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## PAPER

# Effective doses and risks from medical diagnostic x-ray examinations for male and female patients from childhood to old age

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Supplementary material for this article is available [online](#)

## Abstract

The consideration of risks from medical diagnostic x-ray examinations and their justification commonly relies on estimates of effective dose, although the quantity is actually a health-detriment-weighted summation of organ/tissue-absorbed doses rather than a measure of risk. In its 2007 Recommendations, the International Commission on Radiological Protection (ICRP) defines effective dose in relation to a nominal value of stochastic detriment following low-level exposure of  $5.7 \times 10^{-2} \text{ Sv}^{-1}$ , as an average over both sexes, all ages, and two fixed composite populations (Asian and Euro-American). Effective dose represents the overall (whole-body) dose received by a person from a particular exposure, which can be used for the purposes of radiological protection as set out by ICRP, but it does not provide a measure that is specific to the characteristics of the exposed individual. However, the cancer incidence risk models used by ICRP can be used to provide estimates of risk separately for males and females, as a function of age-at-exposure, and for the two composite populations. Here, these organ/tissue-specific risk models are applied to estimates of organ/tissue-specific absorbed doses from a range of diagnostic procedures to derive lifetime excess cancer incidence risk estimates; the degree of heterogeneity in the distribution of absorbed doses between organs/tissues will depend on the procedure. Depending on the organs/tissues exposed, risks are generally higher in females and notably higher for younger ages-at-exposure. Comparing lifetime cancer incidence risks per Sv effective dose from the different procedures shows that overall risks are higher by about a factor of two to three for the youngest age-at-exposure group, 0–9 yr, than for 30–39 yr adults, and lower by a similar factor for an age-at-exposure of 60–69 yr. Taking into account these differences in risk per Sv, and noting the substantial uncertainties associated with risk estimates, effective dose as currently formulated provides a reasonable basis for assessing the potential risks from medical diagnostic examinations.

## 1. Introduction

The UK Ionising Radiation (Medical Exposure) Regulations (DHSC 2018), and equivalent legislation in other countries, require the justification of all medical exposures to ionising radiation, taking account of the characteristics of the individual patient, to demonstrate that the clinical benefits of exposure outweigh any risks. Effective dose,  $E$ , is commonly and increasingly used in medical practice as a measure of risk to health from radiation exposure, although it was not designed for this application. The primary purpose of effective dose, as defined by the International Commission on Radiological Protection (ICRP 2007), has been the

quantification of radiation exposures of workers and members of the public in order to demonstrate compliance with dose limits and to optimise protection against stochastic health effects following low-level exposure (i.e. low doses or low dose-rates), principally cancer (ICRP 1991, 2007, 2021, 2022). For this purpose, the requirement has been for a single quantity appropriately representing the (weighted) summation of organ/tissue-specific absorbed doses from each source of radiation, which is applicable to all workers or all members of the public.

In its application in medicine in the justification of procedures for individual patients, and the understanding and communication of risks, the use of effective dose has two main deficiencies.

The first shortcoming is that the calculation of effective dose involves sex-averaging of organ/tissue doses calculated using male and female reference phantoms of the human body. The use of reference phantoms is a positive step in the provision by ICRP of reference effective dose coefficients, but these calculations do not take account of differences between individuals in body and organ/tissue masses and dimensions. Sex-averaging of organ/tissue doses is also clearly not intended to provide estimates of dose to specific individuals. Second, effective dose is calculated using tissue weighting factors based on sex- and age-at-exposure-averaged risks of cancer and heritable effects. These stochastic risks, as well as being sex- and age-averaged, are calculated using cancer incidence risk estimates that are averaged over two fixed composite (Asian and Euro-American) populations, as representing the global population. The values obtained do not apply to any one particular population group and also, importantly, do not show recognised differences in risks between males and females and by age-at-exposure (ICRP 2021).

This paper applies the cancer risk models used by ICRP (2007) to calculate stochastic detriment values following low-level exposure to radiation, but presents lifetime excess cancer incidence predictions to show the differences in risk estimates between males and females, between children and adults as a function of age-at-exposure, and between the two composite populations. Cancer risks are estimated for a range of diagnostic x-ray examinations and expressed as lifetime excess cancer incidence risk per effective dose for each procedure, using organ/tissue-specific risk models and organ/tissue-specific absorbed doses. Diagnostic procedures target specific organs/tissues and hence result in a highly heterogeneous distribution of absorbed doses between organs/tissues. The total risk resulting from the exposure depends on the sensitivity of the organs/tissues receiving the highest doses, and this sensitivity will vary with sex, age-at-exposure and population group.

## 2. Sex- and age-specific cancer risks and detriment

### 2.1. ICRP risk models

The cancer risk models developed by ICRP for application in its 2007 Recommendations are based primarily on cancer incidence data from the life span study (LSS) of the Japanese atomic bomb survivors, with follow up from 1958 to 1998 for solid cancers and from 1950 to 2000 for leukaemia (ICRP 2007, 2022, Cléro *et al* 2019, 2022, Ban *et al* 2022). Models based on the excess absolute risk (EAR, the additional risk due to the exposure) and excess relative risk (ERR, the proportional increase in risk compared to the background risk) were developed that related EAR and ERR to organ/tissue-specific absorbed dose using the DS02 dosimetry system for the following ten specific organs/tissues: female breast, lung, stomach, colon, red bone marrow (RBM), bladder, liver, thyroid, oesophagus and ovary. The nominal risk estimates from the 1990 Recommendations (ICRP 1991) were used for bone and (non-melanoma) skin cancers because LSS data for these cancers offered only a limited opportunity for the derivation of risk models (ICRP 2007, 2022, Cléro *et al* 2019, Ban *et al* 2022). EAR and ERR models were developed from the LSS incidence data for the remaining solid cancers considered as a single group; risk estimates were not derived for the lymphomas or multiple myeloma, for which evidence of any radiation-related risk is limited. For solid cancers, the models are linear, no-threshold dose-responses, allowing for risk modification by sex, age-at-exposure and attained age. For leukaemia following irradiation of RBM, ICRP (2007) used an EAR model with an explicit linear-quadratic dose-response that allowed for the modifying effects of sex, age-at-exposure and time-since-exposure, similar to that described by Preston *et al* (1994); ERR estimates were derived from this EAR model using the LSS background leukaemia incidence rates (Cléro *et al* 2019, 2022, ICRP 2022, Ban *et al* 2022). Minimum latency periods were set at two years for leukaemia and five years for solid cancers (Cléro *et al* 2019) (although it later transpired that the latency period used for leukaemia was actually five years (Cléro *et al* 2022)).

EAR and ERR models lead to similar predictions of the excess risks of cancer in the population that was used to derive the risk models, but they can lead to markedly different excess risk estimates when applied to other populations with different baseline cancer rates. ICRP (2007) used baseline cancer incidence rates for two fixed composite populations (based on populations with well-established cancer registries) defined by averaging, respectively, Asian (Shanghai, Osaka, Hiroshima and Nagasaki) and Euro-American (Sweden, UK

and USA) populations for specific periods; leukaemia baseline incidence rates excluded chronic lymphocytic leukaemia (CLL, now considered to be a form of non-Hodgkin lymphoma) (Cléro *et al* 2019). Age-averaged lifetime cancer incidence risks were calculated for each composite population and sex by applying the EAR and ERR models to the databases using a single dose of 100 mGy of low-LET radiation delivered to each organ/tissue, and a weighted average of the model estimates was then computed. The relative weights given to the EAR and ERR models varied for the different cancer sites, based on judgements concerning their relative applicability for risk transfer, as follows: EAR:ERR of 0.0:1.0 for thyroid and skin, 1.0:0.0 for breast and bone, 0.7:0.3 for lung, and 0.5:0.5 for all other cancers. For solid cancers, ICRP (2007) applied a dose and dose rate effectiveness factor (DDREF) of 2 to reduce risk estimates for normal applications at low doses ( $< \sim 100$  mGy) or low dose rates ( $< \sim 5$  mGy h<sup>-1</sup>)<sup>4</sup> to organs and tissues, but no DDREF was used for leukaemia. The resulting lifetime risk estimates were further averaged over composite population and sex to produce final nominal risk coefficients consisting of sex-, age-, population- and model-averaged (weighted as appropriate) lifetime excess absolute risks of cancer incidence (LEAR, the EAR of cancer incidence over the remaining lifetime) per Gy for the ten sites, and one grouping of sites (other solid), of cancer; nominal risk coefficients were additionally presented for bone and skin cancers (ICRP 2007). Details may be found in Cléro *et al* (2019, 2022), Ban *et al* (2022) and ICRP Publication 152 (ICRP 2022). ICRP (2007) defines 'nominal' in this context as 'sex-averaged and age-at-exposure-averaged lifetime risk estimates for a representative population'.

