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Radiation Detriment Calculation Methodology

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[GUEST] EDITORIAL

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RADIATION DETRIMENT CALCULATION METHODOLOGY

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Abstract-Radiation detriment is a concept used to quantify the harmful stochastic effects of low-level radiation exposure to the human population. It is determined from lifetime risk of cancer for a set of tissues and organs taking into account their severity in terms of lethality, quality of life, and years of life lost. It also considers heritable effects. The radiation detriment is estimated as a sex- and age-averaged risk indicator for a composite reference population. This report provides a historical review of the detriment calculation methodology adopted by the International Commission on Radiological Protection (ICRP) since *Publication 26* and a detailed description of the whole computation process used in *Publication 103*. It clarifies data sources, risk models, computational methods and rationale for the choice of parameter values. The parameters that have the greatest influence on the radiation detriment calculation are also identified based on a series of sensitivity analyses. They include dose and dose-rate effectiveness factor (DDREF), age at exposure, sex difference and lethality fraction. Although the current scheme of radiation detriment calculation is well established, it may need to evolve to take into account changes in baseline reference data (mortality, cancer incidence and lethality) in recent decades and progress in scientific understanding of radiation health effects. In this perspective, the report suggests ways to update and improve the estimation of key parameters for the calculation of radiation detriment, such as the reference population data and cancer severity. There is also room for improvement in cancer risk models based on the accumulation of recent epidemiological findings. Finally, the importance of improving the comprehensibility of the radiation detriment concept and the transparency of its calculation methodology is emphasised.

Keywords: Radiation detriment; Nominal risk; Sensitivity analysis; Stochastic effects

MAIN POINTS

- **Radiation detriment is a concept used to quantify the health impact of stochastic effects (cancer and heritable effects) from low-dose and low-dose-rate radiation exposures, considering both the probability of occurrence and the severity of these effects.**

- **The method for calculating radiation detriment consists of two main parts: calculation of nominal risk (average estimate of the lifetime cancer risk and the risk of heritable effects associated with radiation exposure) and adjustment for lethality, quality of life and years of life lost.**

- **Sensitivity analysis identified dose and dose-rate effectiveness factor (DDREF), age at exposure, sex and lethality fraction as parameters having a large impact on the estimation of radiation detriment.**

- **Radiation detriment needs to be updated considering changes in reference population data, variation of cancer risk with sex and age and between different populations, cancer severity parameters, improvement in cancer risk models, and review of risk estimates for heritable effects.**

EXECUTIVE SUMMARY

(a) The concept of radiation detriment has been developed by the Commission for the purpose of radiological protection. It is defined as the excess of stochastic health effects in a group of exposed individuals to low-level radiation and their descendants compared to a non-exposed group. It is determined from sex-averaged and age-at-exposure-averaged lifetime risk estimates for a set of organs and tissues, taking into account the severity in terms of lethality, quality of life, and years of life lost.

(b) Radiation detriment at low doses or low dose-rates is quantified assuming a linear-non-threshold (LNT) dose-response relationship for stochastic effects and applying a dose and dose-rate effectiveness factor (DDREF) of 2 for solid cancer.

(c) The methodology for calculating radiation detriment has developed over decades since the concept was first introduced in *Publication 22*. The most recent method in *Publication 103* consists of two main parts. The first part is the calculation of nominal risks, which are average estimates over age groups of the lifetime cancer incidence risks and the risk of heritable effects associated with radiation exposure. The lifetime risk of cancer incidence is calculated separately for four reference populations (males and females of Euro-American and Asian populations) except for bone and skin cancers, and results are averaged across sexes and regions. The second part is the adjustment for the severity of the consequences. All calculation steps are executed separately for individual organs/tissues or group of tissues, and the resulting values are added up to give the total radiation detriment.

(d) The calculation of the nominal cancer risk involves a number of sequential steps. The procedure adopted in *Publication 103* is summarised below:

- Baseline cancer rates are computed using cancer incidence data from selected Asian and Euro-American populations to compile rates for representative populations in different parts of the world.
- Cancer risk models are developed from the analysis of cancer incidence data from the Life Span Study (LSS) of the atomic bomb survivors. The excess relative risk (ERR) and the excess absolute risk (EAR) are modelled with modifying effects of sex, age at exposure, and attained age.
- The minimum latency period is assumed to be five years for solid cancers and two years for leukaemia.
- The risk of exposure-induced cancer incidence (REIC) is calculated for an acute exposure of 0.1 Gy and multiplied by 10 to obtain the lifetime risk at 1 Gy for each cancer site. It is computed for each age at exposure, 0 to 84 years for the whole population and 18 to 64 years for adult workers, by cumulating the risk until the attained age reaches 90 years.
- The weighted mean of REIC for each age at exposure is calculated to give the age-averaged lifetime risk, the weight being proportional to the age distribution of the reference population.
- The ERR and EAR lifetime risks are weight-averaged according to weighting factors specified for each organ or tissue.
- The lifetime risk estimates are adjusted downward by a DDREF of 2 for all cancer sites except for leukaemia for which a linear-quadratic model is used.

- The unweighted mean of the resulting values between the four reference populations yields a nominal risk for each cancer site.
- The total nominal risk is calculated as the sum of nominal risks estimated for 13 categories of cancer with the consideration of additional risk reflecting heritable effects.

(e) The calculation of radiation detriment is based on a weighting procedure in which nominal cancer risks are adjusted by three parameters reflecting lethality, quality of life and years of life lost. These three parameters are independent of radiation dose. Their determination is partly based on expert judgement, and the values used do not consider differences with age, sex, or between populations.

(f) Sensitivity analysis on radiation detriment was conducted for nine solid cancers and a group of other solid cancers to examine the potential impact of assumptions made in the calculations. Depending on their level of impact, three categories were identified.

- Limited impact: minimum latency period, maximum attained age, lifetime risk calculation method, minimum quality-of-life factor, and relative years of cancer-free life lost.
- Noticeable impact on some cancer sites: reference population and transfer model.
- Large impact: DDREF, age at exposure, sex, and lethality fraction.

(g) Considering the variation of cancer risk with sex and age, it is advisable to calculate lifetime risks separately for sexes and selected ages (age groups) and average them in the last stage to obtain a nominal value. This approach distinguishes science-based risk assessment from the subsequent integration of information for protection purposes, thus providing a better understanding of the construction of the radiation detriment. Sex- and age-related variation should also be considered in determining the values of tissue weighting factors, w_T based on the relative detriment. Description of the impact of sex and age at exposure on the relative detriment helps to understand the distribution range and the representativeness of w_T.

(h) Radiation detriment needs to evolve depending on the advances in healthcare and scientific understanding of radiation effects. It will be necessary to update reference population data and cancer severity parameters in the near future. Cancer risk models should be improved and the weighting scheme for transferring risks needs to be validated based on up-to-date epidemiological data. It is also desirable to review the risk estimate for heritable effects taking into account recent studies.

(i) There is considerable uncertainty about the existence or not of a threshold for circulatory disease and cataract and the shape of the dose-response curve at low doses if there is no threshold. Whether or not to include them in the calculation of the radiation detriment currently remains an open question.

(j) Ensuring transparency and traceability of the radiation detriment calculation is important. A full description of the calculation steps of the radiation detriment is necessary, and consideration should be given to the development of an open-source software to perform these calculations. It is also desirable to improve the way of expressing radiation detriment and to illustrate the data of reference populations so that non-specialists can have a balanced perspective on the health risks of radiation.

1. INTRODUCTION

(1) The health effects of radiation are classified into two categories, deterministic effects (harmful tissue reactions) and stochastic effects, i.e., cancer and heritable effects. For low-dose, low-dose-rate exposures, stochastic effects are assumed for radiological protection purposes to follow a dose response with no threshold.

(2) Radiation-associated cancers generally have long latencies, and the length of life lost depends on the distribution of age of onset of the cancers. There are also considerable differences in fatality among cancer sites. To appropriately assess the risk of cancer attributed to radiation exposure, the severity as well as its probability needs to be taken into account. The same holds true for heritable effects as they include a wide range of abnormalities.

(3) The Commission initially introduced the concept of detriment as the mathematical 'expectation' of the harm incurred in a group from a radiation dose (ICRP, 1973, 1977a). It was later replaced by a multi-dimensional concept to properly represent the different aspects of the health impact in order to: (i) assess the consequences of continued or cumulative exposures to recommend dose limits, (ii) compare the consequences of different distributions of equivalent dose within the body and thence to select a set of tissue weighting factors, and (iii) provide a basis for assessing the valuation of a unit of effective dose for use, for example, in the optimisation of protection within a practice (ICRP, 1991).

(4) The Commission has developed a methodology for aggregating different facets of the detriment into a single quantity. It is called radiation detriment, which is calculated as an adjusted excess risk from radiation exposure using this methodology. It is determined from lifetime risk of cancer and heritable effects as an average over different populations, sexes and ages at exposure, taking into account the severity of the disease in terms of lethality, quality of life, and years of life lost. Calculated values for individual organs/tissues or group of tissues are added up to give the total radiation detriment.

(5) Radiation detriment at low doses or low dose-rates is quantified assuming a linear-non-threshold (LNT) dose-response relationship except for leukaemia, which is based on a linear-quadratic dose response. A dose and dose-rate effectiveness factor (DDREF) is applied to solid cancer to adjust the risk estimated from the epidemiological data of high-dose and high-dose-rate exposures. High-dose exposures for which tissue reactions are of concern are strictly out of scope of this methodology, although it does not mean that stochastic effects do not occur at higher dose levels. It is also not recommended to use radiation detriment for assessing the health risk of acute exposures at intermediate dose ranges (e.g. a few hundred millisieverts). At these levels of dose, it would be inappropriate to rely on the LNT model adjusted by the DDREF.

(6) The system of radiological protection applies to any individual who is exposed to ionising radiation, and methods of controlling sources of exposure are usually applied without reference to individual profiles of those exposed. In this regard, it is desirable to set standards and to optimise protection in ways that are independent of age, sex and region of the world. This approach emphasises respect for equity and fairness from an ethical point of view. Radiation detriment is therefore computed by averaging the risk estimates over age groups, both sexes and geographical regions to represent the risk for a nominal population. As the calculation process involves the risk transfer and averaging across populations with differing baseline cancer rates, the nominal population is regarded as a mixture of people with different factors governing individual responses to radiation including not only non-modifiable factors, but also modifiable lifestyle factors. This was clearly demonstrated in ICRP *Publication 115* (ICRP, 2010), in which the nominal risk coefficient for radon exposure was defined for a mixed adult population of non-smokers and smokers.

(7) The Commission believes that the system of radiological protection, which has been developed on the basis of the nominal risk approach, is simple, non-discriminatory and globally applicable while achieving adequate protection for every individual regardless of age, sex and region of the world. Radiation detriment can be used for prospective risk assessment of exposure situations for radiological protection purposes or to assess risks in retrospective situations for exposures of identified individuals. However, it should be noticed that there are significant differences in risk between sexes and in respect of age at exposure. For the estimation of the likely consequences of an exposure of a given individual or population, it is preferable to use specific data relating to the exposed individuals when they are available.

(8) Radiation detriment is intended to be a reliable, robust indicator of the overall burden of stochastic effects, and as such, it needs to reflect the latest scientific information and the changes in population health statistics. The methodology of its calculation has been developed over decades to meet these requirements. This report provides a historical review of the methodology for calculating radiation detriment adopted by ICRP since *Publication 26* (ICRP, 1977) and a detailed description of the computation process used in *Publication 103* (ICRP, 2007). Data sources, risk models, computational methods and the rationale for the parameter values adopted are detailed for each step of the process. This is followed by a series of sensitivity analyses to identify the primary sources of uncertainty in the radiation detriment calculation. Based on the results, some key issues are discussed for future consideration.

2. HISTORY OF RADIATION DETRIMENT CALCULATION

2.1. *Publication 26*

(9) The concept of detriment was first introduced in ICRP *Publication 22* (ICRP, 1973). It was maintained in *Publication 26* (ICRP, 1977a) and defined as follows: '*The deleterious effects of exposure to radiation may be of many kinds. Among the effects on health there may be both stochastic and non-stochastic effects in the exposed individual and stochastic effects in later generations. … The Commission has introduced the concept of detriment to identify, and where possible to quantify, all these deleterious effects. In general, the detriment in a population is defined as the mathematical "expectation" of the harm incurred from an exposure to radiation, taking into account not only the probability of each type of deleterious effect, but also the severity of the effect'*.

(10)In *Publication 26* (ICRP, 1977a), a quantitative value for the detriment at low dose and low dose rate relied on a linear model. *Publication 26* noted that linear extrapolations may lead to an overestimate of the radiation risks at low doses and low dose rates but endorsed this as a cautious assumption. Additionally, while recognising that risks for some cancer sites were age or sex dependent, the Commission judged that for radiological protection purposes sufficient accuracy could be obtained by using an average value for each organ or tissue regardless of age or sex for both workers and the general public. Detriment, specifically called 'risk factor' in *Publication 26*, was expressed as the likelihood of fatal cancers and serious hereditary abnormalities. It was quantified for the following organs/tissues: gonads (including both cancer mortality and hereditary effects in the progeny), red bone marrow, bone, lung, thyroid, breast and 'other tissues'.

(11)The risk factor for leukaemia was taken to be 20 10^{-4} Sv^{-1}. A review by the Commission concluded that bone was much less sensitive than breast, red bone marrow, lung and thyroid and the risk factor for bone cancer was taken to be 5 10^{-4} Sv^{-1}. The risk of lung cancer was about the same as that for the development of leukaemia (e.g. 20 10^{-4} Sv^{-1}). The sensitivity of the thyroid to the induction of cancer by radiation appeared to be higher than that of the red bone marrow for the development of leukaemia. However, the mortality from these thyroid cancers being much lower than for leukaemia, the overall mortality risk factor was considered to be 5 10^{-4} Sv^{-1}. Based on data on the development of female breast cancer following radiation exposure, it was suggested that, during reproductive life, the female breast might be one of the most radiosensitive tissues of the human body. There were indications that, under these circumstances, the risk factor for breast cancer could be a few times higher than that for leukaemia and the risk factor was taken to be 25 10^{-4} Sv^{-1}. In addition to the tissues discussed above, there were other tissues (e.g. stomach, lower large intestine, salivary glands and liver) for which there was evidence that radiation was also carcinogenic at moderate doses, but no risk factors were specified for them. It was estimated that the combined risk of malignancy in all remaining unspecified tissues was unlikely to exceed 50 10^{-4} Sv^{-1}. For gonads, the risk factor for hereditary effects over the first two generations was taken as about 40 10^{-4} Sv^{-1}.

(12)Based on the values described above, the Commission concluded that the mortality risk factor for radiation-induced cancers was about 125 10^{-4} Sv^{-1}, as an average for both sexes and all ages, and that the average risk factor for hereditary effects could be taken as about 40 10^{-4} Sv^{-1}. Results are summarised in Table 2.1.

331 Table 2.1. ICRP *Publication 26* values for nominal mortality risk coefficients.

Organ/tissue	Risk factors (10^{-4} Sv^{-1})
Cancer	
Bone marrow	20
Bone surface	5
Breast	25
Lung	20
Thyroid	5
Remainder*	50
(Total cancer)	(125)
Hereditary effects	
Gonads	40

332 * No specific organs listed.

333 2.2. *Publications 27* and *45*

334 (13)*Publication 27* (ICRP, 1977b) provided supporting guidance to the general
335 recommendations in *Publication 26* (ICRP, 1977a) general recommendations. It aimed to
336 discuss 'the problems entailed in comparing the safety of different industries including those
337 involving radiation exposure, taking account of the fact that the types of injury or induced
338 diseases, and their severity and relative frequencies, might differ completely in different
339 occupations'. By comparing different occupational risk, it aimed to support the value adopted
340 for the occupational dose limit in *Publication 26*.
341 (14)In order to compare different occupational risks, *Publication 27* relied on the calculation
342 of years of life lost for various risks. It concluded that 'If fatal malignancies were induced at a
343 rate of 10^{-4} rem^{-1}, with an equivalent life loss of 15 years for each including the periods of
344 illness from fatal and non-fatal malignancies, the life loss from somatic effects would amount
345 to 1.5 man-years per 1000 man-years per rem of average occupational exposures'. The
346 calculation of the index of harm for ionising radiation took into account fatal cancer as well as
347 non-fatal cancer and associated years of life lost.
348 (15)The assessment of the index of harm in *Publication 27* was revised in *Publication 45*
349 (ICRP, 1985), based on more comprehensive data. For cancers induced by occupational
350 radiation exposure, the risk factors in *Publication 26* were used as the frequency of fatal cases
351 per unit dose, and each case was assumed to bring a mean loss of 15 years of life expectancy
352 plus 1 additional year to take into account the period of illness prior to death (i.e. 16 years of
353 life lost per case). Lethality data of different types of cancer were reviewed to estimate the
354 induction rates and severity of the non-fatal (curable) component, which led to the weighting
355 of 0.29/1.26 for them as shown in Table 2.2. The resultant life-loss detriment from all cancer
356 induction was 0.3 y Sv^{-1} in females and 0.2 y Sv^{-1} in males.
357

Table 2.2. Weighting of detriment from curable cancers in *Publication 45* (ICRP, 1985).

Organ/tissue	Risk of induction (10^{-2} Sv^{-1})			Severity of cure*		Cured (10^{-2} Sv^{-1})
	Fatal	*Curable*				
Breast	0.25	0.15	×	0.6	=	0.09
Bone marrow	0.20	0.01	×	0.95	=	0.01
Lung	0.20	0.01	×	0.95	=	0.01
Thyroid	0.05	1.0	×	0.05	=	0.05
Bone	0.05	0.01	×	0.85	=	0.01
Skin	0.01	1.0	×	0.01	=	0.01
Remainder	0.50	0.15	×	0.75	=	0.11
Total	1.26	2.33				0.29

* The ratio of 'fatal' to 'fatal plus curable' cancers of the same type.

(16)For hereditary effects, the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) 1982 report estimated years of life impaired or lost to be 0.63 years per person.Gy[1] of genetically significant radiation at equilibrium after continuous exposure (UNSCEAR, 1982). The genetically significant fraction of collective dose in the working population was estimated from mean ages at conception, 30.6 years for fathers and 25.9 years for mothers. Based on these parameters, life-loss detriment from occupational exposure at a constant rate was assessed to be about one third in women and three quarters in men of that from cancer. At a dose rate of 2 mSv year^{-1} as a representative exposure scenario for the majority of workers, an index of harm expressed as years lost per 1000 worker-years was thus 0.6 and 0.2 for carcinogenic and hereditary effects in females, and 0.4 and 0.3 in males, respectively.

(17)The effects of exposures during pregnancy were also taken into account on the basis that intra-uterine death, mental retardation, cancer and hereditary effects were induced without threshold. With an assumed frequency of 6.5 pregnancies per 100 worker-years of the female population in employment, the index of harm was calculated to be 1.0 per 1000 female worker-years for exposure at 2 mSv year^{-1}.

2.3. *Publication 60*

(18)In its *Publication 60* (ICRP, 1991), the Commission outlined that new information on the risk of radiation-induced cancer in human populations had emerged since 1977 as well as new experimental data in laboratory animals and cultured cells, leading to a reassessment of *Publication 26* (ICRP, 1977a) estimates of the probability of the carcinogenic effects of radiation. The results for the relative probabilities of fatal cancer for males and females were calculated for China, Japan, Puerto Rico, the U.K. and the U.S. for age 0–89 years and averaged. This yielded the values given in the first column in Table 2.3. These values were used as the

[1] Units of dose are shown as in the original reference. Otherwise, Gy is used for nominal risks and Sv for radiation detriments.

basis of the relative probabilities of cancer in organs for a nominal world population of all ages from which to derive the detriment.

