

EPA 402-R-93-076

ESTIMATING RADIOGENIC CANCER RISKS

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401 M Street S.W.
Washington, DC 20460**

The scientific basis for this report has been reviewed formally by the Radiation Advisory Committee (RAC) of the EPA Science Advisory Board (SAB). The following paragraph is a synopsis of that review.

On January 10, 1992, Margo T. Oge, Director, Office of Radiation Programs (now the Office of Radiation and Indoor Air or ORIA) requested that the RAC review an issues paper comparing health risk estimates due to low level exposures of low-LET radiation based on models recently published by the Radiation Effects Research Foundation, the United Nations, the National Radiological Protection Board of the UK, the National Academy of Sciences, the US Nuclear Regulatory Commission, and the International Commission on Radiation Protection. Following discussions on February 12, 1992 with the RAC, ORIA staff prepared a document titled "Proposed Methodology for Estimating Radiogenic Cancer Risk" and forwarded it to the RAC for their review on May 1, 1992. In their letter to the EPA Administrator dated December 9, 1992, Dr. Raymond C. Loehr, Chairman, SAB Executive Committee, and Dr. Oddvar E. Nygaard, Chairman, SAB Radiation Advisory Committee, provided the Committee's evaluation of the proposed ORIA methodology for estimating radiogenic cancer risks. They concluded that, "Although no single data set and model for predicting radiogenic cancer risk is ideal, the method of analysis chosen by EPA is adequately supported by present evidence." They also offered some comments and suggestions for future consideration. In her letter of April 19, 1993, Carol M. Browner, Administrator, EPA, provided responses to those comments and suggestions.

This report, which was prepared by EPA staff members Jerome S. Puskin and Christopher B. Nelson, Office of Radiation and Indoor Air, Criteria and Standards Division, presents radiation risks calculated with the models proposed to the RAC. It also includes risks due to radionuclide intakes and external exposures calculated with those models.

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The mailing address for the authors is:

U.S. Environmental Protection Agency
Office of Radiation and Indoor Air (6602J)
Washington, DC 20460

ABSTRACT

This document presents a revised methodology for EPA's estimation of cancer risks due to low-LET radiation exposures developed in light of information that has become available since the publication of BEIR III, especially new information on the Japanese atomic bomb survivors. For most cancer sites, the risk model is one in which the age-specific relative risk coefficients are obtained by taking the geometric mean of coefficients derived from the atomic bomb survivor data employing two different methods for transporting risks from Japan to the U.S. (multiplicative and NIH projection methods). Using 1980 U.S. vital statistics, the risk models are applied to estimate organ-specific risks, per unit dose, for a stationary population. With the exception of breast cancer, low-LET radiogenic cancer risks are assumed to be reduced by a dose and dose rate effectiveness factor (DDREF) of 2 at low doses and dose rates compared to risks at high acute dose exposure conditions. The DDREF assumed for breast cancer is 1. For low dose (or dose rate) conditions, the calculated risk of a premature cancer death attributable to uniform, whole-body, low-LET irradiation is about $5.1 \times 10^{-2} \text{ Gy}^{-1}$. The corresponding incidence risk (neglecting nonfatal skin cancers) is about $7.6 \times 10^{-2} \text{ Gy}^{-1}$. High-LET (alpha particle) risks are presumed to increase linearly with dose and to be independent of dose rate. Except for leukemia and breast cancer, a relative biological effectiveness (RBE) factor of 20 is adopted for the risk of high-LET radiation relative to that for low-LET radiation at low dose or low dose rate conditions. For leukemia, an effective high-LET RBE of 1 is used; for breast cancer, the high-LET RBE is 10. Using the revised methodology, lifetime cancer risks associated with constant exposure rate conditions are estimated for over 300 radionuclides.

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I. Introduction

Since 1984, EPA's estimates of risk from low-LET radiation have been based on the 1980 National Academy of Sciences' (NAS) BEIR III Report (NAS 1980, EPA 1984, EPA 1989). Subsequently, important new data have become available, especially revised dosimetry and further epidemiological follow-up on the Japanese atomic bomb survivors. Risk estimates derived in light of the new data have now been presented in several recent reports (Shimizu et al. 1988, 1990; UNSCEAR 1988; Stather et al. 1988; NAS 1990; ICRP 1991; Land and Sinclair 1991; Gilbert 1991). We critically examine here the information in those reports, and some ancillary information, with the aim of developing a revised methodology for EPA's calculations of radiogenic cancer risks. Radiogenic benign neoplasm risks are not considered in this report.

In Section II, the main scientific issues are outlined and discussed. Section III compares the assumptions and numerical projections of risk pertaining to alternative models found in the above reports. Section IV presents EPA's revised methodology for estimating radiogenic cancer risks at low doses and dose rates. The Appendix discusses calculational methods and includes risk estimates for individual radionuclides.

Calculated values in this report are typically shown to three significant figures or one decimal place. This practice is intended to facilitate comparisons and to simplify tabulations only. The number of significant figures should not be considered to indicate the level of certainty in the tabulated values.

II. Scientific Basis for Risk Estimates

A. Epidemiological Data

By far the most important source of data upon which to base estimates of risk from low-LET ionizing radiation is the Atomic Bomb Survivor Study (ABSS). Noteworthy features of this study include: a large, relatively healthy population at the time of exposure; all ages and both sexes; a wide range of doses, believed to be well estimated on an individual basis, to all organs of the body; existence of a good control group, consisting of people who were present in Hiroshima or Nagasaki at the time of bombing but who received only small doses of radiation; detailed, long-term (40 y) epidemiological follow-up. A statistically significant excess cancer mortality associated with radiation has been found among the bomb survivors for the following types of cancer: leukemia, esophagus, stomach, colon, liver, lung, breast, ovary, urinary tract, and multiple myeloma.

There is an extensive body of epidemiological data on radiation-exposed populations, apart from the A-bomb survivors. Most important from the standpoint of developing quantitative estimates of risk from low-LET radiation are the studies of medical exposures of the thyroid and breast. For two other sites, bone and liver, low-LET risk estimates are commonly determined from epidemiological studies of cohorts exposed to alpha particle irradiation, extrapolating a value of RBE from animal studies. Cohorts ingesting or injected

radiogenic liver cancer consists of patients receiving Thorotrast (ThO₂) injections. There are additional important epidemiological studies of persons exposed to low-LET radiation, most notably, perhaps, the ankylosing spondylitis and cervical cancer patients. Currently, however, the main value of these studies is for comparison to the A-bomb survivors; none of the major recent efforts at radiation risk estimation (see below) make direct use of these studies in developing quantitative estimates of risk.

B. Modeling the Epidemiological Data

There are many different ways one could organize and model the ABSS data. Choices can be made regarding: the grouping of cancer sites and age groups, the mathematical form of the dose-response, and the general form of the age and temporal dependence. These choices are generally made after exploratory analyses of the data, which indicate what parameters are most useful to incorporate into the models. By breaking down the data into smaller subgroups, interesting features may be revealed, but, at some point, the concomitant increase in statistical variability precludes any meaningful improvement in the model. This constraint may lead to certain trade-offs; e.g., to obtain a more detailed analysis of the effects of age and temporal factors on risk, the BEIR V Committee combined all types of GI cancers into a single category, even though a single model cannot adequately describe the risk for the different GI cancers.

Age and Temporal Dependence

Information on the variation of risk of site specific radiogenic cancers among the atomic bomb survivors with age and time is limited by sampling uncertainties and by the incomplete period of epidemiological follow-up. For a given age at time of the bomb (ATB), the excess solid tumor mortality has generally been found to increase with the age at death (ATD), roughly in proportion to the age-specific baseline rate for the site of interest. Consequently, models for these tumors are now generally framed in terms of *relative risk*.

For the period of epidemiological follow-up, the highest relative risks are found in the youngest exposure categories. But the lifetime risks of solid tumors due to exposures before age 20 remain highly uncertain. Individuals exposed as children are only now entering the years of life where the risk of cancer is concentrated. While this group has exhibited a high relative risk per unit dose, thus far, the observed excess represents a small number of cancer deaths. Hence, the sampling error for most types of cancers is large for the younger age cohorts. It is, moreover, unclear to what extent the observed high relative risks will persist. Theoretical considerations, arising from carcinogenesis modeling, would suggest that the relative risks will decrease over time (Little and Charles 1991). In addition, there is some epidemiological evidence suggestive of such a temporal fall-off in groups irradiated as children (UNSCEAR 1988, Little et al. 1991).

Conclusions. For cancers other than leukemia, there is strong evidence of an increasing risk with age at expression, roughly in proportion to the increase with age of baseline cancer mortality. The data are generally consistent with a constant relative risk model in which the risk coefficients decrease with age at exposure. There is some suggestive evidence of a fall-off in relative risk with time after exposure, especially for childhood exposures (NAS 1990), but further epidemiological surveillance will be necessary to clarify the pattern of the temporal change (Shimizu et al. 1988).

Transport of Risk Estimates Across Populations

Baseline rates for specific cancer types vary from population to population, as well as over time, within a population. For example, stomach cancer rates are substantially higher in Japan than in the U.S., while the reverse is true for lung, colon, and breast cancer; moreover, the incidence rates for lung and breast cancer, particularly, have been increasing in both populations during recent years. Despite the observed rough proportionality between radiation risk and baseline cancer rates by age, one cannot necessarily infer that the radiation risk will vary in proportion to the baseline rate as one goes from one population to another.

Information on how to "transport" risk estimates across populations is limited by the quality of data available on irradiated populations other than the bomb survivors. Two cancer types for which comparison data exist are thyroid and breast: data on the former suggest that the risk does increase with the baseline rate (NAS 1990), but it would appear that the opposite may be true for the latter (Preston 1991). Some insight into the problem might be gained by looking at subgroups of an irradiated population. For example, lung cancer rates in Japanese males are several times higher than in Japanese females, presumably due in part to the higher smoking rate in males. Nevertheless, the excess absolute risk for lung cancer attributable to radiation does not differ significantly between the male and female bomb survivors. This would suggest that, for lung cancer, absolute risk may be more transportable than relative risk.

Conclusions. Information on how to transport risk estimates between populations is very limited; what information there is suggests that the answer is likely to be cancer site specific.

Dose Response Function and Dependence on Dose Rate

A major issue in radiation risk assessment is how best to quantify the risks and to characterize their uncertainties for small incremental doses above natural background. A comprehensive examination of this question was contained in NCRP Report 64 (NCRP 1980). Based primarily on laboratory studies of cells, plants and animals, the report advocated a linear-quadratic dose response for acute doses up to about 2.5-4 Gy, above which the dose response begins to turn over due to cell killing effects. At low doses, the quadratic term is negligible in comparison to the linear term. The NCRP committee defined the low dose region as 0-0.20 Gy, since significant deviations from linearity are found in *Tradescantia*

experiments and in life shortening in mice above this range. Evidence was also cited to the effect that the D^2 term in the dose response function vanishes when the radiation is delivered at low dose rates, even for total doses above 0.2 Gy.

A theoretical framework for the linear-quadratic dose response model has been developed by Kellerer and Rossi (1972), utilizing concepts originally put forth by Lea (1962). In this theory of "dual radiation action," events leading to "lesions" (i.e., permanent changes) in cellular DNA require the formation of interacting pairs of "sublesions." The interacting pairs can be produced by a single traversing particle, or track, or by two tracks, giving rise, respectively, to a linear and a quadratic term in the dose response relationship. According to the theory, a sublesion may be repaired before it can interact to form a lesion, the probability of such repair increasing with time. Consequently, as the dose rate is reduced, the formation of lesions from sublesions caused by separate tracks becomes less important, and the magnitude of the D^2 term decreases. Hence, the theory predicts that at sufficiently low doses or dose rates, the response should be linear and, in either limit, should have the same slope.

Results of animal tumorigenesis studies are, in general, qualitatively consistent with the theory: low-LET radiation seems to have reduced effectiveness per unit dose at low dose rates (NCRP 1980). However, it is usually not possible from the data to verify that the dose response curve has the linear-quadratic form. Another success of the dual action theory has been in explaining observed differences between the effects of low- and high-LET radiations. In this view, the densely ionizing nature of the latter results in a much greater production of interacting pairs of sublesions by single tracks, leading to a higher biological effectiveness at low doses and a linear dose response relationship (except for deviations at high doses attributable to cell-killing effects).

The dual action theory has nevertheless been challenged on experimental grounds, and observed variations in response with dose, dose rate, and LET can also be explained by other mechanisms, e.g., a theory involving only single lesions and a "saturable" repair mechanism that decreases in effectiveness at high dose rates on the microscopic scale (Goodhead 1982). One property of such a theory is that, in principle, the effectiveness of repair - and therefore the shape of the dose response curve - can vary widely with cell type and species. Hence, results obtained on laboratory animals might not be entirely applicable to humans.

According to either the dual action theory or the saturable repair theory, the dose response should be linear at low doses or low dose rates, and with equal slopes. At higher doses and dose rates, multiple track events become important, and the dose response should bend upward. As a result, the response per unit dose at low doses and dose rates will be overestimated if one extrapolates linearly from observations made at high doses, acutely delivered (NCRP 1980). The degree of overestimation is commonly expressed in terms of a *dose and dose rate effectiveness factor (DDREF)*: e.g., a DDREF of 3 means the risk per unit dose observed at high acute doses should be divided by 3 before being applied to low dose (dose rate) conditions.

Current mechanistic explanations for a DDREF involve DNA repair. The linearity of the dose response at low doses suggests that DNA repair is maximal and independent of dose rate for doses below about 0.2 Gy. Repair of radiation-induced DNA damage is found to be largely complete within a few hours of an acute exposure (Wheeler and Wierowski 1983, Ullrich et al. 1987). Consequently, protracting the dose beyond this time span should have little or no effect on the risk of cancer induction. It is expected, therefore, that repair will be maximal so long as no doses above 0.2 Gy are delivered within a few hours.

When the BEIR III Committee attempted to fit the ABSS data to a linear-quadratic model, the results were not very satisfactory (NAS 1980). Depending on whether one analyzed solid tumors or leukemia, and depending on which city the data were drawn from, the shape of the fitted dose response changed markedly; in some cases no reasonable fit could be obtained. Some of the discrepancy was resolved by attributing a substantial fraction of the excess cancers in Hiroshima to neutrons, but serious problems remained, and the ultimate choice of dose response function seemed to be strongly influenced by supporting laboratory data.

With the revised "DS86" dosimetry, these curve-fitting problems are largely removed (Shimizu et al. 1990, NAS 1990). The data from the two cities are now in reasonable agreement. The combined leukemia data can be fit by a linear-quadratic dose response function; the slope of the function at low doses is about half that obtained by a linear fit to the data. A statistical analysis of the solid tumor data, on the other hand, indicates no departure from linearity in the dose response over the entire range from 10 to 400 rad. Using a linear-quadratic model to fit the data reduces the linear term by, at most, a factor of 2 compared to a simple linear model. Viewing these results through the paradigm adopted by NCRP 64 would indicate that: a best estimate of the DDREF is about 2 for leukemia while, for solid tumors, a DDREF of 2 represents an *upper bound*, and the best estimate is about 1.

The conclusions in the preceding paragraph, however, appear to require some modification in light of a subsequent analysis by Pierce and Vaeth (1991). These authors show that errors in dose estimation will introduce a negative bias in the dose-squared dependence of the response. Assuming dosimetry errors of 30-40%, this has a relatively minor effect on the best estimate of the DDREF, but the upper bound is increased to about 3.

Also pertinent to the issue of what DDREF is applicable to human cancer induction are clinical studies of radiation-induced breast and thyroid cancer, which have shown little or no reduction in risk with dose fractionation (Shore et al. 1984, Davis et al. 1989, Howe 1992). This again suggests a DDREF of about 1 (ICRP 1991). On the other hand, while studies of tuberculosis patients, who had undergone repeated fluoroscopic examinations, clearly show an elevated risk of breast cancer from fractionated doses of x-rays, they show no indication of an excess lung cancer risk (Davis et al. 1989, Howe 1992). When compared with observed lung cancer risks in the atomic bomb survivors, the results of these studies suggest that the DDREF may be quite large for lung cancer induction, although the possibility of confounding by the underlying disease process cannot be ruled out.

The results on human solid tumors appear to differ from those obtained through laboratory studies, including studies of radiation-induced tumorigenesis in mice and rats. For the most part, the laboratory studies suggest a DDREF of about 2 or 3, and sometimes higher, depending on end point; on the other hand, the preponderance of the evidence on humans suggests a lower DDREF, possibly about 1 for most sites. One can resolve this apparent conflict in different ways, with significantly different implications for radiation risk estimation at low doses and dose rates.

If one retains the linear-quadratic model in NCRP 64 and the connection it implies between the shape of the dose response and the effect of dose rate on the response, the Japanese data would seem to dictate an average DDREF no higher than 1 to 3 for radiation-induced carcinogenesis in humans. It could then be argued that, in light of the extensive laboratory data suggesting a DDREF of at least 2, a DDREF of 2-3 should be applied in estimating risks to humans. On the other hand, it could be argued that animal data are an unreliable guide to the response in humans and that a DDREF of 1, an approximate best estimate from the human data, should be used in estimating the risk of solid tumors at low doses and dose rates.

There are experimental data suggesting a difference in DDREF between humans and rodents. Grosovsky and Little (1985) measured x-ray induced mutations in human lymphoblast cells and found a linear dose response over the range 0.05-2 Gy, which was independent of dose rate. Similar experiments performed on rodent cells have shown a curvilinear dose response and a decrease in the response with decreasing dose rate.

Alternatively, one might reconcile the human and laboratory data by abandoning the linear-quadratic model of radiation-induced carcinogenesis and its implied connection between the shape of the dose response for acute doses and the effect of protracting the dose over time. For example, one could envision having a linear response over the dose range accessible to epidemiological observation (above about 0.1 Gy) but a substantial reduction in risk at lower doses, or if these doses are fractionated or delivered chronically.

Studies indicate that repair of radiation-induced DNA damage is completed within a few hours after it is incurred (Wheeler and Wierowski 1983, Ullrich et al. 1987). Hence, for multitrack events to be biologically important, it would appear that at least two tracks would have to pass through a cell nucleus within this time period. For low-LET doses of about 1 mGy, only one track, on average, traverses each cell nucleus. From these considerations, it is expected that the same amount of unrepaired DNA damage per unit dose should occur so long as the dose delivered over a few hours is below about 1 mGy. Such conditions define a region in which the response is expected to be linear with dose and independent of any further fractionation or reduction in dose rate.

Conclusions. Taken together, current scientific data are generally indicative of a DDREF between 1 and 3 for human cancer induction. However, there is an indication that a higher DDREF may apply to the lung.

C. Risk from Alpha Particles

Radiobiological data indicate that high-LET alpha radiation has a larger biological effect than an equal absorbed dose of low-LET radiation. The subject has recently been reviewed in: the BEIR IV Report (NAS 1988); NCRP Report No. 104 (NCRP 1990); and ICRP Publication 60 (ICRP 1991). The radiobiological results, including those for tumor induction, are generally suggestive of a linear nonthreshold dose response for high-LET radiation, except for a possible fall-off in effectiveness at high doses. In contrast to low-LET radiation, the effects of high-LET radiation often increase with fractionation or with a decrease in dose rate.