Detriment (to health) was calculated by ICRP (2007) from these values of the nominal LEAR of cancer incidence per Gy by adjusting for severity of each cancer type, applying weighting factors for lethality, morbidity associated with non-fatal cancers, and years of life lost. As discussed by Cléro *et al* (2019), Ban *et al* (2022) and ICRP (2022), these severity weighting factors were broad evaluations of available data, which involved expert judgement in interpreting limited data for the purpose of these adjustments; for example, lethality fractions were those that had been used for the ICRP 1990 Recommendations (ICRP 1991) and did not vary between the sexes or change when children and the elderly were removed from the age-at-exposure averaging (ICRP 2007). Total cancer detriment was obtained by summing the detriment values for each cancer site. Estimated risks of heritable effects from irradiation of the gonads, derived from animal experiments and knowledge of human genetics, were similarly averaged to derive a nominal value of LEAR per Gy, which was severity weighted to provide a detriment value that was then added to the cancer detriment to provide the overall detriment for stochastic health effects. Tissue weighting factors,  $w_T$ , are simplified values chosen to represent relative detriment from each organ/tissue when averaged over both sexes and all ages. For simplicity, only four different values of  $w_T$  are used: 0.12, 0.06, 0.04 and 0.01, and  $\sum w_T = 1$  when summed over all tissues.

Note that, while nominal risk coefficients are calculated as LEAR per Gy absorbed dose to organs/tissues for the effect resulting from this irradiation, the nominal risk coefficients presented by ICRP (2007) are expressed as LEAR per Sv effective dose by convention as the basis for the use of this protection quantity. Similarly, the detriment is expressed per effective dose. Two overall detriment values were presented in ICRP Publication 103:  $5.7 \times 10^{-2}$  Sv<sup>-1</sup> effective dose for the whole population (both sexes, all ages), and  $4.2 \times 10^{-2}$  Sv<sup>-1</sup> effective dose for a working age population (both sexes and 18–64 yr age-at-exposure). Cancer detriment, for a uniform exposure, dominates at  $5.5 \times 10^{-2}$  Sv<sup>-1</sup> and  $4.1 \times 10^{-2}$  Sv<sup>-1</sup>, respectively.

## 2.2. Application of ICRP models

Wall *et al* (2011) constructed 11 cancer EAR and ERR models from the appropriate LSS cancer incidence data, following the methodology described in ICRP Publication 103 (ICRP 2007) as closely as possible, although later information on this methodology has revealed various aspects of the description provided in Publication 103 that require revision (Cléro *et al* 2019, 2022, ICRP 2022, Ban *et al* 2022). The LEAR of cancer incidence estimates were calculated for each organ/tissue receiving a uniform absorbed dose of 10 mGy of low-LET radiation. Comparison of the results of applying the models of Wall *et al* (2011) with equivalent results presented in Publication 103 show good agreement for most cancer types. However, discrepancies were observed for thyroid cancer (−42%) and leukaemia (+50%), and less so for female breast cancer (−13%), which were largely unexplained (Wall *et al* 2011), although the differences have, at least in part, been clarified by further elucidation of what was done to derive the ICRP models (Cléro *et al* 2019, 2022, ICRP 2022, Ban *et al* 2022). Despite these differences, the overall LEAR of cancer incidence (excluding bone and skin cancers) was calculated by Wall *et al* (2011) as  $665 \times 10^{-4}$  Gy<sup>-1</sup>, (i.e. 665 cases per 10 000 persons per Gy) compared with a value of  $688 \times 10^{-4}$  Gy<sup>-1</sup> presented in Publication 103 (ICRP 2007), results that are within 3% of each other, which is good overall agreement.

<sup>4</sup> A low dose rate for low-LET radiation has been defined by UNSCEAR (2020) as less than 0.1 mGy min<sup>-1</sup> when averaged over approximately one hour, which is effectively the definition adopted in ICRP Publication 147 (ICRP 2021).

**Table 1.** Estimates of lifetime attributable risk (LAR) of cancer incidence per organ/tissue absorbed dose ( $\times 10^{-4} \text{ Gy}^{-1}$ ) from uniform whole-body exposure to low-LET radiation for the ICRP (2007) Euro-American composite population, by organ/tissue, sex and age-at-exposure using ICRP (2007) risk models (adapted, with permission, from ICRP Publication 147 (ICRP 2021), table 2.4). Cancer incidence excludes cancers of the skin and bone. Calculations performed with a uniform whole-body dose of 10 mGy of low-LET radiation.

Organ/tissue	Lifetime attributable risk (LAR) of cancer incidence per organ/tissue absorbed dose ( $\times 10^{-4} \text{ Gy}^{-1}$ ) <sup>a</sup>									
	Age at exposure (years)									
	0–9	10–19	20–29	30–39	40–49	50–59	60–69	70–79	80–89	90–99
<i>Males</i>										
Lung	70	70	70	80	80	80	60	40	20	3
Stomach	100	80	60	40	30	20	10	5	2	0
Colon	160	130	110	80	60	40	20	10	4	0
RBM	130	130	80	70	70	40	30	10	7	2
Bladder	90	80	70	60	50	30	20	10	5	1
Liver	60	50	40	30	20	10	6	3	1	0
Thyroid	40	20	6	3	1	0	0	0	0	0
Oesophagus	10	10	10	10	10	10	10	8	5	1
Other solid	490	320	240	140	90	50	30	10	3	0
<b>All above organs/tissues</b>	<b>1150</b>	<b>880</b>	<b>680</b>	<b>500</b>	<b>400</b>	<b>290</b>	<b>190</b>	<b>100</b>	<b>40</b>	<b>8</b>
<i>Females</i>										
Breast	670	410	250	150	80	40	20	7	2	0
Lung	150	160	170	180	190	190	160	110	50	6
Stomach	170	130	100	70	50	30	20	10	5	0
Colon	80	70	50	40	30	20	10	8	3	0
RBM	50	50	50	40	50	30	20	10	4	1
Bladder	80	70	60	50	40	40	30	20	10	1
Liver	30	20	20	10	9	6	4	2	1	0
Thyroid	190	80	30	10	4	1	0	0	0	0
Oesophagus	10	10	10	10	10	20	20	20	20	3
Ovary	60	40	30	20	20	10	6	3	1	0
Other solid	370	250	170	120	80	50	30	10	5	0
<b>All above organs/tissues</b>	<b>1850</b>	<b>1300</b>	<b>940</b>	<b>710</b>	<b>570</b>	<b>440</b>	<b>320</b>	<b>210</b>	<b>100</b>	<b>10</b>

RBM = Red bone marrow.