(19)In *Publication 60* (ICRP, 1991), the following specific assumptions were made for the thyroid, bone surface, skin and liver.

- For thyroid, the UNSCEAR 1988 Report (UNSCEAR, 1988) and the U.S. National Academy of Sciences' Biological Effectiveness of Ionizing Radiation (BEIR) Report V (NRC, 1990) agreed that the best available estimates of risk to the thyroid were those presented in NCRP Report No. 80 (NCRP, 1985). These estimates gave a lifetime risk for fatal thyroid cancer of 7.5 10^{-4} Gy^{-1}. The fatality rate was stated to be 0.1, thus the incidence was 75 10^{-4} Gy^{-1}.
- For bone surface, based on high linear energy transfer (LET) radiation data, the BEIR IV report (NRC, 1990) provided an estimate of a lifetime incidence of about 133 10^{-4} Gy^{-1}. With a lethality fraction of 0.70, this became 93 10^{-4} Gy^{-1} and about 4.7 10^{-4} Sv^{-1} after application of a quality factor (Q) of 20.
- For skin, *Publication 59* (ICRP, 1992) found the incidence of skin cancer to be 1000 10^{-4} Sv^{-1}, while the fatality (or lethality) fraction was conservatively estimated as 0.2%. The fatal skin cancer risk was presumed to be applicable at low doses and was thus taken to be 2 10^{-4} Sv^{-1}.
- For liver, the data from thorotrast studies in West Germany, Portugal, Japan and Denmark yielded about 300 10^{-4} fatal liver cancers per Gy. With a Q of 20, a nominal risk estimate of 15 10^{-4} Sv^{-1} was derived and applied also for low LET radiation.

407 Table 2.3. Calculation of detriment in *Publication 60*[*] (ICRP, 1991).

Organ/tissue	Probability of fatal cancer F (10^{-4} Sv^{-1})	Severe genetic effects (10^{-4} Sv^{-1})	Relative length of life lost $l/\bar{l}$	Relative non-fatal contribution $(2–k)$	Detriment[**] (10^{-4} Sv^{-1})	Relative contribution to the total detriment
Bladder	30		0.65	1.50	29.4	0.040
Bone marrow	50		2.06	1.01	104.0	0.143
Bone surface	5		1.00	1.30	6.5	0.009
Breast	20		1.21	1.50	36.4	0.050
Colon	85		0.83	1.45	102.7	0.141
Liver	15		1.00	1.05	15.8	0.022
Lung	85		0.90	1.05	80.3	0.111
Oesophagus	30		0.77	1.05	24.2	0.034
Ovary	10		1.12	1.30	14.6	0.020
Skin	2		1.00	2.00	4.0	0.006
Stomach	110		0.83	1.10	100.0	0.139
Thyroid	8		1.00	1.90	15.2	0.021
Remainder	50		0.91	1.29	58.9	0.081
Gonads		100	1.33		133.3	0.183
Total	500				725.3	1.000

408 [*] Definition of symbols
409 F : Probability of fatal cancer
410 l : Expected years of life lost
411 $\bar{l}$: Average of l for all cancers (15.0 years)
412 k : Lethality fraction
413 [**] Detriment is given by F $(l/\bar{l})$ $(2–k)$
414

415 (20)In addition to nominal estimates of fatal cancer, the detriment calculated in *Publication*
416 *60* included three additional components:

417 • A specific allowance for differences in lethality which resulted in different values of
418 expected life lost for fatal cancer originating in different organs;

419 • An allowance for the morbidity resulting from induced non-fatal cancers; and

420 • An allowance for the risk of serious hereditary disease in all future generations descended
421 from the irradiated individual.

422 (21)To allow for the detriment associated with non-fatal cancers, the detriment of each
423 cancer type included a non-fatal component weighted according to the lethality fraction k. Thus,
424 if in a given tissue there were F fatal cancers, the total number of cancers was F / k. The number
425 of non-fatal cancers was then $(1 – k)$ F / k and the total weighted detriment was $F + k$ $[(1 – k)$
426 $F / k]$ or F $(2 – k)$.
427 (22)Steps in the calculation of the detriment are detailed in Table 2.3. It shows how the
428 probability of fatal cancer of 500 (considering only fatal cancer) develops into a detriment of
429 725 per 10,000 person.Sv. This part of the methodology is based on risk characteristics

associated with cancer types and hereditary disease. It is not directly related to radiation exposure.

(23)The relative contributions of the organs to the total detriment (last column) formed the basis of the Commission's tissue weighting factors.

2.4. *Publication 103*

(24)*Publication 103* (ICRP, 2007) adopted a new calculation methodology. While the methods used were broadly similar to those used in *Publication 60* (ICRP, 1991), modifications were made in several aspects of the computations. Of these, one major change was the move to base nominal risk calculations on cancer incidence data rather than on cancer mortality data. For clarification, the detriment calculated using this methodology is specifically called 'radiation detriment', and the term 'detriment' means radiation detriment hereafter unless otherwise noted.

(25)The *Publication 103* methodology of radiation detriment calculation is detailed in Section 3 with an effort to avoid imprecisions and ambiguities (its outline is also provided in Cléro et al., 2019). The description presented herein should be considered an improved and corrected version of that provided in *Publication 103*.

3. CALCULATION OF RADIATION DETRIMENT

(26)The procedure for calculating radiation detriment is described in Annex A.4 of *Publication 103* (ICRP, 2007). Background information about cancer risk estimation is also given in Annex B of *Publication 60* (ICRP, 1991).

(27)This calculation procedure has two major parts each of which consists of sequential steps (Fig. 3.1). The first part is the calculation of the nominal risk: an estimate of the lifetime risk associated with radiation exposure, including the risk of cancer and heritable diseases. Risk estimates of cancer are averaged across sexes, ages at exposure and geographical regions for each cancer site. The second part is the adjustment for severity, which takes into account lethality, quality of life, and years of life lost. As shown in Fig. 3.1, only the first part depends on radiation dose. The second part is virtually independent of radiation exposure, but reflects the severity of cancer (also heritable disease for the gonads) of respective organs or tissues.

(28)In this publication, Gy is used as the dose unit for the calculation of nominal risk (First part) and Sv is used for the calculation of radiation detriment (Second part).

(29)Averaging across sexes, ages or geographical regions is applied at different steps in the process of detriment calculation. The lifetime risk of cancer is calculated separately for males and females, and for the two reference populations (except for bone and skin cancers), and the results are averaged to estimate the nominal risk. The estimate of the excess risk of heritable effects and the adjustment factors including the DDREF, the lethality fraction and the parameters related to quality of life are applied without distinguishing between sexes or population groups. All steps are conducted in parallel for each organ and tissue separately, and the resulting values are finally summed to give the total radiation detriment.

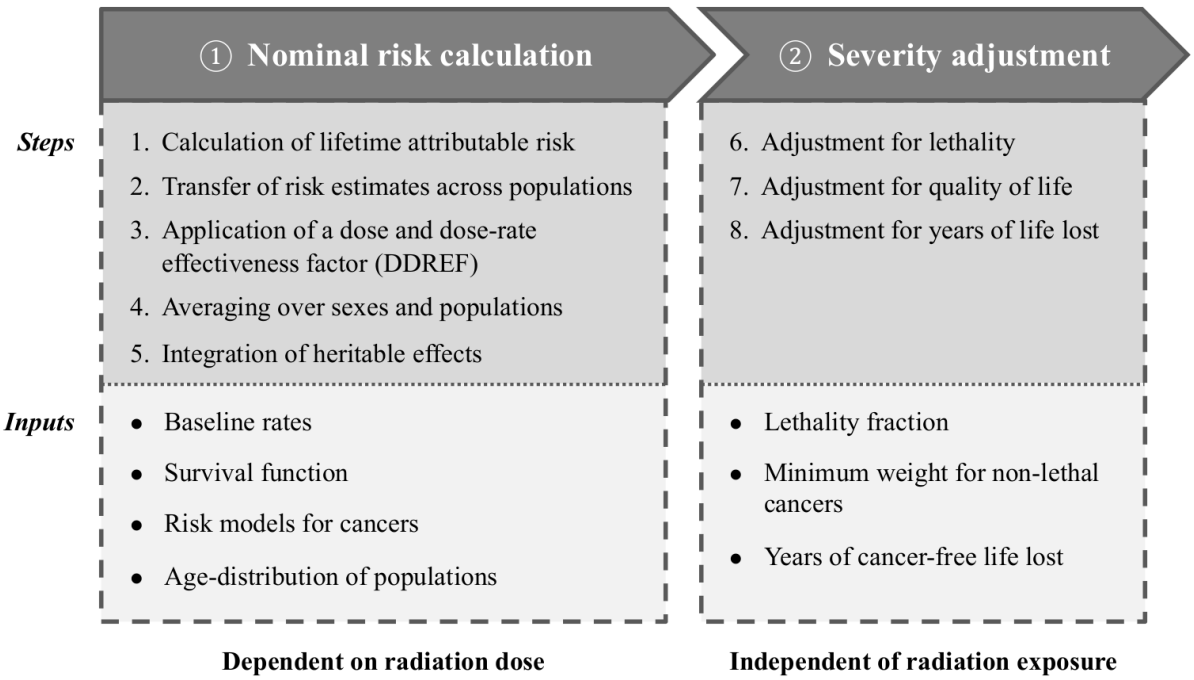


Fig. 3.1. Calculation procedure of radiation detriment in *Publication 103* (ICRP, 2007).

3.1. Nominal risk calculation

3.1.1. Cumulative baseline risk

3.1.1.1. Reference populations

(30)Composite baseline incidence rates of cancer were computed using cancer incidence data from selected Asian and Euro-American populations with long-running cancer registries: Shanghai (China), Osaka, Hiroshima and Nagasaki (Japan), Sweden, United Kingdom, and the Surveillance, Epidemiology, and End Results (SEER) program of the U.S. National Cancer Institute. An unweighted average of the Asian and the Euro-American data was calculated to form a composite population. The aim was to compile rates for representative populations in different parts of the world. Population size data were obtained from the World Health Organization (WHO) international mortality statistics database (WHO population data file downloaded April 22, 2003: http://www.who.int/healthinfo/mortality_data/en/).

3.1.1.2. Baseline cancer rates

(31)Population-based cancer incidence rates were obtained from the 8th edition of Cancer Incidence in Five Continents (cancer rates measured by registries during the period 1993–1997 (Parkin et al., 2002). Incidence data are available for all cancer sites except for bone and skin. Average incidence rates were compiled for the Asian and Euro-American populations, separately for males and females and by 5-year age categories (from 0–4 to 90+), for the oesophagus, stomach, colon, liver, lung, female breast, ovary, bladder, thyroid, leukaemia, leukaemia excluding chronic lymphocytic leukaemia (CLL), all solid cancers and all cancers combined. In addition, mortality rates for each cancer category and for all causes combined were also provided (Tables A.4.10 to A.4.17, in *Publication 103* (ICRP, 2007)).

3.1.1.3. Survival functions

(32)The survival functions (Fig. 3.2) were derived from the mortality rates estimated for the four reference populations (males and females each in Asian and Euro-American populations), obtained from the 8th edition of Cancer Incidence in Five Continents (Parkin et al., 2002).

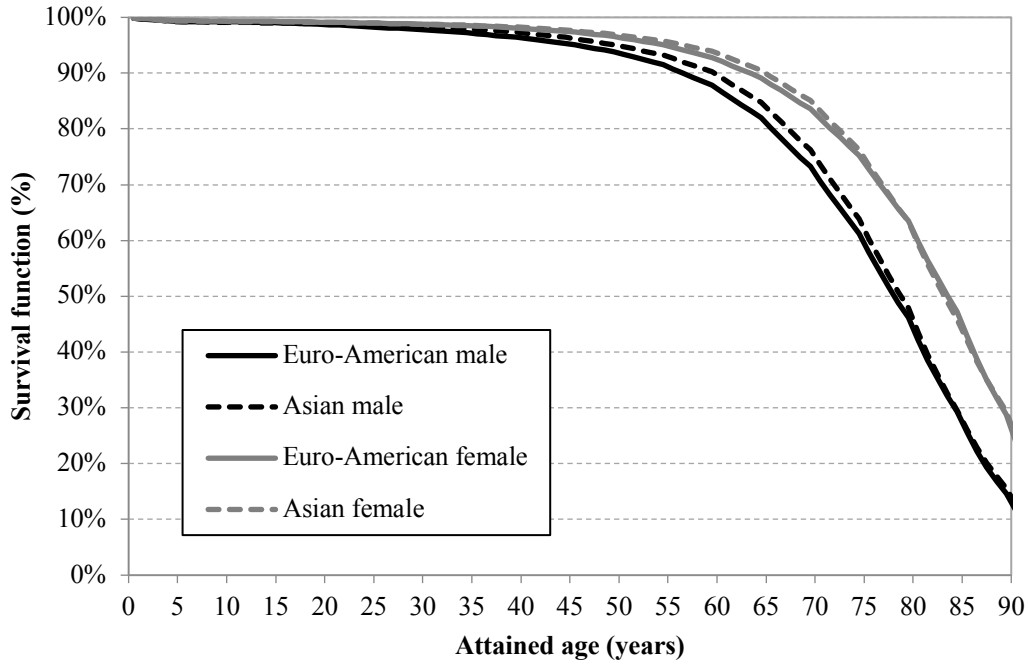


Fig. 3.2. Survival function of reference populations.

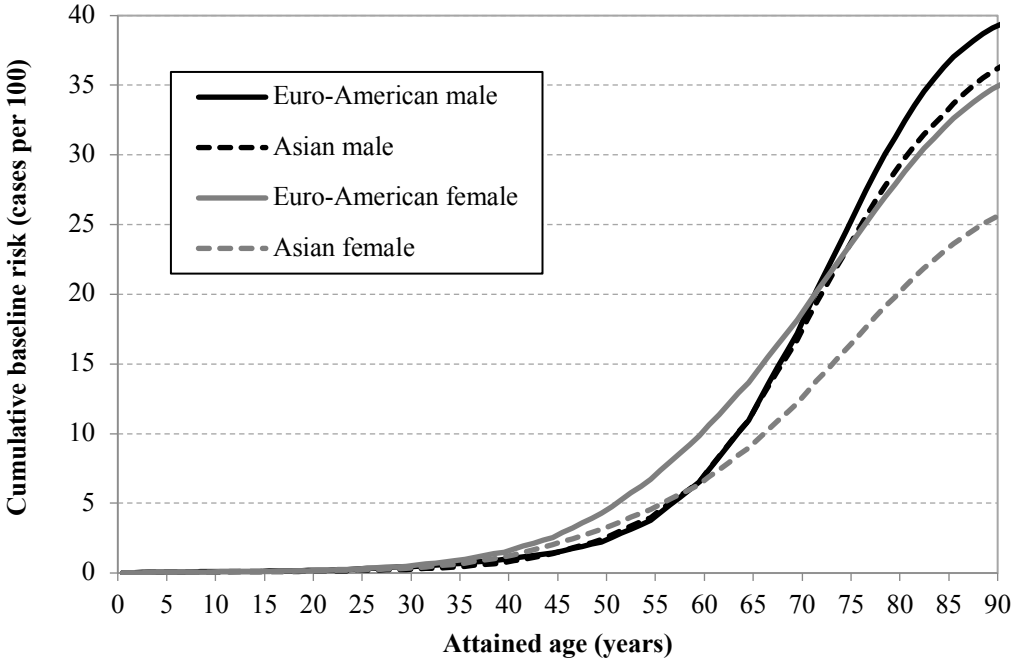
3.1.1.4. Calculation of cumulative baseline cancer risk

(33)The lifetime baseline risk (LBR) is the cancer risk in the absence of radiation exposure cumulated up to reaching the age of 90 years old.

$$LBR(a_{min}, s) = \int_{a_{min}}^{a_{max}} \mu_i(a,s) S(a|a_{min}, s) da$$

where s = sex, a_{min} = age at the beginning of risk, a_{max} = maximum age (*i.e.* 90 years), $\mu_i(a,s)$ = age- and sex-specific baseline cancer incidence rates, and $S(a|a_{min},s)$ = survival function (i.e. the sex-specific probability to be alive at age a for a person alive at age a_{min}).

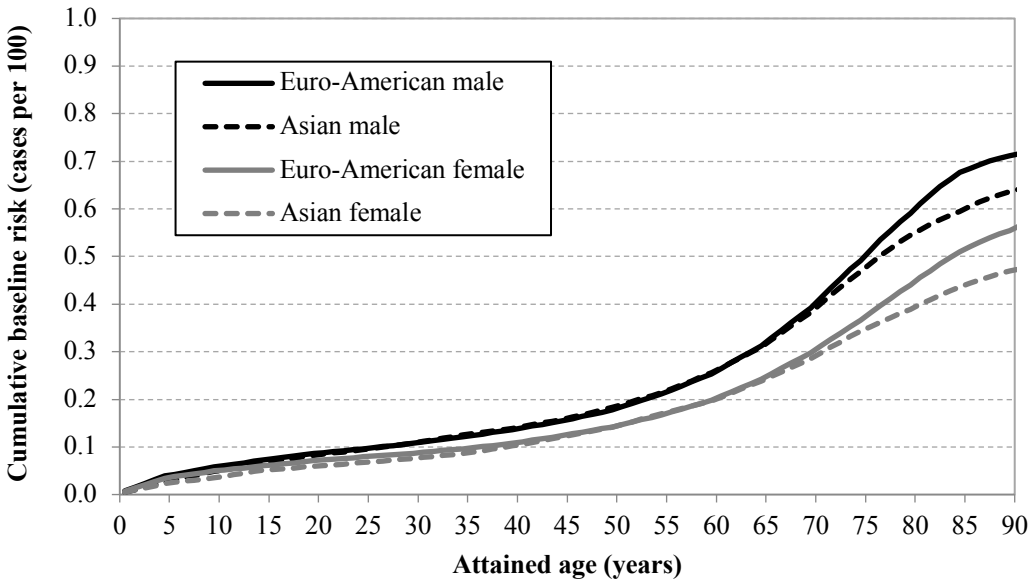
(34)For illustration, cumulative baseline risks are presented in Fig. 3.3 for all solid cancers, Fig. 3.4 for non-CLL leukaemia, and Fig. 3.5 for female breast cancer incidence. For most cancer sites, cumulative baseline risks are higher in males than in females (oesophagus, colon, lung, bladder, non-CLL leukaemia, and all solid cancers). In both sexes, stomach and liver cancer incidence is higher for Asian than for Euro-American populations. For female breast cancer, baseline rates vary and are markedly higher for Euro-American than for Asian populations.



