# UNCERTAINTIES IN RADIATION CANCER RISK ESTIMATES

# UNCERTAINTIES IN LIFETIME RISK PROJECTIONS FOR RADIATION-INDUCED CANCER AND AN ASSESSMENT OF THE APPLICABILITY OF THE ICRP-60 CANCER RISK ESTIMATES TO THE CANADIAN POPULATION

BY

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TITLE: Uncertainties in Lifetime Risk Projections for Radiation-Induced Cancer and an Assessment of Applicability of the ICRP-60 Fatal Cancer Risk Estimates to the Canadian Population

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## ABSTRACT

The BEIR V preferred relative risk models and standard life-table techniques are used to project lifetime fatal cancer risk factors for average members of the Canadian population. Uncertainties associated with projections are evaluated for: (1) sampling variation (statistical error), (2) extrapolation of risks to low doses and low dose rates, (3) projection of excess lifetime cancer risks beyond the current periods of human observation in epidemiological studies, (4) the transfer of site-specific excess risk coefficients between populations with differing baseline cancer rates, and (5) the effect of differences in the age and sex distributions among occupations in the Canadian "radiation" workforce. Results are used to assess the applicability of the fatal cancer risk estimates recommended in ICRP publication 60 to the Canadian population.

It was found that sampling variation, extrapolating to low doses and dose rates, projecting excess risks beyond current periods of observation, and the uncertainty in how to transfer site-specific excess risks between populations all cause substantial variations in lifetime cancer risk projections. Site-specific cancer risk projections may be expected to vary by factors of 2 to 5, depending on the source

of uncertainty.

Site-specific differences were found in the fatal cancer risk factors projected for "average" male and female workers among different occupations in the Canadian workforce. Site-specific worker averages differed by as much as a factor 3. Female average risk factors for digestive cancers were substantially higher than male workers, while male average risk factors tended to be higher for leukemia and respiratory cancer. Overall however, the majority of worker risk factors were within 25% of the site-specific projections for the workforce as a whole.

The ICRP-60 nominal fatal cancer risk estimates, tissue weighting factors, and lifetime risk projections for prolonged radiation exposure were all in good agreement with equivalent values derived in this report for the Canadian population. In view of the uncertainties, the results suggest the ICRP estimated cancer risks are as good as any presently available and supports the use of the ICRP recommended values for the planning and regulation of radiation protection in Canada.

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## 1.0 Introduction

### 1.1 Background

Present knowledge of the carcinogenic effects of ionizing radiation in humans is restricted primarily to effects observed in the Life Span Study of the Japanese survivors of the atomic bombings in Hiroshima and Nagasaki and in studies of populations irradiated for medical reasons. These studies show that external exposure to sufficiently high doses of low-LET[1] radiation delivered in a short time period can increase the subsequent probability of cancer mortality in most organs and tissues of the body. However, the ability of ionizing radiation delivered at low doses and/or at low dose rates to increase the risk of cancer is less clear. Linear extrapolation of effects observed at high doses suggest that any increase cancer risk caused by low dose exposure will be small and difficult to distinguish from the statistical variation expected in the "normal" rate of cancer or from increases caused by other factors. As a result, low dose studies, such as those of occupationally exposed groups, have provided little, if any, reliable quantitative information to base estimates of radiation-induced cancer risks. Therefore

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[1] Linear energy transfer

risk estimation has relied on the extrapolation of effects observed at high doses and high dose rates (Upton 1991, Darby 1991, ICRP 1991, NRC 1990, UNSCEAR 1988).

The Life Span Study of the atomic bomb survivors represents the single largest source of information on the carcinogenic risk of external exposure to high dose and dose rate low-LET ionizing radiation. Studies of medically irradiated populations taken as a whole also provide a substantial amount of information. However, few of the medical studies are adequate enough to provide by themselves enough reliable data for predicting site-specific radiogenic risks (Darby 1991). At present, increased cancer risks at specific cancer sites have only been examined within specific cohort populations. There has been no attempt so far to conduct a comprehensive analysis that combines all the available data from the various studies in order to derive site-specific excess risk coefficients. As a consequence, current risk estimates for radiation-induced cancer use site-specific risk coefficients that are derived almost entirely from the cancer mortality observed in the Life Span Study.

In 1988, the Radiation Effects Research Foundation, or RERF, reanalysed cancer mortality among the atomic bomb survivors using the new individual dose estimates of the 1986 Dosimetry System (DS86) and cancer mortality data for the years 1950-1985. The United Nation Scientific Committee on the

Effects of Atomic Radiations (UNSCEAR 1988) concluded, based on the subsequent RERF report by Shimizu et al. (1988), that the estimated cancer risk following radiation exposure had increased significantly compared to previous estimates made in 1977 (UNSCEAR 1977). In 1989, the fifth National Research Council Committee on the Biological Effects of Ionizing Radiation, or the BEIR V Committee, conducted its own analysis of the carcinogenic effects of low-LET radiation. The analysis used data from the Life Span Study as well as any data that was available to the Committee from other studies. The results of the analysis, published in the BEIR V Report (NRC 1990), found that the predicted lifetime increase risk in cancer mortality for the U.S. population following a hypothetical single whole-body dose of 0.1 Gy was about 3-4 times higher than that predicted in 1980 in the BEIR III report (NRC 1980).

Impelled by the RERF, UNSCEAR, and BEIR results, the International Commission on Radiological Protection, or ICRP, decided to reassess their 1977 risk estimates for the carcinogenic effects of radiation. In early 1990 the Commission circulated to regulatory bodies and radiation protection organizations around the world a draft report summarizing the results of their reassessment. The final report was released a year later as the "1990 Recommendations of the ICRP" in ICRP publication 60 (ICRP 1991). The new recommended estimate for fatal cancer following low-level

whole-body radiation exposure is 5.0 x $10^{-4}$ per Sv for a general population (ages 0-90) and 4.0 x $10^{-4}$ per Sv for a general population (ages 18-65). These estimates are significantly higher than the previous 1977 estimates (ICRP 1977) by a factor of about three. In addition, there is a new set of tissue weighting factors which includes 7 additional organ sites. New effective dose[2] limits for radiation protection were also recommended to reflect the higher estimated cancer risk. The new recommended public limit is 1 millisieverts (mSv) in a year[3] (reduced from 5 mSv per year) and the new occupation limit is 20 mSv per year averaged over defined periods of 5 years[4] (changed from 50 mSv per year).

In view of the potential impact of the new ICRP recommendations on the planning and regulation of radiation protection in Canada, this thesis report was commissioned by the Atomic Energy Control Board of Canada to examine the uncertainties in the risk assessment process with emphasis on assessing the Commission's approach and the suitability of using the new risk factors for the purpose of radiation protection in Canada.

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[2] ICRP 60 uses the term "effective dose" to denote the effective dose equivalent.

[3] In special circumstances, the Commission suggests a higher value of effective dose could be allowed in a single year, provided that the average over 5 years does not exceed 1 mSv per year

[4] With the further provision that the effective dose should not exceed 50 mSv in any single year.

## 1.2 Overview of the Risk Assessment Process

The process of predicting the probability of developing and dying from a radiation-induced cancer involves:

(a) developing risk models or excess risk coefficients to describe the subsequent magnitude and pattern of increase risk of cancer following exposure and

(b) using the risk models and coefficients to predict the increase lifetime risk of cancer for an exposed population.

Both the reports by Shizimu et al. (1988) and the BEIR V Committee (NRC 1990) give models and risk coefficients by which the lifetime mortality risk due to radiation-induced cancer can be calculated for an exposed population. Using DS86 individual dose estimates with a neutron RBE[5] of 10 and an assumed linear dose-response, Shizimu et al. fitted both age-constant absolute risk (AR) and relative risk (RR) models to atomic bomb survivor cancer mortality data for the years 1950 to 1985. Excess absolute risk (AR) and relative risk (RR) coefficients at a dose of 1 Gy were derived for 27 different cancer sites. Cancers were significantly elevated for 9 sites: all cancers except leukemia as a group, leukemia, lung, female breast, stomach, colon, oesophagus, ovary, and bladder. Data was sufficient at six of the cancer sites to allow the derivation of coefficients that described variations of the

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[5] Relative biological effectiveness

excess risk with sex and by 10 year age groups of age at the time of the bombings (age ATB). For the remaining sites (oesophagus, ovary, and bladder), data was only sufficient to produce coefficients which were the average over all ages ATB and both sexes.

The goal of the BEIR V Committee's analysis was to develop risk models which were suitable for projecting the excess lifetime cancer risks resulting from the hypothetical radiation exposure of a U.S. population. The Committee used exposure-time-response risk models which are capable of describing possible variations in the excess risk with sex, exposure age, or time following exposure. These models were fitted to the same A-bomb cancer data used in the RERF analysis[6] and, whenever possible, to data from other studies. The analysis produced "preferred" relative risk models for leukemia, cancers of the respiratory tract, female breast, digestive system, and other remaining cancers combined. A separate model was also developed to predict the possible increase of thyroid cancer using data from the study of Israeli children receiving x-ray irradiation for the treatment of tinea capitis and the study of infants in Rochester N.Y. irradiated in the treatment of supposedly enlarged thymus glands.

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[6] Except the neutron RBE was taken to be 20

Once risk models or coefficients describing the increase risk in cancer has been developed, the last step in the risk assessment process is to transfer excess risks to other populations where the potential lifetime cancer risks resulting from radiation exposure are to be assessed. It is important to recognize that the subsequent increased cancer risk can not be assessed for a specifically exposed individual. The carcinogenic process is very complex, many factors can influence not only radiogenic risks, but also the "normal" risk of cancer. When speaking of an individual's lifetime risk of cancer, it is addressed in terms of the risk to an "average" member in a hypothetical population. For risk assessment, a hypothetical population is normally constructed using standard life-table techniques. A life-table assumes that individuals in the hypothetical population experience the same national age- and sex-specific cancer mortality rates and mortality rates from all causes of death as the country in which they are living, and further assumes that these rates will remain constant throughout their lifetime. By following an initial cohort of 100,000 newborns throughout their life, the life-table can predict the total number of deaths and cancer deaths that can be expected to occur each subsequent year as the cohort ages. The effect of radiation exposure is evaluated by incorporating the additional risk of cancer given by the risk models and/or coefficients.

The ICRP 60 report used the above approach in their reassessment of fatal cancer risk estimates and tissue weighting factors ($w_T$s). In order that their risk estimates be applicable internationally, the Commission chose to average projections over the national populations of Japan, United States, Puerto Rico, the United Kingdom, and China. Although the BEIR V models were acknowledged by the Commission, projections were performed using only the risk coefficients given by Shizimu et al. (1988). Additional estimates were also provided for cancers of the thyroid, bone, liver, and skin using risk estimates made by other reports[7].

For this thesis report, it was decided to perform lifetime risk projections for the Canadian population using the five preferred relative risk models developed by the BEIR V Committee[8]. There are two main reasons for using the Committee's models. First, the BEIR V analysis is the only analysis to date that has attempted to develop risk models

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[7] The other reports were NCRP report 80 (NCRP 1985), UNSCEAR (1988), BEIR IV and V Reports (NRC 1988, 1990), and the report of the ICRP Task Group on the Skin which was still in preparation.

[8] It was decided not to perform risk projections for the increase risk of thyroid cancer. The BEIR V thyroid model is based on a small number of excess thyroid cases observed among the Israeli tinea capitis patients. While the increased relative risk per Gy is large for thyroid cancer, estimated relative risks are very unstable and the variation of relative risks between different ethnic groups is unclear. Since the Committee's analysis, additional data from the continued follow-up of patients (Ron et al. 1989) indicates relative risks are higher than those given in the BEIR V report. Given the instability of results from this cohort, it was concluded the inclusion of projections for thyroid cancer was not warranted and that the ICRP 60 estimate was as good as any presently available. See section 3.7.3 for further detail.

specifically for the purpose of carrying out lifetime risk projections using data of other studies, in addition to the data from the Life Span Study. In contrast, the purpose of the analysis by Shizimu et al. (1988) was to evaluate the excess mortality experienced solely in the LSS cohort. And the second reason is to allow assessment of the applicability of ICRP 60 risk estimates to the Canadian population while at the same time evaluating whether the Commission's more detailed site-specific estimates are consistent with estimates made using the BEIR V models.

## 1.3 Outline of the Report

As mentioned, this report examines the uncertainties associated with the estimation of the carcinogenic effects of low-level radiation exposure and assesses the applicability of the ICRP 60 cancer risk estimates to the Canadian population. This is done by first reviewing the results of the major human epidemiological studies (chapter 2.0) and discussing the issues and uncertainties associated with using epidemiological results for predicting potential low-level radiation effects (chapter 3.0). Chapter 4.0 describes the BEIR V preferred relative risk models, develops the methodology of lifetime risk projections, and defines several risk attributes that can be used to describe the increase cancer risk following exposure. Using the methods developed in chapter 4.0, analyses are performed in chapters 5.0 and 6.0 examining the effect of

two specific elements associated with projecting lifetime cancer risks for the average member of the Canadian population:

(1) the effect of the choice of method for transferring excess relative risk coefficients between populations and

(2) the effect of differences in worker age and sex distributions among occupations in the Canadian radiation workforce.

And finally, chapter 7.0 compares the nominal fatal cancer risk estimates and tissue weighting factors recommended in ICRP 60 with those derived for the Canadian general and working population.

## 2.0 Human Epidemiology Studies

### 2.1 Introduction

Studies of populations exposed to external low-LET radiation delivered at high doses and high dose rates illustrate the ability of ionizing radiation to induce cancer in most organs and tissues of the body. Numerous studies show radiation-related excesses of leukemia (excluding chronic lymphatic leukemia), cancers of the thyroid, female breast, lung, stomach, colon, oesophagus, bladder, and ovary, and of multiple myeloma. Clear radiation-related increases has also been seen in specific irradiated populations for cancers of the salivary glands, rectum, brain and nervous system, kidney, body of the uterus, bone and connective tissue, and also for non-Hodgkin's lymphoma. For most other sites, clear radiation-related excesses have not yet been demonstrated. However, this does not necessarily indicate they are not sensitive to radiation. It may be the case of a lack of studies involving substantial irradiation of the appropriate organs (Darby 1991).

Table 2.1 summarizes the major human studies providing information on the effects of external exposure to high dose rate low-LET radiation. The primary source of information for risk estimation comes from the Life Span Study (LSS) of the

Japanese survivors of the atomic bombings in Hiroshima and Nagasaki. The LSS cohort is composed of about 120,000 individuals of all ages and both sexes exposed to nearly uniform whole-body external gamma and neutron radiation over a large dose range. The Tentative 1965 Doses (T65D) estimated individual doses for 91,228 survivors. This number has been reduced to 75,991 under the new 1986 Dosimetry system (DS86). This group is referred to as the DS86 cohort. The latest period of follow-up for the survivors covers the years 1950 to 1985 in which 5936 cancer deaths were identified in the DS86 cohort, approximately 355 above the expected (Shimizu et al. 1988).

Other epidemiological studies consist mostly of populations irradiated with external low-LET radiation for medical diagnostic or therapeutic reasons. These studies are increasingly playing a more important role in evaluating the radiation-related cancer risks at specific tissues and organs as well as providing information regarding variations in excess risks with exposure age, sex, time sine exposure, and population. Few of these studies however are adequate enough to provide by themselves reliable data to be used to predict radiogenic risks. Extensive analyses using all available information has not yet been carried out. Until such time, risk estimation will continue to rely heavily on the Japanese survivor data (Darby 1991).

The remainder of this chapter reviews the major epidemiological studies providing information on the carcinogenic effects of exposure to low-LET ionizing radiation and describes the analyses and findings of each.

## 2.2 Life Span Study of Japanese Atomic Bomb Survivors

### 2.2.1 Cohort, follow-up, and dosimetry

The Life Span Study of the Japanese atomic bomb survivors consists of a population of 120,128 males and females of all ages at the time of exposure. This includes 26,517 unexposed individuals who were living in either Hiroshima or Nagasaki in 1950 but who were not in the cities at the time of the bombings (ATB). In 1965, tentative doses (T65D) were estimated for 91,228 of the 93,600 exposed survivors. This number has since been reduced to 75,991 under the new 1986 dosimetry system (DS86). The cancer mortality in this latter group, known as the DS86 cohort, has been examined for the years between 1950 and 1985 (Shimizu et al. 1988).

By 1985, nearly 60% of the DS86 cohort were still living. Causes of death among those who had died, were identified through the Japanese Koseki obligatory household registries with almost complete ascertainment. To date, a total of 5936 cancers deaths have been observed, approximately 355 above that expected. The total includes 202 leukemia deaths, 638 lung cancer deaths, 2007 stomach cancer deaths,

and 155 breast cancer deaths, all of which were significantly elevated (see tables 2.2 and 2.3).

Survivors were divided into three subgroups for analysis:

(a) proximal exposed group: survivors located at ground distances within 2,499m of the hypocentre (centre of explosion);

(b) distal exposed group: survivors located at ground distances between 2500 and 10,000m from the hypocentre; and

(c) not exposed group: survivors located at more than 10,000m from hypocentre or who were not-in-city ATB but took up residency in Hiroshima or Nagasaki prior to 1950 (Kerr 1989).

Dose estimates ranged from under 0.01 Gy up to 4 Gy or more. The majority of the excess cancers were restricted to a subgroup of 7600 survivors (10% of the entire cohort) who received doses over 0.5 Gy. Among the remaining cohort, approximately 34,000 members were used as internal controls. These included individuals who were not in the city ATB and distal exposed survivors with estimated doses below 0.005 Gy (NRC 1990, Shimizu et al. 1988).

The DS86 dosimetry system has resulted in more reliable and precise organ dose estimates for individual survivors. The major change in the DS86 from the T65D

dosimetry was the reduction of the neutron component of radiation doses in Hiroshima and Nagasaki by factors of about 10 and 2 respectively. This has resulted in the neutron dose in both cities being no longer considered significant. Other changes include an increased free-in-air gamma-ray kerma in Hiroshima, increased shielding effect of houses, and a decrease in the shielding of organs by the body. The increase in shielding by houses and decrease in shielding by the body tended to offset each other so that estimated organ gamma doses remained about the same as before. However, differences between risk estimates made with the DS86 and T65D are sensitive to the choice of the value of the neutron RBE. An assumed value of 1 gives no difference while an assumed RBE of 20 results in DS86 risk estimates being 35-40% higher than T65D estimates (NRC 1990, Preston and Pierce 1988). Section 2.2.3 discusses further the differences in the two dosimetry systems.

2.2.2 Results

Tables 2.2 and 2.3 summarize excess risks observed at specific cancer sites. Excess cancers attributed to exposure were leukemia (except chronic lymphatic leukemia), cancers of the oesophagus, stomach, colon, lung, breast, ovary, and urinary tract, tumours of the central nervous system (excluding the brain), and multiple myeloma. Cancers were elevated, but not significantly, for the liver, gallbladder,

uterus, skin (except melanoma), bone, and larynx. No increase has yet been observed at any other cancer site. The observed absolute risk for leukemia was 2.94 (2.43, 3.49 90% CI[9]) excess deaths per $10^4$ PYGy[10] and for cancers other than leukemia combined, 10.13 (7.96, 12.44) excess deaths per $10^4$ PYGy.

Leukemia

No information is available on the excess number of leukemia deaths in the first five years following exposure. Excess leukemia deaths observed after this time were seen to peak within 6-8 years after exposure and to decline thereafter, but has remained significantly increased in Hiroshima during the years 1981-1985 (RR at 1 Gy = 2.92 (1.47, 6.33) (Shimizu et al. 1988). Over the entire follow-up period, the relative risk at 1 Gy was significantly higher among those exposed at ages under 10 ATB who experienced a relative risk of 20 compared to the cohort average of 6.2 (Shimizu et al. 1988). The excess number of leukemias was significantly higher for male than for females, 3.14 and 1.80 excess deaths per $10^4$ PYGy, respectively.

The dose-response was described equally as good by a linear and linear-quadratic model when analysis was carried out for both cities and doses restricted to under 4 Gy

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[9] 90% confidence interval.

[10] Person year Grays

(Shimizu et al. 1988).

Cancers other than leukemia

The latest follow-up period (1950-1985) indicates that excess deaths for all cancers other than leukemia combined has continued to increase with time in proportion to the expected increase in baseline rates. This apparent constancy in the relative risk over time is seen for most ages at exposures except for children exposed under the age of 10 ATB. This group experienced very high relative risks at attained ages below 30, which consequently fell to a lower, but constant, level for at older attained ages. As a result of these observations the age-constant relative risk model, rather than the age-constant absolute risk model, appears preferable for describing excess risk experienced in this cohort (see section 3.6.1 for additional detail on risk models).

As mentioned above, relative risks were higher among those exposed at younger ages ATB. Excess relative risks among children exposed under the of 10 were about 2-3 times higher than those seen for adults above the age 30 ATB. However, very few cancers have occurred among these younger survivors and individuals are only now approaching ages where the baseline rate of cancer is expected to start increasing. It is not known whether the relative risks will remain at the same high levels in the future.

At individual cancer sites, relative risks have

appeared to have remain fairly constant over time as well, although a slight decreasing, but not significant, trend has been detected for lung cancer. No significant sex difference has been observed in absolute excess risks, but relative risks were observed to be significantly higher for females than for males for cancers of the oesophagus and lung. When adjustments were made between males and females for the effects of smoking, the sex difference in the relative risk of lung cancer was no longer significant. The dose-response for cancers other than leukemia was best described by a linear function (Shimizu et al. 1988).

2.2.3 T65D and DS86 Dosimetry Systems (Kerr 1989, RERF 1987)

A great deal of effort has gone into assessing the doses received by individual survivors since the atomic bombs were dropped on Hiroshima and Nagasaki in 1945. For completeness, and also for interest sake, the development of A-bomb dosimetry over the past 40 years will be summarized.

Dose assessment has involved the evaluation of:

(1) the yield of the bombs;

(2) the radiation output of the bombs;

(3) the tissue kerma (both neutron and gamma) in air at the location of survivors without adjustment for shielding (free-in-air kerma);

(4) the tissue kerma adjusted for shielding by houses, buildings, and terrain (shielded kerma); and

(5) the organ-absorbed dose adjusted for the absorption and scattering of radiation by an individual's body.

The task of evaluating these five components has involved a number of activities including measurements of physical data by Japanese scientists immediately after the bombings; the questioning of survivors to obtain information on their location, orientation, and shielding ATB; the estimation of free-in-air (FIA) kerma as a function of distance from explosion and shielding provided by houses from measurements made during weapon tests and experiments using gamma and neutron sources; the estimation of free-in-air kerma and shielding provided by houses from calculations performed by computer-based models employing Monte Carlo simulations and numerical methods; and the use of physical phantoms and Monte Carlo simulations to determine the shielding of organs by the body.

Two major dosimetry systems have arose over the last forty years. The first was the tentative 1965 dosimetry (T65D) system which is based entirely on data from weapon tests and experiments using gamma and neutron sources. The second, and most current, is known as the Dosimetry System 1986 (DS86) and is based entirely on computer-based models employing Monte Carlo simulations and numerical methods.

T65D Dose Estimates

The tentative 1965 dosimetry (T65D) system is based on

experimental data obtained from the Nuclear Testing Site (NTS) prior to the 1962 Limited Test Ban Treaty and thereafter from experiments conducted at operation BREN at NTS.

Weapons tests in the 1940s and 1950s were used to estimate the yields of the Hiroshima and Nagasaki bombs. Because bombs detonated during weapon tests were identical to the one dropped on Nagasaki, the yield of the Nagasaki bomb was estimated fairly accurately from the radiochemical evaluation of debris and the measurement of fireball expansion of the test bombs. The bomb dropped on Hiroshima, however, was a one of-a-kind design, and therefore its yield had to be estimated indirectly by comparing blast damage between Hiroshima and Nagasaki. This procedure resulted in yield estimates of 12 ± 1 ktons in Hiroshima and 22 ± 2 ktons for Nagasaki.

Operation BREN consisted of a series of experiments at NTS designed to derive free-in-air kerma versus distance curves and the transmission factors for Japanese houses. BREN used a small unshielded reactor, a large Co-60 source, and a charged particle accelerator mounted on a 500m tower to simulate the neutron and gamma ray output of the atomic bombs.

Free-in-air kerma versus distance curves were derived from an empirical fit of a simple 2-parameter formula to results of FIA kerma measurements made at various distances from source during weapon tests and BREN experiments.

Initially it was believed that the kerma versus distance curve would be used temporarily until further work could be performed. However, it was found that the curves agreed remarkably well with TL measurements of decorative tile and brick that had received gamma irradiation from the atomic bomb expolsions. As the result, the T65D FIA kerma doses were initially used with a great deal of confidence for assessing excess cancer risks.

Operation BREN also produced a set of factors describing the transmission of neutron and gamma rays through typical Japanese houses at over 20 different locations within a house using a nine-parameter method[11]. Information from interviews with survivors were used to determine the position and orientation of individual survivors moments prior to the detonation of bombs. Transmission factors were assigned to as many individual survivors as possible. For survivors who had incomplete or unavailable shielding histories, average transmission factors were used.

For survivors exposed in the open (e.g. outside the house) a globe method was used to determine transmission factors. The globe method determined transmission factors by direct observation using scaled models of houses and terrain and a spherical light projector called the "globe" which

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[11] The nine parameters were: (1) front shielding, (2) front shielding size, (3) unshielded, (4) lateral shielding, (5) internal front wall, (6) internal lateral walls, (7) height above floor, (8) floor number, and (9) slant penetration

simulated a survivor's exact exposure condition. A single transmission factor was used for distal survivors.

Transmission factors and radiation doses were neither calculated nor assigned for 3017 proximal exposed survivors because their shielding conditions were either extremely complex or unknown.

Using the transmission factors and FIA kerma dose, the T65D system estimated the shielded kerma dose to 91,228 survivors. These values were used to approximate organ doses until absorbed-dose factors were available for calculating organ doses in the early 1970s.

Reassessment of Dosimetry

Concerns regarding the accuracy of the T65D system were first raised in the mid-1970s. Calculations of FIA kerma for Hiroshima and Nagasaki using bomb leakage radiation (radiation output) calculations and state-of-the-art computers indicated significant errors in the T65D kerma dose estimates. It was concluded that the neutron kerma was overestimated by nearly a factor of 10 in Hiroshima and a factor of 2 in Nagasaki while the gamma-ray kerma at 2000m in Hiroshima was underestimated by a factor of 4. Re-analysis of house transmission factors also revealed T65D gamma ray transmission factors were too high by a factor of almost 2.

These errors were substantiated by several investigators and were attributed to the failure to account

for the higher humidity in Hiroshima and Nagasaki compared to NTS; the use of inadequate and inappropriate radiation sources in Operation BREN; and the overestimation of transmission factors caused by production of gamma radiation from neutron interactions with housing materials in the BREN experiments. As a result of these errors, there were questions as to how individual dose estimates might be affected. A joint U.S. - Japan research program was therefore establish in early 1983 to thoroughly review and reassess all aspects of A-bomb radiation dosimetry. The reassessment resulted in the development of the Dosimetry System 1986 (DS86), which represents a complete replacement of the T65D system.

Dosimetry System 1986

The Dosimetry System 1986 differed from T65D in that it was produced entirely from computer-based models employing Monte Carlo simulations and numerical methods. The models made use of physical data[12] obtained in Hiroshima and Nagasaki shortly after the bombings in 1945.

Improved blast wave models and physical data indicated that the bomb yield was somewhat higher in Hiroshima (15 kton in contrast to 12 kton) and slightly lower in Nagasaki (21

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[12] Physical data included measurements of fast neutron activation of sulphur used as glue in electric insulators, activity induced in cobalt impurities in iron and Eu-152 induced in rock by thermal neutrons, gamma dose delivered to small quartz inclusions in kiln-fired brick and tile used in buildings using TL techniques, and gamma dose in shell buttons and teeth using electron spin resonance (measurements made in 1980s).

kton in contrast to 22 kton). This resulted in the gamma-ray FIA increasing in Hiroshima and slightly decreasing in Nagasaki.

The FIA kerma versus distance from hypocentre was based on complex calculations of transport and hydrodynamics of exploding bombs, extensive transport Monte Carlo calculations, and comparison to physical data. It was found that the neutron FIA kerma dose decreased by a factor of about 10 in Hiroshima and a factor of 2 in Nagasaki. Besides taking into account the high humidity in the Japanese cities, the reduction in neutron kerma was also due in part to radiation output calculations indicating the average fast neutron energy in Hiroshima was 0.3 MeV whereas the fission neutrons in the BREN experiments had an average energy of 1 MeV, thus resulting in even more absorption of neutrons in air.

The shielding of survivors were determined using detailed computer models of houses and tenements, Monte Carlo calculations, and the nine-parameter and globe shielding data. The calculations indicated a reduction of housing transmission factors for gamma-rays by a factor of about 2. The T65D system had overestimated transmission factors because gamma emitting nuclides in the roof and walls of the house were produced by neutrons from the unshielded reactor at Operation BREN.

The calculation of organ-absorbed doses was based on measurements in physical phantoms using information on the

posture and orientation of survivors at the time of the bombings. Calculations were verified by Monte Carlo simulation. It was found that the shielding of internal organs by the body appeared to be less than was previously thought.

Unlike T65D, DS86 did not use transmission factors or absorbed dose factors to calculate organ-absorbed dose per se. Instead, if the shielding history of a survivor was available, the shielded kerma and organ doses were calculated directly. If shielding histories were not available for proximally exposed survivors, individual doses were estimated indirectly using the average of transmission factors calculated for individuals exposed inside houses. No dose estimates were made for distally exposed individuals without shielding histories.

