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3 **History of radiation detriment and its calculation methodology used in**
4 **ICRP Publication 103**
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Abstract

Over the past decades, the International Commission on Radiological Protection (ICRP) has used radiation detriment, which is a multidimensional concept to quantify the overall harm to health from stochastic effects of low-level radiation exposure of different parts of the body. The tissue-specific detriment is determined from the nominal tissue-specific risk coefficient, weighted by the severity of the disease in terms of lethality, impact on quality of life and years of life lost. Total detriment is the sum of the detriments for separate tissues and organs. Tissue specific detriment values are used to determine tissue weighting factors for the calculation of effective dose. Calculating radiation detriment is a complex process that requires information from various sources and judgements on how to achieve calculations. As such, it is important to document its calculation methodology. To improve the traceability of calculations and form a solid basis for future recommendations, the ICRP Task Group 102 on detriment calculation methodology was established in 2016. It has reviewed the history of radiation detriment and the process of detriment calculation. This article summarizes the work of this Task Group and aims at clarifying the process of detriment calculation currently used by the ICRP.

Keywords: detriment; radiation; health effect; cancer.

Highlights

1. The methodology of radiation detriment has been developed to quantify the harmful health effects of radiation exposure at low doses and low dose rates.
2. Radiation detriment takes into account the radiation induced lifetime risk for 14 cancer sites.
3. The radiation-associated cancer risk models were mostly derived from the Life Span Study of Hiroshima and Nagasaki atomic bomb survivors.
4. The age-averaged lifetime risk was calculated as a weighted mean of the lifetime risk estimated for each age at exposure, using the age distribution from four reference populations (males and females of Asian and Euro-American populations).

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1. Introduction

Exposure to ionising radiation may cause cancer and other health effects, each of which varies greatly in the shape of the dose-response curve, latency, persistency, recurrence, curability, fatality and impact on quality of life (Hamada & Fujimichi 2014). Over the past decades, for radiation protection purposes, the International Commission on Radiological Protection (ICRP) has divided all such diverse radiation effects into either stochastic effects (with no dose threshold) or tissue reactions (formerly termed deterministic effects, which are considered to have a dose threshold). Safety standards aiming to reduce risks of stochastic effects (cancer and heritable effects) are partly based on detriment-adjusted nominal risk coefficients, assuming a linear-non-threshold (LNT) dose response and a dose and dose-rate effectiveness factor (DDREF) of 2 which applies to absorbed doses <0.2 Gy and to higher absorbed doses at <0.1 Gy per hour for low linear energy transfer (LET) radiation (ICRP 1991). The detriment is a quantitative scale of the total harm to health among a group of the population and its descendants as a result of the group's exposure to ionising radiation, and allows for the variations in radiosensitivity of different organs and tissues to the induction of stochastic effects. The tissue weighting factors (w_T) are derived from the relative contribution of a tissue or organ to the total detriment following whole body exposure.

Calculating radiation detriment is a complex process that requires information from various sources and judgments on how the calculations are best performed. The current methodology is outlined in Annex A of ICRP Publication 103 (ICRP 2007), but it is possible that accumulation of scientific evidence for radiation induced effects and evolution of knowledge about cancer may need revised methodology and different judgments in the future. To secure a solid basis for future recommendations, it is important to review the current process of detriment calculation and document it in a reproducible manner, considering ways in which different approaches might be applied when new data become available. In 2016, ICRP therefore established Task Group 102 on detriment calculation methodology to identify sources for the necessary information, to reproduce the calculation in Publication 103 with documentation of the detailed procedure, and to find room for future improvement.

This paper summarizes some of the work of Task Group 102 with a focus on the history of radiation detriment and the calculation methodology used in Publication 103.

2. History of radiation detriment calculation

The multidimensional concept of detriment was introduced in ICRP Publication 26 (ICRP 1977) and defined as follows: 'The Commission has introduced the concept of detriment to

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3 identify, and where possible to quantify, all these deleterious effects. In general, the detriment in
4 a population is defined as the mathematical “expectation” of the harm incurred from an exposure
5 to radiation, taking into account not only the probability of each type of deleterious effect, but
6 also the severity of the effect¹. Quantitative values assessed for the radiation detriment for low
7 dose and low dose rate relied on the assumption of a linear relationship between dose and risk.
8 Recognizing that this linear extrapolation may lead to an overestimate of the radiation risks at
9 low doses and low dose rates, the Commission nevertheless endorsed its view described in ICRP
10 Publication 9 (ICRP 1966)¹. Additionally, the Commission indicated that even if some of the
11 quantitative risk factors were age or sex dependent, it was judged that for radiological protection
12 purposes, sufficient accuracy could be obtained by using dose limits regardless of age or sex for
13 both workers and the general public. Detriment, expressed as the likelihood of fatal cancers and
14 serious hereditary abnormalities, was then used to set up dose limits for workers and the public,
15 as well as tissue weighting factors (w_T) as part of the effective dose concept.
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24 In its Publication 60 (ICRP 1991), ICRP outlined that new information on the risk of
25 radiation-induced cancer in human populations had emerged since 1977 as well as new data in
26 laboratory animals and cultured cells experiments, leading to the need for a reassessment of
27 Publication 26 (ICRP 1977) estimates of the probability of the carcinogenic fatal effects of
28 radiation. In addition to nominal estimates of fatal cancer, the detriment calculated in
29 Publication 60 included also additional components in order to take into account cancer
30 lethality and morbidity resulting from induced non-lethal cancer. ICRP introduced the concept
31 of risk tolerability and proposed new dose limits based on this concept and detriment
32 calculations.
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39 Most recently, in its Publication 103 (ICRP 2007), ICRP provided a new methodology for
40 the calculation of the detriment based on cancer incidence. While the methods used were
41 broadly similar to those used in Publication 60 (ICRP 1991), modifications were made in
42 several aspects of the computations. Annex A of ICRP Publication 103 describes detriment
43 calculation methodology, but not to a degree with which calculations can be fully reproduced.
44 The next section describes details of the calculation methodology used in ICRP Publication
45 103.
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52 ¹ *‘As the existence of a threshold dose is unknown, it has been assumed that even the smallest doses involve a proportionately*
53 *small risk of induction of malignancies. Also, because of the lack of knowledge of the nature of the dose-effect relationship*
54 *in the induction of malignancies in man particularly at those dose levels which are relevant in radiological protection, the*
55 *Commission sees no practical alternative, for the purposes of radiological protection, to assuming a linear relationship*
56 *between dose and effect, and that doses act cumulatively. The Commission is aware that the assumptions of no threshold*
57 *and of complete additivity of all doses may be incorrect but is satisfied that they are unlikely to lead to the underestimation*
58 *of risks’.*
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3. Steps in the calculation of radiation detriment used in ICRP Publication 103

