td>1.576</td>
</tr>
<tr>
<td>0.0025</td>
<td>3.584</td>
<td>1.301</td>
</tr>
</tbody>
</table>

Risk estimates for lung cancer among A-bomb survivors were separately calculated by Don Pierce, applying model (1) above to data from his site-specific study of the joint effects of radiation and smoking history (Pierce et al., 2003). This analysis is discussed later in this report.

Radon-related lung cancer. A report prepared for the Department of Justice (1996) contains tables of cumulative radon exposures, in working level months (WLM), consistent with point estimates and upper 80 per cent and 90 per cent confidence limits for probability of causation greater than or equal to 50 per cent, and the original data set used for these calculations, but restricted to exposures $\leq 3200$ WLM, was made available to the Working Group. The Working Group attempted to approximate Appendix Table 3a of the report, modelling $\text{ERR}$ as follows:

$$
\text{ERR}(\text{wlm},e,t) = \alpha \text{wlm}^\beta \exp\{\gamma f(a) + \delta g(t)\}
$$

(3)
where $wlm$ is cumulative radon exposure in working level months, $a$ is age at diagnosis, $t$ is time since last exposure, $\alpha$, $\beta$, $\gamma$ and $\delta$ are unknown parameters, and

$$
\begin{align*}
f(a) &= 0 \text{ for } a \leq 45, f(a) = a - 45 \text{ for } 45 < a \leq 75, \text{ and } f(a) = 30 \text{ for } a > 75 \\
g(t) &= 0 \text{ for } t \leq 5, g(t) = t - 5 \text{ for } 5 < t \leq 30, \text{ and } g(t) = 20 \text{ for } t > 25
\end{align*}
$$

Thus, ERR was assumed to be proportional to an uncertain power of cumulative exposure in WLM, and to be constant in $a$ (at different levels) for $a \leq 45$ and $a > 75$, and to be constant in $t$ (again, at different levels) for $t \leq 5$ and $t > 25$. Likelihood functions for $ERR_{1\ wlm}$ are given in table IV.D.10 of Land et al. (2003), for smokers and non-smokers, for $a \leq 45$, $a = 63$, and $a > 75$, and for $t \leq 5$, $t = 15$, and $t > 25$, for interpolation in $a$ and $t$. For ERR at arbitrary $wlm$, $ERR_{1\ wlm}$ is multiplied by $wlm^{0.82}$.

## D.2 Correction for random and systematic errors in A-bomb survivor dosimetry

Our treatment of random and systematic errors in A-bomb survivor dosimetry was based mainly on the treatment described in Chapter 3 of a report by the NCRP (1997), and the reader is referred to this material for details. Briefly, a correction factor was obtained incorporating uncertainty in the magnitude of random errors in the doses of individual survivors, in the appropriate choice of neutron RBE in analysing A-bomb survivor data, due to systematic bias in gamma dose estimates, and due to systematic bias in neutron dose estimates in Hiroshima, yielding an overall multiplicative correction factor distributed as a normal random variable with mean 0.83 and standard error 0.084.

Figure D.2 shows the example in figure D.1 adjusted for the dose reconstruction error.

**Figure D.2 Example (female gastric cancer, continued): Effect of adjustment for dose reconstruction error**

Note: The corrected estimate of $ERR(80)$ per Sv is approximately lognormal, with GM 0.375 and GSD 1.341.
D.3 Dependence of risk on dose and dose rate for low-LET radiation

For leukaemia, the shape of the linear-quadratic dose-response function determines a twofold reduction in excess risk per unit dose between $D = 1$ Sv and $D$ near zero. Because there is no uncertainty in the curvature of the fitted dose response, there is no uncertainty in the DDREF for leukaemia. (This decision will be re-evaluated when the estimates are revised.) For other cancers for which a linear dose response is assumed, the Working Group assumed an uncertain DDREF with probabilities 0.01, 0.04, 0.2, 0.3, 0.3, 0.1, 0.04 and 0.01 assigned to DDREF values 0.5, 0.7, 1, 1.5, 2, 3, 4 and 5. Division by this DDREF in effect divides the median risk in half, but adds considerable uncertainty to the estimate. For “chronic” exposure, the DDREF always applies; for “acute” exposure, the DDREF ($\text{DDREF}_{\text{acute}}$) is modelled as a random quantity that approaches $\text{DDREF}_{\text{chronic}}$ as dose decreases to zero. Between zero and an uncertain reference dose, $D_L$ (between 0.03 and 0.2 Gy, distributed as log-uniform over that interval), $\text{DDREF}_{\text{acute}}$ increases smoothly from $\text{DDREF}_{\text{chronic}}$ at zero dose to 1 at $D_L$ and above, according to a logistic function of dose (figure D.3). Figure D.4 presents the stomach cancer example used in figures D.1 and D.2, additionally adjusted for the uncertain DDREF.

