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Lifetime Risks for Lung Cancer due to Occupational Radon Exposure: A Systematic Analysis of Estimation Components

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Lifetime risk estimates play a key role in many areas of radiation research. Here, the focus is on the lifetime excess absolute risk (LEAR) for dying from lung cancer due to occupational radon exposure based on uranium miners cohort studies. The major components in estimating LEAR were systematically varied to investigate the variability and uncertainties of results. Major components of the LEAR calculation are baseline mortality rates for lung cancer and all causes of death, risk model and exposure scenario. Sex-averaged mortality rates were chosen from a mixed Euro-American-Asian population, in addition to mortality rates to represent heavy and light smokers. Seven radon-related lung cancer risk models derived from different uranium miners cohorts were compared. As exposure scenarios, occupational exposure of two working level months (WLM) from age 18–64 years was considered, and three scenarios from the German uranium miners cohort. Further components were modified in sensitivity analyses. The LEAR was compared to other lifetime risk measures. With a range from less than 0.6×10^{-4} to over 8.0×10^{-4}, LEAR per WLM estimates were influenced heavily by the choice of risk models. Notably, mortality rates, particularly lung cancer mortality rates, had a strong impact on LEAR per WLM across all models. The LEAR per WLM exhibited only low variation to changes in exposure scenarios for all risk models, except for the BEIR VI model fitted on the pooled 11 miners study. All assessed lifetime risk measures displayed a monotonically increasing relationship between exposure and lifetime risk at low to moderate exposures, with minor differences between ELR, REID, and LEAR (all per WLM). RADS yields the largest lifetime risk estimates in most situations. There is substantial variation in LEAR per WLM estimates depending on the choice of underlying calculation components. Reference populations and mortality rates should be selected with care depending on the application of lifetime risk calculations. The explicit choice of the lifetime risk measure was found to be negligible. These findings should be taken into consideration when using lifetime risk measures for radiation protection policy purposes. © 2025 by Radiation Research Society

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INTRODUCTION

Lifetime risk measures reflect the probability of developing (or dying from) a specific disease of interest over a lifetime. Lifetime risk measures are highly relevant for different areas of radiation research to quantify the lifetime excess risk due to radiation exposure. For example, they are part of the detriment calculation (*1*) with the calculation of nominal risks or the epidemiological approach for radon dose conversion (*2*–*4*). Here, lung cancer related to occupational radon exposure will be considered. The calculation of (excess) lifetime risks is typically based on one specific combination of calculation components, and in the final estimate, no uncertainties are reflected. The objective of this analysis was to vary the components of the lifetime risk calculation systematically to assess their impacts on the lifetime risk estimate. Therefore, this exploratory analysis contributes to quantifying uncertainties and sensitivities in the lifetime risk of lung cancer related to radon exposure.

Radon exposure is one of the most important causes of lung cancer aside from smoking (*5*). This was demonstrated in uranium miners and residential radon studies (*6*–*8*). Uranium miners studies have shown a linear relationship between occupational radon exposure and excess lung cancer mortality risk which is modified by age, time since exposure and exposure rate. The risk models are complex and differ between studies. Risk model parameter estimates between cohorts are therefore difficult or even impossible to compare. Lifetime risk measures provide a possibility for comparison and interpretation. Hence, such measures can also contribute to clearer and more comprehensible risk communication.

Different measures for excess lifetime risks include lifetime excess absolute risk (LEAR), risk of exposure-induced death (REID) and excess lifetime risk (ELR) (*9*, *10*). Tomasek et al. (*11*) calculated the LEAR of dying from lung cancer due to radon exposure for different risk models, among them an updated risk projection model for the pooled Czech and

French cohort of uranium miners. These updated lifetime risk calculations were considered in the epidemiological approach for radon dose conversion by ICRP (*4*). ICRP eventually recommended new factors for radon dose conversion (*1*). Even if the ICRP now recommends the dosimetric approach, the epidemiological approach using the LEAR is still relevant for comparison purposes (*4*, *12*).

Understanding the importance and consequences of necessary choices when implementing lifetime risk calculations requires elaborating on the sensitivity of the lifetime risk concept and its underlying calculation components. For example, Hunter et al. (*13*) performed a thorough sensitivity analysis on the REID for U.S. mortality rates focusing on effects of risk models from studies of occupational and residential radon exposure and differences in sex and smoking behavior. Chen et al. (*14*) conducted a sensitivity analysis for indoor radon for the Canadian population. A comparison of LEAR, ELR and REID for a linear risk model can be found in Kellerer et al. (*9*). Ulanowski et al. (*15*) introduced radiation-attributed decrease of survival (RADS) as another lifetime risk measure aimed to be less sensitive to the choice of background rates. Besides sensitivity analyses, uncertainties in lifetime risks have been investigated and quantified only rarely (*16*, *17*). Existing literature on sensitivity analysis of lifetime risks often focuses on a subset of components and selected lifetime risk measures. This highlights a research gap for structured sensitivity analyses incorporating all calculation components and a likewise structured comparison between lifetime risk measures, especially for lung cancer related to radon.

We contribute to this by systematically varying (excess) lifetime risk calculation components and quantifying their impact on the corresponding lifetime risk measure. In particular, we consider different risk models, and multiple heterogeneous exposure scenarios and construct different sex-averaged reference populations to account for a variety of situations and individuals, specifically for lung cancer related to occupational radon exposure. Focusing on the LEAR, we also compare results to ELR, REID and RADS. Further methodological issues are considered and discussed.

METHODS

Lifetime Risk Definition

There are various definitions for excess lifetime risks (*10*, *15*). All considered definitions emerge from the difference between risk under exposure and the baseline risk without exposure. Here we focus on the lifetime excess absolute risk (LEAR), sometimes also referred to as lifetime attributable risk (LAR), e.g. (*9*). The LEAR is defined as

$$LEAR_E(a) = LR_E(a) - LR_0(a) = \int_a^\infty (r_E(t) - r_0(t))S(t|a)\ dt = \int_a^\infty r_0(t)ERR(t)S(t\,|\,a)\ dt \tag{1}$$

with lifetime risk of dying from a specific disease of interest (here: lung cancer) under exposure $LR_E(a) = \int_a^\infty r_E(t)S(t|a)\ dt$, baseline lifetime risk $LR_0(a) = \int_a^\infty r_E(t)S(t|a)\ dt$, minimum age at risk a, baseline lung cancer mortality rates $r_0(t)$ and lung cancer mortality rates under exposure $r_E(t)$ at age t. $S(t\,|\,a)$ is the conditional survival function with $S(t\,|\,a) := P(T \geq t\,|\,T \geq a)$ and $T \geq 0$ the unknown random retention time until death. $S(t\,|\,a)$ describes the probability to survive until age t given the survival to age a. We set $S(t) := S(t\,|\,0) = P(T \geq t)$ and model the survival function as $S(t) = e^{-\int_0^t q_0(u)du}$ with baseline mortality rates for all causes of death $q_0(t)$. $ERR(t)$ denotes the excess relative risk at age t. In Eq. (1), the following risk projection model is assumed:

$$r_E(t) = r_0(t)(1 + ERR(t)). \tag{2}$$

Established risk models for lung cancer from radon exposure follow such an ERR structure (*8*, *18*). The $ERR(t)$ depends not only on age t but on further variables such as cumulative lagged exposure to radon progeny, time since exposure or exposure rate. The exact composition and complexity of the $ERR(t)$ depends on the specific risk model.

Lifetime Risk Calculation and Choice of Components

In the computation of LEAR, we distinguish between major and minor components. Minor components are defined with limited freedom of choice or possess minimal influence on the resulting LEAR. Major components, on the other hand, necessitate further decision-making because they are less constrained, and their choice is consequential.

The $LEAR_E(a)$ relies on three major components: mortality rates, risk models, and exposure scenarios. The first component encompasses mortality rates for lung cancer $r_0(t)$ and all-cause mortality rates $q_0(t)$, at each age t. The relative risk projection model shapes $ERR(t)$ at each age. The exposure scenario involves yearly exposure to radon progeny in working level months (WLM) and has an impact on $ERR(t)$ for every age t. Minor components include minimum age at risk a, maximum age a_{max}, minimum latency time L between exposure and risk amplification, and the chosen approximation approach for the survival function $S(t)$.