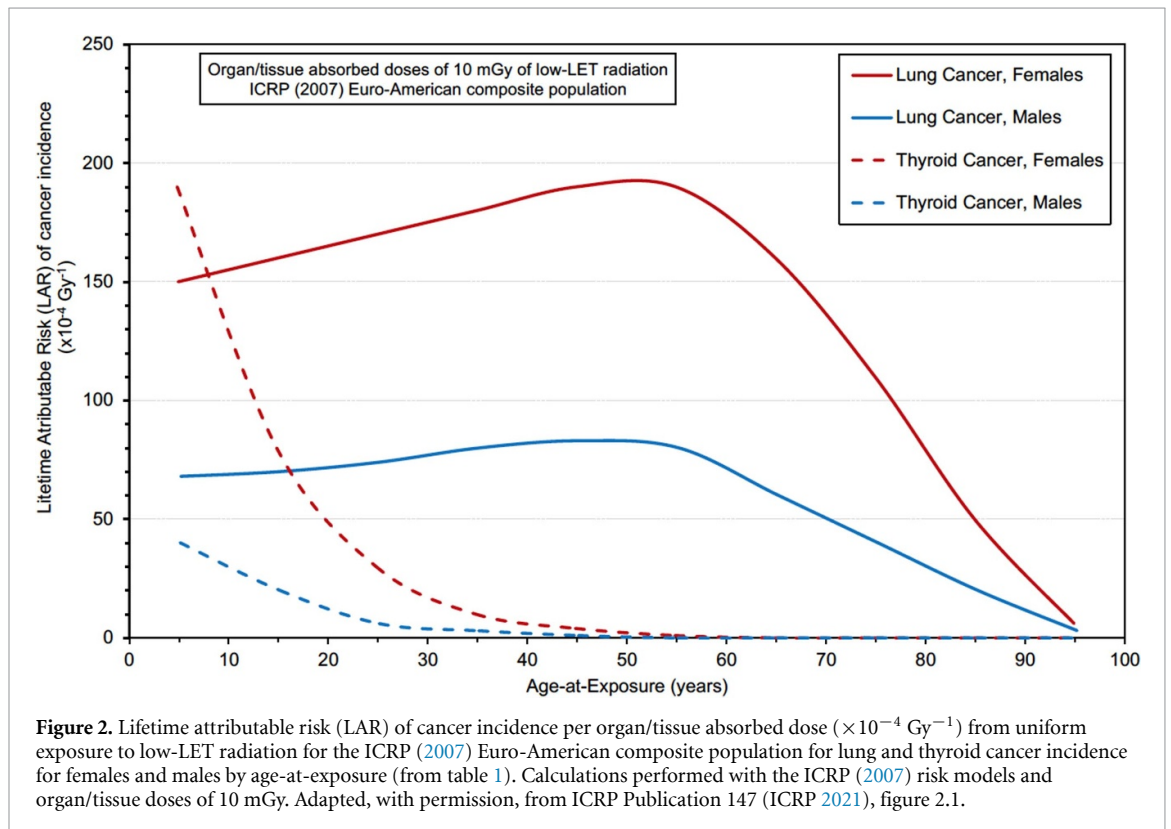
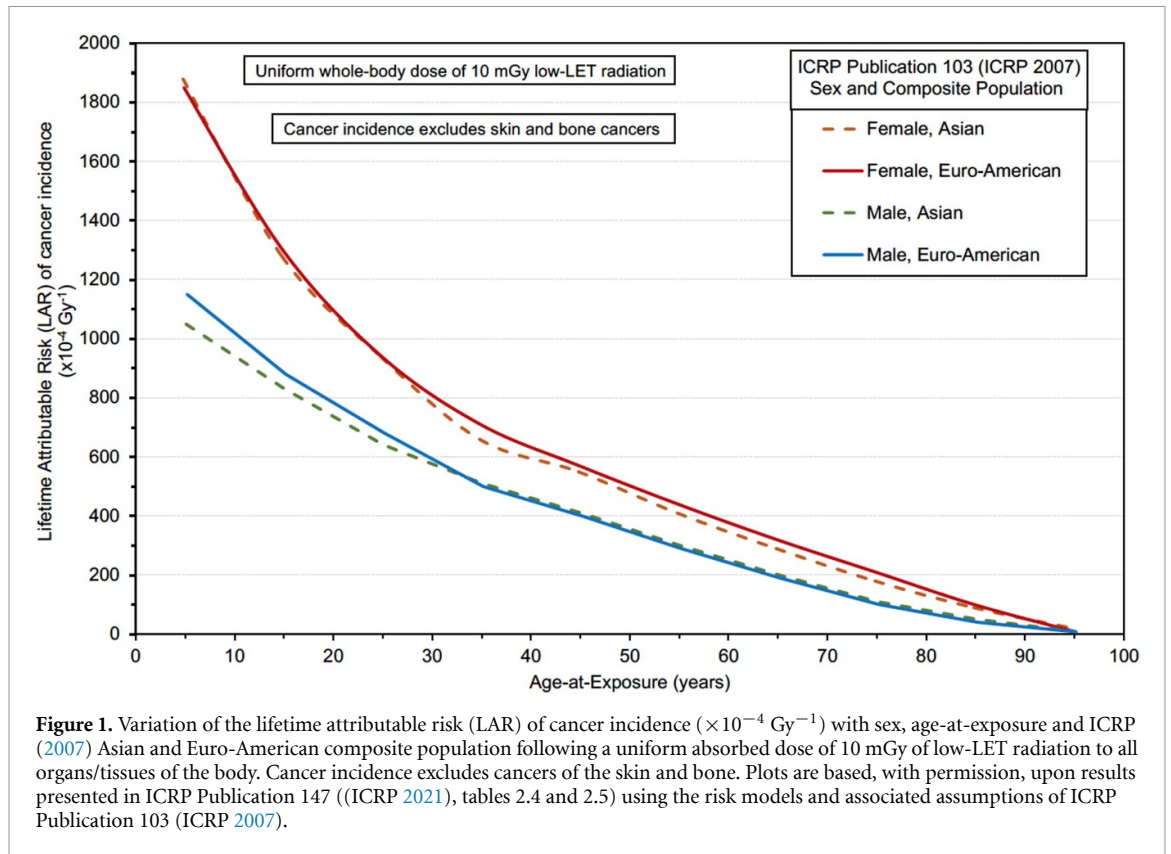
Risks are calculated using EAR and ERR models and applying a DDREF of 2 for all cancer types other than leukaemia, and with ERR/EAR weightings of 1/0 for thyroid, 0.3/0.7 for lung, 0/1 for breast, 0.5/0.5 for all other cancers. Minimum latency periods applied were two years for leukaemia and five years for solid cancers.

<sup>a</sup> cases per 10 000 persons per Gy.

In ICRP Publication 103 (ICRP 2007, Cléro *et al* 2019) LEAR estimates of cancer incidence were calculated using the measure, risk of exposure-induced cancer (REIC) (Thomas *et al* 1992), which was also the measure used by Wall *et al* (2011). ICRP Publication 147 (ICRP 2021) presented results for the LEAR of cancer incidence measured as the lifetime attributable risk (LAR), and LAR is also used in this paper, although the difference between the REIC and LAR estimates are generally small, as noted later.

Table 1 shows results obtained for lifetime EARs of cancer incidence calculated as LAR, considering 11 different cancer types: female breast, lung, stomach, colon, bladder, liver, thyroid, oesophagus, ovary, non-CLL leukaemia, and all other solid cancer sites as a single grouping, excluding skin and bone cancers. The cumulative EAR of cancer incidence per organ/tissue absorbed dose up to an attained age of 100 years was calculated separately for males and females and by category of age-at-exposure (ten age-at-exposure 10 yr groups, from 0–9 yr to 90–99 yr). Calculations were performed for the ICRP Euro-American composite population. The equivalent results using REIC as a measure of LEAR are shown in table S1, and are similar to those shown in table 1.

The LAR values presented in table 1 show, when taking the 30–39 yr age-at-exposure group as a reference, LAR estimates about two to three times higher in the youngest group (0–9 yr at exposure) and about two to three times lower at an age-at-exposure of 60–69 yr. A very similar pattern of results is obtained using the ICRP Asian composite population, as illustrated in figure 1, which shows the variation of the LAR of cancer incidence by sex and age-at-exposure for Euro-American and Asian composite populations.



However, the results show substantial differences between cancer types (table 1), as illustrated in figure 2 for lung and thyroid cancers. While the LAR of cancer incidence values for most cancer types are greatest at younger ages at exposure, this tendency is more pronounced for some (e.g. thyroid cancer) than others (e.g. oesophageal cancer); lung cancer is a notable exception for which risks peak for exposures in middle age (UNSCEAR 2013, Cahoon *et al* 2017). Because of these variations, the contribution of the different cancer

types to overall lifetime risk varies substantially with age-at-exposure as well as between males and females, and the sex-specific differences in LAR estimates are particularly notable for lung and thyroid cancers. Note that variations with age-at-exposure reflect cumulative lifetime risks of cancer incidence, so that reduction of risk with increasing age-at-exposure reflects mainly the reduction in remaining lifetime after exposure during which the risk may be expressed rather than a variation of sensitivity with age-at-exposure, although the latter variation also plays a role, especially in combination with a decrease in risk with time-since-exposure (UNSCEAR 2013).

### 3. Effective dose from diagnostic x-ray examinations

Effective dose, as currently defined, is calculated in reference male and female phantoms of specific ages: newborn, ages 1, 5, 10 and 15 yr, and adult (ICRP 2007, 2020, 2021). Absorbed doses and equivalent doses to organs and tissues are calculated separately for males and females and then sex-averaged for the calculation of effective dose. The final stage in the calculation of effective dose,  $E$ , is the weighted averaging of organ/tissue equivalent doses:  $E = \sum_T w_T H_T$ , where  $H_T$  is the equivalent dose to tissue T and  $w_T$  is the corresponding tissue weighting factor. Effective dose calculated with each of the phantom male and female combinations relates to the two stochastic detriment values of  $574 \times 10^{-4} \text{ Sv}^{-1}$  effective dose for the whole population and  $422 \times 10^{-4} \text{ Sv}^{-1}$  effective dose for a working-age population (age-at-exposure, 18–64 yr).

While effective dose can be used to represent the overall dose received by a particular person from a particular exposure, which can be used for the purposes of radiological protection as intended by ICRP (2007), it clearly does not provide a measure that is specific to that individual, either in terms of total (whole-body) dose or of the related total risk. Nonetheless, effective dose is the reported quantity that allows comparisons of doses from different procedures in different clinical settings. The relationship between effective dose and risks and the quantification of risks using organ/tissue absorbed doses is discussed in other sections of this paper.

Effective dose from medical procedures is calculated using dose coefficients that relate measurable quantities to the protection quantity. ICRP has published dose coefficients for diagnostic procedures in nuclear medicine, but has not yet provided such reference dose coefficients for diagnostic x-ray imaging procedures. However, dose coefficients are available from published studies for the calculation of organ/tissue doses and effective doses from entrance surface air kerma ( $K_e$ ) or kerma-area product ( $P_{KA}$ ) for radiography and fluoroscopy (Jones and Wall 1985, Hart *et al* 1994, Ranniko *et al* 1997, Kramer *et al* 2004), or the dose-length product (DLP) for computed tomography (CT) (Lee *et al* 2011, 2012, Wall *et al* 2011, Ding *et al* 2015, Shrimpton *et al* 2016).