520

521 Fig. 3.3. Cumulative baseline risk for all solid cancer incidence in reference populations.

522



523

524 Fig. 3.4. Cumulative baseline risk for all non-CLL leukaemia incidence in reference populations.

525

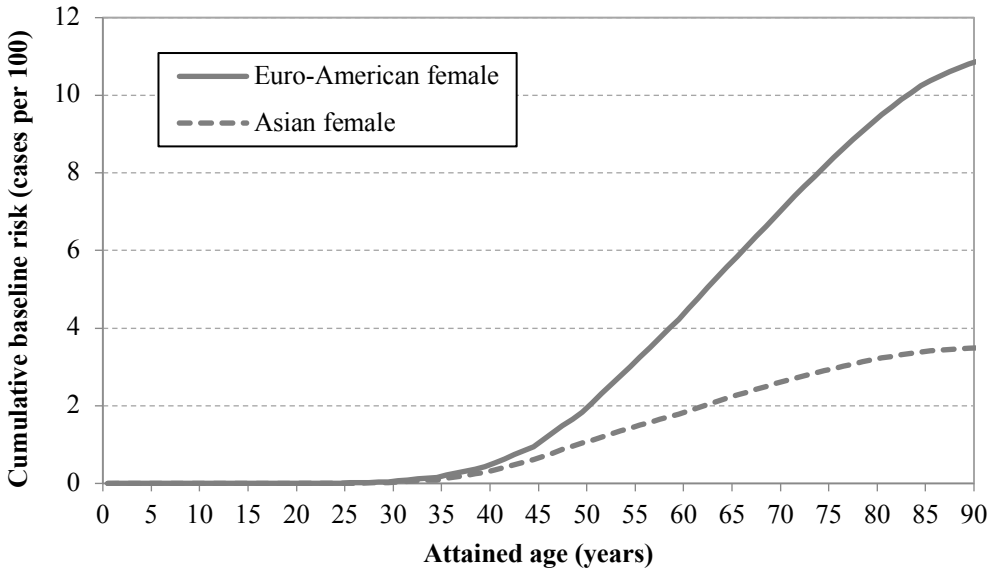


Fig. 3.5. Cumulative baseline risk for female breast cancer incidence in reference populations.

3.1.2. Risk models for radiation-associated cancers

3.1.2.1. Solid cancers

(35)Radiation-associated cancer risk models were developed for ten categories: nine organs or tissues (oesophagus, stomach, colon, liver, lung, female breast, ovary, bladder, thyroid), and a set of other solid cancers (Table 3.1) using data from the analyses of solid cancer incidence risk of the atomic bomb survivor Life Span Study (LSS) published in 2007 (Preston et al., 2007). These models considered cancer incidence data, with a follow-up from 1958 through 1998. Risk estimates were adjusted to reduce the bias in risk estimates arising from uncertainty in individual dose estimates derived from the dosimetry system 2002 (DS02). No specific risk models were derived for brain and salivary glands.

(36)Risk models involved a linear dose response allowing for modifying effects of sex, age at exposure, and attained age. These effects were constrained to equal the values obtained for all solid cancers as a group unless there were indications that these constraints resulted in a marked reduction in the goodness of fit when modelling cause-specific cancer types. Either the excess relative risk (ERR) or excess absolute risk (EAR) was modelled.

(37)The model equation was as follows:

$$Excess\ Risk = \beta . d . exp\left[\alpha_1\left((e-30)/10\right) + \alpha_2 ln(a/70)\right]$$

where d = dose (Gy)[2], e = age at exposure (years) and a = attained age (years). Risk coefficients used for radiation detriment calculation are summarised in Tables A.4.6 and A.4.7 in *Publication 103* (ICRP, 2007). See parameter values by sex, for ERR-based (Table 3.2) and EAR-based models (Table 3.3). The ERR/Gy and EAR/10^4 person-years/Gy for all solid

[2] The dose in Gy is intended to represent that of low LET radiations since DS02 organ dose estimates in the reference (Preston et al., 2007) were calculated as the sum of the γ-ray dose plus 10 times the neutron dose to allow for the greater biological effectiveness of neutron doses.

cancers are illustrated in both sexes by age at exposure and attained age in Figs. 3.6 and 3.7, respectively.

(38)The minimum latency period is the shortest time in which a specified radiation-induced tumour is known or believed to occur after exposure. The minimum latency period used for solid cancers in *Publication 103* was five years.

Table 3.1. Risk models used for each organ/tissue category (ICRP, 2007).

Organ/tissue	Source	Dose-risk relationship[g]	Risk transfer model[h]
Oesophagus	LSS incidence[c]	L	50%ERR:50%EAR
Stomach	LSS incidence[c]	L	50%ERR:50%EAR
Colon	LSS incidence[c]	L	50%ERR:50%EAR
Liver	LSS incidence[c]	L	50%ERR:50%EAR
Lung	LSS incidence[c]	L	30%ERR:70%EAR
Bone	Nominal risk of ICRP 60[d]	L	50%ERR:50%EAR
Skin[a]	Nominal risk of ICRP 59[e]	L	100%ERR
Breast	LSS incidence[c]	L	100%EAR
Ovary	LSS incidence[c]	L	50%ERR:50%EAR
Bladder	LSS incidence[c]	L	50%ERR:50%EAR
Thyroid	LSS incidence[c]	L	100%ERR
Bone marrow	LSS incidence[f]	LQ	50%ERR:50%EAR
Other solid[b]	LSS incidence[c]	L	50%ERR:50%EAR
Gonads (heritable)	–	–	–
Brain	–	–	–
Salivary glands	–	–	–

[a] Non-melanoma skin cancers.
[b] Remainder tissues (14 in total): adrenals, extra-thoracic region, gall bladder, heart, kidneys, lymphatic nodes, muscle, oral mucosa, pancreas, prostate, small intestine, spleen, thymus, uterus/cervix.
[c] LSS, incidence, 1958–1998, DS02 (Preston et al., 2007).
[d] Mortality (ICRP, 1991).
[e] Mortality (ICRP, 1992).
[f] LSS, incidence, 1950–1998, DS02 (a special analysis of leukaemia data, unpublished).
[g] L: linear; LQ: linear-quadratic.
[h] EAR: excess absolute risk; ERR: excess relative risk; see Section 3.1.4 for details on the transfer of risk estimates across populations.

Table 3.2. Coefficients of the ERR-based models for solid cancers incidence (from Table A.4.6, *Publication 103* (ICRP, 2007)).

Cancer site	Sex	ERR per Gy at age 70 for exposure at age 30 (β [a])	Age at exposure: % change in ERR per decade increase (x [b])	Power of attained age by which the ERR varies (α_2 [a])
All Solid	M F	0.35 0.58	–17%	–1.65
Oesophagus	M F	0.40 0.65	–17%	–1.65
Stomach	M F	0.23 0.38	–17%	–1.65
Colon	M F	0.68 0.33	–17%	–1.65
Liver	M F	0.25 0.40	–17%	–1.65
Lung	M F	0.29 1.36	+17%	–1.65
Breast	F	0.87	0%	2.26
Ovary	F	0.32	–17%	–1.65
Bladder	M F	0.67 1.10	–17%	–1.65
Thyroid	M F	0.53 1.05	–56%	0.00
Other	M F	0.22 0.17	–34%	–1.65

[a] β and α_2 are the parameters in the model equation of excess risk.

[b] $\alpha_1 = \ln(1 + x)$, where α_1 is the parameter in the model equation of excess risk.

574 Table 3.3. Coefficients of the EAR-based models for solid cancers incidence (from Table A.4.7,
575 *Publication 103* (ICRP, 2007)).

Cancer site	Sex	Excess cases per 10,000 persons per year per Gy at age 70 for exposure at age 30 (β[a])	Age at exposure: % change in EAR per decade increase (x[b])	Power of attained age by which the EAR varies (α_2[a])
All Solid	M F	43.20 59.83	–24%	2.38
Oesophagus	M F	0.48 0.66	64%	2.38
Stomach	M F	6.63 9.18	–24%	2.38
Colon	M F	5.76 2.40	–24%	2.38
Liver	M F	4.18 1.30	–24%	2.38
Lung	M F	6.47 8.97	1%	4.25
Breast	F	10.9	–39%	3.5[c] 1.0
Ovary	F	1.47	–24%	2.38
Bladder	M F	2.00 2.77	–11%	6.39
Thyroid	M F	0.69 2.33	–24%	0.01
Other	M F	7.55 10.45	–24%	2.38

576 [a] β and α_2 are the parameters in the model equation of excess risk.
577 [b] $\alpha_1 = \ln(1 + x)$, where α_1 is the parameter in the model equation of excess risk.
578 [c] The upper value represents the age effect before age 50 years and the lower is for age greater than 50.
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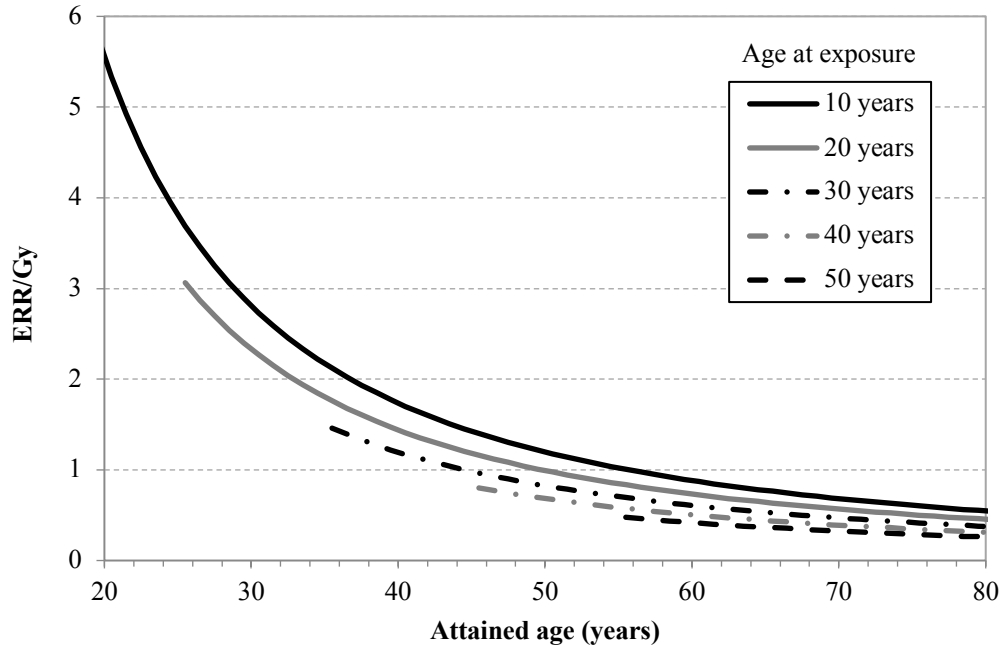

580

581 Fig. 3.6. Modification of the ERR for all solid cancers by age at exposure and attained age.

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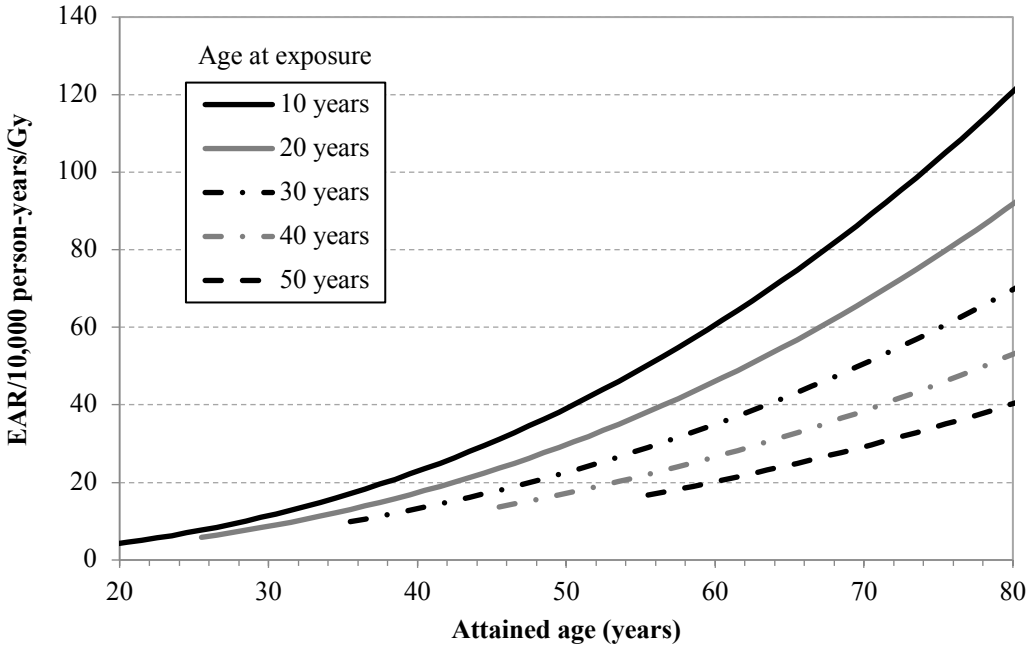


583

584 Fig. 3.7. Modification of the EAR for all solid cancers by age at exposure and attained age.

585

586 *3.1.2.2. Leukaemia*

587 (39)Leukaemia risk estimates were based on LSS incidence data, with a follow-up from 1950
588 to 1998, using the DS02 dosimetry system. The EAR-based model was similar to that derived
589 from the LSS in 1994 (Preston et al., 1994), with a linear-quadratic dose response that allows
590 for effect modification by sex, exposure age, and time following exposure. The ERR estimates

were computed from the LSS leukaemia EAR-based model and from the LSS leukaemia background rate, taking into account sex, age at exposure and attained age. However, the equations of the EAR-based and ERR-based models for leukaemia were not available.

(40)The minimum latency period used for leukaemia in *Publication 103* (ICRP, 2007) was two years.

3.1.2.3. Bone cancer

(41)The nominal risk estimate was taken from *Publication 60* (ICRP, 1991) because there was no LSS data available to derive a specific risk model, and other data sources were extremely limited. The same nominal risk was applied to both males and females. It should be noted that the ICRP risk estimate for bone cancer was based on average bone dose from radium-224 while dosimetric models estimated doses to bone surfaces, using a radiation quality factor of 20. In *Publication 103* (ICRP, 2007), the risk estimate based on the average bone dose was used although its possible conservatism was recognised.

3.1.2.4. Skin cancer

(42)For non-melanoma skin cancer risks, it was judged that LSS derived models may not be adequate for a general population because of differences in risk related to skin pigmentation. Therefore, the Commission used the nominal skin cancer risk estimate from *Publication 59* (ICRP, 1992). The same nominal risk was applied to both males and females. This estimate was also used in *Publication 60* (ICRP, 1991). In *Publication 59*, the risks have been estimated using an absolute and a constant relative risk model (with no modifying effects of age or time since exposure), using both mortality and incidence data, based on epidemiological and experimental results published up to 1990 (ICRP, 1992).

3.1.3. Lifetime excess risk

3.1.3.1. Method of calculation

(43)Several types of lifetime risk estimates can be used to calculate the risk, over a lifetime, for an individual to develop, or die from, a specific disease. The lifetime risk used in *Publication 103* (ICRP, 2007) for the radiation detriment calculation is the risk of exposure-induced cancer incidence (REIC).

(44)The REIC cumulates the excess cases over the background rate of the unexposed individuals. When exposed to dose d at age e, it is expressed in the formula:

$$REIC_c(e,d) = \int_{e+L}^{a_{max}} [\mu_{ic}(a|e,d) - \mu_{ic}(a)] S(a|e,d)\,da$$

where $\mu_{ic}(a|e,d)$ and $\mu_{ic}(a)$ denote incidence rates for a specific cancer c at age a with and without exposure, respectively. L is a minimum latency period, and $S(a|e,d)$ is the cancer-free survival probability. In *Publication 103*, a_{max} was set to 90 years, and REICs were calculated for ten solid cancer sites and leukaemia.

(45)The incidence rate for specific cancer after the exposure is calculated as:

$$\mu_{ic}(a|e,d) = \mu_{ic}(a) \times [1 + ERR_{ic}(a|e,d)]$$

or

$$\mu_{ic}(a|e,d) = \mu_{ic}(a) + EAR_{ic}(a|e,d)$$

where $ERR_{ic}(a|e,d)$ and $EAR_{ic}(a|e,d)$ are the excess relative risk and the excess absolute risk of the specific cancer.

(46)Using the Kaplan-Meier method, the cancer-free survival probability can be calculated as:

$$S(a|e,d) = \prod_{n=e}^{a}[1 - \mu(n|e,d)]$$

where $\mu(n|e,d)$ denotes the rate of developing any type of cancer or dying from causes other than cancer at age n. It can be described as:

$$\mu(n|e,d) = \mu(n) - \mu_{mac}(n) + \mu_{iac}(n|e,d)$$

where $\mu(n)$ and $\mu_{mac}(n)$ are the all-cause mortality and the all-cancer mortality, respectively, at age n in the unexposed. $\mu_{iac}(n|e,d)$ is the all-cancer incidence at age n after exposed to dose d at age e, which is calculated as:

$$\mu_{iac}(n|e,d) = \mu_{iac}(n) \times [1 + ERR_{iac}(n|e,d)]$$

or

$$\mu_{iac}(n|e,d) = \mu_{iac}(n) + EAR_{iac}(n|e,d)$$

where $ERR_{iac}(n|e,d)$ and $EAR_{iac}(n|e,d)$ are the excess relative risk and the excess absolute risk of the all types of cancer.

(47)The risk models and survival function described above were used to compute sex-specific lifetime risk estimates for the Asian and Euro-American composite populations. For each solid cancer site and for leukaemia, the considered exposure scenario was acute exposure to 0.1 Gy. REIC at 1 Gy was computed as the REIC at 0.1 Gy multiplied by 10.

(48)Two nominal populations were considered: the whole population (age at exposure 0 to 84 years) and adult workers (age at exposure 18 to 64 years). REIC was calculated for each age at exposure by cumulating the risk until the attained age reaches 90 years, as in *Publication 60* (ICRP, 1991). This means that the risk was cumulated over an age range 0–89 years (maximum 90 years) for the whole population, and 18–89 years (maximum 72 years) for adult workers.

(49)For the calculation of leukaemia lifetime risk, the risk models derived from the LSS considered all leukaemia (including CLL), whereas the baseline reference rates from Asian and Euro-American populations considered non-CLL leukaemia. This difference has little impact as CLL cases are very rare in Japan. Nevertheless, as the equations of the EAR-based and ERR-based models were not available for leukaemia, calculations of lifetime risk of leukaemia are not presented in the rest of this report.

3.1.3.2. Age-dependence of lifetime excess risk

(50)Figs 3.8 and 3.10 show the excess risk of solid cancers, cumulated up to a given attained age, in Euro-American females with a single exposure to 1 Gy at different ages at exposure (0, 20 and 40 years), using an ERR-based and EAR-based model, respectively. Figs 3.9 and 3.11 show lifetime excess risk of solid cancers (up to the age of 89) in the general population with a single exposure to 1 Gy, using an ERR-based and EAR-based model, respectively.