In summary, the main differences between the DS86 and T65D dosimetry systems are:

(1) increased FIA gamma-ray kerma in Hiroshima;
(2) decreased FIA neutron kerma by a factor of 10 in Hiroshima and a factor of 2 in Nagasaki;
(3) decrease of house transmission factors by a factor of about 2; and
(4) decrease in shielding of organs by the body

The DS86 system has resulted in more reliable and accurate organ dose estimates for individual survivors. However, the increased shielding by houses and reduced shielding by the body tended to compensate so that on average,

therefore, gamma organ-absorbed doses are about the same as before[13].

## 2.3 Ankylosing Spondylitis Study

### 2.3.1 Cohort, Follow-up, and Dosimetry

Between 1935 and 1954 patients with ankylosing spondylitis in Great Britain and Northern Ireland received x-ray treatment in 1 of 87 radiotherapy centres. Mortality among 14,106 patients has been examined up to the end of 1982 by Darby et al. (1987). Patients were kept in the study until they had either died or received a second course of radiotherapy. Retreated patients were removed following 12 and 18 months retreatment for the assessment of leukemia and non-leukemia mortality, respectively. For retreated patients the average period of follow-up was 3.5 years and for other patients, 23.6 years.

Mortality information came from searches of death certificates in the National Health Service Central Registers. Expected deaths were estimated using age-, sex-, and calender-specific deaths rates for the population of England and Wales. By 1983 just over half the patients had been retreated (7,431), 3,175 had died, 346 had emigrated, 171 were not traceable, and 2,983 were still alive and living in the U.K..

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[13] This is not true at high doses where increased accuracy of DS86 dosimetry resulted in estimated doses being reduced by 60%.

In total, 727 cancer deaths were observed compared to 547 expected.

High doses were delivered to many organs as a result of irradiation to the spine in treatment of the disease. Monte Carlo calculations estimated average organ doses using the radiotherapy records of 903, 1 in 15, randomly selected patients. Typical doses were in the order of 5 Gy, 4 Gy, 2.5 Gy, and 2 Gy for the main bronchi, active bone marrow, stomach, and total body, respectively (Lewis et al 1988).

2.3.2 Results

Mortality among the cohort was reported separately for leukemia, colon cancer, and other cancers. Tables 2.4a and 2.4b summarize the observed and expected deaths by individual cancer site. Statistically significant increases were observed for leukemia (excluding chronic lymphatic leukemia) and cancers of the colon, oesophagus, lung, breast, bones (excluding jaw and nose), non-Hodgkin's lymphoma, and tumours of the central nervous system (other than spinal chord).

Leukemia

Observed leukemia deaths, excluding chronic lymphatic leukemia, were significantly elevated above the number expected in the general population. A total of 39 leukemia deaths were observed compared with 12.3 expected, a relative risk of 3.17 ($p$[13] < 0.001). Excess deaths were detected

[13] P-value: represents the probability that the observed excess is not due to random variation (see section 3.2 for further information).

within 2 years following treatment, peaked at about 5 and declined thereafter, but remained statistically elevated following 25 years at nearly twice the level of the general population (Darby et al 1987). Relative risks did not vary significantly with age at time of treatment, but were higher, though not significantly, for males (RR=3.43) than for females (RR=1.79).

Colon Cancer

Colon cancer deaths were significantly elevated by 30%, but may have been due to the association of spondylitis with an increased risk of ulcerative colitis (Darby et al 1987).

Cancers other than leukemia and colon cancer

For all cancers other than leukemia and colon cancer as a group, mortality was found to be 28% higher than expected in the general population (639 deaths observed compared to 499 expected). Relative risks were observed to first increase at about 5 years following exposure, peak at 10 to 15 years, and decline thereafter. After 25 years the excess risk was no longer statistically significant (RR=1.07 (0.92, 1.24 95% C.I.)). Relative and absolute risk projections models with adjustments for age-at-exposure and time-since-exposure fitted the data equally well (Muirhead and Darby 1989). Both the excess relative and absolute risk was observed to tail off following 25 years since first treatment. Lifetime cancer risk

projections for a male U.K. population following a hypothetical exposure to 0.1 Gy found excess lifetime risks to be 82 and 62 excess deaths per 100,000 persons for the relative and absolute projection models, respectively (see table 2.5).

The ankylosing spondylitis study is the first to show an eventual decline of the radiation-related risk for all cancers other than leukemia. Additional analysis has failed to find any artificial explanation for the tailing of the risk (Muirhead and Darby 1989). The condition of spondylitis has not appeared to have had an effect on the subsequent cancer risk. A study of a smaller group of patients who were not treated with x-rays showed that the number of observed cancer deaths were almost identical to that expected in the general population (Darby et al 1987).

Lung Cancer

Among the observed excess cancers at individual sites, lung cancer accounted for approximately 40% of the total. The excess lung cancer risk was observed to peak at around 17 years following first treatment and to decrease significantly thereafter, returning to normal levels after 25 years. The decrease in excess lung cancers over time appears to be the main reason for the overall decline of the excess risk for all cancers other than leukemia and colon cancer (Darby et al 1987).

Stomach Cancer

Early studies of the cohort found the risk of stomach cancer to be elevated in patients nine or more years following exposure (31 observed deaths compared with 20.1 expected) (NRC 1990). However in the most recent analysis (Darby et al 1987), there was no observed increased risk (64 deaths observed compared to 63.2 expected). This is in contrast to the risks observed among the Japanese A-bomb survivors where stomach cancer was the one of the most prominent excess cancers (see section 2.2). One possible explanation for the apparent discrepancy may involve the fact that spondylitics were a much older cohort (average age at treatment in mid-thirties) than the LSS cohort (NRC 1990). In addition, doses to the stomach were quite variable ranging from 0 to 5 Gy (Lewis et al 1988) and the lack of individual dose estimates does not allow the proper analysis of the dose-response. On the other hand, the results may suggest that the relationship between radiation and stomach cancer may be more complicated than previously believed (NRC 1990).

Tumours of the Brain and Central Nervous System

Tumours of the central nervous system (excluding spinal chord) were also significantly increased (RR=1.57 ($P < 0.05$)). Twenty-one of 22 observed tumours occurred in the brain even though the mean brain dose was estimated to be relatively low, under 0.15 Gy. It is thought that the increase

may be the result of secondary tumours from primary growths in the lung (Darby et al. 1987).

## 2.4 Study of Women Treated for Cancer of the Cervix

### 2.4.1 Cohort, follow-up, and dosimetry

Several studies have been conducted examining the mortality among women treated for cancer of the uterine cervix with radium implants or external radiotherapy. Initial studies in the 1950's examined the mortality experienced by 30,000 treated patients, since then the study has expanded to include over 150,000 women. Data on second cancers for these women have been taken from 19 population-based cancer registries and 20 clinics (where women were treated) across the world including Canada, Europe, and the U.S. (Boice et al. 1988).

A case-control study was chosen to evaluate the increased risk of second cancers among treated women. This design study was chosen because of the impracticalities of acquiring dose estimates for each of the 150,000 women. Instead doses were estimated only for those women who died of a second primary cancer as well as for a group of matched control women who had not yet developed a secondary cancer. A cohort of 4,188 women were identified with having a second primary cancer as a cause of death. They were matched by age with 6,880 other women (Boice et al. 1988).

The treatment with radium implants and external

radiotherapy resulted in substantial dose to the bladder, rectum, uterine corpus, large intestine, ovaries, and bone; moderate doses to the stomach, pancreas, gallbladder, and liver; and smaller doses to the lung, breast, brain, salivary gland, and thyroid. (Boice et al. 1984). Individual organ doses were estimated by phantom measurements using original radiotherapy records (NRC 1990). Average estimated doses (see table 2.6) were 2 Gy to the stomach, 7 Gy to the whole bone marrow, 20 Gy to the bone, 30-60 Gy to the rectum, an average of 0.11 Gy to the thyroid, 0.31 Gy to the breast, and 0.35 Gy to the lung (Boice et al. 1988).

2.4.2 Results

Table 2.7 summarizes the excess risks of second cancers associated with radiotherapy for cervical cancer. Statistically significant increased cancer mortality was observed for acute and chronic myeloid leukemia (RR=2.02), cancers of the stomach (RR=2.08), bladder (RR=4.05), vagina (RR=2.65), rectum (RR=1.83), and all female genital (RR=1.50) (Boice et al. 1988). Among women irradiated in adult life, excess risks were generally found to be higher in younger patients irradiated at ages between 35 and 55. Relative risks also tended to be highest following 20 or more years after treatment and the pattern of excess second cancers appeared to be consistent with an age-constant relative risk model (Boice 1988). Despite the large organ doses and the size of the

cohort, however, it is estimated that at most only 5% of all second cancers can be attributed to radiation therapy (Howe 1991a).

Leukemia

While initial studies failed to find any excess in leukemia mortality, the expansion of the study size revealed a two-fold increase in acute and myeloid leukemia. The estimated RR at 1 Gy was 1.7 and the absolute excess risk was 0.10 per $10^4$ PYGy (0.00, 0.31 90% CI). The relative risk was observed to decrease with increasing age at treatment and was greatest 1-4 years following irradiation (RR=8.9) and declined thereafter. The risk decreased for whole bone marrow doses above 4 Gy. Chronic lymphatic leukemia was not found to be elevated (RR=1.03 (0.3, 3.9)) (Boice et al. 1988).

Ovary Cancer

An overall reduction in mortality from ovary cancer was observed among the treated women (RR=0.45). The reduction was greatest within 1-4 years following treatment (RR=0.13), however among long-term survivors, there was an indication of a small, though not significant, radiation-related increase (RR=1.4 (0.3, 5.6)). Because the average dose to the ovaries was about 32 Gy, it has been suggested the low risk observed shortly after irradiation may have been due to the killing of premalignant ovarian tumour cells that would have developed into a detectable cancer within 5 years of therapy (Boice et

al. 1988).

Breast Cancer

Despite breast cancer being the most common second cancer observed among patients (953 cases), no overall association with radiation could be found (RR=0.88 (0.7, 1.1)). This was attributed to sterilization of the ovary and subsequent radiation-induced menopause. When a subgroup of women whose ovaries had been surgically removed were studied, the relative risk was observed to be elevated, RR=1.33 (0.6, 2.8) and there was a suggestion of a dose response (RRs of 1.0 at 0 Gy, 1.1 at 0.01-0.24 Gy, 1.3 at 0.25-0.49 Gy, and 1.4 at 0.50+ Gy) (Boice et al. 1988).

Cancers of the Lung and Stomach

The data for cancers of the lung and stomach observed in the cohort has been difficult to interpret. The relative risk of lung cancer was originally observed to be statistically increased following an average lung dose of 0.35 Gy (RR=3.7 ($p<0.01$)). However, it was discovered that patients treated with radiation tended to smoke more compared to the general population. When smoking was taken into account, the apparent excess no longer existed (NRC 1990). Cancer of the stomach has had just the opposite history. Early studies could find no excess in stomach cancer. Only 3 cancers were observed while an excess of 60 cancers were predicted based on the excess observed among atomic bomb survivors. The latest study

found a statistically significant two-fold increase in stomach cancer. The RR at 1 Gy was 1.69 (1.01, 2.25 90% CI) with an absolute excess risk of 3.16 deaths per $10^4$ PYGy (0.05, 10.4) (Boice et al. 1988).

Thyroid Cancer

Treated women were observed to experience at two-fold, though not statistically significant, increase risk of thyroid cancer following an average dose to the thyroid of 0.11 Gy. The dose-response showed a smooth trend in excess relative risk with increasing dose which is highly suggestive of a causal relationship. The excess relative risk was estimated to be 12.3 at 1 Gy and the excess risk approximately 7.6 per $10^4$ PYGy (Boice et al. 1988).

Bone Cancer

The observed incidence of bone cancer in the cohort strongly suggested a causal relationship with radiation treatment (Howe 1991a). A total of 16 bone cancers were observed. Nine occurred in the heavily irradiated pelvic region, only 2.5 cancers were expected. The total relative risk observed was 1.3 and reached three-fold for bone doses greater than 10 Gy (Boice et al. 1988). Data suggested a threshold of about 1 Gy (Howe 1991a). While most of the expressed bone cancers appeared 10 years or more following treatment, the observed increase occurred within the first ten years (RR=2.1) (Boice et al. 1988).

## 2.5 Massachusetts Women Tuberculosis Study

### 2.5.1 Cohort, follow-up, and dosimetry

The increase in the incidence of breast cancer has been examined among 4,940 female patients treated for tuberculosis in two Massachusetts sanatoriums between the years 1930 and 1956 (Boice et al. 1990). About half the women (2,573 patients) were treated for the disease by pneumothorax, a procedure requiring repeated monitoring by chest fluoroscopy of collapsing of the lung. The other half (2,367 patients) were treated by other means (Howe 1991a, NRC 1990).

Patients were followed using hospital records, death certificates, and periodically mailed questionnaires. By 1980, 97% of the cohort were deceased. A total of 234 breast cancer cases were observed among monitored and non-monitored patients after an average follow-up of 30 years (Howe 1991a, NRC 1990).

Women treated with pneumothorax received an average of 88 fluoroscopies over a period of 5 years (Boice 1988). Doses were reconstructed using Monte Carlo simulations based on information from medical records and interviews with subjects and their physicians. The estimated accumulated breast tissue dose ranged from 0.01 Gy to 6 Gy with a mean of 0.96 Gy (Howe 1991a) and a mean lung dose of 0.85 Gy (Davis et al. 1989).

## 2.5.2 Results

### Breast Cancer Incidence

In the 2,573 irradiated women there were 147 observed breast cancers in contrast to 113.6 expected, a standard incidence ratio (SIR) of 1.29 (1.1, 1.5 95% CI). No excess was found in women not treated with pneumothorax (87 cancers observed in contrast to 100.9 expected[15]) (Boice et al 1990, Howe 1991a).

Table 2.8 shows the excess breast cancer incidence among exposed women with increasing dose. The dose response was consistent with a linear model for doses up to 4 Gy, above which, the response flattened. After a ten year minimum latency period, the estimated relative risk at 1 Gy was 1.61 (Boice et al. 1990). The relative risk was highest for exposure ages between 15 and 19 years and decreased thereafter with no clear excess for exposure ages above 30 (Howe 1991a).

### Lung Cancer Mortality

The effect of fluoroscopy on the subsequent risk of lung cancer mortality was examined in an extended study of 6285 patients receiving an average of 77 fluoroscopies and followed for an average of 25 years. The mean accumulated dose to the lung was estimated to be 0.84 Gy (Davis et al. 1989). No excess lung cancer deaths were found when comparison was made with number of deaths expected in the U.S. general

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[15] Based on baseline incidence rates for Connecticut

population. Sixty-nine lung cancer deaths were observed, 86.3 expected. This produced a standard mortality ratio (SMR) of 0.80 (0.66, 0.97 90% CI). Despite a wide range of doses there was no evidence of a dose-response. Adjustments for smoking habits and the amount of lung tissue at risk also did not appreciably effect the findings.

It has been suggested that the lack of excess lung cancer mortality may be a consequence of an effect of the fractionation of doses delivered at high dose rates. However, it has been noted that this interpretation needs to be viewed cautiously (Darby 1991, Howe 1991a). First, no such effect was observed for the increased incidence of breast cancer. Second, it is not clear whether using national mortality rates to estimate the expected numbers of deaths for these patients is appropriate. However, a similar lack of excess lung cancer has recently been observed among Canadian fluoroscopy patients (Howe 1991b) (see section 2.6).

## 2.6 Canadian Women Fluoroscopy Study

### 2.6.1 Cohort, follow-up, and dosimetry

The Canadian Fluoroscopy Study is the largest study available on radiation exposure and subsequent breast cancer mortality (Howe 1991b, NRC 1990). The study includes 31,710 women with tuberculosis who, in the 1930s and 1940's, received multiple chest fluoroscopy in Canadian sanatoriums during the

treatment of the disease by pneumothorax.

Mortality among women has been monitored for the years 1950 to 1980 using a computerized record linkage to the Canadian National Mortality Data Base. By 1980, a total of 482 breast cancer deaths were observed after 867,541 women years of follow-up (NRC 1990). Women in Nova Scotia showed a significantly higher risk of breast cancer mortality compared to women treated in other provinces. As a result, the cohort has normally been analyzed separately for women treated in Nova Scotia, and women treated in other provinces.

It was not uncommon for patients to receive fluoroscopy every 2 weeks for up to 5 years or more (Boice 1988). Estimates of breast tissue dose have been made for all 31,710 women by phantom measurements and Monte Carlo simulations using information obtained from patient's medical records and interviews with physicians (Sherman et al. 1978). Only the breast and lung received substantial doses. Approximately one quarter of the women (8,380) received estimated breast tissue doses of 0.1 Gy or more with maximum doses being over 20 Gy (NRC 1990).

2.6.2 Results

Patients receiving doses above 0.1 Gy experienced 163 breast cancer deaths compared to 102 expected based on Canadian baseline rates. The SMR was significantly increased at 1.60 (1.37, 1.87 95% CI) (Howe 1991a).

Excluding women from Nova Scotia, the increase in breast cancer mortality with increasing dose was consistent with a linear dose-response. Table 2.9 shows the increase in risk had very strong dependence on age at exposure. The greatest increase was observed for women exposed between ages 10 and 14 years. In contrast, little increase in the risk of breast cancer mortality was observed for women exposed at ages over 30 (Howe 1991a).

As mentioned, there was a highly significant difference ($p<0.0001$) in the excess risks observed between women treated in Nova Scotia and women treated in other provinces. Table 2.10 shows the excess risks observed at 24 years following treatment for women who were treated at the age of 25. Breast cancer mortality for women in Nova Scotia was observed to be approximately three times higher compared to women treated in other provinces. It has been suggested that this difference may be due to a dose rate effect. Women in Nova Scotia were exposed in the anterior/posterior orientation which resulted in a greater higher dose rate to the breast tissue. While the results suggest a dose-rate effectiveness factor of about 3, the existence of a dose rate effect is questionable. The level and pattern of mortality among women treated in provinces other than Nova Scotia is similar to that seen among women A-bomb survivors who were exposed at even higher dose rates than women in Nova Scotia

(Darby 1991, Howe 1991a, NRC 1990).

Lung Cancer

Lung cancer mortality in this study cohort is currently being examined by the National Cancer Institute of Canada. Preliminary findings suggest an absence of excess lung cancer mortality (Howe 1991b). The observation is consistent with those observations made among women receiving fluoroscopy treatment for tuberculosis in Massachusetts (see section 2.5).

## 2.7 New York State Postpartum Mastitis Study

### 2.7.1 Cohort, follow-up, and dosimetry

A study was conducted of 601 women in New York State who were given radiotherapy for the treatment of acute postpartum mastitis[16] during the 1940s and 1950s. The study examined the relationship between irradiation of the breast and the subsequent increase in breast cancer incidence observed among the women. As a control group the study used 1,239 non-exposed women with mastitis not treated by radiotherapy as well as siblings of both irradiated and nonirradiated women (NRC 1990, Shore et al. 1986).

Case ascertainment was determined using mailed questionnaires. Any identified breast cancer was medically verified. After a follow-up period of up to 45 years, 115 breast cancers were identified, 56 among exposed women and 59

[16] A disorder causing inflammations or infections of the breast following childbirth or breast feeding.

among controls.

Treatment of the ailment consisted of 1 to 10 x-ray treatments of the infected breast(s) separated by one or more days. Individual breast tissue doses were estimated for all 601 women using information from original radiotherapy records. Two-thirds of the women received irradiation to only one breast. Dose estimates ranged from 0.6 up to 14 Gy with a mean breast dose of 3.7 Gy (Shore et al. 1986).

Breast cancer data was analyzed in terms of irradiation per breast rather than per person. Incidence rates of exposed subjects and non-exposed controls were compared to the expected rates estimated from the New York State Cancer registry (Shore et al. 1986).

2.7.2 Results

Standardized incidence ratios were significantly elevated for the exposed patients, non-exposed controls, and sibling controls (see table 2.11). Comparison of excess cancer rates between the irradiated cohort and the control group resulted in an age-adjusted relative risk of 2.2 (1.6, 3.0 90% CI) for women who had been irradiated. The fitted relative risk at 1 Gy was 1.4 (1.2, 1.7).

Investigation of the dose response at low doses was limited to doses above 0.6 Gy. The observed response for the relative risk was consistent with a linear model with a downturn in risk for doses above 5 Gy. As most treated women

were in the same age range (75 percent between ages 20 and 34), there was no observable variation of excess risk with age at irradiation. However, it was observed that women irradiated shortly after their first childbirth had a higher increase in risk than did irradiated women who have had two or more pregnancies (Darby 1991).

## 2.8 Israeli Tinea Capitis Study

### 2.8.1 Cohort, follow-up, and dosimetry

The Israeli tinea capitis study consists of 10,834 children who received x-ray therapy for tinea capitis (ringworm of the scalp) between 1948 and 1960 (Ron et al. 1989, 1988, Modan et al. 1989, Ron and Modan 1984). Children in the study were fathered by men who had immigrated to Israel from either Africa or Asia (mostly the middle East). The study made a distinction between child who were actually born in Israel and those who had immigrated to Israel. A control group was formed using 10,834 matched individuals from the general population and 5392 non-irradiated siblings. The latest study of thyroid cancer incidence was for the years 1960-1986 (Ron et al. 1989) while the latest study for cancer mortality was for the years 1960-1982 (Ron et al. 1988).

Thyroid cancers were identified only if children had under gone thyroid surgery in one of 22 possible hospitals in Israel. Cancer mortality was ascertained using computer

matching with the Israel Cancer Registry (Ron et al. 1988, Modan et al. 1989).

Dose estimates were calculated using patient treatment records and phantom measurements. It was assumed younger children received higher thyroid radiation doses because of a smaller gland size. Estimated thyroid doses ranged from 0.04 to 0.5 Gy with an overall estimate of 0.09 Gy (Ron et al 1989). Average doses to other organs were 0.30 Gy for the whole bone marrow, 0.016 Gy for the breast, and 0.1 Gy for the brain (Ron et al. 1988).

2.8.2 Results

Table 2.12 summarizes the excess cancer mortality and incidence observed among children in the study. Significantly increased cancers were observed for leukemia, cancers of the thyroid, head and neck, breast, and tumours of the brain and central nervous system (Ron et al. 1989, 1988, Modan et al. 1989).

Thyroid

The risk of developing thyroid cancer was highly elevated. Overall there were 98 thyroid tumours identified among exposed children (43 malignant and 55 benign) and 57 among population and sibling controls (16 malignant and 41 benign). The overall excess relative risk was 30 and 10 per Gy for malignant and benign thyroid tumours, respectively. The excess absolute risk was 13 and 14 per $10^4$ PYGy. Even for a

mean dose as low as 0.09 Gy, the increased incidence was four-fold (Ron et al. 1989).

The dose response was linear for both malignant and benign thyroid tumours with no evidence of non-linearity. The relative risk, following a 5 year minimum latency period, remained fairly constant with time since exposure and the absolute excess increased continually over the entire study period (Ron et al. 1989).

The level and pattern of risk depended strongly on sex, age-at-irradiation, and ethnic origin. Relative risks were similar between male and female irradiated children, but the excess absolute risk for malignant tumours was 10 times higher for females than for males (statistically significant). There was no significant sex difference in the excess risk for benign tumours.

Table 2.13 shows the relative risks at 1 Gy by ethnic group for children irradiated at ages under 5 and those irradiated between the ages of 5 and 14. For both ethnic groups, relative risks were significantly higher in younger age groups by a factor of about 2. For instance, malignant thyroid cancer in Israeli born children exposed under age 5 the relative risk at 1 Gy was 30 in contrast to a relative risk of 17 for older irradiation ages. The relative risk for non-Israeli born children was significantly higher than Israeli born by a factor of about 3. There were no ethnic

differences for benign tumours (Ron et al. 1989). The reason for the ethnic difference is not clear. There is no evidence of significant differences in the background rates by ethnic origin and genetic differences do not appear to account for the difference since all children were fathered by men who were either Asian or North African (Ron et al 1989). It has been suggested that non-Israeli born children may have been unknowingly treated for tinea capitis before immigrating or that the difference could originate from differences in lifestyle (NRC 1990).

Breast cancers

A recent extended follow-up of the cohort for the years 1982-1986 indicated for the first time a significant increase in breast cancer among irradiated girls. The mean breast dose for children treated between the ages of 5 and 9 was estimated to be 0.016 Gy (Modan et al. 1989). Previous to 1981, the breast cancer incidence was observed to be the same between the irradiated cohort and control groups (exposed: 12 cases, population control: 12 cases, sibling control: 6 cases). In the following years between 1982 and 1986 an additional 13 breast cancer cases were identified among the cohort while only 5 and 4 cases, respectively, were identified in the population and sibling controls. Statistically, this represents over a 2-fold (1.3, 3.8 95% CI) increase in the risk of breast cancer for irradiated girls (Modan et al.

1989).

It is difficult to say whether these results suggest a true increase in risk of breast cancer as a result of the girls' radiation exposure. First, the increase is based on a small number of observed cancers (exposed: 13 cases, controls: 9 cases). Second, it is suspected that controls may have experienced unusually low breast cancer incidence than would normally be expected. And third, dose estimates may be inaccurate. However, if results are depictive of a true increase, it may indicate a high radiosensitivity in young females or an indirect pituitary effect on the induction of breast cancer (Howe 1991a).

Other Cancers

Statistically significant increases in mortality were observed for leukemia and tumours of the brain and nervous system, head and neck, and all bone and connective tissues (see table 2.12) (Ron et al. 1988).

Following an average whole bone marrow dose of approximately 0.3 to 0.6 Gy, the increased relative risk for leukemia was observed to be 2.3 (1.0, 5.6 90% CI) and the estimated excess absolute risk was 0.9 per $10^4$ PYGy. Excesses occurred within five years following exposure (Ron et al. 1988).

A 2.5-fold (0.9, 7.4 95% CI) increase was observed for tumours of the brain and nervous system following a mean

intracranial dose of 1.5 Gy. The excess absolute risk was estimate as 0.15 per $10^4$ per PYGy and was observed to have a minimum latency of about 6 years (Ron et al. 1988).

For tumours of the head and neck, the RR was 2.9 (1.2, 7.2 90% CI) while for all bone and tissue carcinomas, the RR was 9.0 (1.3, 208 90% CI)) (Ron et al. 1988).

## 2.9 Rochester Thymus Study

### 2.9.1 Cohort, follow-up, and dosimetry

Between 1926 and 1957 it was common practice in Rochester New York to treat infants with supposedly enlarged thymus gland with external irradiation in order to shrink the glands. These infants, who were all under a year old at the time of treatment, have been study for increases in the subsequent risk of thyroid and breast cancer. A cohort of 2,652 irradiated infants were used to studied the subsequent thyroid cancer risk and a cohort 1201 irradiated infant girls were studied for an increase in breast cancer. Infants were followed for an average of 25 and 36 years in the thyroid and breast cancer studies, respectively. Controls in the respective studies consisted of 4,823 and 2,469 un-irradiated siblings (Shore et al. 1985, 1986, Hildreth et al. 1989).

Cancer cases were identified using mailed questionnaires and verified with patients physicians and hospitals. Only one thyroid cancer was identified among

controls. Because of this, thyroid cancer rates from the Connecticut Cancer Registry were used to calculate expected number of cases.

Infants received between 1 and 11 treatments. Patients irradiated in earlier decades of the procedure normally experienced higher thyroid doses (Howe 1991a). Rough estimates of thyroid doses ranged from below 0.05 Gy to about 11 Gy with 62% of irradiated infants receiving thyroid doses under 0.5 Gy. For those irradiated in earlier years the mean thyroid dose was 1.4 Gy while for later years the average was 1.2 Gy (Shore et al. 1985).

2.9.2 Results

Table 2.14 summarizes the thyroid and breast cancer risks observed among irradiated infants. In total, 30 malignant and 59 benign thyroid cases were reported among irradiated infants while only 1 malignant and 8 benign cancers were observed among controls (Shore et al. 1985, 1986). There were 22 breast cancer cases identified in irradiated infant girls and 16 cases in the girl controls (Hildreth et al. 1989).

Thyroid

A 45-fold (32, 61 90% CI) and 15-fold (8, 28) increase in the risk of malignant and benign thyroid cancer, respectively, was observed for the irradiated infants. Relative risks were similar between infant boys and girls, but

excess absolute risks were 2.5 times higher for girls compared to boys (5.3 compared to 2.1 per $10^4$ PYGy). The overall excess absolute risk averaged over both sexes was 3.5 per $10^4$ PYGy (Shore et al. 1985). An analysis was also performed that was restricted to infants whose thyroid doses were under 0.3 Gy (mean dose of 0.1 Gy). The absolute risk estimate for this group was found to be 14 per $10^4$ PYGy (Shore et al. 1986), comparable to that observed in the Israeli tinea capitis study (see section 2.8).

The pattern of excess incidence over time was described best by an age-constant absolute model. The relative risk was seen to decrease smoothly following 15 since the time of irradiation. The dose-response was fitted adequately with a linear model, although a linear-quadratic model could not be excluded (Shore et al. 1985).

Effect of fractionation of dose

A separately analysis has also been carried out to investigate the effect of dose fractionation on increased thyroid cancer risks for a subcohort of infants receiving total thyroid doses below 0.6 Gy. No effect on subsequent thyroid risks was found for differences in the dose per fraction, number of fractions, or time interval between fractions administered to infants (Shore et al. 1985).