Figure D.3 Variation of $\text{DDREF}_{\text{acute}}$ as a function of radiation dose for selected values of $\text{DDREF}_{\text{chronic}}$ for a fixed value of $D_L$, the lowest dose at which linearity of dose response is assumed to apply.
D.4 Adjustment for radiation quality

People can be exposed to many different types of ionizing radiation including photons, electrons, alpha particles and neutrons, and the energies of each radiation type can vary widely. Many studies of the effects of ionizing radiation on a wide variety of biological systems, ranging from simple cells to complex whole organisms, have shown that different types of radiation often differ substantially in their biological effectiveness. That is, the probability that a particular biological response is induced by radiation depends on the radiation type, and sometimes its energy, as well as the dose. In estimating cancer risks and probability of causation (assigned share) for an individual who received known exposures to particular radiation types, it is therefore essential that differences in the biological effectiveness of the different radiations be taken into account.

For the purpose of estimating cancer risks and assigned shares in identifiable individuals who received known (estimated) radiation exposures, the term “radiation effectiveness factor”, denoted by REF, has been developed to describe the biological effectiveness of different radiation types (Kocher et al., 2005). There are two reasons why a new term, other than “RBE” or “radiation weighting factor”, is used. First, “RBE” is not appropriate because this quantity strictly applies only to results obtained from specific radiobiological studies and so should not be used to describe an extrapolation of such results to a different biological endpoint, biological system, or condition of exposure. Second, as discussed above, the radiation weighting factor is a prescribed point quantity, without uncertainty, which is used in radiation protection to calculate equivalent doses, but it is not intended to be used to estimate cancer risks and assigned shares in identifiable individuals who received known exposures. Furthermore, cancer risks and assigned shares are estimated based on estimates of dose without the need to estimate equivalent doses, and it is essential that uncertainties in the biological effectiveness of different radiation types relative to a defined reference radiation be taken into account. Figure D.5 shows the applications of these considerations using the example of female gastric cancer. The figure presents the stomach cancer example used in figures D.1, D.2 and D.4, with additional adjustment for medical X-ray dose.

The probability distributions of the radiation effectiveness factors used in the NCI/CDC report were developed by Kocher et al. (2005) under a contract with the National Institute of Occupational Safety and Health (NIOSH), and have taken into account peer
reviews of the work by NIOSH consultants. The assumed probability distributions of the radiation effectiveness factors for photons and electrons are summarized in table D.6, the distributions for alpha particles are summarized in table IV.H.2, and the distributions for neutrons are summarized in table IV.H.3 of Land et al. (2003). For photons and electrons, the probability distributions of the radiation effectiveness factors are applied to all cancers, whereas separate probability distributions are developed for leukaemias (including lymphomas and lymphocytic cancers) in cases of exposure to alpha particles and neutrons. For present purposes, it is assumed that any exposure to proton radiation will be at high proton energies, with RBE = 1 relative to high-energy photons. The probability distributions of the correction for an inverse dose-rate effect are included in the tables for alpha particles and neutrons.

Table D.6 Photons and electrons: Summary of probability distributions of radiation effectiveness factors to be used in estimating cancer risks and assigned shares in accordance with eq. (IV.H.1), (IV.H.3) or (IV.H.4)*

<table>
<thead>
<tr>
<th>Radiation type</th>
<th>Exposure</th>
<th>Probability distribution of radiation effectiveness factor (REF)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Photons</td>
<td>Chronic or acute</td>
<td></td>
</tr>
<tr>
<td>$E &gt; 250$ keV</td>
<td>Single-valued at 1.0 (higher-energy photons are assumed reference radiation)</td>
<td></td>
</tr>
</tbody>
</table>
| $E = 30–250$ keV | Hybrid distribution with:  
|                 | • 25% probability assigned to value 1.0  
|                 | • 75% probability assigned to lognormal distribution with 95% probability between 1.0 and 5.0 |
| $E < 30$ keV   | Product of two distributions:  
|                 | (1) hybrid distribution for $E = 30–250$ keV; and  
|                 | (2) triangular distribution with minimum of 1.0, mode of 1.3, and maximum of 1.6 |
| Electrons      | Chronic or acute |                                                                 |
| $E > 15$ keV   | Single-valued at 1.0 (assumed to be same as value for reference higher-energy photons) |
| $E < 15$ keV   | Lognormal distribution with 95% confidence interval between 1.2 and 5.0 |

* The equations are given in Section IV.H of Land et al. (2003). Equation (IV.H.1) applies to solid tumours, eq. (IV.H.3) applies to leukaemias under conditions of chronic exposure, and eq. (IV.H.4) applies to leukaemias under conditions of acute exposure.

* When eq. (IV.H.1) is used, DDREF is always applied under conditions of chronic exposure. At acute doses greater than 2 mGy, DDREF is assumed to be 1.0. At acute doses less than 2 mGy, a DDREF that can exceed 1.0 is applied, and the distribution of possible values approaches the probability distribution of DDREF that applies to all chronic exposures as the dose approaches zero.

* Probability distribution is based on data on RBE for low-energy beta particles emitted in decay of tritium ($^3$H); distribution is applied to other electrons of energy less than 15 keV, except low-energy Auger electrons emitted by radionuclides that are incorporated into DNA are excluded.