Table 2 provides illustrative examples of effective dose estimates for diagnostic procedures. The highest effective doses for radiographic examinations are in the range of 0.1–1 mSv, while others are substantially lower. For example, table 3 summarises results from a review by Wall *et al* (2011) in which effective dose was calculated using ICRP (2007) methodology, showing that doses from radiographic procedures ranged from 0.1  $\mu\text{Sv}$  (knee, foot) to 0.4 mSv (lumbar spine, thoracic spine). Effective doses from examinations involving fluoroscopy and radiography were in the range of 1–4 mSv.

As shown in table 2, effective doses from CT examinations of adults are estimated as between about 2 mSv (head) to around 20 mSv (chest + abdomen + pelvis, CAP). Differences between countries in values for the same procedure are at least partly due to different methodologies of dose calculation rather than differences in exposures. Shrimpton *et al* (2016) provided a comparison of effective dose estimates from surveys in the UK in 2003 and 2011, as summarised in table 4. The 2003 survey applied ICRP Publication 60 (1991) methodology, whereas the 2011 survey applied ICRP Publication 103 (2007) methodology. While a trend to increased exposures measured as DLP was observed, reflecting the evolution of scanning practices, greater increases in estimates of effective dose for 2011 compared with 2003 were also partly due to increases in effective dose per DLP coefficients. Thus, as shown in table 4, these coefficients were unchanged for some examinations (e.g. head CT) but increased substantially for others (e.g. chest CT). These differences are attributable to changes made by ICRP in the calculation of effective dose, specifically in tissue weighting factors and the introduction of reference male and female dosimetric phantoms (ICRP 2007, 2021, Harrison *et al* 2021). In comparing doses estimated for a particular procedure in different hospitals, it is clearly important to ensure that the same dose coefficients are being used or that differences are recognised. Comparisons have been made more difficult by the absence of a reference set of dose coefficients provided by ICRP. Thus, while ICRP publishes dose coefficients for most situations of exposure of workers and members of the public, and also for the diagnostic use of radiopharmaceuticals, reference coefficients have not yet been provided for medical x-ray examinations. This work is now in progress.

The data presented by Shrimpton *et al* (2016) included effective dose estimates for CT scans of the heads of children, with similar values of around 2 mSv at each age (2.0–2.3 mSv), as for adults (1.8 mSv). Wall *et al*

**Table 2.** Examples of typical effective doses (mSv) for adults in three countries for some common diagnostic x-ray examinations (adapted, with permission, from ICRP Publication 147, ICRP (2021), table 5.1).

Diagnostic procedure	Effective dose (mSv)		
	UK <sup>a</sup>	USA <sup>b</sup>	Russian Federation <sup>c</sup>
<b>Radiography</b>			
Chest PA	0.01	0.03	0.1
Chest Lat	0.04	0.07	0.18
Lumbar spine AP	0.39	2.0	0.6
Lumbar spine Lat	0.21	—	0.6
Abdomen AP	0.43	0.6	1.0
Pelvis AP	0.28	0.4	0.7
<b>Interventional</b>			
Coronary angiography	3.9	15	15
Femoral angiography	2.3	7	5–10
<b>Computed tomography</b>			
CT Head	1.8	1.6	1.8
CT Chest	14	6.1	6.3
CT Abdomen	16	—	9
CT Abdomen + Pelvis	13	7.7	—
CT Chest + Abdomen + Pelvis	19	12	25
<b>Nuclear Medicine</b>			
Bone scan: <sup>99m</sup> Tc	3	4	3
PET whole-body tumour imaging ( <sup>18</sup> F FDG) <sup>d</sup>	7.6	13	5

PA: postero-anterior; AP: antero-posterior; Lat: lateral; PET: positron emission tomography; FDG: fluorodeoxyglucose.

<sup>a</sup> Wall *et al* (2011); Shrimpton *et al* (2016); ARSAC (2020).

<sup>b</sup> Mettler *et al* (2008); Smith-Bindman *et al* (2015); Alessio *et al* (2015); Becker *et al* (2016); Kanal *et al* (2017); NCRP (2019).

<sup>c</sup> Chipiga and Bernhardsson (2016); Vodovatov *et al* (2017); Zvonova *et al* (2015); Balonov *et al* (2018).

<sup>d</sup> Doses are for PET tumour imaging from <sup>18</sup>F only and do not include CT which is frequently performed with PET.

**Table 3.** Typical values of effective dose for adult patients from x-ray examinations involving radiography alone and with fluoroscopy. Adapted from Wall *et al* (2011), tables 7, 11 and 14, by kind permission of UK Health Security Agency.

Effective dose, mSv	X-ray examination
0.0001–0.001	Knee AP, knee lat, foot (dorsi-plantar), foot (oblique)
0.001–0.01	Femur lat, shoulder axial, shoulder AP
0.01–0.1	Femur AP, single hip AP, chest, lat, chest PA, cervical spine lat, cervical spine AP, head lat, head PA, head AP
0.1–1	Both hips AP, pelvis AP, abdomen AP, lumbo-sacral joint lat, lumbar spine lat, lumbar spine AP, thoracic spine lat, thoracic spine AP
1–10	Ba swallow, Ba follow, Ba enema, coronary angiography, femoral angiography, IVU

PA: postero-anterior; AP: antero-posterior; Lat: lateral; Ba: barium; IVU: intravenous urography.

(2011) estimated effective dose for children for CT scans of the chest and head using ICRP Publication 60 (1991) methodology, showing similar values of around 1 mSv for both procedures and all age groups from newborn to adult. In contrast, results for radiographic examinations (chest AP/PA, abdomen AP, pelvis/hips AP) and examinations involving radiography and fluoroscopy (micturating cysto-urethrogram, barium meal, barium swallow) showed substantially lower values for children (newborn to age 15 yr) than for adults (Wall *et al* 2011). In general, dose estimates for children were less than half those for adults and around 10% or less of the adult dose for children aged 5 yr and younger in the case of radiography of the abdomen and pelvis/hips. It was noted that effective doses estimated for persons aged 15 yr were less than half those for adults. Wall *et al* (2011) observed that the extent to which optimisation of patient protection is practiced appears to be very variable around the UK, with specialised paediatric hospitals often achieving far lower doses for children than general hospitals. They concluded that there was particular scope for improvement in the optimisation of doses to children from CT examinations.

The importance of CT in medical diagnosis has continued to grow in recent years, accounting for 62% of the global collective dose received from diagnostic exposures in 2020 (UNSCEAR 2022). Concern has been expressed that substantial numbers of patients are receiving effective doses of greater than 100 mSv as a result of multiple CT scans over short periods (Brambilla *et al* 2020, Rehani *et al* 2020). While these larger cumulative doses apply to a small proportion of overall CT imaging and most patients receiving such doses are in the older age categories, a significant proportion are younger (aged less than 50 yr). Differences



**Table 4.** Typical values of effective dose,  $E$ , estimated for adult and paediatric patients for common CT examinations in the UK.  $E_{60}$ : calculated using ICRP Publication 60 (1991) methodology;  $E_{103}$ : calculated using ICRP Publication 103 (2007) methodology. Adapted, with permission, from Shrimpton *et al* (2016), table 6.

CT examination	2003 survey <sup>a</sup>			2011 survey <sup>b</sup>		
	$E/DLP$ (mSv/mGy·cm)	DLP <sup>c</sup> (mGy·cm)	$E_{60}$ (mSv)	$E/DLP$ (mSv/mGy·cm)	DLP <sup>c</sup> (mGy·cm)	$E_{103}$ (mSv)
Head	0.0021	690	1.5	0.0020	890	1.8
Cervical spine	0.0059	—	—	0.0057	525	3.0
Chest	0.014	400	5.8	0.027	500	14
Chest (Hi res): All	0.014	88	1.2	0.027	230	6.2
Axial	—	—	—	0.027	110	3.0
Helical	—	—	—	0.027	360	9.7
CT Angiography	0.015	—	—	0.024	800	19
CTPA	0.015	—	—	0.027	360	9.7
Abdomen	0.015	350	5.3	0.024	670	16
Abdomen & pelvis	0.015	470	7.1	0.020	645	13
VC	0.015	—	—	0.020	780	16
Enteroclysis	0.015	—	—	0.020	580	12
KUB	0.015	—	—	0.018	355	6.4
Urogram	0.015	—	—	0.018	960	17
CAP	0.015	670	9.9	0.021	900	19
Paediatric head: 0–1 yr	0.011	230	2.5	0.0069 <sup>d</sup>	315	2.2
Paediatric head: >1–5 yr	0.0040	380	1.5	0.0044 <sup>d</sup>	530	2.3
Paediatric head: >5 yr	0.0032	510	1.6	0.0027 <sup>d</sup>	750	2.0

DLP = dose-length product; CTPA = CT pulmonary angiography; VC = virtual colonoscopy; KUB = kidneys-ureters-bladder; CAP = chest-abdomen-pelvis.

