Improved radiation risk models applied to different patient groups in Sweden

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In radiological diagnostics and therapy, it is important that practitioners, referrers, (i.e. radiologists, radiation oncologists and others in health-care) are aware of how much radiation a patient may receive from the various procedures used and associated health risk. The profession has a duty to inform patients or their representatives of the advantages and disadvantages of specific investigations or treatment plans. The need to estimate and communicate risks in connection with medical use of ionizing radiation is highlighted e.g. in the Russian Federation State Law No 3, §17.2, 1996 and in the EU directive (2013/59/EURATOM 2014). The most commonly used way to express harm in relation to low doses of ionizing radiation is use of the quantity effective dose (E). Effective dose, a radiation protection quantity, however is not intended to provide risk estimates for medical exposures. Its purpose is to optimize conditions for radiation workers (18–65 years) or the general public; all groups with age distributions that differ from patients. In this paper the lifetime attributable risk was used to estimate the excess risk of receiving and dying of radiogenic cancer. The lifetime attributable risk estimations are generated from three different variables, gender, attained age and age at exposure giving the possibility to create age and gender specific cancer risk estimations. Initially, the US Environmental Protection Agency lifetime attributable risk coefficients which are intended to predict the cancer risk from ionizing radiation to a normal US population were applied. In this work, the lifetime attributable risk predictions were modified to the normal Swedish population and to cohorts of Swedish patients undergoing radiological and nuclear medicine examinations or treatments with survival times that differ from the normal population. For Swedish males, all organs were given the same absorbed dose, exposed at 20, 40 and 70 years, the lifetime attributable risk coefficients (Gy−1) were 0.11, 0.068, and 0.038, respectively, which is lower than the corresponding figures for US males, 0.13, 0.077, and 0.049. For Swedish females, all organs were given the same absorbed dose, exposed at 40 years of age with a diagnosis of breast, colon or liver cancer, the lifetime attributable risk coefficients are 0.064, 0.034, and 0.0038, respectively, which is much lower than if a 40 years female without known cancer is exposed, 0.073.

Key words: effective dose, life time attributable risk, radiation risk predictions.
1. Introduction

In radiological diagnostics and therapy, it is important that practitioners, referrers, radiologists, radiation oncologists and others in health-care understand how much radiation a patient may receive from the various procedures used and the associated risk. National and international directives state e.g. that “… based on the citizens’ or patients’ request they shall receive full information on expected or received dose and possible consequences due to the x-ray examination …” (Russian Federation Law No 3, § 17.2 “On the radiation safety of the public, 1996”) and the need to “… ensure wherever practicable and prior to the exposure taking place adequate information relating to the benefits and risks associated with the radiation dose from the medical exposure” (EU directive 2013/59/EURATOM 2014) [2].

The most commonly used way to express harm in relation to low doses of ionizing radiation is to use the quantity effective dose (E) defined by the International Commission on Radiological Protection (ICRP) [3]. However, effective dose was not intended to provide risk estimates for medical exposures. Its purpose is to optimize conditions for radiation workers (18–65 years) or the general public – groups with different age distributions than patients. In spite of that, effective dose is also frequently used for risk estimates for patients undergoing medical exposures and even for individual patients. The effective dose is a weighted sum of tissue specific doses. ICRP [3] determined tissue weights by first calculating tissue-specific “nominal risks adjusted for lethality and quality of life” which are the tissue-specific cancer (and hereditary) risk estimates multiplied by a “lethality fractions”, and “relative cancer free life lost”. The tissue weights were then subjected to appropriate rounding and normalized to sum to 1, resulting in values of 0.01, 0.04, 0.08, or 0.12. Based on estimates of detriment for lifetime exposure to uniform whole-body radiation, ICRP established risk coefficients of 5.5% per Sv for a population of all ages and 4.1% per Sv for radiation workers. The effective dose is a robust unit for many populations exposed to environmental and occupational sources of radiation, but it does not account for variability within a population (e.g. due to different radiation mix and different radiation sources).