The calculation of radiation detriment for the ICRP system of radiological protection relies on a sequential procedure presented in Figure 1 (ICRP 1991, ICRP 2007). The first step is the calculation of the nominal risk, which is an estimate of the lifetime risk associated with radiation exposure, averaged over sex, age and region for different types of cancer and combined with the risk of heritable effects. The second step is the calculation of the detriment, which is a global risk indicator based on nominal risk, averaged over sex, and adjusted for lethality, quality of life and years of life lost. This step does not depend on the radiation dose, but reflects aspects of severity inherent to each cancer site.

3.1. Calculation of nominal risks

3.1.1. Health data

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) and population size data by 5-year age categories were obtained from the World Health Organization (WHO) international mortality statistics database (WHO 2003). Incidence rates came from selected Asian (Shanghai in China; Osaka, Hiroshima and Nagasaki in Japan) and Euro-American (Sweden; United Kingdom; the Surveillance, Epidemiology, and End Results (SEER) program of the US National Cancer Institute) populations with long-running cancer registries. 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. 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, all solid cancers, leukaemia excluding chronic lymphocytic leukaemia (CLL), all leukaemia and all cancers combined.

The survival functions were derived from the mortality rates estimated for the four reference populations (*i.e.* Asian males and females, Euro-American males and females).

3.1.2. Lifetime baseline risk

The lifetime baseline risk (LBR) corresponds to the cumulative cancer risk in the absence of radiation exposure. LBR is computed by summing cumulative baseline risks up to the age 90 years (*i.e.* from 0 to 89 years) as follows:

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

where s is the sex, a_{min} is the age at the beginning of risk, a_{max} is the maximum age (*i.e.* 89 years), $m(a, s)$ is the age- and sex-specific baseline cancer incidence rates, and $S(a|a_{min}, s)$ is the survival function.

3.1.3. Risk models for specific cancer sites

For solid cancers, risk models were developed for 10 organs or tissues: oesophagus, stomach, colon, liver, lung, female breast, ovary, bladder, thyroid, and other solid cancers. No specific risk models were derived for brain and salivary glands. The specific models for these 10 solid cancers come from the analyses of solid cancer incidence risk of the Life Span Study (LSS) of Japanese atomic bomb (A-bomb) survivors of Hiroshima and Nagasaki, with follow-up from 1958 through 1998 (Preston et al. 2007). Risk models considered a linear dose response allowing for modifying effects of sex (s), age at exposure (e), and attained age (a). 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). For each cancer site, modelling of the radiation-associated risk has been made using an excess relative risk (ERR) model and/or excess absolute risk (EAR) model with the general forms:

$$\lambda(s, a, e, d) = \lambda_0(s, a, e) [1 + ERR(s, a, e, d)]$$

$$\lambda(s, a, e, d) = \lambda_0(s, a, e) + EAR(s, a, e, d)$$

$$\text{with } ER(s, a, e, d) = \beta d \exp[\alpha_1((e - 30)/10) + \alpha_2 \ln(a/70)]$$

where e is the age at exposure (in years), a is the attained age (in years), d is the dose (in Gy), and λ_0 is the baseline cancer rate. Parameter values for β and α used for the calculation of nominal risks are summarised in Table 1, for ERR and EAR models (ICRP 2007). The modifications of the ERR and the EAR for all solid cancers by age at exposure and attained age are presented in Figure 2 and Figure 3, respectively. The minimum latency period (the shortest time where a specified radiation-induced tumour is known or believed to occur after exposure) was assumed to be five years for solid cancers.

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3 For leukaemia, the LSS incidence data were used, with a follow-up from 1950 through
4 1998 and DS02. The risk estimates were based on an EAR model similar to that derived by
5 Preston et al. (1994), with a linear-quadratic dose response that allows for effect modification
6 by sex, age at exposure, and time since exposure. The ERR estimate was computed from the
7 leukaemia EAR model and from the LSS background rate, taking into account sex, age at
8 exposure and attained age. However, there is no published information about these models
9 and further details are not available. Leukaemia excess risk was estimated for all leukaemia
10 cases (including chronic lymphocytic leukaemia, CLL), but was applied to the Asian and
11 Euro-American populations using non-CLL leukaemia rates. The minimum latency period for
12 leukaemia was considered to be two years.
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20 3.1.4. Lifetime excess risk