Breast Cancer

A statistically significant increase in breast cancer

incidence has also been seen in irradiated infants who have been followed-up for at least 28 years (see table 2.13). Among the 1201 irradiated infant girls there were 22 observed breast cancers while only 16 cases were found among the 2469 sister controls. Following a mean breast dose of 0.69 Gy, the relative risk at 1 Gy was estimated to be 3.48 (2.1, 6.2 95% CI) with an absolute excess of 5.7 per $10^4$ PYGy (2.9, 9.5 95% CI). All but 10% of the irradiated girls were less than 6 months old when treated (Hildreth et al. 1989).

## 2.10 Other Studies

Studies providing additional information on the carcinogenic effects of low-LET ionizing radiation include:

- New York tinea capitis study,
- Late Effects Study Group of children irradiated for childhood cancer
- radiotherapy and diagnostic studies

### 2.10.1 New York Tinea Capitis Study

This study involved 2,215 children in New York State who were treated for tinea capitis by x-ray therapy between 1940 and 1960. A control group was formed using a 1,395 controls who had tinea capitis but did not receive irradiation. The average age of the children was around 8 years. Estimated average organ dose were 0.06 Gy for the thyroid, 4 Gy for the cranial marrow, and 4.5-8.5 Gy for the

scalp (Howe 1991a).

Cancers were statistically elevated risks for thyroid adenomas (8 vs 0), brain tumours (8 irradiated cases vs 0 control cases), and skin cancer (excluding melanoma) (41 vs 4) (Howe 1991a). Increased risks were also observed for leukemia, cancers of the salivary gland, breast, all bone and connective tissues, and central nervous system (Darby 1991). No excesses were detected for thyroid cancer (Howe 1991a).

The excess of skin cancer was not observed until after a minimal latency period of about 20 years and appeared to be greatly enhanced by exposure to ultra-violet radiation. Excesses were more prevalent among white children and occurred four times more often on the face than on the scalp. The estimated excess absolute risk was 0.56 and 0.12 per $10^5$ per $cm^2$ per Gy for the face and scalp, respectively (Darby 1991, Howe 1991a).

2.10.2 Late Effects Study Group

The late effects of radiotherapy and chemotherapy for childhood cancer were examined among children in the Late Effects Study Group who received cancer therapy in 1 of 13 medical centres in Canada, the U.S., and Europe between 1945 and 1979. The study group was comprised of 9,170 children who had survived childhood cancer for more than 2 years following therapy. Therapy included treatment for Wilm's tumour, Hodgkin's disease, retinoblastoma, neuroblastoma, and Ewing's

sarcoma (Tucker et al. 1984).

Children received doses ranging from a few Gy to tens of Gy depending on the treatment and cancer site. Table 2.15 summarizes the observed relative risks. Overall, there were 167 observed second cancers while only 11.4 were expected. Significantly increased cancers were leukemia, thyroid, breast, digestive system, bone, and connective tissue. The increase in bone cancer was seen only for doses over 10 Gy (Howe 1991a). Excess brain tumours was seen only for high cranial doses.

Gender did not appear to influence the subsequent risk of developing a second cancer, but age at therapy did. Children exposed at adolescent ages seemed to have a higher risk of osteosarcoma and younger children appeared more susceptible to thyroid cancer. For some cancers there was a suggested association between genetic susceptibility and radiotherapy of the first cancer. Retinoblastoma patients, for instance, are believed to have a thousand times higher chance of developing osteosarcoma (Tucker et al. 1984).

2.10.3 Other radiotherapy and diagnostic studies

Other studies of populations irradiated for diagnostic and therapeutic reasons are summarised briefly.

A statistically significant increase in breast cancer was observed among teenage girls aged 10 to 14 who were given frequent diagnostic x-ray examinations for spinal monitoring

in the treatment of scoliosis. After an average follow-up of 26 years there were 11 breast cancers observed while 6 were expected, giving a relative risk of 1.82 (1.0, 3.0 90% CI). Increased relative risks were only statistically significant for doses above 0.2 Gy (RR=3.4 (1.2, 7.8)), but were seen to increase smoothly with increasing dose at lower dose levels where the mean breast dose was 0.13 Gy (Darby 1991).

Significant increases in breast cancer were observed among adult women irradiated for the treatment of benign breast disease in Sweden (Boice 1988).

A number of excess cancers were reported among 15,336 infants in Sweden treated for skin haemangioma (birth marks) with either x-ray therapy or Ra-226 sources in flat applicators, needles, or tubes. A total of 224 cancers were observed with significant increases detected for cancers of the brain, bone and connective tissue, and breast. The relative risks for all cancers combined and breast cancer were 1.18 (1.03, 1.35 95% CI) and 1.65 (1.26, 2.13), respectively (Darby 1991).

Studies of patients irradiated for the treatment of Hodgkin's disease have found elevated cancers of the stomach, lung, breast, thyroid (adults), oral cavity, and connective tissue (Boice 1988).

Studies of patients treated for non-Hodgkin's disease have reported significant increase for cancers of the stomach

and connective tissue (Darby 1991).

Adolescents who were given radiotherapy for acne were reported to have significantly increased risks for benign head and neck conditions (Darby 1991).

Two case-control studies of men and women in Los Angeles County given full mouth dental x-rays and x-ray treatment to the head, reported a statistically significant increase for meningiomas. The odds ratio for women under the age of 20 was 4.1 ($p<0.01$) and for men under 20 it was 3.5 ($p<0.02$) (Darby 1991).

Table 2.1
Summary of the major human studies

| Population | Exposure History | Organ Dose | Exposure age | Statistically significant excess cancers |
|---|---|---|---|---|
| Japanese Atomic Bomb Survivors | External gamma and neutron from thermal nuclear explosion | 0 - 6 Gy | All ages Both sexes | leukemia, lung, breast, stomach, oesophagus ovary, urinary bladder, multiple myeloma |
| Ankylosing Spondylitis Patients | Treatment with x-rays to the spine during the years 1935 to 1954 in the U.K. | 1 to 5 Gy | Adult (Ave age: mid 30's) | leukemia, lung, breast, oesophagus, non-Hodgkin's lymphoma, and central nervous system |
| Women treated for cervix cancer | Radium implants and external gamma treatment | 2 to 60 Gy | Adult female (Ave age: 50) | leukemia, stomach, thyroid, bladder, kidney, bone, vagina, caecum, uterine corpus, rectum, non-Hodgkin's lymphoma - a reduction in breast cancer |
| Canadian and Massachusetts Tuberculosis Patients | Multiple fluoroscopy to monitor lung collapse | average of 1 Gy to the breast | All ages (early adult most common) | breast - no excess lung cancers |
| New York Postpartum Mastitis patients | External x-ray treatment for inflammation of the breast following childbirth | about 4 Gy to the breast | 20 - 35 | breast |
| Israeli Tinea Capitis Patients | External x-ray treatment for tinea capitis (ring worm of the scalp) | 0.04 - 0.5 Gy (thyroid) | 0 - 15 | thyroid, leukemia, skin, brain and nervous system, bone and connective tissue, and parotid gland |
| Rochester thymus patient | External x-ray treatment for supposedly enlarged thymus gland | about 1 Gy (thyroid) | infants (< 6 months) | thyroid and breast |

Table 2.2
Number of cancer deaths, estimated relative and absolute risks of cancer death in the Life Span Study cohort (DS86 dosimetry, shielded kerma, both cities, both sex, all ages at exposures, 1950-1985)
( Based on Shimizu et al. 1988, tables 2A, 2B, and 4)

| Site of cancer | Number of deaths in kerma dose group | | | Estimated RR at 1 Gy (kerma) | Excess risk per 10E04 PYGy (kerma) |
|---|---|---|---|---|---|
| | < 0.50 Gy | > 0.50 Gy | Total Deaths | | |
| ALL MALIGANT NEOPLASMS | 5181 | 755 | 5936 | 1.39 (1.32, 1.46) | 10.0 (8.36, 11.8) |
| LEUKEMIA | 130 | 72 | 202 | 4.92 (3.89, 6.40) | 2.29 (1.89, 2.73) |
| ALL EXCEPT LEUKEMIA | 5051 | 683 | 5734 | 1.29 (1.23, 1.36) | 7.41 (5.83, 9.08) |
| DIGESTIVE ORGANS | 2769 | 360 | 3129 | 1.24 (1.16, 1.33) | 3.39 (2.27, 4.59) |
| Oesophagus | 152 | 24 | 176 | 1.43 (1.09, 1.91) | 0.34 (0.08 0.67) |
| Stomach | 1776 | 231 | 2007 | 1.23 (1.13, 1.34) | 2.07 (1.19, 3.05) |
| Colon | 200 | 32 | 232 | 1.56 (1.25, 1.98) | 0.56 (0.26, 0.91) |
| Rectum | 198 | 18 | 216 | 0.93 ( , 1.27) | -0.07( , 0.25) |
| Liver, primary | 68 | 9 | 77 | 1.12 (0.87, 1.70) | 0.05 (-0.05, 0.25) |
| Gallbladder and bile ducts | 132 | 17 | 149 | 1.37 (0.98, 1.96) | 0.22 (-0.01, 0.53) |
| Pancreas | 172 | 19 | 191 | 0.98 ( , 1.27) | -0.10 ( , 0.20) |
| Other, unspecified | 71 | 10 | 81 | 1.32 (0.87, 2.14) | 0.11 (-0.05, 0.35) |
| RESPIRATORY | 652 | 95 | 747 | 1.40 (1.21, 1.63) | 1.29 (0.71, 1.96) |
| Lung | 554 | 84 | 638 | 1.46 (1.25, 1.72) | 1.25 (0.70, 1.89) |
| FEMALE BREAST (a) | 125 | 30 | 155 | 2.00 (1.48, 2.75) | 1.02 (0.53, 1.60) |
| CERVIX UTERI AND UTERUS (a) | 344 | 38 | 382 | 1.22 (1.01, 1.50) | 0.06 (0.04, 1.29) |
| Cervic uteri (a) | 79 | 11 | 90 | 1.43 (0.93, 2.30) | 0.26 (-0.04, 0.70) |
| OVARY (a) | 69 | 13 | 82 | 1.81 (1.16, 2.89) | 0.45 (0.10, 0.90) |
| PROSTATE (a) | 47 | 5 | 52 | 1.05 ( , 1.73) | 0.03 ( , 0.40) |
| URINARY TRACT | 107 | 26 | 133 | 2.02 (1.45, 2.87) | 0.55 (0.26, 0.89) |
| Kidney | 33 | 5 | 38 | 1.58 (0.91, 2.94) | 0.09 (-0.02, 0.26) |
| Bladder | 70 | 20 | 90 | 2.13 (1.40, 3.28) | 0.41 (0.16, 0.70) |
| MALIGNANT LYMPHOMA | 102 | 8 | 110 | 0.95 ( , 1.40) | -0.02 ( ,0.18) |
| MULTIPLE MYELOMA | 30 | 6 | 36 | 2.86 (1.55, 5.41) | 0.21 (0.07, 0.39) |
| OTHER | 805 | 102 | 907 | 1.20 (1.05, 1.38) | 0.77 (0.19, 1.44) |
| OTHER SITES | | | | | |
| Liver (b) | | | | 1.24 (1.06, 1.47) | 0.63 (0.17,1.18) |
| Tongue | 26 | 0 | 26 | 0.83 ( , 1.49) | |
| Pharynx | 20 | 3 | 23 | 0.83 ( , 2.04) | |
| Nose | 40 | 4 | 44 | 0.84 ( , 1.67) | |
| Larynx | 45 | 6 | 51 | 1.51 (0.95, 2.68) | |
| Skin cancer (except melanoma) | 29 | 2 | 31 | 1.17 ( , 2.47) | |
| Bone | 24 | 3 | 27 | 1.22 ( , 2.79) | |
| Brain tumours | 44 | 3 | 47 | 1.03 (0.51, 2.09) | 0.01 (-0.12, 0.20) |
| Tumours of CNS (except brain) | 11 | 3 | 14 | 3.09 (1.06, 9.74) | 0.19 (0.00, 0.24) |

( ) 90% confidence interval given in parentheses
(a) Risk estimation for these sites based on either males or females only
(b) Including not specified as primary cancer

Table 2.3
Oberved and expected number of cancer deaths for kerma doses above 0.5 Gy and relative risk and excess absolute risks for statistically significant sites. Both cites , both sex, all ages ATB (organ absorbed dose)
(Based on Shimizu et al. 1988; Tables 4, 2-3,4,6,7,8,15,16,19,21, and 23)

| Site of cancer | Number of deaths in kerma dose group > 0.5 Gy | | | Mean absorbed-organ dose (Gy) | Estimated RR at 1 Gy (organ-absorbed dose) | Excess risk per 10E04 PYGy (organ-absorbed dose) |
|---|---|---|---|---|---|---|
| | Observed | Expected | O/E | | | |
| All cancers | 755 | 537 | 1.41 (1.32, 1.49) | | | |
| Leukemia | 72 | 19 | 3.82 (2.18, 4.57) | 0.242 | 6.21 (4.83, 8.12) | 2.94 (2.43, 3.49) |
| All except leukemia | 683 | 518 | 1.32 (1.24, 1.40) | 0.223 (a) | 1.41 (1.32, 1.51) | 10.13 (7.96, 12.44) |
| Esophagus | 24 | 17 | 1.44 (1.03, 2.01) | 0.228 (b) | 1.58 (1.13, 2.24) | 0.45 (0.10, 0.88) |
| Stomach | 231 | 185 | 1.25 (1.12, 1.39) | 0.228 | 1.27 (1.14, 1.43) | 2.42 (1.26, 3.72) |
| Colon | 32 | 21 | 1.51 (1.13, 2.01) | 0.223 | 1.85 (1.39, 2.45) | 0.81 (0.40, 1.30) |
| Lung | 84 | 57 | 1.46 (1.22, 1.75) | 0.240 | 1.63 (1.35, 1.97) | 1.68 (0.97, 2.49) |
| Female breast | 30 | 14 | 2.12 (1.58, 2.85) | 0.240 | 2.19 (1.56, 3.09) | 1.20 (0.61, 1.91) |
| Ovary | 13 | 8 | 1.73 (1.10, 2.72) | 0.211 | 2.33 (1.37, 3.86) | 0.71 (0.22, 1.32) |
| Urinary tract | 26 | 12 | 2.13 (1.56, 2.93) | 0.231 | 2.27 (1.53, 3.37) | 0.68 (0.31, 1.12) |
| Multiple myeloma | 6 | 3 | 2.08 (1.08, 4.03) | 0.242 (c) | 3.29 (1.67, 6.31) | 0.26 (0.09, 0.47) |
| Tumours of CNS (except brain) | | | | | 3.09 (1.06, 9.74) (d) | 0.19 (0.00, 0.24) (d) |

( ) 90% confidence interval
(a) colon mean dose
(b) stomach mean dose
(c) mean bone marrow dose
(d) based on kerma dose

Table 2.4
Observed and expected deaths at age less than 85 years from cancers other than leukemia or colon cancer by cancer site and time since exposure among ankylosing spondylitis patients (Re-treated patients included for 18 months after re-treatment)
(Based on Darby et al. 1987, table IV)

| Cancer site | Time since first treatment (years) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | 5.0-24.9 | | | => 25 | | | => 5.0 | | |
| | O | E | O/E | O | E | O/E | O | E | O/E |
| Mouth | 2 | 1.2 | 1.68 | 1 | 0.7 | 1.41 | 3 | 1.9 | 1.58 (0.5, 4.8) |
| Pharynx | 3 | 1.7 | 1.78 | 1 | 0.9 | 1.14 | 4 | 2.6 | 1.56 (0.6,4.1) |
| * Oesophagus | 15 | 7.3 | 2.05 (b) | 13 | 5.4 | 2.41 (b) | 28 | 12.7 | 2.20 (1.53, 3.16) (c) |
| Stomach | 44 | 36.5 | 1.20 | 11 | 17.8 | 0.62 | 55 | 54.3 | 1.01 (0.8, 1.3) |
| Rectum | 16 | 14.0 | 1.14 | 8 | 8.3 | 0.96 | 24 | 22.4 | 1.07 (0.7, 1.6) |
| Liver | 2 | 3.5 | 0.58 | 4 | 2.0 | 2.01 | 6 | 5.4 | 1.10 (0.5, 2.4) |
| Pancreas | 14 | 12.4 | 1.13 | 7 | 8.2 | 0.86 | 21 | 20.5 | 1.02 (0.7, 1.5) |
| Larynx | 4 | 2.9 | 1.37 | 3 | 1.6 | 1.85 | 7 | 4.5 | 1.54 (.7, 3.2) |
| * Lung | 155 | 113.1 | 1.37 (c) | 69 | 71.4 | 0.97 | 224 | 184.5 | 1.21 (1.07, 1.38) (b) |
| * Breast | 21 | 11.2 | 1.88 (b) | 5 | 4.9 | 1.02 | 26 | 16.1 | 1.62 (1.11, 2.37) (a) |
| Uterus | 5 | 4.4 | 1.15 | 1 | 1.5 | 0.65 | 6 | 5.9 | 1.02 (0.5, 2.2) |
| Ovaries | 4 | 3.8 | 1.07 | 1 | 1.6 | 0.62 | 5 | 5.4 | 0.93 (0.4, 2.2) |
| Prostate | 12 | 9.7 | 1.24 | 9 | 8.5 | 1.07 | 21 | 18.2 | 1.16 (0.8, 1.7) |
| Kidney | 8 | 5.0 | 1.61 | 4 | 2.9 | 1.36 | 12 | 7.9 | 1.52 (0.85, 2.65) |
| Bladder | 9 | 9.9 | 0.91 | 11 | 6.8 | 1.62 | 20 | 16.7 | 1.20 (0.8, 1.7) |
| Skin | 3 | 2.4 | 1.23 | 2 | 1.3 | 1.52 | 5 | 3.8 | 1.33 (0.6, 3.2) |
| Spinal chord tumors | 1 | 0.2 | 6.67 | 0 | 0.1 | 0.00 | 1 | 0.2 | 4.76 (0.8, 28.1)) |
| * CNS tumours (excl. spinal chord) | 16 | 10.0 | 1.60 (a) | 6 | 4.0 | 1.49 | 22 | 14.0 | 1.57 (1.04, 2.37) (a) |
| Bone (excl. nose and jaw) | 3 | 1.0 | 2.94 | 1 | 0.3 | 2.94 | 4 | 1.4 | 2.94 (1.16, 7.48) (a) |
| Hodgkin's disease | 5 | 3.0 | 1.66 | 0 | 0.8 | 0.00 | 5 | 3.8 | 1.32 (0.6, 3.2) |
| * Non-Hodgkin's lymphomas | 13 | 4.5 | 2.90 (c) | 3 | 2.7 | 1.13 | 16 | 7.1 | 2.24 (1.39, 3.61) (b) |
| Multiple myeloma | 4 | 2.6 | 1.52 | 4 | 2.0 | 1.97 | 8 | 4.7 | 1.72 (0.87, 3.40) |
| Other neoplasms | 26 | 19.2 | 1.35 | 14 | 12.8 | 1.10 | 40 | 32.0 | 1.25 (0.95, 1.6) |
| TOTAL | 385 | 279.4 | 1.38 (c) | 178 | 166.6 | 1.07 | 563 | 446.0 | 1.26 (1.16, 1.37) (c) |

Table 2.5
Observed and expected deaths from leukemia at age less than 85 years by time since exposure among ankylosing spondylitis patients (Re-treated patients included for 12months after re-treatment)
(Based on Darby et al. 1987. Table IV)

| Type of Leukemia | Time since first treatment (years) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | 1.0-14.9 | | | => 15.0 | | | => 1.0 | | |
| | O | E | O/E | O | E | O/E | O | E | O/E |
| * Acute myeloid | 7 | 1.4 | 4.93 (c) | 10 | 2.9 | 3.42 (c) | 17 | 4.3 | 3.92 (2.5, 6.1) (c) |
| Acute lymphatic | 1 | 0.5 | 2.17 | 1 | 0.5 | 2.13 | 2 | 0.9 | 2.15 (0.6, 8.3) |
| Chronic myeloid | 3 | 0.7 | 4.62 (a) | 0 | 1.4 | 0.00 | 3 | 2.1 | 1.46 (0.5, 4.5) |
| Chronic lymphatic | 0 | 0.5 | 0.00 | 2 | 1.8 | 1.09 | 2 | 2.4 | 0.84 (0.2, 3.4) |
| Unspecified acute | 2 | 0.4 | 5.13 | 0 | 0.4 | 0.00 | 2 | 0.8 | 2.56 (0.7, 9.8) |
| Unspecified chronic | 0 | 0.0 | 0.00 | 0 | 0.0 | 0.00 | 0 | 0.0 | 0.00 |
| * Unspecified myeloid | 4 | 0.5 | 7.84 (b) | 0 | 0.2 | 0.00 | 4 | 0.7 | 5.63 (2.4, 13.4) (b) |
| * Unspecified lymphatic | 2 | 0.3 | 7.14 (a) | 1 | 0.1 | 10.77 | 3 | 0.4 | 8.11 (3.1, 20.9) (b) |
| * Unspecified leukemia | 3 | 0.1 | 25.00 (c) | 0 | 0.2 | 0.00 | 3 | 0.3 | 10.71 (4.3, 26.5) (b) |
| * All types | 22 | 4.4 | 5.01 (c) | 14 | 7.5 | 1.87 (a) | 36 | 11.9 | 3.03 (2.2, 4.1) (b) |

( ) 95% confidence interval
(a) $p < 0.05$
(b) $p < 0.01$
(c) $p < 0.001$
* Statistically significant

Table 2.6
Projected excess lifetime risk for all cancers excluding leukemia and colon cancer resulting from an exposure of 0.1 Gy to a male U.K. populatior (Based on Muirhead and Darby 1989. Table 13.4)

| Age at exposure | Excess deaths per 10E05 RR model | AR model |
|---|---|---|
| 15-24 | 17 | 16 |
| 25-34 | 44 | 56 |
| 25-44 | 101 | 103 |
| 45-54 | 149 | 101 |
| 55-64 | 135 | 49 |
| 15-64 | 82 | 62 |

Table 2.7
Excess cancer risks observed in the study of women treated with radiotherapy for cervix cancer
( Based on Boice et al. 1988. Table V and VI)

| Second cancers | Average organ dose (Gy) | Observed cases | Relative risk (matched) | Statistically significant excess risks | |
|---|---|---|---|---|---|
| | | | | Relative risk at 1 Gy | Excess absolute risk per 10E04 PYGy |
| HEAVILY IRRADIATED SITES (a) | | | | | |
| Small intestine | 10-20 | 22 | 1.00 (0.3, 2.9) | | |
| Colon | 24 | 409 | 1.02 (0.7, 1.6) | | |
| Cecum | 28 | 91 | 1.54 (0.7, 3.5) | | |
| * Rectum | 30-60 | 488 | 1.83 (1.2, 2.8) | 1.02 (1.00, 1.04) | 0.06 (0.00, 0.16) |
| * All female genital | 77 | 650 | 1.50 (0.9, 2.6) | 1.01 (1.00, 1.02) | 0.05 (-0.01, 0.16) |
| Uterus | 165 | 313 | 1.34 (0.8, 2.3) | | |
| Ovary | 32 | 309 | 0.45 (0.2, 1.0) | | |
| * Vagina | 66 | 105 | 2.65 (1.0, 6.3) | 1.03 (1.00, 1.08) | — |
| Other genital | 12 | 90 | 0.82 (0.4, 1.8) | | |
| * Bladder | 30-60 | 273 | 4.05 (1.9, 8.5) | 1.07 (1.02, 1.17) | 0.12 (0.04, 0.30) |
| Bone | 22 | 16 | 1.39 (0.3, 5.6) | | |
| Connective tissue | 7.0 | 46 | 0.67 (0.2, 1.9) | | |
| MODERATELY IRRADIATED SITES (b) | | | | | |
| * Stomach | 2.0 | 348 | 2.08 (1.1, 4.0) | 1.69 (1.01, 3.25) | 3.16 (0.05, 10.4) |
| Pancreas | 1.9 | 221 | 1.34 (0.7, 2.7) | | |
| Kidney | 2.0 | 148 | 1.23 (0.7, 2.2) | 1.71 (1.03, 3.24) | 1.10 (0.05, 3.50) |
| LIGHTLY IRRADIATED SITES (c) | | | | | |
| Breast | 0.31 | 953 | 0.88 (0.7, 1.1) | | |
| Thyroid | 0.11 | 43 | 2.35 (0.6, 8.7) | 12.3 (d) | 7.6 (d) |
| HEMATOLOGIC LEUKEMIA | | | | | |
| Chronic lymphatic | 6.7 | 52 | 1.03 (0.3, 3.9) | | |
| * Acute leukemia and chronic myeloid leukemia | 7.1 | 141 | 2.02 (1.0, 4.2) | 1.14 (1.00, 1.45) | 0.10 (0.00, 0.31) |
| Hodgkin's disease | 8.2 | 14 | 0.63 (0.2, 2.6) | | |
| Non-Hodgkin's lymphoma | 7.1 | 96 | 2.51 (0.8, 7.6) | | |
| Multiple myeloma | 7.1 | 54 | 0.26 (0.0, 2.6) | | |

( ) 90% confidence interval
a Dose > 3 Gy
b Dose > 1 Gy
c Dose < 1 Gy
d Not statistically significant
* Statistically significant

Table 2.8
Observed and expected breast cancer incidence and relative and absolute risks among Massachusetts women with tuberculosis receiving multiple chest x-ray fluoroscopy (Based on Boice et al. 1990)

| Radiation dose to breast (Gy) | Observed | Expected | Fitted relative risk | Fitted excess risk per 10E04 PY |
|---|---|---|---|---|
| 0 | 87 | 100.9 | 1.00 | 0.00 |
| 0.01-0.99 | 75 | 70.6 | 1.18 | 3.36 |
| 1.00-1.99 | 44 | 28.0 | 1.76 | 12.9 |
| 2.00-2.99 | 14 | 6.6 | 2.46 | 24.3 |
| >3 | 9 | 2.4 | 3.60 | 36.2 |
| Unknown | 5 | 6.0 | - | - |
| Total Exposed | 142 | 107.6 | 1.48 | 8.50 |

Table 2.9
Excess breast cancer mortality among Canadian women with tuberculosis receiving multiple chest x-ray fluoroscopy (a)
(Based on Miller et al. 1989)

| Age at first exposure | Relative Risk | Absolute excess risk per 10E04 PY |
|---|---|---|
| 10-14 years | 4.46 (1.12, 41) | 6.06 (0.25, 51) |
| 15-24 years | 1.77 (1.04, 7.1) | 3.05 (0.14, 18) |
| 25-34 years | 1.25 (1.01, 2.8) | 1.72 (0.08, 12) |
| > 35 years | 1.10 (1.00,2.22) | 1.23 (0.00, 15) |

(a) Women in Nova Scotia excluded

Table 2.10
Excess breast cancer mortality among Canadian fluoroscopy patients treated in Nova Scotia and other provinces (a)

| Province of treatment | RR at 1 Gy | Absolute excess risk per 10E04 per PYGy |
|---|---|---|
| Nova Scotia | 2.8 | 2.91 |
| Non-Nova Scotia | 1.53 | 2.59 |

(a) Breast cancer risks 24 years after treatment for women exposed at age 25

Table 2.11
Observed and expected number of breast cancers and standardized incidence ratios for women given radiotherapy in the treatment of postpartum mastitis (Howe 1991a)

| Group | Observed | Expected (a) | SIR (b) |
|---|---|---|---|
| Exposed (n=601) | 56 | 16.5 | 3.4 (2.8, 4.2) |
| Controls (n=1239) (c) | 59 | 36 | 1.6 (1.3, 2.0) |

( ) 90% confidence interval
(a) Based on NY State Cancer Registry
(b) Standardized incidence ratio
(c) Non-exposed patients and sibling controls

Table 2.12
Observed relative risk by cancer site among Israeli children receiving x-ray irradiation for the treatment of tinea capitis
(Based on Modan et al. 1989, table1, Ron et al. 1989, table II, and Ron et al. 1988)

| Cancer site | Exposed (10,834 children) | Nonexposed (a) (16,226 children) | Relative Risk (95% CI) |
|---|---|---|---|
| Thyroid (malignant) (b) | 43 | 16 | 4.0 (2.3, 7.9) |
| Thyroid (benign) (b) | 55 | 41 | 2.0 (1.3, 3.0) |
| Total cancers except thyroid | 49 | 44 | 1.7 (1.1, 2.5) |
| Head and Neck | 20 | 7 (c) | 2.9 (1.2, 7.2) |
| Bone and connective tissue | 6 | 1 | 9.0 (1.3, 208) |
| Lymphoma | 6 | 10 | 0.9 (0.3, 2.5) |
| Leukemia | 14 | 9 | 2.3 (1.0, 5.6) |
| Breast (<1982) | 12 | 18 | 1.0 -- |
| Breast (1982-1986) | 13 | 9 | 2.3 (p<0.01) |

(a) Sibling (5,392 children) and population (10,834 children) controls
(b) Incidence
(c) population controls

Table 2.13
Relative risk for the increase incidence of thyroid cancer among Israeli children treated for tinea capitis by age at exposure and ethnic group
(Based on Ron et al. 1989. Table VI)

| Age at exposure | Relative risk at 1 Gy | | |
|---|---|---|---|
| | Malignant tumours | | Benign Tumours |
| | Israeli born | Non-Israeli born | Ethnic groups combined |
| <= 5 | 30 | 77 | 43 |
| 5 - 14 | 17 | 37 | 21 |

Table 2.14
Thyroid and breast cancer incidence among infants irradiated for supposedly enlarged thymus glands
(Howe 1991a, Hildreth et al. 1989, Ron et al. 1989)

| Cancer | Observed | Expected | Relative Risk | Relative Risk at 1 Gy | Excess absolute risk per 10E04 PYGy |
|---|---|---|---|---|---|
| Thyroid (all doses) | 37 | - | 49.1 | - | 3.5 |
| Thyroid (doses<0.3 Gy) | - | - | 12.9 | - | 14 |
| Female breast (a) | 22 | 7.7 (b) | 2.8 (1.9, 4.1) | 3.48 (2,1, 6.2) | 5.7 (2.9,9.8) |

( ) 95% confidence intervals
(a) Hildreth et al. 1989
(b) Based on 16 cases observed among 2,469 controls

Table 2.15
Observed and expected number of second cancers among children of the Late Effects Study Group surviving 2 or years after therapy for childhood cancer (Based on Tucker et al. 1984. Table 3)

| Second Cancer | Observed | Expected | O/E |
|---|---|---|---|
| All | 167 | 11 | 15 (13, 17) |
| Buccal cavity | 5 | 0.2 | 31 (10, 73) |
| Digestive | 12 | 0.3 | 38 (20, 67) |
| Bone | 48 | 0.4 | 133 (96, 176) |
| Connective tissue | 20 | 0.4 | 41 (24, 67) |
| Breast | 5 | 0.3 | 12 (3, 31) |
| Genitourinary | 7 | 3.9 | 1.8 (0.7, 3.7) |
| Brain | 14 | 0.9 | 15 (8, 26) |
| Thyroid | 23 | 0.4 | 53 (34, 80) |
| Leukemia | 22 | 1.5 | 14 (9, 22) |
| Other | 11 | 3.1 | 3.6 (1.8, 6.4) |

( ) 95% confidence interval

Table 2.16
Studies of cancer in patients irradiated for therapeutic or diagnostic reason
(Based on Darby 199[illegible], tables 4, 5, and 6)

| Original condition | Age at exposure | Statistically significant excess cancer |
|---|---|---|
| Acne | All ages<br>(teen-age exposures common) | Parotid gland tumours |
| Benign head and neck disease | Children<br>(up to age of 15 years) | Thyroid, salivary gland, neural tumours |
| Benign breast conditions | Adults<br>(average age: approx. 30 years) | Breast |
| Benign gynaecological disease | Adults<br>(median age: late 40s) | Leukemia, pelvic sites;<br>reduction for breast in some populations;<br>no bone excess |
| Benign Skin conditions | All ages | Skin (excluding melanoma) |
| Breast Cancer | Adults<br>(Median age: mid-50s) | Connective and soft tissue, non-Hodgkin's lymphoma, lung, uterus, second breast, bone;<br>no leukemia excess |
| Excessive dental x-rays | All ages | Brain, parotid gland tumours |
| Excess diagnostic x-rays | Adults all ages | Leukemia |
| Hodgkin's disease | All ages<br>(median age: 30 years) | Stomach, thyroid, oral cavity, lung, breast, leukemia, bone |
| Non-Hodgkin's lymphoma | All ages<br>(average age: approx. 60 years) | Leukemia, rectum, multiple myeloma |
| Ovarian cancer | Adults<br>(median age: mid-50s) | Colon, bladder, rectum, connective tissue;<br>no leukemia excess |
| Prevention of deafness | Children<br>(Median age: 5-9 years) | Head and neck tumours |
| Skin haemangioma | Children<br>(up to age 20 but median age 6 months) | Breast, soft tissue |
| Testicula cancer | Age range 15-84 years<br>(average age: 37 years) | Tumours in urinary and gastro-intestinal tracts |
| Uterine corpus cancer | Adults<br>(Average age: aprox. 60 years) | Leukemia, rectum, multiple myeloma |

# 3.0 Sources of Uncertainty

## 3.1 Introduction

The epidemiological studies reviewed in chapter 2 demonstrate that low-LET ionizing radiation delivered externally at sufficiently high doses and dose rates can increase the subsequent probability of cancer mortality at most organ and tissue sites in the body. However, using the results of these studies to predict the lifetime increase risk of cancer mortality associated with low-level radiation involves many unresolved issues and uncertainties that limits the accuracy and confidence on such predictions. This chapter discusses the uncertainties arising from:

(1) sampling variation;

(2) bias;

(3) random error in A-bomb dosimetry;

(4) extrapolating effects to low doses and low dose rates;

(5) risk modelling; and

(6) transfer of excess risks between populations.