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Введение

В лучевой диагностике и терапии крайне важно, чтобы медицинский персонал (врачи-рентгенологи, лечащие врачи, радиационные онкологи и пр.) имел представление о том, какую дозу облучения получил пациент от различных рентгенорадиологических исследований и с каким радиационным риском для здоровья эта доза связана. Это условие является обязательным в соответствии с различными национальными и международными законодательством. Так, в Российском Федеральном законе № 3-ФЗ “О радиационной безопасности населения” прописано, что “По требованию гражданина (пациента) ему предоставляется полная информация об ожидаемой или полученной им дозе облучения и о возможных последствиях при проведении медицинских рентгенорадиологических процедур”. Директива Евросоюза 2013/59/EURATOM 2014 предусматривает необходимость предоставления адекватной информации о пользе и рисках от доз медицинского облучения и о возможных последствиях при проведении исследования.

Наиболее распространенной мерой вреда от низких доз ионизирующего излучения является эффективная доза (ЭД), определенная Международной комиссией по радиологической защите (МКРЗ). Однако эффективная доза не была предназначена для оценки риска от медицинского облучения. ЭД использовалась для определения условий работы персонала (в возрасте 18–65 лет) или населения – групп с иными возрастными распределениями и оценка риска. Несмотря на данные ограничения, ЭД часто используется для оценки риска у пациентов при медицинском облучении, в том числе и для индивидуальных пациентов. Эффективная доза является взведенной суммой тканеспецифических доз и поэтому для оценки риска по данным МКРЗ определено, что эффективные дозы в расчете на часть тканеспецифических “номинальных” доз (с поправкой на смертность и качество жизни), которые представляют собой тканеспецифические оценки дозы, используются "доли летальности" и "относительную потерю лет жизни без рака". Данные
take age and sex into account (e.g., for specific individuals undergoing medical procedures). The reason for this, stated by the ICRP, is that the system of protection should be sufficiently simple and robust [3, 4].

A step forward, as it relates to specific patients, could be to define an “index of harm” as the effective dose (keeping the tissue weighting factors) multiplied by a relative risk factor which is age dependent. Almén and Mattsson [5] used a risk factor between 2 and 3 for children and adolescents and Wall et al., [6], Balonov and Shrimpton [7] and Balonov et al. [8] a relative risk factor of 2 for children and adolescents < 18 y, 1 for adults < 65 y and 0.1 for seniors 65+ years. Simple adjustments of ICRP’s nominal risk coefficient to account for age differences have in this way made effective dose a useful tool for the description of the radiation detriment.

An alternative approach to effective dose is to base risk assessments directly on calculations of lifetime attributable risk (LAR). There are different organizations performing risk models for assessing LAR [9, 10]. For these calculations, we adopted the approach used by the United States’ Environmental Protection Agency (EPA) [9]. For most cancer sites, the EPA risk models and other underlying assumptions are identical to those recommended in the United States’ National Research Council BEIR VII report [11]. For example, the models for most cancers were derived from incidence data from the Lifespan Study of the Japanese atomic bomb survivors and a dose and dose rate effectiveness factor of 1.5 assumed for cancers other than leukemia, bone, and skin. Extensions and modifications to the BEIR VII approach include risk estimates for alpha particles and a more extensive analysis of uncertainties associated with the radiogenic risk estimates. These risk models are used to assess both excess risk of cancer incidence and premature cancer death. For most cancer sites, the EPA assumed both relative and absolute excess risk to be proportional to the absorbed dose with a slope that depends on the tissue, age at exposure, attained age and sex. An important exception is leukemia, for which risk depends also on time since exposure.