A number of cohorts exposed occupationally or medically to internally deposited alpha emitters have shown an excess of cancer at heavily irradiated sites. Most important is the observed induction of: (1) lung cancer in miners inhaling radon progeny; (2) bone sarcomas in patients injected with Ra-224; (3) bone sarcomas and head carcinomas in dial painters ingesting mixtures of Ra-226 and Ra-228; and (4) liver cancers in patients injected with Thorotrast, an x-ray contrast medium containing isotopes of Th. Although other organs of the body received doses of alpha radiation in these populations, excess cancers were generally not observed at sites other than those mentioned. As a result, only upper bounds to the risk for these other organs can be estimated from studies of humans exposed to alpha irradiation.

Site specific cancer risk estimates for high-LET radiation (neutrons or alpha particles) are often calculated utilizing human epidemiological data on low-LET radiation (e.g., from the ABSS) and laboratory data on the relative biological effectiveness (RBE) of the high-LET radiation compared to a reference low-LET radiation (NCRP 1990). Since the dose response relationship obtained for low-LET radiation is typically linear or concave upward while that for high-LET radiation is linear or concave downward, the RBE is dose dependent. EPA is primarily concerned with risks at low doses and dose rates, where the acute high dose risk for low-LET radiation is reduced by the DDREF. Under these conditions, the dose responses for both low and high LET radiations are thought to be linear, and the RBE takes on a constant (maximum) value: RBE_M .

Ranges of estimated values for neutron and alpha particle RBE_M are wide, depending on both the biological system and the observed end-point; the uncertainty in the RBE_M estimate from an individual study is also usually large, primarily due to the uncertainty in extrapolation of low-LET data to low doses. The effectiveness of alpha emitters has been found to be 15 to 50 times that of beta emitters for the induction of bone sarcomas, liver chromosome aberrations, and lung cancers (NCRP 1990). Since the LET of secondary protons produced by fission neutrons in living tissue is comparable to that for alpha particles, data on the RBEs of fission neutrons provides ancillary information relevant to the estimation of alpha particle RBE. Where the dose response data on carcinogenic end-points are adequate

to derive an estimate, fission neutrons have been found to have an RBE_M between 6 and 60 times that of low dose gamma rays (NCRP 1990).

III. Comparison of Alternative Risk Models

A. Model Descriptions

This section provides a qualitative description of 6 sets of risk models, all based largely on ABSS data collected through 1985 and incorporating DS86 dosimetry.

RERF. A detailed description of the RERF's data on the atomic bomb survivors is contained in two publications by Shimizu, Kato, and Schull (1988, 1990). The data on solid tumors were analyzed using constant absolute and relative risk models, where the risk coefficients were, however, allowed to vary with the age ATB. From this analysis, the authors concluded that the relative, but not the absolute, risk model is consistent with the observed temporal dependence of excess mortality due to solid tumors.

Most of the major qualitative conclusions that can be drawn from the analysis have been discussed above. These include: (1) a statistically significant increase in cancer at various sites, positively associated with radiation dose; (2) for solid tumors, an increase in risk with age ATD, roughly in proportion to the increase in baseline cancer mortality with age; (3) a substantially higher observed relative risk in those below age 20 ATB; (4) essentially a linear dose response for solid tumors, but with evidence of a relatively small quadratic contribution in the dose response for leukemia.

Age-specific risk coefficients are given for these sites: leukemia, stomach, breast, lung, colon, and nonleukemia. In the Life Span Report, these are tabulated both for an assumed neutron RBE of 1 and 10 (Shimizu et al. 1988).

UNSCEAR 88. The lifetime risks in UNSCEAR (1988) were calculated (for the Japanese population) using coefficients from the RERF *Life Span Study Report II* (Shimizu et al. 1990). Estimates for leukemia and all other malignancies are derived for age-specific, absolute and relative risk projection models. The UNSCEAR report also provides risk estimates for an expanded set of cancer sites, calculated using an "age-averaged" relative risk model. In this model, the observed differences in relative risk with age ATB are ignored, and a single, average risk coefficient is adopted for each site. This approach generally yields somewhat lower estimates of the risk for constant lifetime exposures. While we would reject the age-averaged model, as being less reflective of the epidemiological data collected so far, if the high relative risks observed in the younger exposure groups decrease appreciably over time, the age-averaged model may turn out to provide a better numerical estimate of the lifetime risk than the age-specific model derived from these data.

NRPB. The NRPB report (Stather et al. 1988) represents a further elaboration of the information contained in *Life Span Study Report 11* and in UNSCEAR 88. Although other model estimates are developed, the favored approach for solid tumors seems to be the age-specific, constant relative risk projection model, while an absolute risk model with a 2-40 y expression period is adopted for leukemia.

The NRPB model incorporates some modifications from the model originally derived from the Japanese data. First, it was noted that the data indicate a much higher risk coefficient for childhood irradiation of the colon compared to that for adults, but that the data on childhood exposures are, in this case, sketchy. To avoid the problem this poses (a relatively large contribution from colon cancer with very little observational support), the authors arbitrarily assigned the coefficient obtained for ages 20-29 ATB to the age cohort 0-19. Second, risk estimates were given for several cancer sites (breast, liver, thyroid, and bone) derived from epidemiological data collected on populations other than the bomb survivors.

In the case of breast cancer, the authors argued that data on North American women would be more relevant for the population of interest to them (women of the U.K.). Accordingly, they adopted a risk model published in the Nuclear Regulatory Commission's (NRC) Health Effects Model Document (Evans et al. 1985). This breast cancer model was largely based on studies of women from the U.S. and Canada who received diagnostic or therapeutic doses of x-rays. For thyroid cancer, the NRPB adopted a model contained in the same NRC report, as well as in an NCRP report on thyroid cancer risks (NCRP 1985). For liver and bone cancer, no actual age-specific models are given, but lifetime risk estimates are developed on the basis of Thorotrast and radium exposed populations, respectively, assuming an RBE of 20 for alpha particles.

No model is presented for calculating the risk at sites other than those listed. A risk estimate for the "remainder" sites is calculated by taking the difference between the estimate for total solid tumors and the estimated sum for listed solid tumors. This procedure only makes sense in the context of uniform, whole-body irradiation.

ICRP. As support for its recommendations in Publication 60 (ICRP 1991), the ICRP has made use of risk calculations performed by Land and Sinclair (1991). For the most part, these calculations make direct use of the age- and sex-specific relative risk coefficients listed in Table 5A of RERF Report 11, Part 2 (neutron RBE=10) (Shimizu et al. 1988). The age at exposure groups are 0-9, 10-19, 20-29, 30-39, and 40+ years. Land and Sinclair also use the information in the RERF report to incorporate three additional sites into their model: esophagus, ovary, and bladder. Age-specific risk coefficients for "residual" cancers are obtained by subtraction of specified cancers from the total for the period of follow-up.

The risks are calculated for 5 reference populations: Japan, U.S., Puerto Rico, U.K., and China. Three methods are used to transport risk estimates from the study population of bomb survivors to each of the 5 reference populations. The first (additive) involves a direct

transport of age- and sex-specific absolute risk coefficients. The second (multiplicative) involves a direct transport of relative risk coefficients. The third (NIH) is a hybrid of the additive and multiplicative methods. For solid tumors, the total excess risk after the minimal latency period is projected for the period of epidemiological follow-up (i.e., 10-40 y for the RERF data) using the absolute risk coefficients of the additive model. However, it is considered to be distributed over time after exposure as a multiple of the baseline rate. The NIH model relative risk coefficient yields the same risk over the follow-up period as the absolute risk model. This coefficient is then used to project lifetime risk in the same way as for the multiplicative model. With the NIH method, the excess risk varies with age, in proportion to the baseline rates in the population of interest, but only weakly reflects differences between these baseline rates and those in Japan.

A peculiarity of the NIH projection model is that it can artificially introduce age-dependent variability where none can be discerned from the data. For example, in view of the very limited data on lung and colon cancer mortality among the atomic bomb survivors exposed as children, authors have assigned equal risk coefficients for these cancers to the 0-9 y and the 10-19 y age groups, for both the additive and multiplicative models (Shimizu et al. 1990, Land and Sinclair 1991). However, if these age groupings are maintained, the derived NIH projection model will contain significantly higher risk coefficients for the 0-9 group, and a likely inflation of the risk estimates associated with childhood exposures. To avoid this problem, the NIH risk coefficients for lung and colon are calculated on the basis of treating the 0-19 y age group as a single group. The result is a decrease in the estimated risk for these sites compared to previous calculations (ORP 1992).

In discussing the appropriateness of the three models, the authors note that the multiplicative but not the additive model provides a reasonable approximation to the epidemiological data. On the other hand, they also point out that little information is available pertaining to the transfer of risk across populations. Hence, in developing organ-weighting factors, they advocate an average of the multiplicative and NIH model projections.

BEIR V. The National Academy of Sciences BEIR V Committee conducted an independent analysis of the ABSS data, supplemented by data on breast cancer induced by medical irradiation (NAS 1990). The Committee developed several age- and sex-specific relative risk models for calculating excess mortality due to these types of radiogenic cancers: leukemia, respiratory, digestive, breast, and other; breast cancer incidence was also modeled separately from breast cancer mortality. In each case, a preferred model was designated. Unlike all the other reports discussed here, BEIR V incorporated time-since-exposure dependence into its modeling. In transporting risk estimates from Japan to the U.S., BEIR V assumes a multiplicative model: i.e., it assumes that the same risk models and coefficients derived from a statistical fit to the atomic bomb survivor data can be applied to the U.S. population with its own baseline cancer rates.

For acute doses of 0.1 Gy or more, BEIR V derives a linear-quadratic model for leukemia but a linear model for all other cancers. This finding is consistent with earlier conclusions by the RERF group (Shimizu et al. 1988, 1990).

A number of problems can be identified pertaining to the preferred models and estimates in BEIR V:

First, the definition of "excess" risk adopted in the report excludes radiogenic cancers in individuals projected to die of cancer in the absence of the irradiation - this not only understates the predicted health impact of the radiation, it creates disturbing anomalies such as organ-specific risks that are negative or dependent on doses to other organs (C. Nelson, unpublished results).

Second, in projecting radiogenic leukemia risk from the ABSS to the U.S. population, chronic lymphocytic leukemias were erroneously included in the U.S. baseline rate although this type of leukemia does not appear to be radiogenic (Gilbert 1991). This results in an inflation of the leukemia risk estimate for the U.S.

Third, the BEIR V committee employed a multiplicative projection of breast cancer risk from the Japanese to the U.S. Given the higher breast cancer rates in the latter, this implies a higher radiogenic risk also. In fact, epidemiological data indicate the radiogenic risks are similar in the two populations (Preston 1991).

Fourth, a sharp fall-off in risk with time after exposure is incorporated into the respiratory cancer model. Although the model was developed from a statistical fit to the ABSS data, the existence of such a fall-off is not strongly supported by the ABSS data collected so far (NAS 1990). The main evidence for a temporal fall-off comes from the ankylosing spondylitis study (Darby et al. 1987) for which good dosimetry is lacking and for which there is a question of potential confounding by the disease. A striking consequence of the model's fall-off is that the projected risk of irradiating the respiratory tract of children is very low compared to adults. It is noteworthy that the irradiated spondylitic patients were all at least 15 y old.

Fifth, BEIR V models digestive cancers from the ABSS as a single category. An obvious disadvantage of this approach is that no guidance is provided on how to partition the risk among the digestive organs - most importantly, between stomach and colon. By combining the ABSS data in this way, BEIR V was able to obtain a more accurate and robust statistical modeling of sex, age, and time dependencies. To some degree, the gain may be illusory, however, since there is no guarantee that the various digestive organ risks should vary in the same way with these model parameters. Additional error may be introduced in transporting digestive cancer risks from Japan, where stomach cancer rates are high and colon cancer rates are relatively low, to the U.S., where the reverse is true.

Sixth, BEIR V fails to deal effectively with the issue of projecting risk estimates into the domain of low doses and dose rates. On the one hand, a linear dose response for solid tumors is advocated. On the other, it is suggested that at low *dose rates* risk estimates should be reduced "by a factor of two to ten." Taken together, these two recommendations appear to be at odds with basic radiobiological principles, which would seem to imply no dose rate dependence at low doses (see Section II).

NUREG/CR-4214. Recently, the Nuclear Regulatory Commission has published a revision to NUREG/CR-4214 (Abrahamson et al. 1991), that updates its health effects models and their scientific bases. The estimation of radiogenic cancers due to low-LET radiation is discussed in a chapter by Dr. Ethel Gilbert (1991).

While adhering to the general framework laid out in the previous report (Abrahamson et al. 1989), revised models are developed in light of subsequent information, including the new Japanese data and the analyses performed by the BEIR V, UNSCEAR 88, and ICRP 60 committees. Risk models are given for application to the general population for these cancer sites: breast, lung, GI, thyroid, bone, skin, and other. In addition, models are given for *in utero* cancer induction and benign thyroid tumor risks. Guidance is also included as to how to partition the GI cancer risk among organs and how to separately calculate organ specific risks for males and females.

Age-specific, constant relative risk models are recommended for all sites except leukemia, bone, thyroid, and skin, for which additive risk models are proposed. The risk coefficients do not generally represent statistical "best estimates" obtained from an analysis of the epidemiological data. Compared to the other models discussed here, the NUREG/CR-4214 models more explicitly incorporate expert judgment. Broadly speaking, the models are designed to be as simple as possible, but to yield estimates of the risk on an age and organ specific basis, which are reasonably central in view of the scientific uncertainties outlined in Section II.

B. Comparison of Risk Projections

Three sets of models described above are quite similar: RERF, UNSCEAR 88, and the ICRP (multiplicative model). For simplicity, we will eliminate the first two of these from consideration.

Table 1 compares the site-specific projections of lifetime risks for a stationary U.S. population, calculated using these five alternative sets of models: NRPB, BEIR V, NUREG/CR-4214, ICRP (multiplicative), and ICRP (NIH). For purposes of this comparison, the DDREF in each case is 1. (The choice of a DDREF will be discussed below.) The values in the table are risk of cancer attributable to radiation, not just the "excess" risk calculated in the BEIR V report. The cancer risks have been calculated for each model using 1980 decennial U.S. life table and force of mortality data. Hence, the model comparisons are not

confounded by the effects of different sets of vital statistics or of using the specific age distribution for a population in a particular year. As a consequence, the calculated risks differ slightly from those in the referenced reports.

Leukemia. The numerical estimates of lifetime risk are quite similar, except for BEIR V, which is about a factor of 2 higher. In part, this is due to the fact that BEIR V fails to exclude chronic lymphocytic leukemias, which have not been shown to be radiogenic.

The NRC and NRPB recommend simple absolute risk models, which distribute the risk evenly over a 25 or 38 y time period, respectively. In contrast, both ICRP's NIH model and the BEIR V model for leukemia represent statistical fits to the temporal dependence in the Japanese data. The NIH model, for example, predicts a lognormal temporal response, with a shape dependent on age at exposure. The ICRP multiplicative model is least satisfactory in

Table 1. Low-LET U.S. population fatal cancer risk (deaths per 10⁴ person-Gy) for five models.

Cancer Site	NRPB	BEIR V	NUREG/ CR-4214	ICRP	
				Mult.	NIH
Nonleukemia ¹	966	—	—	966	773
Digestive	179 ²	263	297	457 ³	434 ³
Esophagus	—	—	14.9 ⁴	6.2	21.3
Stomach	29.3	—	74.3 ⁴	29.3	274
Colon	120	—	149 ⁴	381	109
Liver	30 ⁵	30 ⁵	29.7 ⁴	30 ⁵	30 ⁵
Respiratory	—	187	—	—	—
Lung	265	—	149	265	78.7
Bone	10 ⁶	14 ⁷	8.1	9.3 ⁸	9.3 ⁸
Skin	3.0	—	1.8	2.0 ⁹	2.0 ⁹
Breast	78.0	33.5	46.2	116	32.7
Ovary	—	—	32.2 ¹⁰	47.5	25.0
Bladder	—	—	49.6 ¹⁰	64.0	38.9
Thyroid ¹¹	6.3	22.5	6.4	7.5	7.5
Leukemia	81.7	164 ¹²	89.9	110	97.9
Remainder	424 ¹³	329	248	374	276

Totals:					
Solid tumors ¹⁴	—	812	754	1293	855
All sites ¹⁵	—	977	845	1403	953
Nonleukemia + leukemia ¹⁶	1048	—	—	1076	871

Notes for Table 1.

General: All risks are calculated for a stationary population using 1980 decennial U.S. vital (see Appendix A.) The DDREF is one. Risks not in italics are calculated directly from the models given in the references. Those in italics are sums of sites or make use of supplementary information.

Notes for Table 1. (Cont.)

1. NRPB and ICRP provide explicit models for all solid tumor (nonleukemia) mortality risk. These nonleukemia models presume a uniform dose to all tissues.
2. Sum of stomach, colon, and liver risks.
3. Sum of esophagus, stomach, colon, and liver risks.
4. NUREG/CR-4214 esophagus, stomach, colon, and liver risks are 0.05, 0.25, 0.50, and 0.10 times the digestive risk, respectively.
5. NRPB, BEIR V, and ICRP liver risks are the corresponding alpha dose risks divided by an RBE of 10.
6. NRPB alpha dose bone risk divided by an RBE of 10.
7. BEIR V incidence value multiplied by a lethality factor of 0.7 for comparability.
8. ICRP alpha dose bone risk divided by an RBE of 10.
9. ICRP also recommends this value for the low-dose, low-dose-rate region.
10. NUREG/CR-4214 ovary and bladder risks are 0.13 and 0.20 times the remainder risk, respectively.
11. Except for BEIR V, all thyroid risks are based on NCRP Report 80 (NCRP 1985).
12. The linear coefficient of the BEIR V linear-quadratic model for leukemia has been doubled for comparability with the other model estimates.

13. NRPB remainder risk is calculated as the nonleukemia risk less the sum of the stomach, colon, liver, lung, bone, breast, and thyroid values.
14. Sum of site specific risks for each model as follows:
BEIR V: Digestive, respiratory, breast, and remainder.
NUREG/CR-4214: Digestive, lung, bone, breast, thyroid, and remainder.
ICRP: Esophagus, stomach, colon, lung, breast, ovary, bladder, and remainder.
15. Sum of all solid tumor and leukemia risks (BEIR V, NUREG/CR-4214, and ICRP, only).
16. Sum of nonleukemia and leukemia risks (NRPB and ICRP, only).

describing the temporal response, concentrating the excess risk at the end of the expression period, opposite to what is actually observed.

Breast. A major issue with respect to breast cancer is in the transport of risk from Japan to the U.S., where the baseline rates are much higher. For example, the ICRP multiplicative and NIH projections of breast cancer risk for the U.S. differ by almost a factor of 4 (Land and Sinclair 1991). The NRPB and NUREG/CR-4214 models do not have this problem since they are based on North American data. These model projections agree fairly well with ICRP's NIH projection but are substantially lower than the projection made with ICRP's multiplicative model.

Lung. Lung cancer risks are highly uncertain due to uncertainties in age and temporal dependencies, and in transporting risk from Japan to the U.S., where the baseline lung cancer rates are considerably higher. The NRPB relative risk and ICRP multiplicative models are identical and project the highest risks; these models both presume that the age-specific constant relative risk coefficients derived from the Japanese data apply to other populations. The BEIR V respiratory model yields a lower estimate because of its temporal fall-off. ICRP's NIH projection is lower because of the difference between Japanese and U.S. lung cancer rates. The NUREG/CR-4214 model produces a higher estimate of risk for childhood exposures but a lifetime risk projection similar to that from BEIR V or an average of the two ICRP model projections.