21 Several methods can be used to calculate the lifetime risk of developing (or dying from) a
22 specific disease caused by a radiation exposure. In Publication 103, lifetime risk estimates
23 were calculated using the risk of exposure-induced cancer (REIC) method (Thomas et al.
24 1992).
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29 REIC refers to the cumulated excess disease cases in an exposed population over a follow-
30 up period. In Publication 103, REIC was calculated for 10 solid cancer sites and leukaemia.
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$$32 \text{ REIC}(e, d) = \int_{a=e+L}^{89} [\mu(a|e, d) - \mu(a)] S(a|e, d) da$$

33 where e is the age at exposure (in years), d is the dose (in Gy), a is the attained age (in years),
34 L is the latency period (in years), μ is the annual risk of cancer incidence and $S(a|e, d)$ is the
35 probability of the individual cancer-free surviving to age a (*i.e.* alive and without cancer)
36 given exposure d at age e . The risk models and survival functions were used to compute sex-
37 specific lifetime risk estimates in the Asian and Euro-American composite populations, for
38 each cancer site.
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45 Considered exposure scenario was acute exposure to 0.1 Gy of low-LET radiation. REIC
46 was calculated for each year of age at exposure from 0 to 84 years for the whole population
47 (estimated 85 times) and from 18 to 64 years for workers (estimated 47 times). The risk was
48 cumulated up to the day each individual turns 90 years old, that is over age range 0–89 years
49 (90 years of life) for the whole population and 18–89 years (72 years of life) for workers. The
50 age-averaged lifetime risk was calculated as a weighted mean of the lifetime risk estimated
51 for each age at exposure, the weight being derived from the age distribution of the four
52 reference populations. REIC at 1 Gy was computed as 10 times the REIC at 0.1 Gy.
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3.1.5. *Transfer of risk estimates across populations*

To provide a reasonable basis for transferring risk across populations with different baseline rates, weighted average of the ERR and EAR lifetime risk estimates was used for each cancer site. As indicated in Table 1, 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.

For lung cancer, the ERR model was given a weight of 0.3 because A-bomb survivors data suggested that the EAR was more comparable across sexes than the ERR and that radiation dose and smoking history interacted additively as lung cancer risk factors (Pierce et al. 2003).

For female breast cancer, risks were based solely on an EAR model derived from the LSS incidence data (Preston et al. 2007) because a pooled analysis of radiation effects provided strong evidence against the use of common ERR models (Preston et al. 2002).

For thyroid cancer, risks were based solely on an ERR model derived from the LSS incidence data (Preston et al. 2007), according to an analysis of radiation associated thyroid cancer risks (Ron et al. 1995). This is because variation in screening intensity was thought to have a marked effect on the rate of radiation-associated thyroid cancers, making it problematic to use EAR models.

3.1.6. *Application of a Dose and Dose-Rate Effectiveness Factor*

Experimental studies generally show that fractionation or protraction of dose is associated with reduced risk per unit dose compared to acute high dose of low-LET radiation. For radiological protection purposes, a dose and dose-rate effectiveness factor (DDREF) of 2 was introduced in Publication 60 (ICRP 1991) to be applied to low-dose, continuous or fractionated exposures. The lifetime risk estimates were adjusted downward by a factor of 2, except for leukaemia for which variation in the risk per unit dose was built into the linear-quadratic model. The same DDREF was applied for males and females, general population and workers.

3.1.7. *Integration of heritable effects*

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. ICRP risk estimates for all classes of genetic diseases (Mendelian diseases, chronic diseases and congenital abnormalities) were derived from the UNSCEAR 2001 Report (UNSCEAR 2001),

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3 based mainly on results from animal experiments (in rodents). Risks were expressed as the
4 predicted number of additional cases (*i.e.* over the baseline) of different classes of genetic
5 disease per million live births per Gy for a population exposed to low-LET, low-dose or
6 chronic irradiation, generation after generation. In ICRP Publication 103, risk estimates were
7 based on results observed on the first two generations (ICRP 2007). The risk of heritable
8 effects in the general population associated with gonadal dose was estimated to be around 20
9 cases per 10,000 persons per Gy. The risk in workers was estimated to be 60% of that for the
10 general population, leading to an estimated nominal risk of 12 per 10,000 per Gy.
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18 *3.1.8. Final calculation of nominal risks*

19 The nominal risk coefficients R are presented in Table 2, for the general population and for
20 workers of both sexes (ICRP 2007). The nominal risk estimate for bone cancer was taken
21 from Publication 60 (ICRP 1991) because data sources were extremely limited and the LSS
22 provided no usable data to derive a specific risk model. Regarding non-melanoma skin cancer,
23 the nominal risk estimate was taken from Publication 59 (ICRP 1992) because models derived
24 from LSS were judged to be not adequate for a general population due to differences between
25 countries in risk related to skin pigmentation. The risks of skin cancer have been estimated
26 using a constant relative risk model, with no modifying effects of age or time since exposure,
27 using both mortality and incidence data, based on the epidemiological and experimental
28 results published before 1990.
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35 As shown in Table 2, the global nominal risk coefficient is 1715 cancer cases per 10,000
36 persons per Gy in the general population and 1179 cancer cases per 10,000 persons per Gy in
37 workers.
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42 **3.2. Calculation of radiation detriment**

43 *3.2.1. Adjustment for lethality*

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45 Nominal risk coefficients based on the excess incidence of cancer were converted to fatal
46 cancer risks by multiplying by their lethality fractions (factor k in Table 2). The lethality
47 fraction has been derived as judgment-based values reflecting the impact of medical treatment
48 for some types of cancer, based on the analysis of 5-year survival rates by cancer site for the
49 1980–1985 and 1950–1970 periods (US DHHS 1989), which were obtained from the SEER
50 program providing information on cancer statistics in the US population. The same lethality
51 fraction values were applied to males and females, general population and workers. As shown
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3 in Table 2, highly lethal cancers received a relatively greater weight (*e.g.* liver or lung cancer)
4 than those that seldom cause death (*e.g.* skin or thyroid cancer).
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7 8 3.2.2. *Adjustment for quality of life*