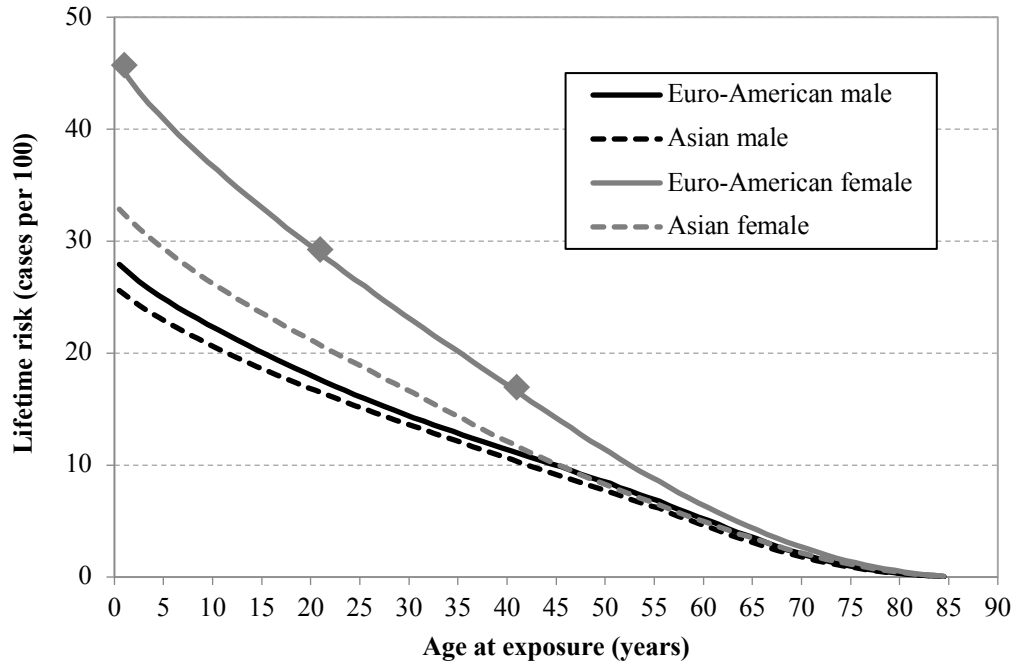
(51)Figs 3.8–3.11 illustrate the change of the cumulative excess risk with respect to the attained age and age at exposure. No DDREF was applied at this step of calculation. The data points shown by diamonds in Fig. 3.8 for a radiation exposure to 1 Gy at 0, 20 or 40 years of age in Euro-American females correspond to those in Fig. 3.9. Similarly, the data points marked by circles in Fig. 3.10 for a radiation exposure to 1 Gy at 0, 20 or 40 years of age in

Euro-American females correspond to those in Fig. 3.11. The cross markers in Figs. 3.8 and 3.10 indicate the cumulative excess risk 20 years after the exposure.

(52)Figs 3.8 and 3.10 show that the cumulative excess risk increases gradually from 5 years after exposure (reflecting the minimum latency period of 5 years) up to the age of 89 years. This increase is due to the increase in the cumulative baseline risk. It should also be noted that the cumulative excess risk 20 years after exposure (represented by the crosses) is slightly higher for exposure at the age of 40 years than at the age of 20 years and at the age of 0 year. This is the result of the counter-balancing effects between the increase in the cumulative baseline risk with attained age and the decrease in the risk coefficient with attained age for an ERR-based model (Fig. 3.6), or with age at exposure for an EAR-based model (Fig. 3.7). The cumulative excess risk at age 89 years is lower for exposure at age 40 years than at age 20 and 0 years; this is due to the shorter remaining duration of life for older ages at exposure.

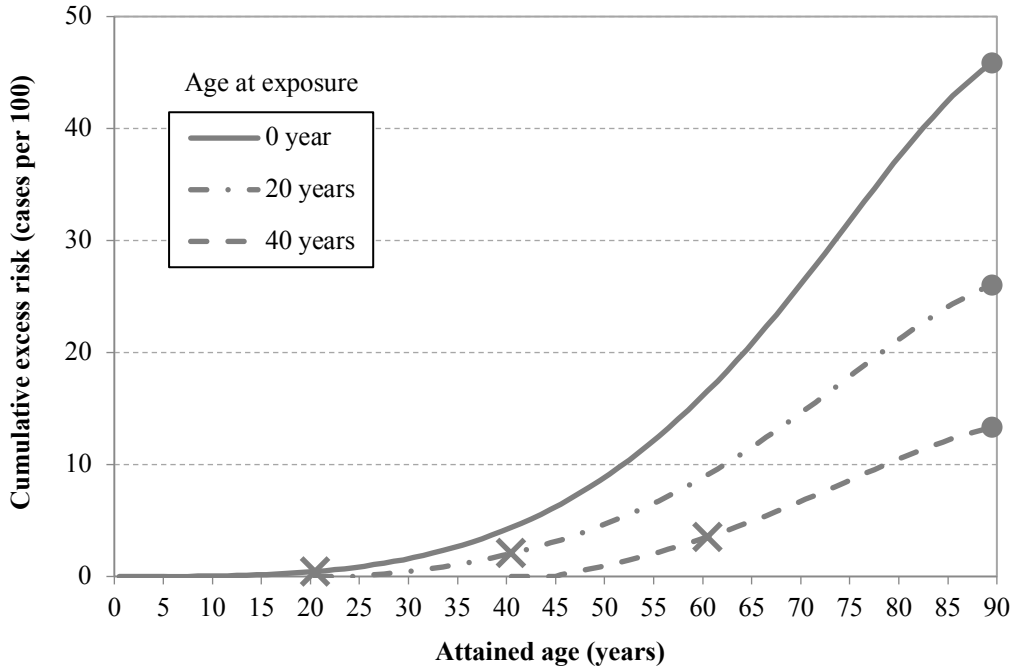
(53)Figs 3.9 and 3.11 show that the lifetime excess risk decreases gradually with age at exposure from birth to the age of 85. This decrease is mainly due to the reduction of remaining duration of life with increasing age at exposure, and also partly due to the decrease in the risk coefficient with age at exposure. For age at exposure 85 years or more, the lifetime excess risk is zero (due to the minimum latency period of 5 years). These figures also show the difference between sexes and geographical regions. The lifetime excess risk is higher among females than among males. Using an ERR-based model, the lifetime excess risk is higher in the Euro-American population than in the Asian population (Fig. 3.9), whereas such difference is not apparent when the EAR-based model was used (Fig. 3.11). Nevertheless, the decrease of the lifetime excess risk with age at exposure is similar in all populations.

Fig. 3.8. Cumulative excess risk at 1 Gy for all solid cancers in Euro-American females by age at exposure, using an ERR-based model. The data points shown by diamonds correspond to those in Fig. 3.9. The cross markers indicate the cumulative excess risk 20 years after the exposure.



699

700 Fig. 3.9. Lifetime excess risk for all solid cancers after exposure to 1 Gy, using an ERR-based model.
701 The data points shown by diamonds correspond to those in Fig. 3.8.
702



703

704 Fig. 3.10. Cumulative excess risk at 1 Gy for all solid cancers in Euro-American females by age at
705 exposure, using an EAR-based model. The data points marked by circles correspond to those in Fig.
706 3.11. The cross markers indicate the cumulative excess risk 20 years after the exposure.
707

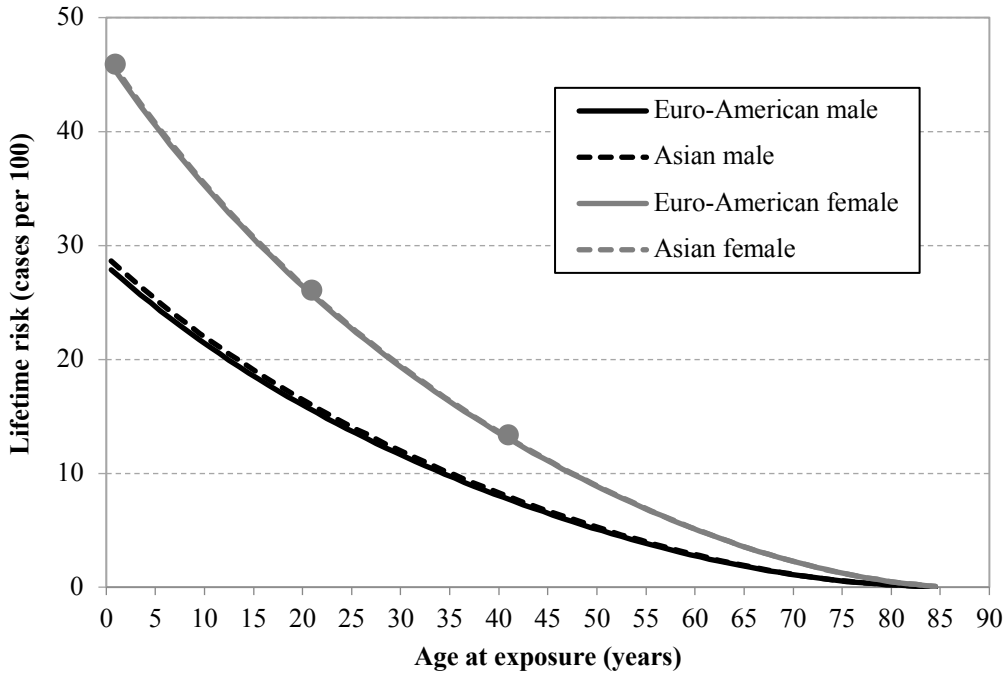


Fig. 3.11. Lifetime excess risk for all solid cancers after exposure to 1 Gy, using an EAR-based model. The data points marked by circles correspond to those in Fig. 3.10.

3.1.3.3. Averaging lifetime excess risk

(54)The age-averaged lifetime excess risk was calculated as a weighted mean of REIC for overall ages at exposure. The weight was assigned in proportion to the population of each age group in the reference population as shown in Fig. 3.12, which illustrates the population distribution by 5-year age categories for Asian and Euro-American populations.

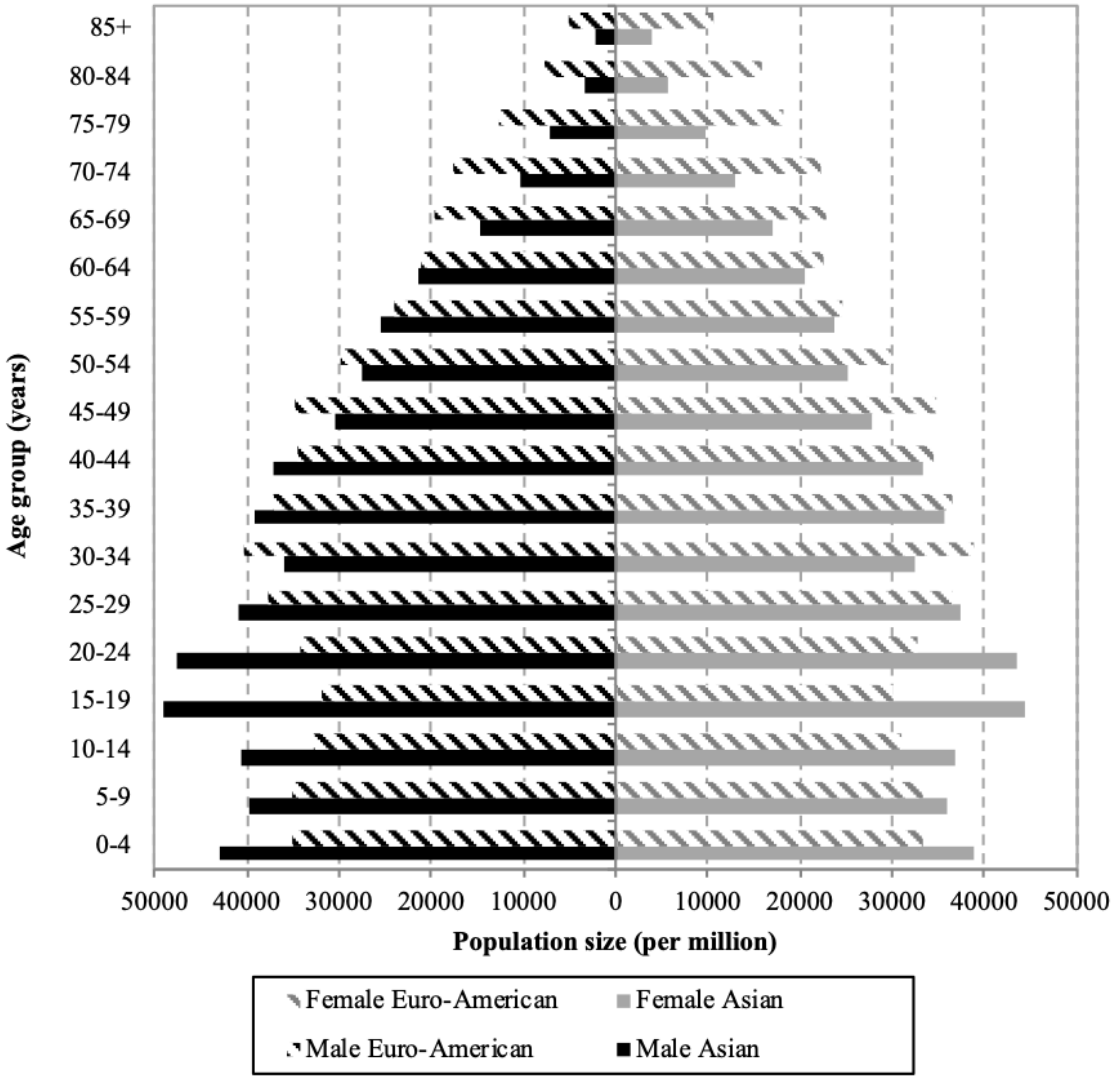


Fig. 3.12. Euro-American and Asian population size by age group.

(55)Table 3.4 summarises the averaged lifetime excess risk for solid cancers by site calculated for the general population (0–89 years of age) with ERR-based and EAR-based models, in Euro-American and Asian populations. They were calculated as an unweighted mean of the lifetime excess risks for both sexes, each of which was the weighted mean of REICs for ages at exposure of 0 to 84 years.

727 Table 3.4. Sex- and age-averaged lifetime excess risk for the whole population.

Cancer	Cases per 100 per Gy using an ERR-based model		Cases per 100 per Gy using an EAR-based model	
	Euro-American	*Asian*	*Euro-American*	*Asian*
Oesophagus	0.24	0.43	0.22	0.20
Stomach	0.27	1.61	2.08	2.38
Colon	1.34	1.73	1.01	1.20
Liver	0.11	0.90	0.67	0.80
Lung	3.05	2.60	1.77	1.84
Breast	–	–	2.04	2.45
Ovary	0.28	0.14	0.20	0.23
Bladder	1.34	0.66	0.60	0.63
Thyroid	0.49	0.73	–	–
Other solid	3.95	2.29	2.36	2.71
All solid	14.86	13.37	13.37	15.32

728

729 ### 3.1.4. Transfer of risk estimates across populations

730 (56)It is problematic to transfer site-specific risk estimates of radiation-associated cancers
731 from one population to the other if the corresponding baseline rates differ. To address this issue,
732 the population risks were defined as weighted averages of the EAR- and ERR-based risk
733 estimates with weights based on judgements concerning the relative applicability of the two
734 risk estimates (Table 3.1). Weights of 0.5 were used for all tissues except the breast, thyroid,
735 skin and lung.
736 (57)For female breast cancer, a pooled analysis of radiation effects (Preston et al., 2002)
737 provided evidence against the use of common ERR-based models. Therefore, female breast
738 cancer risks were based solely on an EAR-based model derived from recent incidence data
739 from the LSS (Preston et al., 2007).
740 (58)For thyroid cancer, the use of EAR-based models appeared to be problematic because
741 variation in screening intensity has a marked effect on the rate of radiation-associated thyroid
742 cancers. Therefore, based on an analysis of radiation-associated thyroid cancer risks (Ron et
743 al., 1995) and on the most recent available results from the LSS (Preston et al., 2007), thyroid
744 cancer risks were based solely on an ERR-based model. The same weighting scheme was
745 applied to skin cancer as well.
746 (59)For lung cancer, the atomic bomb survivor data suggested that the EAR was more
747 comparable across sexes than the ERR, and also that radiation dose and smoking history
748 interacted additively as lung cancer risk factors (Pierce et al., 2003). Consequently, the ERR-
749 based model was given a weight of 0.3 and the EAR-based model a weight of 0.7.
750 (60)For leukaemia, transfer to other populations was done using both EAR and ERR
751 estimates. The detriment computations used an average (50:50%) of the EAR and ERR transfer
752 risk estimates (a 100% EAR transfer was erroneously indicated in *Publication 103* (ICRP,
753 2007)). Nevertheless, as the equations of the EAR-based and ERR-based models were not
754 available for leukaemia, calculations of lifetime risks of leukaemia are not presented in the rest
755 of this report.

(61)In summary, ERR:EAR weights of 0:100% were assigned for breast, 100:0% for thyroid and skin, 30:70% for lung, and 50:50% for all others including leukaemia (Table 3.1).

3.1.5. Application of DDREF

(62)Experimental studies show that biological effectiveness of radiation exposure at low doses and low dose rates is usually lower compared with exposures at high doses and high dose rates, suggesting that dose-specific estimates based on high-dose, acute exposure data should be divided by a DDREF for applications to low-dose, continuous, or fractionated exposures. Recognising uncertainties, the Commission recommended in *Publication 103* (ICRP, 2007) that a DDREF of 2 continued to be used for radiological protection purposes. The Commission stressed that its recommendation was a broad judgement including elements of both subjectivity and probabilistic uncertainty.

(63)The lifetime risk estimates were adjusted downward by a factor of 2 to account for a DDREF, except for leukaemia for which the linear-quadratic dose-response model already takes into account the risk modification at low doses. The same DDREF applied to males and females, the whole population and adult workers.

(64)The DDREF applies specifically to doses below 0.2 Gy or dose rates less than 0.1 Gy per hour (ICRP, 1991). This means the radiation detriment assumes low-dose and/or low-dose-rate exposures.

3.1.6. Integration of heritable effects

(65)To estimate the risk of heritable effects, the relative importance of genetic components as well as the frequency of transmissible mutations needs to be taken into account. The UNSCEAR 2001 Report provided risks expressed as the predicted number of additional cases (i.e. over the baseline) of different classes of genetic disease per million live births per Gy for a population exposed to low-LET, low-dose or chronic irradiation, generation after generation (UNSCEAR, 2001). For all classes except congenital abnormalities, the estimates were based on a doubling dose (DD) of 1 Gy and the respective values of baseline frequency, mutation component and potential recoverability correction factor for the different classes of genetic diseases. For congenital abnormalities, the risk estimate came from mouse data and was not based on the DD method.

(66)On the basis of UNSCEAR (2001), the Commission derived ICRP estimates of risks for all classes of genetic diseases: Mendelian diseases, chronic diseases and congenital abnormalities (Tables A.6.4 and A.6.6, *Publication 103* (ICRP, 2007)). While based on the state of knowledge in this area, the strengths and limitations of these estimates need to be borne in mind, in view of various underlying assumptions.

(67)The Commission decided to use risk estimates for the first two generations (c.f. two generations in *Publication 26* (ICRP, 1977a) and all generations in *Publication 60* (ICRP, 1991)). The risk of heritable effects in the whole population associated with gonadal dose was estimated to be around 20 cases per 10,000 people per Gy. The risk for adult workers was estimated to be 60% of that for the whole population, leading to an estimated nominal risk of 12 per 10,000 per Gy. These values were applied to both males and females.

3.1.7. Nominal risk coefficient

(68)Following the steps mentioned above, the nominal risk coefficient was computed for 14 organs or tissues, which include 12 cancer sites (oesophagus, stomach, colon, liver, lung, bone,

skin, female breast, ovary, bladder, thyroid, red bone marrow), a set of the remaining cancer sites grouped into one 'remainder' category, and the gonads for heritable effects.

(69)Some radiation-related cancers are sex-specific, and for many others, sex is a major modifier of radiation-related risk. Nominal cancer risks were calculated separately for males and females, and for the whole population and for adult workers (Tables A.4.18 and A.4.19, *Publication 103* (ICRP, 2007)).

(70)In accordance with ICRP procedures, intermediate and final numerical risk estimates have been sex-averaged as an unweighted mean between male and female estimates. For ovaries, the average was calculated considering that lifetime risk among males was zero. For breasts, the average was calculated given that lifetime risk among males was zero. This assumption was made because of the rare occurrence of male breast cancer.[3] Sex-average nominal cancer risks for the whole population and for adult workers are presented in Table 3.5.