Figure D.5 Example (female gastric cancer, continued): The radiation effectiveness factor for 30–250 keV photons is distributed according to a hybrid distribution that assigns 25% probability to one and 75% probability to a lognormally distributed random variable with GM 5% and GSD 1.51

Note: The corrected estimate of ERR(60) for a chronic X-ray dose of 0.12 Sv is approximately lognormal, with GM 0.0435 and GSD 2.13.
APPENDIX D

D.5 Transfer of ERR from the Japanese to the US population

A major concern in using data from Japanese A-bomb survivors to estimate risks for specific cancers in a US (or other) population is that baseline risks differ between the two populations and the dependence of radiation risks on baseline risks is not known with certainty. For example, baseline cancer rates for breast, lung and colon cancer are lower in Japan than in the United States, while rates for stomach and liver cancer are much higher in Japan. Estimation of risk for a US population based on the dose response coefficients derived from A-bomb survivor data is commonly referred to as the “transfer” or “transportation” problem. A more detailed discussion of the transfer problem appears in NCRP (1997).

Two simple solutions are the so-called “multiplicative” and “additive” transfer models, in which estimates of excess relative risk (the ratio between excess and baseline risk) and absolute risk (the difference between the estimated cancer rates with and without exposure), are respectively applied to the second population (in this case, the US population). The multiplicative transfer model is biologically plausible to the extent that ionizing radiation exposure can be assumed to act as an “initiator” of a process whose likelihood of resulting in cancer depends upon the action of “promoting” agents, if these “promoting” agents are responsible for the difference in baseline rates between the two populations, or, alternatively, if radiation were to act as a promoter of the carcinogenic effects of other agents that are differentially effective in the two populations. In this view, the excess risk from radiation exposure would be greater in a normally high-risk population than in a normally low-risk population. The additive transfer model is plausible to the extent that radiation can be assumed to act mainly as an initiator and the difference between population baseline rates can be assumed to be due to the differential effects in the two populations of other “initiator” carcinogens that act similarly to radiation. In this view, the additional cancer risk burden of radiation exposure would be independent of the population baseline rate.

Several approaches have been used to transfer risk estimates based on the Japanese A-bomb survivor data to other populations. The multiplicative transfer model was used by UNSCEAR (1988) for the world population and in the BEIR V report (NRC, 1990) for the US population. The additive transfer model was used in the BEIR III report (NRC, 1980) and the NIH report (1985). The two transfer models can lead to very different estimates of radiation-related risk for certain cancers for which baseline risks differ greatly between Japan and the United States (Land, 1990). Each model receives some support from site-specific comparisons, but there are few sites for which meaningful analytic comparisons can be made. If population differences in cancer rates may be due to both initiating and promoting agents, it is likely that both additive and multiplicative model interactions with radiation may take place, and that some kind of mixture model may be appropriate. For example, the ICRP (1991) used the arithmetic mean of the ERR values obtained by the two transfer models for all solid cancer types combined (Land and Sinclair, 1991), and the Environmental Protection Agency (Puskin and Nelson, 1995) used the geometric mean (except for liver cancer associated with exposure to the radioactive contrast medium thorotrast and bone cancer from exposure to injected 224Ra, for which an additive transfer model was chosen). More recent reports have used uncertain (i.e. randomized) linear or geometric combinations, weighted in various ways, of the additive and multiplicative transfer models for the estimation of total risk of cancer mortality (EPA1999).
Mortality rates for all types of cancer combined vary relatively little by nation, compared to site-specific variation. The initial ERR1Sv value for mortality from all cancers combined used in the NCRP (1997) report was the rounded average of multiplicative and additive transfer model estimates from the LSS mortality data for five different national populations (ICRP, 1991; Land and Sinclair, 1991). Thus, the problem for that report was not how to estimate ERR_{1Sv} for a US population, but to determine the uncertainty associated with estimating ERR_{1Sv} in a particular way. Their solution was an uncertainty factor f(T), distributed as ln(1, 1.3).

For Land et al. (2003), the problem was how to estimate site-specific and age-specific values of ERR_{1Sv} for a given population in the presence of possibly large differences in baseline rates and the absence of useful information about which model might be correct. The approach chosen was to use a random linear combination between the additive and multiplicative models,

\[(ERR_{1Sv})_{US} = y \times (ERR_{1Sv})_{mult} + (1-y) \times (ERR_{1Sv})_{add}\]

where the random variable y varies between –0.1 and 1.1. Here, (ERR_{1Sv})_{mult} is the site-, gender- and age-specific excess relative risk at 1 Sv obtained from statistical analysis of the Japanese A-bomb survivor data and adjusted for random and systematic errors in dose to individual A-bomb survivors (see above). (ERR_{1Sv})_{add} is the same value, adjusted for the corresponding ratio between baseline rates in the two countries:

\[(ERR_{1Sv})_{add} = (ERR_{1Sv})_{add} \cdot \left( \frac{B_{Japan}}{B_{US}} \right)\]

Here, B_{Japan} and B_{other} are the gender- and site-specific, age-adjusted background cancer incidence rates in Japan (a surrogate for the A-bomb survivor cohort) and the target population, respectively, both age-standardized to the world population age distribution (Parkin et al., 2002).