Discussion will be focused on the use of cancer mortality data from the Life Span Study of the atomic bomb

mortality data from the Life Span Study of the atomic bomb survivors.

## 3.2 Sampling Variation

Sampling variation refers to the statistical error in the measured cancer risk caused by expected variations in the normal expression of cancer in the population. It is a significant source of uncertainty in most epidemiological studies. For any increase in cancer caused by exposure to radiation, or any other carcinogen, to be detected, the increase in risk must at least be as large as the statistical error.

The statistical error in measured cancer risks is usually given in terms of a confidence interval (CI). The CI gives the range of risks that would be expected to be measured as a consequence of natural variation of the number of cancers occurring in a population. A 95 percent CI, for instance, would give the range of risks that would be expected to be measured in 95 of 100 identical populations. The statistical error in observed excesses, or deficiencies, in cancers in a population can also be given in terms of a "p-value". The p-value gives the probability that an excess, or deficiency, is not due to statistical error. An increase, or decrease, is considered "statistically significant" if the 90 percent confidence interval for the relative risk does not include the value of 1 or if the p-value is less than 0.1.

The effect of sampling variation places severe limitations on the design of epidemiological studies and on conclusions that can be made from results regarding the effects of radiation exposure. Variations in the cancer risk measured in a population from year to year is expected to follow a Poisson distribution (Dolson and Gaudette 1987, Ahlbom and Norell 1984). The statistical error in the observed cancer risk in any particular year will therefore be approximately proportional to the inverse of the square-root of the number of cancers observed in that year. The only way to reduce the statistical error is to increase the number of cancers in the population. This could be achieved by increasing the size of the population, however most irradiated populations available for study are of fixed size. Therefore, the only alternative way of detecting an increase risk of cancer following radiation exposure is to study populations who receive sufficiently high organ doses so that any subsequent increase in cancer will be large enough to detect. The difficulty to distinguish small increases from normal statistical variations is the main reason why low dose studies, which are of limited population size and involve very small radiation doses, are limited in their ability to demonstrate significant associations between cancer and low-level radiation exposure. Instead, inferences of low-level radiation risks must rely on the effects observed in high dose

studies, such as the Life Span Study of atomic bomb survivors.

## 3.3 Bias

Another important source of error in the cancer risk measured by epidemiological studies is bias. Bias refers to any feature of the design, execution, or analysis of the epidemiological study that introduces a systematic error that can cause the measured risk to artificially deviate from the "true" risk. Bias arises mainly because epidemiological studies are observational in nature. Since exposures do not occur under ideal experimental conditions, there is a possibility that other factors not directly related to the exposure can affect the subsequent identification of excess cancers. If the study cannot control for these factors, bias can be introduced and the measured risk will never equal the actual risk no matter how small the uncertainty due to sampling variation may be (Darby 1991, Howe 1991a).

There are three general categories of bias:

(1) selection,

(2) information, and

(3) confounding.

### 3.3.1 Selection Bias

Ideally, it is desirable that the cohort under study be a randomly selected group representative of the population. However this is not usually the case since there is always

some underlying reason why a population was exposed or why some individuals are included and others excluded from a cohort. This may introduce bias (Howe 1991a).

For instance, the Life Span Study cohort are a unique group in that they had survived a situation of high mortality. In some sense, the cohort may represent the hardiest and healthiest persons living in Hiroshima and Nagasaki at the time of the bombings (the possible effect of this is discussed below). There is also doubt whether male survivors are depictive of the average Japanese man. Because it was a time of war in 1945, most young males were away in military service. The young men that were present probably had some health reason for not being in the military (Howe 1991a). The influence this might have on the subsequent cancer risk in the male cohort is not clear. In studies of medically exposed populations there is also a potential for selection bias. Irradiations always resulted as a consequence of the medical treatment for some pre-existing disease or health condition. It is difficult to know how the original disease, or the effect of other treatments, may influence a cohorts susceptibility to developing cancer (Jablon 1984).

Healthy Survivor Effect

Stewart and Kneale (1990, 1989, 1984) have argued that the Life Span Study cohort is not a representative selection of the populations present in Hiroshima and Nagasaki at the

time of the 1945 bombings. Except for those survivors incurring bone marrow damage at higher doses, Stewart and Kneale believe the cohort would have a much stronger immune system than the population present before the bombings and experience fewer deaths from infectious disease. Presuming survivors would also have a greater immunity for cancer induction, Stewart and Kneale suggest that this so-called "healthy survivor effect" will result in a smaller number of radiation-induced cancers being expressed in the cohort.

To provide evidence of a selection bias, Stewart and Kneale examined the dose response for infectious disease for the years 1950-1982 using T65D dosimetry. If a bias did exist, they postulated that the dose-response should be U-shaped. Disease rates being lower for moderately exposed survivors where the selection effect would be greatest, and gradually return to normal for higher exposed individuals because of the compensating effect of the increase injury to the immune system caused by radiation damage to the bone marrow. Their analysis showed that a U-shaped response did exist for all non-cancer causes of death, excluding cardiovascular disease, combined as a group. They therefore concluded that radiogenic risk estimates based on the atomic bomb survivors underestimates the "true" radiation risk.

Other investigators have dismissed Stewart and Kneale's interpretation of the results of their analysis. The

Committee of the BEIR V report found that lower death rates were due to a reduction of a variety of diseases not just infectious ones[17]. More recently, Little and Charles (1990) conducted their own analysis, but used DS86 dosimetry as well as T65D dose estimates. It was concluded that the response observed by Stewart and Kneale may in part be an artifact of the T65D dose estimates. In addition, when analysis was restricted to only tuberculosis there was no evidence of a U-shaped response.

### 3.3.2 Information Bias

Misclassification of disease or loss of individuals from a cohort can introduce information bias into the results of epidemiological studies. The potential of information bias is minimized if the exclusion or loss of persons is random, and if the accuracy of identifying the cause of death is the same for both the cohort and control individuals (Howe 1991a).

The effect of information bias on the measured risk depends on the study design and the statistical models used to describe the data. Cohort studies employing relative risk models are usually less susceptible to information bias than ones employing absolute risk models (Howe 1991a, NRC 1990).

### 3.3.3 Confounding

Confounding refers to the effect on the measured

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[17] The Committee's findings were described in Howe 1991a

cancer risk caused by an intermediate factor associated with both exposure to ionizing radiation and the subsequent health effect. The consequence of not identifying and adjusting for the effects of confounders is the potential of wrongly attributing any increase in risk to the effects of radiation exposure (Howe 1991a).

The greatest potential confounder for radiation exposure is the effect of cigarette smoking on the risk of lung cancer. For instance, the relative risk of lung cancer was found to be significantly higher for female rather than male survivors in the LSS cohort. However, when adjustments were made for differences in smoking habits, there was no longer a significant difference (Shimizu et al. 1988). Unfortunately, most epidemiological studies have little or no information on the smoking habits of cohort subjects (Darby 1991).

## 3.4 Random Errors in A-bomb Dosimetry

Under the T65D dosimetry system, some survivors were estimated to have received doses in the range of 6-10 Gy. Few, if any, would have been expected to survive the acute effects associated with such high doses. Under the DS86 system, the upper range of doses has been reduced, but remains high at 4-6 Gy.

The high dose estimates are due mainly to

nonsystematic error (random error) caused by inaccuracies in the information regarding the location and shielding of survivors. The net effect of the random error is to introduce systematic bias in cancer risk estimates and distortion of the shape of the dose-response curve (Pierce and Vaeth 1991, 1989, Pierce, Stram, and Vaeth 1990). To minimize the effect, most analyses of the LSS cohort excludes survivors with estimated doses above 4 Gy (NRC 1990, Shimizu et al. 1988). However, few analyses actually compute the uncertainty introduced into the estimated risk.

One analysis that has examined the variation in cancer risk estimates caused by random error in dosimetry was performed by Pierce, Stram, and Vaeth (1990). The random error was modelled by assuming 35% error in doses on a log scale[18]. Using a constant relative risk model, the effect was examined for the increased risk in all cancers other than leukemia as a group. Analyses were perform with and without restricting doses to under 4 Gy. It was found that random error caused the excess cancer risk to be underestimated by 5% when the doses were restricted to under 4 Gy and underestimated by 15% when doses were not restricted.

## 3.5 Extrapolation to Low Doses and Low dose Rates

The investigation of dose and dose rate effects in

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[18] i.e. the error in dose estimates will be greater at higher doses

humans is hindered by statistical errors in cancer data, inaccurate dose estimates, little information on dose rate effects, and a shortage of reliable information for doses under 0.2 to 0.5 Gy. Present knowledge and understanding on possible effects is therefore based mostly on radiobiology and information taken from cellular and animal experiments. These strongly suggest the dose-response for low-LET radiation is best described by a linear-quadratic model and that doses delivered at low dose rates are less effective in inducing cancer than at high dose rates. Animal studies indicate reductions of 2 to 10 in the risk per unit dose of low-LET radiation exposure when dose rates are reduced.

The current theories from radiobiology and evidence in animal experiments, and indications in human studies, of a dose and dose rate effect are reviewed in further detail below.

### 3.5.1 Information from radiobiology

Theories of the mechanisms of carcinogenesis suggest the development of cancer is comprised of three stages:

(1) initiation stage: modification of a normal cell,

(2) promotion stage: promotion of a modified cell to a cancer cell, and

(3) progression stage: progression of a cancer cell to a full tumour.

The effect of ionizing radiation on carcinogenesis is

believed to take place in the initiation phase. It is generally accepted that initiation can be caused by direct or indirect radiation damage to the DNA. Direct damage includes single and double DNA breaks by discrete tracks of charged particles traversing a cell. Indirect damage is caused by the production of highly reactive free radicals by radiation which in turn, react chemically with base molecules in the DNA. This changes the chemical make-up of the DNA. The possible effect of ionizing radiation in the promotion and progression stages is still not understood (Cox 1991).

The probability that a cell will be modified depends on whether DNA damage is caused by way of a single-track or multiple-track process. In a single-track process, DNA damage is caused by a single particle track passing through a cell. The dose-response is expected to be linear with dose and, provided that tracks act totally independently of each other, independent of dose rate. In addition, no threshold for an effect is expected because even one particle track has at least some probability of modifying the cell since the repair of damage is not always 100 percent efficient (Bennison 1991, Strather and Goodhead 1991). In a multiple-track process, damage is caused by two or more particle tracks. The response is expected to be quadratic or proportional to the square of the dose and, since DNA repair can occur in the time interval

between consecutive tracks, dependent on dose rate[19] (Strather and Goodhead 1991, ICRP 1991, NRC 1990, NCRP 1980). Taking into account the additional probability of cell death, the general expression of the dose-response is of the form

$$I(d) = (a_1 d + a_2 d^2)\ e^{-(b_1 d + b_2 d^2)} \qquad (1)$$

where

$I(d)$ is the initiation rate at dose d;

$a_1$ is the probability of induction per unit dose (linear term) and is independent of dose rate;

$a_2$ is the probability of induction per unit dose square (quadratic term) and is dependant on dose rate;

$e^{-(b1\ d + b2\ d2)}$ is the probability of cell inactivation, or death; and

$b_1$ and $b_2$ are probability of cell inactivation per unit dose and per unit dose square, respectively

Assuming that the probability of cancer induction is directly proportional to the number of initiated cells, the dose-response for radiation-induced cancers will also be expected to linear-quadratic.

Figure 3.1 depicts the dose-response relationships expected for high linear energy transfer (LET) radiation (e.g.

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[19] Cells are able to repair both single and double stand breaks in DNA over a period of a few hours (Bennison 1991, Strather and Goodhead 1991)

slow neutrons and alpha particles) and low-LET radiation (e.g. gamma rays and beta particles). For high-LET radiation, single-track processes are expected to be predominant over most of the dose range (i.e. $a_2$=0) and the dose response should therefore be linear and independent of dose rate. A flattening of the response is expected at higher doses as a result of cell-killing. For low-LET radiation, single-track processes are expected to be predominant at lower doses and multi-track processes at higher doses (i.e. $a_1$>0 and $a_2 \geq 0$). Therefore the response should be linear and independent of dose rate at lower doses and curve upwards at higher doses. At very high doses a cell-killing effect is also expected. As dose rates are reduced, the response will become linear over the entire dose range as response at high doses approaches that of low doses as multi-track processes become to resemble single-track processes (i.e. $a_2$->0) (see figure 3.2). Therefore the effect of reducing doses and/or dose rates on the probability of initiation should be the same (ICRP 1991, Strather and Goodhead 1991, NCRP 1980).

3.5.2 Dose and dose rate effectiveness factor

The general approach used for estimating cancer risks at low doses and dose rates is to extrapolate linearly and then apply a correction factor to correct for the dose and dose rate effect. The correction factor has been called various names including dose rate effectiveness factor (DREF),

dose and dose rate effectiveness factor (DDREF), linear extrapolation overestimation factor (LEOF), and low dose extrapolation factor (LDEF). This report will use the term DDREF.

Use of the DDREF has the advantage in that dose response, as well as dose rate, information from a wide range of sources can be used to evaluate the reduced biological effectiveness at low doses of low-LET radiation. In terms of the dose-response, the DDREF can be thought of as the ratio of the risk per unit dose observed at high doses to the risk per unit dose observed at low doses (DDREF = Slope A/Slope B, see figure 3.2). By mathematically fitting a linear-quadratic model to experimental or human data, the DDREF can be given by the linear and quadratic terms in the expression for dose-response by

$$DDREF = 1 + \theta\, d_o \qquad (2)$$

where

$\theta$ is the ratio $a_2/a_1$, where $a_1$ is the linear term and $a_2$ is the quadratic term; and

$d_o$ depends on the range and distribution of doses

The inverse of $\theta$, $a_1/a_2$, represents a "crossover" dose above which the quadratic term dominates and below which the linear term dominates (Pierce and Vaeth 1989a).

3.5.3 Information from experimental studies

Most dose and dose rate information comes from

experiments with cellular systems and animals. Cellular systems indicate that the dose-response for most biological endpoints, such as mutations, chromosomal aberrations in mammalian cells, and cell transformation, are best described by a linear model for high-LET radiation and a linear-quadratic model for low-LET radiation, with an exponential cell-killing term at high doses for both radiation types (Strather and Goodhead 1991, ICRP 1991, NCRP 1980).

Animal studies demonstrate a dose rate effect on radiation-induced life shortening and tumour induction. Life shortening in mice is observed to be reduced with increasing protraction of dose from low-LET radiation. Assuming life shortening is mainly due to increased tumour induction, reductions are consistent with a dose rate effectiveness factor between 2 and 5 (Strather and Goodhead 1991, ICRP 1991, UNSCEAR 1986). Animal studies show the rate of tumour induction for a given dose of low-LET radiation delivered at high dose rates can be reduced by 2 to 10 times by lowering the dose rate[20] (see table 3.1) (Strather and Goodhead 1991, UNSCEAR 1988, NCRP 1980). These studies also demonstrate the difficulty in resolving the linear and quadratic terms in the dose-response observed in statistical data. Although a study may have revealed a dose rate effect, the dose-response observed at high dose rates would normally be consistent with

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[20] Values vary depending on the exposure conditions, animal strain, tissue/tumour type, and dose range.

a linear model (Strather and Goodhead 1991).

### 3.5.4 Information from human studies

#### Atomic bomb survivors

Pierce and Vaeth (1991) have estimated using atomic bomb survivor cancer mortality data the range of dose and dose rate effectiveness factors that could be considered plausible given the statistical errors that exists in cancer observations and the inaccuracies in individual dose estimates. Age-constant relative risk models were fitted to cancer mortality data separately for leukemia and for all cancers except leukemia combined as a group[21], using a linear-quadratic response function[22]. This was done for the case when allowances are and are not made for uncertainties in the cancer data and dose estimates. Possible values of the DDREF were estimated from the response using the equation DDREF = $1 + \theta d_0$ (see above section).

Figure 3.3 shows the observed dose-response for the excess relative risk of leukemia and all cancers except leukemia when no corrections for uncertainties are made. The leukemia response was best fit by a linear-quadratic function which had an inherent DDREF of 2. For other cancers, the response was best describe by a linear function, with a DDREF

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[21] Excess relative risk averaged over city, sex, and age at time of the bombings

[22] The intestinal dose equivalent (neutron 10) was used and the range of doses restricted to under 4 Sv.

equal to 1. When uncertainties in the data and doses were allowed, the best estimate of the DDREF for leukemia was 2.2 with values of up to 5 consistent with the data. For all cancers except leukemia as a group, plausible DDREFs consistent with the data ranged from 1 to 2.5, depending on the chosen level of random error in dose estimates which was allowed to range up to 40% (on the log scale).

Studies of medically irradiated populations

Studies of populations irradiated for medical reasons provide some site-specific information on the increase risk of cancer for doses below 0.5 Gy, fractionation of high dose rate exposures, and possible dose rate effects.

Women in Massachusetts and Canada treated with pneumothorax for tuberculosis received an average of 88 fluoroscopies over a period of five years. In both studies, there was no evidence that fractionation had an effect on the subsequent increase in breast cancer. However, a lack of excess lung cancers have been reported for both cohorts (Darby 1991, Howe 1991b).

In the Canadian study, it has been suggested that the higher risk of breast cancer mortality experienced by women treated in Nova Scotia compared to women treated in other provinces, may be due to a difference in the dose rate delivered to the breast. The difference in the subsequent breast cancer risk could possibly be interpreted as suggesting

a DDREF of about 3. However, such interpretation must be reviewed cautiously since excesses were similar to that seen among female atomic bomb survivors (Darby 1991, Howe 1991a, NRC 1990).

In the study of infants treated with radiotherapy to shrink supposedly enlarged thymus glands, there was no evidence of a fractionation effect on the subsequent risk of thyroid cancer when data was analyzed by dose per fraction, number of fractions, or time interval between fractions (Shore et al. 1985). However, an analysis did show an increased incidence of thyroid cancer with increasing dose for doses below 0.3 Gy (Shore et al. 1986).

The study of Israeli children has shown the thyroid and breast to be highly radiosensitive at doses below 0.1 Gy. A four-fold increase in the incidence of thyroid cancer was observed for a mean thyroid dose of 0.09 Gy (Ron et al. 1989) and a two-fold increase in breast cancer observed following a mean breast cancer dose of 0.016 Gy (Modan et al. 1989). In the study of children irradiated for tinea capitis in New York State children received a mean thyroid dose of 0.06 Gy. While there was a significant increase in thyroid adenoma, no increase in thyroid cancer has yet been detected (Howe 1991a).

A possible DDREF of 3 or more has also been suggested for radiation-induced thyroid cancer in a study of patients in Sweden given I-131 for diagnoses of hyper-active thyroids or

thyroid cancer. The subsequent number of excess thyroid cancers was about 3 times smaller than would be expected based on the excess risk observed in the Israeli tinea capitis study. This was originally attributed to the thyroid dose from I-131 being delivered at a low dose rate. However, it appears the excess is not attributable to radiation, but isolated to only patients who were originally suspected of having thyroid cancer (Holm et al. 1989, 1988, Strather and Goodhead 1991).

3.5.5 Summary

Radiobiology and experimental studies support the use of a non-threshold linear-quadratic model for extrapolating the effects of low-LET radiation to low doses. The rate of leukemia and tumour induction in animals following a given radiation dose delivered at a high dose rate is reduced by a factor 2 to 10 when the dose rate is lowered.

Some human studies suggest that cancers associated with high relative risks at 1 Gy (e.g. leukemia, thyroid cancer, and female breast cancer) may have a dose and dose rate effectiveness factor of 2 or more. In the study of A-bomb survivors, the dose-responses for other cancers appear to be best described by a linear model. However, allowing for uncertainties in observed risks and estimated doses, the dose response could plausibly be considered consistent with the DDREFs up to 2.5. Values greater than this would be difficult to justify (Pierce and Vaeth 1991).

The range of DDREFs that has been suggested by various organizations in the past 10 years are listed in table 3.2. The International Commission on Radiological Protection recently concluded in its 1990 Recommendation of the ICRP (ICRP 1991) that sufficient evidence exists to justify apply a DDREF of 2 to estimate the cancer risks at organ doses below 0.2 Sv and dose rates below 0.1 Sv per hour. The ICRP recommendation will be used in this report.

## 3.6 Risk Modelling

An important aspect of assessing the excess cancer risk is the proper description of the level and pattern of excess cancers appearing in the lifetime of an exposed population. However, very few epidemiological studies have followed cohorts until all members have died. For individuals who are still living it is necessary to use risk projection models to extrapolate their cancer risks in time and age beyond the current state of knowledge.

The present section will describe the risk projection models that have been used in the past and how they have gradually evolved from simple age-constant risk models averaged over all ages and both sex which assume the excess risk will remain constant over time to exposure-time-response models that allow for any variations with sex, attained age, or time following exposure. Discussion will be focused on the

uncertainties in the models used to describe the excess cancer risk in the Life Span Study of the atomic bomb survivors.

### 3.6.1 Age-constant absolute and relative risk models

The Japanese survivors of the atomic bombings have been followed for only about 40 years and as result, the vast majority of persons exposed at ages under 30 ATB are still living. Generally, either an age-constant absolute risk (AR) or relative risk (RR) model has been used to describe the magnitude and pattern of excess cancers experienced by survivors who have died as well as to project the future excess cancer risk for survivors who are still living.

The age-constant AR model assumes, following an initial latency period, that the number of excess cancer deaths will be constant in each subsequent year following exposure and independent of the natural background cancer risk (see figure 3.4). That is,

$$\lambda'(u) = \lambda(u) + AR \qquad (3)$$

where

$u$ is age at risk;

$\lambda'(u)$ is the cancer risk after exposure;

$\lambda(u)$ is the background cancer risk without exposure; and

AR is the absolute excess risk caused by the radiation exposure and is assumed to remain constant over time.

The age-constant RR models assumes, following an initial

latency period, that the number of excess cancer deaths in each subsequent year following exposure will be a constant proportion of the natural background cancer risk (see figure 3.4). That is,

$$\lambda'(u) = \lambda(u) \times RR \qquad (4)$$

where

RR is the increased proportion of cancers, i.e. the relative risk, caused by the radiation exposure and is assumed to remain constant over time.

In the most simple form, the values of the AR and RR can be averaged over both sexes and all ages at exposure. However when sufficient data is available, it is more common to allow the risk to vary with sex and age-at-exposure. The relative risk model will project significantly higher excess risk than the absolute risk model for survivors who are still alive. However, the difference in projections between the models has decreased as follow-up of survivors has become more complete (ICRP 1991).

Models used in the past 20 years

Table 3.3 summarizes the models used during the past 20 years to describe the cancer mortality in the Life Span Study and the estimated cancer risk made. Estimates based on the relative risk model have been fairly robust, varying by less than a factor of 2 since 1972 while estimates based on the absolute risk model has increased by about 3-4 times.

Before the latest follow-up period (1950-1985), the preference of one risk model over another was a contentious issue. In 1972 the BEIR I Report (NRC 1972) could find no basis for preferring one model over the other. Although they used both the absolute and relative risk models, they decided to base their risk estimates using the more conservative RR model. The 1977 reports by UNSCEAR (UNSCEAR 1977) and the ICRP (ICRP 1977) both based their risk estimates on an age-constant absolute risk model whose coefficients were averaged over sex and age-at-exposure. The BEIR III report (NRC 1980) used both models.

Muirhead and Darby (1987)[23] tested a hybrid absolute/relative risk model on mortality data for all cancers other than leukemia as a group for the follow-up period 1950-1978 and using T65D dose estimates. The model featured a mixing parameter that took on the value of one if the absolute model fitted the data and the value of zero if the relative risk model fitted the data. They found that neither the AR or RR adequately fitted the data nor do they necessarily provide an upper and lower bound for the projected risk.

The last extended period of follow-up (1982-1985) shows that the absolute excess risk for cancers other than leukemia has increased compared to earlier periods of follow-up while the relative risk has remained fairly constant. As a

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[23] Results described in UNSCEAR 1988

result, it is now the generally agreement that the age-constant absolute risk is inadequate and should no longer be used to project lifetime cancer risks (Darby 1991, NRC 1990, Thomas 1990, Pierce and Vaeth 1990a, 1990b, 1989b, UNSCEAR 1988, Shimizu et al. 1988, Preston et al 1986). The preference of the relative risk model over the absolute risk model is the main reason why current risk estimates by UNSCEAR and the ICRP are 3-4 times higher than estimates made in 1977.

3.6.2 Exposure-time-response risk models

While the age-constant relative risk model appears to adequately describe the mortality for all cancers other than leukemia for the LSS cohort up to the period 1950-1985, it appears to be too simple for describing the variation of excess risks with time at different cancer sites. Nearly all high dose epidemiological studies report the excess relative risk for leukemia mortality to begin at about 2 years following exposure, peak within 5 years, and declining thereafter (see section 2.0). The ankylosing spondylitis study shows a significant reduction in the excess relative and absolute risks for lung cancer following 17 years after exposure, with risks returning to normal following 25 years (Darby et al. 1987). A slight decreasing trend over time in the relative risk for lung cancer is also suggested by the Life Span Study of the atomic bomb survivors (Shimizu et al. 1988). A parallel analysis of the LSS cohort, Canadian and

Massaschusetts fluoroscopy patients, and postpartum mastitis patients by the BEIR V Committee showed excess relative risk of breast cancer to decline following about 20 years after exposure (NRC 1990). The relative risk has also been observed to decrease smoothly for thyroid cancer following 15 years after exposure in the study of infants receiving thymus irradiation (Shore et al. 1985).