The LEAR is subsequently estimated and calculated through the following approximation:

$$LEAR_E(a) \approx \sum_{t=a}^{a_{max}} r_0(t)ERR(t)\widetilde{S}(t\,|\,a), \tag{3}$$

where the approximation $\widetilde{S}(t\,|\,a) = e^{-\sum_{u=a}^{t-1} q_0(u)}$ is utilized for the survival function $S(t\,|\,a)$. This approximation is based on the Nelson-Aalen estimator of the cumulative hazard rate (*19*).

The minimum age at risk a was set to $a = 0$ to account for the full lifetime of an individual. The maximum age a_{max} was set to $a_{max} = 94$ for comparability to previous studies (*20*). For readability, we write $LEAR = LEAR_E(0)$. For the latency period L between exposure to radon progeny and death from lung cancer, the cumulative exposures were lagged by $L = 5$ years since all considered risk projection models also assume $L = 5$. LEAR estimates were computed and compared for different combinations of major calculation components: three sex-averaged reference populations, seven risk models and four exposure scenarios, resulting in $3 \times 7 \times 4 = 84$ distinct LEAR estimates. Besides the total LEAR, the LEAR per WLM (LEAR/WLM) is considered, defined as the LEAR divided by total cumulative exposure accrued over the entire exposure scenario in WLM. All statistical or numerical analyses were conducted with the statistical software R (*21*).

Mortality Rates

The following sex-averaged mortality rates were chosen: ICRP reference rates reflecting a mixed Euro-American-Asian population derived from population data from the years 1993–1997 (*3*, *22*); rates from Greece 2018, the Netherlands 2018 and Norway 2016 based on population data provided by the WHO Mortality Database (*23*) reflecting a heavy smoker reference population; and rates from Costa Rica 2019, USA 2019 and Sweden 2016 reflecting a light smoker population. The country selection was based on smoking behavior following the OECD Health Statistics (*24*) on tobacco consumption (percentage of the population aged 15+ who are daily smokers) from the year 2000 to account for a latency of around 20 years between smoking and the

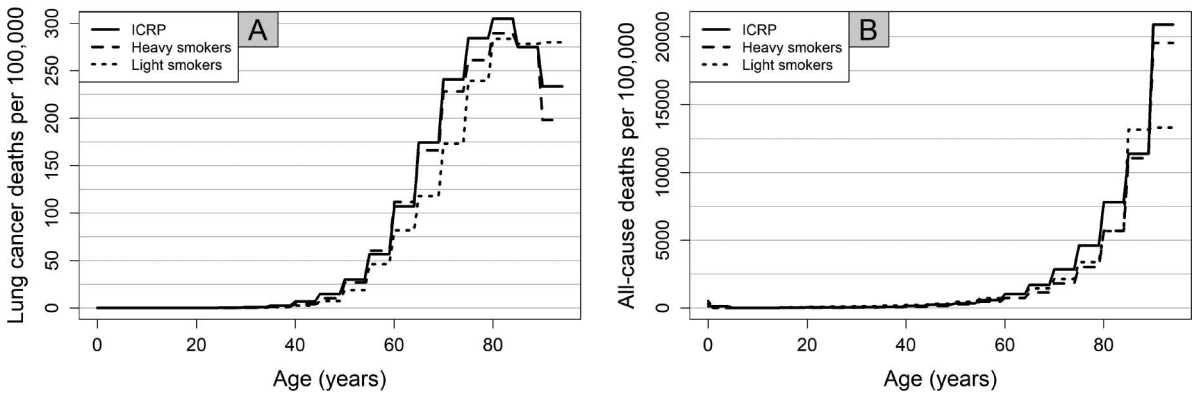

FIG. 1. Lung cancer deaths (panel A) and all-cause deaths (panel B) per 100,000 persons by age in the sex-averaged ICRP reference population (*3*) and in the constructed reference populations for heavy and light smokers.

development of lung cancer. This latency time was chosen based on several studies (*25–27*). The three countries with the highest percentage of daily smokers aged 15 and older (32–35%) were chosen to represent the heavy smoker population, while the three countries with the lowest percentage of daily smokers (12–19%) were chosen to represent the light smoker population. Further, the countries were chosen with the objective to employ complete and recent data.

The rates of heavy and light smokers were constructed by aggregating death cases d_i and population sizes n_i from different countries and sexes, where $i = 1, \ldots, N$ indexes both the country and sex for every age:

$$m = \frac{\sum_{i=1}^{N} d_i}{\sum_{i=1}^{N} n_i}. \tag{4}$$

If d corresponds to lung cancer deaths at age t, this yields the baseline rate $m = r_0(t)$, and if d denotes all-cause death counts at age t, $m = q_0(t)$. The full derivation of Eq. (4) is shown in the Supplementary Materials, Section A (https://doi.org/10.1667/RADE-24-00060.1.S1).[2]

Figure 1 shows the difference in lung cancer deaths (panel A) and all-cause deaths (panel B) per 100,000 persons for all three sex-averaged reference populations (exact numerical values in the Supplementary Table S1; https://doi.org/10.1667/RADE-24-00060.1.S1). Population data is given in 5-year age intervals. Light smoker and heavy smoker populations are similar in all-cause deaths per 100,000, with visible differences only at ages 85+. The ICRP reference population shows considerably more all-cause deaths per 100,000 compared to the smoker populations until age 85. There are notably more lung cancer deaths per 100,000 for heavy smokers than for light smokers (as expected). However, at ages 85+ heavy smoker lung cancer deaths per 100,000 decreased whereas for light smokers, they stayed approximately constant. The ICRP reference population yields relatively many lung cancer deaths, comparable to the heavy smoker population.

Unless explicitly stated otherwise, all lifetime risk estimates are calculated with mean mortality rates for males and females (sex-averaged mortality rates). Lifetime risks with male-specific mortality rates are analyzed in Supplementary Materials, Section B (https://doi.org/10.1667/RADE-24-00060.1.S1).

Risk Models and Cohorts

The LEAR calculation is based on a risk projection model and data from a cohort study, on which the risk model was fitted. Established risk models for lung cancer due to radon exposure in uranium miners cohort studies are based on the general structure in Eq. (2) and are fitted with internal (or sometimes also external) Poisson regression on grouped data. The preferred risk model contains a linear relationship between cumulative occupational radon exposure in WLM and excess relative risk of lung cancer, which is additionally modified by time since exposure, attained age or age at exposure, and in some cases also exposure rate (*8*). Recently, more focus has been given to more recent periods with low-radon exposures or exposure rates ("sub-cohorts 1960+") (*28–30*).

Here, the considered risk models are the categorical BEIR VI exposure-age-concentration model fitted on the pooled 11 miners cohort (BEIR VI model) (*6*) as well as on the Pooled Uranium Miners Analysis (PUMA) cohort (PUMA model) (*18*, *28*), the adjusted Jacobi model fitted on six cohort studies (*2*, *31*), and parametric risk models fitted on the German Wismut cohort (*7*) as well as on the Joint Czech and French miners cohort (*32*).

These models were selected for the following reasons. The initial factors for radon dose conversion were computed using the Jacobi model (*2*). The Joint Czech and French risk model, along with the BEIR VI model fitted to the pooled 11 miners cohort, contributed to a novel radon dose conversion proposal by ICRP (*4*). The German Wismut cohort is the world's largest single cohort of uranium miners. Notably, this extensive cohort was not included for the BEIR VI risk model (as the cohort was only established later). Suitable parametric models are used for both the Wismut 1960+ sub-cohort and full cohort, containing a continuous exposure rate effect for the full cohort that contrasts with the categorical exposure rate considerations in the BEIR VI and PUMA models. Note that we deliberately included Wismut models from the follow-up period 1946–2013 (*7*) rather than from the latest follow-up 1946–2018 (*29*) to ensure a broader variety of risk model structures for sensitivity analyses. While the more recent models offer slightly more precise estimates, such as by additionally stratifying the baseline by duration of employment, they are structurally similar to the other models compared here and do not contribute to the diversity of our model selection. The PUMA study is the largest uranium miners cohort worldwide, encompassing twice as many uranium miners and roughly three times as many lung cancer deaths (*33*) as the pooled 11 miners cohort (*6*). In particular, it includes the German Wismut cohort.