Digestive. All of the stomach and colon cancer risk models are constant relative risk models derived from the ABSS data, each incorporating a substantially higher coefficient for the younger age-at-exposure groups. Nevertheless there are important differences among them. For example, as shown below from a comparison of Land and Sinclair's multiplicative and NIH projections (1991), the results for stomach and colon are very sensitive to how one transports the risk from Japan to the U.S.

Table 1a. Low-LET U.S. population risks (deaths per 10⁴ person-Gy) of stomach and colon cancer for two ICRP Publication 60 projection models.

Cancer type	Transport model			
	Multiplicative		NIH	
	Male	Female	Male	Female
Stomach	32	45	221	333
Colon	223	602	178	143

These results highlight the large colon cancer contribution in the multiplicative model - especially for females. As noted previously, the NRPB reduced its colon cancer risk estimate by decreasing the risk coefficient for childhood exposures. It is also instructive to compare estimates of total digestive cancers made with the BEIR V model and the ICRP multiplicative model. (See Table 1.) Although both are age-specific constant relative risk models, which

transport relative risk coefficients directly from the bomb survivors to the U.S. population, the former projects only about 65% of the digestive cancer risk. This seems to be largely a consequence of BEIR V's modeling digestive cancers as a single group, coupled with the differences in baseline stomach and colon cancer rates between Japan and the U.S. The same concern would probably apply to Gilbert's estimate of digestive cancer risk.

Other/Remainder. Since the sites for which risk estimates are developed differ, the sites included in the remainder category differ among the sets of models considered here. The NRPB does not develop a risk model for this category; instead the projection is obtained as a difference between the model projection for total (nonleukemia) cancers and for modeled (nonleukemia) sites. This approach yields a higher estimate for the remainder sites than obtained by modeling the risk for these sites directly. The lack of an explicit model and the assignment of about 40% of the total risk to this category might be regarded as weaknesses of the NRPB approach.

Miscellaneous Specific Organs. Each of the reports cites high-LET Thorotrast data as the basis for its liver cancer risk estimate. Assuming an RBE of 10, for the purpose of this comparison, all the models yield a liver cancer risk estimate of 30 per 10^4 Gy. The reports recommend bone cancer risk estimates derived from Ra-224 studies. Based again on an assumed RBE of 10 and a bone cancer lethality of 0.7, the estimates are in reasonably good agreement with one another.

ICRP 60 recommends a risk estimate for skin cancer incidence of 980 per 10^4 Gy; 0.2% of these cancers are assumed to be fatal. The NRC bases its skin cancer model on ICRP 60 and, therefore, projects essentially the same risk. (The two reports differ on the risk at low dose rates; the ICRP makes no adjustment in extrapolating from high dose rates, whereas the NRC applies a DDREF of 2.) The NRPB employs an older ICRP model, which projects about 30% of the incidence but 150% of the mortality as ICRP 60.

The NRC, NRPB, and ICRP 60 all calculate thyroid cancer using the absolute risk model recommended in NCRP 80 (NCRP 1985). From the Israeli Tinea Capitis data (Ron and Modan 1984), BEIR V develops its own (relative risk) model, which projects about 3 times higher risk than the NCRP model.

The ICRP estimates risk for the bladder and ovary, using the age-average relative risk coefficients derived from the ABSS data. None of the reports provides an estimate for kidney, which receives a relatively high dose from certain radionuclides, especially uranium. The ABSS shows a non-statistically-significant elevation in kidney cancer associated with radiation. The existence of a radiogenic risk for this site seems to be confirmed by the cervical cancer study (NAS 1990). The estimate of kidney cancer risk here is based on the ABSS kidney data. An alternative would be to calculate the risk using a model developed for remainder sites. A preliminary examination indicates this approach would yield roughly similar results.

C. Extrapolation to Low Doses and Dose Rates

As discussed in Section I.B, it is widely assumed that the risks of low-LET radiation are reduced by a DDREF at low doses and dose rates. For leukemia, BEIR V advocates a linear-quadratic model, consistent with a DDREF of 2. For other sites, the report recommends no reduction in risk for low, acute doses but suggests that a reduction by a factor of 2 or more may be appropriate at low dose rates. ICRP 60 contains a fairly detailed discussion of the issue [ICRP 1991: pp. 108-112] and recommends that a DDREF of 2 be used for radiation protection purposes at this time. The NRC concurs with the ICRP recommendation, except in the case of breast or thyroid, for which a DDREF of 1 is adopted. The NRPB recommends a DDREF of 1, 2, and 3, respectively, for thyroid, breast, and all other sites.

D. High-LET Risk Estimates (RBE)

The ICRP (1991) assumes that alpha radiation produces 20 times the risk, per unit absorbed dose (Gy or rad), as low-LET radiation. This relationship is meant to hold in the limit of low doses and dose rates. Thus, it already takes into account the assumed DDREF of 2 for low-LET radiation; at high acute doses, the RBE would be 10. This must be kept in mind both when calculating alpha particle risks using models derived from low-LET epidemiological data and when estimating low-LET risks (for bone and liver) based on high-LET studies. The NRC and NRPB reports also assume that at low doses the risk per Gy from alpha particles is 20 times that from gamma rays.

IV. Methodology for Estimating Radiogenic Cancer Risks

A. Selection of Risk Models

In our opinion, the models developed by Gilbert for the NRC, and by Land and Sinclair for the ICRP, are preferable for EPA's needs to the others considered here. BEIR V reveals novel features of the Japanese data, but for reasons outlined above, was not deemed to provide a good basis for EPA's nominal "best estimates" of radiogenic cancer risk. The NRPB models are very similar to the ICRP multiplicative models, but the NRPB makes a somewhat arbitrary adjustment to its colon cancer model for childhood exposures and appears deficient in its handling of the "remainder" category. It could also be argued that, in view of the uncertainties, weight should be given to an "additive transport" of risk estimates, where the radiogenic risk is assumed to be insensitive to differences in baseline cancer rates between populations. An additive type of transport (such as that provided by ICRP's NIH projection) leads to somewhat lower estimates of risk for most radionuclides, especially for those retained in the lung. (See Table 1.)

To a large extent, the ICRP approach reflects a well defined, predetermined procedure in which the excess cancer mortality observed in the A-bomb survivors, by site, age, and sex, are used to calculate risk in the U.S. population. However, Land and Sinclair express no preference between the multiplicative and NIH methods of projecting risk from one population to another, and the ICRP ended up adopting an arithmetic average of the two methods for each site. The NRC approach, on the other hand, rests to a degree on judgments, reached after an examination of

all the epidemiological data and a consideration of alternative modeling approaches, on a site specific basis.

Land and Sinclair's models involve more detailed age and site specific information. For example, Gilbert does not provide age and sex-specific risk coefficients for individual types of digestive cancers. Instead she develops a model for uniform irradiation of the digestive organs and then indicates what fraction of the total risk should be assigned to each. Since the age and sex dependencies differ among the various digestive organs, considerable manipulation is required to obtain organ-specific relative risk coefficients that will result in calculated lifetime risks partitioned as Gilbert describes. Some might prefer Gilbert's approach, however, since it points up the sketchiness of the organ-specific epidemiological data. The BEIR V Committee, for this reason, also chose to model digestive cancers together as a single class.

Similar considerations apply with respect to the dependence of risk on age at exposure, where the ICRP incorporates a more detailed breakdown. Due probably in large part to sampling errors, the ICRP models display some anomalous variations in risk with age at exposure. Such variations are smoothed out in the NUREG/CR-4214 models, but significant real features of the age dependence may be lost in the process.

Gilbert's breast cancer model has the advantage of being derived from North American data; however, Land and Sinclair's NIH projection of lifetime risk for breast cancer is comparable to Gilbert's and, qualitatively, exhibits the same sharp decrease in risk with age at exposure.

Conclusions. Having reviewed the scientific information and models outlined here, we have chosen to base the risk estimates for most organs on Land and Sinclair's models. (A notable exception is breast cancer where Gilbert's model is adopted.) In view of the uncertainty over the transport of risk estimates from Japan to the U.S., we have adopted a methodology, described below, which yields risk estimates intermediate between the multiplicative and NIH projections of Land and Sinclair. This methodology has been reviewed and judged acceptable by EPA's Radiation Advisory Committee (Loehr and Nygaard 1992).

B. Transport of Risk Estimates from Japan

Land and Sinclair present two models for calculating risk in a population, differing in how risk estimates are to be transported from the Japanese atomic bomb survivor study (ABSS) population. Both models assume a constant excess relative risk coefficient beginning 10 years after an exposure and continuing throughout the rest of life for each cancer site (except for leukemia). One model (multiplicative) assumes that the relative risk coefficient is the same across populations. The other (NIH) assumes that the relative risk coefficients for the target population should yield the same risks as those calculated with the additive risk coefficients from the original population over the period of epidemiological follow-up (excluding the minimal latency period.) These excess relative risk coefficients are then used to project the risk over the remaining years of life. Projections made for the U.S. using the NIH model are much less sensitive to differences in site specific baseline rates between Japan and the U.S. than are those using the multiplicative model.

Data on North American women irradiated for medical purposes indicate a similar attributable risk for breast cancer induction as the ABSS study data, despite the substantially higher breast cancer rates found in the U.S. or Canada, compared to Japan. For breast cancer, therefore, the NIH model projection agrees with observation better than the multiplicative model projection. Comparative data on other radiation-induced cancers, however, are generally lacking or are too weak to draw any conclusions regarding the transportation of risk estimates from the ABSS population to the U.S.

Both transportation models have a degree of biological plausibility. For example, the multiplicative model is consistent with the hypothesis that radiation acts as an initiator while the factors responsible for differences in baseline rates act as promoters. Alternatively, if both radiation and these factors act independently, but at the same stage in the carcinogenesis process, their effects should be additive, and radiation risks should be similar between populations despite differences in baseline rates. In actuality, the situation is likely to be more complex than either of these alternatives, leading to an interaction that is intermediate between multiplicative and additive. In this case, the radiation risk for the U.S. population may lie between the multiplicative and NIH projections from the ABSS.

Given the uncertainty in the transportation of risk across populations, we adopt here a model in which most age and site specific risk coefficients are taken to be geometric means of the corresponding coefficients from the multiplicative and NIH models of Land and Sinclair. Our choice of a geometric mean coefficient (GMC) model reflects a judgment regarding the distribution of uncertainty associated with the transportation of risk. We believe this approach provides a reasonable central estimate of the risk, by organ and by age at irradiation. While giving weight to both the multiplicative and NIH approaches, it tends to deemphasize extreme values (e.g., the multiplicative projection for colon cancer) which may reflect large extrapolations based on a few excess cancers observed among those exposed as children. (Note: since the risk coefficients for the respective models are age and sex specific, the average lifetime risks calculated with the GMC model are, in general, not the same as the geometric means of the average lifetime risks calculated with the multiplicative and NIH models.)

Risk estimates for esophagus, stomach, colon, lung, ovary, bladder, leukemia, and residual, derived using the multiplicative, NIH, and geometric mean projection models are given in Table 2. Shown for comparison are the estimates derived using Gilbert's models for these same organs. Gilbert's models were, as discussed previously, developed primarily from the same epidemiological data, but incorporate the author's judgment as to what constitutes a reasonable central estimate of the risk in light of all available information and uncertainties. As can be seen from Table 2, the GMC model yields lifetime risks that agree well with those calculated using Gilbert's models.

Table 2. Comparison of fatal cancer risks (deaths per 10⁴ person-Gy) for the geometric mean coefficient (GMC) projection model with Land & Sinclair (ICRP) and NUREG/CR-4214 models.

Cancer type	Land & Sinclair		GMC	NUREG/CR-4214
	Mult	NIH		
Esophagus	16.2	21.3	18.1	<i>14.9</i>
Stomach	29.3	274	88.7	<i>74.3</i>
Colon	381	109	196	<i>149</i>
Lung	265	78.7	143	149
Breast	116	32.7	60.8	46.2
Ovary	47.5	25.0	33.2	32.2
Bladder	64.0	38.9	49.7	<i>49.6</i>
Leukemia	110	97.9	99.1	89.9
Residual	374	276	297	238
Total	<i>1403</i>	<i>953</i>	<i>986</i>	<i>844</i>

Notes for Table 2.

Model risks are calculated with 1980 U.S. vital statistics for a DDREF of one.

Values in roman type are calculated directly from the models given in the references. Those in italic type are sums of sites or make use of supplementary information.

The sex and age at exposure specific risk model coefficients for the GMC model are the geometric means of the corresponding coefficients for the Land and Sinclair (1991) multiplicative and NIH risk transportation models (see text). Note that since the risk model coefficients are age and sex specific, the average lifetime risks for the GMC model are not simply equal to the geometric mean of the risks for the corresponding multiplicative and NIH models.

The residual risk shown for the NUREG/CR-4214 model (Gilbert 1991) includes the balance of the digestive site risk.

Table 3. Residual site mortality risk coefficients for the additive and multiplicative projections of Land and Sinclair (1991).

Age group (y)	Additive (10 ⁻⁴ per Gy-y)		Multiplicative (per Gy)	
	Male	Female	Male	Female
0-9	0.096	1.73	0.091	2.370
10-19	2.28	1.60	0.762	0.876
20-29	5.42	4.98	0.682	0.969
30-39	4.18	4.16	0.246	0.403
40+	2.09	2.96	0.052	0.151

C. Adjustments to Models

For all sites except leukemia, the same temporal response was employed by Land and Sinclair in both the multiplicative and NIH models; i.e., a constant relative risk with a 10 y minimal latency period. For leukemia, the temporal dependencies are different. The multiplicative model presumes a constant relative risk over an expression period extending from 2 y to 40 y after exposure; the NIH model, on the other hand, presumes a lognormal variation in relative risk with a width dependent on the age at exposure (National Institutes of Health 1985). Averaging the risk coefficients corresponding to these two different temporal models is nonsensical. Before taking the geometric mean of the model coefficients, therefore, we first adjusted the multiplicative model so that it would exhibit the same form of temporal response as the NIH model, holding constant the lifetime risk for a given age at exposure. The NIH temporal response was chosen over that of the multiplicative model because it conforms better to the epidemiological data. [The BEIR V Committee preferred a model incorporating two steps, with the temporal width of the steps dependent on age at irradiation (NAS 1990)].

In view of the statistical limitations in the data, analysts have assumed a constant ratio of male to female risk coefficients across ages for all specific cancer sites (Shimizu et al. 1988, Land and Sinclair 1991). Land and Sinclair did not, however, constrain their models for "residual" cancers in this way. As shown in Table 3, this leads to apparent anomalies in the risk coefficients for the 0-9 y age group. Due probably to large sampling errors in this group, the estimated risk coefficient appears to be low for males but high for females. Accordingly, we have replaced the 0-9 y values given by Land and Sinclair with those given for the age group 10-19 y. The overall effect of this change on U.S. population risk estimates is very slight: about 10% for the residual organ risk and about 2% for the uniform whole body risk.

D. Other Cancer Sites

For all the sites listed in Table 2, a statistically significant excess of cancer mortality (at the 95% confidence level) has been observed in the ABSS (Shimizu et al. 1988). The excess for breast cancer is also statistically significant, but it seems preferable to base the risk estimate on the available North American data, bypassing the question of transporting risk estimates across populations. For kidney, the ABSS data are suggestive of a risk but narrowly miss statistical significance. The existence of a radiogenic kidney cancer risk is, however, borne out by the cervical cancer study (NAS 1990; Boice et al. 1988). Given the importance of the kidney as a possible target organ for uranium and some other radionuclides, we have developed a risk estimate for this site based on the ABSS data. We also present risk estimates for liver, bone, and thyroid.

Breast. Using the multiplicative model projection from the Japanese data, a breast cancer (mortality) risk of $116 \times 10^{-4} \text{ Gy}^{-1}$ is calculated. The NIH projection is $32.7 \times 10^{-4} \text{ Gy}^{-1}$. Gilbert's model, based on the North American data, yields a lifetime risk of $46.2 \times 10^{-4} \text{ Gy}^{-1}$, much closer to the NIH projection. The geometric mean model projection ($60.2 \times 10^{-4} \text{ Gy}^{-1}$) also agrees reasonably well with Gilbert's model estimate.

Kidney. The age- and sex-averaged excess relative risk (ERR) coefficient for kidney cancer mortality from the ABSS is 0.5829 Gy^{-1} , with a 90% confidence interval of (-0.09, 1.94). The corresponding absolute risk (AR) coefficient from Table 2B of Shimizu et al. (1988) is $9 \times 10^{-6} (\text{Gy y})^{-1}$. However, these values are based on shielded kerma rather than on organ-absorbed dose as needed for the risk model. Risk coefficients based on kidney-absorbed dose were calculated assuming that the ratio of the organ-absorbed dose ERR coefficients to the shielded kerma values is the same as for the bladder. The bladder-absorbed dose ERR from Table 1 of Land and Sinclair (1991) is 1.3395 Gy^{-1} and the corresponding shielded kerma value from Table 2-27 of Shimizu et al. (1988) is 1.1343 Gy^{-1} . Accordingly, the kidney-absorbed dose ERR and AR coefficients for kidney cancer are calculated to be 0.6883 Gy^{-1} and $1.06 \times 10^{-5} (\text{Gy y})^{-1}$, respectively. Using this AR coefficient, the NIH model ERR coefficient is calculated to be 0.2663 Gy^{-1} . The geometric mean model ERR projection (see Table 4) is 10.9 kidney cancer deaths per 10^4 Gy , about 20% of bladder cancer mortality.

Liver. Based on Thorotrast data, BEIR III and BEIR IV estimate the risk of fatal liver cancer induction by alpha particle radiation to be $300 \times 10^{-4} \text{ Gy}^{-1}$ (NAS 1980, 1988). Assuming an RBE of 10 for alpha particles (see below), a low-LET risk of $30 \times 10^{-4} \text{ Gy}^{-1}$ is derived. For purposes of calculation, we have adopted a constant relative risk model independent of age-at-exposure and sex.

Bone. As a basis for estimating radiation-induced bone sarcomas, we adopt BEIR IV's estimate of $2 \times 10^{-2} \text{ Gy}^{-1}$ for alpha irradiation by ^{224}Ra (NAS 1988), derived from the work of Mays and Speiss (1984). This estimate, however, refers to *average skeletal dose* rather than *endosteal cell dose* (NAS 1980, 1988; Mays and Speiss 1984). Taking the endosteal dose to be 7.5 times that of the whole skeleton, the alpha risk estimate is 2.7×10^{-3} cases per Gy; for

low-LET, the risk is again assumed to be 10 times lower ($2.7 \times 10^{-4} \text{ Gy}^{-1}$). About 70% of bone sarcomas are fatal (ICRP 1991); hence, for mortality, the low-LET risk estimate is $1.9 \times 10^{-4} \text{ Gy}^{-1}$. The ICRP 60 estimate of bone cancer risk is higher because it confuses endosteal and average skeletal doses (ICRP 1991, Puskin et al. 1992); unfortunately, the NRC erroneously adopted the ICRP estimate (Gilbert 1991). In a subsequent report (Gilbert 1993), the NRC has addressed this error. Following BEIR III (NAS 1980), a constant absolute risk model was selected for projecting risk, with an expression period extending from 2 to 27 years after exposure.