With the advancement in recent years of statistical techniques for modelling radiation risks, it is no longer considered appropriate not to examine, or adjust if necessary, for apparent time variations in the relative risk (Darby 1991, NRC 1990, Thomas 1989). If models are allowed to adjust for age and time effects, there is no longer a real difference mathematically between the relative and absolute risk model. They merely represent two simple alternative ways of describing observed effects (Preston 1990). Investigators have found that the absolute risk model usually requires more parameters to adjust for age and time effects compared to the relative risk model (NRC 1990, Thomas 1989, Muirhead and Darby 1989) and that absolute risk models tend to be more susceptible to bias from incomplete or poor quality cancer data and from errors in dosimetry (Howe 1991a, NRC 1990). Because of this, exposure-time-response relative risk models are normally preferred over the absolute model.

The BEIR IV and V Reports (NRC 1988, 1990) were the

first comprehensive analyses of the biological effects of ionizing radiation to employ exposure-time-response models. The BEIR IV Committee examined the increase risk of lung cancer mortality due to radon daughter exposure experienced by four cohorts of underground miners. Analyses indicated the excess relative risk declined with both attained age and time-since-exposure. The age specific lung cancer mortality rate at attained age a following radon daughter exposure was modelled by

$$r(a) = r_0(a)\,[1 + 0.025\, I(a)\,(W_1 + 0.5W_2)] \qquad (5)$$

where

$r_0(a)$ is the age specific baseline lung cancer rate at attained age a for persons of a given sex and smoking status;

$I(a)$ = 1.2 when age a is less than 55 yr

= 1.0 when a is 55-64 yr

= 0.4 when a is 65 yr or more;

$W_1$ is the cumulative exposure in Working Level Month (WLM) incurred between 5 and 15 yr before attained age a; and

$W_2$ is the WLM incurred 15 or more years before this age.

The BEIR V Committee examined the increased lifetime risk of mortality from all cancers following exposure to low-LET ionizing radiation. Their analysis resulted in five preferred

relative risk models which adjust for variations with sex, age-at-exposure, and time-since-exposure. Models are described in section 4.2.

3.6.3 Variation of excess risks with age at exposure

Most epidemiological studies indicate children and young adults may have a greater susceptibility to the carcinogenic effects of ionizing radiation compared to adults exposed at older ages. In the study of women treated for cervix cancer the increased risk of second cancers was concentrated among those adults irradiated at younger ages (Boice et al. 1988). Studies of the radiogenic risk of breast cancer show women are most radiosensitive at ages below 15 years while increased risks are small for women exposed at ages above 30. Studies of infants irradiated for the treatment of supposedly enlarged thymus glands (Shore et al. 1985) and for skin haemangioma (Darby 1991), shows radiation can significantly increase the risk for cancers of the thyroid, breast, brain, and bone and connective tissue. The Israeli tinea capitis study (Ron et al. 1989) showed children irradiated under 5 years of age had nearly twice the relative risk for subsequent thyroid cancer compared to children exposed between the ages of 5 and 14. In the ankylosing spondylitis study the relative risk for all cancers other than leukemia and colon cancer showed little variation with age-at-exposure, but the absolute excess risk did. For patients who

were under the age of 25 at the time of treatment, excess absolute risks were about 10 times lower compared to those patient treated at ages above 45 (Muirhead and Darby 1989).

In the Life Span Study of atomic bomb survivors, nearly all the survivors who were exposed under the age of 20 at the time of the bombings (ATB) were still living in 1985. During the years 1950-1985, the absolute excess risk for all cancers except leukemia for this under 20 ATB group was about 4 times smaller than survivors in the over 40 ATB group, while relative risk were 4 times higher (Shimizu et al. 1988). It is evident that any assumptions regarding the future behaviour of the relative risk will greatly influence the projected excess lifetime cancer risk for younger age groups. For instance, if the relative risk is assumed to remain elevated for the survivors' entire lifetimes, then the total excess cancer risk for those under age 20 ATB will increase by 10-fold and will be higher by about a factor 3 compared to the projected excess risk for over 40 ATB group (Pierce and Vaeth 1989b). Charles and Little (1990) showed that lifetime excess risk projections for a U.K. general and working population following a single whole-body dose of 1 Gy are reduced by 15-40 percent if the relative risk for all cancers other than leukemia is allowed to decay exponentially with time following 40 years.

### 3.6.4 Summary

The BEIR V preferred relative risk models are presently considered to be the most suitable models for lifetime risk projection (Darby 1991) and will be used in this report. The BEIR IV models for lung cancer mortality following radon exposure are not considered[24].

It should be noted that the BEIR V relative risk models are only mathematical models. Adjustments for age or time effects have no clear relevance to biological mechanisms and were made only to provide the best fit to the data (Howe 1991b). Because the behaviour of the excess relative risk beyond 40 years following exposure is unclear, age-specific lifetime projections performed in this report involving young exposure ages should be interpreted cautiously.

## 3.7 Transfer of Excess Risk Coefficients Between Populations

### 3.7.1 Introduction

An important issue associated with predicting radiogenic risks using excess risk coefficients or models derived from the Life Span Study of the atomic bomb survivors is how to transfer derived excess risk coefficients to other populations where baseline cancer rates are substantially different from those in Japan. For excess relative risk coefficients, there are two plausible methods of transfer. The

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[24]It was felt that although such projections would be interesting, it was beyond the main purpose of the report, namely, to assess the suitability of the ICRP-60 fatal cancer risk estimates in Canada

first is a multiplicative method that simply applies the relative risk directly to the baseline cancer rates in other populations. The other is an additive method that transfers the absolute risk by first applying the relative risk to the baseline cancer rates of Japan. If baseline rates are similar, the choice of transfer method should not matter. But if baseline rates differ substantially, as it does between Japanese and North American populations (see section 5.3), the choice of method might significantly influence the projected lifetime cancer risk.

Presently it is unclear which, if either, of these two possible transfer methods should be used. The remainder of this section will review the information currently available from (a) biological mechanisms of cancer induction and (b) studies examining the excess risks at a specific cancer site for different exposed cohort populations, that might provide some assistance in choosing the best method. The effect of the choice of transfer method on projected lifetime risks is examined in chapter 5.0.

### 3.7.2 Biological mechanisms of cancer induction

Conjectures as to which might be the best method for transferring risks can be attempted by examining the possible biophysical reasons as to why baseline rates between populations might differ. As discussed in section 3.5.1, the process of cancer induction can be sub-divided into three

stages: initiation, promotion, and progression. Ionizing radiation is widely accepted to be an initiator; its possible role as a promoter is not yet understood (Cox 1991). Assuming that radiation acts only as an initiator, inferences can be made depending on whether variations in baseline rates are caused by the action of cancer initiators or by cancer promoters. If differences are caused by initiators alone, the risk caused by radiation will only add to the baseline cancer risk and the excess absolute risk should therefore be the same between populations. The additive transfer method would seem preferable. But if differences are caused by promoters, the relative difference between baseline rates would indicate differences in the probability of a radiation-initiated cell being promoted to a cancer. The multiplicative transfer method would therefore seem more preferable. However, the development of cancer is a very complex process. Present understanding of the role of genetic, physiological, and environmental factors in the different cancer stages is not yet sufficient to explain unequivocally explain why baseline rates differ (Cox 1991, UNSCEAR 1988). It is most likely the case that variations are caused by combined effects of differences in initiator and promoter agents.

### 3.7.3 Information from Human Studies

#### Breast Cancer

In 1980, Land et al. (1980) compared the excess

incidence of breast cancer observed in cohort studies of Japanese A-bomb survivors, Massachussetts fluoroscopy patients, and New York postpartum mastitis patients. It was found that the relative risk model described the excess breast cancer best in each cohort. However, the absolute excess risk was found to be more comparable between cohorts. In 1989, the BEIR V Committee performed a similar analysis, but in addition, also compared the increase breast cancer mortality in Canadian non-Nova Scotia tuberculosis patients with that experienced by female A-bomb survivors. In contrast to Land et al., the Committee's analysis suggested the relative risk for breast cancer incidence was more comparable between cohorts. The Committee attributed the discrepancy with Land et al. to the additional follow-up of the U.S. cohorts, introduction of the DS86 dosimetry system, changes in the make-up of the LSS cohort, and the use of exposure-time-response risk models (NRC 1990). For breast cancer mortality, cohort differences in the both absolute and relative risk were large but not statistically significant. The excess relative risk per unit dose was 2-3 times higher for Japanese women compared to non-Nova Scotia Canadians while the relative difference in the absolute excess risk was somewhat lower (NRC 1990).

Thyroid Cancer

The BEIR V Committee also compared the excess incidence of thyroid cancer experienced by Israeli tinea

capitis and New York thymus patients. The Committee found that the relative risk was more comparable between the two children cohorts than the absolute risk.

Further follow-up of the tinea capitis cohort by Ron et al. (1989) has shown discrepancies with the BEIR V results. For children irradiated between the ages of 5 and 14, the relative risk has increased from the BEIR V estimate of 8.3 per Gy (page 236, NRC 1990) to a value of 17 per Gy estimated by Ron et al.. In addition, when the excess absolute risk estimated by Ron et al. for the under 5 age group is compared to that estimated in the Rochester thymus study for thyroid doses under 0.3 Gy, the estimates were in very good agreement, 13 compared to 14 per $10^4$ PYGy (Ron et al. 1989, Shore et al. 1986). Thus suggesting absolute risks might be more comparable.

A different conclusion can also be made by comparing the increased risk experienced between boys and girls in the Israeli study. Israeli girls, who have a higher baseline risk of thyroid cancer than boys, experienced a significantly higher absolute excess incidence rate of thyroid cancer. In contrast, the relative risk between sexes was the same (Ron et al. 1989).

As Ron et al. (1989) noted, the excess number of thyroid cancer cases in these cohorts are still too small to make any stable risk estimates or firm conclusions.

### 3.7.4 Approaches Used by Recent Radiological Reports

The 1988 UNSCEAR report (UNSCEAR 1988) compared the lifetime cancer risk projected to three populations (Japan, United Kingdom, and Puerto Rico) when the absolute and excess relative risk coefficients obtained from age-constant absolute and relative risk models were transferred between populations. When absolute risks were transferred, it was found that were virtually no differences projected risk estimates between populations. When the relative risk was transferred, the maximum difference between any two populations was 20 percent. The report considered this to "clearly show that the lifetime risk projections are very insensitive to differences" in baseline cancer rates. However, it was noted later that "much larger proportional differences may apply to site-specific cancers such as female breast, stomach, large bowel, and lung" (UNSCEAR 1988).

It was the judgement of the BEIR V Committee that there was no particular reason as to why the absolute risk should be transferred (Thomas 1990). Based on the findings of the comparison of excess risks between different breast cancer cohorts and thyroid cancer cohorts, the Committee chose to transfer the excess relative risk from its preferred risk models directly to the U.S. population. However, the report did acknowledge that it is not clear whether the relative or absolute risks should be transferred across populations and

that it may be the case that neither can be extrapolated with any assurance (NRC 1990).

The 1990 ICRP recommendations (ICRP 1991) could not agree on which, if any, transfer method should be used or whether the same method should be used for every cancer site. They opted to transfer risks using both methods and then to average the resulting estimates.

3.7.5 Summary

There is no general agreement on which, if any, type of risk transfer method should be used for estimating excess cancer risks in populations other than Japan. It is likely that neither the additive or multiplicative method will be appropriate in all circumstances and probably may depend on the particular cancer and possibly on age and sex. The issue is simply not resolvable at this time (Land et al. 1991).

Table 3.1
Dose and dose rate effectiveness factor (DDREF) for tumour induction in experimental animals (Strather and Goodhead 1991, table 8)

| Effect | Animal studied | Dose rate (mGy/min) High | Low | DDREF |
|---|---|---|---|---|
| Myeloid leukemia | CBA/H male mice | 250 | 0.04-0.11 | 2.2-5 |
| | RFM male mice | 800 | 0.04-0.6 | 5.6 |
| | RFM/Un female mice | 450 | 0.06 | 9.7- inf |
| | Rf female mice | 67 | 0.004-0.7 | 14 |
| Lung adenocarcinomas | BALB/c female mice | 400 | 83 | 2.8 |
| Lung cancer (a) | Beagle dogs | | | 3 |
| Mammary tumours | BALB/c mice | 450 | 0.06 | 1.9 |
| | Sprague-Dawley rats | 10-30 | 0.02-0.14 | 1.6-1.7 |
| | Spague-Dawley rats | 100 | 30 | 1 |
| | WAG/RU rats | (2 Gy) | (2 Gy) (b) | 1 |
| Mammary adenocarcinomas | Sprague-Dawley rats | 100 | 30 | 4 |
| Pituitary tumours | BALB/c mice | 450 | 0.06 | 6 |
| Thyroid tumours (c) | CBA mice | (15 Gy) | (64-160 Gy) | 2-10 |
| | Rats | (11 Gy) | (100 Gy) | 10 |
| | Long Evans rats | 2800 | (0.8-8.5 Gy) | 1 |
| Harderian gland tumours | RFM female mice | 450 | 0.06 | 3 |
| Ovarian tumours | RFM mice | 450 | 0.06 | 5.5 |
| | RFM mice | 400 | 83 | 6.7 |
| Thymic lymphomas | RFM female mice | 450 | 0.06 | 5.8 |
| | RF male mice | 800 | 0.04-0.6 | 2.6 |

(a) High dose rate from Y-91; low dose rate from Ce-144 or Sr-90
(b) Ten fractions of 0.2 Gy each
(c) High dose rate from x rays; low dose rate from I-131

Table 3.2
Summary of suggested dose and dose rate effectiveness factors

| Investigators | DDREF | Reference |
|---|---|---|
| ICRP 60 | 2 | ICRP 1991 |
| Pierce and Vaeth | 1-2.5 (a) | Pierce and Vaeth 1991, 1990a |
| BEIR V Report | 2 or more | NRC 1990 |
| 1988 UNSCEAR Report | 2-10 | UNSCEAR 1988 |
| 1986 UNSCEAR Report | up to 5 | UNSCEAR 1986 |
| BEIR III Report | 2.25 | NCRP 1980 |
| NCRP Report 64 | 2-10 | NCRP 1980 |
| ICRP 26 | 2.5 | ICRP 1977 |
| 1977 UNSCEAR Report | 2.5 | UNSCEAR 1977 |

(a) Range was 1-1.5 if no allowances made for imprecision in dose estimates and 2-2.5 if allowances are made

Table 3.3
Derived fatal cancer risk estimates over the past 20 years

| Source of estimate | Lifetime risk of fatal cancer per 10E04 Gy | |
|---|---|---|
| | AR model | RR model |
| 1972 BEIR I | 120 | 620 |
| 1977 UNSCEAR | 250 | - |
| 1977 ICRP 26 | 100 (a) | - |
| 1980 BEIR III | 80 - 250 | 230 - 500 |
| 1988 UNSCEAR | 420 | 1070 |
| 1990 BEIR V | - | 885 (b) (1060 ) (c) |
| 1991 ICRP 60 | - | 500 (d) |

(a) DDREF=2.5 was used
(b) 'Low dose' leukemia component multiplied by 2
(c) Excess lifetime risk multiplied by 1.2 to obtain lifetime risk of fatal cancer (see section 4.4)
(d) DDREF= 2 was used

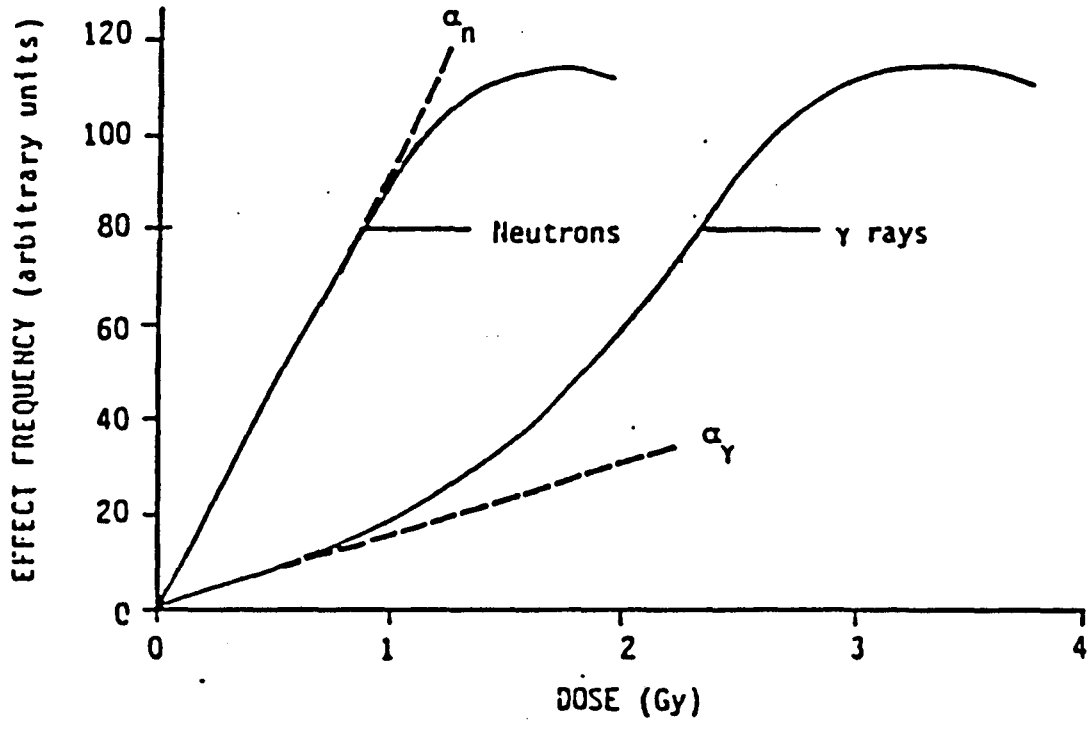


Figure 3.1
Expected dose-response relationships for cancer induction by high- and low-LET ionizing radiations

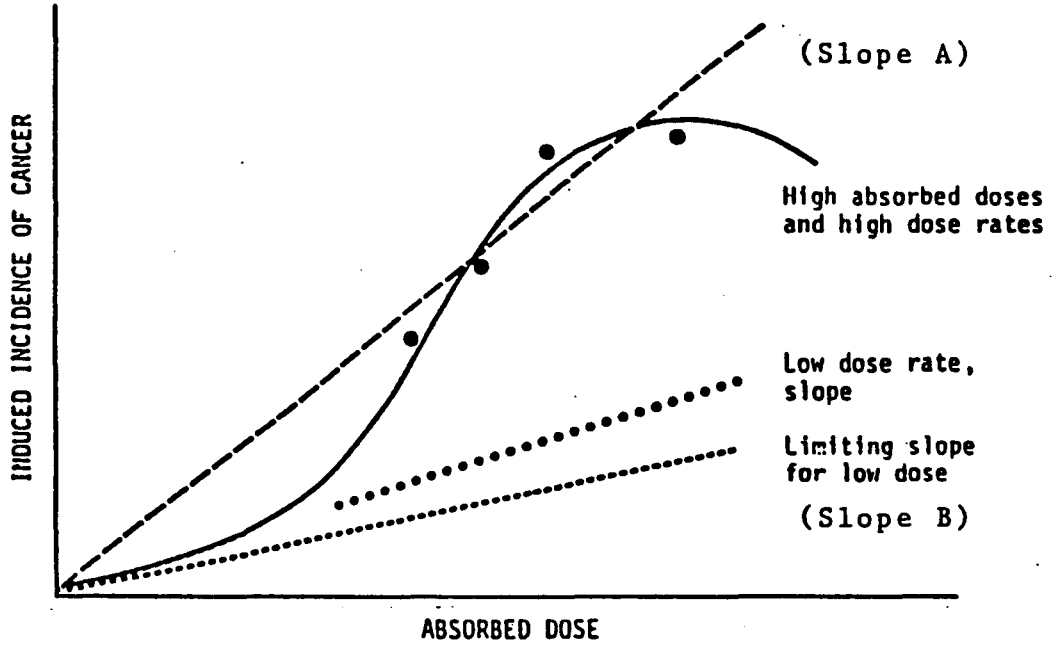


Figure 3.2
Expected dose-response relationships for cancer induction by low-LET ionizing radiation at different doses and dose rates

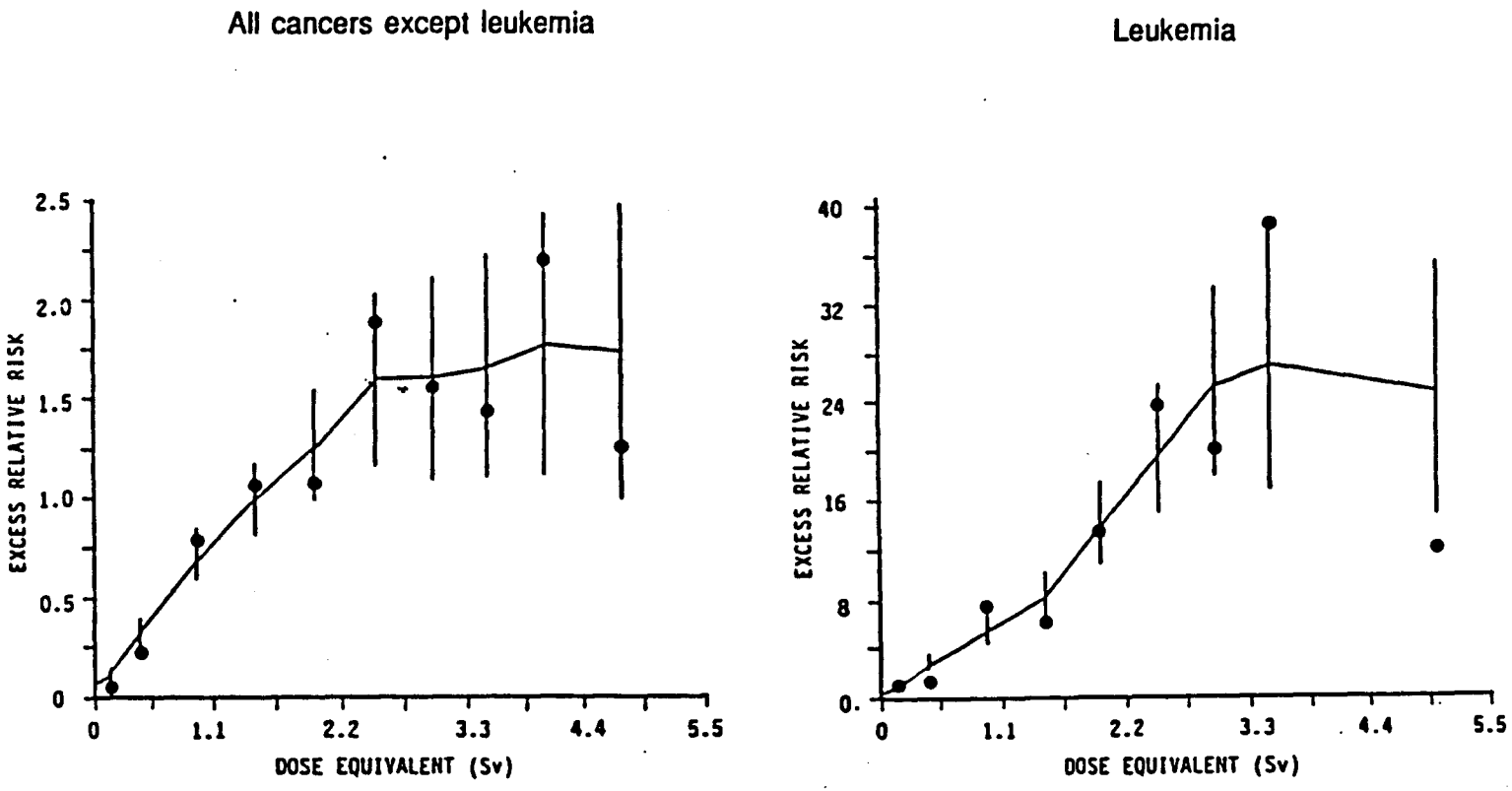


Figure 3.3
Dose response among LSS cohort for all cancers except leukemia as a group and leukemia in terms of intestinal dose equivalent (neutron RBE=10)
(Points are the excess relative risk and the lines moving averages of these. Error bars refer to the smoothed points. Risks are average over city, sex, and age at exposure)

(Adapted from Pierce and Vaeth 1991)

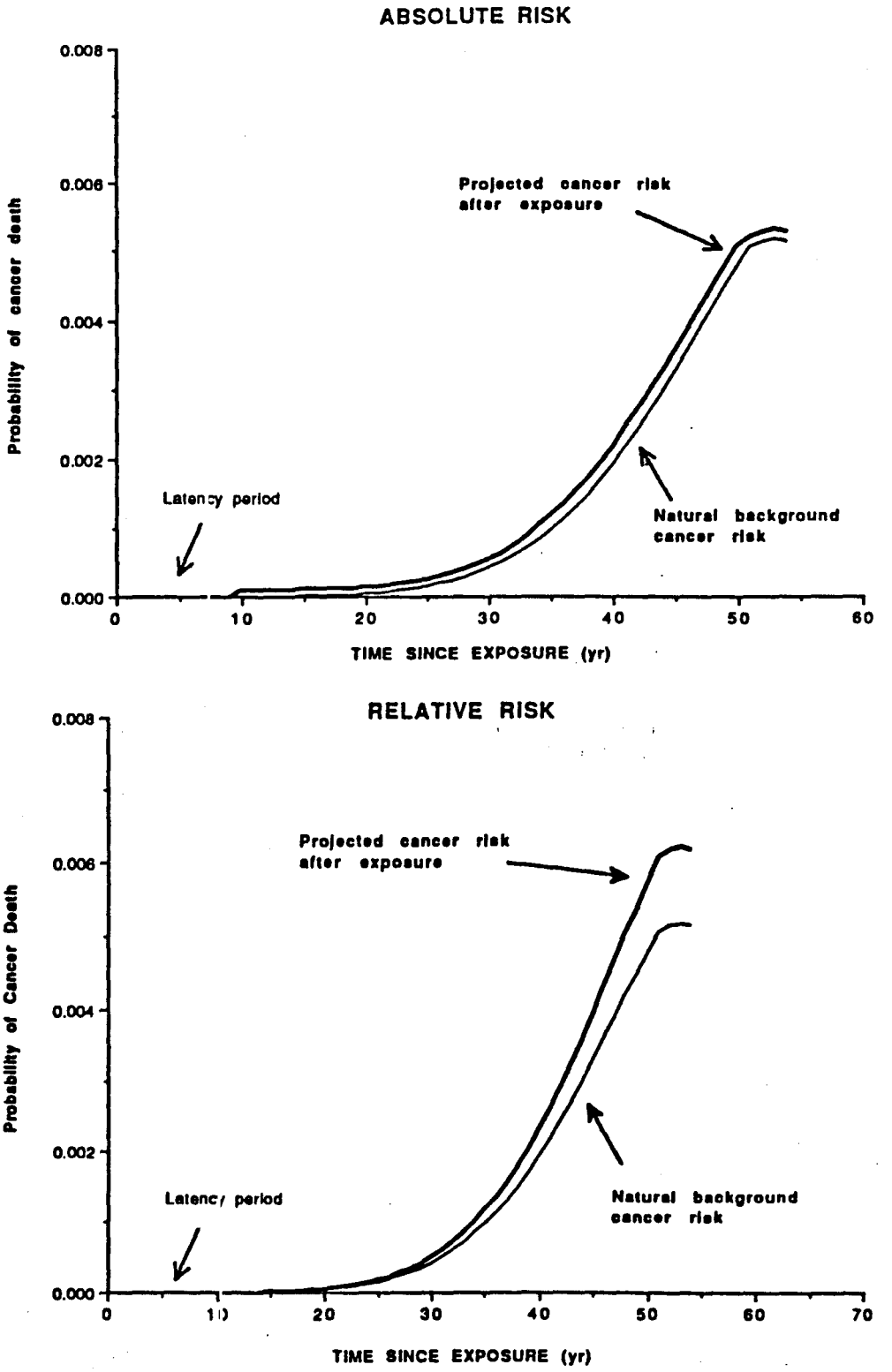


Figure 3.4
Age-constant absolute and relative risk projection models

# 4.0 Lifetime Risk Projections

## 4.1 Introduction

The process of projecting lifetime cancer risks in populations following exposure to ionizing radiation consists of performing two tasks. The first is to develop risk models describing the subsequent magnitude and pattern of excess risk of cancer mortality following radiation exposure. The second is to use the risk models along with life-table techniques to project the lifetime cancer risks for the exposed population under consideration.

Models and risk coefficients are provided by the ICRP (based on Shimizu et al. 1988) and BEIR V. For reasons given in chapter 1.0 and section 3.6, this report will use the risk models developed by BEIR V. The models, and their development, are described in section 4.2. The remainder of the chapter describes how life-table techniques can be used to evaluate the potential effects radiation exposure might have on the level of cancer mortality in a population. The life-table allows the cancer risk to be described in various ways. Six risk attributes will be defined that can describe the magnitude of increased risk of cancer mortality or the

temporal distribution of the increase over time. Examples are given for projections for a 1988 Canadian population following a hypothetical single whole-body exposure of 0.1 Gy.