The generic categorical BEIR VI exposure-age-concentration model at age t reads

$$ERR(t) = \beta(W_{5-14}(t) + \theta_{15-24} W_{15-24}(t) + \theta_{25+} W_{25+}(t))\phi_{age}\gamma_z, \tag{5}$$

where W_{5-14}, W_{15-24}, W_{25+} is the cumulative radon exposure in the windows 5–14, 15–24 or 25+ years before age t with corresponding parameters θ_{15-24}, θ_{25+} and ϕ_{age} and γ_z are factors for attained age and exposure rate, respectively. We refer to Eq. (5) as the "BEIR VI" model when fitted to the pooled 11 miners cohort, and as "PUMA full" or "PUMA sub" when fitted to the full PUMA cohort or to the 1960+ sub-cohort, respectively, the latter comprising miners hired in 1960 or later. Note that in PUMA models the exposure rate factor γ_z

[2] Editor's note. The online version of this article (DOI: https://doi.org/10.1667/RADE-24-00060.1) contains supplementary information that is available to all authorized users.

TABLE 1
Overview of all Considered Risk Models and Associated Cohort Data

Model name	Reference	Cohort	Equation	Miners	PYR*	Lung cancer deaths
BEIR VI	NRC 1999 (*6*)	Pooled cohort of 11 studies	(*5*)	67,897	1,155,453	2,799
PUMA full	Kelly-Reif et al. 2023 (*18*)	PUMA cohort	(*5*)	119,709	4,125,533	7754
PUMA sub	Richardson et al. 2022 (*28*)	PUMA 1960+ sub-cohort	(*5*)	57,873	1,887,092	1217
(Adjusted) Jacobi	Jacobi 1993 (*31*)	Pooled cohort of 6 studies	(*6*)	28,702	584,072	912
Joint CZ+F	Tomasek et al. 2008 (*32*)	Pooled Czech and French cohort	(*7*)	10,100	248,782	574
Wismut full	Kreuzer et al. 2018 (*7*)	German uranium miners cohort	(*8*)	58,974	2,332,008	3,942
Wismut sub	Kreuzer et al. 2018 (*7*)	German uranium miners 1960+ sub-cohort	(*7*)	26,765	956,776	495

* PYR, person-years at risk.

accounts for the annual exposure rate, whereas in the classical BEIR VI model γ_z corresponds to the cumulative exposure rate.

The adjusted Jacobi model is the classical Jacobi model in (*31*) adjusted by the factor 0.83 to account for overestimation (*2*). It reads with time since exposure TE, age at exposure $AE = t - TE$, and cumulative exposure $W(t)$ in WLM at age t in years,

$$ERR(t) = 0.83 \sum_{TE \le t} \alpha(AE)\theta(TE)W(AE), \tag{6}$$

with AE-specific parameters $\alpha(AE)$ and TE-specific parameters $\theta(TE)$. Note that although this model is based on six cohorts, the modifying effect structure of time since exposure and age at exposure was estimated solely from the Czech cohort. The parameter estimates in $\alpha(AE)$ are adjusted to match the meta-estimate for ERR per WLM derived from all six cohorts (*31*).

The generic parametric risk models for ERR at age t read,

$$ERR(t) = \beta W(t)\exp\{\alpha(AME(t) - 30) + \varepsilon(TME(t) - 20)\}, \tag{7}$$

$$ERR(t) = \beta W(t)\exp\{\alpha(AME(t) - 30) + \varepsilon(TME(t) - 20) + \psi(ER(t) - 3)\} \tag{8}$$

with cumulative exposure $W(t)$ in WLM and continuous effect modifiers age at median exposure $AME(t)$ in years, time since median exposure $TME(t)$ in years and cumulative exposure rate $ER(t)$ in WL with corresponding parameters β, α, ε and ψ. We consider Eq. (7) fitted on two different cohorts, namely the joint Czech and French and the German uranium miners sub-cohort with miners hired in 1960 or later (Wismut 1960+ sub-cohort). Equation (7) fitted on the joint Czech and French cohort is referred to as the "Joint CZ+F" risk model. We call Eq. (7) fitted on the Wismut 1960+ sub-cohort with follow-up 2013 "Wismut sub", whereas Eq. (8) was fitted to the full German uranium miners cohort and is referred to as 'Wismut full". The parameter estimates differ between cohorts and only Wismut full incorporates an exposure rate effect with $\psi \neq 0$.

All considered risk models include unknown parameters (indicated by Greek letters) which are estimated using Maximum-Likelihood methods based on miners cohort data. In total, we consider four categorical risk models (BEIR VI, PUMA full, PUMA sub, adjusted Jacobi) and three parametric continuous risk models (Joint CZ+F, Wismut full, Wismut sub) (Table 1). Here, the terms "categorical" and "parametric/continuous" refer to the categorical or continuous nature of the effect-modifying variables. Among all seven considered models, three categorical models (BEIR VI, PUMA full, PUMA sub) and one parametric model (Wismut full), account for an exposure rate effect. The explicit parameter estimates for all risk models can be found in Supplementary Materials, Section C (https://doi.org/10.1667/RADE-24-00060.1.S1) or the corresponding references. For the actual calculation of lifetime risk estimates, parameter estimates are plugged into the corresponding risk model structure.

Exposure Scenario

As exposure scenarios, we consider the internationally well-accepted default choice for LEAR calculation with occupational exposure of 2 WLM from age 18–64 (94 WLM total cumulative exposure over lifetime (WLM/life), moderate exposure) as used in (*11*). Furthermore, we use three scenarios calculated from mean exposures during employment of miners from the Wismut cohort depending on the period of begin of employment (1946–1954: 1,750 WLM/life, "very high exposure"; 1955–1970: 352 WLM/life, "high exposure"; and 1971–1989: 54 WLM/life, "low exposure"). The mean exposure was determined by averaging the annual exposures of miners (with WLM > 0) by age. The constructed exposure scenarios differ considerably in shape and yearly exposure (Fig. 2).

Comparison of Lifetime Risk Measures

Three additional lifetime risk measures were calculated: The risk of exposure-induced death REID [first introduced in (*34*) and employed in (*13*, *35*)], the excess lifetime risk ELR (*36*) and the radiation attributable decrease of survival RADS (*15*). The central difference of the additionally considered lifetime risk measures compared to the LEAR is the explicit accounting for radon exposure in the survival function. The LEAR approach assumes that radon exposure affects only the explicit lung cancer risk but not the survival function. Survival under exposure shall be denoted by $S_E(t \mid a)$ and baseline survival by $S_0(t \mid a) = S(t \mid a)$, emphasizing that $S_0(t \mid a)$ does not depend on exposure. It holds,

$$REID_E(a) = \int_a^\infty r_E(t)S_E(t|a)\ dt - \int_a^\infty r_0(t)S_E(t|a)\ dt$$

$$= \int_a^\infty r_0(t)ERR(t)S_E(t|a)\ dt,$$

$$ELR_E(a) = \int_a^\infty r_E(t)S_E(t|a)dt - \int_a^\infty r_0(t)S_0(t|a)dt,$$

$$RADS_E(a) = \lim_{t\to\infty} \frac{S_0(t|a) - S_E(t \mid a)}{S_0(t \mid a)} = 1 - \lim_{t\to\infty} \frac{S_E(t|a)}{S_0(t|a)}.$$

As for the LEAR, we investigate $a = 0$ and write $REID = REID_E(0)$, $ELR = ELR_E(0)$ and $RADS = RADS_E(0)$. To calculate these additional lifetime risk measures, assumptions on the survival function affected by exposure are necessary. Analogously to $S_0(t) = e^{-\int_0^t q_0(u)\ du}$ we set $S_E(t) = e^{-\int_0^t q_E(u)\ du}$ where $q_E(u)$ describes the all-cause mortality rate at age u affected by exposure. For computation of $S_E(t)$ we employ the approximation $\widetilde{S}_E(t) = e^{-\sum_{u=0}^{t-1} q_E(u)}$ and assume that radon exposure only influences the risk for lung