The coefficient y of the linear combination can be used to favour one model or the other according to the weight of evidence. For instance, y = 0 corresponds to the additive model, y = 1 to the multiplicative model, and y = ½ to the arithmetic average of the two. A Monte Carlo simulation is used to express uncertainty about y, with y values sampled according to the following probability density distribution:

\[f(y) = 0.9091 \times \begin{cases} 
(y + 0.1) & -0.1 < y < 0 \\
1 & 0 \leq y \leq 1 \\
(1.1 - y) & 1.0 < y < 1.1
\end{cases}\] (3)

The constant probability density shown above for y values between 0 and 1 reflects a complete lack of knowledge about the appropriateness of particular weighted averages of the additive and multiplicative transfer models, and the assignment of a small probability weight (9 per cent) to values less than 0 and larger than 1 allows for the (subjectively unlikely) possibility that radiation-related cancer risk might be negatively correlated with population baseline risk.

For breast, thyroid and stomach cancer, more information is available and so the “uninformed” trapezoidal density given above and in Land et al. (2003) may be modified by redistributing some of the weight to the additive transfer model in the case of breast cancer (Preston et al., 2002; Little and Boice, 1999; Land et al., 1980) or the multiplicative model for thyroid cancer and stomach cancer (Carr et al., 2002; Ron et al., 1995; Griem et al., 1994). Thus, for breast cancer, a probability weight of 50 per cent was assigned to the additive transfer model (y = 0), and 50 per cent was assigned to the trapezoidal probability density distribution. For stomach cancer, a probability weight of 33 per cent was
assigned to the multiplicative model \((y = 1)\), and 66 per cent to the trapezoidal distribution in figure D.6 (Land et al., 2003). The cumulative distribution functions for these distributions are compared with that for the “uninformed” distribution in figure IV.G.2 of Land et al. (2003). For thyroid cancer, the multiplicative model was used, reflecting the international basis of Ron et al. (1995).

Figure D.6 Trapezoidal probability density function \(f(y)\) for the uncertain linear mixture coefficient \(y\) between additive \((y = 0)\) and multiplicative \((y = 1)\) models for transfer of excess relative risk from one population to another, for most types of cancer.

Figure D.7 Example (female gastric cancer, concluded): Effect of adjustment for population transfer.

Note: The ratio of age-standardized gastric cancer rates for women in Japan compared to the United States is about 12. Thus, the multiplicative transfer model gives the same ERR(60) value for both countries, while additive transfer assigns a value 12 times as high to ERR(60) for an American woman. By assigning 1/3 probability to the multiplicative transfer model and 2/3 probability to the random mixture of that value and the additive transfer model shown in the previous paragraph (equation 3), we obtain an adjusted estimate, the distribution of which is very roughly lognormal with GM 0.10 and GSD 4.0. This is an extreme but realistic example, illustrating the extent to which ignorance about site-specific radiation-related risk in different populations can lead to extreme uncertainty in estimated risk.
Figure D.7 shows the concluding example of female stomach cancer, here also adjusted for population transfer.

As discussed below, Pierce et al. (2003) found that, among A-bomb survivors with both radiation dose estimates and smoking history data, lung cancer risk was “quite consistent” with an additive model for interaction between radiation and tobacco smoking, but statistically inconsistent with a multiplicative model. Given this result, and the strong dependence of population lung cancer rates on cigarette consumption (Blot and Fraumeni, 1996), the Working Group concluded that the “informed” transfer model used for breast cancer, with 50 per cent probability assigned to the additive model, was also appropriate for lung cancer.

D.6 Modification by epidemiological risk factors

Site-specific studies of radiation dose and cancer risk, in the LSS sample and in other exposed populations continually followed up over time, generally proceed in a series of steps beginning with the evaluation of evidence that a dose-related excess risk actually exists. Usually, the first modifiers of dose response to be considered are gender, age at exposure, age at observation (attained age), and time following exposure, because information about them is usually obtained at the same time as information on radiation exposure and disease occurrence. Modification of dose response by other factors is a more difficult problem, because it usually requires special data-gathering efforts, such as with an embedded case-control study. Informative studies of interaction between radiation dose and epidemiological risk factors have been carried out for reproductive history in the case of breast cancer and for smoking history in the case of lung cancer.

D.6.1 General formulation

If radiation dose $D$ and factor $f$ are multiplicative in effect, then the excess relative risk associated with exposure $D$ is independent of $f$, i.e. $\text{ERR}_{D|f} = \text{ERR}_D$. If $D$ and $f$ are additive in effect, then the conditional ERR associated with $D$ given exposure $f$ is

$$\text{ERR}_{D|f} = \frac{\text{ERR}_D}{1 + \text{ERR}_f}$$

D.6.2 Breast cancer: Interaction of radiation and age at first full-term pregnancy