[3] Although a recent analysis of the LSS data (Brenner et al., 2018) suggested a significant positive dose response for male breast cancer, this assumption continues to be valid considering the very small number of cases.

Table 3.5. Nominal risk coefficients in *Publication 103* (ICRP, 2007): by sex for the general population and for workers (from Tables A.4.1, A4.18 and A4.19).

Organ/tissue	Nominal risk coefficient (R[a])		
	Men	*Women*	*Both sexes*
Whole population (age 0–84 years at exposure)			
Oesophagus	15	16	15
Stomach	68	91	79
Colon	91	40	65
Liver	41	19	30
Lung	76	153	114
Bone (surface)	7	7	7
Skin[b]	1000	1000	1000
Breast	0	224	112
Ovary	0	21	11
Bladder	46	41	43
Thyroid	12	53	33
Bone marrow[c]	48	36	42
Other solid[d]	157	131	144
Gonads (heritable)	20	20	20
Total	*1580*	*1851*	*1715*
Adult workers (age 18–64 years at exposure)			
Oesophagus	14	16	16
Stomach	51	70	60
Colon	73	33	50
Liver	31	16	21
Lung	84	174	127
Bone (surface)	5	5	5
Skin[b]	670	670	670
Breast	0	116	49
Ovary	0	16	7
Bladder	40	39	42
Thyroid	4	20	9
Bone marrow[c]	24	22	23
Other solid[d]	94	88	88
Gonads (heritable)	12	12	12
Total	*1103*	*1297* [e]	*1179*

[a] R is expressed in cases per 10,000 persons per Gy.
[b] Non-melanoma skin cancers.
[c] Non-CLL leukaemia.
[d] Remainder tissues (14 in total): adrenals, extra-thoracic region, gall bladder, heart, kidneys, lymphatic nodes, muscle, oral mucosa, pancreas, prostate, small intestine, spleen, thymus, uterus/cervix.
[e] This value corresponds to the sum of the above lines and is slightly different from that (1242) in ICRP *Publication 103* (Table A.4.19).

3.2. Severity adjustment

(71)Table 3.6 summarises the parameters for severity adjustment by which the nominal risk was converted into the radiation detriment.

3.2.1. Adjustment for lethality

(72)Since the nominal risk coefficient was calculated based on the excess incidence, the lethality fraction (k) was applied to take account of cancer severity.

(73)Lethality fractions were derived as judgement-based values reflecting the impact of medical treatment for some types of cancer. In *Publication 60* (ICRP, 1991), the choice of the values was based on the analysis of two sets of data from the US SEER programme: 5-year survival rates by cancer site for 1980–1985 and 20-year survival rates for 1950–1970 (U.S. DHHS, 1989). They were updated in *Publication 103*（ICRP, 2007), but remained close to the previous values. The same set of values were applied to males and females, the whole population and adult workers.

(74)The lethality adjustment was performed by multiplying the nominal risk coefficient R by the factor k. Highly lethal cancers received a relatively greater weight (e.g. 0.95 for liver cancer, 0.89 for lung cancer) than those that seldom cause death (e.g. 0.002 for skin cancer, 0.07 for thyroid cancer) (Table 3.6).

Table 3.6. Construction of radiation detriment in *Publication 103* (ICRP, 2007): from nominal risk coefficient to radiation detriment for the whole population and for adult workers (from Tables A.4.1 and A.4.5).

Organ/tissue	Nominal risk coefficient R^*	Lethality fraction k	Min weight for non-fatal cancers q_{min}	Non-fatal case weight q	Relative cancer free life lost l	Radiation detriment D^*	Relative radiation detriment
Whole population (age 0–84 years at exposure)							
Oesophagus	15	0.93	0.1	0.935	0.87	13.1	0.023
Stomach	79	0.83	0.1	0.846	0.88	67.7	0.118
Colon	65	0.48	0.1	0.530	0.97	47.9	0.083
Liver	30	0.95	0.1	0.959	0.88	26.6	0.046
Lung	114	0.89	0.1	0.901	0.80	90.3	0.157
Bone	7	0.45	0.1	0.505	1.00	5.1	0.009
Skin[c]	1000	0.002	0.0	0.002	1.00	4.0	0.007
Breast	112	0.29	0.1	0.365	1.29	79.8	0.139
Ovary	11	0.57	0.1	0.609	1.12	9.9	0.017
Bladder	43	0.29	0.1	0.357	0.71	16.7	0.029
Thyroid	33	0.07	0.2	0.253	1.29	12.7	0.022
Bone marrow[d]	42	0.67	0.1	0.702	1.63	61.5	0.107
Other solid[e]	144	0.49	0.1	0.541	1.03	113.5	0.198
Gonads (heritable)	20	0.80	0.1	0.820	1.32	25.4	0.044
Total	1715					574	1.000
Adult workers (age 18–64 years at exposure)							
Oesophagus	16	0.93	0.1	0.935	0.91	14.2	0.034
Stomach	60	0.83	0.1	0.846	0.89	51.8	0.123
Colon	50	0.48	0.1	0.530	1.13	43.0	0.102
Liver	21	0.95	0.1	0.959	0.93	19.7	0.047
Lung	127	0.89	0.1	0.901	0.96	120.7	0.286
Bone	5	0.45	0.1	0.505	1.00	3.4	0.008
Skin[c]	670	0.002	0.0	0.002	1.00	2.7	0.006
Breast	49	0.29	0.1	0.365	1.20	32.6	0.077
Ovary	7	0.57	0.1	0.609	1.16	6.6	0.016
Bladder	42	0.29	0.1	0.357	0.85	19.3	0.046
Thyroid	9	0.07	0.2	0.253	1.19	3.4	0.008
Bone marrow[d]	23	0.67	0.1	0.702	1.17	23.9	0.057
Other solid[e]	88	0.49	0.1	0.541	0.97	65.4	0.155
Gonads (heritable)	12	0.80	0.1	0.820	1.32	15.3	0.036
Total	1179					422	1.000

[*] R and D are expressed in cases per 10,000 persons per Gy and Sv, respectively.

$q = k + q_{min} \times (1 - k)$ $\quad D = [(R \times k) + (R \times (1 - k) \times q)] \times l$

3.2.2. Adjustment for quality of life

(75)Cancer survivors generally experience adverse effects on their quality of life. Thus, the Commission judged that cancers should be weighted not only by lethality but also for pain, suffering, and any adverse effects of cancer treatment. To achieve this, a factor termed q_{min} was applied to the non-lethal fractions of cancers to produce a quality of life factor termed q. It is expressed in a formula $q = k + q_{min} \times (1 - k)$, where k is the lethality faction and q_{min} is a tissue-specific constant representing the minimum weight for non-lethal cancers.

(76)q_{min} is a judgment-based parameter. The value of q_{min} was set equal to 0.1 except for the skin and thyroid. The q_{min} adjustment has an impact upon radiation detriment calculations in proportion to the fraction of cancers that are non-lethal. Accordingly, highly lethal cancers such as lung and stomach cancer are little affected by q_{min} compared to less lethal cancers such as breast or thyroid.

(77)No q_{min} adjustment was used for skin cancer because radiogenic skin cancers (i.e. non-melanoma skin cancers) are almost exclusively of the basal cell type, which is usually associated with very little pain, suffering or treatment sequelae. For thyroid cancer, q_{min} was set to 0.2.

3.2.3. Adjustment for years of life lost

(78)To take into consideration the difference in the distribution of age at diagnosis among cancer sites, the loss of life expectancy (LLE) was calculated for a specific cancer c by a formula:

$$LLE_c(e,d) = \int_{e+L}^{a_{max}} S(a|e)da - \int_{e+L}^{a_{max}} S_c(a|e,d)da$$
$$= \sum_{a=e+L}^{a_{max}} S(a|e) - \sum_{a=e+L}^{a_{max}} S_c(a|e,d)$$

where the notations are the same as those in Section 3.1.3.1, and the cancer-free survival probability $S_c(a|e,d)$ allows for an alteration in the incidence of cancer c following radiation exposure. The years of life lost for cancer c was given by dividing $LLE_c(e,d)$ by $REIC_c(e,d)$, in which the effect of dose d is cancelled out.

(79)Average years of life lost were computed for each sex in each composite population as the weighted average over ages at exposure. These were converted to relative values (factor l) by division by the average years of life lost for all cancers. The average number of years of life lost for all cancers was equal to 15 years as in *Publication 60* (ICRP, 1991). The factor l reflects the relative years of cancer-free life lost, with the value of less than 1 for cancers occurring late in life (e.g. 0.71 for bladder cancer, 0.80 for lung cancer) and more than 1 for those occurring early in life (e.g. 1.63 for red bone marrow, 1.29 for thyroid or breast cancer).

(80)The years of life lost for bone and skin cancer cannot be obtained in the same way and therefore were arbitrarily set at the average years of life lost for all cancers. The value of l was therefore equal to 1 for these two cancer sites. The gonads were assigned a value of 20 years of life lost on average for severe genetic disorders, which was equivalent to l of 1.32.

3.2.4. Calculation of radiation detriment

(81)As shown in Table 3.6, the radiation detriment D for each organ or tissue was calculated by applying the above-mentioned factors to the nominal risk coefficient R using the formula:

$$D = [R \times k + R \times (1 - k) \times q] \times l$$

(82)The overall radiation detriment was calculated as an unweighted sum of the 14 tissue-specific detriments. The result is shown in terms of the number of cases per 10,000 persons per Sv. It represents not the real number, but the weighted number of excess cases per unit dose of radiation. 'Sv' is used to express the radiation dose since the radiation detriment is intended for the purpose of radiological protection at low doses and low dose rates.

3.3. Relation between radiation detriment and effective dose: tissue weighting factors w_T

(83)The relative radiation detriments for the whole population, which are the normalised radiation detriments of respective organs/tissues to sum to unity, form the basis of the tissue weighting factors w_T used for calculation of the effective dose. In *Publication 60* (ICRP, 1991), the Commission selected a very simplified system of weights, which used no more than four groups of weights and required no more than about a factor of 2 rounding between the relative radiation detriments and the assigned weights. In *Publication 103* (ICRP, 2007), the numerical values changed as shown in Table 3.7, but the basic concept remained unchanged.

Table 3.7. Tissue weighting factors used for each organ/tissue category in *Publication 103* (ICRP, 2007).

Organ/tissue	Relative radiation detriment		w_T
	Whole population	*Adult workers*	
Oesophagus	0.023	0.034	0.04
Stomach	0.118	0.123	0.12
Colon	0.083	0.102	0.12
Liver	0.046	0.047	0.04
Lung	0.157	0.286	0.12
Bone	0.009	0.008	0.01
Skin	0.007	0.006	0.01
Breast	0.139	0.077	0.12
Ovary	0.017	0.016	
Bladder	0.029	0.046	0.04
Thyroid	0.022	0.008	0.04
Bone marrow	0.107	0.057	0.12
Other solid*	0.198	0.155	0.12
Gonads (heritable)	0.044	0.036	0.08
Brain	–	–	0.01
Salivary glands	–	–	0.01
Total	1.000	1.000	1.00

* Remainder tissues (14 in total): adrenals, extra-thoracic region, gall bladder, heart, kidneys, lymphatic nodes, muscle, oral mucosa, pancreas, prostate, small intestine, spleen, thymus, uterus/cervix.

(84)The Commission has defined a single set of w_T values that is applied to both sexes and all ages. Since the detailed relative radiation detriments in Table 3.6 and 3.7 were imprecise because of uncertainties associated with their estimation, they were grouped into four categories broadly reflecting the relative detriments.

(85)For the organs with the highest radiation detriments (lung, breast, stomach, red bone marrow, colon, remainder tissues), the w_T was set to 0.12. The gonads were assigned a w_T of 0.08 based on the relative detriment for heritable effects and ovarian cancer. For the organs with intermediate radiation detriments (bladder, oesophagus, liver, thyroid), the w_T was set to 0.04. The w_T value for the thyroid was set to 0.04 to take account of the concentration of cancer risk in childhood (i.e. young children are considered to be a particularly sensitive subgroup for thyroid cancer). For the organs with the lowest radiation detriments (skin, bone), the w_T was set to 0.01. Cancer risks in salivary glands and brain, whilst not specifically quantifiable, were judged to be greater than that of other tissues in the remainder fraction and, for this reason, each was also assigned a w_T of 0.01.

(86)A group of ‘remainder tissues’ was included to account for radiation detriments to organs or tissues for which detailed radiation-risk calculations were uninformative. To make the sum of w_T equal to unity, the remaining value (0.12) was assigned to them. This category denoted as ‘other solid cancers’ or ‘remainder tissues’ includes 14 organs or tissues, and the w_T of 0.12 has to be considered as equally distributed between them.

4. SENSITIVITY OF RADIATION DETRIMENT CALCULATION

(87)Many parameters are involved in the calculation of the radiation detriment, and the variation in the values adopted for these parameters can have effects on the total detriment, which in turn could have implications on radiation protection practice. In order to examine the effects of these variations on the radiation detriment, a sensitivity analysis was conducted for a variety of parameters. The analysis focuses on solid cancers other than bone and skin cancers.

(88)To reproduce the radiation detriment calculation as similarly as possible to that in *Publication 103*, the following parameters were chosen:

- The 100% ERR-based and the 100% EAR-based models were used for thyroid cancer risk and breast cancer risk, respectively. A mixed model of 50% ERR-based and 50% EAR-based was used for the rest of solid cancer risks, except for lung cancer where a model of 30% ERR-based and 70% EAR-based was used. For solid cancers, lifetime risk was divided by a DDREF of 2 to take into account the effect from protracted radiation exposure.
- Population averaged lifetime risk with age at exposure of 0–84 years were calculated with attained age set at 89 years.
- Nominal risks were calculated at 0.1 Sv, and then were linearly extrapolated to 1 Sv through multiplication by a factor of 10.

(89)For the sensitivity analysis, the parameters were set differently from *Publication 103* as below and were changed one at a time to examine their impact on the radiation detriment.

- DDREF: 1.
- Age at exposure: 0–14, and 18–64 years.
- Sex: male and female, separately.
- Reference population: Euro-American and Asian, separately.
- Transfer model: 100% ERR and 100% EAR, separately.
- Minimum latency period for solid cancers: 10 years.
- Maximum attained age: 99 years.
- Lifetime risk calculation method: lifetime attributable risk (LAR) and excess lifetime risk (ELR).
- Lethality fraction: 1 for all cancer sites.
- Minimum quality of life factor: 0 for all cancer sites.
- Relative years of cancer-free life lost: 1 for all cancer sites.

4.1. Parameters involved in the calculation of the nominal risk

(90)In *Publication 103* (ICRP, 2007), the nominal risk for solid cancers was divided by a DDREF of 2 to take into account the possible effects of low-dose and low-dose-rate exposures of the general population and workforce. However, the value of DDREF has become a topic of discussion in recent years within the radiological protection community (Rühm et al., 2015, 2016; Shore et al., 2017). The National Academy of Sciences-National Research Council

proposed a DDREF value of about 1.5 in BEIR VII (NRC, 2006), and some even consider that a DDREF of 1 should be used (SSK, 2014). The radiation detriment calculated with a DDREF of 1 and 2 are presented in Fig. 4.1. As the radiation detriment is inversely proportional to the DDREF, this leads to a difference of a factor of two for all solid cancers.

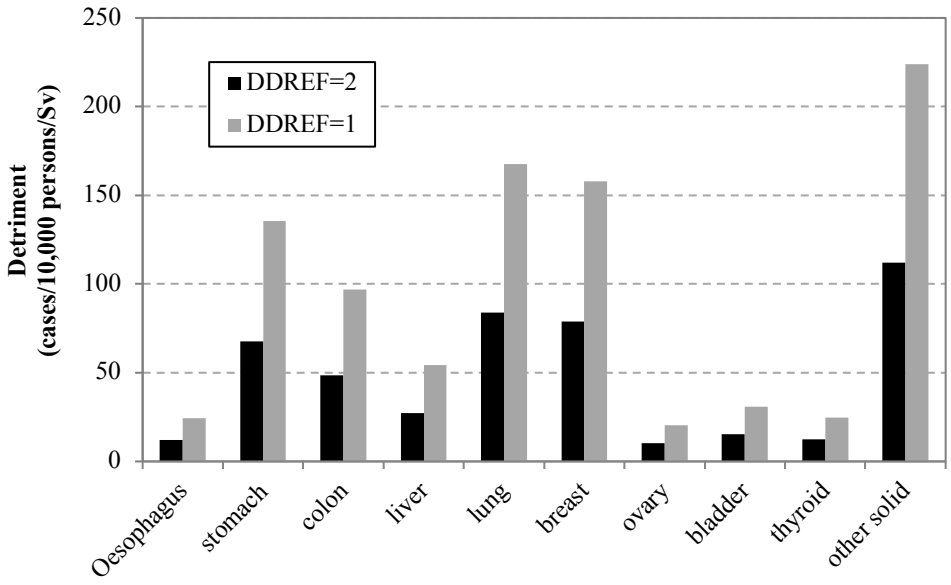


Fig. 4.1. Results of cancer detriment for DDREF values of 1 and 2. Taken from Zhang et al. (2020).

(91)The nominal risk was averaged over age-at-exposure 0–84 years in *Publication 103* (ICRP, 2007). Fig. 4.2. shows the radiation detriment for different groups of age at exposure. Comparisons are made between three groups of age at exposure: 0–14 years, 18–64 years and 0–84 years which represent the young age population, working age population and whole population, respectively. For most cancer sites, the detriment for the young ages-at-exposure population (0–14 years) is higher than that for a whole population averaged (0–84 years). In some cases (i.e. stomach cancer, breast cancer, thyroid cancer, and other solid cancer), the detriment for 0–14 years is more than double compared with that for 0–84 years.

(92)The radiation detriment was also averaged over sexes and two composite populations which were derived from four Asian and three Euro-American populations in *Publication 103* (ICRP, 2007). This methodology applied to all cancer sites, despite the fact that some cancer incidences are higher in one population than others and some cancers are sex-specific, such as ovary cancer and female breast cancer. Fig. 4.3 shows radiation detriment averaged over 0–84 years of age at exposure for the sexes separately, and Fig. 4.4 shows the radiation detriment for the composite Euro-American and Asian populations separately. For lung cancer, the detriment for females appears to be higher than for males. For stomach and liver cancers, the detriment for the Asian population appears to be higher than for the Euro-American population. For breast and ovary cancers, the differences in radiation detriments between the populations are relatively small, but using the male detriment of zero reduces the overall detriment by 50%. For other solid cancers, the detriment for the Euro-American male is somewhat higher than that for other population groups.