9 Because cancer survivors generally experience adverse effects on their quality of life,
10 ICRP judged that cancers should be weighted not only for lethality, but also for pain,
11 suffering and any adverse effects of cancer treatment. To achieve this, a factor termed q_{min}
12 was applied to the non-lethal fractions of cancers to produce an adjusted lethality fraction
13 termed q , which is defined by:
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$$16 \quad q = (1 - q_{min}) \times k + q_{min}$$

17 where k is the lethality fraction and q_{min} is the minimum weight for non-fatal cancers
18 (judgment-based values). Table 2 presents the values of q and q_{min} . q_{min} was set equal to 0.1
19 for all cancer sites, except for thyroid (0.2) and skin (0). No q_{min} adjustment was used for skin
20 cancer because radiogenic skin cancers are non-melanoma skin cancers and are almost
21 exclusively of the basal cell type which is usually associated with very little pain, suffering or
22 treatment sequelae. The same values of q_{min} and thus q were applied to males and females,
23 general population and workers. The q adjustment has an impact upon detriment calculations
24 in proportion to the fraction of cancers that are non-lethal. Accordingly, highly lethal cancers
25 such as lung and stomach cancer are little affected by q_{min} , whereas relatively non-lethal
26 cancers such as breast or thyroid are much affected.
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37 3.2.3. *Adjustment for the duration of life lost*

38 The distribution of age at diagnosis differs among cancer types. For each type of cancer,
39 the average number of years of life lost was computed for each sex in each composite
40 population as the average over ages at exposure and subsequent attained ages of the residual
41 lifetime. The relative duration of life lost, factor l in Table 2, was calculated with the average
42 number of years of life lost for the specific cancer dividing by the average number of years of
43 life lost for all cancers which was equal to 15 years in Publication 60 (ICRP 1991). Factor l
44 reflects the relative cancer free life lost varying from <1 for cancers occurring late in life (*e.g.*
45 0.71 for bladder cancer and 0.80 for lung cancer in the general population) to >1 for cancers
46 occurring early in life (*e.g.* 1.63 for leukaemia and 1.29 for thyroid or breast cancer in the
47 general population). For bone and skin cancers, the years of life lost cannot be obtained in the
48 same way and therefore were arbitrarily set at the average years of life lost for all cancers (*i.e.*
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3 equal to 1). For gonads, a number of 20 years of life lost was assigned for severe genetic
4 disorders (*i.e.* a factor l of 1.32).
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7 8 3.2.4. Final calculation of radiation detriment

9 Based on the nominal risk coefficient R , the lethality fraction k , the quality of life factor q
10 and the relative life lost factor l , radiation detriment D associated with each type of cancer
11 was calculated as follows:
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$$14 \quad D = [(R \times k) + (R \times (1 - k) \times q)] \times l$$

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16 The global radiation detriment was calculated as the unweighted sum of the 14 site-specific
17 detriments. The detriment D is expressed in terms of numbers of cases per 10,000 persons per
18 Sv of low dose radiation. As shown in Table 2, the global radiation detriment is 574 cases per
19 10,000 persons per Sv in the general population and 422 cases per 10,000 persons per Sv in
20 workers. Lung cancer is the major contributor to the global detriment for workers (relative
21 detriment of 0.286), whereas it is the other solid cancers in the general population (relative
22 detriment of 0.198).
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27 The site-specific relative radiation detriments for the whole population form the basis of
28 the tissue weighting factors w_T .
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32 4. Discussion and perspectives