Reproductive history is known to be an important breast cancer risk factor. In particular, early age at first full-term pregnancy has been shown, in virtually every population that has been studied, to be protective. A case-control interview study of female A-bomb survivors examined the interaction of this risk factor with radiation dose (Land, 1994), and found that an additive interaction model was rejected, whereas a multiplicative interaction model was consistent with the data. A general risk model,

$$R_{\text{mix}}(D; X; \beta, \xi) = (1 + a_{E}D)(1 + \beta X/(1 + a_{E}D) \xi)$$

was used to distinguish between the multiplicative model (corresponding to $\xi = 0$),

$$R_{\text{mult}}(D; X; \beta) = (1 + a_{E}D)(1 + \beta X)$$

and the additive model (corresponding to $\xi = 1$),

$$R_{\text{add}}(D; X; \beta) = 1 + a_{E}D + \beta X$$
Here, $D$ is radiation dose, $X$ is age at first full-term pregnancy, $a_E$ is a parametric function describing radiation dose response as a function of age at exposure $E$, and $\beta$ is an unknown parameter corresponding to $X$. The maximum likelihood estimate of the parameter $\xi$ was negative (–0.25) (Land, 1994) and the likelihood distribution placed less than 10 per cent probability on values greater than zero in calculations performed for the present report. Thus, it appears that very little additional uncertainty would be contributed by allowing for deviations from the multiplicative interaction model, for which no adjustment of $\text{ERR}_{1\text{Sv}}$ is required for age at first full-term pregnancy. Land et al. (2003) therefore makes no uncertainty adjustment for this factor.

D.6.3 Lung cancer: Interaction of radiation dose with smoking history

Interaction analyses of A-bomb survivors (Blot et al., 1984) and uranium miners (NRC, 1988) failed to discriminate between additive and multiplicative interaction models, although the BEIR IV committee concluded that the data were more consistent with a multiplicative interaction (NRC, 1988).

More recently, Lubin and Steindorf (1995) modelled joint relative risks for smoking history (ever vs. never) and exposure to inhaled radon decay products among six cohorts of US uranium miners for which such information was available. They concluded that, at that level of smoking history detail, the best-fitting interaction model was intermediate between the additive and multiplicative interaction models. The BEIR VI committee (NRC, 1999) applied the approach of Lubin and Steindorf (1995) using more recent data, and concluded that both the multiplicative and (especially) the additive interaction models were statistically inconsistent with the data. Treatment of smoking status for radon-related lung cancer risk is discussed above.

A new analysis of lung cancer and smoking history among A-bomb survivors by Pierce and Preston (1993) was based on 45,113 survivors followed through 1994, including 592 lung cancer cases, for whom smoking history information was available from questionnaire responses and clinical interviews. The main finding was that radiation and smoking effects on lung cancer risk were statistically inconsistent with a multiplicative interaction model, and “quite consistent” with an additive model. At the Working Group’s request, Dr Pierce kindly carried out dose-response analyses on his data set according to model (1), which showed that the values $\beta = 0.843$, $\gamma = –0.5255$, and $\delta = –1.626$ used in approach (2) in Section IV.D.1 of Land et al. (2003) were statistically consistent with the lung cancer data. He also estimated the likelihood profile distribution for the parameter $\alpha$ assuming the above parameter values, so that approach (2) could be applied to lung cancer (table D.7) as described in Section IV.D.1 of Land et al. (2003). However, because the analysis clearly supported the additive interaction model, the analysis was adjusted for smoking and the tabulated profile pertains to risk among lifetime non-smokers. Also, for lung cancer the tabulated profile is adjusted to be midway between the values for the two genders corresponding to $\beta = 0.843$.

In the NIH (1985) report, it was assumed that the interaction of smoking and exposure to low-LET radiation was additive, with appropriate assigned shares obtained by multiplying the ERRs by the factors indicated in columns 2 and 3 of table IV.I.1 of Land et al. (2003). These factors were calculated as described on pp. 48–51 of NIH (1985) and based on lung cancer relative risks by smoking category given by Rogot and Murray (1980) and the distribution of the US population by smoking status in 1964–65 as published by the National Center for Health Statistics (1967). For Land et al. (2003), these factors
were updated using 1993 information on the smoking status distribution provided by the Centers for Disease Control (1995). The updated distribution differs substantially from that used in the 1985 report, as shown in table D.8. Because the CDC report did not provide data on amount smoked, it was assumed that among current smokers the distribution by amount smoked was the same as that used in the 1985 report (NIH, 1985, p. 50). It was also assumed that the relative risks by smoking category remained appropriate. The revised factors for additive interaction, with the total population as the standard, are given in the last two columns of table D.8.

Not taking into account the findings of Pierce et al. (2003), an approach guided by the BEIR VI findings for radon-related lung cancer risk would be to multiply the ERR$_{1Sv}$ for lung cancer, unadjusted for smoking, by a factor $W_s x (1 - x)$ $W_5 \delta$, where $S$ indexes smoking categories, the $W_s \delta$ are the factors given in columns 4 and 5 of table D.7, and $x$ is assumed to follow a triangular distribution (0, 1, 1.1). This uncertainty distribution for $x$ allows the ERR$_{1Sv}$ for lung cancer to range from that obtained with an additive interaction ($x = 0$) to that obtained with a multiplicative interaction ($x = 1$), with a probability of about 0.10 for a super-multiplicative interaction ($x > 1$). The median of the uncertainty distribution is 0.74, and at this value, $W_5 = 1.97$ for male never-smokers, 0.87 for male ever-smokers, 1.75 for female never-smokers, and 0.85 for female ever-smokers. Thus, at the median value, the estimated ERR$_{1Sv}$ for never-smokers would be a little more than twice that for ever-smokers. A ratio of 2 was used by the BEIR VI committee, and was obtained from analyses of uranium miner data (NRC, 1999, p. 154).