<sup>a</sup> Shrimpton *et al* (2005).

<sup>b</sup> Shrimpton *et al* (2014).

<sup>c</sup> Mean values from distributions of typical doses observed for sample in national survey.

<sup>d</sup> Based on Deak *et al* (2010).

between countries were observed, with the numbers of high cumulative dose patients per capita being about three times higher in the USA than in the UK or the Netherlands (Rehani and Hauptmann 2020). Substantial progress has been made in recent years in dose reduction for paediatric CT, but comparisons indicate that further improvements are possible. Strauss *et al* (2019) compared CT doses for paediatric patients in US hospitals with and without specialist paediatric facilities. They found significant differences in doses, with academic paediatric facilities using lower CT doses with less variation than non-academic paediatric and adult facilities for all brain and the majority of chest and abdomen-pelvis examinations. For example, while abdomen CT doses for the smallest patients were about 50% of the adult doses, corresponding doses in academic paediatric facilities average about 35% of adult doses.

## 4. Lifetime cancer risks and effective dose for common x-ray examinations

### 4.1. Variation of lifetime cancer risk per Sv with diagnostic procedure

Applying the methodology described in section 2 to calculate the sex- and age-at-exposure-specific LEAR of cancer incidence per organ/tissue absorbed dose for each of 11 organs/tissues (table 1), and using UK estimates of organ/tissue doses from a range of medical diagnostic procedures, as illustrated in section 3, Wall *et al* (2011) derived sex- and age-at-exposure-specific LEAR estimates for overall cancer incidence (excluding bone and skin cancers) for each procedure using the ICRP (2007) Euro-American composite population. For each procedure, Wall *et al* (2011) also calculated the effective dose and divided the sex- and age-specific LEAR estimates of overall cancer incidence by the effective dose to obtain the LEAR of cancer incidence per effective dose for each sex and age-at-exposure group. Wall *et al* (2011) used the LEAR of cancer incidence calculated as the REIC, as in table S1, with results shown in table S2, whereas ICRP Publication 147 (ICRP 2021) presented results using the corresponding LEAR estimates calculated as LAR, as in table 1, which are shown in table 5. Variations in the LEAR of cancer incidence per Sv effective dose reflect the combination of organ/tissue doses received during each procedure, which can vary considerably between different x-ray examinations. A chest x-ray examination, for example, results in doses to a number of organs/tissues, including the liver and stomach, as well as the lungs and breasts, while a head CT scan mainly delivers dose to the brain.

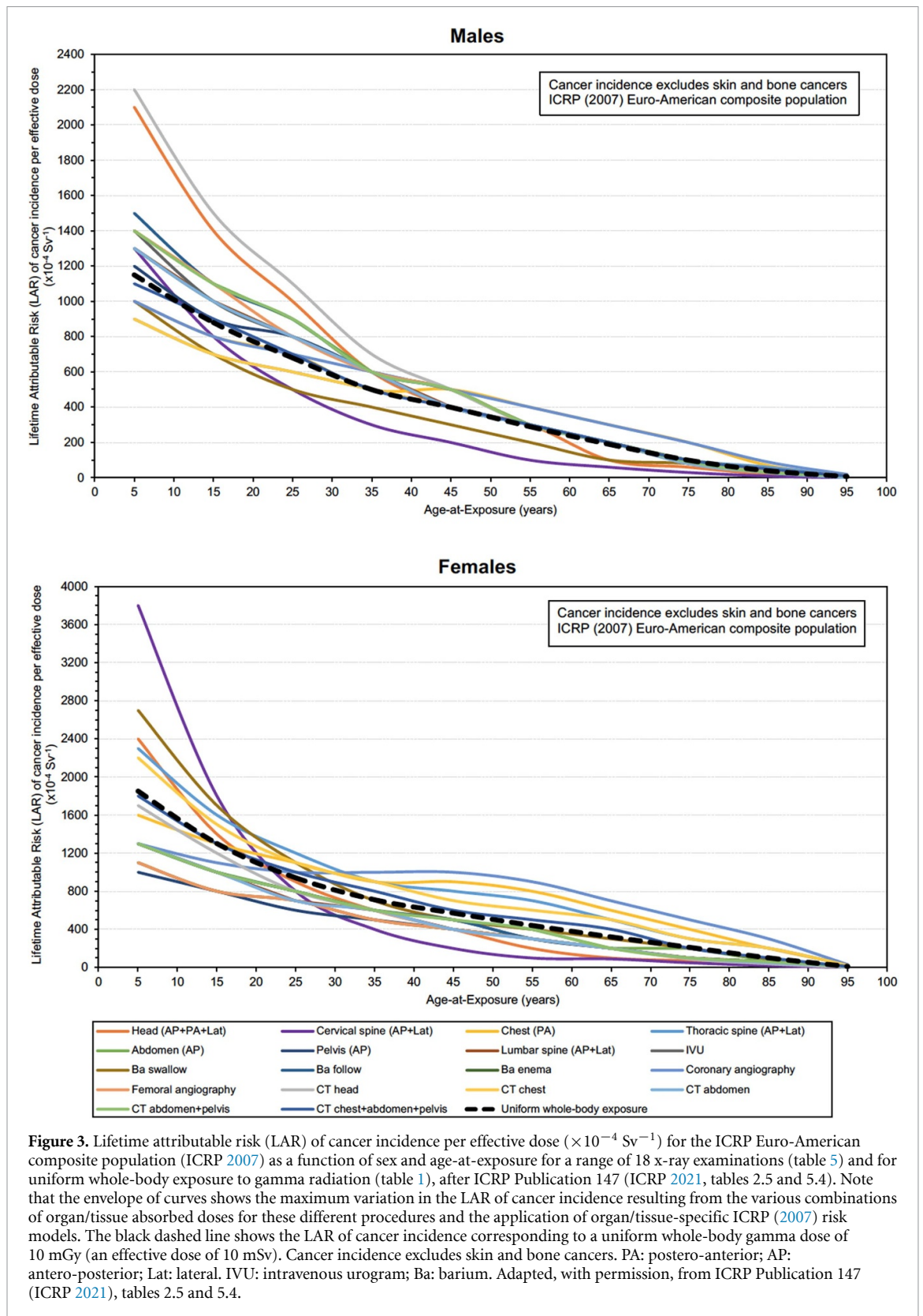
**Table 5.** Estimates of lifetime attributable risks (LAR) of cancer incidence per effective dose ( $\times 10^{-2} \text{ Sv}^{-1}$ ) by sex and age-at-exposure for a range of 18 medical diagnostic x-ray examinations, calculated using ICRP (2007) risk models applied to the ICRP (2007) Euro-American composite population (adapted, with permission, from ICRP Publication 147, ICRP (2021), table 5.3). Cancer incidence excludes cancers of the bone and skin.

		Lifetime attributable risk (LAR) of cancer incidence per effective dose ( $\times 10^{-2} \text{ Sv}^{-1}$ )									
Examination	Sex	Age at exposure (years)									
		0–9	10–19	20–29	30–39	40–49	50–59	60–69	70–79	80–89	90–99
Head	M	21	14	10	6	4	3	1	0.6	0.2	0
(AP + PA + Lat)	F	24	14	9	6	4	2	1	0.7	0.3	0
Cervical spine	M	13	8	5	3	2	1	0.6	0.3	0.1	0
(AP + Lat)	F	38	18	8	4	2	1	0.9	0.5	0.2	0
Chest	M	10	8	7	5	5	4	3	2	0.7	0.1
(PA)	F	16	13	11	9	9	8	6	4	2	0.3
Thoracic spine	M	9	7	6	5	4	3	2	1	0.6	0.1
(AP + Lat)	F	23	16	12	9	8	7	5	3	2	0.2
Abdomen	M	14	11	9	6	5	3	2	1	0.4	0.1
(AP)	F	13	10	8	6	5	4	2	2	0.7	0.1
Pelvis	M	12	9	8	6	4	3	2	1	0.4	0.1
(AP)	F	10	8	6	5	4	3	2	1	0.6	0.1
Lumbar spine	M	13	10	8	6	4	3	2	0.8	0.3	0.1
(AP + Lat)	F	13	10	7	6	4	3	2	1	0.6	0.1
IVU	M	14	10	8	6	4	3	2	0.9	0.3	0.1
	F	13	10	8	6	5	3	2	1	0.6	0.1
Ba swallow	M	10	7	5	4	3	2	1	0.8	0.3	0.1
	F	27	17	11	7	5	4	3	2	0.9	0.1
Ba follow	M	15	11	9	6	5	3	2	0.9	0.3	0.1
	F	13	10	8	6	5	3	2	1	0.6	0.1
Ba enema	M	13	10	8	6	5	3	2	1	0.4	0.1
	F	11	8	7	5	4	3	2	1	0.6	0.1
Coronary	M	10	8	7	6	5	4	3	2	0.9	0.2
Angiography	F	13	11	10	10	10	9	7	5	3	0.3
Femoral	M	14	11	8	6	5	3	2	0.9	0.4	0.1
Angiography	F	11	8	7	5	4	3	2	1	0.5	0.1
CT head	M	22	15	11	7	5	3	2	0.8	0.3	0.1
	F	17	12	8	6	4	3	2	0.9	0.4	0
CT chest	M	9	7	6	5	4	3	2	1	0.5	0.1
	F	22	15	11	9	7	6	5	3	2	0.2
CT abdomen	M	13	10	8	6	4	3	2	0.8	0.3	0
	F	13	10	7	6	4	3	2	1	0.5	0.1
CT abdomen +	M	14	11	9	6	5	3	2	0.9	0.3	0.1
Pelvis	F	13	10	8	6	5	4	2	1	0.6	0.1
CT chest +	M	11	9	7	5	4	3	2	1	0.5	0.1
Abdomen + pelvis	F	18	13	10	8	6	5	4	2	1	0.1

PA: postero-anterior; AP: antero-posterior; Lat: lateral. IVU: intravenous urogram; Ba: barium.