33 Radiation detriment is an indicator used to quantify the harmful effects of low-dose
34 exposure. Based on scientific evidence, it quantifies the risk of stochastic effects of low-level
35 radiation exposure, taking into account variability among sexes, ages and populations.
36 Resultant nominal risk coefficients, reflecting the radiation induced lifetime risk for more than
37 10 cancer sites, are adjusted for the severity of the disease in terms of lethality, quality of life
38 and years of life lost. Total detriment is the sum of the detriment for each part of the body
39 (tissues and/or organs) and of potential heritable effects.
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45 The current scheme of detriment calculation is carefully designed to quantify and integrate
46 key aspects of health effects related to low-dose radiation exposure. This scheme has been
47 updated several times in the past (ICRP 1977, ICRP 1991, ICRP 2007) to reflect the evolution
48 of scientific knowledge and integrate new findings. It is expected to evolve in the future in
49 accordance with changes in baseline cancer incidence rates, improvement in cancer treatment
50 and progress in scientific understanding of radiation-induced health effects.
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55 In Publication 103 (ICRP 2007), baseline rates correspond to the period 1993–1997.
56 However cancer incidence rates and all-cause mortality rates have changed significantly since
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3 then, due to changes in lifestyle, advances in diagnostic methods and improvement in cancer
4 treatment, especially for certain cancer types such as thyroid cancer or breast cancer.
5 Updating these reference rates will provide a more realistic basis for the system of
6 radiological protection in the future (Breckow et al. 2018). Furthermore, incidence and
7 mortality vary considerably around the world reflecting genetic difference and differences in
8 lifestyle and healthcare provision. Integration of other reference populations in addition to
9 those currently considered could provide an improved representation of the world population.
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14 In Publication 103 (ICRP 2007), radiation-associated cancer risk models were derived
15 from the LSS for 11 organs or tissues, based on a follow-up from 1958 through 1998 (Preston
16 et al. 2007). Since then, new models with longer duration of follow-up have been published
17 and could be used to update risk models. Risk models derived from studies other than the LSS
18 (*e.g.* Chernobyl clean-up workers, nuclear workers (including Mayak workers) and residents
19 along the Techa river, *etc.*) should be considered, based on a detailed analysis of their
20 respective limitations and advantages, and discussion of the consistency of their results with
21 those from the LSS. For bone marrow, it would be desirable to extend this category to
22 multiple myeloma, because the only hematologic malignancies currently included are non-
23 CLL leukaemia. For bone cancer and non-melanoma skin cancer, nominal risk estimates
24 come from Publications 60 and 59 (ICRP 1991, ICRP 1992), respectively. It would be
25 worthwhile to investigate if specific risk models are available for these cancer types.
26 Furthermore, it is desirable to explore the possibility of developing risk models for the brain
27 and salivary glands, because no specific risk models are considered for these two organs in
28 the current procedure for detriment calculation, whereas values of tissue weighting factors are
29 assigned specifically to them.
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40 The construction of the radiation detriment relies on the LNT model, which has been a
41 basis of radiation protection since Publication 9 (ICRP 1966). Relevance of this model was
42 reinforced by the recent epidemiological literature (NCRP 2018). A DDREF of 2 is used in
43 the calculation of the detriment. The validity of this factor is under examination with
44 reference to current biological and epidemiological findings under the ICRP Task Group 91
45 on radiation risk inference at low-dose and low-dose rate exposure for radiological protection
46 purposes (Rühm et al. 2015, Rühm et al. 2016, Rühm et al. 2017, Shore et al. 2017, Tran et al.
47 2017, Rühm et al. 2018, Wakeford et al. 2019). It is also desirable to validate the current
48 scheme of risk transfer between populations, which is a weighted average between an ERR
49 and an EAR transfer, based on the recent epidemiological data.
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3 Consideration of possible heritable effects is based on the estimate of UNSCEAR 2001
4 report (UNSCEAR 2001). Scientific developments have been made since then, and an up-to-
5 date review of recent findings is desirable.
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8 In the current calculation scheme of detriment calculation, cancer lethality fractions are
9 derived from US data in the 1980s (US DHHS 1989), and are applied without distinction
10 between males and females, general population and workers. The same applies to relative
11 duration of life lost, which was derived in from the data of 1980s (ICRP 1991). More recent
12 data are available today and they will provide a basis for more realistic estimates for each
13 cancer site, with due consideration of variation with age and sex and between populations.
14 Moreover, adjustment for quality of life is judgment-based in the current methodology, but
15 there are attempts to determine the weights in more reasonable way. For example, elaborated
16 approaches have been developed to obtain parameter values for disability-adjusted life years
17 (DALY) (Chen et al. 2015, Shimada & Kai 2015). A review of these methods and available
18 data could be considered, taking into account variation with cancer type, age, sex and
19 population, to improve the assessment of radiation detriment.
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27 To conclude, the global radiation detriment in Publication 103 is 574 cases per 10,000
28 persons per Sv in the general population and 422 cases per 10,000 persons per Sv in workers
29 (ICRP 2007). Detriment is constructed as an integrated indicator of the stochastic risks and
30 health impact associated with low dose radiation exposure, which constitutes an easy to use
31 and robust parameter for the practical application of radiation protection. Nevertheless,
32 calculation of detriment consists of many steps, in which a wide range of information is
33 processed, including risk models and health statistics along with other parameters related to
34 cancer severity. Over time, the methodology has become increasingly elaborated, and it
35 appears more and more important to accurately document and publish the calculation
36 procedure for ensuring transparency and traceability.
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References

- Baselet B, Rombouts C, Benotmane A M, Baatout S, Aerts A 2016 Cardiovascular diseases related to ionizing radiation: The risk of low-dose exposure (Review). *Int J Mol Med.* **38** (6) 1623–41
- Breckow J, Emami S, Amalhaf S, Beshgard A, Buermeyer J, Spruck K 2018 Impact of updating the non-radiation parameters in the ICRP 103 detriment model. *Radiat Environ Biophys.* **57** (2) 89–98
- Chen A, Jacobsen KH, Deshmukh AA, Cantor SB 2015 The evolution of the disability-adjusted life year (DALY). *Socioecon Plann Sci.* **49** (C) 10–5
- Hamada N, Fujimichi Y 2014 Classification of radiation effects for dose limitation purposes: history, current situation and future prospects. *J Radiat Res.* **55** (4) 629–40
- ICRP 1959 Recommendations of the International Commission on Radiological Protection. ICRP Publication 1
- ICRP 1966 Recommendations of the International Commission on Radiological Protection. ICRP Publication 9
- ICRP 1977 Recommendations of the International Commission on Radiological Protection. ICRP Publication 26 *Ann. ICRP* **1** (3)
- ICRP 1991 The 1990 Recommendations of the International Commission on Radiological Protection. ICRP Publication 60 *Ann. ICRP* **21** (1–3)
- ICRP 1992 The Biological Basis for Dose Limitation in the Skin. ICRP Publication 59 *Ann. ICRP* **22** (2)
- ICRP 2007 The 2007 Recommendations of the International Commission on Radiological Protection. ICRP Publication 103 *Ann. ICRP* **37** (2–4)
- ICRP 2012 ICRP Statement on Tissue Reactions / Early and Late Effects of Radiation in Normal Tissues and Organs – Threshold Doses for Tissue Reactions in a Radiation Protection Context. ICRP Publication 118 *Ann. ICRP* **41** (1–2)
- Kreuzer M, Auvinen A, Cardis E, Hall J, Jourdain JR, Laurier D, Little MP, Peters A, Raj K, Russell NS, Tapio S, Zhang W, Gomolka M 2015 Low-dose ionizing radiation and cardiovascular diseases--Strategies for molecular epidemiological studies in Europe. *Mutat Res Rev Mutat Res.* **764** 90–100
- Little MP, Azizova TV, Bazyka D, Bouffler SD, Cardis E, Chekin S, Chumak VV, Cucinotta FA, de Vathaire F, Hall P, Harrison JD, Hildebrandt G, Ivanov V, Kashcheev VV, Klymenko SV, Kreuzer M, Laurent O, Ozasa K, Schneider T, Tapio S, Taylor AM, Tzoulaki I, Vandoolaeghe WL, Wakeford R, Zablotska LB, Zhang W, Lipshultz SE 2012