Thyroid. Thyroid risk estimates are based on NCRP Report 80 (NCRP 1985). Both the NRC and the ICRP have also adopted this approach (Gilbert 1991, ICRP 1991). The estimated fatality risk is calculated to be $6.4 \times 10^{-4} \text{ Gy}^{-1}$, 1/10 the incidence risk. The estimated incidence and mortality risks are each reduced by a factor of 3 in the case of exposures to iodine-125, -129, and -131. [This reduction includes the effect of lowered dose rate on the risk, as well as possible other factors; hence, the DDREF of 2 applied to organ specific risk estimates (see below) should not be applied in the case of these radioiodine exposures.]

In addition to thyroid cancers, radiation has been found to induce benign thyroid nodules. Maxon et al. (1985) has estimated that: for external X or gamma rays, the risk of a radiogenic thyroid nodule is about 3.7 times that of a radiogenic thyroid cancer; for iodine-131, the nodule risk is about 2.1 times the cancer risk.

Skin. Estimates of skin cancer risks are highly uncertain, but the mortality risk is known to be relatively low. For acute exposures, we have adopted the mortality risk estimate in ICRP 60, $2 \times 10^{-4} \text{ Gy}^{-1}$; however, in contrast to ICRP, we have applied a DDREF of 2 in estimating the skin cancer risk at low doses and dose rates (see below).

Radiation induced skin cancers are of two types: basal cell and squamous cell carcinomas. The former are nearly all curable, perhaps about 99.99%, but about 1% of the latter may be fatal (ICRP 1991, 1992). As an upper bound, the ICRP estimates that one of six radiogenic skin cancers would be squamous cell. Based on these considerations, the ICRP estimates that only 0.2% of all radiation-induced skin cancers are fatal (ICRP 1991, 1992).

The great majority of radiation induced skin cancers should be easily curable and result in little trauma for the patient (ICRP 1992). However, left unattended, some of these cancers, though still not fatal, may require more intensive medical treatment or be disfiguring. In the absence of data on the fraction of radiogenic skin cancer cases that might be regarded as serious, we have excluded nonfatal radiogenic skin cancers from the estimates of risk.

E. Summary of Site Specific Cancer Mortality Risk Estimates

Table 4 summarizes an extended set of organ risk estimates calculated using the revised EPA set of models described above, and using the NUREG/CR-4214 and ICRP models (Gilbert 1991, Land and Sinclair 1991, ICRP 1991). A detailed listing of age- and site-specific risk coefficients for the EPA revised models is given in the Appendix. For most sites, the ICRP estimates reflect the multiplicative and NIH projections of the ABSS estimates to the U.S.; the basis for ICRP estimates of risk for liver, bone, thyroid, and skin are described separately in ICRP 60 (ICRP 1991). For comparison purposes, a DDREF of 1 is again assumed for all sites. The resulting whole body risk calculated using the revised EPA set of models is 9.72×10^{-2} fatal cancers per person-Gy.

F. Incidence Risk Estimates

To obtain estimates of radiation-induced cancer incidence, each site specific mortality risk estimate is divided by its respective lethality fraction, i.e., the fraction of radiogenic cancers at that site which are fatal. Aside from thyroid cancer, the lethality fraction is generally assumed to be the same for radiogenic cancers as for the totality of other cancers at that site. [An exception is sometimes made for thyroid cancer because the radiogenic cases are confined to specific types, which have a somewhat lower than average lethality (NCRP 1985)].

Table 5 reproduces a list of lethality fractions recommended by ICRP 60. Two limitations with respect to this list should be noted. First, the values reflect only cancers appearing in adults. It appears that leukemia is now often curable in children. However, most radiogenic leukemias in the atomic bomb survivors occurred before successful treatment became available. Hence, the leukemia mortality risks derived from the Japanese may more properly reflect incidence rather than mortality for children. Second, the values listed in Table 5 are, in part, judgments, since there is no completely reliable way to determine long term survival based on current (or future) treatment modalities. As in the case of childhood leukemia, however, improved survival would imply an overestimation of mortality risks rather than an underestimation of incidence.

Recognizing these limitations, we calculate site specific incidence using the ICRP 60 lethality fractions, except for skin, which is projected by this method to contribute most of the nonfatal cancers induced by uniform whole body irradiation. At least 83% are basal cell carcinomas ($\approx 0.01\%$ lethality) and the remainder squamous cell carcinomas ($\approx 1\%$ lethality). The incidence estimate employed here reflects only fatal cases and omits the much larger number of nonfatal cases, most of which are easily treated (see Section D).

Table 4. Site specific mortality risk estimates (deaths per 10⁴ person-Gy) for proposed EPA model compared with those for ICRP and NUREG/CR-4214 models (male and female combined, DDREF=1).

Cancer site	ICRP		EPA	NUREG/CR-4214
	Mult	NIH		
Esophagus	16.2	21.3	18.1	14.9
Stomach	29.3	274	88.7	74.3
Colon	381	109	196.4	149
Liver	30	30	30.0	29.7
Lung	265	78.7	143.2	149
Bone	9.3	9.3	1.9	8.1
Skin	2.0	2.0	2.0	1.8
Breast	116	32.7	46.2	46.2
Ovary	47.5	25.0	33.2	32.2
Bladder	64.0	38.9	49.7	49.6
Kidney	—	—	10.9	—
Thyroid	7.5	7.5	6.4	6.4
Leukemia	110	97.9	99.1	89.9
Residual	325	227	246.2	193
Total	1403	953	972.0	844

Notes for Table 4

The ICRP risk estimates for liver, bone, skin, and thyroid are general (rather than sex-specific) estimates from ICRP Publication 60 (ICRP 1991).

For those sites above (other than breast) that are also shown in Table 1, the proposed EPA risk model coefficients are the same as those for the geometric mean coefficient (GMC) model. If the GMC model for the female breast had been used, the breast cancer risk would be 118.5 and 60.2 per 10⁴ Gy for the female and combined male and female populations, respectively. The total risk estimates would change accordingly.

The residual risk estimate is different from that in Table 1 because it does not include risks for sites (viz., liver, bone, skin, kidney, and thyroid) that are specifically estimated.

Table 5. Lethality data for adult cancers by site.

Cancer site	Lethality fraction <i>k</i>
Esophagus	0.95
Stomach	0.90
Colon	0.55
Liver	0.95
Lung	0.95
Bone	0.70
Skin	0.002
Breast	0.50
Ovary	0.70
Bladder	0.50
Kidney	0.65
Thyroid	0.10
Leukemia (acute)	0.99
Residual	0.71

Notes for Table 5.

Lethality fractions (mortality:morbidity ratios) except for residual are from Table B-19 of ICRP Publication 60 (ICRP 1991). Residual lethality fraction is calculated from the corresponding value of $(2-k)$ in Table B-20 of the same document.

G. Dose and Dose Rate Effectiveness Factor (DDREF)

The issue of DDREF was discussed in Section II.B. After reviewing the information and arguments pertinent to the choice of a DDREF, we concluded that a value of 2.0 provides a reasonable "best estimate." The Agency's Radiation Advisory Committee agreed "that this choice is reasonable and ... consistent with current scientific judgment" (Loehr and Nygaard 1992). A DDREF of 2 has recently been adopted by the ICRP (1991), as well as by other organizations (Gilbert 1991, CIRRPC 1992), and is expected to be widely applied for purposes of risk assessment and radiation protection worldwide. The uncertainty in the DDREF will be factored into EPA's assessments of uncertainty in radiation risk, however.

The DDREF will be applied to all organ-specific risks except for the breast. There is epidemiological evidence that dose fractionation has little or no effect on risk to the breast (NAS 1988); moreover, the risk model we have adopted is based mainly on fluoroscopy studies in which the doses were in fact delivered as multiple small fractions (NAS 1990, Gilbert 1991). Hence, we have adopted a DDREF of 1 for breast cancer. This choice assumes that the risk (per unit dose) of highly fractionated exposures approximates the risk at low doses and dose rates.

The conditions under which the DDREF should be applied remain to be defined. Although the biological mechanisms are not yet elucidated, according to current thinking: at low doses, repair is maximal, and the unrepaired DNA damage reflects single track events; at higher (acute) doses, repair decreases due to damage caused by multiple tracks passing through the cell nucleus in close temporal proximity. It would appear that repair efficiency is maximal for all doses below about 0.2 Gy (NCRP 1980). It also appears that DNA repair is essentially completed within a few hours after radiation-induced damage (Wheeler and Wierowski 1983, Ullrich et al. 1987). Consequently, maximum repair efficiency should occur so long as the dose does not exceed 0.2 Gy over a few hours. In view of these considerations, we have adopted UNSCEAR's recommendation that the DDREF should be applied whenever *the total dose is below 0.2 Gy or the dose rate is below 0.1 mGy/min* (UNSCEAR 1993).

H. Alpha Particle RBE

With the exception of radiation-induced breast cancer and leukemia, we have followed the ICRP recommendation (ICRP 1991) and assumed that the RBE for alpha particles is 20, in comparison to low-LET radiation at low doses and dose rates. Where the comparison is made against acute high doses of low-LET radiation, however, a value of 10 will be assumed for the alpha particle RBE. Thus the low-LET radiation DDREF of 2 we have used for these cancers is implicitly incorporated into the RBE value for alpha radiation.

For breast cancer induction, a DDREF of 1 has been adopted (see above). Therefore, the RBE will be independent of dose and dose rate. Since there is no DDREF correction of the low-LET breast cancer risk estimates at low doses and dose rates, it is assumed that the acute high dose RBE of 10 is also applicable to breast cancer at low doses and dose rates.

There is evidence that alpha particle leukemia risks estimated on the basis of an RBE of 20 are too high (EPA 1991). For this reason, an alpha particle leukemia risk estimate of $5.0 \times 10^{-3} \text{ Gy}^{-1}$ is employed, consistent with the available high-LET epidemiological data (NAS 1988, EPA 1991). Quantitatively, this would correspond to an RBE of 1 for this site (relative to low dose, low-LET radiation). This is not to imply that alpha radiation is no more carcinogenic than low-LET radiation in inducing leukemia. At least in part, the lower than expected leukemia risk produced by alpha emitters may result from a nonuniform distribution of dose within the bone marrow (i.e., average doses to sensitive target cells may be substantially lower than calculated average marrow doses). Thus the RBE of 1 should be

regarded as an "effective RBE," that reflects factors other than just the relative biological sensitivity to high- and low-LET radiations. Finally, we recognize that since the spatial distribution of the dose within the marrow will differ among alpha emitters, depending on the distribution of the radionuclide within bone and the energies of the emitted alpha particles, the effective RBE may be radionuclide dependent. However, this issue cannot be resolved with current data.

Our radon decay product risk estimates will continue to be based directly on radon epidemiological data. Currently, EPA's radon risk estimate is 2.2×10^{-4} fatal lung cancers per working level month (EPA 1992, Puskin 1992).

I. Summary of Revisions to EPA Low Dose Risk Estimates

Table 6 lists the revised EPA site specific cancer risk estimates (incidence and mortality), applicable at low doses and dose rates. For comparison, the corresponding estimates from EPA's 1989 Background Information Document for the Radionuclides NESHAPS are shown (EPA 1989). These revised EPA site specific mortality risk estimates are generally quite close to those in NUREG/CR-4214 (Gilbert 1991).

For low-LET radiation at low doses or dose rates, the lifetime fatal cancer risk estimate associated with uniform, whole-body irradiation of the U.S. population has increased by 24%, from 392 to 509 per 10^4 person-Gy. This value is similar to those determined by NRPB, ICRP, and NRC, and BEIR V, assuming a DDREF of 2. It is estimated that about 70% of all cancers induced by whole-body irradiation are fatal (nonfatal skin cancers excluded), corresponding to an incidence risk estimate of 7.61×10^{-2} Gy⁻¹. These increases occur despite the change from a DDREF of 1 in NESHAPS to a value of 2 here; without this change, the risk estimates would have more than doubled.

It should be emphasized that EPA's previously published lifetime risk estimates for chronic radionuclide exposures (EPA 1989) cannot be simply scaled by the ratio of whole-body risk estimates to arrive at risk estimates based on the revised models. In general, such exposures produce a non-uniform dose distribution within the body, which may also be time varying. As a result, estimation of revised radionuclide specific risks requires more detailed calculations.

Table 6. EPA low dose, low dose rate cancer risks (10^{-4} per Gy).

Cancer site	Mortality		Morbidity	
	NESHAPs	Revised	NESHAPs	Revised
Esophagus	9.1	9.0	9.1	9.5
Stomach	46.0	44.4	60.1	49.3
Colon	22.9	98.2	42.9	178.5
Liver	49.6	15.0	49.6	15.8
Lung	70.1	71.6	74.5	75.4
Bone	2.5	0.9	2.5	1.3
Skin	—	1.0	—	1.0
Breast	55.4	46.2	142.0	92.5
Ovary	—	16.6	—	23.7
Bladder	11.8	24.9	21.4	49.7
Kidney	5.9	5.5	21.4	8.4
Thyroid	6.4	3.2	64.3	32.1
Leukemia	44.8	49.6	44.8	50.1
Remainder	67.8	123.1	90.5	173.4
TOTAL	392.1	509.1	623.0	760.6

Notes for Table 6.

The Dose and Dose Rate Effectiveness Factor (DDREF) is 1 for breast and 2 for all other sites. These risk coefficients are applicable to all doses less than 200 mGy and for total doses greater than 200 mGy from dose rates less than 0.1 mGy/min. The revised model morbidity estimate for skin shown is for fatalities only. The entire morbidity risk for skin would be about 500 times greater. The thyroid morbidity risk includes only malignant neoplasms and does not include benign tumors or nodules. For most cancer sites, high-LET (alpha particle) risk estimates have increased by more than the corresponding low-LET estimates, reflecting the change in RBE from 8 to 20, which comes from adopting a DDREF correction at low doses of low-LET radiation (EPA 1989, NCRP 1990, ICRP 1991).

For occupational exposures, the mortality and incidence risk estimates are 3.94×10^{-2} Gy^{-1} and 5.67×10^{-2} Gy^{-1} , respectively. (Occupational risks were calculated assuming a constant exposure rate for both sexes between the ages of 18 and 65.)

References

- S. Abrahamson, M. Bender, S. Book, C. Buncher, C. Denniston, E. Gilbert, F. Hahn, V. Hertzberg, H. Maxon, B. Scott, W. Schull, S. Thomas. *Health Effects Model for Nuclear Power Plant Accident Consequence Analysis. Low LET Radiation, Part II: Scientific Bases for Health Effects Models*. NUREG/CR-4214, SAND 85-7185, Rev. 1, Part II, U.S. Nuclear Regulatory Commission, Washington, DC, May 1989.
- S. Abrahamson, M.A. Bender, B.B. Boecker, E.S. Gilbert, B.R. Scott. *Health Effects Models for Nuclear Power Plant Accident Consequence Analysis. Modifications of Models Resulting from Recent Reports on Health Effects of Ionizing Radiation, Low LET Radiation, Part II: Scientific Bases for Health Effects Models*. NUREG/CR-4214, Rev. 1, Part II, Addendum 1, LMF-132, August 1991.
- J.D. Boice, Jr., G. Engholm, R.A. Kleinerman, M. Blettner, M. Stovall, H. Lisco, W.C. Moloney, D.F. Austin, A. Bosch, D.L. Cookfair, E.T. Kremetz, H.B. Latourette, J.A. Merrill, L.J. Peters, M.D. Schulz, H.H. Storm, E. Bjorkholm, F. Pettersson, C.M.J. Bell, M.P. Coleman, P. Fraser, F.E. Neal, P. Prior, N.W. Choi, T.G. Hislop, M. Koch, N. Kreiger, D. Robb, D. Tobson, D.H. Thomson, H. Lochmuller, D.V. Fournier, R. Frischkorn, K.E. Kjorstad, A. Rimpela, M.H. Pejovic, V.P. Kirn, H. Stankusova, F. Berrino, K. Soigurdsson, G.B. Hutchison, and B. MacMahon. Radiation dose and second cancer risk in patients treated for cancer of the cervix. *Radiat. Res.* **116**, 3-55, 1988.
- CIRRPC. *Use of BEIR V and UNSCEAR 1988 in Radiation Risk Assessment*. 1992. Committee on Interagency Radiation Research and Policy Coordination; Office of Science and Technology Policy. Available as ORAU 92/F-64 through NTIS, Springfield, VA 22161.
- S.C. Darby, R. Doll, S.K. Gill and P.G. Smith. Long-term mortality after a single treatment course with X-rays in patients treated for ankylosing spondylitis. *Br. J. Cancer* **55**, 179-190, 1987.
- F.G. Davis, J.D. Boice, Jr., Z. Hrubek and R.R. Monson. Cancer mortality in a radiation-exposed cohort of Massachusetts tuberculosis patients. *Cancer Res.* **49**, 6130-6136, 1989.
- Environmental Protection Agency. *Radionuclides: Background Information Document for Final Rules. Volume I*. EPA 520/1-84-022-1, 1984.
- Environmental Protection Agency. *Risk Assessment Methodology. Environmental Impact Statement. NESHAPS for Radionuclides. Background Information Document — Volume 1*. EPA/520/1-89-005, 1989.
- Environmental Protection Agency. *Final Draft for the Drinking Water Criteria Document on Radium*. Prepared by Life Systems, Inc., NTIS: PB 91225631, EPA, 1991.

Environmental Protection Agency. *Technical Support Document for the 1992 Citizen's Guide to Radon*. EPA 400-R-92-011, 1992.

J.S. Evans, D.W. Moeller and D.W. Cooper. *Health Effects Model for Nuclear Power Plant Accident Consequence Analysis*, NUREG/CR-4214, SAND 85-7185, U.S. Nuclear Regulatory Commission, Washington, DC, 1985.

E.S. Gilbert. "Chapter 3: Late somatic effects." In: S. Abrahamson, M.A. Bender, B.B. Boecker, E.S. Gilbert, B.R. Scott. *Health Effects Models for Nuclear Power Plant Accident Consequence Analysis. Modifications of Models Resulting from Recent Reports on Health Effects of Ionizing Radiation, Low LET Radiation, Part II: Scientific Bases for Health Effects Models*. NUREG/CR-4214, Rev. 1, Part II, Addendum 1, LMF-132, August 1991.

E.S. Gilbert. "Chapter 3: Late somatic effects." In: S. Abrahamson, M.A. Bender, B.B. Boecker, E.S. Gilbert, B.R. Scott. *Health Effects Models for Nuclear Power Plant Accident Consequence Analysis. Modification of Models Resulting from Addition of Effects of Exposure to Alpha-Emitting Radionuclides, Part II: Scientific Bases for Health Effects Models*. NUREG/CR-4214, Rev. 1, Part II, Addendum 2, LMF-136, May 1993.

D.T. Goodhead. An assessment of the role of microdosimetry in radiobiology. *Radiat. Res.* **91**, 45-76, 1982.

A.J. Grosovsky and J.B. Little. Evidence for linear response for the induction of mutations in human cells by x-ray exposures below 10 rads. *Proc. Natl. Acad. Sci. USA* **82**, 2092-2095, 1985.