Table D.7 Computation of the uncertainty distribution for ERR at 1 Sv. Likelihood profile distributions $\alpha$ for cancers of the lung and female genital organs other than the ovary associated with exposure to low-LET radiation

<table>
<thead>
<tr>
<th>Profile quantiles</th>
<th>Lung (never-smokers)</th>
<th>Female genital (excluding ovary)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Both genders $^a$</td>
<td>Females $^b$</td>
</tr>
<tr>
<td>0.9975</td>
<td>1.822</td>
<td>0.172</td>
</tr>
<tr>
<td>0.995</td>
<td>1.724</td>
<td>0.136</td>
</tr>
<tr>
<td>0.9875</td>
<td>1.590</td>
<td>0.0866</td>
</tr>
<tr>
<td>0.975</td>
<td>1.482</td>
<td>0.0791</td>
</tr>
<tr>
<td>0.95</td>
<td>1.368</td>
<td>0.0607</td>
</tr>
<tr>
<td>0.875</td>
<td>1.200</td>
<td>0.0463</td>
</tr>
<tr>
<td>0.8413</td>
<td>1.152</td>
<td>0.030</td>
</tr>
<tr>
<td>0.5</td>
<td>0.8603</td>
<td>-0.189</td>
</tr>
<tr>
<td>0.1587</td>
<td>0.6127</td>
<td>-0.278</td>
</tr>
<tr>
<td>0.125</td>
<td>0.5792</td>
<td>-0.289</td>
</tr>
<tr>
<td>0.05</td>
<td>0.4750</td>
<td>&lt;0</td>
</tr>
<tr>
<td>0.025</td>
<td>0.4133</td>
<td>&lt;0</td>
</tr>
<tr>
<td>0.0125</td>
<td>0.3610</td>
<td>&lt;0</td>
</tr>
<tr>
<td>0.005</td>
<td>0.3024</td>
<td>&lt;0</td>
</tr>
<tr>
<td>0.0025</td>
<td>0.2642</td>
<td>&lt;0</td>
</tr>
</tbody>
</table>

$^a$ For lung cancer, ERR at 1 Sv = $\alpha \times h^s(s, e, a, \beta, \gamma, \delta \)}$, where independence is assumed between $\alpha$ and $h^s(s, e, a, \beta, \gamma, \delta \}) = \exp(\beta x s + \gamma f(e) + \delta g(a))$, and where $s = -0.5$ for males and 0.5 for females. $h^s(s, e, a, \beta, \gamma, \delta \})$ is assumed to be lognormally distributed with:

$GM = \exp(0.843 s - 0.0525 f(e) - 1.626 g(a))$

$GSD = \exp[(0.0625 s^2 - 2 \times 0.000047 s \times f(e) + 0.000033 s \times g(a) + 0.00830 s \times f(e)^2 - 2 \times 0.00708 f(e) \times g(a) + 0.562 g(a)^2)]^{1/2}$

$^b$ For female cancers other than ovary, for which $\gamma$ and $\delta$ were assumed to be zero, the statistical uncertainty distribution of $\alpha = ERR$ at 1 Sv is completely specified by the tabulated likelihood profile distribution.
Table D.8 Factors for adjusting the lung cancer ERR$_{1Sv}$ for smoking status under the assumption of an additive interaction model

<table>
<thead>
<tr>
<th>Smoking category (S)</th>
<th>Used in the 1985 report</th>
<th>Used in deriving uncertainty distribution for this report (WS$^*$)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Males</td>
<td>Females</td>
</tr>
<tr>
<td>Total</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Never-smokers</td>
<td>6.81</td>
<td>4.64</td>
</tr>
<tr>
<td>Former smokers</td>
<td>1.71</td>
<td>1.17</td>
</tr>
<tr>
<td>Present smokers (all)</td>
<td>0.604</td>
<td>0.411</td>
</tr>
<tr>
<td>&lt;10 cigarettes/day</td>
<td>1.75</td>
<td>1.19</td>
</tr>
<tr>
<td>10–20 cigarettes/day</td>
<td>0.71</td>
<td>0.48</td>
</tr>
<tr>
<td>21–39 cigarettes/day</td>
<td>0.41</td>
<td>0.28</td>
</tr>
<tr>
<td>40+ cigarettes/day</td>
<td>0.29</td>
<td>0.20</td>
</tr>
<tr>
<td>Ever-smoker (present and former smokers)</td>
<td>0.73</td>
<td>0.47</td>
</tr>
</tbody>
</table>

*These percentages were obtained by assuming that the distribution by amount smoked among current smokers was the same as that used in the 1985 report (Land et al., 2003, p. 41).

However, the analysis of Pierce et al. (2003) suggests that the radiation-smoking interaction among LSS subjects is more nearly additive than that estimated for uranium miners. Accordingly, for external radiation the Working Group adopted an uncertainty model for interaction that puts 50 per cent probability on the additive model and 50 per cent on the model described in the preceding paragraph. Of course, because the profile in table D.7 corresponds to never-smokers, the tabulated values WS$^*$ were normalized to the never-smoker standard, i.e. they were divided by 4.74 for males and by 3.90 for females.