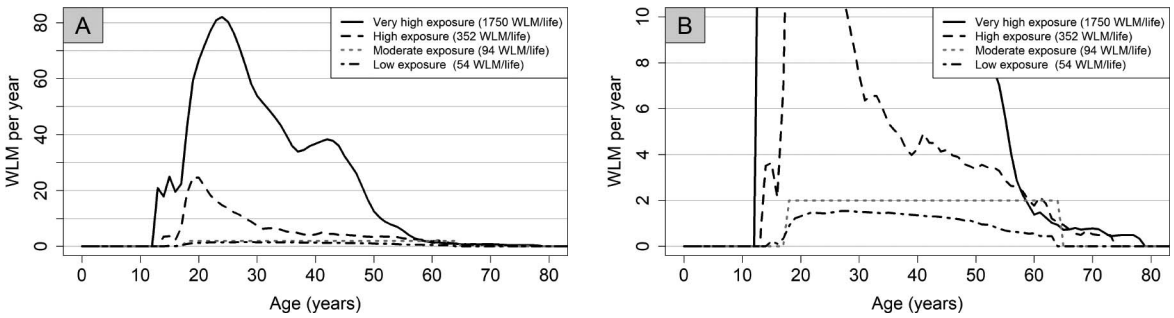

FIG. 2. Exposure to radon progeny in WLM per year by age for the four considered exposure scenarios with total cumulative exposure in parentheses (panel A). Panel B differs in the scale of the y-axis only and gives a more focused view on lower exposures per year.

cancer mortality. Therewith, $q_E(u)$ differs from $q_0(u)$ by an increased lung cancer mortality rate. Hence, $q_E(u) = q_0(u) + r_0(u)ERR(u)$ and

$$\widetilde{S}_E(t) = \widetilde{S}_0(t)e^{-\sum_{u=0}^{t-1} r_0(u)ERR(u)}.$$

Employing the same approximation as for the LEAR, the final approximated formulas for all considered lifetime risk measures are

$$LEAR \approx \sum_{t\geq 0} r_0(t)ERR(t)\widetilde{S}_0(t),$$

$$REID \approx \sum_{t\geq 0} r_0(t)ERR(t)\widetilde{S}_E(t),$$

$$ELR \approx \sum_{t\geq 0} r_0(t)(1+ERR(t))\widetilde{S}_E(t) - \sum_{t\geq 0} r_0(t)\widetilde{S}_0(t),$$

$$RADS \approx 1 - e^{-\sum_{t\geq 0} r_0(t)ERR(t)}.$$

RESULTS

All possible variations of considered mortality rates, risk models and exposure scenarios result in $3 \times 7 \times 4 = 84$ different LEAR and LEAR per WLM estimates (Table 2 and Fig. 3). Note that LEAR estimates are obtained from LEAR per WLM estimates by multiplying the LEAR per WLM by the scenario-specific cumulative exposure in WLM. The LEAR estimates themselves vary heavily from 0.45% to 151.27% and increase monotonically with exposure (as would be expected). Notably, LEAR estimates exceeding 100% are observed for the PUMA sub-risk model and the very high-exposure scenario for all three reference populations. Although absolute risks (i.e., probabilities) are typically bounded by 100%, the LEAR methodology allows for unbounded values, reflecting substantial risk increases under extreme exposure scenarios (see discussion). The LEAR per WLM spans from 0.58×10^{-4} to 8.80×10^{-4}. Roughly, this implies that among 100 individuals with a cumulative occupational radon exposure of 100 WLM, there would be an additional 0.58 to 8.80 (excess) lung cancer deaths attributed to this exposure over their lifetime.

Effects of Reference Populations

The LEAR per WLM estimates for the population of heavy smokers closely align with those of the ICRP reference population, whereas light smokers consistently yield lower estimates. The heavy smoker population (baseline lifetime risk of 5.27%), exhibits higher LEAR per WLM estimates than light smokers (baseline lifetime risk of

TABLE 2
Results for LEAR per WLM $\times 10^4$ Estimates for All Considered Exposure Scenarios, Reference Populations and Risk Models

Exposure scenario	Population	BEIR VI	PUMA full	PUMA sub	Adj. Jacobi	Joint CZ+F	Wismut full	Wismut sub
Very high	Heavy smokers	2.13	4.82	8.64	2.75	4.64	0.81	3.75
	ICRP	2.10	4.47	8.06	2.61	4.67	0.80	3.56
	Light smokers	1.54	3.70	6.50	2.07	3.36	**0.58**	2.81
High	Heavy smokers	3.70	5.08	**8.80**	2.75	4.33	0.87	3.45
	ICRP	3.57	4.80	8.36	2.60	4.36	0.87	3.28
	Light smokers	2.73	3.89	6.60	2.09	3.14	0.63	2.59
Moderate	Heavy smokers	6.15	5.38	7.98	3.41	4.68	1.17	4.50
	ICRP	5.97	5.74	7.50	3.20	4.58	1.13	4.21
	Light smokers	4.42	4.39	6.02	2.56	3.41	0.85	3.40
Low	Heavy smokers	6.25	5.55	8.51	3.19	4.80	1.14	4.28
	ICRP	6.12	5.24	8.04	3.01	4.74	1.12	4.03
	Light smokers	4.50	4.23	6.40	2.39	3.48	0.83	3.22

Note. Minimum and maximum values are bolded.

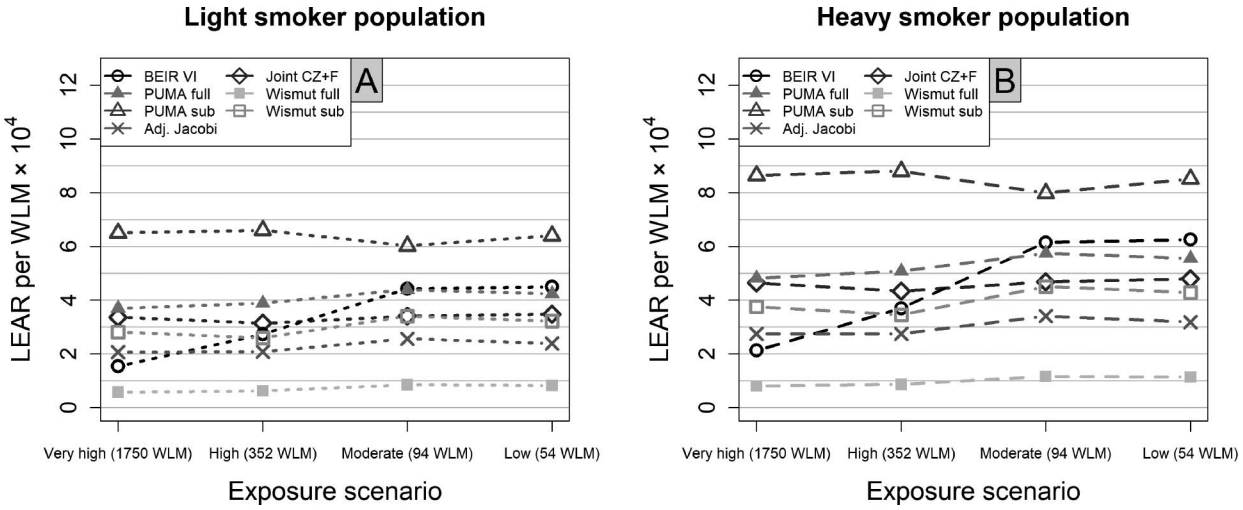

FIG. 3. LEAR per WLM $\times$ 10^4 for the four considered exposure scenarios and all considered risk models. Results of different smoker populations in different plots (panel A: light smokers, panel B: heavy smokers). A plot for the ICRP population is omitted here as it closely mirrors the heavy smokers panel.

4.12%). For comparison, the baseline lifetime risk of the ICRP reference population is 4.83%. The differences between reference populations can be explained when the factors $r_0(t)$ and $S(t)$ are interpreted as weighting factors $r_0(t)S(t)$ for the $ERR(t)$ in the LEAR calculation [Eq. (3)] since these weights completely depend on mortality rates (Fig. 4). The weights $r_0(t)S(t)$ are notably lower for light smokers than for heavy smokers and ICRP reference at all ages except for ages 80+. This characteristic roughly translates to the age-specific contributions $r_0(t)S(t)ERR(t)$ to the final LEAR estimate. Thus, the population of light smokers produces lower LEAR per WLM estimates because lung cancer rates are lower among light smokers, whereas the results for the ICRP reference population and the heavy smoker population are almost similar. Using male-specific compared to sex-averaged mortality rates results in higher lifetime risk estimates across all reference populations, risk models, exposure scenarios, and lifetime risk measures (Supplementary Materials, Section B; https://doi.org/10.1667/RADE-24-00060.1.S1).