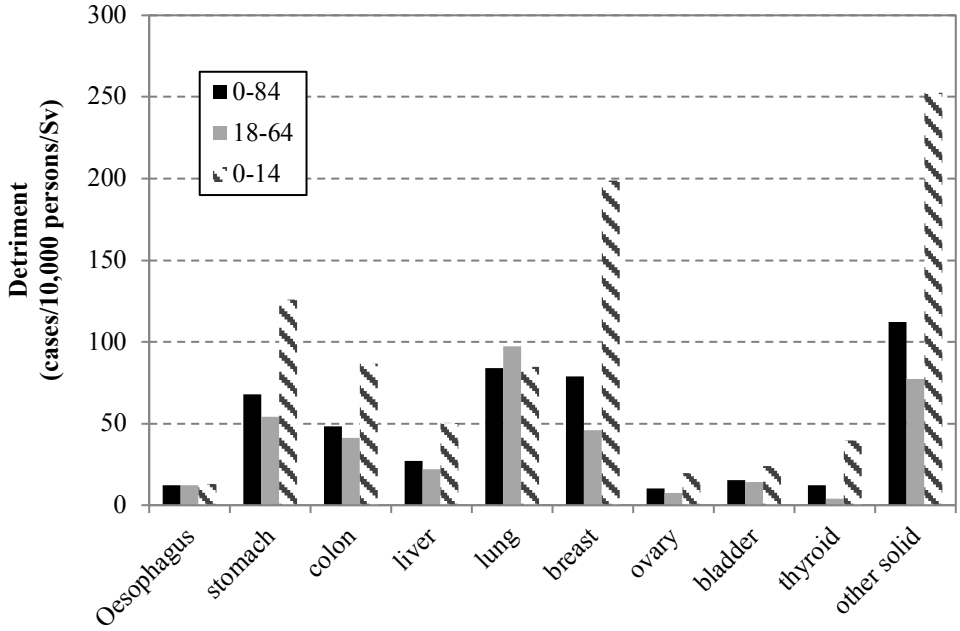


Fig. 4.2. Cancer detriment for different groups of age at exposure. Taken from Zhang et al. (2020).

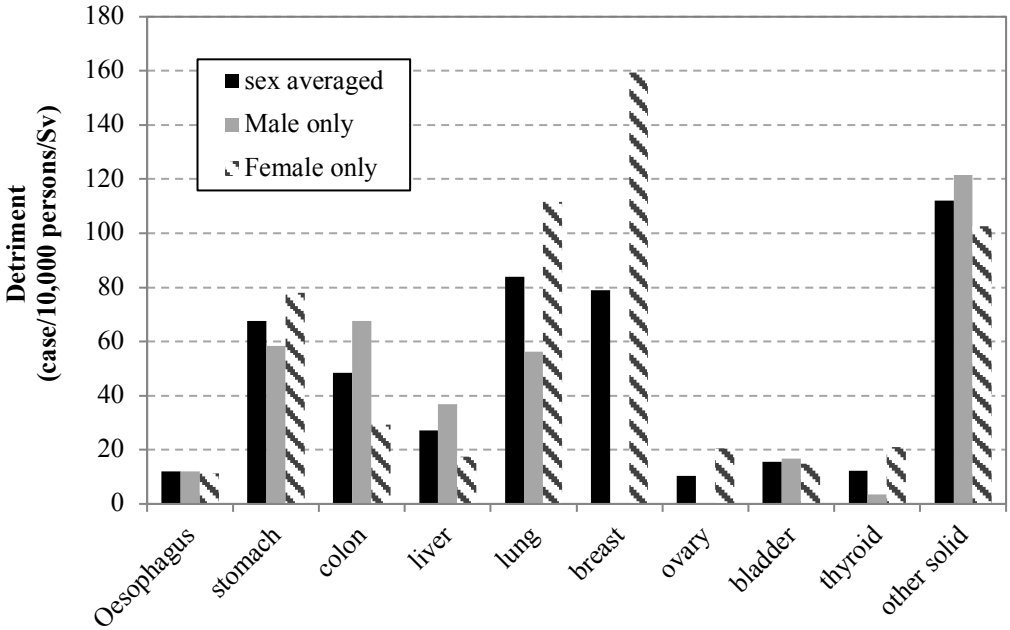


Fig. 4.3. Cancer detriment calculated for males and females separately, compared with sex averaged values. Taken from Zhang et al. (2020).

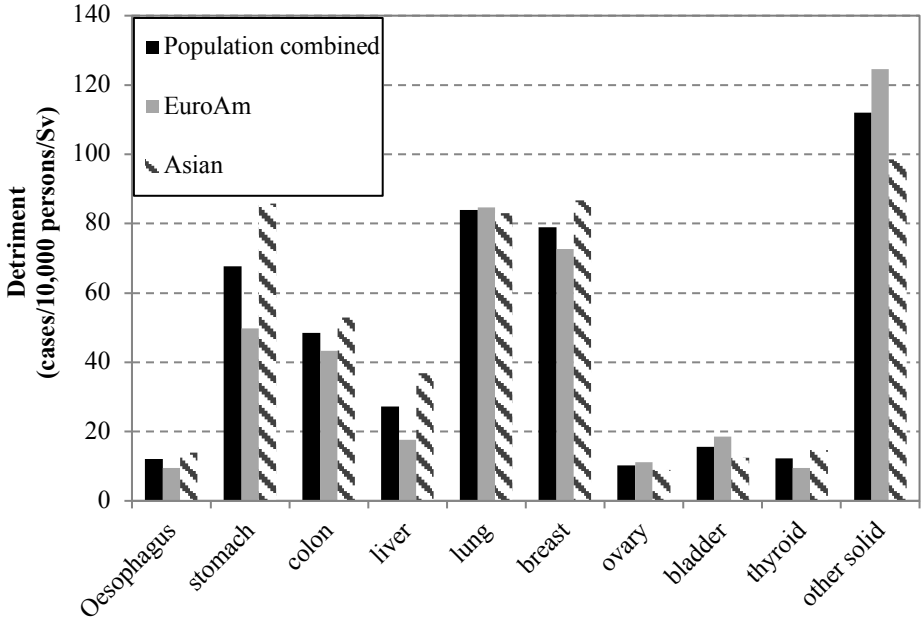


Fig. 4.4. Comparison of cancer detriment calculated for Euro-American and Asian populations. Taken from Zhang et al. (2020).

(93)In *Publication 103* (ICRP, 2007), the nominal risks were derived based on the 100% ERR-based model for thyroid cancer, the 100% EAR-based model for breast cancer, the 30% ERR-based + 70% EAR-based models for lung cancer, and the 50% ERR-based + 50% EAR-based models for the rest of solid cancer sites (Table 3.1). This weighting scheme is related to the different cancer baseline rates between populations and it would be problematic to transfer radiation risk from one population to another using either the ERR-based or the EAR-based model uniformly across all cancer sites. Therefore, the contribution to nominal risk from the two models varies depending on the cancer sites (see Section 3.1.4). Fig. 4.5 shows the radiation detriment calculated based on the 100% ERR-based and the 100% EAR-based models for sex and ages-at-exposure averaged population in comparison with that derived using the method described in *Publication 103*. As shown in Fig. 4.5, the contribution from the ERR-based model is relatively small for stomach cancer, liver cancer and relatively large for lung cancer, bladder cancer and other solid cancer.

(94)There is a minimum time required between induction of a cancer and its detection. This latent period is expected to differ with cancer site, but information is limited to only a few cancers. There are uncertainties associated with this parameter as it can depend on the diagnostic techniques available. The minimum latency period is considered to be 5–10 years for solid cancer. Fig. 4.6 shows the comparison of radiation detriments using a different latency period. The minimum latencies of 5 and 10 years produce little difference.

(95)As life expectancy increases, the cumulative lifetime radiation risk is also increasing, and this results in predicted additional deaths from radiation exposure. In this detriment calculation, the maximum attained age to 99 years instead of 89 years was used to examine its impact on the radiation detriment calculation. Fig. 4.7 shows the comparison of cancer detriments for the two different attained ages. The increase in radiation detriment with the maximum attained age of 99 years depends on cancer sites, from 3% for the colon and liver, to 10% for the bladder.

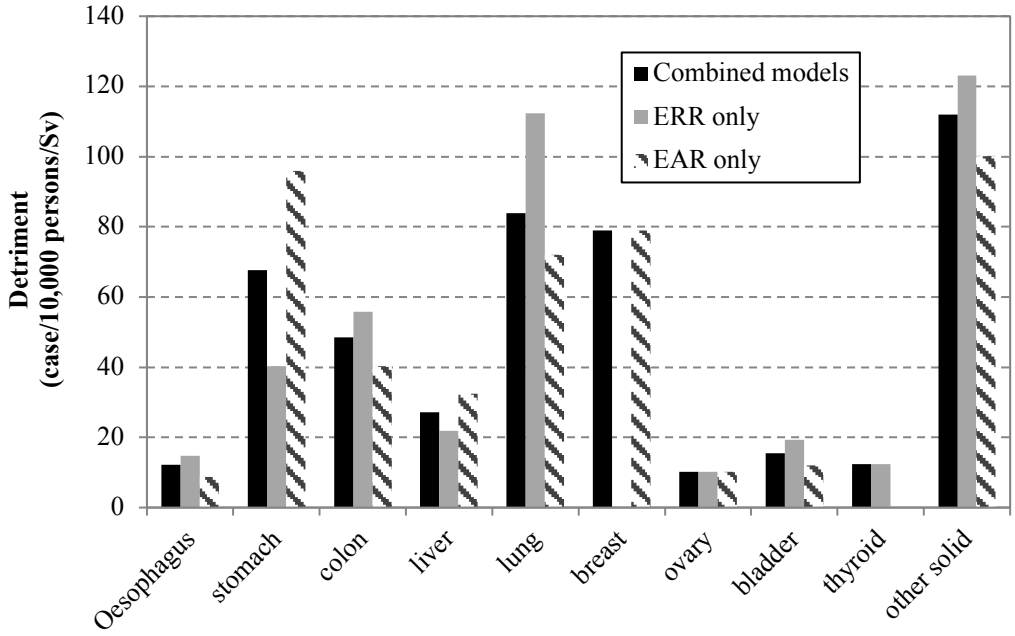


Fig. 4.5. Radiation detriment calculated using a 100% ERR-based model and a 100% EAR-based model, in comparison with the combined models used in *Publication 103*. Taken from Zhang et al. (2020).

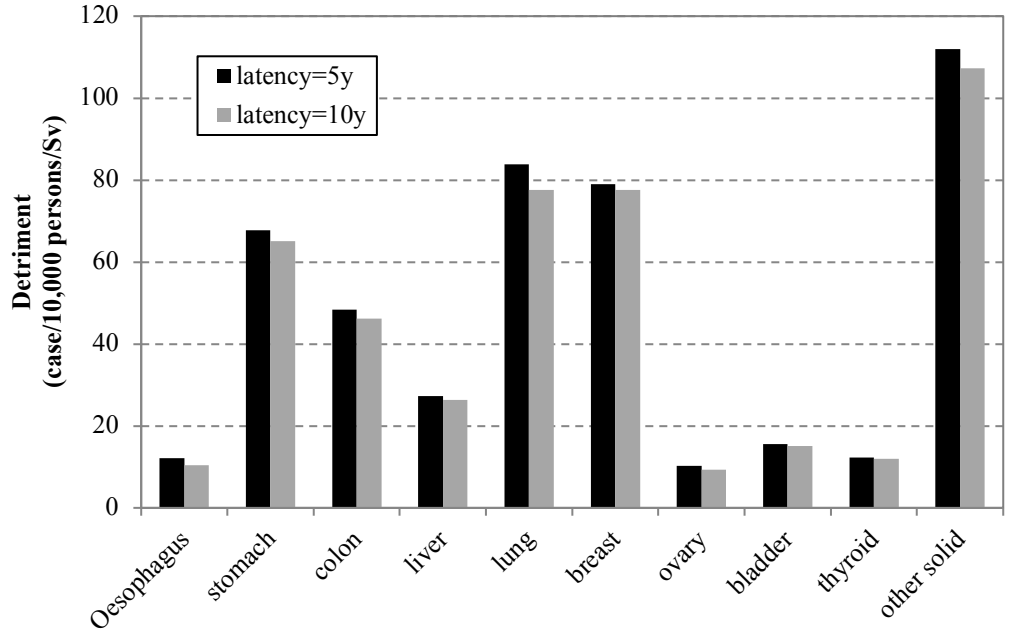


Fig. 4.6. Variation in radiation detriment with different latency periods. Taken from Zhang et al. (2020).

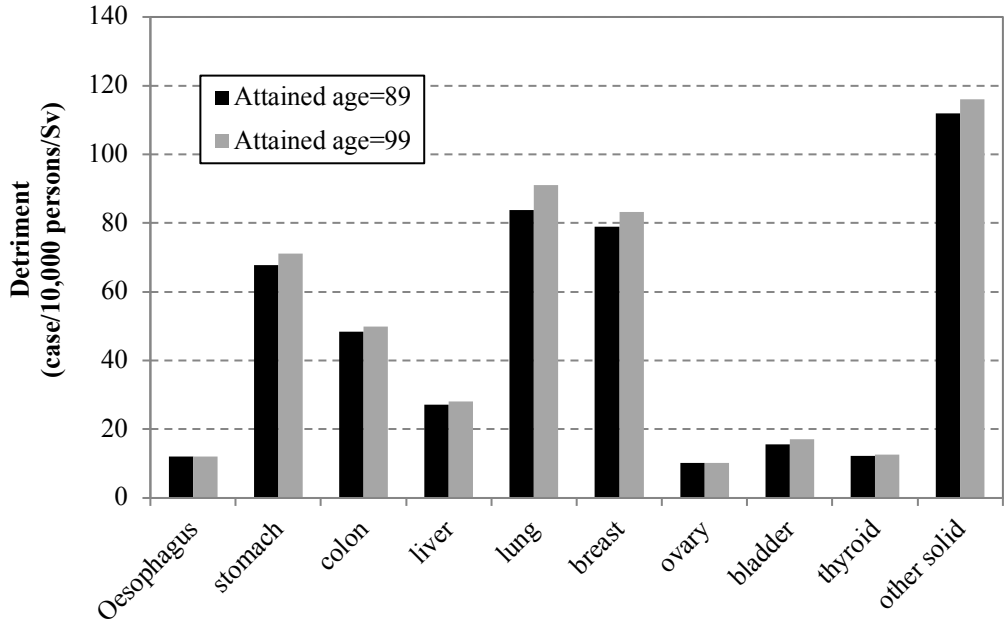


Fig. 4.7. Comparison of cancer detriment calculated for different maximum attained ages. Taken from Zhang et al. (2020).

(96)The effects of varying the values of the parameters used in the radiation detriment calculation are summarised in Table 4.1. The second column of Table 4.1 shows the results calculated based on the methodology of *Publication 103* (ICRP, 2007) (hereafter referred to as 'standard detriment') for various cancers, along with the ratio of radiation detriments under varying conditions (those with the relevant parameter change). In reference to the detriment of *Publication 103*, the ratios illustrate the sensitivity of the radiation detriment with respect to changes in the value of the parameters used for the calculation. For example, the radiation detriment from thyroid cancer in the group of 0–14 years of age at exposure is 3 times higher than that of the group of 0–84 years of age-at-exposure. The detriment calculation for breast cancer was based on the 100% EAR model, which does not depend on the baseline rate of breast cancer incidence. Although the baseline rate for breast cancer is higher for the Euro-American population than that of the Asian population, the detriment from radiation exposure for the Asian population was higher than the Euro-American population as shown in Table 4.2. This is because the radiation detriment is proportional to the product of the EAR model and the survival curve, while the EAR model produces the same results for the Euro-American and the Asian population, the survival curve used in the calculation decreases more slowly for the Asian population than for the Euro-American population between ages 50 and 75 years as shown in Fig. 3.2.

Table 4.1. Standard detriment and ratio of radiation detriment for changed parameter values compared with standard detriment.

Cancer site	Standard detriment	Relative change in radiation detriment due to variation of input parameter values										
		DDREF = 1	Age at exposure 18–64	Age at exposure 0–14	Male only	Female only	Euro-American	Asian	ERR-based model only	EAR-based model only	Latency = 10 years	Attained age = 99 y
Oesophagus	12.13	2	1.00	1.07	1.00	0.93	0.79	1.14	1.21	0.71	0.86	1.00
Stomach	67.71	2	0.80	1.86	0.86	1.15	0.73	1.27	0.59	1.42	0.96	1.05
Colon	48.44	2	0.85	1.79	1.39	0.61	0.89	1.09	1.15	0.83	0.95	1.03
Liver	27.22	2	0.81	1.84	1.35	0.65	0.65	1.35	0.81	1.19	0.97	1.03
Lung	83.88	2	1.16	1.01	0.67	1.33	1.01	0.99	1.34	0.86	0.92	1.08
Breast	78.93	2	0.58	2.52	0	2.02	0.92	1.10	0	1.00	0.98	1.05
Ovary	10.27	2	0.73	1.91	0	2.00	1.09	0.87	1.00	1.00	0.91	1.00
Bladder	15.52	2	0.93	1.55	1.08	0.95	1.20	0.80	1.25	0.78	0.98	1.10
Thyroid	12.32	2	0.32	3.23	0.29	1.71	0.77	1.19	1.00	0	0.97	1.03
Other solid	112.02	2	0.69	2.25	1.08	0.92	1.11	0.88	1.10	0.89	0.96	1.04

Note: The first column is the standard cancer detriment derived for whole population 0–84 years. They were newly calculated for this report and are slightly different from the values in Table 3.6, which are quoted from *Publication 103*. The rest of columns represent the ratio of radiation detriment for the special condition defined in the column title over the standard detriment in the first column. Taken from Zhang et al. (2020).

(97)Although the lifetime risk calculations in *Publication 103* and this report were based on the risk of exposure induced cancer/death (REIC/REID), there are variations with slightly different methods (Thomas et al., 1992). Alternative methods are the lifetime attributable risk (LAR) method or the excess lifetime risk (ELR) method. Comparisons of radiation detriment based on different lifetime risk calculation methods are shown in Fig. 4.8. There are small differences (1–4%) in detriments for stomach, colon, liver, breast cancers, while the differences become greater for other solid cancers (4–10%).

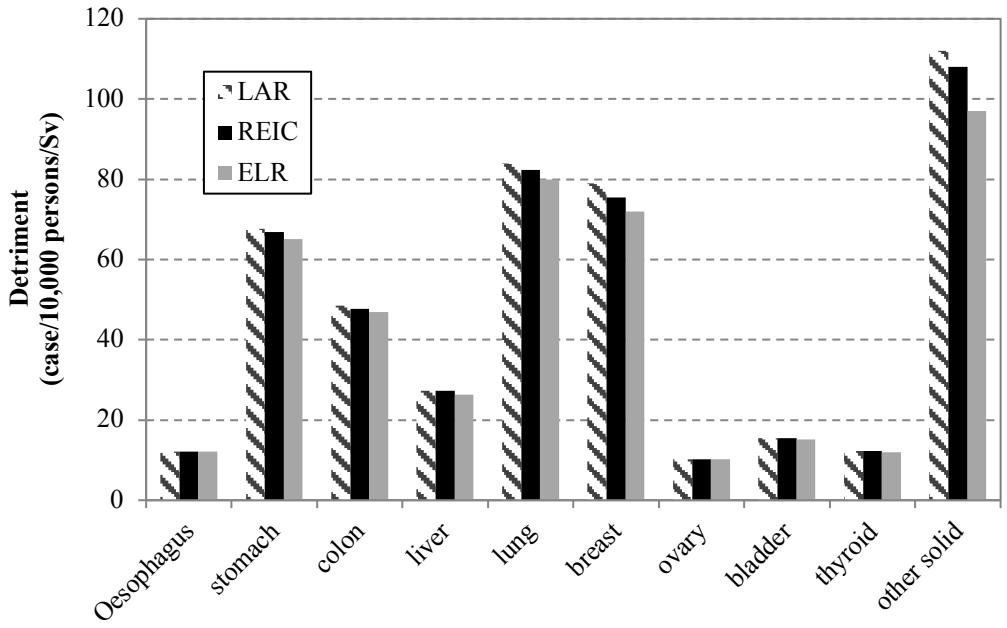


Fig. 4.8. Comparison of cancer detriment based on lifetime risk calculated by three different methods (LAR, REIC and ELR). Taken from Zhang et al. (2020).
LAR: lifetime attributable risk; REIC: risk of exposure induced cancer; ELR: excess lifetime risk

4.2. Parameters related to adjustment for severity

(98)Apart from the nominal risks that are the most important part in the calculation of radiation detriment, adjustment factors can also contribute to variation in the values of the detriment. They include the lethality fraction (k), minimum quality-of-life factor (q_{min}), and relative years of cancer-free life lost (l). The lethality fraction is used to compute the nominal risk of fatal cancers, and at the same time, it serves as a parameter to adjust the quality of life of non-fatal cancers. The parameter q_{min} represents the minimum weight due to pain, suffering, and any adverse effects of treatment that are commonly experienced by cancer survivors. According to the formulation in Section 3.2.2, the adjustment factor q increases with the values of k and q_{min}, both of which are expressed as a value between 0 and 1. Setting q_{min} at the maximum value ($q_{min} = 1$) produces the same effect as $k = 1$, demonstrating the worst level of quality of life that is comparable to the loss of life (fatal cancers). In view of this relationship, potential impact of these parameters has been tested for two extreme scenarios $k = 1$ and $q_{min} = 0$, respectively. The values of relative cancer-free life lost vary between organs and tissues, ranging from 0.71 to 1.29 for solid cancers as shown in Table 3.6. To illustrate the effect of changes in this parameter, a calculation was made by setting l at 1 for every cancer site.