G.R. Howe. Effects of dose, dose-rate and fractionation on radiation-induced breast and lung cancers: the Canadian fluoroscopy study. In: *International Conference on Radiation Effects and Protection*, pp. 108-113. Japan Atomic Energy Research Institute, Tokyo, 1992.

ICRP. *ICRP Publication 60. 1990 Recommendations of the International Commission on Radiological Protection. Annals of the ICRP* **21**, No. 1-3, 1991.

ICRP. *ICRP Publication 59. The Biological Basis for Dose Limitation in the Skin. Annals of the ICRP* **22**, No. 2, 1992.

A.M. Kellerer and H.M. Rossi. The theory of dual radiation action. *Curr. Topics Radiat. Res. Quart.* **8**, 85-158, 1972.

C.E. Land and W.K. Sinclair. The relative contributions of different organ sites to the total cancer mortality associated with low-dose radiation exposure. In: *Risks Associated with Ionising Radiations, Annals of the ICRP* **22**, No. 1, 1991.

D.E. Lea. *Actions of Radiations on Living Cells*, Cambridge University Press, 1962.

- M.P. Little and M.W. Charles. Time variations in radiation-induced relative risk and implications for population cancer risks. *J. Radiol. Prot.* **11**, 91-110, 1991.
- M.P. Little, M.M. Hawkins, R.E. Shore, M.W. Charles and N.G. Hildreth. Time variations in the risk of cancer following irradiation in childhood. *Radiat. Res.* **126**, 304-316, 1991.
- R.C. Loehr and O.E. Nygaard. Evaluation of EPA's proposed methodology for estimating radiogenic cancer risks. Letter to William K. Reilly, EPA Administrator, Dec. 9, 1992.
- H. Maxon, S. Thomas, C. Buncher, S. Book and V. Hertzberg. Thyroid effects. In: *Health Effects Model for Nuclear Power Plant Accident Consequence Analysis. Part II: Scientific Basis for Health Effects Models*, pp. 181-226 (J.S. Evans, D.W. Moeller and D.W. Cooper, eds.) NUREG/CR-4214, U.S. Nuclear Regulatory Commission, Washington, D.C., 1985.
- C.W. Mays and H. Speiss. Bone sarcomas in patients given radium-224. In: *Radiation Carcinogenesis. Epidemiology and Biological Significance*, pp. 241-252 (J.D. Boice and J.F. Fraumeni, eds.) New York: Raven, 1984.
- National Academy of Sciences. *The Effects on Populations of Exposure to Low Levels of Ionizing Radiation (BEIR III)*. National Academy Press, Washington, D.C., 1980.
- National Academy of Sciences. *Health Risks of Radon and Other Internally Deposited Alpha-Emitters (BEIR IV)*. National Academy Press, Washington, D.C., 1988.
- National Academy of Sciences. *Health Effects of Exposure to Low Levels of Ionizing Radiation (BEIR V)*. National Academy Press, Washington, D.C., 1990.
- National Institutes of Health, *Report of the National Institutes of Health Ad Hoc Working Group to Develop Radioepidemiological Tables*, NIH Publication 85-2748, Superintendent of Documents, U.S. Government Printing Office, Washington, DC, 1985.
- NCRP. *Influence of Dose and Its Distribution in Time on Dose-Response Relationships for Low-LET Radiations. NCRP Report 64*. National Council on Radiation Protection and Measurements, Bethesda, MD, 1980.
- NCRP. *Induction of Thyroid Cancer by Ionizing Radiation. NCRP Report 80*. National Council on Radiation Protection and Measurements, Bethesda, MD, 1985.
- NCRP. *The Relative Biological Effectiveness of Radiations of Different Quality. NCRP Report No. 104*. National Council on Radiation Protection and Measurements, Bethesda, MD, 1990.
- Office of Radiation Programs. Reevaluation of EPA's Methodology for Estimating Radiogenic Cancer Risks. Transmittal from M. Oge to D.G. Barnes, Jan. 10, 1992.

D.A. Pierce and M. Vaeth. The shape of the cancer mortality dose-response curve for the A-bomb survivors. *Radiat. Res.* **126**, 36-42, 1991.

D.L. Preston. Breast cancer: Discussion. In: *The Future of Human Radiation Research, BIR Report 22*, p. 56 (G.B. Gerber, D.M. Taylor, E. Cardis and J.W. Theissen, eds.) British Institute of Radiology, London, 1991.

J.S. Puskin. An analysis of the uncertainties in estimates of radon-induced lung cancer. *Risk Analysis* **12**, 277-285, 1992.

J.S. Puskin, N.S. Nelson and C.B. Nelson. Bone cancer risk estimates. *Health Phys.* **63**, 579-580, 1992.

E. Ron and B. Modan. Thyroid and other neoplasms following childhood scalp irradiation. In: *Radiation Carcinogenesis: Epidemiology and Biological Significance*, pp. 139-151 (J.D. Boice Jr. and J.F. Fraumeni, Jr., eds.) Raven Press, N.Y., 1984.

Y. Shimizu, H. Kato and W.J. Schull. *Life Span Study Report 11. Part 2. Cancer Mortality in the Years 1950-85 Based on the Recently Revised Doses (DS86)*. RERF TR 5-88. 1988.

Y. Shimizu, H. Kato and W.J. Schull. Studies of the mortality of A-bomb survivors: 9. Mortality, 1950-1985: Part 2. Cancer mortality based on the revised doses (DS86). *Radiat. Res.* **121**, 120-141, 1990.

R.E. Shore, E.D. Woodward and L.H. Hempelman. Radiation induced thyroid cancer. In: *Radiation Carcinogenesis: Epidemiology and Biological Significance*, pp. 131-138 (J.D. Boice, Jr. and J.F. Fraumeni, Jr., eds.) Raven Press, N.Y., 1984.

J.W. Stather, C.R. Muirhead, A.A. Edwards, J.D. Harrison, D.C. Lloyd and N.R. Wood. *Health Effects Models Developed from the 1988 UNSCEAR Report*. NRPB-R226. 1988.

R.L. Ullrich, M.C. Jernigan, L.C. Satterfield and N.D. Bowles. Radiation Carcinogenesis: Time-dose relationships. *Radiat. Res.* **111**, 179-184, 1987.

UNSCEAR. *Sources, Effects and Risks of Ionizing Radiation*. United Nations, N.Y., 1988.

UNSCEAR. *Sources and Effects of Ionizing Radiation*. United Nations, N.Y., 1993.

K.T. Wheeler and J.D. Wierowski. DNA repair kinetics in irradiated undifferentiated and terminally differentiated cells. *Radiat. Environ. Biophys.* **22**, 3-19, 1983.

Appendix A: Calculational Methods

A. Introduction

A radiogenic cancer risk model defines the relationship between radiation dose and the subsequent force of mortality (or morbidity) attributable to that dose. As such, the model provides the basis for calculating a time (or age) varying rate coefficient in a death or disease process model. [General methods for structuring and solving the differential equations representing such stochastic processes can be found in Chiang (1980).] Thus, to calculate risks, the radiogenic risk model and other relevant quantities must be incorporated into a suitable calculational procedure.

The risk calculations in this appendix are for attributable risk. Attributable risk can be defined as the likelihood of death from (or development of) cancer that, according to the risk model, is caused by a radiation exposure. By way of comparison, the excess risk calculated in BEIR V (National Research Council 1990, Vaeth and Pierce 1989) excludes the fraction of the attributable risk that represents deaths or cases among persons who would be expected to die from (or to develop) cancer at a later age even if they had not been exposed.

The use of the attributable risk-per-unit-dose coefficients calculated here is limited to the asymptotic case, i.e., these coefficients can only be used for applications where the survival function is not significantly affected by the doses being assessed. When this is not the case, risks must be calculated explicitly for the specific doses under consideration.

Male and female survival data (up to an age of 110 y) are from the *U.S. Decennial life Tables for 1979-1981* (National Center for Health Statistics 1985). These data were used to calculate a combined life table for a male:female live birth ratio of 1.051. U.S. mortality data were extracted from 1979-1981 Vital Statistics Mortality Data, Detail Tapes (National Center for Health Statistics 1982, 1983, 1984). Deaths in these data files are classified according to the 9th edition of the International Classification of Disease (ICD) codes (Public Health Service 1980).

Radiogenic risk calculations require integrating functions of the risk model and vital statistics. The vital statistics are discrete data, typically tabulated at one or five year intervals. Radiogenic risk models are usually defined for several different age intervals and are inherently discontinuous. Previously, such risk model calculations were implemented by adapting actuarial methods developed for life table calculations, e.g., the CAIRD program (Cook et al. 1978). The method used here is to fit a cubic spline to discrete data and then to calculate interpolated values, derivatives, and integrals directly from the spline coefficients (de Boor 1978, Fritsch and Butland 1982). This method admits almost any form of risk model and eliminates most of the ad hoc approaches that were necessary with CAIRD.

B. Risk Model Formulation

There are two basic types of radiogenic cancer risk projection models: absolute risk and relative risk. An absolute risk model presumes that the age-specific excess force of mortality (or morbidity) due to a radiation dose is independent of cancer mortality or morbidity rates in the population. It can be written as

$$\epsilon(x, x_e) = \alpha(x_e) \zeta(t, x_e) \gamma(x) \quad (1)$$

where $\epsilon(x, x_e)$ is the excess force of mortality (or morbidity) ($y^{-1} \text{ Gy}^{-1}$) at age x due to a dose at age x_e ($x > x_e$),

$\alpha(x_e)$, the absolute risk coefficient ($y^{-1} \text{ Gy}^{-1}$), is a function of age at exposure, x_e ,

$\zeta(t, x_e)$, the time since exposure ($t = x - x_e$) response function, can also be a function of x_e ,

and $\gamma(x)$ is the age at expression response function,

The radiogenic risk models for bone, skin, and thyroid cancer in the Revised Methodology are all absolute risk models.

A relative risk model presumes that the age-specific excess force of mortality (or morbidity) due to a radiation dose is the product of an exposure-age-specific relative risk coefficient and baseline cancer mortality or morbidity rates in the population. The model can be written as

$$\eta(x, x_e) = \beta(x_e) \zeta(t, x_e) \gamma(x) \quad (2)$$

where $\eta(x, x_e)$ is the relative risk (Gy^{-1}) at age x due to a dose at age x_e ($x > x_e$),

$\beta(x_e)$, the relative risk coefficient (Gy^{-1}), is a function of age at exposure, x_e ,

$\zeta(t, x_e)$, the time since exposure ($t = x - x_e$) response function, may also be a function of x_e ,

and $\gamma(x)$ is the age at expression response function.

C. Revised Methodology Risk Models

Risk model coefficients

Risk coefficients for the Revised Methodology mortality risk models are shown in Table A.1. Absolute risk models are used for bone, skin, and thyroid cancers. Relative risk models are used for all other cancers.

Table A.1 Coefficients for the Revised Methodology mortality risk model (male and female by age group).

Cancer type	Risk model type*	Age group				
		0-9	10-19	20-29	30-39	40+
Male:						
Esophagus	R	0.2239	0.2312	0.2517	0.2892	0.3258
Stomach	R	1.2337	1.9165	1.9051	0.2881	0.2524
Colon	R	2.1565	2.1565	0.2809	0.4275	0.0899
Liver	R	1.3449	1.3449	1.3449	1.3449	1.3449
Lung	R	0.4060	0.4060	0.0453	0.1342	0.1794
Bone	A	0.0927	0.0927	0.0927	0.0927	0.0927
Skin	A	0.0672	0.0672	0.0672	0.0672	0.0672
Breast	R	0.0	0.0	0.0	0.0	0.0
Ovary	R	0.0	0.0	0.0	0.0	0.0
Bladder	R	1.2191	1.1609	1.0736	1.0544	0.9639
Kidney	R	0.3911	0.3911	0.3911	0.3911	0.3911
Thyroid	A	0.1667	0.1667	0.0833	0.0833	0.0833
Leukemia	R	672.16	244.07	323.47	228.86	142.51
Residual	R	0.7115	0.7140	0.1735	0.1754	0.1847
Female:						
Esophagus	R	1.0418	1.0896	1.2492	1.5831	2.0211
Stomach	R	3.4469	4.2721	4.0533	0.5797	0.4887
Colon	R	2.9680	2.9680	0.5755	0.8186	0.1870
Liver	R	1.3449	1.2449	1.3449	1.3449	1.3449
Lung	R	1.3753	1.3753	0.1921	0.5440	0.8048
Bone	A	0.0927	0.0927	0.0927	0.0927	0.0927
Skin	A	0.0672	0.0672	0.0672	0.0672	0.0672
Breast	R	0.7000	0.7000	0.3000	0.3000	0.1000
Ovary	R	1.3163	1.0382	0.8829	0.7678	0.6367
Bladder	R	1.0115	0.9296	1.0124	1.1032	0.9792
Kidney	R	0.3911	0.3911	0.3911	0.3911	0.3911
Thyroid	A	0.3333	0.3333	0.1667	0.1667	0.1667
Leukemia	R	761.07	225.81	281.76	153.12	154.28
Residual	R	0.7119	0.7174	0.2932	0.2963	0.3031

*Notes:

<u>Risk model type</u>	<u>Coefficient units</u>
Absolute (A)	$10^{-4} (\text{Gy y})^{-1}$
Relative (R)	Gy^{-1}

Time since exposure response function

The time since exposure (TSE) response function for all cancers except bone, thyroid, and leukemia has a 10 y minimal latency period and a lifetime plateau, i.e.,

$$\begin{aligned}\zeta(t) &= 0, & t < 10 \\ &= 1, & 10 \leq t\end{aligned}\quad (3)$$

while for bone cancer,

$$\begin{aligned}\zeta(t) &= 0, & t < 2 \\ &= 1, & 2 \leq t < 27 \\ &= 0, & 27 \leq t\end{aligned}\quad (4)$$

and for thyroid cancer,

$$\begin{aligned}\zeta(t) &= 0, & t < 5 \\ &= 1, & 5 \leq t\end{aligned}\quad (5)$$

For leukemia, the TSE function developed by the NIH Working Group for the Radioepidemiology Tables (National Institutes of Health 1985) is used. The Working Group fitted lognormal response functions for time since exposure greater than a minimal latency of 2 years to A-bomb survivor data for both chronic granulocytic leukemia (CGL) and acute leukemia (AL). These response functions can be expressed as follows:

$$\begin{aligned}\zeta(t, x_e) &= 0, & t < 2 \\ &= \phi(t, \xi(x_e), \sigma^2), & 2 \leq t\end{aligned}\quad (6)$$

where

$$\phi(t, \xi(x_e), \sigma^2) = \frac{\exp\left(-0.5(\ln(t-2) - \xi(x_e))^2 / \sigma^2\right)}{(t-2)\sqrt{2\pi\sigma^2}}. \quad (7)$$

The values of $\xi(x_e)$ and σ^2 are 2.68 and 1.51, respectively, for CGL. For AL, they are the value of the expression $1.61+0.151x_e+0.0005x_e^2$ and 0.65, respectively. The total leukemia response function is a weighted mean of the CGL and AL response functions:

$$\zeta(t, x_e) = 0.32 \phi(t, \xi_{cgl}(x_e), \sigma_{cgl}^2) + 0.68 \phi(t, \xi_{al}(x_e), \sigma_{al}^2). \quad (8)$$

Since this TSE function has a maximum value that is much less than 1, the risk model coefficients for leukemia in Table A.1 are much larger than those for other sites.

Age at expression function

The age at expression function, $\gamma(x)$, is equal to one for all models in the Revised Methodology.

D. Risk Calculations

Basic quantities

$S(x)$, the survival function, is the fraction of live born individuals expected to survive to age x . $S(0)=1$, and decreases monotonically for increasing values of x . $S(x)$ is obtained by fitting a cubic spline to the decennial life table values to provide a continuous function of x .

$e(x)$ is the expected lifetime (years) remaining for an individual who has attained age x . It is given by

$$e(x) = \frac{1}{S(x)} \int_x^{\infty} S(u) du . \quad (9)$$

$\mu(x)$ is the force of mortality or hazard rate (y^{-1}) at age x . Without a subscript, it is usually the total rate from all causes. A subscript is used (unless it is clear by context) to indicate a specific cause, i.e.,

$$\mu(x) = \sum_{\text{all } i} \mu_i(x) . \quad (10)$$

$S(x)$ is directly dependent on $\mu(x)$ since

$$S(x) = \exp\left(-\int_0^x \mu(u) du\right) . \quad (11)$$

When the baseline force of mortality $\mu_0(x)$ is incremented by $\mu_i(x)$, $S(x)$ becomes

$$\begin{aligned} S(x) &= \exp\left(-\int_0^x (\mu_0(u) + \mu_i(u)) du\right) \\ &= S_0(x) S_i(x) , \end{aligned} \quad (12)$$

where

$$S_0(x) = \exp\left(-\int_0^x \mu_0(u) du\right) , \quad (13)$$

and

$$S_i(x) = \exp\left(-\int_0^x \mu_i(u) du\right) . \quad (14)$$

For sufficiently small values of $\mu_i(x)$, $S_i(x)$ approaches a value of 1 for all values of x , i.e., $S_0(x)$ and $S(x)$ are essentially the same. For most environmental radiation risk assessment cases of practical interest, the increment of risk due to radiation satisfies this condition.

Attributable lifetime risk coefficient

The age-specific attributable lifetime risk (ALR) coefficient, $r(x)$, is the risk per unit dose of a subsequent cancer death (Gy^{-1}) due to radiation received at age x . For an absolute risk model, the asymptotic ALR coefficient is

$$r_i(x) = \frac{1}{S_0(x)} \int_x^\infty \epsilon_i(u) S_0(u) du . \quad (15)$$

Similarly, for a relative risk model,

$$r_i(x) = \frac{1}{S_0(x)} \int_x^\infty \eta_i(u) \mu_i(u) S_0(u) du . \quad (16)$$

These age-specific coefficients are principally used to calculate age-averaged coefficients and risks from radionuclide intakes or exposures.

Attributable lifetime loss coefficient

The attributable lifetime loss (ALL) coefficient $e(x)$, at age x , is the expected lifetime loss per unit dose (y Gy^{-1}) for a radiation dose at age x . For an absolute risk model, the asymptotic ALL coefficient is

$$e_i(x) = \frac{1}{S_0(x)} \int_x^\infty \epsilon_i(u) \varrho(u) S_0(u) du . \quad (17)$$

For a relative risk model,

$$e_i(x) = \frac{1}{S_0(x)} \int_x^\infty \eta_i(u) \mu_i(u) \varrho(u) S_0(u) du . \quad (18)$$

Age-averaged coefficients

The lifetime age-averaged risk and life loss coefficients are

$$\begin{aligned} \bar{r} &= \frac{\int_0^\infty r(x) S(x) dx}{\int_0^\infty S(x) dx} \\ &= \frac{\int_0^\infty r(x) S(x) dx}{\varrho} , \end{aligned} \quad (19)$$

and

$$\bar{e} = \frac{\int_0^{\infty} e(x) S(x) dx}{\int_0^{\infty} S(x) dx} \quad (20)$$

$$= \frac{\int_0^{\infty} e(x) S(x) dx}{\bar{e}} ,$$

respectively. Since the age distribution of a stationary population is proportional to $S(x)$, the stationary-population weighted average values are identical to the lifetime age-averaged ones. When the coefficients are averaged over a specific age interval, e.g., for assessing childhood or occupational exposures, the limits of integration in both the numerators and the denominators of these expressions are changed accordingly.

