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Projecting radiation-induced cancer risks across time and populations

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Information on the risks of cancer following radiation exposure comes from a large number of epidemiological studies, and has recently been reviewed by the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR)[1] and by the U.S. Committee on the Biological Effects of Ionising Radiation (BEIR V)[2]. The populations that have been studied include:

(i) over 90,000 survivors of the atomic bombings of Hiroshima and Nagasaki[3];
(ii) 14,000 mostly male patients in the UK treated for ankylosing spondylitis with x-rays[4];
(iii) 83,000 women in eight countries treated for cervical cancer with x- or gamma radiation[5].

Very few epidemiological studies have followed the entire study population until the end of life. For example, the Japanese atomic bomb survivors have so far been followed up to 40 years post exposure, and about two-thirds of this population are still alive. Those who were exposed when young are only now reaching the ages when solid cancers would normally be more likely to occur. Consequently some means is required of projecting risks beyond the period of follow-up in such studies in order to estimate the cancer risk over a whole lifetime.

Another problem concerns how risk estimates derived in one population should be transferred for application to another population. This topic is important in view of the large variation between different countries in the baseline rates for several types of cancer. For example, stomach cancer rates are much higher and lung and breast cancer rates are lower in Japan than the corresponing rates in western populations. Therefore whether or not account is taken of these variations when, say, projecting radiation risks from Japan to the UK can have a large influence on the estimates for individual cancer types, if not on the total cancer risk.

Two models have commonly been used to project risks over time and across populations. Under the absolute (or additive) risk model, the annual absolute excess risk estimated in a study is assumed to remain constant across time and/or across populations. Under the relative (or multiplicative) risk model, the relative risk is assumed to remain constant across time and/or populations. In both cases, the risks may depend on sex and age at exposure. Since the baseline rates for solid cancers increase rapidly with age, the use of a relative risk model over time yields a higher estimate of lifetime risk than does the absolute risk model. More generally, a hybrid model that reduces to either the relative or the absolute model as a special case can be implemented[6]. It is also possible to allow the relative risks to depend on time since exposure as in the BEIR V report[2].

The aim of these models is primarily to provide an empirical fit to the data rather to describe the carcinogenic process, although they might suggest ways in which models for carcinogenesis might be improved. In this paper the epidemiological evidence from various studies concerning the choice of models is reviewed, both for projection across time and projection across populations. The estimates of radiation-induced cancer risk that arise from the application of various projection models are then presented and compared.

Epidemiological evidence

(a) Projection of risks over time

For leukaemia, the temporal pattern of risk observed in studies such as the Life Span Study (LSS) of A-bomb survivors[3] and the Ankylosing Spondylitis Study (ASS)[4] is of a peak within about 4–7 years of exposure followed by a tailing-off in risk. Other studies show a similar pattern for bone cancer. Virtually all of the radiation-induced risk seems to have been expressed within the period of follow-up of these studies, and so the topic of projecting risks beyond the period of follow-up is not of major importance.

However, for the grouping of all cancers other than leukaemia, Shimizu et al.[3] showed that excess deaths in the LSS in the most recent follow-up to 1985 continued to increase over time in direct proportion to the increase in natural cancer mortality with age. Thus, after adjustment for age at exposure, the relative risks appeared to be constant over time. A possible exception concerns those aged less than 10 years at time of exposure, for whom there is a suggestion of a fall in the relative risk. This is especially important since the relative risks increase with decreasing age at exposure, and so this cohort would show the highest lifetime risk if the relative risk were to remain constant until the end of life. However, since those irradiated at less than 10 years are only now attaining the ages at which solid tumours would normally occur, the

absolute excess risk observed to date is lower than in older cohorts. Consequently there is substantial uncertainty in the eventual lifetime risk for the youngest cohort.

The study of the incidence of second cancers in women treated with radiation for cervical cancer [5] also suggested that the relative risk for solid tumours is approximately constant up to at least 30 years following exposure. However, analyses based on the follow-up to 1982 of the ASS [4, 7] revealed a tailing-off in the radiation-induced risk for the grouping of all cancer except leukaemia and colon cancer, both on relative and absolute scales, from about 20 years following exposure. This discrepancy might, in part, be explained by differences in temporal patterns of risk for different cancer types. Whilst the data for individual cancer types are often not sufficiently strong to allow these patterns to be distinguished, some of the analyses that have been conducted will now be reviewed.