In a previous analysis [12] we used EPA LAR coefficients – as an alternative to effective dose – to quantify sex and age specific cancer risks for patients undergoing x-ray examinations, nuclear medicine examinations and treatments. The study also assessed differences in outcome between effective dose and LAR risk estimation, as illustrated in Fig. 1. The effective dose-based risk for adults (4.1% per Sv) and the LAR risk are presented for a 99mTc bone scintigraphy, for ages at exposure between 0 and 110 years. We also showed that if the difference in cancer risk for different ages at exposure and sex is known, it is possible to perform bone scintigraphy procedures with the same risk for stochastic effects by varying the activity administered for patients of different age and sex. However, for that study, the LAR coefficients that were applied had been developed for a standard (healthy) US population. The aim of the current project was to explore the need to modify the approach by applying LAR coefficients that are appropriate for a standard Swedish population and also for specific cohorts of Swedish patients.

2. Materials and Methods

2.1. Investigated cohorts of patients in medicine

The cohorts of patients range from diagnostic radiology/subject – especially those examined using computed tomography (CT) – to cohorts of patients undergoing nuclear medicine...
and external beam radiation therapies. To properly assess the risk associated with medical procedures, one must consider the range of circumstances under which the radiation exposures occurred. CT examinations are administered to many patients for a wide range of medical indications. The purpose of the exposures is to provide information on the disease, (e.g., to exclude or stage disease) as part of treatment planning or as an aid in executing the treatment. Other exposures are performed to verify treatment results in connection with the treatment but also used to follow-up disease – sometimes up to several years after the actual treatment ends. Thus, medical exposures involve healthy (normal), diseased, and potentially cured persons (which are assumed to re-enter the cohort of healthy individuals), and for these three types of patient groups there are different sets of survival rates. Exposure levels can vary greatly, depending both on the type of examination and the hospital where the examination is performed.

For radiation treatments (e.g., therapy with radiopharmaceuticals and external beam radiation therapy) concern about acute radiation effects takes precedence and less interest is devoted to the mitigation of radiation induced cancer. Before an external beam radiation therapy treatment is performed the absorbed dose is planned, using a sophisticated dose planning calculation, and the dose distribution is optimized in the planning target volume, with attention to doses in tissues at risk for acute effects. Outside this volume, absorbed dose and radiation risk levels are to a less extent assessed and considered. However, larger volumes of healthy tissues are exposed to doses of varying magnitude in patients undergoing external beam radiation therapy [13]. Also, in nuclear medicine therapy, the absorbed dose distribution in the patient is not routinely assessed and the dose distribution for the individual patient is to a less extent optimized. Radiopharmaceuticals include both alpha and beta emitting radionuclides resulting in high doses to small volumes of tissues. However, organs outside targeted tissues can receive a high radiation absorbed dose resulting in acute radiation effects in some nuclear medicine procedures.

Fig. 1. Left: Age at exposure and sex dependent cancer morbidity and mortality risks from an intravenously administered \( ^{99m}\text{Tc} \)-phosphonates for bone scintigraphy using recommended age dependent administrations [12]. Right: Adjusted administered activity to get the same radiation risk independent of age and gender [12]

[Рис. 1. Слева: возраст при облучении и риски заболеваемости и смертности от рака с учетом пола пациента при внутривенном введении \( ^{99m}\text{Tc} \)-фосфоанта для сцинтиграфии костей с использованием рекомендованных активностей для соответствующих возрастных категорий. Справа: скорректированные значения активности данного препарата для достижения одного и того же риска вне зависимости от пола и возраста]
2.2. Risk estimates using LAR

For cancer patients the lifetime attributable risk of a secondary primary cancer caused by radiation and the risk of dying of this cancer is estimated. The term “secondary primary cancer” describes, in this case, a new primary cancer that occurs in a person who has had cancer in the past. The second primary cancers may occur years after the original (primary) cancer was diagnosed and is independent of the first cancer. This is important as most radiation treatments for cancer involve many examinations. For non-cancer patients the LAR coefficients for estimating the morbidity and mortality risk for radiation induced primary cancer for 14 specific cancers: bone, breast, colon, kidney, leukaemia, liver, lung, ovary, prostate, skin, stomach, thyroid, urinary bladder, uterus; and the category ‘residual site’ cancers are available [9]. The residual site cancers include cancers for which there were insufficient data from the Life Span Study of Japanese Atomic Bomb Survivors or other epidemiological studies to reliably quantify radiogenic site-specific risks. The LAR cancer risk predictions for a specific cancer site are presented in Eq. 1, which basically can be described in three steps. The first step is to calculate, for each of the 15 different cancer types, age specific excess rate of cancer diagnosis, \( M(D, b, a) \). The \( M(D, b, a) \) is a function of three variables, the absorbed dose \( D \) of the specific organ, the age \( e \) at the exposure and the attained age \( a \) of cancer diagnosis. The next step is to multiply the excess cancer rates by the probability of being alive at age \( a \), \( S(a) \), each year after the exposure, normalized by the probability of being alive at exposure. Finally, the LAR is obtained by integrating these adjusted excess cancer rates though integration over attained age starting post a latent period of five years (two for leukemia) after the age of exposure. Thus,