Sex-averaged coefficients

Since radiogenic cancer risk models are generally sex-specific, the resulting risk coefficients must be averaged for use in assessing risk to a combined population. This is accomplished by presuming a male:female sex ratio for live births of 1.051. Since $S(x)$ is sex-specific, the sex ratio is a function of age. The combined (sex-averaged) survival function is

$$S_c(x) = \frac{1.051 S_m(x) + S_f(x)}{2.051} , \quad (21)$$

where the subscripts m, f , and c refer to the male, female, and combined values respectively.

Similarly, the expected combined lifetime is

$$\bar{e}_c = \frac{1.051 \bar{e}_m + \bar{e}_f}{2.051} . \quad (22)$$

The combined age-specific force of mortality must reflect the age-specific contribution of each sex. Hence,

$$\mu_c(x) = \frac{1.051 S_m(x) \mu_m(x) + S_f(x) \mu_f(x)}{1.051 S_m(x) + S_f(x)} . \quad (23)$$

Combined age-specific ALR and ALL coefficients are

$$r_c(x) = \frac{1.051 S_m(x) r_m(x) + S_f(x) r_f(x)}{1.051 S_m(x) + S_f(x)} , \quad (24)$$

and

$$e_c(x) = \frac{1.051 S_m(x) e_m(x) + S_f(x) e_f(x)}{1.051 S_m(x) + S_f(x)} . \quad (25)$$

respectively. Combined age averaged ALR and ALL values must reflect the expected lifetime over the age interval. The lifetime average combined ALR and ALL coefficients are

$$r_c = \frac{1.051 \bar{e}_m r_m + \bar{e}_f r_f}{1.051 \bar{e}_m + \bar{e}_f} , \quad (26)$$

and

$$e_c = \frac{1.051 \bar{e}_m e_m + \bar{e}_f e_f}{1.051 \bar{e}_m + \bar{e}_f} . \quad (27)$$

Continuity considerations

While the integration of a smoothly varying function using a spline is straightforward, the radiogenic cancer models are inherently discontinuous. For example, the time since exposure function for most solid cancers typically has a value of zero for times since exposure that are less than the 10 y minimal latency and a value of one for times equal to or greater than the minimal latency. Suppose that the function to be integrated (the integrand) is fitted at one year increments. For the Revised Methodology models, the function will change abruptly from a value of zero for times since exposure less than 10 y to a generally smoothly varying function of time for times equal to or greater than 10 years. However fitting a spline to the integrand provides a continuous transition from the value at 9 y to the value at 10 y. If the integral is evaluated on the basis of these spline coefficients, it will include an unintended contribution from this interval.

One way to solve the problem is to integrate functions in piece-wise continuous intervals. This method is exact and would work well for the simple example considered above. In general, however, the value of the integrand at each discontinuity depends on the interval of integration; the method becomes unwieldy for situations with many discontinuities. An alternative method for situations where the function is reasonably smooth on either side of a discontinuity is to replace the value of the function at the discontinuity with the average of the values immediately above and below it. For the case above, the value of the time since exposure response function at 10 y is changed from 1 to $(0+1)/2=0.5$. The reduced excess in the integral between 9 and 10 y is then compensated for by a comparable reduction in the 10 to 11 year interval. This discontinuity smoothing method was used to calculate the risks and lifetime losses for this report.

Cancer type and dose location associations

The dose locations associated with each cancer type are shown in Table A.2. When more than one dose location is shown in the table, risks are calculated for a weighted mean of the doses at these locations using the weights shown in the table. The residual cancer category represents a composite of primary and secondary cancers that are not otherwise considered in the model. The dose location associated with these cancers, the pancreas, was chosen to be generally representative of soft tissues; the pancreas is not considered the origin of all these neoplasms.

Table A.2 Dose regions associated with cancer types in the Revised Methodology risk models.

Cancer type	Dose region	Weighting factor
Esophagus	Esophagus	1.0
Stomach	Stomach wall	1.0
Colon	Upper Large Intestine wall	0.5
	Lower Large Intestine wall	0.5
Liver	Liver	1.0
Lung	Tracheo-bronchial region	0.8
	Pulmonary region	0.2
Bone	Bone surface	1.0
Skin	Dermis	1.0
Breast	Female breast	1.0
Ovary	Ovary	1.0
Bladder	Urinary bladder wall	1.0
Kidney	Kidney	1.0
Thyroid	Thyroid	1.0
Leukemia	Red bone marrow	1.0
Residual	Pancreas	1.0

Cancer morbidity calculations

While the calculational methodology outlined above could be used with incidence models and force of morbidity data, the method used for the Revised Methodology is to divide the mortality risk coefficient by a corresponding lethality factor, k , (see Section IV.F). An exception is made for skin; only mortality is considered for calculating skin cancer morbidity, i.e., k is considered to be 1. The lifetime loss coefficient is not recalculated for morbidity.

E. Baseline Force of Mortality Calculations

Age-specific mortality rates (force of mortality) were calculated at one year intervals using U.S. death data for the period 1979-1981 (National Center for Health Statistics 1982, 1983, 1984). These calculations assume that the fraction of the recorded deaths in each age group due to a given cause, e.g., a specific ICD code, is the same as the probability of death in that age interval for a birth cohort with the corresponding age-specific death rate. In summary,

let n_i be the number of deaths due to all causes between ages x_{i-1} and x_i ,

n_{ij} be the number of deaths due to cause j between ages x_{i-1} and x_i ,

m_i be the probability in a birth cohort of dying from all causes between ages x_{i-1} and x_i ,

and m_{ij} be the probability in a birth cohort of dying from cause j between ages x_{i-1} and x_i .

Then, given the age-specific forces of mortality, $\mu(x)$ and $\mu_j(x)$, and the survival function, $S(x)$,

$$m_i = \int_{x_{i-1}}^{x_i} \mu(x) S(x) dx = S(x_{i-1}) - S(x_i) , \quad (28)$$

and

$$\begin{aligned} m_{ij} &= \int_{x_{i-1}}^{x_i} \mu_j(x) S(x) dx \\ &= \frac{n_{ij} m_i}{n_i} \\ &= \frac{n_{ij}}{n_i} [S(x_{i-1}) - S(x_i)] . \end{aligned} \quad (29)$$

(For $i=0$, x_i , n_i , n_{ij} , m_i , and m_{ij} are all equal to 0 as well.) Let $M_j(x_i)$ be the probability in a birth cohort of dying from cause j by age x_i , i.e.,

$$\begin{aligned}
M_j(x_i) &= \int_0^{x_i} \mu_j(x) S(x) dx \\
&= \sum_{k=0}^i m_{kj} .
\end{aligned}
\tag{30}$$

Differentiating the expression for $M_j(x)$ with respect to x ,

$$\frac{dM_j(x)}{dx} = \mu_j(x) S(x) .
\tag{31}$$

Solving for the force of mortality,

$$\mu_j(x) = \frac{1}{S(x)} \frac{dM_j(x)}{dx} .
\tag{32}$$

Hence, point estimates of $\mu_j(x)$ can be calculated by fitting a spline to $M_j(x)$, calculating its derivative with respect to x from the spline coefficients, and dividing the derivative by the value of the survival function at x .

The ICD-9-CM codes for malignant neoplasms used to define each cancer type in the model are listed in Table A.3. Even though baseline mortality rates are not necessary for absolute risk model calculations, these definitions serve to clarify which ICD codes are considered to comprise each cancer type.

F. Radionuclide Risk Coefficients

Age-specific radionuclide risk calculations

The age-specific cancer risk attributable to a unit intake of a radionuclide (Bq^{-1}) is calculated from the absorbed dose rate due to a unit intake of activity and the age-specific risk per unit dose coefficient. The calculation is specific for each cancer and associated absorbed dose site in the risk model. The complete calculation may involve the sum of contributions from more than one tissue (see Table A.2) and from both low- and high-LET absorbed doses. Except for leukemia, the Revised Methodology radiogenic cancer risk relative biological effectiveness (RBE) for a high-LET absorbed dose from alpha radiation is 20 times that for a low dose, low dose rate low-LET absorbed dose. For leukemia, the effective alpha dose RBE is 1, as discussed in the main text. Each risk contribution is calculated as follows:

$$r_a(x_i) = \frac{1}{S(x_i)} \int_{x_i}^{\infty} d(x) r(x) S(x) dx ,
\tag{33}$$

where $r_a(x_i)$ is the cancer risk coefficient (Bq^{-1}) for a unit intake of activity at age x_i ,

$d(x)$ is the absorbed dose rate (Gy y^{-1}) at the site at age x due to a unit intake of activity at age x_{iii} ,

$r(x)$ is the cancer risk due to a unit absorbed dose (Gy^{-1}) at the site at age x ,

and $S(x)$ is the survival function at age x .

Sex-averaged risk coefficient

Age-specific male and female risk coefficients are combined by calculating a weighted mean:

$$r_a(x_i) = \frac{1.051 r_{ma}(x_i) S_m(x_i) + r_{fa}(x_i) S_f(x_i)}{1.051 S_m(x_i) + S_f(x_i)}, \quad (34)$$

where $r_a(x)$ is the combined cancer risk coefficient (Bq^{-1}) for a unit intake of activity at age x_i ,

1.051 is the presumed sex ratio at birth (male:female),

$r_{ma}(x)$ is the male risk per unit activity at age x_i ,

$r_{fa}(x)$ is the female risk per unit activity at age x_i ,

$S_m(x_i)$ is the male survival function at age x_i ,

and $S_f(x_i)$ is the female survival function at age x_i .

This formulation weights each sex-specific risk coefficient by the proportion of that sex in a stationary combined population at the desired age of intake.

Average lifetime risk coefficient

The average lifetime risk coefficient (Bq^{-1}) for a unit intake of a radionuclide is calculated from the age-specific value, $r_a(x)$, by the equation:

$$\bar{r}_a = \frac{\int_0^{\infty} r_a(x) S(x) dx}{\bar{\varrho}}, \quad (35)$$

where \bar{r}_a (Bq^{-1}) is the average lifetime risk per unit intake of activity

and $\bar{\varrho}$ is the expected lifetime at age 0.

Table A.3 ICD codes used to define cancer types.

Cancer type	ICD-9-CM codes
Esophagus	150.0-150.9
Stomach	151.0-151.9
Colon	153.0-153.9
Liver	155.0-155.2
Lung	162.0-162.9
Bone	170.0-170.9
Skin	173.0-173.9 except 172.0-172.9 (melanoma)
Breast	174.0-174.9 (female) and 175.0-175.9 (male)
Ovary	183.0
Bladder	188.0-188.9
Kidney	189.0-189.1
Thyroid	193.0-193.9
Leukemia	204.0-208.9 except 204.1 (chronic lymphoid)
EPA residual	140-208 except 181 (placenta), and all codes included in the above definitions.

Equation (35) also provides the lifetime risk per unit intake for a lifetime intake at a constant intake rate. For a stationary population, the expected incidence or mortality per unit activity intake (cases per Bq or deaths per Bq) is also given by equation (35).

The sex-weighted average is given by

$$\bar{r}_a = \frac{1.051 \bar{r}_{ma} \bar{e}_m + \bar{r}_{fa} \bar{e}_f}{1.051 \bar{e}_m + \bar{e}_f} . \quad (36)$$

Radionuclide risk coefficients for external exposures

Lifetime risks for external radionuclide exposures are calculated in a similar manner to those for radionuclide intakes. Since the organ and tissue doses occur at the same time as the exposure and are not considered to be age dependent, the calculations are simpler. Given the age specific cancer risk per unit dose, $r(x)$, from equation (15) or (16) and the corresponding

dose per unit exposure coefficient, d_e (e.g., Gy per Bq y/m² to the thyroid from ground surface exposure to ⁶⁰Co), the lifetime risk is simply

$$r_e(x) = d_e r(x) \quad (37)$$

for an exposure at age x . The average lifetime risk, \bar{r}_e is just

$$\bar{r}_e = d_e \bar{r} . \quad (38)$$

Equation (38) can also be used in the same manner as equation (35) to calculate lifetime risk from lifetime exposure at a constant exposure rate or population risk from an external exposure.

Radionuclide Risk Coefficient Tables

Average lifetime mortality and morbidity (incidence) risk coefficients for the 321 radionuclides in Tables A.4a and A.4b, respectively, were calculated with CRDARTAB (Sjoreen 1994) using RADRISK dose rates and Revised Methodology risk coefficients. The coefficients correspond to the sums of risks for all cancers using equations (35) or (38) for intakes and external exposures, respectively. The default clearance class for inhalation and the digestive tract to blood transfer factor, f_1 , for each element (or radionuclide when necessary) are shown in Table A.5. These clearance class and f_1 defaults were chosen conservatively, i.e., among the standard values that might be appropriate for environmental exposures, they yield the greatest risks.

Table A.4a Revised Methodology radionuclide mortality risk coefficients. (cont.)

Nuclide	Internal			External		
	Ingestion (Bq ⁻¹)	Inhalation (Bq ⁻¹)	Submersion Gnd Surface (per Bq y/m ²)	Ingestion (Bq ⁻¹)	Inhalation (Bq ⁻¹)	Submersion Gnd Surface (per Bq y/m ²)
Nb-97	3.34E-12	5.38E-12	3.92E-08	7.82E-10	1.23E-11	4.38E-13
Nb-97m	6.41E-14	8.41E-14	4.36E-08	8.54E-10	1.88E-08	3.39E-10
Mo-99	3.88E-11	8.60E-11	9.15E-09	1.82E-10	2.36E-09	5.89E-11
Tc-95	1.22E-12	7.59E-13	4.72E-08	9.21E-10	2.57E-08	5.21E-10
Tc-95m	2.22E-11	4.38E-11	3.91E-08	7.85E-10	1.16E-07	2.05E-09
Tc-96	3.95E-11	3.86E-11	1.51E-07	2.90E-09	2.43E-08	5.01E-10
Tc-96m	4.66E-13	4.83E-13	2.50E-09	4.90E-11	1.63E-07	3.24E-09
Tc-97	2.83E-12	8.02E-12	3.05E-11	4.13E-12	9.22E-12	1.85E-09
Tc-97m	2.10E-11	4.49E-11	4.33E-11	3.71E-12	3.88E-08	7.78E-10
Tc-99	2.49E-11	6.75E-11	2.63E-14	6.22E-16	8.69E-08	1.63E-09
Tc-99m	8.87E-13	7.66E-13	7.02E-09	1.56E-10	3.42E-10	1.48E-11
Ru-97	9.18E-12	7.83E-12	1.28E-08	2.82E-10	2.74E-10	5.79E-12
Ru-103	5.09E-11	9.66E-11	2.76E-08	5.70E-10	1.12E-10	4.74E-12
Ru-105	1.83E-11	1.82E-11	4.62E-08	9.24E-10	3.08E-09	6.38E-11
Ru-106	5.28E-10	2.75E-09			1.94E-09	4.02E-11
Rh-103m	1.66E-13	3.26E-13	6.59E-12	7.46E-13	2.44E-08	4.91E-10
Rh-105	2.91E-11	2.38E-11	4.37E-09	9.40E-11	8.59E-08	1.63E-09
Rh-105m	1.98E-14	2.11E-14	1.51E-09	3.66E-11	1.19E-08	2.66E-10
Rh-106	8.78E-14	1.18E-13	1.21E-08	2.41E-10	5.59E-08	1.14E-09
Pd-100	5.76E-11	6.71E-11			8.58E-09	1.94E-10
Pd-101	5.89E-12	4.82E-12	6.03E-11	6.62E-12	3.86E-10	1.77E-11
Pd-103	1.58E-11	2.23E-11			2.65E-08	5.38E-10
Pd-107	3.14E-12	3.61E-11	3.94E-11	8.11E-13	1.26E-07	2.50E-09
Pd-109	5.05E-11	4.17E-11			2.98E-10	1.56E-11
Ag-105	2.67E-11	4.75E-11			1.26E-07	4.55E-10
Ag-108	1.03E-10	1.49E-09	1.02E-09	2.07E-11	2.16E-08	4.55E-10
Ag-108m	1.69E-13	2.42E-13	9.41E-08	1.90E-09	1.37E-07	2.64E-09
Ag-109m	6.55E-15	8.87E-15	2.04E-10	7.24E-12	3.52E-08	7.06E-10
Ag-110	5.94E-14	8.10E-14	1.80E-09	3.59E-11	1.60E-07	2.99E-09
Ag-110m	1.43E-10	6.78E-10	1.65E-07	3.11E-09	9.74E-08	1.69E-09
Ag-111	1.03E-10	1.01E-10	1.48E-09	3.19E-11	5.60E-14	7.54E-11
Cd-109	1.38E-10	4.29E-10	7.25E-11	6.22E-12	1.39E-14	7.08E-10
Cd-115	1.10E-10	9.45E-11	1.17E-08	2.40E-10	1.93E-14	1.38E-08
Cd-115m	2.19E-10	3.64E-10	1.33E-09	2.44E-11	7.37E-15	1.44E-08
In-113m	1.50E-12	1.43E-12	1.44E-08	3.07E-10	1.09E-14	1.01E-09
In-114	1.10E-13	1.49E-13	1.85E-09	3.71E-11	3.65E-10	1.42E-11
In-114m	3.18E-10	5.39E-10	5.15E-09	1.08E-10	7.87E-15	1.64E-09
In-115	8.14E-10	5.00E-09			9.81E-15	1.54E-09
In-115m	5.46E-12	4.09E-12	8.92E-09	1.93E-10	1.44E-14	1.39E-08
Sn-113	5.66E-11	1.46E-10	3.92E-10	1.26E-11	3.81E-15	2.45E-08
Sn-121	1.84E-11	1.20E-11			3.17E-14	1.10E-08
					4.21E-14	7.33E-08
						1.23E-09

Table A.4a Revised Methodology radionuclide mortality risk coefficients. (cont.)