Figure 3 presents the LAR of cancer incidence for 18 different diagnostic x-ray examinations and the resulting distributions of organ/tissue doses, together with that for a uniform whole-body gamma dose of 10 mGy (i.e. an effective dose of 10 mSv). The variation of the LEAR/Sv with sex and age-at-exposure for uniform whole-body exposure is a consequence of the explicit application of sex- and age-specific risk models in contrast to the sex- and age-averaging of risk model results by ICRP (2007), which led to the relative detriment values that formed the basis of the broad groupings of tissue weighting factors used in the calculation of effective dose (see section 2). For most diagnostic procedures, the estimates of LEAR/Sv are within about  $\pm 50\%$  of those for uniform whole-body irradiation for the particular sex and age-at-exposure group. The variation of LEAR/Sv estimates about those for a uniform whole-body exposure reflects the application of organ/tissue-specific risk models to the different organ/tissue absorbed doses delivered in the various procedures, and the extent to which tissue weighting factors represent the sex- and age-specific LEAR/Gy estimates for the variously exposed organs/tissues in particular diagnostic x-ray examinations.

By way of comparison, figure S1 shows the results of an equivalent exercise using the ICRP (2007) Asian composite population (ICRP 2021, table 5.4). The patterns of LAR of cancer incidence values are broadly similar to those shown in figure 3 for the ICRP (2007) Euro-American composite population, with differences due to the differences in baseline cancer incidence rates in the two composite populations.



**Figure 3.** Lifetime attributable risk (LAR) of cancer incidence per effective dose ( $\times 10^{-4} \text{ Sv}^{-1}$ ) for the ICRP Euro-American composite population (ICRP 2007) as a function of sex and age-at-exposure for a range of 18 x-ray examinations (table 5) and for uniform whole-body exposure to gamma radiation (table 1), after ICRP Publication 147 (ICRP 2021, tables 2.5 and 5.4). Note that the envelope of curves shows the maximum variation in the LAR of cancer incidence resulting from the various combinations of organ/tissue absorbed doses for these different procedures and the application of organ/tissue-specific ICRP (2007) risk models. The black dashed line shows the LAR of cancer incidence corresponding to a uniform whole-body gamma dose of 10 mGy (an effective dose of 10 mSv). Cancer incidence excludes skin and bone cancers. PA: postero-anterior; AP: antero-posterior; Lat: lateral. IVU: intravenous urogram; Ba: barium. Adapted, with permission, from ICRP Publication 147 (ICRP 2021), tables 2.5 and 5.4.

#### 4.2. Cancer severity adjustment

The sex- and age-specific LEAR of cancer incidence per Sv estimates presented in table 5 (as LAR) and table S2 (as REIC), and illustrated in figures 3 and S1, respectively, were obtained by summing the LEAR/Sv estimates for each of the 11 cancer types for which ICRP (2007) risk models are available (see table 1), and depend on the organ/tissue doses received in each procedure. These LEAR/Sv estimates for each cancer type are not weighted by severity, which was done by ICRP (2007) to obtain the detriment values that are the basis of the  $w_T$  values used in the calculation of effective dose. Ideally, to provide additional information on

lifetime risks, the cancer incidence LEAR/Sv estimates derived here would be weighted by severity, but as noted in section 2, the severity adjustments carried out by ICRP (2007) were broad factors applied to nominal cancer incidence risk coefficients that were the result of sex-, age- and population-averaging for each cancer type. The severity adjustments cannot be disaggregated into specific weightings by sex, age-at-exposure and composite population (Cléro *et al* 2019, ICRP 2022, Ban *et al* 2022). As a consequence, the severity adjustment factors presented in ICRP Publication 103 provide little additional information on the variation of cancer risk by sex and age-at-exposure beyond that available from the cancer incidence LEAR/Sv estimates.

For illustrative purposes, for the 11 separate cancer models derived by ICRP (2007), the overall LEAR of cancer incidence per Sv reduces by 21.6% when weighted by severity to produce the equivalent detriment value (from  $688 \times 10^{-4} \text{ Sv}^{-1}$  to  $540 \times 10^{-4} \text{ Sv}^{-1}$ ). The largest proportional changes to the relative contributions to the overall nominal risk coefficient and detriment are reductions of  $\sim 50\%$  for bladder and thyroid cancers and an increase of  $\sim 87\%$  for leukaemia, but these cancer types each contribute only  $\sim 5\%$ – $6\%$  to the overall LEAR/Sv. Otherwise, the relative contributions of cancer types to the detriment are within 15% of the relative contributions to the nominal risk coefficient. Although differences will result from a particular combination of organ/tissue doses and associated severity weightings, substantial changes to the sex/age patterns of LEAR/Sv shown in figures 3 and S1 if cancer types were weighted by (sex- and age-averaged) severity would not, in general, be expected. Nonetheless, as emphasised above, severity weighting factors are broadly based values derived from limited data and applied across the whole population (ICRP 2022), and although severity adjustment by sex, age and population would be desirable, this is not possible from the information available in Publication 103 (ICRP 2007); improvements in the derivation of detriment are currently under consideration by ICRP (2022).

#### 4.3. Skin and bone cancers

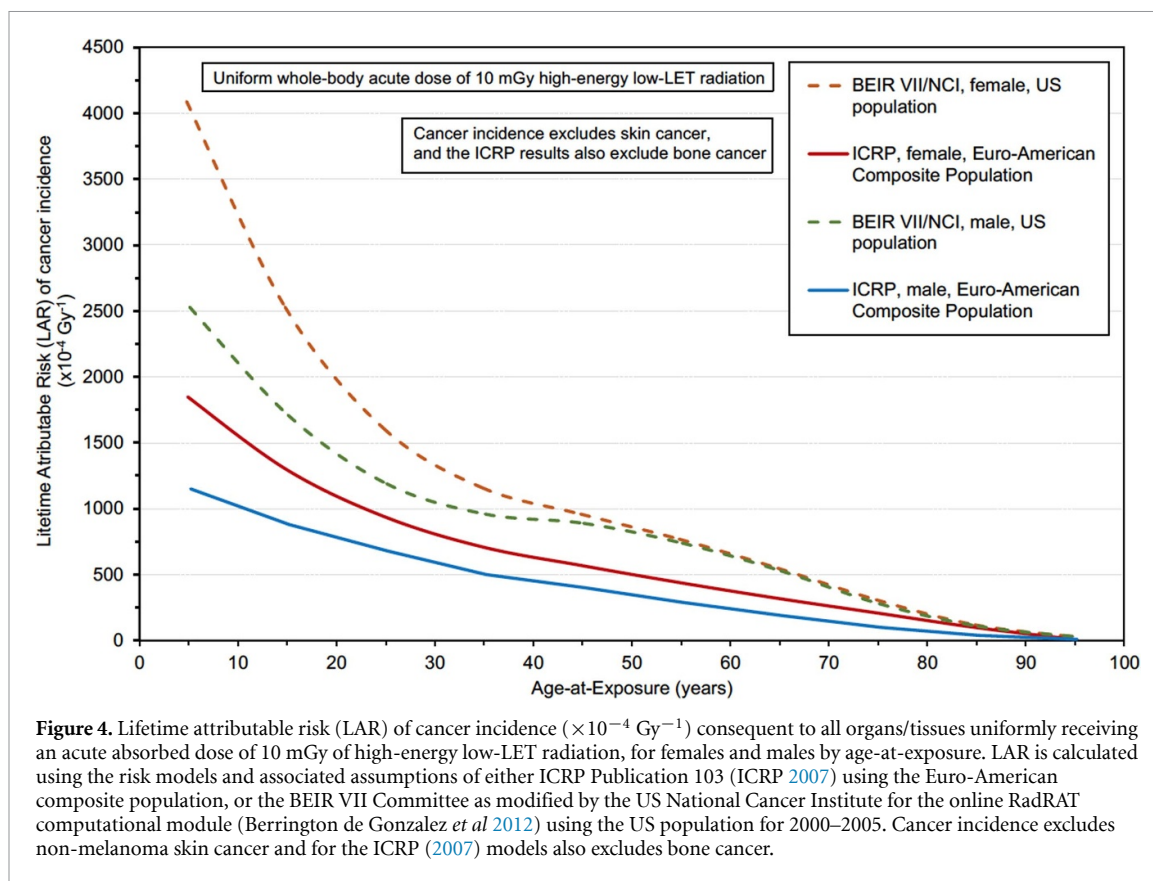
The exclusion of bone and (non-melanoma) skin cancers from the cancer incidence LEAR/Sv estimates of tables 5 and S2 (and figures 3 and S1) will have little impact on the overall conclusions. In Publication 103, bone cancer contributes just 0.4% of the total cancer incidence LEAR/Sv and 0.9% of the cancer detriment for the whole population. While skin cancer accounts for 59% of the total cancer incidence LEAR/Sv, non-melanoma skin cancer is easily treated (often as an outpatient), and once weighting for severity is incorporated, skin cancer contributes just 0.7% of the total cancer detriment. This is reflected in  $w_T$  values of 0.01 for both bone surface and skin so that doses to these tissues generally make a relatively small contribution to the effective dose. Further, as noted in ICRP Publication 103, the bone cancer risk coefficient was an overestimate based on average dose of radium-244 to bone volume rather than the appropriate higher dose (x9) to the bone surface.