## 4.2 BEIR V Risk Models

The fifth National Research Council Committee on the Biological Effects of Ionizing Radiation (NRC 1990), or BEIR V, carried out an extensive statistical analysis of the carcinogenic effects of low-LET ionizing radiation. Analysis was performed using cancer mortality primarily from the Life Span Study of atomic bomb survivors. Data from other studies (see table 4.1) were used whenever possible. The Japanese LSS data was made available to the Committee by the Radiation Effects Research Foundation for mortality in the years 1950-1985. Data was stratified by sex, city, ten exposure groups (DS86 individual dose estimates, RBE=20), and five-year age intervals of attained age, age at exposure, and time-since-exposure (NRC 1990)

Both absolute and relative risk exposure-time-response models with parameters adjusting for variations with sex, age-at-exposure, time-since-exposure, and attained age were fitted to data for ten cancer sites or groups of sites using maximum likelihood methods. Relative risk models were of the form

$$RR = [1 + f(d)\, g(\beta)] \qquad (6)$$

and absolute risk models,

$$AR = f(d)\, g(\beta) \qquad (7)$$

where

d is organ absorbed dose

f(d) is the dose-response function, either linear or linear-quadratic

$g(\beta)$ is the excess risk function and $\beta$ is a vector of coefficients describing the dependency of the risk on sex, age-at-exposure, time-since-exposure, and attained age.

It was found that relative risk models needed less parameters for describing age and temporal variations and were more stable than absolute models. The Committee therefore used only the relative risk models for its formal assessment. The analysis of cancer sites found that there was only sufficient data to provide stable risk models for five separate cancer sites or groups of sites. These were

(1) leukemia,

(2) respiratory tract,

(3) female breast,

(4) digestive system, and

(5) other remaining sites

The sites within each cancer group is shown in table 4.2.

4.2.1 Leukemia (ICD[18] 204-207, excluding CLL)

The Committee's preferred risk model for leukemia represents the total relative risk of mortality for all types of leukemia, excluding chronic lymphatic leukemia (CLL). The model is given by

$$RR(\text{leukemia}) = [1 + f(d)\, g(T,TSE)] \qquad (8)$$

where

d is the bone marrow dose in Gy (or Sv);

T is the age at exposure

TSE is the time since exposure in years;

$f(d) = 0.243\ d + 0.271\ d^2$;

$g(T,TSE) = \exp[4.885\ I(TSE \leq 15) + 2.380\ I(15 < T \leq 25)]$ for $T \leq 20$;

$g(T,TSE) = \exp[2.367\ I(TSE \leq 15) + 1.638\ I(15 < T \leq 25)]$ for $T > 20$;

and

$I(T \leq 15)$ equals 1 when $T \leq 15$ and 0 otherwise

$I(15 < T \leq 25)$ equals 1 when $15 < T \leq 25$ and 0 otherwise

The minium latency period is taken to be 2 years. Figure 4.1 plots the model as a function of time-since-exposure for ages at exposure under and above 20.

There is a distinct difference in excess risk exhibited for those exposed under the age of 20 and those at older ages. A simple step function with two steps fitted both groups rather well. There did not appear to be any differences

[18] International code of diseases

between males and females.

The Committee had to model the 2 to 5 year period because the study of the LSS cohort did not begin until five years after the exposure (by which time the peak in excess leukemia would have been expected to have already occurred). This was done by extrapolating the excess relative risk observed in the 5-10 years period back to 2 years. The Committee noted that the extrapolation procedure may lead to an underestimation of the actual risk and this should be kept in mind when interpreting results of risk projections. Because the age dependence in baseline rates differs among different leukemia types, the Committee cautioned that the model is a gross simplification.

4.2.2 Respiratory tract (ICD 160-163)

The increased relative risk of respiratory cancer mortality was modelled by

$$RR(\text{respiratory cancer}) = [1 + 0.636\, d\, g(TSE,S)] \qquad (9)$$

where

d is the lung dose in Gy (or Sv);

S indicates male or female;

TSE is time since exposure;

$g(TSE,S) = \exp[-1.437 \ln(TSE/20) + 0.711\, I(S)]$; and

$I(S) = 1$ for females and 0 for males

A 10 year minimum latency period is assumed. Figure 4.2 plots the model as a function as age-since-exposure for

both males and females.

The preferred dose-response model was linear. Women had a somewhat higher relative risk than men, however, the models do not adjust for the possible confounding by smoking among male and female survivors. Adjustment for smoking would tend to push the excess relative risks for females upward and downward for males (Darby 1991, Howe 1991a). Analysis of data showed little effect of age-at-exposure on the excess RR but did show a decrease in excess RR with time after exposure. Although an age-constant relative risk gave almost as good fit to the data, it was decided to include a time dependant term because of a similar temporal pattern seen among spondylitic patients (see section 2.3). The term results in a decrease of the RR by a factor of 5 over a 10 to 30 year time period after exposure.

It should be noted that survivors exposed in childhood, under the age of 10 ATB, have not yet experienced any increase in lung cancer deaths and are just now approaching the ages where lung cancer mortality is prevalent in the Japanese population (Shimizu et al. 1988). It is not known how valid the above models are for this age group.

4.2.3 Female breast (ICD 174)

The breast cancer models were fitted using the pooled mortality data for the female atomic bomb survivors and Canadian tuberculosis patients. Data for Nova Scotia patients

was excluded for describing the magnitude of the relative risk, but was included for describing age and temporal variations. The minimum latency period is taken to be five years. Data was analyzed separately for exposure ages under and above 15 years. The model is given by,

$$RR(\text{breast cancer}) = [1 + 1.220d\, g(T,TSE) \qquad (10)$$

where

d is the breast dose in Gy (or Sv);

T is the age at exposure;

TSE is the time since exposure;

$g(T,TSE) = \exp [1.385 - 0.104 \ln(TSE/20) + 2.212 \ln^2(TSE/20)]$ for $T \leq 15$; and

$g(T,TSE) = \exp [-0.104 \ln(T/20) - 2.212 \ln^2(T/20) - 0.0628 (T-15)]$ for $T>15$.

Figure 4.3 plots the model as a function of time since exposure for a number of ages at exposure.

The studies of the different female cohorts all suggest that women exposed before the age of puberty (< age 16) have a much greater relative risk of cancer mortality with the risk decreasing with increasing age at exposure. For all ages, the relative risk was seen to peak at about 15-20 years after exposure and decline thereafter.

4.2.4 Digestive system (ICD 150-159)

For digestive cancers, the relative risk was modelled by a linear age-constant relative risk model. The RR decreased

with increasing age at exposure above age 25 with the risk higher for women compared to males. The model is given by,

$$RR(\text{digestive cancer}) = [1 + 0.809 d\, g(S,T)] \qquad (11)$$

where

d is the stomach dose in Gy (or Sv);

S is male or female;

T is the age at exposure;

$g(S,T) = \exp[0.553\, I(S) + h(T)]$;

$I(S) = 1$ for females and 0 for males; and

$h(T) = 0$ if $T \leq 25$,

$= -0.198\,(T-25)$ if $25 < T \leq 35$, or

$= -1.98$ if $T > 35$

A 10-year minimum latency period is used. Figure 4.4 plots the model as a function age at exposure for males and females. A significantly higher excess RR was observed for those exposed under the age of 25 and decreased thereafter with increasing age at exposure. While there is no apparent biological basis for such an abrupt change in the RR at this age, it does not appear to be an artifact in the data or analysis (NRC 1990). While the RR have remained constant with time after exposure for those exposed at older ages, it is unclear whether this will hold true for the younger exposure groups.

4.2.5 Other remaining sites (ICD 140-209 less those listed above)

The preferred model, after a minimum latency period of 10 years, is a linear age-constant relative risk model with the RR decreasing exponentially with increasing age at exposure after the age of 10. There was no evidence of either an effect by sex or time after exposure. The model is given by

$$RR(\text{othercancer}) = [1 + 1.220d\ g(T)] \qquad (12)$$

where

d is the appropriate organ dose in Gy (or Sv);

T is age at exposure; and

$g(T) = 1$, if $T \leq 10$, or

$= \exp[-0.0464\ (T-10)]$, if $T > 10$

Figure 4.5 plots the model as a function of age at exposure. The Committee found that the mortality data at other cancer sites were not sufficient to provide stable modelling for any further breakdown of cancers.

## 4.3 Life-table Methodology

### 4.3.1 Cohort and Stationary Population

A life-table is a hypothetical population that models the level and pattern of mortality in a given population. By incorporating the additional cancer risks caused by radiation exposure, the table can be used to evaluate the potential lifetime cancer mortality risks of radiation exposure.

The life-table can be treated either as a "cohort" or "stationary" population. As a cohort population, the life-table is constructed by following individuals throughout their lifetime under the assumption that they are subject to the same age- and sex-specific mortality rates as the population they are living in. Normally the table is constructed by following an initial cohort of 100,000 newborns through their entire lifetime. The number of persons alive, number of deaths, and the expected years to live at any subsequent age can be calculated (Sullivan and Weng 1987, Bunger et al. 1980, Shyrock and Siegel 1973).

If it is assumed that birth rates remain constant, the cohort population can also be treated as stationary (Shyrock and Siegel 1973). The age distribution will be given by the number of the initial 100,000 newborns surviving to each subsequent age. If m(u-1) denotes the probability of cohort members of age u-1 dying within the next year (i.e., the age-specific mortality rate) and l(u-1) the number of persons of age u-1 living at the beginning of the year, then the number of persons alive the next year, l(u), will be given by

$$l(u) = l(u-1)\ e^{-m(u-1)} \qquad (13)$$

In terms of the initial number of newborns, l(0), this may be rewritten as

$$l(u) = l(0)\ \exp\left(-\sum_{x=0}^{u-1} m(x)\right) \qquad (14)$$

or

$$l(u) = l(0)\ S(u) \qquad (15)$$

where

S(u) is the survival function denoting the probability of surviving to age u from birth;

If m(u) is always less than one, the survival function may be approximated by

$$S(u) = \prod_{x=0}^{u-1} (1-m(x)) \qquad (16)$$

The value of m(u) is taken to be 1 for the maximum age in the life-table population, normally assumed to be the age of 105 (Shylock and Siegel 1973). Figure 4.6 shows the age- and sex-specific mortality rates from all causes of death in a 1988 Canadian population[27]. Figure 4.7 shows the age distribution[28] of the 1988 Canadian population (StatsCan 1990b) and the distribution for the corresponding stationary

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[27] Rates derived using data from 1988 Canadian causes of death tables and population estimates (see appendix B for further detail).

[28] Averaged over both sex and expressed in terms of the proportion of the total population.

life-table population[29]. The age distribution of the life-table population provides a fair representation of the true age distribution of the Canadian population except in the 30 to 50 age range. Here, the 1988 Canadian distribution has a "blip" as a consequent of birth rates not being constant during the late 1940s and in the 1950s, the so-called "baby boom" years, when birth rates were higher than normal.

4.3.2 Assessment of Radiation Risks

The lifetime cancer risks in a population following radiation exposure is evaluated by incorporating the additional risk of cancer mortality given by radiation risk models into the life-table. Because the life-table can be treated either as a cohort or stationary population, potential lifetime risks can be assessed for (a) a single radiation exposure for males, females, or both at a specific exposure age; (b) a single radiation exposure in a population with a given age and sex distribution (c) prolonged radiation exposure over a number of years; and (d) a specific exposure scenario. Section 4.4 and 4.5 describes further how the life-table is used to assess radiation-induced cancer risks.

4.3.3 Limitations

The assumption that cancer rates and overall mortality rates will remain constant over the lifetime of a life-table population limits the accuracy in lifetime risk projections.

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[29] Distribution calculated using the above expression for the number of newborns surviving to each age.

In reality, rates vary annually for statistical reasons and over a period of time may change significantly as lifestyle, environmental conditions, and health care changes.

In industrialized countries, the average life expectancy has increased substantially as the result of improved hygiene, cleaner water, better living conditions, and advances in medicine and public health (ICRP 1991). Cancer rates can also change over time. The incidence and mortality rate for lung cancer has doubled in Canada for women in the last 20 years while for men, there is evidence lung cancer rates have levelled off since 1985 and may be starting to decline (NCIC 1990). Lung cancer trends are attributed to changes in smoking habits in the past 30 years. The rate of melanoma of the skin has also been on the rise and is likely the consequence of increased sunbathing and exposure to the sun in the 1970s and 80s (NCIC 1990). In contrast, stomach cancer rates has decreased by almost half in the past 20 years. The reason for the decline is not known, but it has been suggested that it could be due increased availability of fresh and frozen food (NCIC 1990). Cancer of the cervix for women has also been declining and is generally attributed to improved hygiene and more recently, the effect of pap smear screening (NCIC 1990).

It is clear that the baseline rates for a population that is exposed today will probably not be exactly the same in

ten years as it is now or might even differ significantly in 20 or more years. However, mortality rates for all cancers as a group have remained fairly constant since 1970. In view of the difficulty in assessing what the future trends could be, the assumption of rates staying the same over time could be considered as good as any for projecting lifetime cancer risks.

It must also be remembered that national mortality rates represent the "average" rate in the population. Cancer development is a complex biological process that involves many factors, such as genetic, physiological, and environmental, that may influences the subsequent risk of cancer for a given individual. The life-table, therefore, only models the cancer risk for the "average" individual.

## 4.4 Life-table Quantities

The life-table uses the following quantities in evaluating the lifetime cancer risks following radiation exposure:

u age at risk or attained age;

T age at exposure;

d(T) absorbed dose received at age T;

$\lambda_i(u)$ age- and sex-specific baseline cancer mortality rate for cancer site i;

$f_i(d)$ dose response for cancer site i following an

absorbed dose d;

$g_i(\beta)$ excess relative risk for cancer site i, where $\beta$ depends on sex, age-at-exposure, and time-since-exposure;

$dp_i(u,T)/du$ conditional risk coefficient: conditional probability of radiation-induced death for cancer site i at attained age u resulting from an exposure at an earlier age T;

$R(u,T)$ unconditional risk coefficient: unconditional probability of radiation-induced death for cancer i at attained age u resulting from an exposure at an earlier age T;

$m(u)$ age- and sex-specific baseline mortality rate for all causes of death in unexposed cohort;

$m_E(u,T)$ age- and sex-specific baseline mortality rate from all causes of death in cohort exposed at an earlier age T;

$S(u|T_0)$ conditional survival function: probability of a cohort alive at age $T_0$ surviving to age u;

$S_E(u|T_0)$ conditional survival function for an exposed cohort;

$e(u)$ expected remaining lifetime for a cohort at age u;

$e_E(u)$ expected remaining lifetime for an exposed cohort at age u;

Note: The term "individual" will be used to refer to a cohort

of individuals at a specific age or range of ages in the life-table population, that is, the "average" individual.

4.4.1 Conditional risk coefficient, dp(u,T)/du

The conditional risk coefficient refers to the increased probability of cancer death at attained age u resulting from a radiation exposure at an earlier age T. The risk is conditional in that it can only be expressed if an individual is alive at age u. It may also be thought of as a differential probability expressing the fraction of the total committed lifetime risk that will be expressed at age u.

For an individual receiving an absorbed dose d at cancer site i at age T, the conditional risk coefficient at attained age u is given by

$$\frac{dp_i(u,T)}{du} = \lambda_i(u)\ f_i(d)\ g_i(\beta) \qquad (17)$$

where

$dp_i(u,T)/du$ is the conditional risk coefficient for cancer type i;

$\lambda_i(u)$ is the age- and sex-specific baseline cancer mortality rate for cancer site i of the exposed population;

$f_i(d)\ g_i(\beta)$ is the excess relative risk coefficient given by the BEIR V preferred risk models.

The conditional risk for all cancers will be the sum of the coefficients at the individual cancer sites.

Chronic exposure

Typical exposures relevant to radiation protection are usually spread out over a number of years. Assuming each increment of dose contributes independently to the cancer risk[29], the conditional risk coefficient at cancer site i for a chronic exposure starting at age $T_o$ will be given by

$$\frac{dp_i(u)}{du} = \lambda_i(u) \int_{T_o}^{u} f_i(d(t))\, g_i(\beta)\, dt \qquad (18)$$

Note that a dose and dose rate effectiveness factor may be needed for assessing low-level prolonged exposures.

4.4.2 Age-specific mortality rate following exposure, $m_E(u)$

The age- and sex-specific mortality rates for an exposed cohort will include the additional risk of all radiation-induced cancers combined. If m(u) denotes the baseline rate before exposure, the rate after exposure, $m_E(u)$, is

$$m_E(u) = m(u) + \frac{dp(u)}{du}$$

The background age- and sex-specific rates of the 1988 Canadian population are illustrated in figure 4.6.

4.4.3 Conditional survival function, S(u|T)

In section 4.3, the survival function, S(u), was

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[29] If additional radiation dose does not influence the promotion or progression of initiated cells produced by earlier doses, then this assumption will be valid at low doses where the dose-response is independent of dose rate.

defined as the probability of an individual surviving from birth to age u. It is also convenient to define the probability of an individual living at age T surviving to an older age u. This quantity is known as the conditional survival function and is given by

$$S(u|T) = \frac{S(u)}{S(T)} \qquad (20)$$

Similarly for an exposed cohort,

$$S_E(u|T) = \frac{S_E(u)}{S_E(T)} \qquad (21)$$

where

$S(u|T)$ is the probability of surviving to age u given that an individual is alive at age T; and

$S_E(u|T)$ is the survival probability for an exposed individual.

For convenience, this quantity will be referred to simply as the survival function.

4.4.4 Unconditional risk coefficient, R(u,T)

As explained above, the conditional risk coefficient represents the excess risk that is committed for an older age in the future. The unconditional risk coefficient takes into account than at exposed individual must be able to survive to that older age by multiplying the conditional risk by the survival function. The probability of dying from a radiation-induced cancer at site i as a result of an earlier exposure at age T is then

$$R_i(u,T) = S_E(u|T) \frac{dp_i(u)}{du} \qquad (22)$$

where

$dp_i(u,T)/du$ is the conditional risk coefficient for cancer site i; and

$S_E(u|T)$ is the survival probability for the exposed individual.

4.4.5 Expected remaining lifetime, e(u)

The expected remaining lifetime, e(u), expresses the average remaining years to be lived by an individual of age u. In terms of a life-table population, it is defined as the ratio of the number of person years to be lived by all individuals living at age u in the stationary population to the total number of persons living at that age, that is

$$e(T) = \frac{1}{l(T)} \int_T^\infty L(u)\, du \qquad (23)$$

where

e(u) is the expected remaining lifetime for an individual of age u;

l(u) is the number of persons living at age u; and

L(u) is the person years to be lived in the next year.

Person years to be lived in the next year can be estimated by assuming the number of deaths occurring in the year is uniformly distributed. That is, those individuals dying would, on average live a half year. Therefore the person

years lived in the next year will be given by the number of people alive at the beginning of the year subtracted by the person years not lived by those who died,

$$\begin{aligned} L(u) &= l(u) - \frac{1}{2} d(u) \\ &= l(u) - \frac{1}{2}[l(u+1) - l(u)] \\ &= \frac{l(u) + l(u+1)}{2} \end{aligned}$$

Alternatively, the expected remaining lifetime may be approximated by[30],

$$e(T) = \int_T^\infty S(u|T)\, du \qquad (25)$$

4.5 Cancer Risk Attributes

The life-table quantities defined in the previous section allows the lifetime cancer risk following exposure to be measured by a number of different attributes. Six attributes will be defined and described, including

(1) lifetime risk of fatal cancer (R),

(2) excess lifetime risk (ELR),

(3) loss of life expectancy (LLE),

(4) average years lost per fatal cancer (Y),

(5) average age at radiation-induced death (A), and

(6) fraction of radiation-induced cancer deaths expressed

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[30] Pierce and Vaeth 1989b

between the attained ages $a_1$ and $a_2$

Examples are given for each attribute in terms of the projected risk for a 1988 Canadian population receiving a single hypothetical whole-body dose of 0.1 Gy (no DDREF applied).

4.5.1 Lifetime risk of fatal cancer (R(T))

The lifetime risk of fatal cancer following exposure of an individual at age T, R(T), measures the probability the person will die of radiation-induced cancer during the person's remaining lifetime. The lifetime risk for fatal cancer at site i is given by

$$R_i(T) = \int_T^{\infty} S_E(u|T) \frac{dp_i(u,T)}{du} du \qquad (26)$$

where

$S_E(u|T)$ is the survival function and

$dp_i(u,T)/du$ is the conditional risk coefficient

The lifetime R for all cancers combined is given by the sum of the projections at each individual site.

Figure 4.8 shows the variation of the lifetime risk of fatal cancer with sex, age-at-exposure, and cancer site. For leukemia, the lifetime R exhibits a wave-like pattern with age at exposure. The female breast cancer lifetime R rises quickly with increasing age at exposure under age 15 and then drops sharply for exposure ages above 15. The lifetime probability of radiation-induced respiratory cancer mortality increases

with exposure age for ages above 35 and peaks at about age 50. The lifetime risk of digestive cancers is greatest for exposure ages under 30 and the lifetime R for "other" cancers is also most prevalent at younger exposure ages.

4.5.2 Excess lifetime risk (ELR)

The excess lifetime risk following an exposure of an individual of age T measures the individual's excess probability of dying from cancer in the their lifetime. It is defined as the difference between the lifetime cancer risk projected for an exposed individual and lifetime cancer risk projected for an unexposed individual of the same age.

If the lifetime risk of dying from cancer i for an individual exposed at age T is given by

$$r_{iE}(T) = \int_T^\infty S_E(u|T) \left( \lambda_i(u) + \frac{dp_i(u,T)}{du} \right) du \qquad (27)$$

and for an unexposed individual,

$$r_i(T) = \int_T^\infty S(u|T)\ \lambda_i(u)\ du \qquad (28)$$

The excess lifetime risk will be the difference,

$$
\begin{aligned}
ELR_i(T) &= r_{iE}(T) - r_i(T) \\
&= \int_T^{\infty} S_E(u|T) \left( \lambda_i(u) + \frac{dp_i(u,T)}{du} \right) du \\
&\quad - \int_T^{\infty} S(u|T)\ \lambda_i(u)\ du \\
&= \int_T^{\infty} S_E(u|T) \frac{dp_i(u,T)}{du}\ du - \int_T^{\infty} \lambda_i(u) \left[ S(u|T) - S_E(u|T) \right] du
\end{aligned}
$$

Figure 4.9 shows the variation of the ELR with sex, age-at-exposure, and cancer site.

The first term in the above equation is simply the lifetime risk of fatal cancer following exposure. The second term corrects for the cancer risk that would have been expressed anyhow had there been no exposure, albeit later in life. The fraction of the radiation-induced cancer risk that is expected to be expressed anyhow is roughly equal to the "normal" lifetime cancer risk in the unexposed cohort. As an example, consider a cohort of 100,000 Canadian males 25 years of age receiving a single whole-body dose of 0.1 Gy. Figure 4.10 shows the projected distribution of all cancer deaths with attained age for an exposed and unexposed cohort. Assuming a DDREF=1, the number of cancer deaths occurring in the lifetime is

Exposed: 28,000 cancer deaths[32] (28.0%)

Unexposed: 26,900 cancer deaths (26.9%)

There are 1400 projected radiation-induced cancer

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[32] Computed using the computer code "Radrisk" (see section 4.8)

deaths for the exposed cohort (i.e. lifetime R = 1.4%), only 1100 of these deaths are expected to be over and above the normal number projected for the unexposed cohort (i.e. ELR = 1.1%). The relative difference between the two cancer measures indicates that 27 percent of the radiation-induced cancers would have occurred any how if there been no exposure. Note that this is equal to the value of the normal lifetime risk (27%). On average, the lifetime R for all cancers will be roughly a factor 1.2 higher than the ELR (Pierce and Vaeth 1989b).

The "correction" term in the expression for the excess lifetime risk makes site-specific projections using the ELR difficult to interpret because the expression of an excess risk at an individual site must compete with the excess risks at other sites. It is conceivable that the lifetime mortality risk at a specific cancer site could be less following whole-body exposure compared the lifetime cancer risk before exposure. That is, it is possible to have a negative excess lifetime risk. This would come about as a consequence of radiation-induced mortality risk at other cancer sites reducing both the survival probability and conditional risk of cancer death at older ages. For instance, consider the excess lifetime risk for respiratory cancer mortality in a 1988 Canadian male population following a single whole body exposure of 0.1 Gy (DDREF=1). Figure 4.11 plots both the

excess lifetime and lifetime risk of fatal cancer with age-at-exposure. For exposure ages between 10 and 30, the ELR is 2-10 times lower than the lifetime R. For exposure ages below 5, the ELR is negative although there exists a probability of radiation-induced respiratory cancer death.

The ELR has been used in the BEIR and RERF reports while the lifetime R has been used in UNSCEAR and ICRP reports. Chapter 5.0 will examine further the differences between these two measures when different methods are used for transferring excess risks between populations.

4.5.3 Loss of life expectancy (LLE(T))

The excess lifetime risk and lifetime risk of fatal cancer expresses only the magnitude of the increased cancer risk but do not convey any information regarding when the risk is expressed. The loss of life expectancy (LLE) is an attribute that does. It represents the average number of remaining years to be lived that will be lost as a result of the higher probability of dying from cancer following radiation exposure. For an individual exposed at age T, the LLE is simply the difference in the expected remaining lifetime before exposure, e(T), and the expected remaining lifetime after exposure, $e_E(T)$ (Pierce and Vaeth 1989b). This is given by

$$LLE(T) = e(T) - e_E(T)$$
$$= \int_T^\infty S(u|T)\,du - \int_T^\infty S_E(u|T)\,du \qquad (30)$$

where

$e(T)$ is the expected remaining lifetime at age T before exposure;

$e_E(T)$ is the expected remaining lifetime at age T after exposure.

The above expression can be used to calculate the loss of life expectancy resulting from all radiation-induced cancers combined, but not the LLE caused by the increase cancer risk at individual cancer sites. The LLE caused by radiation-induced cancers at site i needs to be computed as the difference in the expected years of life lost caused by cancers at that site in an unexposed cohort, $y_i(T)$ and the years lost in an exposed cohort, $y_{E,i}(T)$. The expected years of life lost can be estimated by integrating over attained age the product of the expected remaining lifetime and the conditional probability of dying from cancer i. For instance, the years of life lost due to cancer i in an unexposed cohort is given by

$$y_i(t) = \int_T^\infty e(u)\ S(u|T)\ \lambda_i(u)\ du \qquad (31)$$

and in an exposed cohort by

$$y_{E,i}(T) = \int_T^\infty e_E(u)\ S_E(u|T)\left(\lambda_i(u) + \frac{dp_i(u,T)}{du}\right) du \qquad (32)$$

The loss of life expectancy is the difference,

$$\begin{aligned} LLE(T) &= y_{E,i}(T) - y_i(T) \\ &= \int_T^\infty e_E(u)\ S_E(u|T)\left(\lambda_i(u) + \frac{dp_i(u,T)}{du}\right) du \\ &\quad - \int_T^\infty e(u)\ S(u|T)\ \lambda_i(u)\ du \\ &= \int_T^\infty e_E(u)\ S_E(u|T)\ \frac{dp_i(u,T)}{du}\ du \\ &\quad - \int_T^\infty \lambda_i(u)\ (\ e(u)\ S(u|T) - e_E(u)\ S_E(u|T)\ )\ du \\ &= \int_T^\infty S'_E(u|T)\ \frac{dp_i(u)}{du}\ du - \int_T^\infty \lambda_i(u)\left(S(u|T) - S'_E(u|T)\right) du \end{aligned} \qquad (33)$$

where

$$\begin{aligned} S'_E(u|T) &= e_E(u)\, S_E(u|T), \text{ and} \\ S'(u|T) &= e(u)\, S(u|T) \end{aligned} \qquad (34)$$

The above expression of the LLE is equivalent to the expression for the ELR except the survival function has been weighted by the expected remaining lifetime.

Figure 4.12 shows the variation of the LLE with age at exposure, sex, and cancer site. The pattern is similar to that of the lifetime R and ELR except leukemia and breast cancer have a greater weighting because these cancers are expressed earlier than other radiation-related cancers.

4.5.4 Average years lost per fatal cancer

The expression of the loss of life expectancy takes into account that there is a probability of dying of a radiation-induced cancer. The actual years of life expected to be lost when the radiation-induced cancer risk is expressed is obtain by dividing the loss of life expectancy, LLE(T), by the lifetime risk of radiation-induced cancer death, R(T). That is,

$$Y(T) = \frac{LLE(T)}{R(T)} \qquad (35)$$

where

Y(T) is the average years lost per fatal cancer

Figure 4.13 illustrates the variation of Y with age-at-exposure, sex, and cancer site. The years of life lost for radiation-induced leukemia and breast cancer depends strongly on exposure age. For exposures under the age of 20, the years lost if the radiation-induced leukemia risk is expressed is 40-55 and for breast cancer, it is about 30 years. At older exposure ages the years lost per fatal cancer decreases. The years lost for other cancers are fairly constant up to the exposure age of 50 (12-18 years), above which it declines. In general, the years of life lost is greater for females than males by 2-3 years, a result which is mainly a consequence of females having a longer life expectancy than males.

4.4.5 Average age at radiation-induced cancer death, A(T)

The age at which a radiation-induced cancer death is most likely to occur may be estimated by taking the weighted average of the distribution of radiation-induced cancer deaths with attained age. For exposure at age T, this will be given by

$$A(T) = \int_{T}^{\infty} \frac{R(u,T)}{R(T)} \, u \, du \qquad (36)$$

where

A(T) is the average expected age at radiation-induced cancer death;

R(u,T) is the unconditional risk coefficient;

R(T) is the lifetime risk of fatal cancer; and

u is age at risk

Figure 4.14 shows the variation of average age at radiation-induced cancer death with age-at-exposure, sex, and cancer site following a single hypothetical whole-body dose of 0.1 Gy to a 1988 Canadian population. For leukemia the expected age at radiation-induced death ranges from 30 to 55 for exposure under age 20; for breast cancer the average age ranges from 55 to 60 for exposure ages under 30; for respiratory cancer it is above age 65 regardless of age at exposure; and for digestive and other cancers the average expected age at radiation-induced cancer death is above 75, irrespective of exposure age.