Effects of Risk Models and Exposure Scenarios

Despite large differences in the four considered exposure scenarios, the resulting respective LEAR per WLM is notably constant (reading Fig. 3 horizontally). Only estimates with BEIR VI deviate considerably and exhibit an increasing trend with decreasing exposure. Notably, this does not apply to PUMA models, although they have a very similar model structure. Generally, the risk models influence LEAR estimates essentially (Fig. 5). There are large differences in age-at-exposure effects and the magnitude and shape of $ERR(t)$, depending on the chosen risk model. All risk models peak at different ages at exposure. The Joint CZ+F and Wismut sub model exhibit distinct $ERR(t)$ patterns, despite originating from an identical risk model structure [Eq. (7)]. This affects the age-specific contribution $r_0(t)ERR(t)S(t)$ to the LEAR (Fig. 5B). Multiplying the $ERR(t)$ (Fig. 5A) with $r_0(t)S(t)$ at every age t yields the curves from Fig. 5B. Integrating these curves over all ages t yields the LEAR estimate for each risk model.

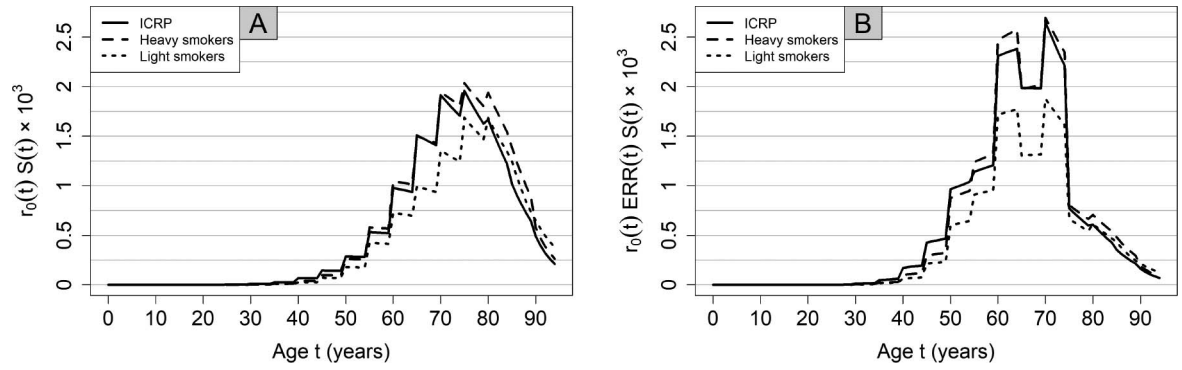

FIG. 4. Illustration of the influence of the three reference populations on LEAR calculation. Panel A: Product of baseline lung cancer mortality rates $r_0(t)$ and survival $S(t)$ $\times$ 10^3; panel B: age-specific contribution to the *LEAR*, $r_0(t)ERR(t)S(t)$ $\times$ 10^3, both for every age t with the BEIR VI risk model and the moderate exposure scenario of 2 WLM from age 18–64 years.

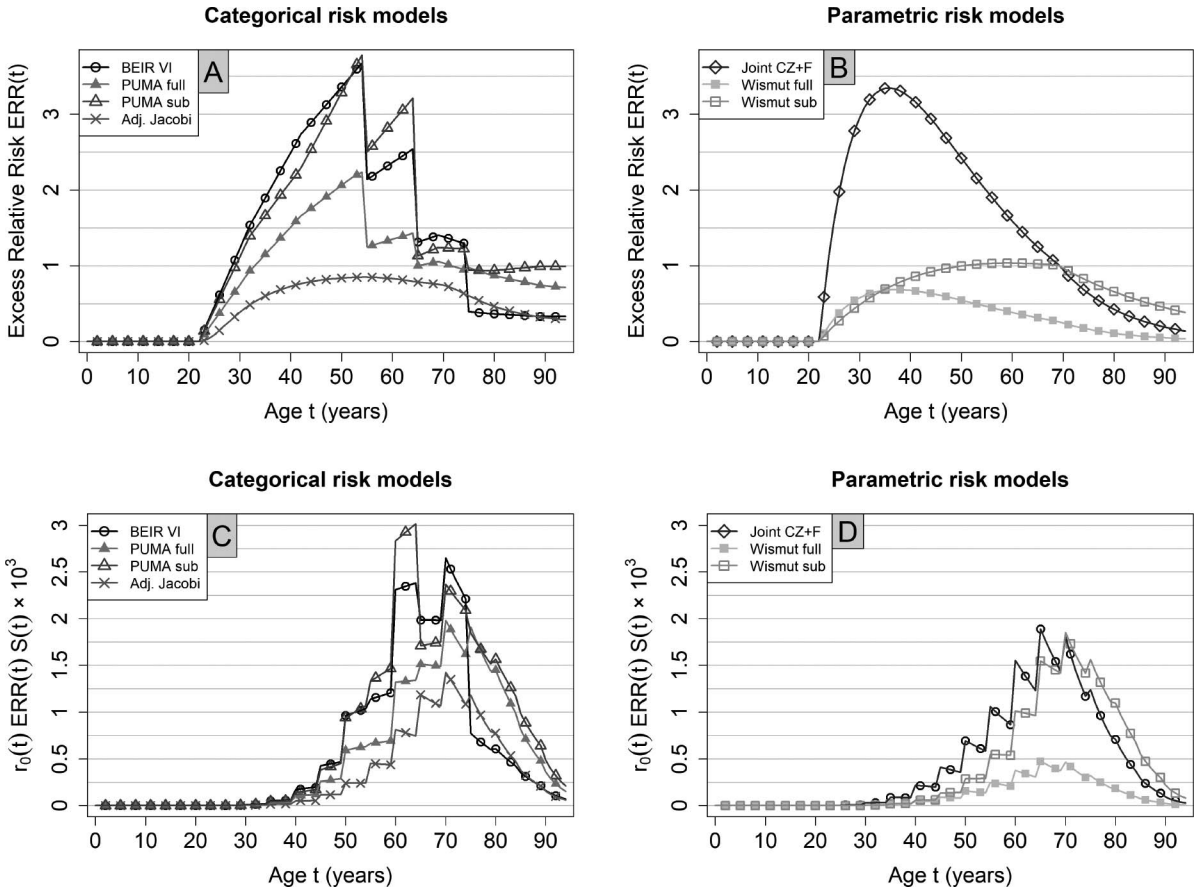

FIG. 5. Excess relative risks of different risk models (panel A: categorical models, panel B: parametric models) and their age-specific contribution to the *LEAR*, $r_0(t)ERR(t)S(t) \times 10^3$ (panels C and D, respectively) for the moderate exposure scenario of 2 WLM from 18–64 years and the ICRP reference population. For readability, data points are displayed only for every third age.

In the Supplementary Materials, Section D (https://doi.org/10.1667/RADE-24-00060.1.S1), further sensitivity analysis on the effects of varying annual exposure and differences between single acute and protracted homogeneous exposure across age are shown for all lifetime risk measures and risk models. Results show stable or slightly declining lifetime risk estimates per WLM for varying annual exposure for all risk models, except for the BEIR VI risk model. Comparing acute exposure at different ages to protracted homogeneous exposure across age reveals substantial differences in lifetime risk estimates especially influenced by the consideration of exposure rate in risk models. Depending on age at acute exposure, the excess lifetime risks (per WLM) differ roughly by a factor of two for all risk models and all lifetime risk measures.

Comparison of Lifetime Risk Measures

Excess lifetime risks were calculated for three additional lifetime risk measures for all combinations of reference populations, risk models and exposure scenarios (Fig. 6 and Table 3 for moderate exposure). There are only slight differences in results for ELR, REID and LEAR, except for very high exposures. RADS estimates are larger than results for the other three measures. We observe the monotonicity ELR $\leq$ REID $\leq$ LEAR $\leq$ RADS (per WLM) for all combinations except for the very high-exposure scenario. At this very high exposure, the monotonicity between lifetime risk measures is ELR $\leq$ REID $\leq$ RADS $\leq$ LEAR (per WLM) for all reference populations and risk models.