\[
LAR(D, e)_sex = \int_{e+L}^{110} M(D, e, a) \cdot \frac{S(a)}{S(e + L)} \, da \quad (1)
\]

where \( D \) is the absorbed dose, \( e \) is the age (year) at exposure, \( L \) is the latency period (year) after exposure for which stochastic effects occurs, \( a \) is the attained age (year), \( S(a) \) and \( S(e) \) is the survival rate at age \( a \) and \( e \) respectively.

2.3. Risk Modification of LAR

Our radiogenic cancer risk projections are combinations of projections based on excess absolute risk (EAR) and excess relative risk (ERR). A projection based solely on an EAR model assumes that radiation risks for the “target” population is the same as for the Life-Span Study, whereas, a projection based solely on the ERR assumes risks are proportional to baseline cancer risks. The BEIR VII Committee concluded that “mechanistic considerations” suggested that for most cancer sites more emphasis should be placed on an ERR model. It can be noted that ICRP used equal weights for ERR and EAR models. The EPA [9], following the advice of the BEIR VII Committee, assigned a weight of 0.7 to the ERR model for most cancers. e.g. the EPA predicted excess cancer rate for male stomach cancer is:

\[
M(D, e, a)_{Male(St)} = 0.7 \cdot (ERR(D, e, a)_{Male(St)} + \lambda(a)) + 0.3 \cdot (EAR(D, e, a)_{Male(St)}) \quad (2)
\]

where \( M(D, e, a)_{Male(St)} \) is the excess incidence cancer risk, \( \lambda(a) \) is the base line cancer risk at age \( a \) and the ERR of (normal),zzarella и возможно вылеченный индивидуум (предполагается что данная категория в дальнейшем войдет в состав когорты здоровых); для всех этих категорий индивидуумов коэффициенты выживаемости существенно отличаются. Также существенно будут отличаться уровни облучения пациентов, в зависимости от типа исследования и материально-технического оснащения медицинской организации.

Для лучевой терапии (как для радионуклидной, так и для дистанционной терапии) в первую очередь актуально исключение детерминированных эффектов; оценка развития радиогенных раков уделяется меньше внимания. До проведения дистанционной терапии производится оценка поглощенной дозы с использованием комплексных алгоритмов; проводится оптимизация распределений доз в планируемом объеме лечения с учетом поглощенных доз в тканях, близких к уровню развития детерминированных эффектов. За пределами данной области оценка и учет поглощенных доз и уровней риска проводятся крайне редко.

Однако большие объемы здоровых тканей облучаются различными дозами при проведении дистанционной лучевой терапии. Также в радионуклидной терапии крайне редко проводится оценка распределений поглощенных доз у индивидуальных пациентов. Радиофармпрепараты для радионуклидной терапии содержат альфа- и бета-излучатели, что приводит к высоким дозам в небольших объемах тканей. Ткани за пределами органа-мишеней могут получать высокие уровни поглощенной дозы, что будет приводить к развитию детерминированных эффектов.