D.6.4 Non-melanoma skin carcinoma: interaction between ionizing and ultraviolet radiation

Ron et al. (1998) found significantly different ($p < 0.02$) ERR$_{1Sv}$ values for basal cell skin carcinoma (BCSC) occurring on the face and hands (0.4, 90 per cent CI –0.1 to 2.1) and on the rest of the body (4.7, 1.2 to 1.3), suggesting a sub-multiplicative, or possibly even additive, interaction between UV and ionizing radiation. This finding suggests that ERR$_{1Sv}$ in lighter-skinned, and therefore more UV-sensitive, populations could be less than that observed in the LSS population. On the other hand, Shore et al. (2002) reported 124 BCSC cases among 1,699 white patients treated by X-ray during childhood for scalp ringworm, compared to 21 among 1035 white non-exposed patients. Among African-Americans, however, only 3 BCSC cases were seen among 525 exposed patients compared to 0 among 345 non-exposed patients. This result, unlike that of Ron et al. (1998), is inconsistent with additive interaction between ionizing radiation and protection from ultraviolet radiation by skin pigmentation or clothing, as risk factors for BCSC. Judging that we do not now have a good basis for evaluating this interaction, the Working Group has chosen to use the general “complete ignorance” uncertainty model discussed in section IV.G of Land et al. (2003) for transfer of risk estimates from one population to another, for transfer of ERR$_{1Sv}$ estimates for non-melanoma skin cancer from the LSS population to identifiable US sub-populations with (on average) different levels of skin pigmentation.
Table D.9 shows population non-melanoma skin cancer incidence rates (cases per 100,000 per year, directly standardized to the age distribution of the 1970 US population) for African-American, Hispanic and non-Hispanic White Americans (Scotto et al., 1996, table 60-4) and Japanese (Muir et al., 1987).

<table>
<thead>
<tr>
<th></th>
<th>United States</th>
<th></th>
<th>Japan</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>African-American</td>
<td>Hispanic</td>
<td>Non-Hisp. White</td>
</tr>
<tr>
<td>Males Rate</td>
<td>4.1</td>
<td>61.6</td>
<td>461.2</td>
</tr>
<tr>
<td>Males Std. error</td>
<td>0.83</td>
<td>4.77</td>
<td>4.38</td>
</tr>
<tr>
<td>Females Rate</td>
<td>4.5</td>
<td>45.1</td>
<td>246.1</td>
</tr>
<tr>
<td>Females Std. error</td>
<td>0.76</td>
<td>3.49</td>
<td>2.86</td>
</tr>
</tbody>
</table>

Thus, for additive interaction model transfer of LSS-based ERR$_{1Sv}$ to US Hispanic males, ERR$_{1Sv}$ was multiplied by the ratio $6.05/61.6 = 0.098$ and, for additive transfer to US African-American females, the multiplier was $4.42/4.5 = 0.98$. Non-melanoma cancer rates were not available for the remaining two US Census racial/ethnic groups, Asians and Pacific Islanders, and Native Americans, and the LSS ERR$_{1Sv}$ estimate was applied to those groups without correction for transfer (i.e. a multiplicative interaction was assumed). Finally, the additive interaction model multiplier for an optional category, “all races/race not specified”, was computed as the weighted mean of subpopulation-specific multipliers according to the projected 2000 distribution of the US population: 12 per cent African-American, 11 per cent Hispanic, 72 per cent non-Hispanic White, and 5 per cent Native Americans, Asians and Pacific Islanders.

**D.6.5 IREP**

The NCI/CDC report (Land et al., 2003) replaced the extensive tables in the original radioepidemiology tables report (NIH, 1985) by IREP, which uses Monte Carlo simulation to calculate the assigned share that might pertain to individual cases. The resulting uncertainty distribution contains essentially all the relevant information, epidemiological (i.e. based on the original data), subjectively interpretive (e.g. with respect to the relativeness of different qualities of radiation, DDREF, or population transfer of risk), pertaining to assigned share and its uncertainty. IREP was intended as an interim update of the 1985 report, requiring revision after publication of the 2006 BEIR VII report (NRC, 2006) and the second comprehensive RERF report based on the RERF Tumor Registry, and was therefore designed to incorporate new data, both epidemiological and interpretive, and new risk models, as they are developed.
Appendix E: The ASQRAD software

E.1 General presentation of the calculation tool

ASQRAD (Assessment System for the Quantification of Radiation Detriment) is a simple Windows-based tool for PCs developed jointly by CEPN (Centre d’étude sur l’Évaluation de la Protection dans le domaine Nucléaire) and HPA (Health Protection Agency, former NRPB) within the EC radiation protection research programme in the mid-1990s (Degrange et al., 1997; Schneider et al., 1994). This software is devoted to the calculation of the lifetime radiation risk allowing for the combination of two main databases: demographic parameters, and radiation health effect risk coefficients.

• The demographic parameters include the age and sex distribution of the population, together with the death rates from all causes and from different types of cancer, and cancer lethality fractions or incidence rates.