For lung cancer, the BEIR IV Committee's [8] joint analysis of miner cohorts exposed to radon indicated a decrease in relative risk from about 15 years following exposure. The BEIR V [2] model for respiratory cancer also allowed for a tailing-off in the relative risk. Although this effect was not statistically significant in the LSS, it was included on the basis of the pattern observed in the ASS [4].

For breast cancer, studies of incidence among the Japanese atomic bomb survivors [9], women in New York State treated for acute postpartum mastitis with x-rays [10], and tuberculosis patients in Massachusetts exposed to multiple chest fluoroscopies [11] suggested constancy of relative risk up to around 40 years following exposure. However, among Canadian tuberculosis patients [12], there appeared to be a decrease in the relative risk of mortality after 35 years following fluoroscopic examinations. The BEIR V combined analysis of these data sets [2], however, suggested that the relative risk for incidence and mortality peaked at about 15–20 years after exposure and then decreased. It will be of interest to see whether such patterns become evident in the individual studies with increased follow-up. A common feature of these studies is that the relative risks were highest at the youngest ages at exposure.

For thyroid cancer, the study of incidence among patients in New York State treated with x-rays for enlarged thymus [13] was consistent with the constancy of absolute rather than relative risks over time, although there was an indication of a decrease in the absolute risk beyond 25 years following exposure. However, thyroid cancer incidence among Israeli children irradiated for ringworm of the scalp [14] indicated constancy of the relative risks, both across time and sexes. For skin cancer incidence, the study of children in New York treated with x-rays for ringworm of the scalp [15] yielded evidence for the constancy of relative risks based on a mean follow-up of 25 years. For digestive cancers and the grouping of all cancers other than breast, lung and digestive cancers, the BEIR V analysis of the LSS data gave rise to models under which the relative risks are constant over time but decrease with increasing age at exposure.

(b) Transfer of risks across populations

There have been relatively few epidemiological analyses of how radiation-induced cancer risks should be transferred across populations. The parallel analysis of breast cancer incidence by Land et al. [16], based on previous follow-ups of the LSS and the two groups of US women mentioned above who were irradiated for medical reasons, suggested that the absolute rather than relative risks were more stable between Japan and the US. However, the BEIR V analysis [2] based on the most recent follow-ups showed that the excess relative risk in the LSS was about 50% greater than that in the two US cohorts ($p = 0.4$), whereas the absolute excess risk in the former was about half of that in the latter groups ($p = 0.01$). For breast cancer mortality, the excess relative risk in the LSS was 2–3 times that in the Canadian tuberculosis patients [12] (other than in Nova Scotia), whereas the absolute risk in the former was less than that in the latter; neither comparison achieved statistical significance. On the basis of these results, the BEIR V Committee adopted a relative risk model to transfer risks from Japan to North America. However, in view of the possible discrepancies under either model, it may be worth considering more general models that lie between the relative and absolute models [17].

There is some other evidence favouring the transfer of relative risks. An analysis [18] based on published results from the LSS [3] and ASS [4] showed that the ratio of lung to stomach cancer risks is similar across studies when based on relative risks, but is an order of magnitude greater in the ASS than in the LSS when based on absolute risks (reflecting the differences in baseline rates between the UK and Japan). The BEIR IV analysis [8] of the joint effect of radon exposure and smoking on the risk of lung cancer in miners showed that the data were consistent with multiplicative as well as sub-multiplicative relationships, but not with an additive relationship. This provides evidence for transferring relative risks of lung cancer across populations with different levels of smoking. Among US children irradiated for ringworm of the scalp [15], the radiation-induced absolute risk of skin cancer incidence appeared to vary according the baseline risk in different ethnic groups. Finally, studies of cancer in mice have indicated that relative rather than absolute risks tend to be stable across strains and sexes [19].

Tab. 1. 1988 UNSCEAR [1] Estimates of the lifetime risk of fatal cancer following whole body exposure at high dose rates (based on a Japanese population).

	Deaths, 10^{-2} Sv^{-1}	
	Relative Risk Model	Absolute Risk Model
Population of all ages	7[b]–11[a]	4[a]–5[b]
Working population (25–64 years)	7[a]–8[b]	4[b]–6[a]

Notes:
[a] Based on age-specific risk coefficients from the LSS [3].
[b] Based on age-averaged risk coefficients from the LSS.