Расчет пожизненного атрибутивного риска

Для онкологических пациентов оцениваются пожизненный атрибутивный риск развития “вторичного первичного рака” от облучения и риск смерти от данного рака. Термин “вторичный первичный рак” в данном случае относится к новому первичному раку, который возникает у индивидуума, у которого в прошлом уже был рак. Вторичный первичный рак может возникнуть спустя несколько лет после того, как исходный (первичный) рак был диагностирован; он развивается независимо от первого рака. Это является очень важным, так как большинство схем лучевой терапии рака предусматривают большое количество исследований с применением лучевой диагностики. Для нераковых пациентов доступны значения LAR для оценки заболеваемости и смертности от первичного радиогенного рака 14 локализаций: кожная ткань, мочевая железа, прямая кишка, лейкемия, печень, легкое, яичники, простаты, кожа, железы, щитовидная железа, мочевая пузьры, матка. Дополнительная категория “раки прочих локализаций” включает в себя раки, для которых было собрано недостаточно данных в пожизненных исследованиях на японской когорте выживших после атомных бомбардировок и прочих эпидемиологических исследований для достоверной количественной оценки радиационных рисков, специфичных для локализации.

Расчет LAR для специфической локализации рака производится по формуле (1). Процедуре расчета LAR можно разбить на три этапа. Первый этап заключается в расчете возраст-специфической избыточной частоты диагностики рака \( M(D, e, a) \) для каждой из 15 локализаций. \( M(D, e, a) \) является функцией трех переменных: поглощенной дозы в конкретном органе \( D \), возраста на момент облучения (e) и...
and EAR are the two different cancer risk estimates of male stomach cancer for an absorbed stomach dose, at an age of exposure e and an attained age of a.

To apply the excess cancer risk on populations other than the U.S., the baseline cancer statistics for the ERR predictions should be changed. The LAR predictions published by the EPA [9] were recalculated for a Swedish population, based on survival rates from the Swedish National Board of Health and Welfare [14] and cancer statistics from NORDCAN [15]. It should also be noted that cancer risk projections shown in Fig. 2. for environmental exposures are based on survival functions for normal populations. For specific populations of patients, e.g., patients suspected to have a life-shortening illness, the survival fraction $S(a)$ in eq.1 often needs to be modified. For example, in the data analyzed by Critz et al. [16], the 10, 15, 20, and 25-year disease-free survival for prostate cancer treated with brachytherapy and external beam radiotherapy is 75%, 73%, 73%, 73%, respectively. As the data indicates, no further recurrence was detected between 15 and 25 years, which indicates that no further follow up for this type of cancer is needed after 15 years to fully evaluate any prostate cancer treatment and that after 15 years patients can be treated as normal. These assumptions were used to estimate excess cancer risk for these patients, which will not follow the same survival rate as a normal population. The survival rates in the LAR equations has been modified with the longest time duration age and sex dependent cancer statistics data for the creation of population specific patient survival rates, as shown in Fig. 3. The EPA ERR and EAR models with Swedish survival rates in Fig. 3 were modified to account for the shorter expected survival for subpopulations of cancer patients as compared to survival rates for standard (relatively healthy) populations. This shows the effect modification of survival rates for different groups of cancer patients has on the predicted LAR. For calculating LAR, the EPA risk models were applied to survival rates and female incidence cancer data for three groups of cancer patients.

Fig. 2. LAR incidence as function of the age at exposure for a Swedish male population based on the last 30 years of Swedish cancer statistic [15] and a U.S male population both based on the risk prediction given by US EPA [9]. All organs are given the same absorbed dose.

$$LAR(D,e)_{sex} = \int_{e+L}^{10} \left( \frac{S(a)}{S(e+L)} \right) da \quad (1)$$

where $D$ – pоглощенная доза; $e$ – возраст на момент облучения (лет); $L$ – латентный период после облучения (лет), после которого начнут проявляться стохастические эффекты; $a$ – достигнутый возраст (лет); $S(a)$ и $S(e)$ – вероятности дожития до возрастов $a$ и $e$ соответственно.