#### 4.4. Heritable effects

An additional component of stochastic health risk considered by ICRP (2007) is heritable effects following irradiation of the gonads. For the whole population (both sexes, all ages) ICRP (2007) derived a nominal risk coefficient of  $20 \times 10^{-4} \text{ Sv}^{-1}$  for heritable effects, by averaging a risk coefficient of  $54 \times 10^{-4} \text{ Gy}^{-1}$  gonadal dose for the reproductive population over the population of all ages. Wall *et al* (2011) adopted a risk coefficient of  $50 \times 10^{-4} \text{ Gy}^{-1}$  for those of reproductive capacity and zero otherwise. ICRP (2007) derived a detriment for heritable effects of  $25.4 \times 10^{-4} \text{ Sv}^{-1}$  for the whole population (an increase of 27% over the nominal heritable risk coefficient) to give a relative detriment of 0.044 (leading to a  $w_T$  for the gonads of 0.08, which also included a somatic component for cancer in the exposed individual). Even taking a severity-weighted risk coefficient for a reproductive population of  $70 \times 10^{-4} \text{ Gy}^{-1}$  (i.e. one predominantly of relevance to younger ages at exposure) the contribution of the risk of heritable effects to the total stochastic effects risk is comparatively small when compared to the cancer detriment of  $549 \times 10^{-4} \text{ Sv}^{-1}$  (both sexes, all ages at exposure). However, the contribution would be somewhat greater for diagnostic procedures giving higher gonadal doses, such as CT examinations of the abdomen and pelvis of young females.

#### 4.5. Other risk models

It is important that the point estimates presented in table 5 (and table 1) do not impart a false impression of the precision of estimates of cancer risk from low-level radiation exposures. Point estimates are presented here to provide the broad overall pattern of sex and age-at-exposure differences in estimated risks. By way of illustration, other expert groups used essentially the same database as ICRP (the LSS cancer incidence data available in the mid-2000s) to generate cancer risk models and lifetime excess cancer incidence risks. One of these expert groups was the BEIR VII Committee of the US National Academy of Sciences (NRC 2006). The BEIR VII risk models have been modified by the US National Cancer Institute (NCI) for the purposes of producing the online adiation risk assessment tool (RadRAT) (Berrington de Gonzalez *et al* 2012).



#### 4.5.1. RadRAT

RadRAT (<https://radiationcalculators.cancer.gov/radrat/>) has here been used to generate cancer incidence LAR/Gy estimates for comparison with the LAR/Gy estimates shown in table 1 that were obtained using the ICRP (2007) cancer risk models. This entailed, for both the ICRP and RadRAT models, using a 10 mGy gamma radiation dose received uniformly by all organs/tissues. For the ICRP models, the Euro-American composite population was used, and for RadRAT the US population during 2000–2005 was selected as an input option. Cancer incidence LAR estimates exclude non-melanoma skin cancer, and the ICRP estimates also exclude bone cancer. Figure 4 presents the results of this comparison, showing that the BEIR VII/NCI cancer incidence LAR/Gy estimates are greater than the ICRP estimates by a factor that approaches 2.5 for the youngest ages at exposure, for both females and males. Important factors in this difference are that RadRAT uses a central estimate of the DDREF for solid cancers of 1.5 whereas the ICRP calculations use a fixed DDREF of 2, and the combination of EAR/ERR model results for most cancers is 0.3/0.7 for BEIR VII/NCI in contrast to 0.5/0.5 for ICRP, but differences also arise from the forms of the risk models used, such as the treatment of modifying factors. This comparison exercise illustrates the considerable uncertainties that arise from modelling and associated decisions, such as the value of the DDREF and the EAR/ERR model ratios used, which must be borne in mind when viewing any one set of risk estimates. Although bone cancer is excluded from the ICRP cancer incidence estimates this will have little impact in comparing the BEIR VII/NCI and ICRP results because bone cancer accounts for only 1% of the overall LEAR/Sv of cancer incidence (excluding skin cancer) in ICRP Publication 103 (ICRP 2007).

Further, RadRAT output includes ‘90% uncertainty ranges’ for the cancer incidence LAR estimates, which permits an assessment of the precision of these estimates produced by the BEIR VII/NCI models and associated assumptions. The 90% uncertainty ranges output by RadRAT account not only for sampling uncertainty in the model parameters, produced by the limited data available for the generation of the risk models, but also for modelled uncertainties in the values of the DDREF, minimum latency period and transfer of risk between populations (Berrington de Gonzalez *et al* 2012). Table 6 presents the cancer incidence LAR/Gy point and interval estimates produced by RadRAT for a uniform whole-body acute dose of 10 mGy of high-energy low-LET radiation received at 5, 35 and 65 yr of age by typical females and males from the US population for 2000–2005. An equivalent exercise for the Japanese population (for 2010) showed similar results (see table S3 and figure S2). The point LAR estimates in table 6 are for the BEIR/NCI models shown in figure 4, but what is striking is the considerable absolute widths of the interval estimates of

**Table 6.** LAR of cancer incidence ( $\times 10^{-4} \text{ Gy}^{-1}$ ), the lifetime baseline risk (LBR) of cancer incidence ( $\times 10^{-4}$ ), and the proportional increase in risk expressed as a fraction of the baseline risk (LAR/LBR) ( $\text{Gy}^{-1}$ ) for the US population for 2000–2005, for each sex and three ages-at-exposure (5, 35 and 65 yr), following a uniform whole-body acute dose of 10 mGy of high-energy low-LET radiation (received in 2005). The calculations were performed using the online radiation risk assessment tool, RadRAT (Berrington de Gonzalez *et al* 2012), which employs BEIR VII cancer risk models and associated assumptions (NRC 2006) as modified by the US National Cancer Institute, <https://radiationcalculators.cancer.gov/radtrat/>. Cancer incidence excludes non-melanoma skin cancer.

Age-at-exposure (years)	Sex	LAR of cancer incidence <sup>a,b</sup> , $\times 10^{-4} \text{ Gy}^{-1}$ (90% uncertainty range) <sup>d</sup>	LBR of cancer incidence from time of exposure <sup>a</sup> , $\times 10^{-4}$	Proportional increase in cancer incidence risk (LAR/LBR) <sup>a,c</sup> , $\text{Gy}^{-1}$ (90% uncertainty range) <sup>d</sup>
5	Male	2530 (1280–4060)	4862	0.52 (0.26–0.84)
	Female	4090 (2300–7050)	4144	0.99 (0.56–1.70)
35	Male	964 (474–1650)	4923	0.20 (0.10–0.34)
	Female	1160 (663–1970)	4090	0.28 (0.16–0.48)
65	Male	526 (274–863)	4293	0.12 (0.06–0.20)
	Female	548 (317–906)	2990	0.18 (0.11–0.30)

<sup>a</sup> Excluding non-melanoma skin cancer.

<sup>b</sup> Lifetime excess absolute risk, LEAR, the probability of developing a cancer over the remaining lifetime as a result of the exposure.

<sup>c</sup> Lifetime excess relative risk, LERR, the fractional increase in the probability of developing a cancer over the remaining lifetime as a result of the exposure in comparison to the baseline probability of developing a cancer over the remaining lifetime in the absence of the exposure.