(99)Fig. 4.9 shows the comparison between the radiation detriments calculated using the methodology described in *Publication 103* (ICRP, 2007) and that with the lethality fraction equal to one. For colon, breast, bladder, thyroid cancers and other solid cancer, there is a noticeable reduction in radiation detriment using the lethality data from *Publication 103*, compared to detriment using a lethality fraction equal to one. The Commission set the value of q_{min} to 0.2 for thyroid cancer and 0.1 for other types of cancer in *Publication 103*. Fig. 4.10 shows the comparison between the use of these values and q_{min} of zero, showing that the differences in radiation detriment are small for most cancer sites, except for thyroid cancer, for which q_{min} of zero results in more than a 50% decrease in detriment.

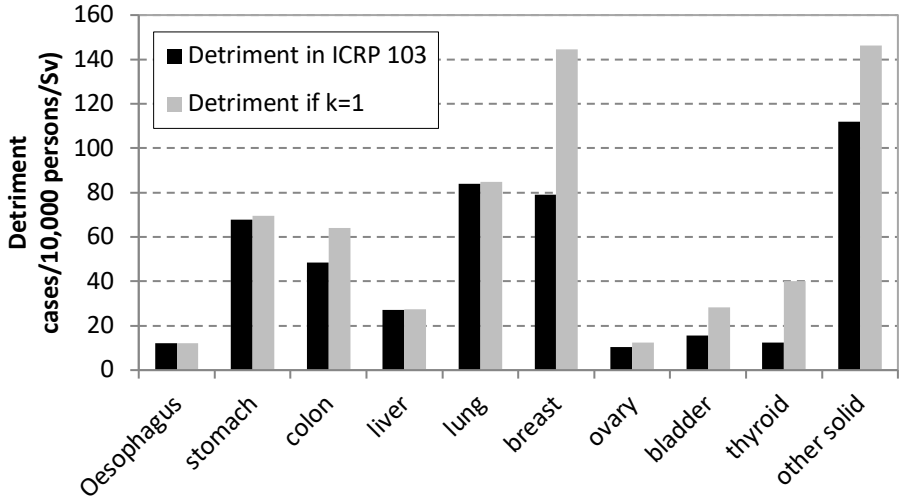


Fig. 4.9. Comparison of standard cancer detriment with that calculated for lethality fraction (k) of one. Taken from Zhang et al. (2020).

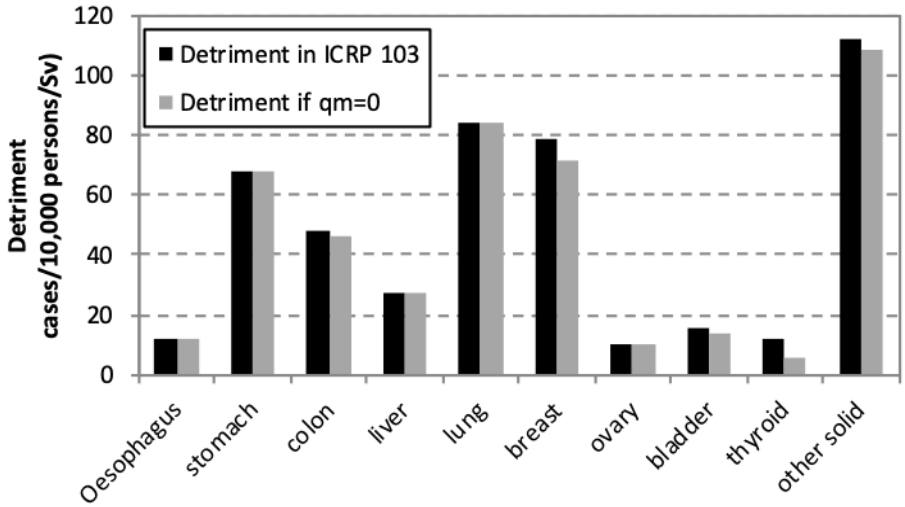


Fig. 4.10. Comparison of standard cancer detriment with that calculated for minimum quality-of-life factor equal to zero. Taken from Zhang et al. (2020).

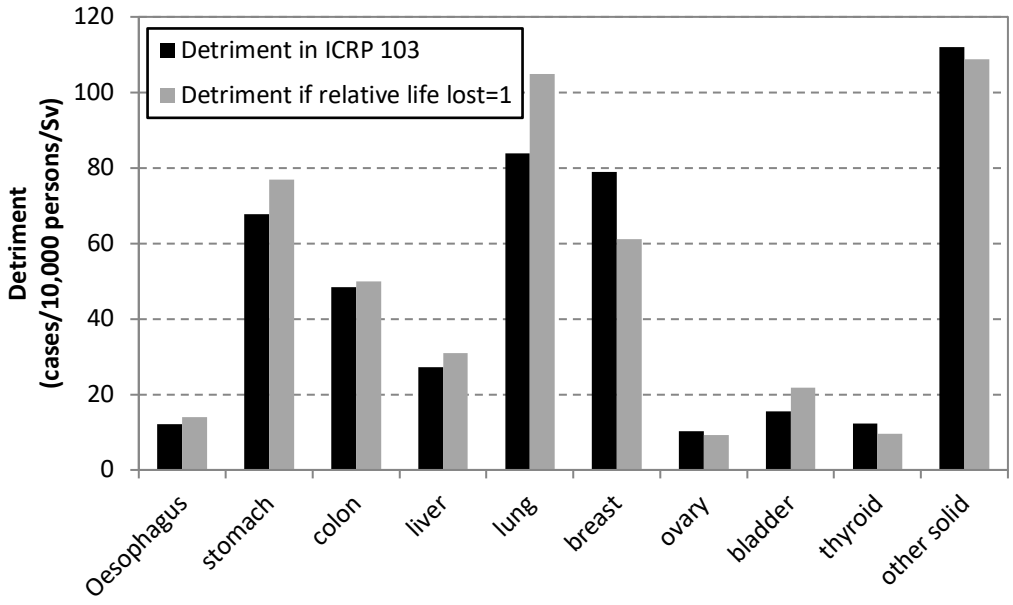


Fig. 4.11. Comparison of standard cancer detriment with that for relative cancer-free life lost (*l*) equal to one. Taken from Zhang et al. (2020).

(100) Fig. 4.11 shows the comparison of results using relative cancer-free life lost as presented in *Publication 103* (ICRP, 2007) and equal to one respectively. Variations in radiation detriment are particularly pronounced for breast, stomach and lung cancers, with increases or decreases of up to about 30–40%.

(101) Table 4.2 summarises the ratios of radiation detriments over the standard detriment in relation to the variations of the lethality fraction, the minimum quality-of-life factor and the relative cancer-free life lost parameters. The impact is noticeable for thyroid cancer when the minimum quality-of-life factor is set to zero. The radiation detriment varies for different cancer sites. When the cancer-free life lost is set to be the same as that of all cancers combined, detriment increases for some cancer sites, such as oesophagus and bladder, but decreases for other cancer sites, such as the breast.

(102) With the improvement in diagnostic techniques and treatment, the cancer death rate has declined during recent decades. Publication of U.S. cancer statistics (Siegel et al., 2019) show that the cancer death rate has declined by 27% from 1991 to 2016. The decline is pronounced in cancers with high lethality: in the case of lung cancer, the death rate has dropped by 48% in men between 1990 and 2016, and by 23% in women from 2002 to 2016. The situation may lead to a considerable change in the values of lethality fraction, and this should be taken into consideration in the future. A more detailed discussion about this issue is found in a study by Breckow et al (2018).

Table 4.2. Standard detriment and ratio of radiation detriment for different settings in lethality fraction, minimum quality-of-life factor and relative years of cancer-free life lost, over the standard detriment.

Cancer site	Standard detriment	Ratio of detriment of lethality = 1 over standard detriment	Ratio of detriment of q_{min} = 0 over standard detriment	Ratio of detriment of relative life lost = 1 over standard detriment
Oesophagus	12.13	1.00	1.00	1.15
Stomach	67.71	1.03	1.00	1.14
Colon	48.44	1.32	0.96	1.03
Liver	27.22	1.00	1.00	1.14
Lung	83.88	1.01	1.00	1.25
Breast	78.93	1.83	0.91	0.78
Ovary	10.27	1.20	0.98	0.89
Bladder	15.52	1.83	0.91	1.41
Thyroid	12.32	3.24	0.44	0.78
Other solid	112.02	1.31	0.97	0.97

Note: The first column is the standard cancer detriment derived for whole population 0–84 years, as in Table 4.1 The rest of columns represent the ratio of radiation detriment for the special condition defined in the column title over the standard detriment in the first column. Taken from Zhang et al. (2020).

4.3. Summary of sensitivity analysis

(103) Based on the calculation result presented above, the parameters can be classified into three categories according to their level of impact on radiation detriment: limited, noticeable and large.

(104) Parameters of limited impact: minimum latency period, maximum attained age, lifetime risk calculation method, minimum quality-of-life factor, and relative years of cancer-free life lost. Changing these parameters results in changes in radiation detriment by a factor of less than 1.5. An exception is the minimum quality-of-life factor for thyroid cancer, but it has little influence on the overall detriment.

(105) Parameters of noticeable impact: reference population and transfer model. Changing the setting of these parameters shows changes in radiation detriment by a factor of 1.5 or more, and less than 2 for some cancer sites. To transfer radiation risk from one population to another, both additive and multiplicative projections are plausible in terms of biological mechanism. Nevertheless, for most cancer sites, the best way to transfer estimates of risk from radiation exposure between populations is still unknown (UNSCEAR, 2012). The choice of the transfer model is particularly important for cancers with varying baseline risks between populations. In this regard, there is a significant difference in baseline rates between Asian and Euro-American populations for female breast, stomach and liver cancer. Depending on the combination of the transfer model and the population, radiation detriment can vary considerably for these cancers.

(106) Parameters of large impact: DDREF, age at exposure, sex and lethality fraction. Changing the setting of these parameters demonstrates changes in radiation detriment by a factor of 2 or more for some cancer sites. The choice of DDREF value has a direct impact, resulting in a two-fold increase in detriment for solid cancers when it is set to 1 instead of 2. In a broad sense, the issue is not limited to the choice of the DDREF value, but is related to the

shape of the dose-response curve. UNSCEAR assumed a linear-quadratic dose-response relationship in estimating the solid cancer risk instead of using the LNT model combined with a DDREF (UNSCEAR, 2006). As for the influence of sex and age at exposure, assuming a female-only population doubles the radiation detriment for breast and ovarian cancers in comparison with the sex-averaged detriment; the exposure to children at 0–14 years of age shows larger detriments, 3.2 times increase for the thyroid, 2.5 times for the breast and almost double for several cancer sites. Finally, the lethality fraction can have a large impact on the radiation detriment. By increasing the lethality fraction to 1 results in a significant increase in the detriment mainly for relatively non-lethal cancers such as thyroid, bladder and breast cancers. Conversely, the progress in diagnostic techniques and treatments should bring about a decrease in radiation detriment as of today and may lead to a significant decrease in the future.

(107) The sensitivity analyses presented here should be regarded as illustrative of the effect of the various factors involved in the calculation of radiation detriment. Bone cancer, skin cancer and leukaemia were excluded from the analysis because of missing information to perform calculations. The parameter settings were not necessarily realistic due to a paucity of available data. For example, the lethality fraction and relative years of cancer-free life lost were set to 1 and the minimum quality-of-life factor to 0, which oversimplifies the real-life scenarios. Finally, the baseline mortality and incidence were as assumed in *Publication 103* although they changed over time.

5. POTENTIAL EVOLUTION

(108) Radiation detriment is an indicator of the overall harm to health resulting from low-dose and low-dose-rate exposures. Based on scientific evidence, it takes into account key aspects of human health and variability among sexes, ages and populations, forming a solid basis for the system of radiological protection. Although the current scheme of radiation detriment calculation is carefully designed to achieve this aim, it needs to evolve according to advances in healthcare and scientific understanding of radiation health effects, as has been the case in the past (see Section 2). There is also scope for further improvement in methodology. In this section, the direction of future evolution and possible ways of improvement are discussed.

5.1. Input information

5.1.1. Reference population data

(109) The calculation of the radiation detriment requires the use of reference population data for baseline cancer rates, mortality and age- and sex-structure.

- Baseline rates used in *Publication 103* (ICRP, 2007) correspond to the period 1993–1997 (Parkin et al., 2002). Cancer incidence rates and mortality rates have changed significantly since then due to the changes in lifestyle, advances in diagnostic methods and improvement in cancer treatment, especially for certain cancers. Updating these reference rates will provide a more realistic basis for the system of radiological protection in the future (Breckow et al., 2018). Furthermore, no baseline rates were provided in *Publication 103* for skin and bone cancers.
- It should be noted that incidences and mortalities vary considerably around the world reflecting genetic/lifestyle difference and differences in healthcare provision. In *Publication 103*, two reference populations were considered: Asian (composite rates from China (Shanghai), Japan (Osaka, Hiroshima and Nagasaki)) and Euro-American (composite rates from Sweden, United Kingdom and the Surveillance, Epidemiology and End Results (SEER) program of the US National Cancer Institute). Extension to other populations will provide a broader representation of the world population based on available data.

5.1.2. Cancer risk models

(110) The calculation of the radiation detriment requires the use of models describing the relationship between the organ/tissue dose and cancer risk for specific cancer sites. The following points provide a summary of cancer risk models in *Publication 103* (ICRP, 2007) and possible ways of updating them.

- Radiation-associated cancer risk models for 11 categories of organs or tissues (oesophagus, stomach, colon, liver, lung, female breast, ovary, bladder, thyroid, other solid cancers and red bone marrow) were derived from the LSS, based on a follow-up from 1958 through 1998 (Preston et al., 2007). Since then, new models with longer follow-up have been published, that can be used to update the risk models.

- For most solid cancers, risk modifying factors (age at exposure and attained age) were parameterised as all solid cancers as a group, and the same values were used for both sexes. The longer follow-up of the LSS will provide more detailed information to establish models that better reflect the variation of risk with sex, age at exposure and attained age for respective cancer sites.
- The bone marrow category includes leukaemia other than CLL. It is desirable to explore the possibility of extending this category to other types of haematological malignancy, such as lymphoma and multiple myeloma.
- Nominal risk estimates for bone cancer and non-melanoma skin cancer were taken from *Publications 60* and *59* (ICRP, 1991, 1992), respectively. These risk estimates are based on early studies, with large uncertainties, and do not allow for variation of risk with sex and age. For better internal consistency in the calculation, it is desirable to investigate whether more up-to-date risk models are available for these two tissues.
- No specific risk models were derived for the brain and salivary glands, whereas tissue weighting factors were assigned specifically to these two organs. To clarify the rationale for these values, it is also desirable to explore the possibility of developing risk models for these two organs.
- The category 'other cancer sites' accounts for about 20% of the total radiation detriment. When additional data are accumulated in the future, it is desirable to quantify the risk for some of them as separate cancer sites in order to reduce the contribution of this heterogeneous category.
- Most of the risk models were derived from the LSS without incorporating findings from other sources. During the last decade, many reports provided risk models derived from other epidemiological studies, especially for populations with protracted exposures (e.g. nuclear workers, Mayak workers, residents along the Techa river, and Chernobyl clean-up workers). Evaluation of the models derived from these studies should be performed based on a detailed analysis of their respective limitations and advantages, and discussion of the consistency of their results.
- The models to calculate the nominal risks rely on several assumptions, including the LNT model, application of a DDREF, and the use of a transfer scheme based on the weighting of ERR and EAR models. The validity of these assumptions must be examined in the light of the latest scientific findings. In this regard, recent epidemiological literature has been reviewed by the National Council on Radiation Protection and Measurements to examine the validity of the LNT model (NCRP, 2018; Shore et al., 2018, 2019). The Commission has launched a Task Group to review the scientific basis of the DDREF in terms of epidemiology, animal experiments and cell biology. Several papers have already been published (Rühm et al., 2015, 2016, 2018; Shore et al., 2017; Tran and Little, 2017; Wakeford et al., 2019) and a dedicated report will be released in due course.

5.1.3. Cancer severity parameters

(111) Calculation of radiation detriment from nominal risks involves three parameters reflecting the severity of the diseases: lethality, quality of life, and years of life lost.

- Lethality fractions per cancer site have been provided as judgement-based values derived from U.S. population data for the 1980–1985 and 1950–1970 periods (U.S. DHHS, 1989). The same lethality fraction values were used for males and females, the general population

and workers. Recent data exist, that provide much better estimates of current cancer lethality, with variations with age and sex. Also, collection of lethality estimates from other populations outside of the USA is desirable to better reflect variation of lethality among different populations in the world.

- Relative estimates of years of life lost were calculated from values used in *Publication 60* (ICRP, 1991). As in the case of the lethality fractions, a review of recent data sources will provide better estimates of current years of life lost due to the specific cancers, with variations with age and sex, and among different populations.
- Adjustment for quality of life of cancer patients was based on the use of very approximate value judgements. More elaborate approaches such as disability-adjusted life years (DALY) are now available to estimate and characterise the quality of life for a wide range of conditions (Chen et al., 2015; Shimada and Kai, 2015). A review of these methods and of available data can help, taking into account the variation with age, sex and geographical region. Some of these approaches combine the quality of life with lethality and years of life lost indicators. Such methods should make the severity adjustment simpler and more reliable.
- The current scheme of the severity adjustment relies mainly on the lethality fraction, and this method gives little weight to non-lethal cancers such as thyroid cancer. They would be better handled if based on the characteristics of each type of cancer.

5.1.4. Heritable effects

(112) The risk of heritable effects in the radiation detriment is derived from the estimate in the UNSCEAR 2001 Report for all classes of genetic diseases up to the first two generations (UNSCEAR, 2001). In recent years, new findings have been obtained, including epigenetic inheritance. It is desirable to review the current literature on the mechanism of inheritance, available data and the methods that can be used for estimating risks of heritable diseases. Advances in this field should help integrate heritable effects into detriment calculation in a manner more consistent with the current methodology that was developed for cancer.