- 1
2
3 Systematic review and meta-analysis of circulatory disease from exposure to low-level
4 ionizing radiation and estimates of potential population mortality risks. *Environ Health*
5 *Perspect.* **120** (11) 1503–11
6
7 NCRP 2016 Guidance on Radiation Dose Limits for the Lens of the Eye. *NCRP Commentary*
8 **No. 26** National Council on Radiation Protection and Measurements, Bethesda: Maryland,
9 USA
10
11 NCRP 2018 Implications of Recent Epidemiologic Studies for the Linear-Nonthreshold
12 Model and Radiation Protection. *NCRP Commentary No. 27* National Council on
13 Radiation Protection and Measurements, Bethesda: Maryland, USA
14
15 Parkin D M, Whelan S L, Ferlay J, Teppo L, and Thomas D B 2002 Cancer Incidence in Five
16 Continents. Volume VIII *IARC Scientific Publications No. 155* Lyon: France
17
18 Pierce D A, Sharp G B, and Mabuchi K 2003 Joint effects of radiation and smoking on lung
19 cancer risk among atomic bomb survivors *Radiat. Res.* **159** (4) 511–20
20
21 Preston D L, Kusumi S, Tomonaga M, Izumi S, Ron E, Kuramoto A, Kamada N, Dohy H,
22 Matsui T, Nonaka H, Thompson D E, Sodak M, and Mabuchi K 1994 Cancer incidence in
23 atomic bomb survivors. Part III. Leukaemia, lymphoma and multiple myeloma, 1950-1987.
24 *Radiat. Res.* **137** (2 Suppl) S68–97
25
26 Preston D L, Mattsson A, Holmberg E, Shore R, Hildreth N G, Boice J D Jr 2002 Radiation
27 effects on breast cancer risk: a pooled analysis of eight cohorts. *Radiat. Res.* **158** (2) 220–
28 35
29
30 Preston D L, Ron E, Tokuoka S, Funamoto S, Nishi N, Soda M, Mabuchi K, and Kodama K
31 2007 Solid cancer incidence in atomic bomb survivors: 1958-1998. *Radiat. Res.* **168** (1) 1–
32 64
33
34 Rühm W, Woloschak GE, Shore RE, Azizova TV, Grosche B, Niwa O, Akiba S, Ono T,
35 Suzuki K, Iwasaki T, Ban N, Kai M, Clement CH, Bouffler S, Toma H, Hamada N 2015
36 Dose and dose-rate effects of ionizing radiation: a discussion in the light of radiological
37 protection. *Radiat Environ Biophys.* **54** (4) 379–401
38
39 Rühm W, Azizova TV, Bouffler SD, Little MP, Shore RE, Walsh L, Woloschak GE 2016
40 Dose-rate effects in radiation biology and radiation protection. *Ann ICRP.* **45** (1_suppl)
41 262–79
42
43 Rühm W, Eidemüller M, Kaiser JC 2017 Biologically-based mechanistic models of radiation-
44 related carcinogenesis applied to epidemiological data. *Int J Radiat Biol.* **93** (10) 1093–117
45
46
47
48
49
50
51
52
53
54
55
56
57
58
59
60