Модификация моделей для расчета локального \наблюдаемого риска

Наши модели риска радиогенного риска являются комбинацией моделей, основанных на избыточном абсолютном риске (EAR) и избыточном относительном риске (ERR). Модель, основанная исключительно на EAR, подразумевает, что радиационный риск для выбранной группы населения идентичен таковому для LSS-когорты. В свою очередь, модель, основанная на ERR, предполагает, что радиационный риск пропорционален фоновому уровню риска рака данной локализации в популяции. Комитет BEIR VII заключил, что для большинства локализаций риска целесообразно использовать модель ERR. Следует отметить, что MKPЗ использовала модели EAR и ERR с одинаковыми взвешивающими коэффициентами. EPA, в соответствии с рекомендациями Комитета BEIR VII, присвоила взвешивающий коэффициент, равный 0,7 модели EAR для раков большинства локализаций. Например, предсказанная избыточная частота развития рака желудка для мужчин составит:

$$M(D,e,a)_{1,Male(Sl)} = 0.7 \times \left( \frac{ERR(D,e,a)_{1,Male(Sl)} + \lambda(a)}{ERR(D,e,a)_{1,Male(Sl)}} \right)$$

где $M(D,e,a)_{1,Male(Sl)}$ – избыточная частота заболевания раком желудка среди мужчин; $\lambda(a)$ – фоновый уровень заболеваемости раком желудка в возрасте $a$; $ERR$ и $EAR$ – два различных показателя избыточного риска развития рака желудка для мужчин при поглощенной дозе в желудке $D$ в возрасте на момент облучения $e$, рассчитанные для достигнутого возраста $a$.

Для применения избыточного риска развития рака для других (не американских) популяций необходимо использовать другие уровни фоновой заболеваемости раком в модели избыточного относительного риска ERR. Результаты расчетов локального избыточного риска, опубликованные EPA, были пересчитаны для шведского населения, основываясь на вероятностях дожития, публикуемых Шведским национальным советом по здоровью и благополучию, и онкологической статистике.
dependent cancer statistics data for the creation of population specific patient survival rates. The probability of survival for age $a$ is calculated as the number of persons alive at year $a$ divided by the initial persons alive at age 0. The patient specific survival rates, $S(a)_{Pat,Spec}$, for e.g. liver are modified for the first five years after exposure as from:

$$S(a)_{Pat,Spec} = \frac{N(a)^*}{N(0)} = \frac{N(a) + \text{ASP}_5(\text{Liver})}{N(0)}$$  \hspace{1cm} (3)$$

where $N(a)$ and $N(0)$ are the total number of health sex specific person alive at age $a$ and 0. $N(a)^*$ is the total number of patients live at age $a$ generated by multiplying $N(a)$ with the annual 5-year survival probability of liver cancer patients. The results of three different cancer patients are shown in Fig. 3. The EPA ERR and EAR models with Swedish survival rates in Fig. 3 were modified to account for the shorter expected survival for subpopulations of cancer patients as compared to survival rates for standard (relatively healthy) populations. This shows the effect modification of survival rates for different groups of cancer patients has on the predicted LAR. For calculating LAR, the EPA risk models were applied to survival rates and female incidence cancer data for three groups of patients (i.e., healthy, breast, colon and liver). Here the survival rate for female breast cancer is high while the survival rate for liver cancer is low.

The survival rates in the LAR equations has been modified with the longest duration age and sex dependent cancer statistics data for the creation of population specific patient survival rates. The probability of survival rate for age $a$ is calculated as the number of persons alive at year $a$ divided by the initial persons alive at age 0. The patient specific survival rates, $S(a)_{Pat,Spec}$, for e.g. liver are modified for the first five years after exposure as from:

$$S(a)_{Pat,Spec} = \frac{N(a)^*}{N(0)} = \frac{N(a) + \text{ASP}_5(\text{Liver})}{N(0)}$$  \hspace{1cm} (3)$$

where $N(a)$ and $N(0)$ are the total number of health sex specific person alive at age $a$ and 0. $N(a)^*$ is the total number of patients live at age $a$ generated by multiplying $N(a)$ with the annual 5-year survival probability of liver cancer patients. The results of three different cancer patients are shown in Fig. 3. The EPA ERR and EAR models with Swedish survival rates in Fig. 3 were modified to account for the shorter expected survival for subpopulations of cancer patients as compared to survival rates for standard (relatively healthy) populations. This shows the effect modification of survival rates for different groups of cancer patients has on the predicted LAR. For calculating LAR, the EPA risk models were applied to survival rates and female incidence cancer data for three groups of patients (i.e., healthy, breast, colon and liver). Here the survival rate for female breast cancer is high while the survival rate for liver cancer is low.