DISCUSSION

Lifetime risk estimates depend on several calculation components and assumptions, each being potentially the source of variability and uncertainty. The extensive variation in the calculated LEAR and LEAR per WLM across different reference populations, risk models and exposure scenarios highlight the complexity of assessing the health risks associated with radon exposure. In case of the LEAR for lung cancer related to occupational radon exposure, the observed LEAR per WLM estimates range from 0.58×10^{-4} to 8.80×10^{-4}, underscoring the considerable impact of the calculation components. We identified mortality rates and risk models as the most influential components. LEAR per WLM exhibits only low variation across different exposure scenarios for all risk models except for the BEIR VI model.

Tomasek et al. (*11*) contributed to understanding the variability of LEAR estimates by comparing effects of

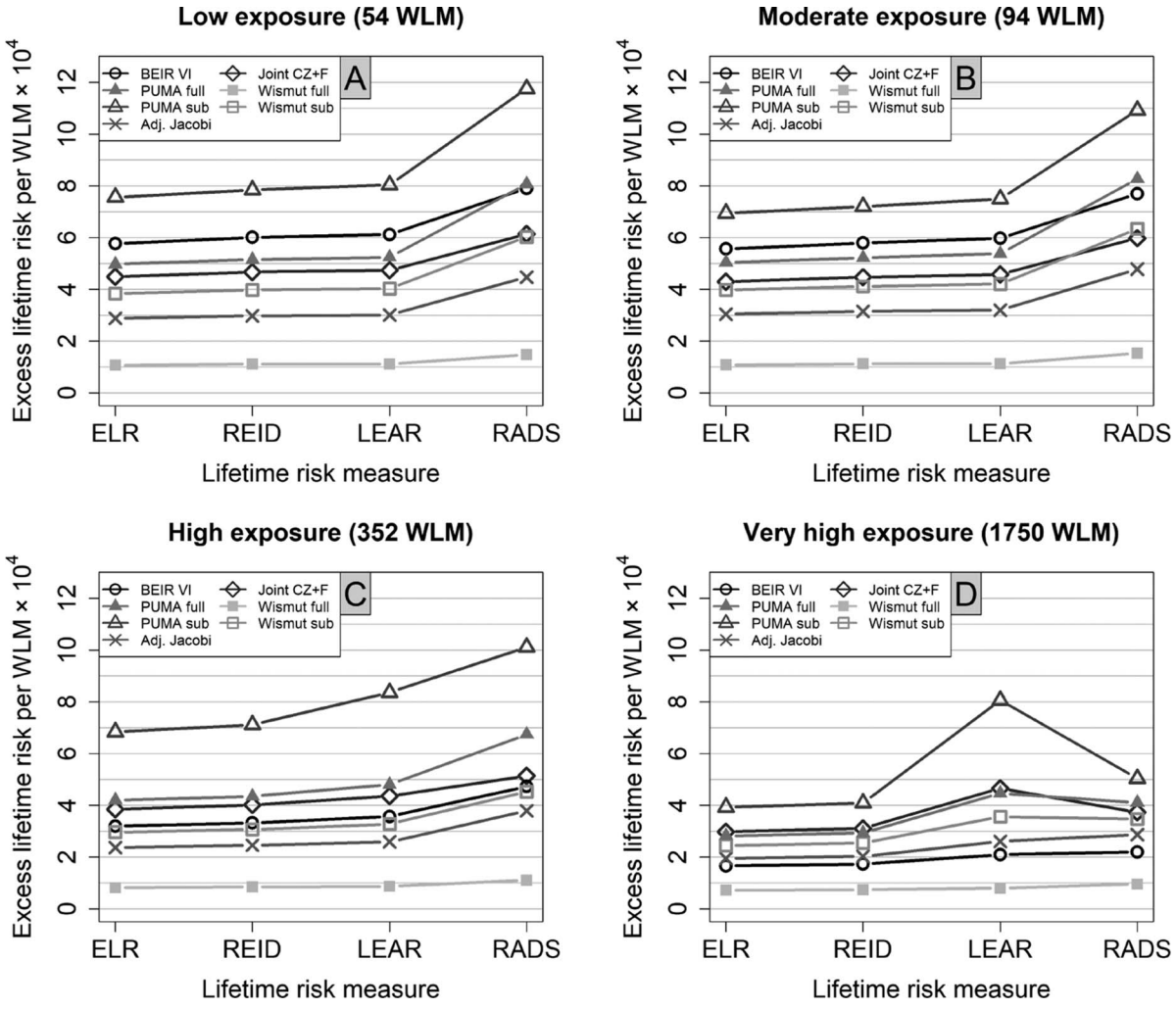

FIG. 6. Excess lifetime risk estimates per WLM $\times$ 10^4 for different lifetime risk measures and the four considered exposure scenarios (panels A–D) were calculated with the ICRP reference population.

different risk models from uranium miners studies and their impact on the radon dose conversion factor. For the REID, Hunter et al. (*13*) performed a sensitivity analysis by additionally accounting for differences in exposure, sex and smoking behavior in the risk model for a U.S. population. The lifetime risk measures LEAR, ELR and REID for occupational exposure were compared for a simple linear risk model in Kellerer et al. (2001) showing clear differences

TABLE 3

Results for Excess Lifetime Risks per WLM $\times$ 10^4 for Different Lifetime Risk Measures, Reference Populations and Risk Models for the Moderate Exposure Scenario of 2 WLM from Age 18–64 Years

Lifetime risk measure	Population	BEIR VI	PUMA full	PUMA sub	Adj. Jacobi	Joint CZ+F	Wismut full	Wismut sub
RADS	Heavy smokers	7.34	7.84	10.43	4.54	5.66	1.45	6.04
	ICRP	7.69	8.27	**10.92**	4.78	5.98	1.53	6.34
	Light smokers	5.99	7.03	9.23	3.96	4.64	**1.19**	5.30
LEAR	Heavy smokers	6.15	5.74	**7.98**	3.41	4.68	1.17	4.50
	ICRP	5.97	5.38	7.50	3.20	4.58	1.13	4.21
	Light smokers	4.42	4.39	6.02	2.56	3.41	**0.85**	3.40
REID	Heavy smokers	5.96	5.57	**7.66**	3.35	4.57	1.16	4.40
	ICRP	5.79	5.22	7.20	3.15	4.47	1.13	4.11
	Light smokers	4.32	4.28	5.82	2.52	3.35	**0.85**	3.34
ELR	Heavy smokers	5.72	5.38	**7.37**	3.23	4.39	1.11	4.24
	ICRP	5.56	5.04	6.94	3.04	4.30	1.08	3.98
	Light smokers	4.17	4.15	5.63	2.45	3.23	**0.82**	3.23

Note. Minimum and maximum values are bolded for every lifetime risk measure.

only at higher exposures (*9*). In the analysis at hand, for the first time variability in the lifetime risk estimates were investigated by directly comparing four lifetime risk measures ELR, REID, LEAR and RADS for different reference populations, risk models and heterogeneous occupational exposure scenarios from real data (German uranium miners cohort).

Effect of Mortality Rates and Reference Populations

Smoking is the greatest risk factor for lung cancer (*37*, *38*) and the interaction effect of smoking and radon on lung cancer is not yet fully understood (*8*). We investigated whether strong differences in the smoking behavior of reference populations are reflected in the corresponding LEAR estimates by constructing a sex-averaged light and heavy smoker reference population. For comparison, the widely accepted ICRP sex-averaged reference population from (*3*) was also considered. In the Supplementary Materials (Section B; https://doi.org/10.1667/RADE-24-00060.1.S1), lifetime risk sensitivities with male-specific mortality rates are additionally investigated.