Results of risk projections

(a) UNSCEAR

In its 1988 report, UNSCEAR [1] calculated risks for individual cancer sites, both under relative and absolute projections over time and using data from the follow-up of the Japanese atomic bomb survivors [3]. However, the relative and absolute risk coefficients used in this instance were averaged over all ages at exposure, whereas information from the LSS and other studies indicates that these coefficients tend to vary with age at exposure. The influence of this factor is illustrated in Table 1. For all cancers, the range on the lifetime cancer risk in a population of all ages is 5–7% Sv^{-1} if based on age-averaged coefficients and 4–11% Sv^{-1} if based on age-specific coefficients. The range is greater in the latter case due to the influence of those in the LSS irradiated early in life; these persons have the highest relative risks but, because they are only now reaching the ages at which solid cancers would tend to occur, currently show the lowest absolute excess risks. In view of the age variation in risk coefficients, the range based on age-specific coefficients is to be preferred. The corresponding range for a working population, namely 6–7% Sv^{-1}, is not as great as for the general population, since children are excluded in the former case.

For application at low dose rates, a dose rate effectiveness factor may be applied to these and the following risk estimates. Based on animal and human data, UNSCEAR [1] have suggested values in the range 2–10. Although the human data on low dose extrapolation point towards values at the lower end of this range [20], virtually all these data relate exposures at high dose rates.

It should also be emphasised that UNSCEAR used Japanese baseline cancer rates in their projections, and so their calculations are specific to a Japanese population.

(b) NRPB-R226

Stather et al. [18] in NRPB-R226 developed health effects models from the 1988 UNSCEAR report and calculated cancer risks that are applicable to a UK population. The R226 models were based on age and sex-specific risk coefficients, generally from the LSS [3], but with information from other studies for some specific cancers (see footnotes to Table 2). For cancers other than leukaemia relative risks were generally projected both across populations and over time, in one instance until the end of life and in the other up until 40 years following exposure (the current period of follow-up for the LSS). In view of the uncertainty in the temporal pattern of the relative risks, these two calculations may provide bounds on the lifetime risk. For leukaemia, since baseline rates are stable across populations and the radiation-induced risk tails off over time, an absolute risk model with an expression period of 2–40 years after exposure was used.

Table 2 shows the risks under the R226 models for a UK population of all ages and both sexes, based on exposure at high doses and high dose rates. The disease groupings are those used in the BEIR V report [2]. The total cancer risk under a lifetime relative risk projection, namely 13% Sv^{-1}, is slightly larger than the corresponding UNSCEAR value due to the slightly higher baseline rate for cancer overall in the UK compared with Japan. Also, as pointed out in Section 3(d), the proportions of the individual cancer types under this form of projection model, differ substantially between the UK and Japan. It should also be noted that the total cancer risk up to 40 years following exposure is only about

Tab. 2. Radiation-induced cancer risks in a UK population (all ages, both sexes) exposed at high doses and high dose rates.

	Deaths, 10^{-2} Sv^{-1}		
	BEIR V Models	NRPB-R226 Models	
		Lifetime	To 40 years
Leukaemia[a]	1.2[b]	0.84	0.84
Breast	0.4[c]	1.1[d]	0.42[d]
Respiratory[a]	2.3[e]	3.6	1.2
Digestive[a]	3.5	3.3[f]	0.84[f]
Remainder[a]	3.6	4.0[g]	0.65[g]
Total	11.0	12.9	3.9

Notes:
[a] Based on the LSS.
[b] Obtained by multiplying the risks at low doses under the BEIR V linear-quadratic dose-response model for leukaemia by 2, the low dose extrapolation factor calculated by BEIR V. All the other risks are based on linear dose-response models.
[c] Based on the LSS and the Canadian cohort [12]; relative risk decreases over time.
[d] Based on the US cohorts [16].
[e] Relative risk decreases over time.
[f] Calculated as the sum of the risk of stomach, colon and all other digestive cancers. For the latter grouping, the age and sex-specific relative risks (RRs) for stomach cancer [3] were applied to the baseline rates for these other digestive cancers.
[g] Calculated by difference from the risks for all cancers other than leukaemia, based on the RRs of Shimizu et al [3].

Tab. 3. Radiation-induced cancer risks in a UK population (both sexes) exposed at high doses and high dose rates, based on the NRPB-R226 models.