<sup>d</sup> A '90% uncertainty range' associated with a particular point estimate of the LAR of cancer incidence is output by RadRAT (Berrington de Gonzalez *et al* 2012). RadRAT also provides a 90% uncertainty range for a LBR point estimate, but proportionally is small in comparison to that for the LAR and so is ignored in the calculation of the 90% uncertainty range for LAR/LBR.

LAR/Gy at an age-at-exposure of 5 yr. Of note is that at this age-at-exposure, the point LAR/Gy estimates for cancer incidence generated by the ICRP (2007) models (table 1) are less than the lower 90% uncertainty limits for the estimates obtained using the BEIR VII/NCI models (table 6). The wide interval estimates are a reflection of the imprecision of LAR/Gy estimates based on models derived from the LSS data (and associated assumptions, such as the value of the DDREF) for those exposed at young ages. When comparing the point estimates of the LAR/Gy of cancer incidence predicted by the ICRP and BEIR VII/NCI risk models, as illustrated in figure 4, the uncertainty ranges shown in table 6 for the BEIR VII/NCI risk models and associated assumptions, as generated by RadRAT, must be recognised; Moreover, these indicators of uncertainty in risk estimates are most unlikely to take account of all the sources of uncertainty that are inherent in the ICRP and BEIR VII/NCI risk modelling and associated assumptions.

#### 4.5.2. Lifetime baseline risk (LBR) of cancer incidence

RadRAT runs also output estimates of the lifetime baseline risk (LBR) of cancer incidence following exposure, that is, the lifetime absolute risk of cancer incidence that would have been experienced by a typical member of the population in the absence of the specific exposure under scrutiny. The ratio of the LAR to LBR estimates provides an estimate of the lifetime excess relative risk (LERR) of cancer incidence following exposure. Table 6 shows values of LBR and LAR/LBR ratios, corresponding to the LAR predictions. Wide uncertainty ranges for the absolute LAR/LBR ratios for young ages at exposure are apparent (see also figures S2 and S3). The advantage of LERR estimates is that they provide context for the LEAR estimates, in that the LAR is expressed as a proportional increase over the background risk of cancer incidence over the expected remaining lifetime. So, from table 6 it can be appreciated that a uniform whole-body acute dose of 10 mGy of high-energy low-LET radiation received by a girl aged 5 yr produces, on the basis of RadRAT and the US population for 2000–2005, a proportional increase in the lifetime risk of cancer incidence of about 1%, with an uncertainty interval of around 0.6%–1.7%.

#### 4.5.3. Other sources of uncertainty

Apart from ICRP and BEIR VII (and as modified by NCI), other expert groups have produced risk models based on the LSS cancer incidence data available in the mid-2000s. These groups include the radiation effects research foundation in Japan (Preston *et al* 2007), the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR 2008) and the US Environmental Protection Agency (EPA 2011). These models differ to varying degrees, and this emphasises the notable uncertainties associated with the approach to model construction, and also those associated with assumptions about, *inter alia*, DDREF and risk transfer. Further sources of uncertainty include the relative biological effectiveness (RBE) of low-energy photons (x-rays) in comparison to high-energy photons (gamma rays), because of evidence that low-energy low-LET radiation has a RBE > 1 relative to high-energy low-LET radiation (NCRP 2018). It should also be borne in mind that risks are estimated for typical members of population groups and that individual patients

may well have shorter life expectancy and hence lower risks of developing radiation-related cancer over their lifetimes; some patients may also suffer from medical conditions (or experience other circumstances, such as treatments with certain drugs) that increase their risk of radiation-related cancer.

In the calculations of excess cancer risks presented above, it has been assumed that the organ/tissue absorbed doses are exact. Of course, this is an idealisation because dose estimates are uncertain, to a greater or lesser extent. RadRAT has a facility to input various uncertainty distributions for organ/tissue dose estimates, and the use of inexact doses will widen the LAR/Gy interval estimates to a degree that depends on the level of organ/tissue dose uncertainties.

ICRP (1991, 2007) does not explicitly consider uncertainties in the derivation of stochastic risk models and dosimetric models. Rather, the protection system relies on the use of periodically updated models that reflect the best contemporary science and their application in the optimisation of protection by keeping effective doses as low as reasonably achievable, social and economic factors being taken into account. However, as discussed by Puncher *et al* (2017) in considering the example of doses and risks from intakes radioisotopes of iodine by children and adults, knowledge of uncertainties can be an important input to decisions over appropriate optimisation.

## 5. Conclusions

Effective dose,  $E$ , has gained worldwide acceptance as the quantity that enables different sources of low-level radiation exposure of different organs/tissues to be meaningfully combined and controlled on a common basis for protection against stochastic effects, principally cancer. This approach has provided a single quantity,  $E$ , to successfully represent the overall dose received from different types of radiation by the various organs/tissues of the body, including when these exposures are highly heterogeneous. However, unlike the well-established use of effective dose to optimise protection for workers and members of the public, its use to assess doses to medical patients has led to suggestions that it is not well-suited for this purpose (Brenner 2008, 2012, Andersson *et al* 2017). The main defect identified is that effective dose relates to values of population-averaged stochastic detriment and thus does not take account of the sex and age-at-exposure of individual patients. Results presented in this paper and in ICRP Publication 147 (ICRP 2021) show that while the age-dependence of lifetime cancer risks varies between organs/tissues, and between males and females, the overall variations in risk can be summarised quite simply. For a range of x-ray examinations, including radiography and CT, average lifetime risks of cancer incidence per Sv may be around two or three times higher for exposures at age 0–9 yr than at age 30–39 yr. For patients exposed in their 60s, the estimated lifetime risks are about half those for patients in their 30s, falling to about one-tenth for those in their 80s.

Since effective dose per examination is generally less in children than in adults by a factor of two or more (see section 3), and given the considerable uncertainties in risk estimates for low doses, particularly for younger ages at exposure, it will be reasonable in most cases to assume that risks for a particular procedure are similar for all young to middle-aged patients, with risks reducing in old age. While it is possible to calculate risks using organ/tissue-absorbed doses and sex- and age-at-exposure-specific risk models, as done, for example, by Andersson *et al* (2017), this will not be necessary in the majority of cases when a simple indication of risk is required, as illustrated in table 7. ICRP (2021) concluded that effective dose can be used for such purposes as ‘an approximate indicator of possible risk’. While somewhat tautological, this term is intended to underline the uncertainties in the estimation of risk at low doses and low dose-rates that are very often below levels at which excess cancer rates have been reliably demonstrated in epidemiological studies.

ICRP (2021) recognises that there is potential to improve the formulation of effective dose so that it more closely relates to stochastic risks under various circumstances. Given that absorbed doses to organs and tissues are calculated as best estimates using sophisticated biokinetic and dosimetric models, it is arguably inconsistent to then apply simplified radiation and tissue weighting factors in the calculation of effective dose. It would be possible to use suitable inferences from the results of scientific studies to calculate effective dose separately for males and females and for different ages at exposure, and specify the corresponding values of stochastic detriment. However, the implications of such a development for the system of protection would have to be carefully considered. ICRP has provided reference dosimetric phantoms for adults and children of different ages, which will help ensure that doses are calculated using standard methodology to facilitate comparisons; modern phantoms are readily deformable and so also adaptable for the calculation of doses for individuals of different sizes.

ICRP is in the early stages of preparation for new recommendations and will consult on possible changes that will include such considerations of the use of effective dose (Clement *et al* 2021). There are good practical reasons, including continuity, for keeping the current formulation of effective dose. ICRP could emphasise, as discussed in Publication 147 (ICRP 2021), that effective dose as currently defined can be used not only in the optimisation of protection and demonstration of compliance with limits, but also as an

**Table 7.** Effective dose ranges and terminology for describing risks from different medical diagnostic procedures for adult patients aged 30–39 yr (based, with permission, on ICRP Publication 147 (ICRP 2021), table 5.2).

Effective dose (mSv)	Cancer risks <sup>a</sup>	Proposed term	X-ray examination type <sup>b</sup>
< 0.1	<10 <sup>-5</sup>	Negligible	Radiography
0.1–1	10 <sup>-5</sup> –10 <sup>-4</sup>	Minimal	Radiography
1–10	10 <sup>-4</sup> –10 <sup>-3</sup>	Very low	Radiography and Fluoroscopy; CT scanning
10–100	10 <sup>-3</sup> –10 <sup>-2</sup>	Low	CT scanning
100s	>10 <sup>-2</sup>	Moderate	Multiple CT scans

<sup>a</sup> Risk bands are LEAR of cancer incidence to nearest order of magnitude, relying on the linear no-threshold dose-response (LNT) model for doses below a few 10s mSv.

<sup>b</sup> See section 2 for effective dose estimates for individual procedures.

approximate measure of the risk of stochastic effects. In parallel, ICRP could promote the use of specific organ/tissue risk models to be applied to organ/tissue-absorbed doses for situations in which best estimates of risk are required.

### Data availability statement

All data that support the findings of this study are included within the article (and any supplementary information files).

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