• The radiation health effect risk coefficients are the updated risk estimates recommended in recent years by international committees (UNSCEAR, BEIR).

The software provides risk estimates to an individual or to a population in various situations of exposures, i.e. acute or extended exposure; whole body or specific organ exposure. The detriment can be expressed as the expected number of excess deaths as well as the loss of life expectancy and the number of excess non-fatal cancers. The software provides tables and graphics and allows for sensitivity analysis on the population parameters, health effect risk coefficients, age at exposure, level of dose, and so on. Figure E.1 provides the main screen for individual risk calculation.

Furthermore, a database management system allows the introduction of new demographic data and radiation health effect risk coefficients.

E.2 Example of application

In the perspective of assessing the risk attributable to occupational radiation exposure, the use of ASQRAD allows us to describe the different steps of calculation for a specific population, to put them into perspective, and to perform a sensitivity analysis on the main parameters (Lepicard et al., 2004). In order to illustrate this type of result, an example of a calculation is presented below, based on French demographic data for the reference year of 1997 (WHO, 2000). The calculations have been performed with the UNSCEAR model for leukaemia (absolute risk model) and all cancer except leukaemia (relative risk model) (UNSCEAR, 1994) and considering a male or a female exposed at work from age 20 to 55 at 20 mSv per year. A dose and dose rate effectiveness factor (DDREF) of 2 was applied on the initial model for calculations.
Concerning the background mortality rates, figure E.2 presents the distribution of death rates by age and sex, assuming the individual is alive at age $a$. Among all causes of death, cancers represent 30 per cent for males and 19 per cent for females, while leukaemia represents 0.97 per cent for males and 0.82 per cent for females.

Figure E.2 Distribution of background mortality for the specific cancer (France, 1997)
From these demographic data, and applying the UNSCEAR model published in 1994, the following values have been derived from ASQRAD for the excess risk of mortality (being relative for all cancer except leukaemia and absolute for leukaemia and applying a DDREF of 2), corresponding to:

\[
\text{ERR (} D, s, e, a \text{)} \text{ for all cancer except leukaemia (excess relative risk)}
\]

\[
\text{EAR (} D, s, e, a \text{)} \text{ for leukaemia (excess absolute risk)}
\]

with: \( D = \text{dose} \), \( s = \text{sex} \), \( e = \text{age at exposure} \), \( a = \text{attained age} \)

**Figure E.3** Distribution of excess relative risk/absolute risk for occupational exposure at 20 mSv/year from age 20 to 55 (France, 1997)

<table>
<thead>
<tr>
<th>Age range</th>
<th>Excess relative risk (solid cancers)</th>
<th>Excess absolute risk (leukaemia)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-20</td>
<td>2.0E-04</td>
<td>0.0E+00</td>
</tr>
<tr>
<td>20-40</td>
<td>1.5E-04</td>
<td>5.0E-05</td>
</tr>
<tr>
<td>40-60</td>
<td>1.0E-04</td>
<td>1.0E-04</td>
</tr>
<tr>
<td>60-80</td>
<td>5.0E-05</td>
<td>1.5E-04</td>
</tr>
<tr>
<td>80-100</td>
<td>2.5E-04</td>
<td>2.0E-04</td>
</tr>
</tbody>
</table>

Considering the lifetime excess risk, the following results were derived:

<table>
<thead>
<tr>
<th>All cancer except leukaemia (%)</th>
<th>Leukaemia (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male 3.3</td>
<td>0.51</td>
</tr>
<tr>
<td>Female 3.6</td>
<td>0.49</td>
</tr>
</tbody>
</table>

Based on these results, it is possible to derive the assigned share using the following formulae:

\[
\text{AS} = \frac{\text{ERR}}{\text{ERR} + 1} \text{ (for all cancer except leukaemia)}
\]

\[
\text{AS} = \frac{\text{EAR}}{B_c + \text{EAR}} \text{ (for leukaemia)}
\]

The results are presented in figure E.4 for males and females according to different ages attained for all cancer except leukaemia and for leukaemia.
From this figure, one can derive that for a man (respectively a woman) developing a cancer (except leukaemia) at age 60, the assigned share would be about 10 per cent (respectively 17 per cent) for occupational exposure at 20 mSv per year from age 20 to 55. For leukaemia, the result would be around 70 per cent.

### E.3 Concluding remarks

In the scope of the calculations of attributable risk, ASQRAD provides the ability to describe the distribution of risk according to age and puts them into perspective with the background risk for each cause of death. It also allows the use of different types of indicators and performing specific calculations for the concerned population. This approach could be useful to open the dialogue on risk calculations with exposed workers.

Furthermore, as already mentioned, ASQRAD provides a useful database management system in order to perform sensitivity analyses on the different risk models (ability to cope with new models), demographic data (regular update and ability to adapt to the characteristics of the concerned population), and exposure scenario.

Finally, ASQRAD can be used for prospective risk calculations on the occurrence of radiation-induced cancers among exposed populations, taking into account the past exposure of the population and according different exposure scenarios for the future.

In the future, new developments could be envisaged in order to update the available data (demographic tables and risk models) and the software environment, as well as to cope with incidence calculations.
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