Age at exposure (years)	Deaths, 10^{-2} Sv^{-1}	
	Lifetime	To 40 years
0– 9	32.8	2.1
10–19	20.5	2.6
20–29	18.5	5.8
30–39	8.8	5.4
40–49	6.0	5.2
50–59	5.0	4.8
60–69	3.5	3.5
70–79	1.8	1.8
80+	0.72	0.72

30% of that based on the assumption that the relative risks will remain constant throughout life. This reflects the uncertainties in the lifetime cancer risks for those in the LSS irradiated when young, who are only now reaching the ages at which solid cancers would normally occur. This age variation in the uncertainty for the lifetime cancer risk is illustrated further in Table 3, taken from Muirhead[21] and based on the R226 models. For those aged less than 10 years at exposure, the risk up to 40 years following exposure and that projected over a lifetime differ by over an order of magnitude.

(c) BEIR V

The BEIR V[2] models were derived by modelling of detailed data from the LSS and certain other studies, such as of breast cancer in women in North America irradiated for medical reasons. These models specified relative risks as functions of sex, age at exposure and time since exposure; in particular, allowance was made for the possibility that relative risks may either remain constant or ultimately decrease with increasing time since exposure. A relative risk projection across populations from the LSS was assumed.

Based on the BEIR V relative risk models, Muirhead[21] calculated cancer risks that are applicable to a UK population of all ages and both sexes. Table 2 shows the calculated numbers of radiation-induced cancer deaths associated with high dose rate exposure under the BEIR V models, along with the corresponding values under the NRPB-R226[18] models. It should be noted that the number of radiation-induced cancer deaths is a different quantity from the *excess* number of cancer deaths that was studied in the BEIR V report. The latter number excludes the proportion among those who die of a radiation-induced cancer who would have otherwise died from a naturally-occurring cancer at a later time. Thus, for all cancers, the number of excess deaths that would be calculated under the BEIR V approach is about 20–25% less than the number of radiation-induced deaths presented here.

The following points can be made from Table 2.

(i) Although the leukaemia risk coefficients in the R226 and BEIR V models were both based on the LSS, there are differences in the range of doses over which models were fitted and in the projection of risks, both before and after the current follow-up period. It is notable that the risk calculated here under the BEIR V model is somewhat smaller than that implied in the BEIR V report (1.9% Sv^{-1}). This is due largely to the inclusion of chronic lymphatic leukaemia (CLL) in BEIR V's analysis and risk projection (D. G. Hoel, Personal Communication), whereas CLL (which appears not to be radiation-inducible) was excluded from the calculations presented here.

(ii) The breast cancer risk under the BEIR V model is at the lower end of the two R226 estimates, not only because the former model incorporated a decrease in the relative risk with time, but also because it was based on both the LSS and Canadian data (see section 2(b)). In view of the differences in relative risk between these data sets, it may be better (as in R226) to use only data from North America when calculating risks for western populations.

(iii) The BEIR V model for respiratory cancer allowed for a decrease in the relative risk over time. Consequently the corresponding estimate lies between the R226 values.

(iv) For digestive and remaining cancers, BEIR V assumed constancy of the relative risk over time. Hence these estimates are similar to the upper R226 values.

The overall risk estimate under the BEIR V models for a population of all ages, namely 11% Sv^{-1}, lies towards the upper end of the R226 range, 4–13% Sv^{-1}. For a working population of both sexes aged 20–64 years, the risks are 9.1% Sv^{-1} under BEIR V compared with 5.2–9.7% Sv^{-1} under R226.

(d) ICRP

The International Commission on Radiological Protection (ICRP) published a draft of its new recommendations in February 1990[22]. In an annex dealing with the biological effects of ionising radiation, provisional estimates of radiation-induced cancer risks were presented, based on a forthcoming paper by C. E. Land and W. K. Sinclair. These were based on age and sex-specific risk coefficients derived from published data on the LSS[3]. Projection models were used that involved (i) constancy of relative risks across both time and populations, (ii) relative risks constant over time but absolute risks

constant across populations (the so-called "NIH" model), and (iii) absolute risks constant across both time and populations. Of these three models, the first two were preferred.
Calculations were performed for several countries. For the UK, the results under the relative risk model were similar to those given in NRPB-R226[18], due to the similarity in methodology. However, while the total cancer risk did not vary greatly across countries under the relative risk model (and was approximately 10% Sv^{-1} for high dose rate exposure), the risks for some individual cancers varied substantially. For example, the risk of stomach cancer was about 4 times higher in Japan than in the UK, whereas the lung cancer risk in the UK was more than double that in Japan and the UK breast cancer risk was about 6 times the Japanese risk; these differences reflected the corresponding differences in the baseline rates between these countries. Under the NIH model the individual cancer risks were similar for different countries, because of the assumption of constancy of absolute risk coefficients across countries. Overall risk estimates were derived by averaging values over countries and the two models (relative and NIH), since the view was taken that there is not enough information to decide how the transfer of risk coefficients across populations should be performed. Whilst, as indicated in section 2(b), there is some information which would suggest that a transfer based on relative risks may be preferred, more work is clearly needed.