This analysis clearly showed that employing a smoking reference population results in elevated LEAR per WLM estimates. This confirms the results published in the literature (*39*) that smoking behavior in a reference population influences lifetime risk estimates heavily. This can be explained by acknowledging that smoking amplifies baseline lung cancer risk $r_0(\cdot)$, which enters the LEAR calculation linearly. In particular, the risk models used are not adjusted for smoking, resulting in the implicit assumption of a multiplicative interaction between smoking and radon on the lung cancer risk here, in line with the findings (*8*, *40*). However, other epidemiological studies suggest a sub-multiplicative (*6*, *7*, *30*, *41*) or additive interaction (*42*). While we retain the multiplicative model due to the heterogeneous nature of smoking adjustments in the existing literature and compatibility issues with our heavy- and light-smoker reference rates, exploring lifetime risk estimates with smoking-adjusted risk models could offer interesting insights.

In that manner, if a LEAR for a smoker is of interest, it may be reasonable to compute smoking-specific LEAR estimates with mortality rates from smoker populations and risk models fitted on suitable cohorts of smoking persons with comparable smoking behavior. Especially between countries heavy differences in lung cancer mortality rates may occur not only due to smoking behavior, but also because of variable health care and medical standards (*43*, *44*). Likewise, if a LEAR for a specific country and sex is of interest, country- and sex-specific lifetime risk estimates with corresponding sex-specific mortality rates should be calculated, as similarly recommended for detriment calculations (*45*). Additional analyses (Supplementary Materials, Section B; https://doi.org/10.1667/RADE-24-00060.1.S1) showed an overall increased lifetime risk when calculated with male-specific baseline mortality rates, with lifetime risk variability closely aligning with results obtained using sex-averaged rates. It is expected that lifetime risks calculated with female-specific mortality rates would be lower correspondingly. However, calculating lifetime risks with female-specific rates and risk models derived from male uranium miners amplifies the risk transfer issue, which is why we excluded female-specific analyses in this study.

This analysis further revealed that the ICRP reference population results in remarkably similar LEAR estimates as when using the heavy smoker population and produces generally high LEAR and LEAR per WLM estimates, too. The same result was observed for male-specific reference populations (Supplementary Materials, Section B; https://doi.org/10.1667/RADE-24-00060.1.S1). This indicates that ICRP reference rates rather represent smokers than non-smokers. In particular, LEAR estimates with the ICRP reference population overestimate absolute risks for non-smokers and underestimate (but to a lesser extent) risks for smokers, compare (*2*). However, smoking behavior has evolved over the years and the smoking population rates in this analysis are derived from population data from more recent years (2016–2019) compared to the ICRP reference rates (1993–1997). This may contribute to explaining the results obtained for the ICRP population. Hence results incorporating ICRP rates must be interpreted with care.

Effect of Risk Models

Varying risk models lead to a large variability in LEAR estimates. This becomes clear when interpreting the LEAR as a weighted average of the ERR(·) term (which depends on risk models) with weights $r_0(\cdot)S(\cdot)$. Even risk models with identical ERR(·) term structures (Joint CZ+F and Wismut sub) inherit different parameter estimates and result in distinct LEAR estimates. The BEIR VI model imposes high variation of the LEAR per WLM for different exposure scenarios due to its strong inverse exposure rate effect. The PUMA models use fewer categories for exposure rate and annual exposure rate instead of cumulative mean exposure rate compared to the BEIR VI model. This explains the lower variability of results from PUMA models for different exposure scenarios despite the structural model similarity to BEIR VI. Although Wismut full also incorporates an inverse exposure rate effect, the impact on LEAR per WLM is considerably weaker because of the continuous nature of the model. In considered risk models without exposure rate effect modifiers, the ERR(t) (and therefore the LEAR) increases linearly when the exposure is increased. This results in remarkably stable LEAR per WLM estimates (Fig. 3).

The Wismut full risk model results in remarkably small lifetime risk estimates, particularly compared to the PUMA full model. This is a direct consequence of the fact that parameter estimates of Wismut full are also comparatively small. Compared to other cohorts in PUMA, the Wismut full cohort is characterized by longer duration of employment combined with rather high-cumulative radon exposure at low-exposure rates [(*20*) see table 1]. These structural differences might result in the substantial

differences between parameter and lifetime risk estimates from PUMA full and Wismut full risk models, despite the fact that the Wismut cohort makes up over half of the PUMA study data (2.3 out of 4.1 million person-years at risk) (*33*). In models from the 1960+ sub-cohorts (Wismut sub, PUMA sub), differences between parameter and lifetime risk estimates are less pronounced, which supports that uncertainties in exposure assessment in the early years of the Wismut cohort might also play an important role.

Exposure assessment in the early years of uranium mining relied on expert ratings rather than on direct measurements, increasing the potential for measurement error (*20*). Inconsistencies in exposure assessment across different periods can lead to differences in risk estimates. Improved exposure assessment quality, such as in the 1960+ sub-cohorts of PUMA and Wismut, reduces measurement error and yields more accurate risk estimates at low exposures and exposure rates. Ongoing research explores the effects of measurement error in the early years within the Wismut cohort (*46*, *47*). Measurement errors are one of many possible explanations for the differences in risk estimates at low exposures and exposure rates between the full and the 1960 + sub-cohort (*29*).

Note that the inverse exposure-rate effect, also known as the protraction enhancement effect, plays a critical role in the observed LEAR variability. This effect describes a decrease in (excess) relative risk for higher exposure rates and was demonstrated in many miners studies (*6*, *8*). However, this effect diminished when the analyses were limited to miners with low levels of cumulative exposure in WLM or those employed in more recent times (*30*, *32*), but it was shown to be statistically significant for the first time at such exposure levels in the PUMA 1960+ sub-cohort (*28*).

Generally, variations in LEAR and LEAR per WLM depending on the choice of risk model emerge from differences in underlying cohorts, risk model structures and assumptions for the fitting process of risk model and cohort. Especially decisions in the necessary data grouping process prior to applying Poisson regression on cohort data are highly susceptible to influencing risk model estimates (*48*). Also, the design of baseline stratification, e.g. with the statistical software Epicure (*49*), influences risk model parameter estimates and corresponding lifetime risk estimates (*50*). In categorical risk models, ERR(t) curves might exhibit abrupt changes at specific ages, times since exposure, and exposure rates as given by the model. This raises concerns about the discontinuity of these models. On the other hand, parametric risk models, which also incorporate effect modifiers, provide a smoother and more intuitive transition over age. Therefore, it seems more reasonable and plausible to use parametric risk models (or generally models with a continuous structure), such as those fitted on a representative cohort (cf. (*28*)), for calculating LEAR estimates. Especially for the BEIR VI model, there were attempts to use a smooth version of this categorical risk model by employing spline functions, see e.g. (*51*).

While our analyses focus on established excess relative risk (ERR) models, we acknowledge that lifetime risk estimates incorporating excess absolute risk (EAR) results for lung cancer related to occupational radon exposure are available, as in the electronic attachment of (*8*). The EAR approach offers an alternative perspective on lifetime risk assessment, although comprehensive application across all our studied cohorts is technically constrained. Future research may explore further EAR models, where possible, potentially enriching the interpretation of radon-related risks.

Effect of Exposure Scenario

In the early years of uranium mining at Wismut after 1945, miners were exposed to high levels of radon and its progeny, and had very different exposure situations than miners later. Due to improved measures for occupational safety like air ventilation, the mean exposure at the Wismut reduced constantly from 1955 and reached levels of international radiation protection standards in the 1970s (*52*). The large size of the Wismut cohort study enabled us to construct realistic occupational exposure scenarios with heterogeneous exposure rates over age (low, high, and very high exposure) additional to the default choice of 2 WLM from age 18–64 years (moderate exposure). In particular, the exposure scenario reflecting begin of employment in 1946–1954 (very high exposure) shows very high exposures at early ages due to missing protective measures in the mines. Likewise, but to a considerably lesser extent, this holds for the exposure scenario with begin of employment 1955–1970 (high exposure). On the other hand, the scenarios for begin of employment 1970–1989 (low exposure) and the ICRP default (moderate exposure) show homogeneous exposure over age without clear peaks.

Despite substantial differences in exposure scenarios, the LEAR per WLM remains relatively constant for all risk models except for BEIR VI with a threefold increase from highest to lowest exposure scenario.