Nuclide	Internal			External		
	Ingestion (Bq ⁻¹)	Inhalation (Bq ⁻¹)	Submersion Gnd Surface (per Bq y/m ²)	Ingestion (Bq ⁻¹)	Inhalation (Bq ⁻¹)	Submersion Gnd Surface (per Bq y/m ²)
Cs-131	3.28E-12	1.91E-12	2.51E-10	1.39E-11	9.22E-08	1.82E-09
Cs-134	8.49E-10	5.01E-10	9.22E-08	1.82E-09	2.03E-08	4.46E-10
Cs-134m	9.17E-13	7.09E-13	1.04E-09	2.71E-11	2.18E-10	5.82E-12
Cs-135	8.27E-11	4.78E-11			2.33E-11	6.94E-13
Cs-136	1.38E-10	8.10E-11	1.30E-07	2.47E-09	1.45E-08	3.55E-10
Cs-137	5.71E-10	3.34E-10			2.18E-09	4.71E-11
Cs-138	3.96E-12	3.21E-12	1.49E-07	2.52E-09	1.87E-09	4.21E-11
Ba-131	2.64E-11	8.35E-12	2.56E-08	5.46E-10	3.05E-08	6.45E-10
Ba-133	4.53E-11	7.90E-11	2.02E-08	4.52E-10	7.70E-08	1.41E-09
Ba-133m	4.17E-11	9.49E-12	3.03E-09	7.19E-11	1.47E-09	4.17E-11
Ba-137m	5.35E-14	3.90E-14	3.52E-08	7.01E-10	1.46E-12	3.26E-14
Ba-139	5.72E-12	3.59E-12	1.96E-09	4.19E-11	2.74E-08	5.59E-10
Ba-140	1.78E-10	5.49E-11	1.07E-08	2.23E-10	7.12E-09	1.76E-10
La-140	1.44E-10	9.75E-11	1.44E-07	2.49E-09	1.04E-09	2.44E-11
Ce-141	5.86E-11	8.99E-11	4.07E-09	9.21E-11		
Ce-143	8.91E-11	7.48E-11	1.46E-08	3.14E-10	3.31E-09	6.85E-11
Ce-144	4.42E-10	2.60E-09	9.32E-10	2.18E-11	4.08E-08	8.25E-10
Pr-142	1.05E-10	8.44E-11	3.64E-09	6.02E-11	3.44E-09	8.36E-11
Pr-143	9.88E-11	1.10E-10	5.33E-16	1.04E-17	1.89E-10	5.45E-12
Pr-144	1.91E-12	3.37E-12	2.10E-09	3.49E-11	3.65E-09	7.95E-11
Pr-144m	7.46E-13	1.44E-12	2.01E-10	7.63E-12	7.98E-08	1.68E-09
Nd-147	8.82E-11	9.47E-11	7.23E-09	1.59E-10	4.64E-08	9.80E-10
Nd-149	7.60E-12	9.89E-12	2.12E-08	4.53E-10	5.28E-09	1.07E-10
Pm-147	2.12E-11	1.84E-10	1.91E-13	4.27E-15	1.51E-08	3.37E-10
Pm-148	2.17E-10	2.00E-10	3.46E-08	6.24E-10	1.70E-12	4.31E-13
Pm-148m	1.52E-10	6.26E-10	1.17E-07	2.33E-09	4.56E-10	1.22E-11
Pm-149	8.27E-11	6.83E-11	6.59E-10	1.40E-11	1.14E-09	2.74E-11
Sm-147	5.12E-10	1.69E-07			4.06E-09	9.42E-11
Sm-151	6.99E-12	1.15E-10	3.02E-14	3.48E-15	2.60E-08	5.65E-10
Sm-153	6.04E-11	4.17E-11	2.45E-09	6.57E-11	2.29E-08	4.83E-10
Eu-152	9.17E-11	1.69E-09	6.79E-08	1.28E-09	2.99E-09	7.60E-11
Eu-154	1.47E-10	2.01E-09	7.51E-08	1.39E-09	1.27E-08	2.77E-10
Eu-155	2.94E-11	2.23E-10	2.81E-09	6.94E-11	2.58E-08	5.51E-10
Eu-156	1.65E-10	1.83E-10	8.59E-08	1.46E-09	4.84E-11	1.23E-12
Gd-153	2.01E-11	7.03E-11	4.18E-09	1.15E-10	1.32E-10	2.91E-12
Gd-159	3.93E-11	2.48E-11	2.04E-09	4.62E-11	2.33E-07	3.56E-09
Tb-158	6.54E-11	1.53E-09	4.55E-08	8.62E-10	1.28E-07	2.27E-09
Tb-160	1.16E-10	2.37E-10	6.48E-08	1.22E-09	1.62E-08	3.61E-10
Dy-165	5.54E-12	5.54E-12	1.34E-09	2.93E-11		
Dy-166	1.40E-10	1.49E-10	1.48E-09	4.04E-11	5.49E-11	2.41E-12
Ho-166	1.14E-10	7.92E-11	1.54E-09	3.04E-11	3.00E-09	5.99E-11

Table A.4a Revised Methodology radionuclide mortality risk coefficients. (cont.)

Nuclide	Internal		External	
	Ingestion (Bq ⁻¹)	Inhalation (Bq ⁻¹)	Submersion Gnd Surface (per Bq y/m ³)	Submersion Gnd Surface (per Bq y/m ²)
Pb-212	2.99E-10	8.68E-10	7.98E-09	1.77E-10
Pb-214	6.02E-12	1.56E-10	1.39E-08	2.98E-10
Bi-206	1.11E-10	9.61E-11	1.98E-07	3.72E-09
Bi-207	7.82E-11	1.95E-10	9.16E-08	1.74E-09
Bi-210	1.10E-10	1.28E-09		
Bi-211	4.42E-13	4.48E-11	2.66E-09	5.69E-11
Bi-212	1.22E-11	9.35E-10	1.11E-08	2.06E-10
Bi-213	8.90E-12	7.92E-10	7.90E-09	1.65E-10
Bi-214	4.45E-12	3.74E-10	9.48E-08	1.64E-09
Po-210	5.88E-09	5.14E-08	5.13E-13	9.95E-15
Po-212	1.10E-21	1.52E-19		
Po-213	1.57E-20	2.00E-18	1.83E-12	3.57E-14
Po-214	5.16E-19	7.12E-17	5.02E-12	9.74E-14
Po-215	1.13E-17	1.15E-15	8.46E-12	1.77E-13
Po-216	1.59E-15	7.56E-14	8.75E-13	1.69E-14
Po-218	1.10E-12	9.44E-11		
At-217	1.98E-16	1.32E-14	1.39E-11	2.80E-13
Rn-219		1.74E-12	3.22E-09	6.91E-11
Rn-220		4.42E-12	3.01E-11	6.16E-13
Rn-222		3.21E-11	2.22E-11	4.59E-13
Fr-221	3.04E-12	2.05E-10	1.70E-09	3.75E-11
Fr-223	7.91E-12	1.17E-11	2.34E-09	5.88E-11
Ra-223	3.91E-09	9.12E-08	7.18E-09	1.61E-10
Ra-224	2.46E-09	5.70E-08	5.57E-10	1.21E-11
Ra-225	2.88E-09	6.01E-08	2.63E-10	1.16E-11
Ra-226	5.26E-09	6.62E-08	3.71E-10	8.21E-12
Ra-228	4.48E-09	2.22E-08	2.48E-18	5.19E-19
Ac-225	2.19E-09	1.06E-07	7.03E-10	1.68E-11
Ac-227	8.17E-09	1.78E-06	6.41E-12	1.95E-13
Ac-228	2.55E-11	8.33E-10	5.58E-08	1.05E-09
Th-227	6.11E-10	1.10E-07	5.72E-09	1.28E-10
Th-228	9.84E-10	2.41E-06	1.01E-10	2.69E-12
Th-229	9.94E-10	1.91E-06	4.42E-09	1.05E-10
Th-230	6.39E-10	4.22E-07	1.88E-11	7.72E-13
Th-231	2.70E-11	2.18E-11	5.60E-10	1.71E-11
Th-232	5.61E-10	4.73E-07	8.51E-12	5.31E-13
Th-234	2.88E-10	3.89E-10	3.78E-10	9.65E-12
Pa-231	2.86E-09	5.85E-07	1.65E-09	3.81E-11
Pa-233	7.05E-11	1.01E-10	1.17E-08	2.58E-10
Pa-234	3.37E-11	2.91E-11	1.17E-07	2.25E-09
Pa-234m				
U-232	1.40E-09	1.35E-06	1.23E-11	8.11E-13
U-233	7.44E-10	3.63E-07	1.21E-11	4.38E-13
U-234	7.38E-10	3.58E-07	6.72E-12	6.12E-13
U-235	7.53E-10	3.33E-07	8.29E-09	1.84E-10
U-236	6.99E-10	3.39E-07	5.12E-12	5.47E-13
U-237	6.01E-11	5.99E-11	6.96E-09	1.65E-10
U-238	7.31E-10	3.19E-07	4.36E-12	4.81E-13
U-240	8.29E-11	6.98E-11	2.79E-11	2.90E-12
Np-236	1.43E-11	8.96E-11	6.80E-09	1.61E-10
Np-237	6.65E-09	8.04E-07	1.13E-09	3.05E-11
Np-238	6.92E-11	9.80E-11	3.36E-08	6.24E-10
Np-239	6.42E-11	4.61E-11	8.96E-09	2.03E-10
Np-240	3.42E-12	3.26E-12	6.81E-08	1.34E-09
Np-240m	5.76E-13	7.24E-13	1.94E-08	3.82E-10
Pu-236	1.56E-09	3.39E-07	4.66E-12	7.14E-13
Pu-238	6.64E-09	6.76E-07	3.32E-12	6.11E-13
Pu-239	7.12E-09	6.82E-07	3.83E-12	2.89E-13
Pu-240	7.11E-09	6.81E-07	3.26E-12	5.86E-13
Pu-241	1.19E-10	6.55E-09		
Pu-242	6.76E-09	6.47E-07	2.78E-12	4.86E-13
Pu-243	5.80E-12	6.29E-12	1.16E-09	2.83E-11
Pu-244	6.95E-09	6.53E-07	1.94E-12	4.11E-13
Am-241	7.39E-09	8.96E-07	8.46E-10	2.61E-11
Am-242	2.25E-11	2.52E-10	7.03E-10	1.78E-11
Am-242m	6.74E-09	8.08E-07	2.01E-11	2.00E-12
Am-243	7.36E-09	8.89E-07	2.43E-09	6.21E-11
Am-244	6.60E-10	7.81E-08	3.62E-12	6.51E-13
Am-245	5.58E-09	6.75E-07	6.82E-09	1.55E-10
Am-246	4.64E-09	5.70E-07	3.04E-12	5.80E-13
Am-247	7.54E-09	9.12E-07	3.69E-09	8.68E-11
Am-248	7.48E-09	9.08E-07	2.49E-12	5.10E-13
Am-249	6.93E-09	8.34E-07	1.79E-08	3.80E-10
Am-250	2.90E-08	3.39E-06	2.26E-12	4.11E-13
Am-251	3.50E-09	6.59E-07	2.79E-12	4.37E-13

Table A.4b Revised Methodology radionuclide incidence risk coefficients.

Nuclide	Internal			External		
	Ingestion	Inhalation	Submersion Gnd Surface	Submersion Gnd Surface	Inhalation	Submersion Gnd Surface
	(Bq ⁻¹)	(Bq ⁻¹)	(per Bq y/m ²)	(per Bq y/m ²)	(Bq ⁻¹)	(per Bq y/m ²)
H-3	1.93E-12	2.59E-12			4.11E-11	3.59E-08
Be-7	2.33E-12	4.81E-12	4.3E-09	8.94E-11	2.26E-11	1.23E-08
C-11	1.21E-12	9.14E-13	8.87E-08	1.83E-09	1.29E-10	5.87E-11
C-14	2.79E-11	1.89E-13			3.19E-13	1.58E-12
C-15	1.79E-14	2.18E-14	4.05E-07	4.69E-09	2.22E-11	3.69E-11
N-13	8.25E-13	6.84E-13	8.87E-08	1.83E-09	1.14E-10	1.25E-10
O-15	2.57E-13	2.60E-13	8.87E-08	1.83E-09	1.77E-10	1.17E-10
F-18	2.94E-12	1.77E-12	8.59E-08	1.77E-09	4.46E-11	3.10E-11
Na-22	2.17E-10	1.32E-10	1.96E-07	3.68E-09	1.77E-10	1.33E-10
Na-24	3.74E-11	2.03E-11	4.28E-07	6.36E-09	1.77E-10	1.33E-10
Si-31	1.36E-11	8.90E-12	8.13E-11	1.42E-12	3.83E-11	2.12E-11
P-32	1.65E-10	7.92E-11			9.40E-16	6.48E-12
P-33	2.11E-11	1.07E-11			7.75E-15	1.94E-10
S-35	1.12E-11	5.01E-12			7.42E-15	1.36E-08
Cl-36	6.04E-11	3.51E-11	6.32E-16	6.95E-17	3.26E-14	7.93E-08
Cl-38	5.58E-12	4.41E-12	1.54E-07	2.37E-09	5.96E-14	2.03E-07
Ar-41	1.27E-14		1.17E-07	2.05E-09	4.34E-14	1.81E-07
K-40	3.39E-10	2.02E-10	1.44E-08	2.43E-10	4.38E-14	1.19E-07
K-42	3.48E-11	2.04E-11	2.57E-08	4.30E-10	2.84E-13	3.17E-13
Ca-45	5.46E-11	6.79E-11	1.13E-18	2.98E-19	1.92E-10	1.14E-10
Ca-47	1.80E-10	1.41E-10	9.74E-08	1.71E-09	9.96E-11	6.12E-11
Sc-46	1.55E-10	3.53E-10	1.85E-07	3.43E-09	3.96E-12	3.69E-12
Sc-47	7.97E-11	5.44E-11	9.42E-09	2.08E-10	2.34E-12	1.87E-12
Sc-48	1.80E-10	1.13E-10	3.10E-07	5.58E-09	6.97E-10	2.40E-10
V-48	2.04E-10	1.85E-10	2.67E-07	4.88E-09	3.78E-11	3.08E-11
Cr-51	3.74E-12	4.70E-12	2.71E-09	5.83E-11	4.87E-13	1.93E-13
Mn-52	1.62E-10	1.19E-10	3.16E-07	5.79E-09	2.79E-10	9.94E-11
Mn-54	5.30E-11	9.98E-11	7.68E-08	1.48E-09	1.10E-09	1.61E-09
Mn-56	2.32E-11	1.41E-11	1.65E-07	2.85E-09	7.62E-11	2.11E-11
Fe-55	9.50E-12	1.51E-11	1.71E-12	3.30E-13	5.48E-11	1.27E-11
Fe-59	1.59E-10	1.91E-10	1.09E-07	1.94E-09	4.05E-10	2.68E-10
Co-57	2.62E-11	7.78E-11	1.03E-08	2.33E-10	3.64E-10	5.01E-10
Co-58	7.62E-11	1.40E-10	8.88E-08	1.73E-09	9.98E-13	8.09E-13
Co-58m	2.56E-12	2.41E-12	2.55E-12	5.31E-13	5.26E-11	4.36E-11
Co-60	5.11E-10	1.86E-09	2.30E-07	4.03E-09	1.55E-10	9.40E-11
Ni-59	5.00E-12	1.08E-11	2.85E-12	6.21E-13	1.41E-11	1.42E-10
Ni-63	1.49E-11	2.74E-11			1.06E-10	1.75E-10
Ni-65	1.52E-11	9.71E-12	5.07E-08	8.78E-10	2.80E-10	1.28E-10
Cu-64	1.42E-11	1.13E-11	1.65E-08	3.38E-10	1.79E-11	1.17E-10
Zn-65	2.68E-10	2.70E-10	5.34E-08	9.68E-10	1.87E-10	2.22E-09
Zn-69	1.67E-12	2.82E-12	5.18E-13	1.09E-14	6.09E-11	8.40E-11
					8.27E-11	6.08E-11

Table A.4b Revised Methodology radionuclide incidence risk coefficients. (cont.)

Nuclide	Internal		External	
	Ingestion (Bq ⁻¹)	Inhalation (Bq ⁻¹)	Submersion (per Bq y/m ³)	Gnd Surface (per Bq y/m ²)
Nb-97	4.74E-12	5.75E-12	5.96E-08	1.19E-09
Nb-97m	8.85E-14	9.03E-14	6.62E-08	1.30E-09
Mo-99	6.13E-11	1.21E-10	1.39E-08	2.78E-10
Tc-95	1.84E-12	9.13E-13	7.17E-08	1.40E-09
Tc-95m	3.36E-11	5.67E-11	5.95E-08	1.20E-09
Tc-96	6.17E-11	5.25E-11	2.29E-07	4.41E-09
Tc-96m	7.05E-13	6.11E-13	3.81E-09	7.53E-11
Tc-97	4.27E-12	9.31E-12	5.69E-11	7.72E-12
Tc-97m	3.24E-11	5.30E-11	7.56E-11	6.78E-12
Tc-99	3.79E-11	7.81E-11	4.14E-14	9.78E-16
Tc-99m	1.51E-12	9.43E-13	1.09E-08	2.42E-10
Ru-97	1.59E-11	1.10E-11	1.96E-08	4.34E-10
Ru-103	8.98E-11	1.24E-10	4.20E-08	8.67E-10
Ru-105	3.12E-11	2.17E-11	7.02E-08	1.40E-09
Ru-106	9.32E-10	3.11E-09		
Rh-103m	2.21E-13	3.45E-13	1.20E-11	1.37E-12
Rh-105	5.22E-11	3.30E-11	6.66E-09	1.43E-10
Rh-105m	2.92E-14	2.50E-14	2.34E-09	5.79E-11
Rh-106	9.80E-14	1.25E-13	1.84E-08	3.66E-10
Pd-100	1.01E-10	9.60E-11		
Pd-101	1.01E-11	6.18E-12		
Pd-103	2.85E-11	2.92E-11	1.09E-10	1.21E-11
Pd-107	5.66E-12	3.94E-11		
Pd-109	9.00E-11	5.37E-11	5.98E-11	1.23E-12
Ag-105	4.41E-11	6.28E-11		
Ag-108	1.88E-13	2.55E-13	1.55E-09	3.14E-11
Ag-108m	1.64E-10	1.90E-09	1.43E-07	2.89E-09
Ag-109m	7.32E-15	9.34E-15	3.31E-10	1.22E-11
Ag-110	6.60E-14	8.53E-14	2.74E-09	5.45E-11
Ag-110m	2.28E-10	8.69E-10	2.50E-07	4.72E-09
Ag-111	1.85E-10	1.42E-10	2.26E-09	4.86E-11
Cd-109	2.16E-10	5.00E-10	1.31E-10	1.13E-11
Cd-115	1.97E-10	1.33E-10	1.77E-08	3.65E-10
Cd-115m	3.84E-10	4.60E-10	2.02E-09	3.71E-11
In-113m	2.24E-12	1.56E-12	2.20E-08	4.68E-10
In-114	1.23E-13	1.57E-13	2.81E-09	5.64E-11
In-114m	5.56E-10	6.83E-10	7.87E-09	1.66E-10
In-115m	9.43E-10	5.60E-09		
In-115	9.24E-12	4.74E-12	1.36E-08	2.96E-10
Sn-113	1.01E-10	1.79E-10	6.29E-10	2.10E-11
Sn-121	3.31E-11	1.66E-11		
Sn-125	5.40E-11	2.02E-10	2.08E-11	7.39E-13
Sn-125m	4.53E-10	3.23E-10	2.85E-08	5.14E-10
Sn-126	5.73E-10	1.15E-09	3.74E-09	9.36E-11
Sb-122	2.38E-10	1.47E-10	3.91E-08	7.91E-10
Sb-124	2.91E-10	3.57E-10	1.76E-07	3.12E-09
Sb-125	8.02E-11	1.41E-10	3.69E-08	7.64E-10
Sb-126	2.63E-10	2.27E-10	2.47E-07	4.92E-09
Sb-126m	1.97E-12	1.74E-12	1.40E-07	2.82E-09
Sb-127	2.29E-10	1.63E-10	5.89E-08	1.18E-09
Sb-129	5.02E-11	2.32E-11	1.32E-07	2.47E-09
Te-125m	6.78E-11	7.70E-11	5.94E-10	2.57E-11
Te-127	2.31E-11	1.17E-11	4.17E-10	8.81E-12
Te-127m	1.62E-10	3.53E-10	1.94E-10	8.24E-12
Te-129	4.00E-12	3.96E-12	4.70E-09	9.74E-11
Te-129m	3.18E-10	3.60E-10	2.97E-09	6.18E-11
Te-131	1.05E-11	6.71E-12	3.72E-08	7.49E-10
Te-131m	2.38E-10	2.27E-10	1.31E-07	2.48E-09
Te-132	3.29E-10	2.26E-10	1.83E-08	4.10E-10
I-122	5.84E-13	6.05E-13	8.49E-08	1.73E-09
I-123	1.47E-11	7.94E-12	1.33E-08	3.00E-10
I-125	6.98E-10	4.62E-10	6.63E-10	2.94E-11
I-126	1.30E-09	8.52E-10	3.97E-08	8.06E-10
I-129	4.98E-09	3.30E-09	5.03E-10	2.64E-11
I-130	1.31E-10	7.06E-11	1.91E-07	3.80E-09
I-131	9.77E-10	6.31E-10	3.24E-08	6.82E-10
I-132	1.79E-11	9.51E-12	2.09E-07	4.02E-09
I-133	2.86E-10	1.63E-10	5.35E-08	1.07E-09
I-134	6.25E-12	3.74E-12	2.43E-07	4.54E-09
I-135	6.14E-11	3.19E-11	1.48E-07	2.56E-09
Xe-122	8.34E-14	4.99E-14	4.99E-09	1.18E-10
Xe-123	2.41E-14	5.53E-08	1.08E-09	
Xe-125	3.25E-14	2.12E-08	4.65E-10	
Xe-127	1.10E-14	2.22E-08	4.94E-10	
Xe-129m	1.55E-14	1.65E-09	6.31E-11	
Xe-131m	1.12E-14	5.99E-10	2.39E-11	
Xe-133	1.12E-14	2.62E-09	7.38E-11	
Xe-133m	1.38E-14	2.39E-09	6.32E-11	
Xe-135	2.01E-14	2.12E-08	4.59E-10	
Xe-135m	5.09E-15	3.72E-08	7.68E-10	
Xe-137	3.76E-14	1.67E-08	3.29E-10	
Xe-138	5.58E-14	1.11E-07	1.87E-09	

Table A.4b Revised Methodology radionuclide incidence risk coefficients. (cont.)