Discussion

Taken overall, the evidence from epidemiological studies tends to favour the use of relative rather than absolute risk models to project risks across time and populations for most solid cancers. However, there are clear indications for certain solid cancers (eg. lung) of a tailing-off in the relative risk over time. Also, there is the possibility that risks may vary across populations in a sub-multiplicative manner. Further parallel analyses such as those in the BEIR IV and V reports would be of great help in understanding these topics.
However, to reduce the large uncertainty in the evaluation of lifetime risks for those irradiated when young, as shown in Table 3, requires the continued follow-up of such groups. In the next 10 years or so, the baseline rates among those in the LSS irradiated at ages less than 10 years will increase further and so it should be easier to discern any decrease in the relative risk over time. In the meantime, it may be better to consider ranges (as in NRPB-R226[18]) as well as best estimates for cancer risks, given the uncertainties involved.

Summary

Various methods can be used to project the risks of radiation-induced cancer estimated in cohort studies beyond the period of follow-up and to other populations. The epidemiological evidence for the choice of risk projection model is reviewed based on data from studies such as those of the Japanese atomic bomb survivors and UK ankylosing spondylitis patients given x-ray therapy. The results of risk projections based on various approaches are presented, including those suggested by UNSCEAR and by the BEIR V Committee. It is emphasised that the continued follow-up of populations such as the Japanese atomic bomb survivors is of great importance in estimating lifetime risks, and that further parallel analyses are required to examine how risks vary across populations.

Zusammenfassung

Die Extrapolation von Strahlenkrebs-Risiken aus den beobachteten Daten auf spätere Lebensabschnitte und andere Populationen
Für die Projektion von Strahlenkrebs-Risikowerten aus Kohortenstudien auf Zeitabschnitte jenseits der bisherigen Nachbeobachtungsdauer und auf andere Bevölkerungen stehen verschiedene Methoden zur Verfügung. Die epidemiologischen Fakten, die für die Wahl der Risikoprojektionsmodelle mitbestimmend sind, werden diskutiert, wobei auf die Daten der Atombomben-Überlebenden in Japan und der röntgenbestrahlten Bechterewpatienten in Grossbritannien sowie auf andere Follow-up-Studien Bezug genommen wird. Die mit den verschiedenen Modellen erzielten Risikowerte – darunter auch die Risikoschätzungen des UNSCEAR-Berichtes 1988 und des BEIR V-Berichts von 1990 – werden einander gegenübergestellt. Weitere Nachbeobachtung und Parallelanalysen der verschiedenen Studienbevölkerungen sind notwendig, um verlässlichere Schätzungen des strahlenbedingten Krebsrisikos für eine ganze Lebensdauer (lifetime-risks) zu erhalten und Gemeinsamkeiten und Unterschiede der Risikomuster zwischen den Bevölkerungen besser zu charakterisieren.

Résumé

La projection des risques de cancer radiogénique dans différentes populations et différentes périodes
Diverses méthodes peuvent être utilisées pour projeter les risques de cancer radiogénique estimés dans les études de cohortes au-delà des périodes de suivi ou dans d'autres populations. Les bases épidémiologiques permettant de choisir le modèle de projection de risque sont passées en revue sur la base d'études comme celles sur les survivants de la bom-

be atomique japonaise et sur l'étude britannique concernant les patients atteints de spondylite ankylosante traitée par rayons X. Les résultats des projections de risque basés sur différentes approaches sont présentés, y compris ceux suggérés par UNSCEAR et par le Comité BEIR V. Le suivi continu de populations comme celle des survivants japonais à la bombe atomique est d'une grande importance pour estimer les risques «lifetime»; d'autres études sont nécessaires pour mieux connaître la variation des risques entre les populations.