- 1
2
3 Rühm W, Azizova T, Bouffler S, Cullings HM, Grosche B, Little MP, Shore RS, Walsh L,
4 Woloschak GE 2018 Typical doses and dose rates in studies pertinent to radiation risk
5 inference at low doses and low dose rates. *J Radiat Res.* **59** (suppl_2) ii1–10
6
7
8 Ron E, Lubin J H, Shore R E, Mabuchi K, Modan B, Pottern L M, Schneider A B, Tucker M
9 A, Boice J D Jr 1995 Thyroid cancer after exposure to external radiation: a pooled analysis
10 of seven studies. *Radiat. Res.* **141** (1) 259–77
11
12 Shimada K, Kai M 2015 Calculating disability-adjusted life years (DALY) as a measure of
13 excess cancer risk following radiation exposure. *J Radiol Prot.* **35** (4) 763–775
14
15
16 Shore R, Walsh L, Azizova T, Rühm W 2017 Risk of solid cancer in low dose-rate radiation
17 epidemiological studies and the dose-rate effectiveness factor. *Int J Radiat Biol.* **93** (10)
18 1064–78
19
20
21 Thomas D, Darby S, Fagnani F, Hubert P, Vaeth M, Weiss K 1992 Definition and estimation
22 of lifetime detriment from radiation exposures: principles and methods. *Health Phys.* **63**
23 (3) 259–72
24
25
26 Tran V, Little MP 2017 Dose and dose rate extrapolation factors for malignant and non-
27 malignant health endpoints after exposure to gamma and neutron radiation. *Radiat Environ*
28 *Biophys.* **56** (4) 299–328
29
30
31 UNSCEAR 2001 Heritable Effects of Radiation, United Nations Scientific Committee on the
32 Effects of Atomic Radiation 2001 Report to the General Assembly with Scientific Annex.
33 New York: United Nations
34
35
36 US DHHS 1989 Cancer Statistics Review 1973-86 including a report on the status of cancer
37 control. *NIH Publication No. 89-2789* US Department of Health and Human Services,
38 PHS, NIH, NCI, Bethesda: Maryland, USA
39
40
41 Wakeford R, Azizova T, Dörr W, Garnier-Laplace J, Hauptmann M, Ozasa K, Rajaraman P,
42 Sakai K, Salomaa S, Sokolnikov M, Stram D, Sun Q, Wojcik A, Woloschak G, Bouffler S,
43 Grosche B, Kai M, Little MP, Shore RE, Walsh L, Rühm W 2019 The Dose and Dose-
44 Rate Effectiveness Factor (DDREF). *Health Phys.* **116** (1) 96–9
45
46
47 WHO 2003 World Health Organization Mortality Database, Health statistics and information
48 systems. Data file downloaded April 22, 2003:
49 http://www.who.int/healthinfo/mortality_data/en/
50
51
52
53
54
55
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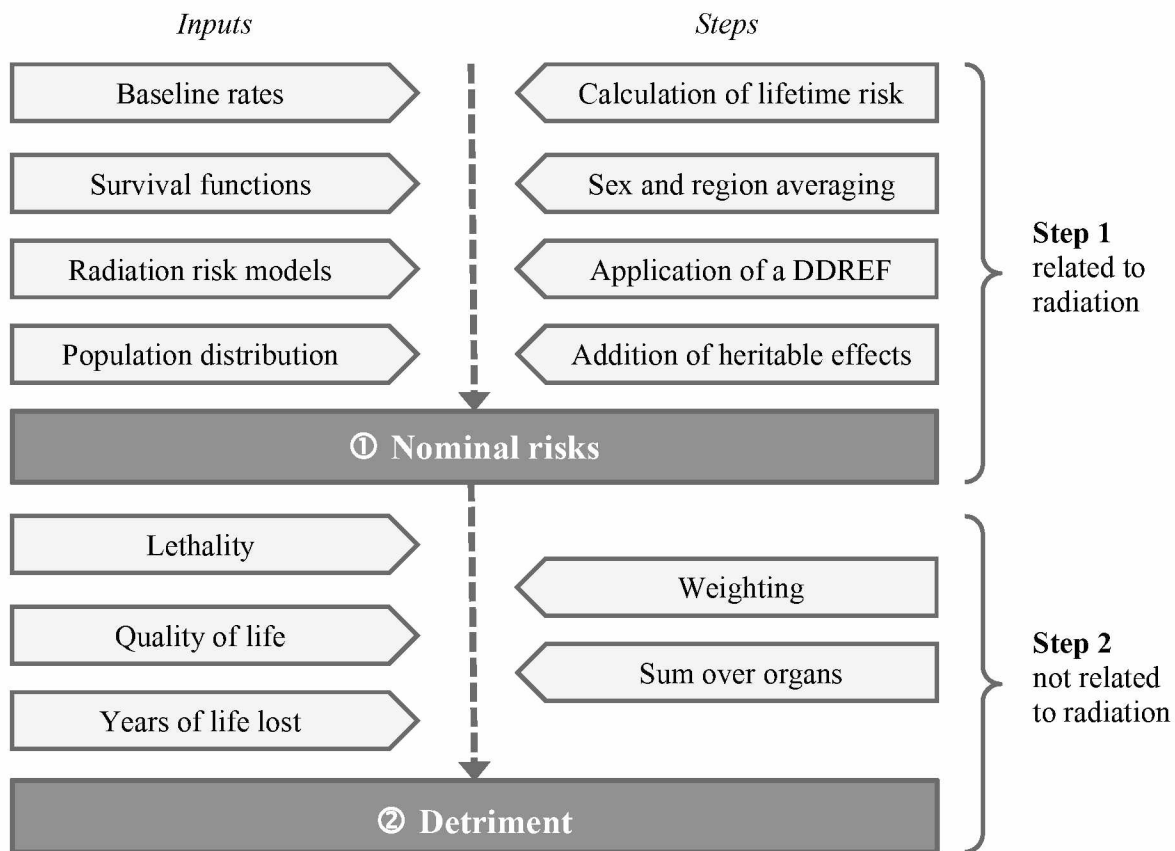


Figure 1. Steps in the calculation of radiation detriment in Publication 103 (ICRP 2007)

Table 1. Linear risk models and coefficients in the current incidence-based ERR and EAR models (from Publication 103, ICRP 2007)

Cancer site	Model type EAR/ERR	Sex	EAR model			ERR model		
			Excess deaths per 10,000 persons per year per Gy at age 70 for exposure at age 30 (β)	Age at exposure: % change in EAR per decade increase (α_1)	Power of attained age by which the EAR varies (α_2)	ERR per Gy at age 70 for exposure at age 30 (β)	Age at exposure: % change in ERR per decade increase (α_1)	Power of attained age by which the ERR varies (α_2)
All solid	50%/50%	M	43.20	-24%	2.38	0.35	-17%	-1.65
		F	59.83			0.58		
Oesophagus	50%/50%	M	0.48	64%	2.38	0.40	-17%	-1.65
		F	0.66			0.65		
Stomach	50%/50%	M	6.63	-24%	2.38	0.23	-17%	-1.65
		F	9.18			0.38		
Colon	50%/50%	M	5.76	-24%	2.38	0.68	-17%	-1.65
		F	2.40			0.33		
Liver	50%/50%	M	4.18	-24%	2.38	0.25	-17%	-1.65
		F	1.30			0.40		
Lung	70%/30%	M	6.47	1%	4.25	0.29	+17%	-1.65
		F	8.97			1.36		
Breast	100%/0%	F	10.9	-39%	3.5 / 1.0 *	–	–	–
Ovary	50%/50%	F	1.47	-24%	2.38	0.32	-17%	-1.65
Bladder	50%/50%	M	2.00	-11%	6.39	0.67	-17%	-1.65
		F	2.77			1.10		
Thyroid	0%/100%	M	–	–	–	0.53	-56%	0.00
		F	–	–	–	1.05		
Other	50%/50%	M	7.55	-24%	2.38	0.22	-34%	-1.65
		F	10.45			0.17		

* The left term is the effect for age before 50 and the right term is the effect for age greater than 50.