4.5.6 Fraction of radiation-induced cancer deaths expressed between the attained ages a1 and a2

The age at expression of radiation-induced cancer deaths can also be examined by computing the fraction of deaths expected to occur between different intervals of attained age. For example, the fraction of radiation-induced deaths expected to occur between the ages a1 and a2 , $F_{a1,a2}(T)$, would be given by

$$F_{a1,a2}(T) = \int_{a1}^{a2} \frac{R(u,T)}{R(T)} du \qquad (37)$$

Figures 4.15 - 4.20 shows the variation of the fraction of radiation-induced mortality for specific cancers occurring at attained ages below 65, 65-75, 75-85, and above age 85 with exposure age for an hypothetically exposed 1988 male population[33]. For all cancers combined, over 70% of all radiation-induced are expected to be expressed above the age of 65, irrespective of exposure age. In contrast, about 80% of radiation-induced leukemia and breast cancers are expected to be expressed before age 65 for exposures under the age of 20. The expression of respiratory, digestive, and other radiation-induced cancer is similar to that for all cancers combined.

4.5.7 Summary

For a population of individuals exposed to a single

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[33] Female population used for breast cancer.

whole-body dose at age T, the expression of risk attributes that can be calculated using life-table methods are:

$$R(T) = \int_T^{\infty} S_E(u|T) \frac{dp(u,T)}{du} du \tag{38}$$

$$ELR(T) = R(T) - \int_T^{\infty} \lambda(u) (S(u|T) - S_E(u|T)) du \tag{39}$$

$$LLE(T) = \int_T^{\infty} S_E(u|T)\, du - \int_T^{\infty} S(u|T)\, du \tag{40}$$

$$Y(T) = \frac{LLE(T)}{R(T)} \tag{41}$$

$$A(T) = \int_t^{\infty} u \frac{R(u,T)}{R(T)} du \tag{42}$$

$$F_{a1,a2}(T) = \int_{a1}^{a2} \frac{R(u,T)}{R(T)} du \tag{43}$$

where

R(T) is the lifetime risk of fatal cancer;

ELR(T) is the excess lifetime cancer risk;

LLE(T) is the loss of life expectancy;

Y(T) is the average years lost per fatal cancer;

A(T) is the average age at radiation-induced cancer death; and

$F_{a1,a2}(T)$ is the fraction of radiation-induced cancer deaths

expressed between the attained ages a1 and a2

## 4.6 Exposure Scenarios

This section expands the methodology to include other types of exposure scenarios besides a single whole body-exposure at a single exposure age, including

1. single whole-body exposure to a population with a given age and sex distribution,
2. continuous annual exposures, and
3. past and (possible) future exposures.

### 4.6.1 Single whole-body exposure to a population with given age and sex distribution

For an exposed population with a mixed distribution of age and sex, lifetime cancer risks for the population can be summarized as the age and sex weighted average of the age- and sex-specific lifetime risk attributes. If X(s,T) denotes the lifetime risk attribute for a cohort of sex s exposed at age T, the weighted population average is given by

$$X = \sum_{s=1}^{2} \sum_{i=T_{min}}^{T_{max}} \frac{P(s,T)}{P_{total}} X(s,T) \qquad (44)$$

where

$P(s,T)$ is the number of persons of sex s living at age T;

$P_{total}$ is the total population (both sexes and all ages);

X is the risk attribute (R, ELR, LLE, Y, A, or $F_{a1,a2}$)

4.6.2 Continuous annual exposures

Radiation doses normally experienced in every day life and at work are at low doses, low dose rates, and protracted over many years. For the purpose of radiation protection, the lifetime risks associated with prolonged exposures are of interest. Assuming each increment of dose contributes independently to the subsequent increase in the risk of cancer mortality, the conditional risk coefficient for a constant annual dose of $d_o$ received between the ages a1 and a2 is given by

$$\frac{dp_i(u)}{du} = \frac{\lambda_i(u)}{DDREF} \int_{a1}^{u} f_i(d_o(T))\ g_i(\beta)\ dT \qquad (45)$$

where

DDREF is the dose and dose rate effectiveness factor correcting for the overestimation of the excess risk by linear extrapolation from high doses and high dose rates

Lifetime cancer risk attributes are calculated in the same manner as described in section 4.5 except the above expression is used for computing the conditional risk coefficient.

4.6.3 Past and Future Exposures

The projection of lifetime cancer risk for specific exposure histories and for future exposures could possibly be a practical tool in (a) the risk management of workers

receiving doses above the annual dose limit or in the management of workers in unplanned emergency situations or (b) in determining probabilities of causation.

If an individual living at age $T_o$ had been receiving radiation exposure starting at an age a1 and if it is assumed the living individual will receive an annual dose of $d_o$ in the future until age a2, then the conditional risk coefficients for cancer i at will be given by

$$\frac{dp_i(u)}{du} = \frac{\lambda_i(u)}{DDREF}\left[\int_{a1}^{T_o} f_i(d(T))\, g_i(\beta)\, dT + \int_{T_o}^{a_{max}} f_i(d_o)\, g_i(\beta)\, dT\right] \quad (46)$$

where

d(T) is the dose (Sv) received at age T;

$d_o$ is the annual dose (Sv) assumed to be received in the future;

a1 is the age exposure first began;

$T_o$ is the current age of individual;

a2 is the assumed age that future exposure will end; and

$a_{max}$ = u, if u<a2

= a2, if u>a2

Lifetime cancer risk attributes are calculated in the same manner as described in section 4.5 except the above expression is used for computing the conditional risk coefficient.

For an individual who had died of cancer i at age $T_o$

and whose radiation exposure started at a1, the projected probability that the cancer was caused by the radiation exposure is given by

$$P.C. = \left[ \frac{1}{\lambda_i(T_o)} \times \frac{dp_i(T_o)}{du} \right] \times 100\% = \left[ \frac{1}{DDREF} \int_{a1}^{T_o} f_i(d(T))\, g_i(\beta)\, dT \right] \times 100\% \quad (47)$$

where

P.C. is the probability of causation[33]

## 4.7 Uncertainty Analysis and Confidence Intervals

### 4.7.1 Approach used by BEIR V Committee

The BEIR V Committee found it difficult to assess the uncertainty in its excess lifetime risks projected for the U.S. population caused by the statistical errors in its preferred risk models. Difficultly arose because the distribution of the statistical error in age and time modifying parameters in models were quite skewed with different parameters within the same model often correlated (Thomas 1990, NRC 1990). The Committee chose to use Monte Carlo simulation to compute 90% confidence interval (CI) for its excess lifetime risk projections. Parameters in risk

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[33] The expression is for the case when the multiplicative method is used for transferring the excess risk between populations. For the case when the additive method is used, the baseline rate in the expression of the conditional probability would be that of the reference population. (See section 3.9 or chapter 5.0 for further detail on transferring excess risks between populations)

models were assumed to have a log normal distribution with the value of the parameter as the mean. Standard errors and correlation coefficients in parameters were computed using likelihood maximum methods. The standard errors are given in BEIR V report, but the covariant matrix containing correlation coefficients is not. Corrections were also made for parameter distributions which were not log normal but highly skewed. A total of 1,000 Monte Carlo trials were performed by random sampling of parameters from their respective distributions. For each trial, excess lifetime risks were projected for the U.S. population using life-table methods. Lower and upper bounds of the 90% confidence interval were taken as the 5th and 95th percentile of the distribution of the 1,000 ELR projections.

4.7.2 Approach used in this report

Because of time constraints, limitations in developing the necessary computer code, and the lack of a covariant matrix, confidence intervals were not computed using the BEIR V Monte Carlo approach. Alternatively, an indirect approach was used. It was assumed that the ratio of the BEIR V upper and lower 90% CI limits to the ELR point estimates given in the BEIR V Report are invariant between populations and cancer measures. Approximate 90% confidence interval were approximated by multiplying projected risks by these ratios. If the upper ratio is denoted by CI(u) and the lower ratio by

CI(l), 90% confidence intervals would be given by

Upper 90% CI limit: CI(u) x ELR or

CI(u) x R

Lower 90% CI limit: CI(l) x ELR or

CI(l) x R

No confidence intervals will be approximated for other risk attributes.

Discussion with Duncan Thomas, Professor at the University of Southern California School of Medicine and statistician for the BEIR V Committee's analyses, confirmed the approach would be valid for providing approximate values of the confidence limits (Thomas 1990).

Table 4.5 lists the values of CI(u) and CI(l) corresponding to an acute exposure to a population, lifetime continuous annual dose, and working lifetime continuous lifetime. Values are given for males, females and both sex for leukemia, non-leukemia cancers as a group, and all cancers as a group. The non-leukemia as a group values will be used to approximate the CIs for cancers of the respiratory, female breast, digestive, and other cancers.

## 4.8 Computer Code "Radrisk"

A computer code, known as "Radrisk", was developed to perform the lifetime cancer risk projections in this report. This section describes that program and its verification.

4.8.1 Description

Radrisk uses the BEIR V five preferred relative risk models and the life-table techniques described in this chapter to evaluate the various lifetime cancer risk attributes for a life-table population following the various exposure scenarios defined in section 4.6.

The program is divided into 6 parts:

1. setting of the dose record for the chosen exposure scenario;
2. calculation of the condition risk coefficients for the chosen exposure scenario using the national age- and sex-specific cancer mortality rates of the population of interest and BEIR V relative risk models (for additive transfer method baseline rates for Japan are used);
3. construction of a life-table population for an exposed and unexposed cohort using the national mortality rates for all causes of death from the population of interest, taking into account the additional risk of death from radiation-induced cancers for the exposed cohort;
4. calculation of lifetime cancer risk attributes;
5. for a single exposure at various ages at exposure, parts (2), (3), and (4) are repeated for each age at exposure and the population average computed based on the age- and sex- distribution of a general stationary life-table population (ages 1-85), working stationary life-table

population (ages 18-65), or a user defined population; and

6. choice of printout of results: (1) all risk attributes for all cancers as a group as a function of age-at-exposure and sex plus the population average; (2) distribution of radiation-induced cancer deaths with attained age; and (3) distribution of each risk attribute with age-at-exposure for each cancer site or group.

The program is also capable of computing life-table quantities and the distribution of total cancer deaths with sex and attained age by cancer site for an exposed and unexposed population.

Appendix C gives the computer code for each of the six parts.

4.8.2 Verification

The Radrisk program was verified in several steps. First, a spreadsheet was used to verify that the excess risk coefficients of the BEIR V relative risk models were being computed correctly by Radrisk. Second, life-table quantities (e.g. number of persons alive at each age, number of deaths at each age, expected remaining lifetime at each age, etc.) were computed by Radrisk using the age- and sex-specific mortality rates for all causes of death obtained from the 1980-1982 Canadian current life-table (StatsCan 1985b) and compared to the values given in the Statistics Canada publication. And

finally, lifetime risk projections were performed with Radrisk using the U.S. baseline age-, sex-, and site-specific mortality rates for cancer and all causes of death used by the BEIR V Committee and compared with the results given in the BEIR V Report[35].

Comparison with the spreadsheet calculations and Canadian current life-tables showed calculations were being performed correctly. Table 4.4 gives the comparison of Radrisk projections with those given in the BEIR V Report for three different exposure scenarios: (a) single whole-body exposure of 0.1 Gy to a U.S. life-table population, (b) continuous annual whole-body exposure of 1 mSv per year from birth over a lifetime, and (c) continuous annual whole-body exposure of 10 mSv per year from age 18 to 65. For the single-whole body exposure, the age-, sex-, and site-specific excess lifetime risks projected by Radrisk were within ± 1-3% of the values projected in the BEIR V report. The site-specific population average[36] ELRs are in good agreement except for (i) male digestive cancers (Radrisk 6% lower than BEIR V), (ii) female breast cancer (Radrisk 20% lower than BEIR V), and (iii) female other cancers (Radrisk 5% higher than BEIR V). The reason for the discrepancy in the average excess risks at these sites was discovered to be a result of BEIR V averages

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[35] Baseline rates supplied by the U.S. National Institute of Health.

[36] Age-weighted average for a 1980 U.S. life-table population

being over exposure ages separated by ten year intervals (i.e. ages 5, 15, 25,...) while Radrisk averages were performed over single ages. The discrepancy was resolved when Radrisk was made to average over ten year intervals as well. Excess lifetime risk projections by Radrisk for continuous annual exposures are in agreement with the BEIR V report as are projections of the loss of life expectancy and years lost per excess death.

From the above verifications, it was concluded that lifetime cancer risks projected by Radrisk are reliable and credible.

Table 4.1
Human epidemiological data used in the BEIR V cancer risk analysis
(Adapted from Thomas 1989)

| | |
|---|---|
| ALL SITES | |
| Atomic bomb survivors | Mortality |
| Ankylosing spondylitis patients | Mortality (excluding colon) |
| BREAST CANCER | |
| A-bomb survivors | Mortality and Incidence |
| Canadian fluoroscopy patients | Mortality |
| Massachusetts fluoroscopy patients | Incidence |
| New York postpartum mastitis patients | Incidence |
| THYROID CANCER | |
| Israeli tinea capitis irradiation patients | Incidence |
| Rochester thymus irradiation patients | Incidence |

Table 4.2
Cancer groupings used in the BEIR V analysis of cancer mortality among irradiated human populations

| Cancer Group | ICD (a) | Cancer site |
|---|---|---|
| Leukemia | 204 | Acute lymphoid leukemia only (b) |
| | 205 | Myeloid leukemia |
| | 206 | Monocytic leukemia |
| | 207 | Other specified leukemia |
| Respiratory | 160 | Nasal cavities, middle ear, sinuses |
| | 161 | Larynx |
| | 162 | Trachea |
| | 163 | Pleura |
| Breast | 174 | Female breast |
| Digestive | 150 | Oesophagus |
| | 151 | Stomach |
| | 152 | Small intestine |
| | 153 | Colon |
| | 154 | Rectum |
| | 155 | Liver and intrahepatic bile ducts |
| | 156 | Gall bladder and extrahepatic bile ducts |
| | 157 | Pancreas |
| | 158 | Retroperitoneum and peritoneum |
| | 159 | Other and ill-defined sites |
| Other | 140-209 less those above | |

(a) International Code of Diseases
(b) Chronic lymphatic leukemia not included

Table 4.3
Lower and upper limits of 90% confidence interval estimated from BEIR V ELR point estimates for various exposure scenarios

| Type of exposure | Cancer type | BEIR V ELR point estimates and 90% CI | Estimated 90% CI limits CI(l) | CI(u) |
|---|---|---|---|---|
| Single exposure to 0.1 Sv | | | | |
| Male | leukemia | 110 (50, 280) | 0.45 | 2.54 |
| | nonleukemia | 660 (420, 1040) | 0.64 | 1.57 |
| | all cancers | 770 (540, 1240) | 0.72 | 1.60 |
| Female | leukemia | 80 (30, 190) | 0.38 | 2.37 |
| | nonleukemia | 730 (550, 1020) | 0.76 | 1.39 |
| | all cancers | 810 (630, 1160) | 0.77 | 1.43 |
| Both sexes | leukemia | -- | 0.42 | 2.47 |
| | nonleukemia | -- | 0.70 | 1.48 |
| | all cancers | -- | 0.74 | 1.52 |
| Continuous lifetime exposure to 1 mSv/yr | | | | |
| Male | all cancers | 520 (410, 980) | 0.79 | 1.88 |
| Female | all cancers | 660 (500, 930) | 0.76 | 1.41 |
| Continuous exposure to 10 mSv/yr from age 18 until age 65 | | | | |
| Male | all cancers | 2880 (2150, 5460) | 0.75 | 1.90 |
| Female | all cancers | 3070 (2510, 4580) | 0.82 | 1.49 |

Table 4.4
Validation of Radrisk computer code. Comparison of excess lifetime risk (excess cancers per 100,000 persons) for a U.S. population following a single whole-body exposure of 0.1 Gy given in the BEIR report and that projected by the Radrisk computer code

Single exposure to 0.1 Sv (a, b, c)

Males

| Age at exposure | All cancers combined. | | | Leukemia | | | Respiratory | | | Breast | | | Digestive | | | Other | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Radrisk | BEIR V | R/B | Radrisk | BEIR V | R/B | Radrisk | BEIR V | R/B | Radrisk | BEIR V | R/B | Radrisk | BEIR V | R/B | Radrisk | BEIR V | R/B |
| 5 | 1282 | 1276 | 1.00 | 110 | 111 | 0.99 | 16 | 17 | 0.94 | | -- | | 364 | 361 | 1.01 | 794 | 787 | 1.01 |
| 15 | 1150 | 1144 | 1.01 | 108 | 109 | 0.99 | 52 | 54 | 0.96 | | -- | | 373 | 369 | 1.01 | 617 | 612 | 1.01 |
| 25 | 921 | 921 | 1.00 | 35 | 36 | 0.97 | 121 | 124 | 0.98 | | -- | | 392 | 389 | 1.01 | 374 | 372 | 1.01 |
| 35 | 556 | 566 | 0.98 | 60 | 62 | 0.97 | 232 | 243 | 0.95 | | -- | | 28 | 28 | 1.00 | 236 | 233 | 1.01 |
| 45 | 604 | 600 | 1.01 | 106 | 108 | 0.98 | 358 | 353 | 1.01 | | -- | | 22 | 22 | 1.00 | 118 | 117 | 1.01 |
| 55 | 624 | 616 | 1.01 | 169 | 166 | 1.02 | 399 | 393 | 1.02 | | -- | | 15 | 15 | 1.00 | 41 | 42 | 0.98 |
| 65 | 488 | 481 | 1.01 | 194 | 191 | 1.02 | 278 | 272 | 1.02 | | -- | | 10 | 11 | 0.91 | 6 | 7 | 0.86 |
| 75 | 259 | 258 | 1.00 | 168 | 165 | 1.02 | 89 | 90 | 0.99 | | -- | | 5 | 5 | 1.00 | -- | -- | -- |
| 85 | 106 | 110 | 0.96 | 96 | 96 | 1.00 | 14 | 17 | 0.82 | | -- | | -- | -- | -- | -- | -- | -- |
| Average (c) | 765 | 770 | 0.99 | 110 | 110 | 1.00 | 190 | 190 | 1.00 | | -- | | 160 | 170 | 0.94 | 305 | 300 | 1.02 |

Females

| Age at exposure | All cancers combined | | | Leukemia | | | Respiratory | | | Breast | | | Digestive | | | Other | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Radrisk | BEIR V | R/B | Radrisk | BEIR V | R/B | Radrisk | BEIR V | R/B | Radrisk | BEIR V | R/B | Radrisk | BEIR V | R/B | Radrisk | BEIR V | R/B |
| 5 | 1546 | 1532 | 1.01 | 74 | 75 | 0.99 | 49 | 24 | 2.04 | 129 | 129 | 1.00 | 662 | 655 | 1.01 | 632 | 625 | 1.01 |
| 15 | 1577 | 1566 | 1.01 | 71 | 72 | 0.99 | 71 | 70 | 1.01 | 295 | 295 | 1.00 | 660 | 653 | 1.01 | 480 | 476 | 1.01 |
| 25 | 1190 | 1178 | 1.01 | 29 | 29 | 1.00 | 126 | 125 | 1.01 | 52 | 52 | 1.00 | 687 | 679 | 1.01 | 296 | 293 | 1.01 |
| 35 | 563 | 557 | 1.01 | 46 | 46 | 1.00 | 210 | 208 | 1.01 | 44 | 43 | 1.02 | 74 | 73 | 1.01 | 189 | 187 | 1.01 |
| 45 | 547 | 541 | 1.01 | 74 | 73 | 1.01 | 280 | 277 | 1.01 | 21 | 20 | 1.05 | 71 | 71 | 1.00 | 101 | 100 | 1.01 |
| 55 | 512 | 505 | 1.01 | 119 | 117 | 1.02 | 276 | 273 | 1.01 | 6 | 6 | 1.00 | 65 | 64 | 1.02 | 45 | 45 | 1.00 |
| 65 | 393 | 386 | 1.02 | 149 | 146 | 1.02 | 175 | 172 | 1.02 | -- | -- | -- | 52 | 52 | 1.00 | 16 | 16 | 1.00 |
| 75 | 232 | 227 | 1.02 | 129 | 127 | 1.02 | 74 | 72 | 1.03 | -- | -- | -- | 27 | 26 | 1.04 | 3 | 3 | 1.00 |
| 85 | 92 | 90 | 1.02 | 75 | 73 | 1.03 | 14 | 15 | 0.93 | -- | -- | -- | 4 | 4 | 1.00 | -- | -- | -- |
| Average (c) | 810 | 810 | 1.00 | 85 | 80 | 1.06 | 155 | 150 | 1.03 | 55 | 70 | 0.79 | 285 | 290 | 0.98 | 230 | 220 | 1.05 |

(a) DDREF=1
(b) Average weighted over age distribution of a 1980 U.S. stationary lifetable population (ages 1-85)
(c) BEIR V values taken from table 4-3, NRC 990
R/B: Ratio of Radrisk projections to BEIR V

Continuous annual exposures (a)

| Type of exposure | Excess lifetime risk (b,c) | | | LLE (d) | | |
|---|---|---|---|---|---|---|
| | Radrisk | BEIR V | R/B | Radrisk | BEIR V | R/B |
| Continuous lifetime exposure to 1 mSwyr | | | | | | |
| Male | 525 | 520 | 1.01 | 9080 | 9100 | 1.00 |
| Female | 600 | 600 | 1.00 | 10580 | 10500 | 1.01 |
| Continuous exposure to 10 mSwyr from age 18 until age 65 (e) | | | | | | |
| Male | 2870 | 2880 | 1.00 | 42690 | 42200 | 1.01 |
| Female | 3075 | 3070 | 1.00 | 52680 | 51600 | 1.02 |

(a) BEIR V values taken from table 4-2, NRC 1990
(b) All cancers combined
(c) DDREF=1
(d) Loss of life expectancy (years lost per 10,000 persons)
(e) Computed for exposures at ages 18 to 64
R/B: Ratio of Radrisk projections to BEIR V