References

1 UNSCEAR. Sources, Effects and Risks of Ionising Radiation. 1988 Report to the General Assembly. New York: United Nations, 1988.

2 Committee on the Biological Effects of Ionising Radiation (BEIR V). Health Effects of Exposure to Low Levels of Ionising Radiation. National Academy of Sciences, National Research Council. Washington DC: National Academy Press, 1990.

3 *Shimizu Y, Kato H, Schull WJ.* Life Span Study Report 11, Part II: Cancer mortality in the years 1950–1985 based on the recently revised doses (DS86). Hiroshima: Radiation Effects Research Foundation, 1988; RERF TR5–88.

4 *Darby SC, Doll R, Gill SK, Smith PG.* Long term mortality after a single treatment course with x-rays in patients treated for ankylosing spondylitis. Br J Cancer 1987; *55*: 179–190.

5 *Boice JD, Day NE, Andersen A,* et al. Cancer risk following radiation treatment for cervical cancer. An international collaboration among cancer registries. J Natl Cancer Inst 1985; *74*: 955–975.

6 *Muirhead CR, Darby SC.* Modelling the relative and absolute risks of radiation-induced cancers (with discussion). J Roy Statist Soc 1987; *A150*: 83–118.

7 *Muirhead CR, Darby SC.* Relative and absolute risk models for cancer mortality in ankylosing spondylitis patients. In: KF Baverstock, JW Stather, eds. Low Dose Radiation: Biological Bases of Risk Assessment. London: Taylor and Francis, 1989: 162–170.

8 Committee on the Biological Effects of Ionising Radiation (BEIR IV). Health Risks of Radon and Other Internally Deposited Alpha Emitters. Washington DC: National Academy Press, 1988.

9 *Tokunaga M, Land CE, Yamomoto T,* et al. Incidence of female breast cancer among atomic bomb survivors, Hiroshima and Nagasaki, 1950–1980. Radiat. Res. 1987; *112*: 243–272.

10 *Shore RE, Hildreth N, Woodard E,* et al. Breast cancer among women given x-ray therapy for acute postpartum mastitis. J Natl Cancer Inst 1986; *77*: 689–696.

11 *Hrubec Z, Boice JD, Monson R, Rosenstein M.* Breast cancer after multiple chest fluoroscopies: Second follow-up of Massachusetts women with tuberculosis. Cancer Res 1989; *40*: 229–234.

12 *Miller AB, Howe GR, Sherman GJ,* et al. Mortality from breast cancer after irradiation during fluoroscopic examinations in patients being treated for tuberculosis. N Eng J Med 1989; *321*: 1285–1289.

13 *Shore R, Woodard E, Hildreth N,* et al. Thyroid tumours following thymus irradiation. J Natl Cancer Inst 1985; *74*: 1177–1184.

14 *Ron E, Modan B, Preston D,* et al. Thyroid neoplasia following low-dose radiation in childhood. Radiat Res 1989; *120*: 516–531.

15 *Shore RE, Albert RE, Reed M,* et al. Skin cancer incidence among children irradiated for ringworm of the scalp. Radiat Res 1984; *100*: 192–204.

16 *Land CE, Boice JD, Shore RE,* et al. Breast cancer risk from low-dose exposures to ionising radiation: Results of parallel analysis of three exposed populations of women. J Natl Cancer Inst 1980; *65*: 353–365.

17 *Muirhead CR.* Transfer of risk estimates between populations. In: Risk Estimates for Radiation Carcinogenesis: Proceedings of the International Workshop, Bad Münstereifel, Germany. Köln: Institut für Strahlenschutz, 1990: 50–54.

18 *Stather JW, Muirhead CR, Edwards AA,* et al. Health effects models developed from the 1988 UNSCEAR report. Chilton: NRPB-R226 (London: HMSO), 1988.

19 *Storer JB, Mitchell TJ, Fry RJM.* Extrapolation of the relative risk of radiogenic neoplasms across mouse strains to man. Radiat Res 1988; *114*: 331–353.

20 *Pierce DA, Vaeth M.* The shape of the cancer mortality dose-response curve for atomic bomb survivors. Hiroshima: Radiation Effects Research Foundation, 1989; RERF TR7–89.

21 *Muirhead CR.* Projection of radiation-induced cancer risks across time and populations. Radiat Prot Dosim 1991; *36*: 321–325.

22 International Commission on Radiological Protection. Recommendations of the Commission – Draft, February 1990. ICRP/90/G-01.

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