Nuclide	Internal			External		
	Ingestion (Bq ⁻¹)	Inhalation (Bq ⁻¹)	Submersion Gnd Surface (per Bq y/m ²)	Ingestion (Bq ⁻¹)	Inhalation (Bq ⁻¹)	Submersion Gnd Surface (per Bq y/m ²)
Cs-131	4.87E-12	2.87E-12	4.35E-10	5.73E-11	4.08E-11	1.52E-13
Cs-134	1.28E-09	7.80E-10	1.40E-07	4.4E-11	2.03E-11	3.12E-08
Cs-134m	1.23E-12	8.39E-13	1.63E-09	2.03E-10	2.96E-10	3.47E-10
Cs-135	1.22E-10	7.33E-11		1.58E-11	4.97E-11	3.75E-11
Cs-136	2.09E-10	1.26E-10	1.98E-07	9.27E-11	1.08E-10	2.27E-08
Cs-137	8.54E-10	5.18E-10		6.49E-11	4.54E-11	3.33E-09
Cs-138	4.75E-12	3.51E-12	2.26E-07	7.97E-11	5.94E-11	2.89E-09
Ba-131	4.59E-11	1.30E-11	3.91E-08	1.48E-10	1.67E-10	4.65E-08
Ba-133	7.29E-11	1.09E-10	3.09E-08	1.90E-10	4.47E-10	1.17E-07
Ba-133m	6.58E-14	4.24E-14	5.34E-08	7.36E-12	2.17E-12	2.37E-09
Ba-139	8.23E-12	4.13E-12	3.02E-09	5.50E-11	1.15E-11	2.27E-12
Ba-140	3.18E-10	8.57E-11	1.62E-08	4.26E-11	6.94E-11	4.17E-08
La-140	2.56E-10	1.38E-10	2.18E-07	8.12E-11	7.06E-11	1.12E-08
Ce-141	1.06E-10	1.17E-10	6.31E-09	2.47E-13	5.10E-13	1.63E-09
Ce-143	1.60E-10	1.04E-10	2.24E-08	8.63E-11	6.25E-11	5.07E-09
Ce-144	7.99E-10	2.91E-09	1.46E-09	4.87E-11	1.25E-10	6.21E-08
Pr-142	1.89E-10	1.12E-10	5.52E-09	8.22E-11	7.30E-11	5.41E-09
Pr-143	1.78E-10	1.51E-10	8.09E-16	1.34E-11	8.97E-12	3.03E-10
Pr-144	2.18E-12	3.55E-12	3.18E-09	1.18E-10	7.24E-11	5.59E-09
Pr-144m	8.72E-13	1.52E-12	3.39E-10	1.34E-11	7.24E-11	1.22E-07
Nd-147	1.59E-10	1.31E-10	1.11E-08	1.74E-10	3.04E-10	7.06E-08
Nd-149	1.23E-11	1.14E-11	3.24E-08	1.89E-10	1.13E-10	8.04E-09
Pm-147	3.82E-11	2.02E-10	2.96E-13	4.05E-11	1.12E-11	2.32E-08
Pm-148	3.90E-10	2.83E-10	5.25E-08	4.38E-12	2.13E-12	3.28E-12
Pm-148m	2.68E-10	7.96E-10	1.78E-07	6.77E-11	1.56E-11	7.26E-10
Pm-149	1.49E-10	9.64E-11	1.01E-09	5.74E-11	1.23E-11	1.79E-09
Sm-147	6.78E-10	1.87E-07		8.79E-12	2.72E-12	6.28E-09
Sm-151	1.24E-11	1.25E-10	5.54E-14	3.52E-11	2.81E-11	3.97E-08
Sm-153	1.09E-10	5.88E-11	3.88E-09	1.43E-10	9.83E-11	3.49E-08
Eu-152	1.55E-10	2.14E-09	1.03E-07	3.20E-11	1.88E-11	4.75E-09
Eu-154	2.53E-10	2.47E-09	1.14E-07	7.15E-11	8.18E-11	1.94E-08
Eu-155	4.46E-11	2.59E-10	4.43E-09	2.74E-11	1.64E-11	3.94E-08
Eu-156	2.95E-10	2.50E-10	1.30E-07	5.33E-11	3.12E-11	7.68E-11
Gd-153	3.56E-11	8.66E-11	6.67E-09	2.90E-13	3.34E-13	2.01E-10
Gd-159	7.03E-11	3.34E-11	3.14E-09	4.73E-13	3.68E-13	3.54E-07
Tb-158	1.14E-10	1.90E-09	6.92E-08	3.80E-13	3.04E-13	1.95E-07
Tb-160	2.06E-10	3.08E-10	9.85E-08	2.79E-11	8.38E-12	2.50E-08
Dy-165	8.80E-12	6.06E-12	2.06E-09	5.66E-12	1.85E-12	1.85E-12
Dy-166	2.55E-10	2.11E-10	2.36E-09	1.82E-08	4.51E-08	9.09E-11
Ho-166	2.05E-10	1.10E-10	2.38E-09	9.13E-12	2.79E-10	4.56E-09

Table A.4b Revised Methodology radionuclide incidence risk coefficients. (cont.)

Nuclide	Internal		External	
	Ingestion (Bq ⁻¹)	Inhalation (Bq ⁻¹)	Submersion (per Bq y/m ³)	Gnd Surface (per Bq y/m ²)
Pb-212	4.87E-10	1.04E-09	1.23E-08	2.74E-10
Pb-214	7.94E-12	1.68E-10	2.12E-08	4.56E-10
Bi-206	1.92E-10	1.37E-10	3.01E-07	5.66E-09
Bi-207	1.37E-10	2.54E-10	1.39E-07	2.65E-09
Bi-210	1.97E-10	1.38E-09		
Bi-211	4.93E-13	4.71E-11	4.05E-09	8.69E-11
Bi-212	1.68E-11	9.88E-10	1.69E-08	3.13E-10
Bi-213	1.19E-11	8.36E-10	1.20E-08	2.51E-10
Bi-214	5.27E-12	3.94E-10	1.44E-07	2.49E-09
Po-210	8.80E-09	5.79E-08	7.79E-13	1.51E-14
Po-212	1.22E-21	1.60E-19		
Po-213	1.81E-20	2.11E-18	2.79E-12	5.42E-14
Po-214	5.74E-19	7.50E-17	7.63E-12	1.48E-13
Po-215	1.35E-17	1.21E-15	1.29E-11	2.70E-13
Po-216	2.38E-15	7.98E-14	1.33E-12	2.57E-14
Po-218	1.37E-12	9.96E-11		
At-217	2.43E-16	1.39E-14	2.11E-11	4.26E-13
Rn-219	1.87E-12	4.91E-09	4.91E-09	1.05E-10
Rn-220	5.18E-12	4.58E-11	4.58E-11	9.35E-13
Rn-222	4.96E-11	3.38E-11	3.38E-11	6.97E-13
Fr-223	3.91E-12	2.17E-10	2.61E-09	5.76E-11
Fr-225	1.21E-11	1.60E-11	3.66E-09	9.23E-11
Ra-223	6.33E-09	9.74E-08	1.11E-08	2.50E-10
Ra-224	4.04E-09	6.08E-08	8.54E-10	1.86E-11
Ra-225	4.25E-09	6.44E-08	4.40E-10	1.96E-11
Ra-226	7.98E-09	7.35E-08	5.72E-10	1.27E-11
Ra-228	6.64E-09	2.60E-08	4.77E-18	9.99E-19
Ac-225	3.85E-09	1.12E-07	1.10E-09	2.65E-11
Ac-227	9.52E-09	1.91E-06	1.01E-11	3.24E-13
Ac-228	4.38E-11	8.84E-10	8.49E-08	1.60E-09
Th-227	1.09E-09	1.16E-07	8.78E-09	1.97E-10
Th-228	1.70E-09	2.55E-06	1.58E-10	4.34E-12
Th-229	1.53E-09	2.05E-06	6.90E-09	1.66E-10
Th-230	1.01E-09	4.66E-07	3.00E-11	1.33E-12
Th-231	4.85E-11	2.97E-11	8.91E-10	2.83E-11
Th-232	8.85E-10	5.21E-07	1.39E-11	9.57E-13
Th-234	5.20E-10	5.15E-10	5.98E-10	1.54E-11
Pa-231	4.02E-09	6.54E-07	2.54E-09	5.92E-11
Pa-233	1.27E-10	1.33E-10	1.80E-08	3.97E-10
Pa-234	5.76E-11	3.51E-11	1.78E-07	3.42E-09
Pa-234m				
U-232	2.19E-09	1.43E-06	2.01E-11	1.47E-12
U-233	1.21E-09	3.82E-07	1.91E-11	7.46E-13
U-234	1.20E-09	3.77E-07	1.13E-11	1.13E-12
U-235	1.22E-09	3.51E-07	1.28E-08	2.84E-10
U-236	1.14E-09	3.57E-07	8.72E-12	1.02E-12
U-237	1.08E-10	8.43E-11	1.08E-08	2.58E-10
U-238	1.15E-09	3.36E-07	7.46E-12	8.97E-13
U-240	1.48E-10	9.06E-11	4.84E-11	5.36E-12
Np-236	2.52E-11	1.05E-10	1.06E-08	2.53E-10
Np-237	7.98E-09	9.33E-07	1.79E-09	4.92E-11
Np-238	1.23E-10	1.26E-10	5.11E-08	9.48E-10
Np-239	1.15E-10	6.52E-11	1.38E-08	3.15E-10
Np-240	4.79E-12	3.55E-12	1.04E-07	2.04E-09
Np-240m	6.54E-13	7.64E-13	2.95E-08	5.80E-10
Pu-236	2.08E-09	3.61E-07	8.30E-12	1.34E-12
Pu-238	7.98E-09	7.42E-07	6.05E-12	1.16E-12
Pu-239	8.53E-09	7.51E-07	6.29E-12	5.28E-13
Pu-240	8.52E-09	7.51E-07	5.93E-12	1.11E-12
Pu-241	1.40E-10	7.59E-09		
Pu-242	8.11E-09	7.13E-07	5.04E-12	9.20E-13
Pu-243	9.97E-12	7.22E-12	1.82E-09	4.46E-11
Pu-244	8.46E-09	7.20E-07	3.63E-12	7.81E-13
Am-241	8.87E-09	1.04E-06	1.36E-09	4.26E-11
Am-242	3.96E-11	2.81E-10	1.10E-09	2.84E-11
Am-242m	7.90E-09	9.43E-07	3.42E-11	3.69E-12
Am-243	8.84E-09	1.03E-06	3.85E-09	9.92E-11
Am-244	1.03E-09	8.54E-08	6.63E-12	1.23E-12
Am-245	6.79E-09	7.81E-07	1.05E-08	2.40E-10
Am-246	5.69E-09	6.58E-07	5.60E-12	1.10E-12
Am-247	9.05E-09	1.06E-06	5.75E-09	1.36E-10
Am-248	8.97E-09	1.05E-06	4.65E-12	9.67E-13
Am-249	8.35E-09	9.68E-07	2.72E-08	5.78E-10
Am-250	3.54E-08	3.95E-06	4.13E-12	7.76E-13
Am-251	4.86E-09	7.01E-07	5.04E-12	8.22E-13

Table A.5 Default inhalation clearance class and ingestion f_1 values by element.

Element	Inhalation clearance class	Ingestion f_1	Element	Inhalation clearance class	Ingestion f_1
H	V	1.0E+00	I	D	9.5E-01
Be	Y	5.0E-03	Xe	*	
N	D	9.5E-01	Cs	D	9.5E-01
C [*]	D	9.5E-01	Ba	D	1.0E-01
O	D	9.5E-01	La	W	1.0E-03
F	D	9.5E-01	Ce	Y	3.0E-04
Na	D	9.5E-01	Pr	Y	3.0E-04
Si	W	1.0E-02	Nd	Y	3.0E-04
P	D	8.0E-01	Pm	Y	3.0E-04
S	D	8.0E-01	Sm	W	3.0E-04
Cl	D	9.5E-01	Eu	W	1.0E-03
Ar	*		Gd	W	3.0E-04
K	D	9.5E-01	Tb	W	3.0E-04
Ca	W	3.0E-01	Dy	W	3.0E-04
Sc	Y	1.0E-04	Ho	W	3.0E-04
V	W	1.0E-02	Er	W	3.0E-04
Cr	Y	1.0E-01	Tm	W	3.0E-04
Mn	W	1.0E-01	Yb	Y	3.0E-04
Fe	W	1.0E-01	Lu	Y	3.0E-04
Co	Y	3.0E-01	Hf	W	2.0E-03
Ni	W	5.0E-02	Ta	Y	1.0E-03
Cu	Y	5.0E-01	W	D	3.0E-01
Zn	Y	5.0E-01	Re	W	8.0E-01
Ga	W	1.0E-03	Os	Y	1.0E-02
Ge	W	9.5E-01	Ir	Y	1.0E-02
As	W	5.0E-01	Pt	D	1.0E-02
Se	W	8.0E-01	Au	Y	1.0E-01
Br	D	9.5E-01	Hg	W	2.0E-02
Kr	*		Tl	D	9.5E-01
Rb	D	9.5E-01	Pb	D	2.0E-01
Sr	D	3.0E-01	Bi	W	5.0E-02
Y	Y	1.0E-04	Po	W	1.0E-01
Zr	W	2.0E-03	At	D	9.5E-01
Nb	Y	1.0E-02	Rn	*	
Mo	Y	8.0E-01	Fr	D	9.5E-01
Tc	W	8.0E-01	Ra	W	2.0E-01
Ru	Y	5.0E-02	Ac	Y	1.0E-03
Rh	Y	5.0E-02	Th	Y	2.0E-04
Pd	Y	5.0E-03	Pa	Y	1.0E-03
Ag	Y	5.0E-02	U	Y	5.0E-02
Cd	Y	5.0E-02	Np	W	1.0E-03
In	W	2.0E-02	Pu	Y	1.0E-03
Sn	W	2.0E-02	Am	W	1.0E-03
Sb	W	1.0E-01	Cm	W	1.0E-03
Te	W	2.0E-01	Cf	Y	1.0E-03

*For ¹⁴C, clearance class is *, f_1 is 1.0.

References for Appendix A

C.L. Chiang, *An Introduction to Stochastic Processes and Their Applications*, Robert E. Krieger Publishing Company, Inc., Huntington, NY, 1980.

J.R. Cook, B.M. Bungler, and M.K. Barrick, *A Computer Code for Cohort Analysis of Increased Risks of Death (CAIRD)*, EPA 520/4-78-012, U.S. Environmental Protection Agency, Washington, DC, June 1978.

C. de Boor, *A Practical Guide to Splines*, Applied Mathematical Sciences Vol. 27, Springer-Verlag, New York, NY, 1978.

F.N. Fritsch and J. Butland, *A Method for Constructing Local Monotone Piecewise Cubic Interpolants*, UCRL-87559, Lawrence Livermore National Laboratory, April 1982.

National Center for Health Statistics, *Vital Statistics Mortality Data, Detail, 1979*, PB82-132340, U.S. Department of Health and Human Services, Public Health Service, National Center for Health Statistics, Hyattsville, MD, 1982.

National Center for Health Statistics, *Vital Statistics Mortality Data, Detail, 1980*, PB83-261545, U.S. Department of Health and Human Services, Public Health Service, National Center for Health Statistics, Hyattsville, MD, 1983.

National Center for Health Statistics, *Vital Statistics Mortality Data, Detail, 1981*, PB84-213008, U.S. Department of Health and Human Services, Public Health Service, National Center for Health Statistics, Hyattsville, MD, 1984.

National Center for Health Statistics, *U.S. Decennial life Tables for 1979-1981*, Vol. 1, No. 1, United States Life Tables, (PHS) 85-1150-1, U.S. Department of Health and Human Services, Public Health Service, National Center for Health Statistics, Hyattsville, MD, August 1985.

National Institutes of Health, *Report of the National Institutes of Health Ad Hoc Working Group to Develop Radioepidemiological Tables*, NIH Publication 85-2748, Superintendent of Documents, U.S. Government Printing Office, Washington, DC, 1985.

National Research Council, *Health Effects of Exposure to Low Levels of Ionizing Radiation (BEIR V)*, Committee on the Biological Effects of Ionizing Radiations, Board of Radiation Effects Research, Commission on Life Sciences, National Research Council, National Academy Press, Washington, DC, 1990.

Public Health Service, *The International Classification of Diseases, 9th Revision, Clinical Modification (ICD-9-CM)*, Vol. 1 Diseases: Tabular List, DHHS Publication No. (PHS) 80-1260, U.S. Department of Health and Human Services, Public Health Service—Health Care

Financing Administration, Superintendent of Documents, U.S. Government Printing Office, Washington, DC, 1980.

A.L. Sjoreen, *Reconstruction of the RADRISK Database*, Oak Ridge National Laboratory, Oak Ridge, TN, (to be published) 1994.

M. Vaeth and D.A. Pierce, *Calculating Excess Lifetime Risk in Relative Risk Models*, RERF CR 3-89, Editorial Office, Radiation Effects Research Foundation, Hiroshima, Japan, November 1989.