Fig. 3. LAR incidence as function of the age at exposure for a normal Swedish female population and for three different groups of Swedish female cancer patients (diagnosed with any stage of, and treated against colon cancer, breast cancer and liver cancer, respectively). SR means years of included survival rate. All organs were given the same absorbed dose.

[Рис. 3. Зависимость пожизненного атрибутивного риска от возраста при облучении для нормального (здорового) шведского населения и для трех различных групп шведских онкобольных женского пола (с установленным диагнозом или проходящих лечение при раке прямой кишки, молочной железы и печени). SR – годы учётного периода дожития. Все органы получили одинаковую поглощенную дозу]
3. Results and discussion

3.1. Changing the cancer base line and survival fractions of the LAR estimations

A comparison between the LAR (Gy⁻¹) incidence for a Swedish male population, based on survival rates from the Swedish National Board of Health and Welfare [14] and the last 30 years of Swedish cancer statistic from NORDCAN [15] and for a US population is shown in Fig 2. Both estimates are based on risk predictions given by EPA [9]. All tissues received the same absorbed dose. For newborn, children and adolescents of the Swedish male population, the LAR is lower than for similar US groups. This is probably due to differences in cancer statistics. For adult and senior (60+ year) males, there is no significant difference between the countries. The impact of any differences in cancer statistics will be less for the elderly due to shorter survival.

3.2. Changing LAR estimations from a healthy Swedish population to a cohort of Swedish patients

In Fig 3 LAR (Gy⁻¹) predictions are shown for a normal Swedish female population and for three different groups of Swedish female cancer patients. The LAR risks are the risk of receiving a cancer, incidence, for a cohort of breast, colon and liver cancer subjects. All tissues received the same absorbed dose. The result can be interpreted in two ways. For colon cancer, patients who have a low probability of survival, additional diagnostic imaging would have a low impact. In some cases, examinations are performed on both normal (healthy) populations and a cohort of a specific (potentially diseased) patient groups with an average risk somewhere in between the risk for these two subgroups. Optimization tools would include as input the fractions associated with each of these groups.

4. Conclusions

In radiological diagnostics and therapy, it is important that practitioners and referrers (i.e., radiologists, radiation oncologists and other health-care professionals) understand the radiation doses and the associated risk for patients undergoing various procedures. The risk for radiation induced cancer should be known and considered for all types of medical exposures and patient cohorts, but unfortunately the knowledge among health care professionals is limited. There is a need to include both more valid assessments of absorbed dose, and reasonable estimates of induced number of radiation cancer cases that would occur later in life for specific patient cohorts.

The most commonly used quantity to estimate the stochastic effects (mainly cancer) in medical radiology is the effective dose (E). However, LAR estimates offer a more suitable and direct approach for assessing cancer risk from the exposure to ionizing radiation associated with medical procedures. For cohorts of Swedish patients, LAR values shall be based on Swedish statistics on survival rates with adjustments to account for the patient’s health status. Assuming the same ab—

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and 0.0038, respectively, which is much lower than the LAR coefficient (0.073) for a 40-year-old female without known cancer. Appropriate LAR predictions can help improved radiation risk estimates for normal (healthy) populations as well as for various groups of patients, and simplify the justification process for referring physicians as well as professionals in diagnostic radiology, nuclear medicine and radiation therapy. The information to patients will also be improved.

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Disclaimer:

The views expressed in this article are those of the author and do not necessarily represent the views or policies of their organizations.

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