Table 2. Steps in the construction of the radiation detriment, from nominal risk coefficient to detriment for the general population and for workers (from Publication 103, ICRP 2007)

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	Detriment D^*	Relative detriment
General population (ages 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	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	42	0.67	0.1	0.702	1.63	61.5	0.107
Other solid	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					574.2	1.000
Workers (ages 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	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	23	0.67	0.1	0.702	1.17	23.9	0.057
Other solid	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					422.0	1.000

* R is expressed in cases per 10,000 persons per Gy and D in cases per 10,000 persons per Sv.

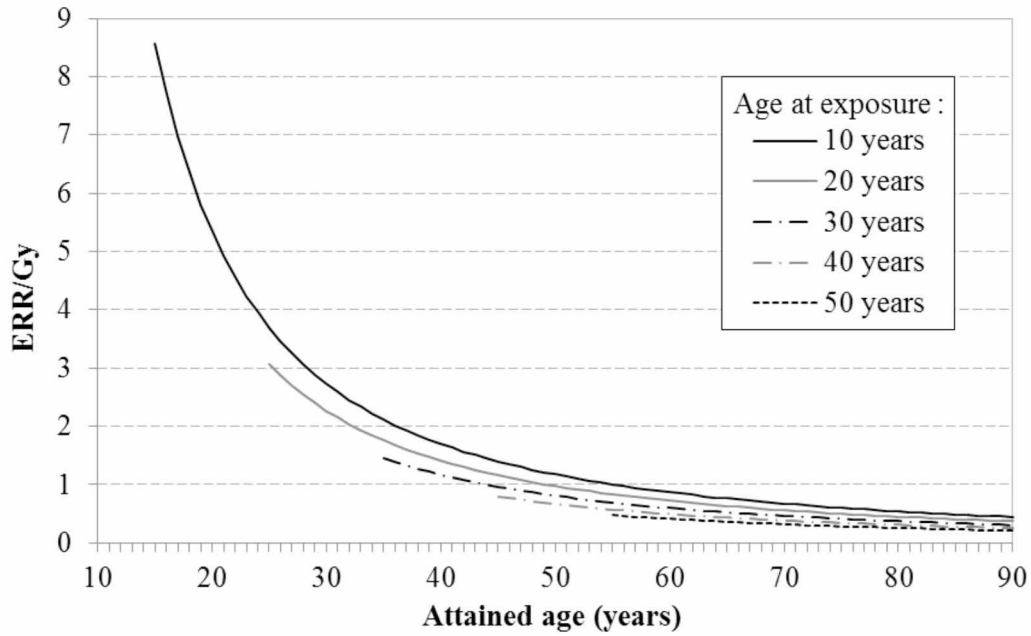


Figure 2. Modification of the ERR for all solid cancers by age at exposure and attained age

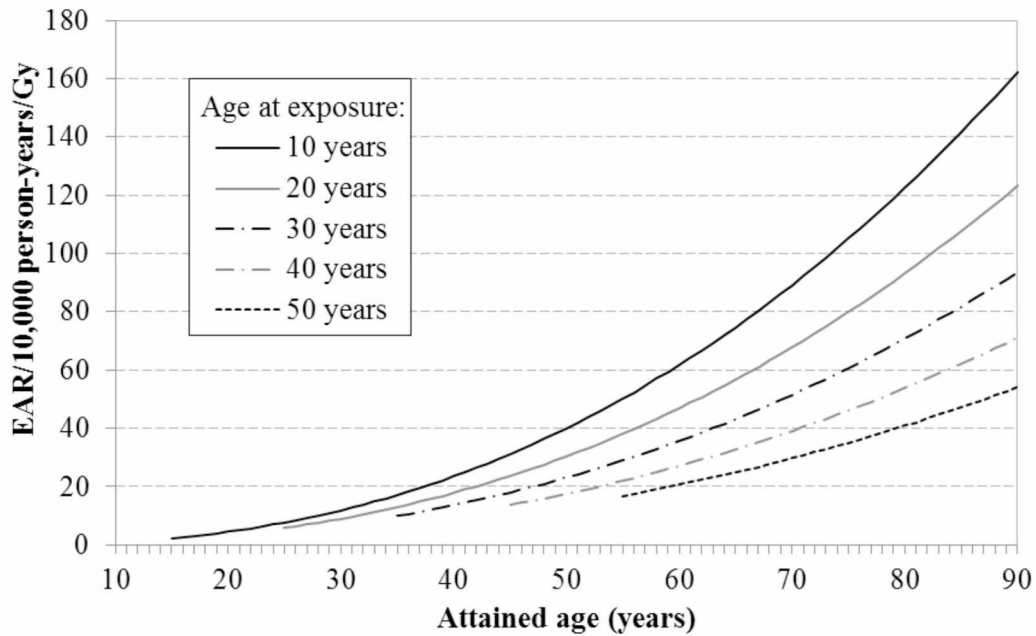


Figure 3. Modification of the EAR for all solid cancers by age at exposure and attained age