5.2. Variation with sex and age

(113) The sensitivity analysis in Section 4 as well as the cancer risk estimate in *Publication XXX* has demonstrated that the age at exposure has a large impact on radiation detriment. In particular, an exposure during childhood substantially increases the lifetime risk for most cancer sites compared to exposure during adulthood, which therefore results in a larger calculated detriment value than that for adult exposure. Differences due to sex are also notable for some tissues, with the most extreme examples of the ovary and the breast. It is advisable to calculate lifetime risks separately for sexes and selected ages (age groups) and average in the last stage to obtain a nominal value. While this approach requires the development of sex- and age-specific values at each step of the radiation detriment calculation (as far as possible) to avoid averaging at intermediate steps, the results should allow to delineate the variation of risk with sex, age at exposure and attained age, and potentially among different populations.

(114) This above approach distinguishes science-based risk assessment from the subsequent integration of information for protection purposes, thus providing a better understanding of the construction of radiation detriment. This may also apply to other influential factors, including modifiable lifestyle factors. The Commission defines the nominal population as a mixture of

people with different factors governing individual response to radiation. A new ICRP Task Group has been set up to review scientific information relevant to the topic of individual response. If factors that greatly influence the sensitivity to cancer induction are identified in the future, whether or not modifiable, the variation of risk with them should be assessed.

(115) Considering situations where the radiation detriment is used for simplified risk estimation, illustrating the variation of lifetime risk estimates with sex and age will help to understand potential deviations from individual risks in specific situations. This is especially the case in healthcare situations involving individual patients or specific groups of patients (Anderson et al., 2017).

(116) The current set of tissue weighting factors, w_T was determined based on the site-specific relative radiation detriments for the whole population (ICRP, 2007). Although the relative contribution of each cancer site to the detriment varies considerably with sex and age at exposure, these variations were not presented in *Publication 103*. A detailed description of them, providing different sets of relative detriments, will help to understand the distribution range and the representativeness of w_T.

5.3. Exposure scenario

(117) Lifetime risks are particularly high for childhood exposure, but the inclusion of adults in the radiation detriment calculation dilutes and offsets the higher lifetime risks in children. A similar situation could occur in a protracted exposure that lasts beyond young ages. The relative contribution of childhood exposure to the total risk becomes smaller as years go by.

(118) With the use of DDREF, the radiation detriment for an acute exposure averaged over the whole population is assumed to be equivalent to that for a lifelong continuous exposure of an individual. Similarly, the radiation detriment of workers represents a constant occupational exposure throughout the working life. While these two are the most typical patterns, other exposure scenarios may be possible.

(119) In-utero exposure is not considered currently in the radiation detriment calculation. If there is not much difference in cancer risk between antenatal exposure and childhood exposure, the lifetime risk for the foetus can be regarded comparable to that for the newborn. This suggests a limited impact on the nominal risk, but nevertheless, special consideration may be needed from an ethical point of view.

(120) The sensitivity analysis demonstrated that length of life has little impact on the radiation detriment. Nevertheless, to reflect increased longevity in the recent decades, extension of the lifetime beyond 90 years will be reasonable. Future demographic changes may also have an impact on the detriment through the alteration of age distribution in the reference populations.

(121) The dose for the radiation detriment calculation should continue to be 0.1 Gy to demonstrate it is intended for the low-dose, low-dose-rate exposure.

5.4. Consideration of non-cancer effects

(122) In *Publication 118* (ICRP, 2012), the Commission made a comprehensive review of accumulating evidence that circulatory disease and cataracts might be induced at much lower doses than previously considered, and therefore recommended a threshold dose of 0.5 Gy for the heart, brain and the lens of the eye irrespective of dose rate. Some recent epidemiological studies suggest a dose-dependent increase of the risk for these effects below 0.5 Gy, but there

is considerable uncertainty about the shape of the dose-response curve at doses below this value and the existence or not of a threshold (Baselet et al., 2016; NCRP, 2016).

(123) For circulatory disease, epidemiological data at low doses are varying according to the health outcome considered and whether analyses are based on incidence or mortality (Yamada et al., 2004; Ozasa et al., 2016). Difficulties are also encountered in quantifying the baseline risk. Health statistics on mortality from circulatory disease exhibit large variations between countries and within each country over time. Data sources of morbidity or incidence are limited and not as standardised as those for cancer. Adjustment for severity is not straightforward, considering the large variation of symptoms and conditions of circulatory disease among patients.

(124) For cataracts, evidence of the risk increase due to radiation exposure is more compelling than circulatory disease. However, heterogeneity of epidemiological data is reported for lens opacities (NCRP, 2016), and the choice of the endpoint and diagnostic method greatly influence the shape and slope of the dose-response curve. There is no reliable source for baseline statistics on vision impairing cataracts. Furthermore, regional variation in health care development is a significant factor in adjusting for quality of life since cataract is a leading cause of blindness in many developing countries where surgery is hardly accessible.

(125) In addition to the aspects discussed above, the determination of underlying biological mechanism and the identification of target tissues related to these effects should be clarified. Whether or not to include them in the calculation of the radiation detriment currently remains an open question.

5.5. Transparency and comprehensibility

(126) As described in Section 3, the calculation of radiation detriment involves many steps in which a wide range of information is processed, including risk models, health statistics and various other parameters. As the methodology becomes increasingly elaborate, it becomes important to accurately document and publish the calculation procedure to ensure transparency and traceability. A full description of the calculation steps is necessary, and it is desirable to develop and share an open-source software to perform these calculations.

(127) Radiation detriment relates to stochastic effects and requires probabilistic representation. In the current method, it takes the form of a risk value expressed as a percentage, which is interpreted as the burden imposed on a nominal population. However, such a metric is difficult to understand for non-specialists. Another possible way is to express the detriment in terms of the lengths of time lost from normal health and activity as a result of harmful effects of radiation. This approach was taken in the assessment of the index of harm, which was defined as years lost per 1000 worker-years (ICRP, 1977b, 1985). Expressing detriment based on the expected values may give a wrong impression that the burden of disease is evenly distributed in the population. Nevertheless, it is much more intelligible and applicable to any deteriorated health condition. Indeed, a similar concept, DALY, is widely used in the fields of welfare, public health and medical services. It combines mortality and morbidity in a single metric, and efforts have been made to assign reasonable weights to a wide range of non-fatal conditions and impairments (Chen et al., 2015). This approach proved applicable to radiation detriment as well (Shimada and Kai, 2015).

(128) There is no simple way to express the multidimensional nature of detriment, and it will be necessary to improve its presentation in the future so that the make-up of radiation detriment becomes more comprehensible to non-specialists. It is also desirable to provide graphical presentation of key components of detriment, which will give a wider, balanced

perspective on the health risks of radiation. They include information about reference populations, absolute years of cancer-free life lost, and a baseline for the radiation detriment calculation (calculation assuming no radiation exposure).

6. SUMMARY AND CONCLUSIONS

6.1. Calculation of radiation detriment

(129) The concept of detriment was first introduced in *Publication 22* (ICRP, 1973). In a broad sense it includes any form of deleterious effects, but the methodology has been developed to quantify the harmful health effects of radiation exposure at low doses and low dose rates. Its principal components are probability of attributable cancer, probability of adverse heritable effects, and weighting to adjust for the severity of these conditions. When the detriment is calculated as an adjusted excess risk from radiation exposure using the Commission's methodology, it is specifically called radiation detriment.

(130) The calculation process of radiation detriment consists of two main parts. The first part is the calculation of nominal risks, which is an estimate of the lifetime risk of stochastic effects averaged over both sexes, all ages at exposure and populations. The second part is the calculation of the radiation detriment in which the nominal risk is adjusted for severity. The second part is independent of radiation dose.

(131) Although Annex A of *Publication 103* (ICRP, 2007) describes the data and models for the radiation detriment calculation, the details were not fully documented. Section 3 of this report has provided full details of the detriment calculation procedure, clarifying the following points.

- After verification, the risk transfer model for leukaemia turned out not to be 100% EAR as indicated in *Publication 103*, but 50:50% ERR:EAR. The EAR-based model was developed using an LSS dataset with a follow-up from 1950 through 1998 and based on the DS02 dosimetry system. The ERR-based model was derived from the EAR-based model and the baseline risk, but details about the models are not available.
- The lifetime risk estimate was REIC, rather than ELR or LAR. It was cumulated over an age range of 0–89 years (90 years of life) for the whole population, and 18–89 years (72 years of life) for adult workers.
- To estimate a lifetime risk per Gy, REIC at 0.1 Gy was calculated and multiplied by 10, for each age at exposure.
- The age-averaged lifetime risk was calculated as a weighted mean of the lifetime risk estimated for each age-at-exposure, the weights being calculated using the age distribution derived from the four reference populations (males and females of Asian and Euro-American populations).

6.2. Sensitivity of radiation detriment

(132) In the calculation of radiation detriment, the lifetime risk estimates were adjusted downward by applying a DDREF of 2 for solid cancer. The choice of the DDREF value thus directly affects the detriment. For example, if the DDREF is set to 1, it doubles the radiation detriment of solid cancers.

(133) Age at exposure and sex are influential factors as well. The radiation detriment for the young age-at-exposure group (0–14 years) is higher than that for the whole population (0–84 years) by more than a factor of 2 for some cancer sites (i.e. stomach, breast, thyroid and other solid cancers). Sex-averaging results in a halving of risks from ovary and breast cancers.

There are also significant differences in lifetime risk between males and females for other organs such as the lung, liver, colon and thyroid.

(134) The sensitivity analysis also demonstrated a large impact of the lethality fraction on the radiation detriment. The lethality fractions currently used in the detriment calculation are based on the data from the US in the 1980s. There is a need for updating these data as the progress in diagnostic techniques and treatments since then may lead to a substantial decrease in lethality fraction.

(135) Another important result of the sensitivity analysis is the significance of the transfer model. As was demonstrated in Section 4, the transfer model has a noticeable impact in the calculation of radiation detriment. It is particularly important for cancers with varying baseline risks between populations. The choice of the transfer model, together with the reference populations, continues to be a fundamental issue in the estimation of radiation-associated cancer risks and requires further research with updated information.

6.3. Suggestions for future improvements

(136) Based on the result of the sensitivity analysis, DDREF, age at exposure and sex are key factors to be considered to improve radiation risk estimation in the future. Efforts should be made to better characterise the dose-response relationship for each cancer site at low doses and low dose-rates. This may include promoting epidemiological studies of populations exposed to chronic radiation exposure with good individual records, both for incidence and mortality data. More research is needed to refine the risk estimates for childhood exposures. Elucidating the difference in sensitivity between males and females is another important priority.

(137) Considering the variation of cancer risk with sex and age, it is advisable to calculate lifetime risks for both sexes and selected ages separately, and then to average them only in the last stage to obtain a nominal value. This may also apply to other influential factors, including modifiable lifestyle factors. If factors that greatly influence the sensitivity to cancer induction are identified in the future, the variation of risk with them should be assessed.

(138) Age dependence of the risk also has relevance to the representativeness of the nominal population. If a situation arises in which children and young people are mainly exposed, due consideration should be given to the validity of the radiation detriment for the whole population.

(139) Radiation detriment needs to evolve depending on changes in cancer incidence and survival rate, and on advances in scientific understanding of radiation health effects. From this viewpoint, reference population data and cancer severity parameters need to be updated and improved. There is also scope for improvement in cancer risk models, including use of the LSS data with a longer follow-up, models derived from other epidemiological studies, especially for populations with protracted exposures, and specific risk models for the bone, skin, brain, salivary gland and haematological malignancies other than leukaemia. Consideration of recent findings regarding heritable effects of radiation is also necessary.

(140) The Commission recommended a lower threshold dose for circulatory disease and cataracts in *Publication 118* (ICRP, 2012) than before, but there is considerable uncertainty about the existence or not of a threshold for these effects and the dose response at low doses if there is no threshold. Whether or not to include them in the calculation of the radiation detriment currently remains an open question.

(141) As the methodology of detriment calculation changes, ensuring transparency and traceability is important. A full description of calculation steps is necessary, and consideration should be given to the development of an open-source software for calculating radiation

1499 detriment. It is also desirable to improve the presentation of the radiation detriment so that non-
1500 specialists can have a balanced perspective on the health risks of radiation.
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ABBREVIATION LIST

BEIR: Biological Effectiveness of Ionizing Radiation

CLL: chronic lymphocytic leukaemia

D: detriment

DALY: disability-adjusted life years

DD: doubling dose

DDREF: dose and dose-rate effectiveness factor

DS: dosimetry system

EAR: excess absolute risk

ELR: excess lifetime risk

ERR: excess relative risk

DHHS: Department of Health and Human Services

Gy: Gray

ICRP: International Commission on Radiological Protection

k: lethality adjustment factor

l: years of life lost adjustment factor

LAR: lifetime attributable risk

LET: linear energy transfer

LSS: life span study

LQ: linear-quadratic

NCRP: National Council on Radiation Protection and Measurements

NRC: National Research Council

q: quality of life adjustment factor

R: nominal risk coefficient

REIC / REID: risk of exposure-induced cancer incidence / death

rem: Röntgen Equivalent Man, old unit of dose measuring the equivalent dose and effective dose (1 rem = 0.01 Sv)

SEER: Surveillance, Epidemiology, and End Results

Sv: Sievert

UNSCEAR: United Nations Scientific Committee on the Effects of Atomic Radiation

WHO: World Health Organization

w_T: tissue weighting factor

YLL: years of life lost

GLOSSARY

Absorbed dose, D

The fundamental dose quantity given by:

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

where $\mathrm{d}\bar{\varepsilon}$ is the mean energy imparted to matter of mass $\mathrm{d}m$ by ionising radiation. For radiological protection purposes, the absorbed dose D_T, averaged over the organ or tissue T, is often used, which is given by:

$$D_T = \frac{1}{m_T}\int D\,\mathrm{d}m = \frac{\varepsilon_T}{m_T}$$

where m_T is the mass of the organ or tissue T, D is the absorbed dose in the mass element $\mathrm{d}m$, and ε_T is the mean total energy imparted in the organ or tissue T. The SI unit for absorbed dose is joule per kilogram (J kg^{-1}) and its special name is gray (Gy).

Active (red) bone marrow

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

Baseline rates

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

Deterministic effect

Injury in populations of cells, characterised by a threshold dose and an increase in the severity of the reaction as the dose is increased further. The term means ‘causally determined by preceding events’ in contrast to ‘stochastic effect’. In some cases, however, deterministic effects are modifiable by post-irradiation procedures including biological response modifiers. The more directly descriptive term ‘tissue reaction’ is also used for this reason.

Detriment (See ‘radiation detriment’).

Disability-adjusted life years, *DALY*

A metric to quantify the burden of disease from mortality and morbidity. It is calculated as the sum of the years of life lost (YLL) due to premature mortality in the population and the years lost due to disability (YLD) for people living with the health condition or its consequences.

Dose and dose-rate effectiveness factor, *DDREF*

A judged factor that adjusts biological effectiveness (per unit of dose) of radiation exposures at low doses and low dose rates as compared with exposures at high doses and high dose rates. The DDREF applies specifically to doses below 0.2 Gy or dose rates less than 0.1 Gy per hour.

Dose limit

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

Doubling dose, *DD*

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

Effective dose, *E*

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

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

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

ELR

See ‘Lifetime risk estimates’.

Equivalent dose, H_T

The dose in a tissue or organ *T* given by:

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

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

Excess absolute risk

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

Excess relative risk

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

Gray (Gy)

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

Incidence (incidence rate)

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

LAR

See 'Lifetime risk estimates'.

Lethality fraction

Unitless judgement-based factor reflecting, for a cancer of specific organ or tissue, the ratio between mortality and morbidity.

Life Span Study, *LSS*

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

Lifetime risk estimates

Estimates of the risk, over a lifetime, that an individual will develop, or die from, a specific disease caused by an exposure. Several types of lifetime risk estimates can be used: 1) the excess lifetime risk (ELR) which is the difference between the proportion of people who develop or die from the disease in an exposed population and the corresponding proportion in a similar population without the exposure; 2) the risk of exposure-induced cancer death (REID) which is defined as the difference in a cause-specific death rate for exposed and unexposed populations of a given sex and a given age at exposure, as an additional cause of death introduced into a population; 3) the risk of exposure-induced cancer incidence (REIC) which replaces the cause-specific death rate in the REID calculation with a cancer incidence rate; 4) loss of life expectancy (LLE) which describes the decrease in life expectancy due to the exposure of interest; and 5) lifetime attributable risk (LAR) which is an approximation of the REID (or REIC) and describes excess deaths (or disease cases) over a follow-up period with population background rates determined by the experience of unexposed individuals.

Linear dose response

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

Linear-non-threshold (LNT) model

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

Linear-quadratic dose response

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

LLE

See 'Lifetime risk estimates'.

Mendelian diseases

Heritable diseases attributable to single-gene mutations.

Nominal risk coefficient

Sex-averaged and age-at-exposure-averaged lifetime risk estimates for an organ or tissue for a representative population. It is quantified assuming a linear-non-threshold (LNT) dose-response relationship for stochastic effects and applying a dose and dose-rate effectiveness factor (DDREF) of 2 for solid cancer.

Non-cancer diseases

Somatic diseases other than cancer, *e.g.* circulatory diseases and cataracts.

Quality of life factor

Unitless judgement-based factor representing adverse effects experienced by cancer survivors on their quality of life, in terms of pain, suffering, and any adverse effects of cancer treatment.

Radiation detriment

The concept of radiation detriment has been developed by the Commission for the purpose of radiological protection. It is defined as the excess of stochastic health effects in a group exposed to low-level radiation and its descendants compared to a non-exposed group. It is determined from nominal risk coefficients for a set of organs and tissues, taking into account the severity in terms of lethality, quality of life, and years of life lost.

Radiation weighting factor, w_R

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

Relative years of life lost

The years of life lost (YLL) represent an average shortening of life expectancy in years among those developing a cancer due to radiation exposure in comparison with a nominal value for the unexposed. The relative years of life lost is the ratio of YLL due to a cancer of a specific organ or tissue to YLL that is averaged over all cancer sites. For the incidence-based calculations, years of cancer-free life lost are used instead of YLL.

REIC/REID

See ‘Lifetime risk estimates’.

Sievert (Sv)

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

Stochastic effects of radiation

Health effects for which the probability of occurrence in a population, but not the severity, is regarded as a function of dose without threshold. Stochastic effects contributing to radiation detriment are cancers and heritable effects.

Threshold dose for tissue reactions

Absorbed dose value (in Gy) to an organ or tissue below which it is considered that the incidence of tissue reactions in a population is less than 1%.

Tissue reaction

See 'Deterministic effect'.

Tissue weighting factor, w_T

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

$$\sum_T w_T = 1$$

Transfer of risk (also called transport of risk)

Taking a risk model estimated for one population and applying it to another population with different characteristics. Usually, the transfer mode is multiplicative (based on an excess relative risk model), additive (based on an excess absolute risk model), or a weighted average of them.

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ICRP Task Group 102 was established in March 2016 to form a basis for future recommendations, by reviewing and documenting the current process of detriment calculation so that it may be carried out in a reproducible manner, considering ways in which different approaches might be applied when new data become available. This report was prepared by Task Group 102 (a) to prepare a document explaining the detailed procedure of detriment calculation and identifying sources for the necessary information; (b) to reproduce the calculation in *Publication 103*, identify any difficulties in reproducing the results, and comment on the approaches taken; (c) to identify potential modifications and improvements in the detriment calculation procedures; and (d) to establish and propose a methodology for future detriment calculation.

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