The LEAR per WLM tends to slightly increase (except for the PUMA sub-model) for the two exposure scenarios with moderate and low cumulative exposure compared to the other two exposure scenarios. Regarding risk models without an exposure rate effect modifier (Adj. Jacobi, Wismut sub and Joint CZ+F), this is because of the more homogeneous exposure in age in these two scenarios – in contrast to the other two scenarios with high and very high-cumulative exposure where the majority of exposure is at earlier ages. At these earlier ages, LEAR and LEAR per WLM are less affected by exposure (see Fig. 4). However, the effect is small. On the other hand, risk models with an exposure rate modifier (BEIR VI, Wismut full, PUMA full and PUMA sub) are additionally affected by variable cumulative exposure. LEAR estimates with the BEIR VI model are heavily affected by this categorical-inverse-exposure rate effect as mentioned before. PUMA sub behaves differently because of its unique feature of an increasing factor for time since exposure 25–34 years

ago Supplementary Materials, Section C; https://doi.org/10.1667/RADE-24-00060.1.S1). This parameter estimate is likely an artifact stemming from the reduced statistical power of the PUMA sub-cohort compared to the PUMA full cohort.

Note that stable LEAR per WLM estimates translate to a roughly linear relationship between LEAR and exposure, e.g. a doubling in yearly exposure roughly doubles the LEAR as well. The LEAR measure is technically unbounded and may result in unreasonable large values for extreme exposure scenarios. LEAR estimates exceeding 100% are to be interpreted cautiously.

Combining risk models derived from low-exposure cohorts with extreme exposures may not seem reasonable at first glance. Risk models without an exposure rate effect modifier tend to be suitable only for low-exposure scenarios (*7*, *53*). However, the goal of this sensitivity analysis was to particularly investigate and combine extreme cases for a better understanding of LEAR drivers.

In summary, the stability of LEAR per WLM for changing exposure scenarios implies no benefit from employing complex over simple exposure scenarios when calculating lifetime risks for realistic exposures. This confirms the default exposure scenario of 2 WLM from age 18–64 years for a working population as a suitable and reasonable choice.

Effect of Lifetime Risk Measures

Comparisons between the different lifetime risk measures ELR, REID, LEAR and RADS, provide valuable insights. The monotonicity observed for all combinations of major components except for the very high-exposure scenario, i.e. ELR $\leq$ REID $\leq$ LEAR $\leq$ RADS (per WLM), underscores the relationship between these measures.

All four measures preserve mostly the behavior as seen for the LEAR regarding risk model and reference population effects. RADS is the only measure where estimates with ICRP mortality rates are higher than estimates with the heavy smoker reference population. This is because RADS estimates are independent of all-cause mortality rates in contrast to ELR, REID and LEAR.

The three measures ELR, REID and RADS (per WLM) are more severely affected by the very high-exposure scenario than the LEAR because these measures account for excess risk in the survival function.

This can be observed by comparing LEAR to, for example, RADS (per WLM) across varying exposure scenarios, moving from lower to higher levels (Fig. 6). For the very high-exposure scenario, LEAR per WLM stands out as it is barely affected by exposure. RADS per WLM decreases considerably for higher levels of exposure. This confirms the stability of LEAR per WLM in capturing the exposure-response relationship for varying exposure scenarios.

The observed relation ELR $\leq$ REID $\leq$ LEAR (per WLM) can be mathematically proven to hold for all combinations of calculation components. Assuming a harmful effect of radon exposure, it holds $r_E(t) \geq r_0(t)$ and $S_E(t|a) \leq S_0(t \mid a)$ for all $t, a \geq 0$. Evidence shown in Supplementary Materials, Section E (https://doi.org/10.1667/RADE-24-00060.1.S1),

$$ELR(a) \leq REID(a) \leq LEAR(a) \ (per\ WLM),$$

$$REID(a) \leq RADS(a) \ (per\ WLM).$$

For moderate excess absolute risks, it even holds ELR $\leq$ REID $\leq$ LEAR $\leq$ RADS (per WLM). At higher exposures the indefinite growth of LEAR exceeds RADS, breaking the inequality.

A critical aspect in estimating ELR, REID, and RADS is the modeling choice of $S_E(t)$. Since there is currently no reliable evidence that radon can cause diseases other than lung cancer (*54*, *55*), we assume that radon exposure affects solely the lung cancer risk, i.e. $q_E(u) = q_0(u) + r_0(u)ERR(u)$ for all ages u.

LEAR exhibits linear growth for increase in lung cancer mortality rates r_0 or yearly exposure whereas the other three measures grow sublinear due to the additional exponential term in the survival function. This mathematical elegance makes the LEAR particularly appealing (Supplementary Materials, Section D; https://doi.org/10.1667/RADE-24-00060.1.S1). Further, for low lung cancer mortality rates r_0 or yearly exposure, values for LEAR, REID and ELR (per WLM) are similar, while RADS values deviate. The similarity of REID, ELR and LEAR is also observed in detriment calculations (*45*).

We conclude that LEAR and REID are the most practicable lifetime risk measures, in accordance with previous findings (*9*, *10*). Both quantities behave very similar for low to moderate exposures and the LEAR is easier to compute since it avoids the ambiguous radiation-affected survival $S_E(t)$. The ELR has a convenient statistical interpretation but is not linear in increase in lung cancer mortality rates r_0 or yearly exposure and may even turn negative. We recommend sticking with the LEAR approach for its broad applicability across most exposure scenarios encountered today. However, for notably higher exposures, the linearity of LEAR and its indefinite growth is unrealistic, and we recommend employing the REID for such situations. RADS serves well as a comparative tool between risk models, by being less influenced by external baseline mortality rates compared to the other lifetime risk measures (*15*).

Calculation Components with Minor Influence

Prior analyses showed that latency time L, minimum age at risk a, the choice of approximation formula for the survival curve $S(t)$ and maximum age a_{max} have negligible impact on lifetime risk estimates similar to results of sensitivity analyses on radiation detriment (*45*). However, in our lifetime risk calculations for lung cancer related to radon exposure the choices of the lag time $L = 5$ and minimum age at risk $a = 0$ are predetermined by the risk model and to not discard early years of life, respectively.

Strengths and Limitations

For the first time in a sensitivity analysis on excess lifetime risks for lung cancer related to radon, new reference

populations mirroring smoking behavior were constructed from WHO data. Further, realistic exposure scenarios derived from the Wismut cohort study were employed in the calculation. This provides a more accurate representation of the actual conditions and radon concentrations that individuals experience in their working environments, and enhances the reliability of risk assessments.

Since no confidence intervals are presented, it is difficult to evaluate whether the presented lifetime risk estimates are statistically compatible. Further, variations in risk models emerge from differences in the underlying cohort and model structure. Smoking is not accounted for in the risk models and thus, any effects of smoking behavior come from amplified baseline lung cancer risks r_0. In that manner, also the risk transfer from miners cohorts to reference populations stays ambiguous [multiplicative risk transfer (*56*)].

Likewise, it is not accounted for sex-specific risks or further individual characteristics. Data for radon effects on females are sparse. Based on the published literature (*57*) we assumed the same *ERR* for females and males.

Future Perspectives

The results on the large impact of reference lung cancer mortality rates on the LEAR encourage to calculate country-specific lifetime risk estimates in future work. Moreover, quantitative estimates for the underlying uncertainty of lifetime risk estimates will sharpen the understanding of variability in lifetime risk estimates.

CONCLUSION

In the calculation of lifetime risk measures, the choice of lifetime risk measure itself and the specific exposure scenario is considerably less important than the used reference population and risk model. The current study confirms the LEAR as a suitable lifetime risk measure for low and moderate exposures and adds evidence that the LEAR is substantially affected by mortality rate changes, especially for lung cancer mortality rates. Thus, reference populations and mortality rates should be selected with care depending on the application of lifetime risk calculations. Further, the internationally typical moderate exposure scenario of 2 WLM from age 18–64 years to represent a working population is further confirmed as a suitable choice. These findings should be considered when using and interpreting lifetime risk measures for radiation protection policy purposes.

SUPPLEMENTARY MATERIALS

Section A: Mortality rates and mixing of populations.
Section B: Lifetime risks for male-specific mortality rates.
Section C: Risk models.
C1: BEIR VI exposure-age-concentration risk model.
C2: PUMA exposure-age-concentration risk model.
C3 Adjusted Jacobi risk model.
C4: Parametric risk models.
Section D: Comparison of lifetime risk measures and additional analyses.
Section E: Mathematical proofs from main paper statements.

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