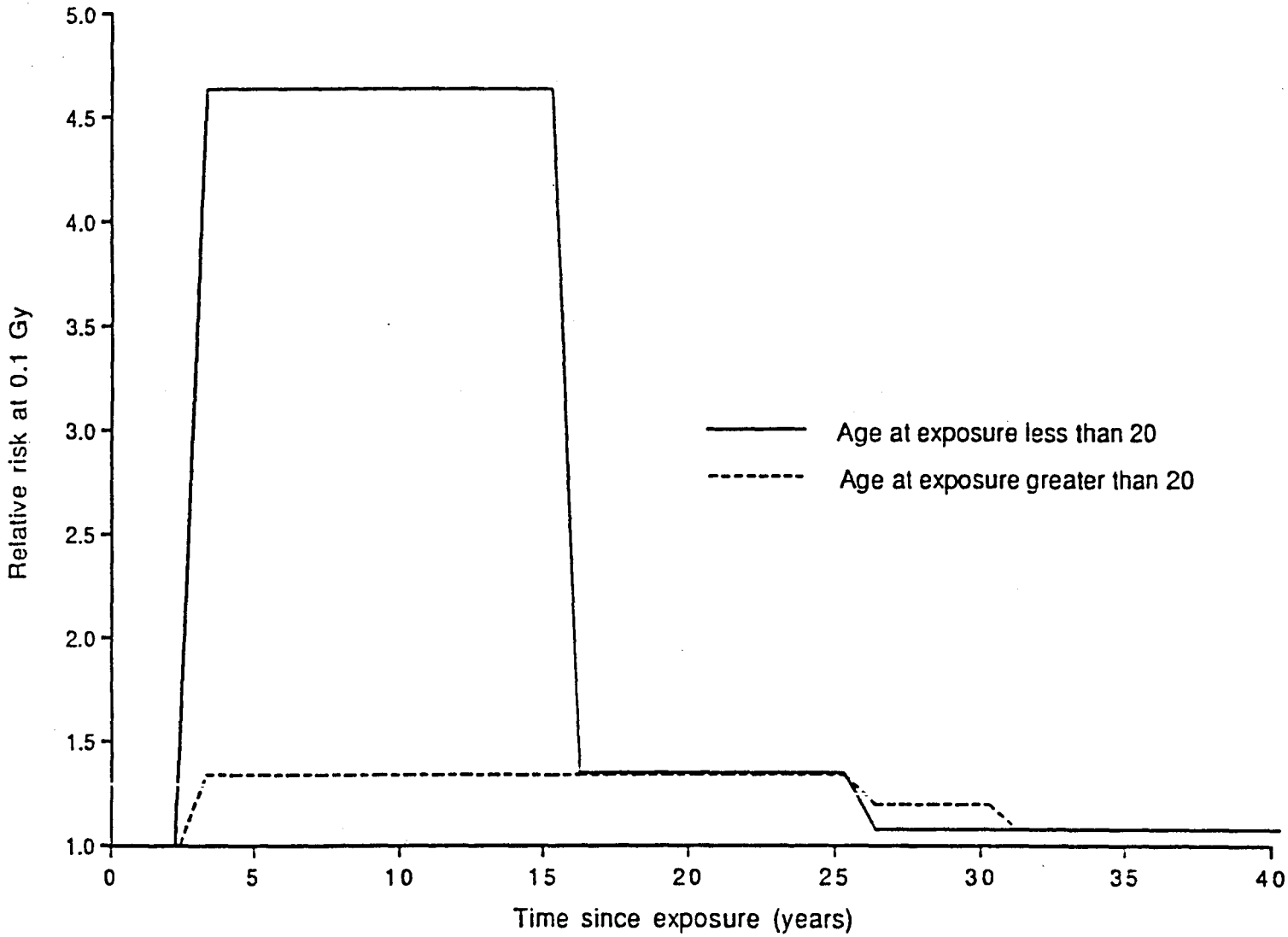



Figure 4.1
BEIR V relative risk model for leukemia mortality

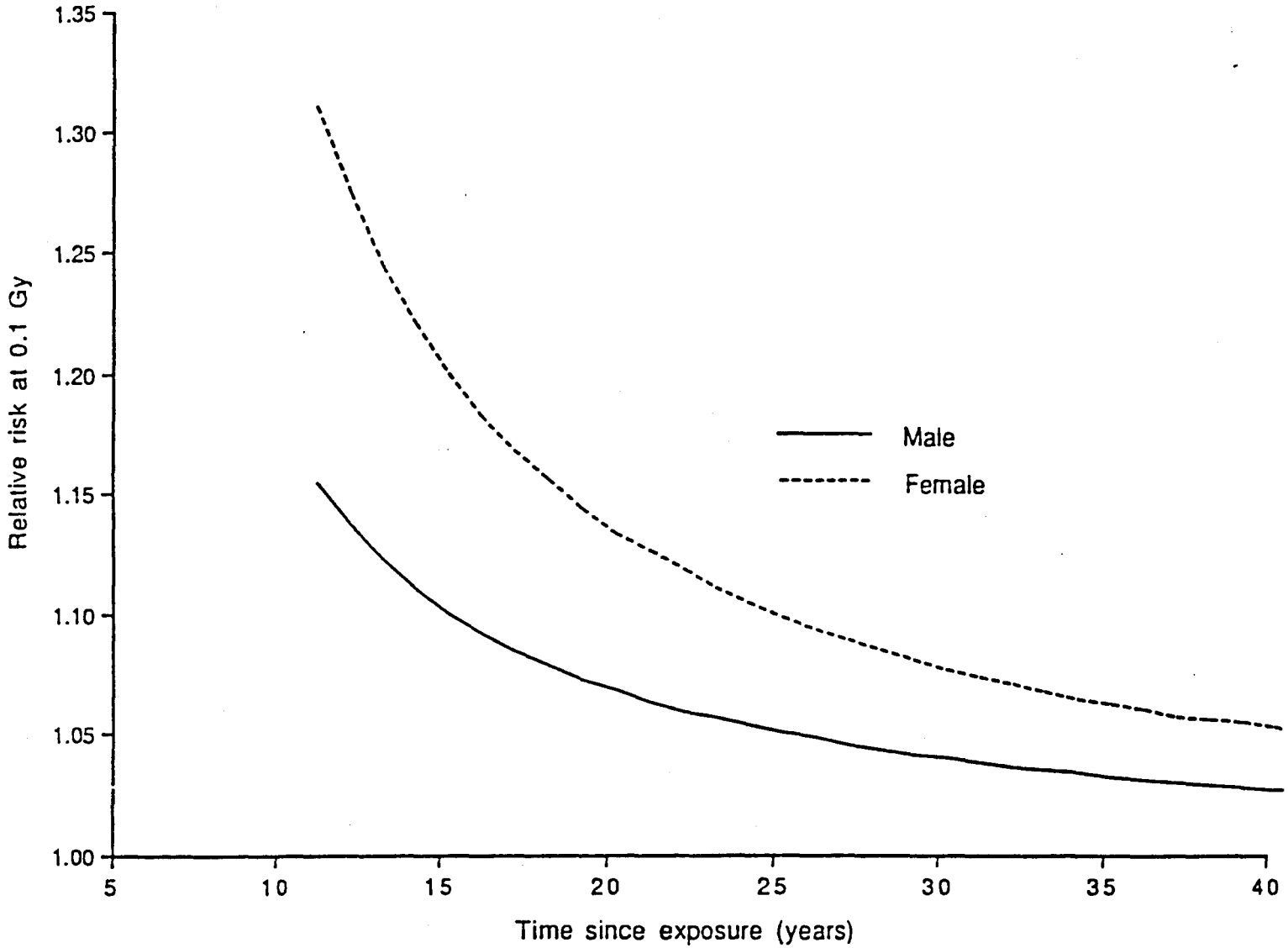



Figure 4.2
BEIR V relative risk model for respiratory cancer mortality

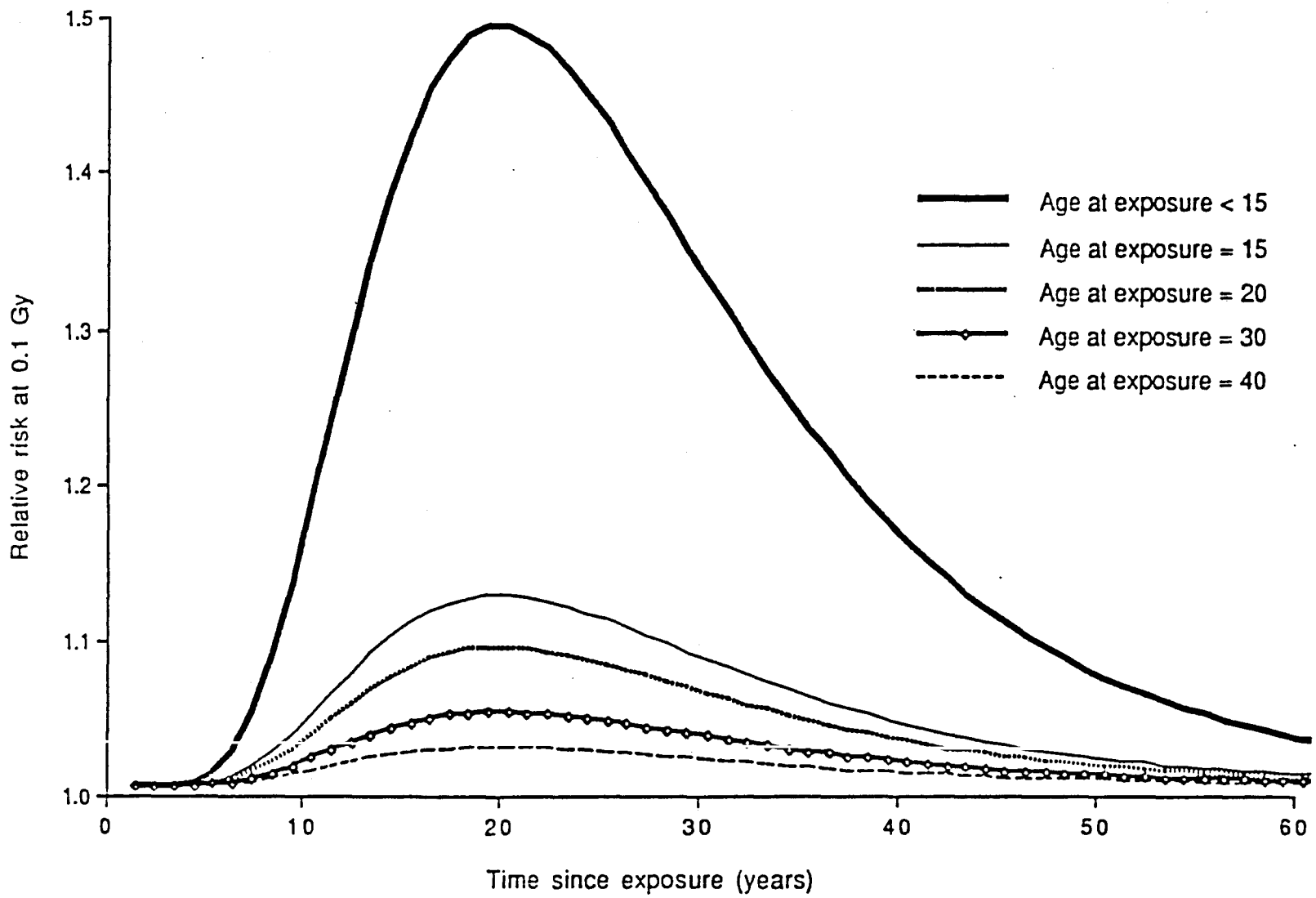



Figure 4.3
BEIR V relative risk model for breast cancer mortality

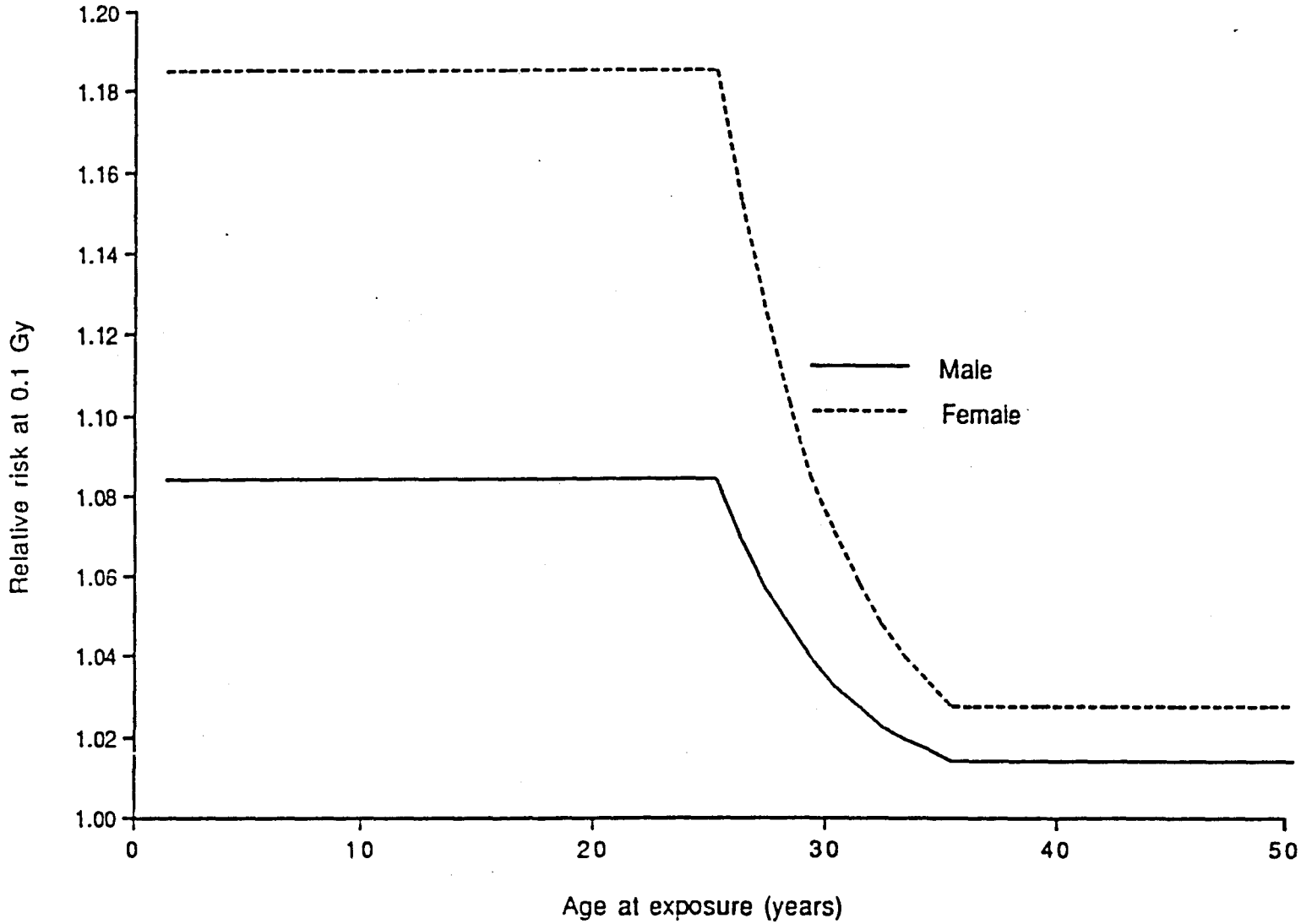



Figure 4.4
BEIR V relative risk model for digestive cancer mortality

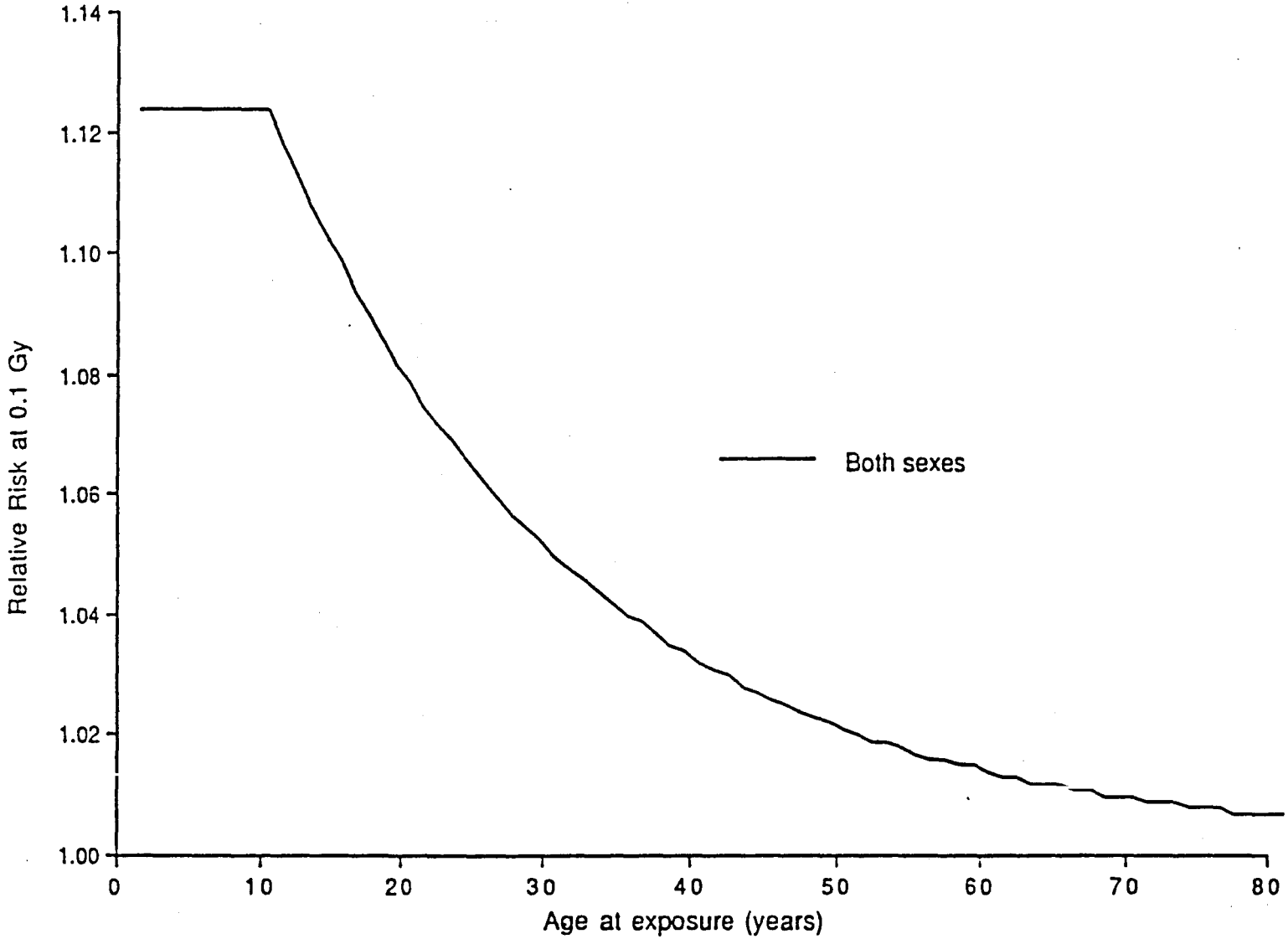



Figure 4.5
BEIR V relative risk model for other cancer sites combined mortality

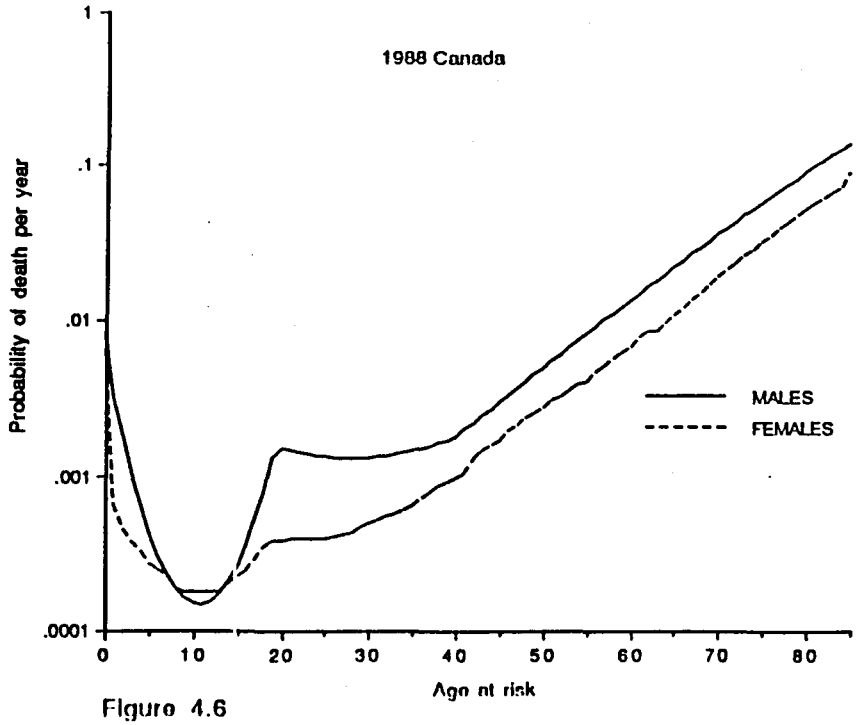



Figure 4.6
1988 Canada national age- and sex-specific mortality rates for all causes of death

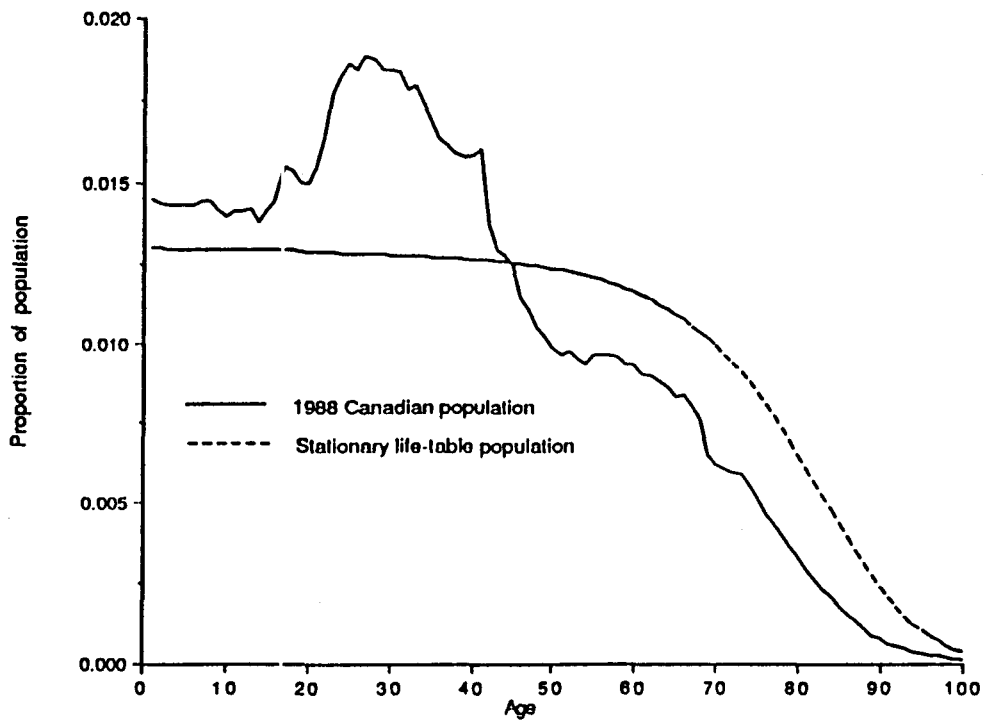



Figure 4.7
Age distribution of the 1988 Canadian population
and the corresponding life-table age distribution (Average over both sexes)

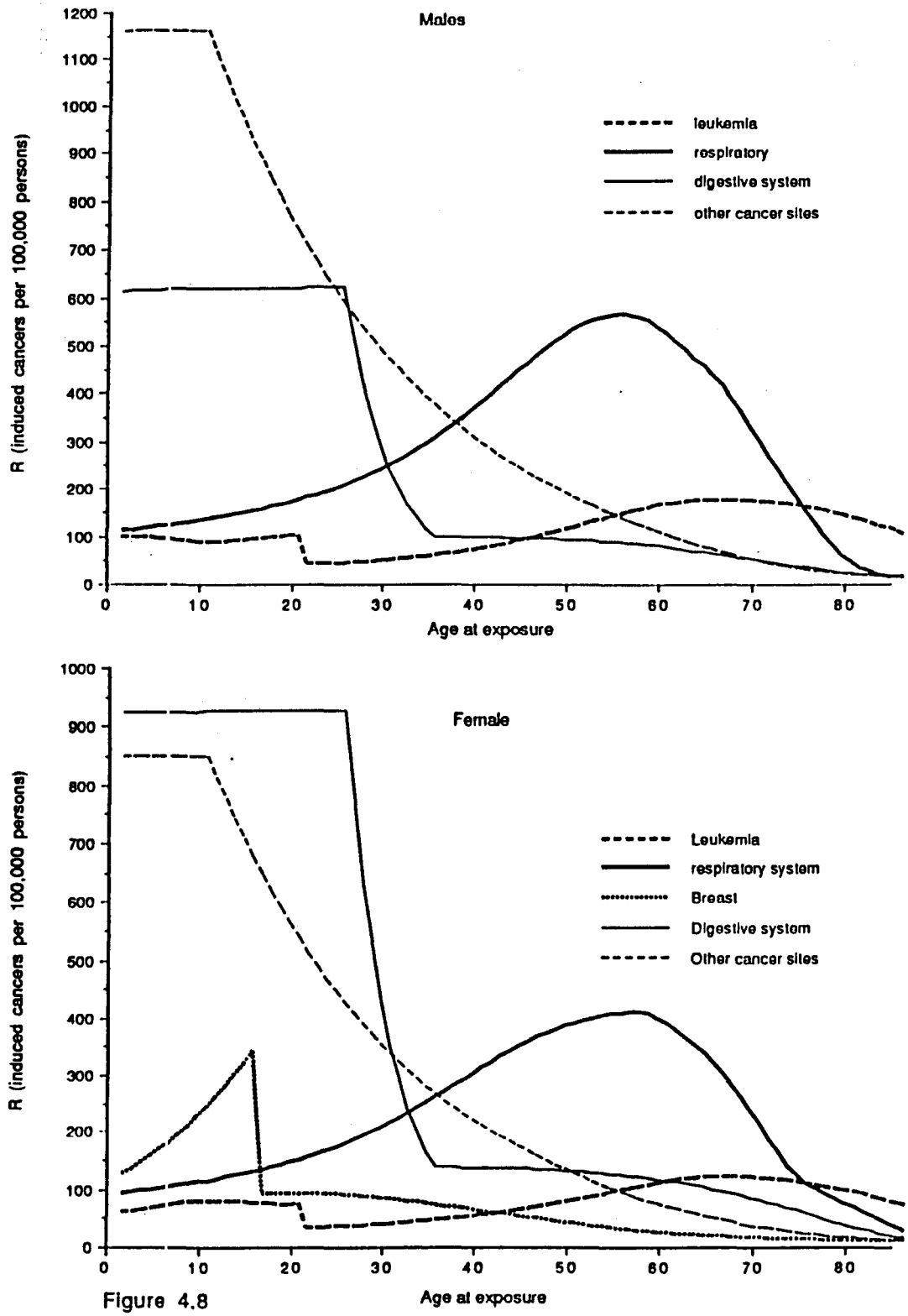
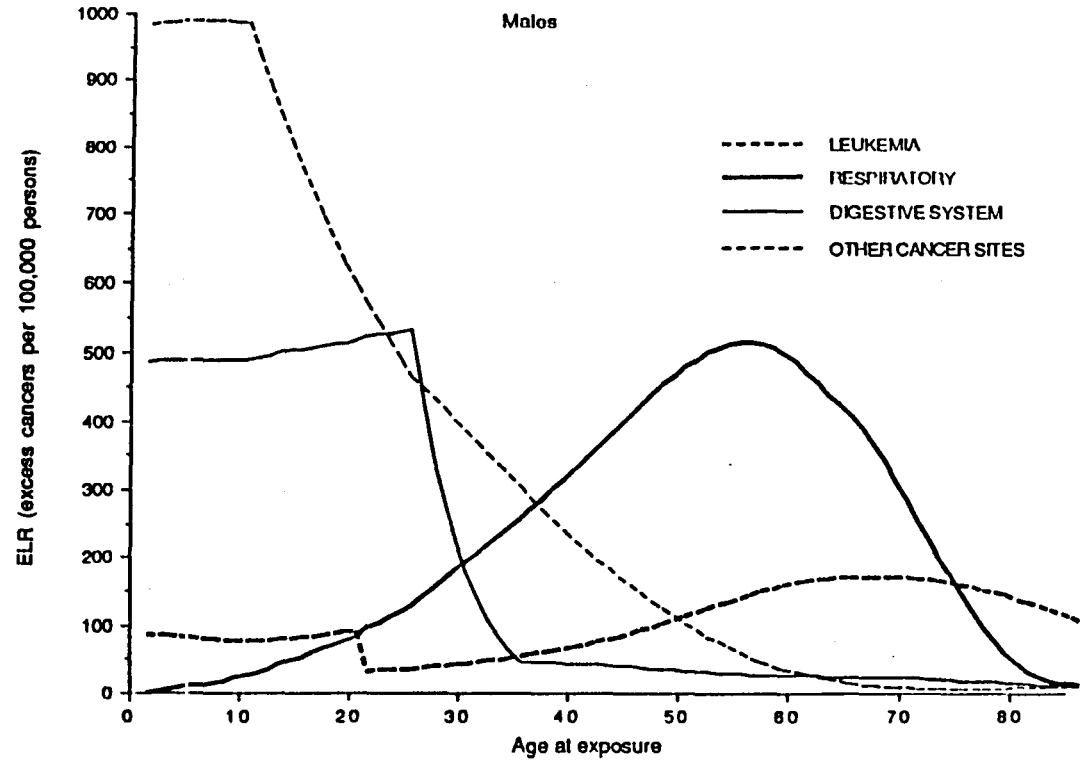



Figure 4.8
Projected lifetime risk of fatal cancer by age, sex, and cancer group resulting from a single whole-body exposure of 0.1 Gy (DDREF=1)



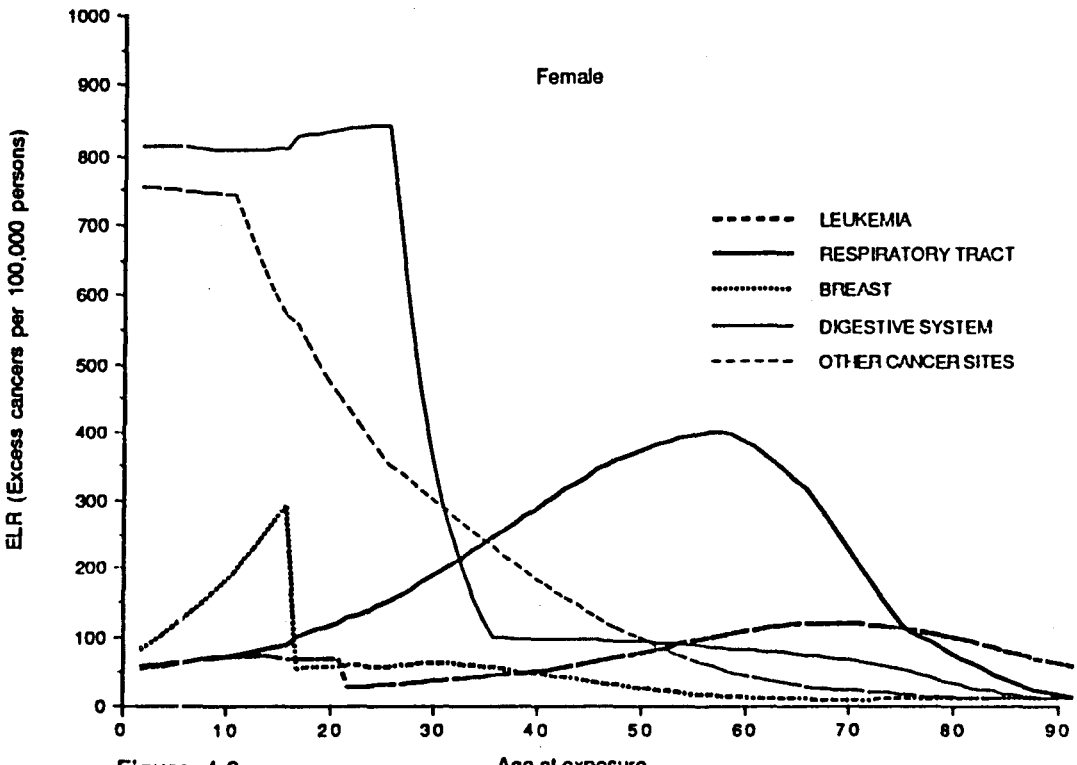




Figure 4.9
Projected excess lifetime risk by age, sex, and cancer group resulting from a single whole-body exposure of 0.1 Gy (DDREF=1)

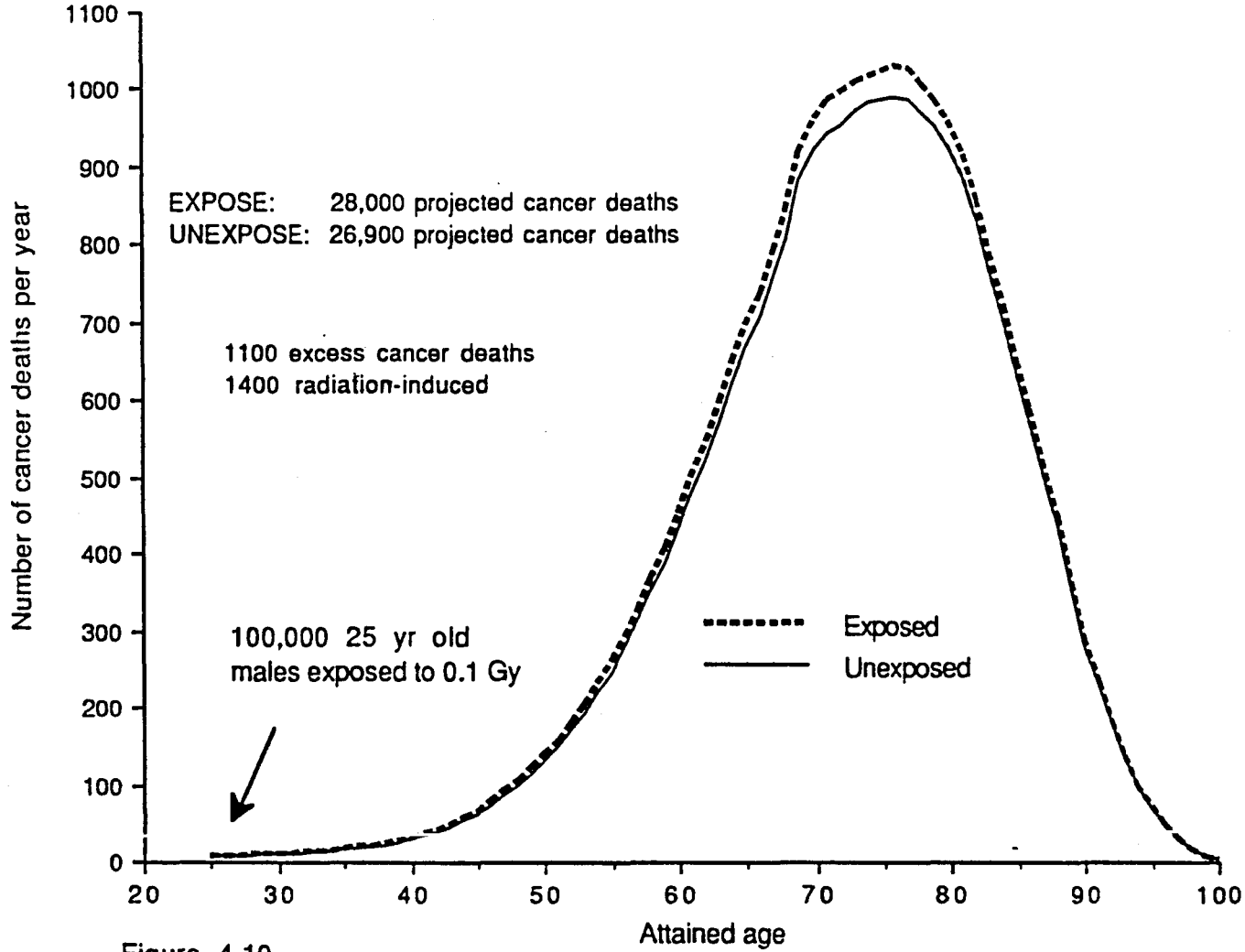



Figure 4.10
Projected distribution of total cancer deaths with attained age for 100,000 unexposed 25 year old Canadian males and 100,000 25 year old Canadian males receiving a single whole-body exposure of 0.1 Gy (DDREF=1)

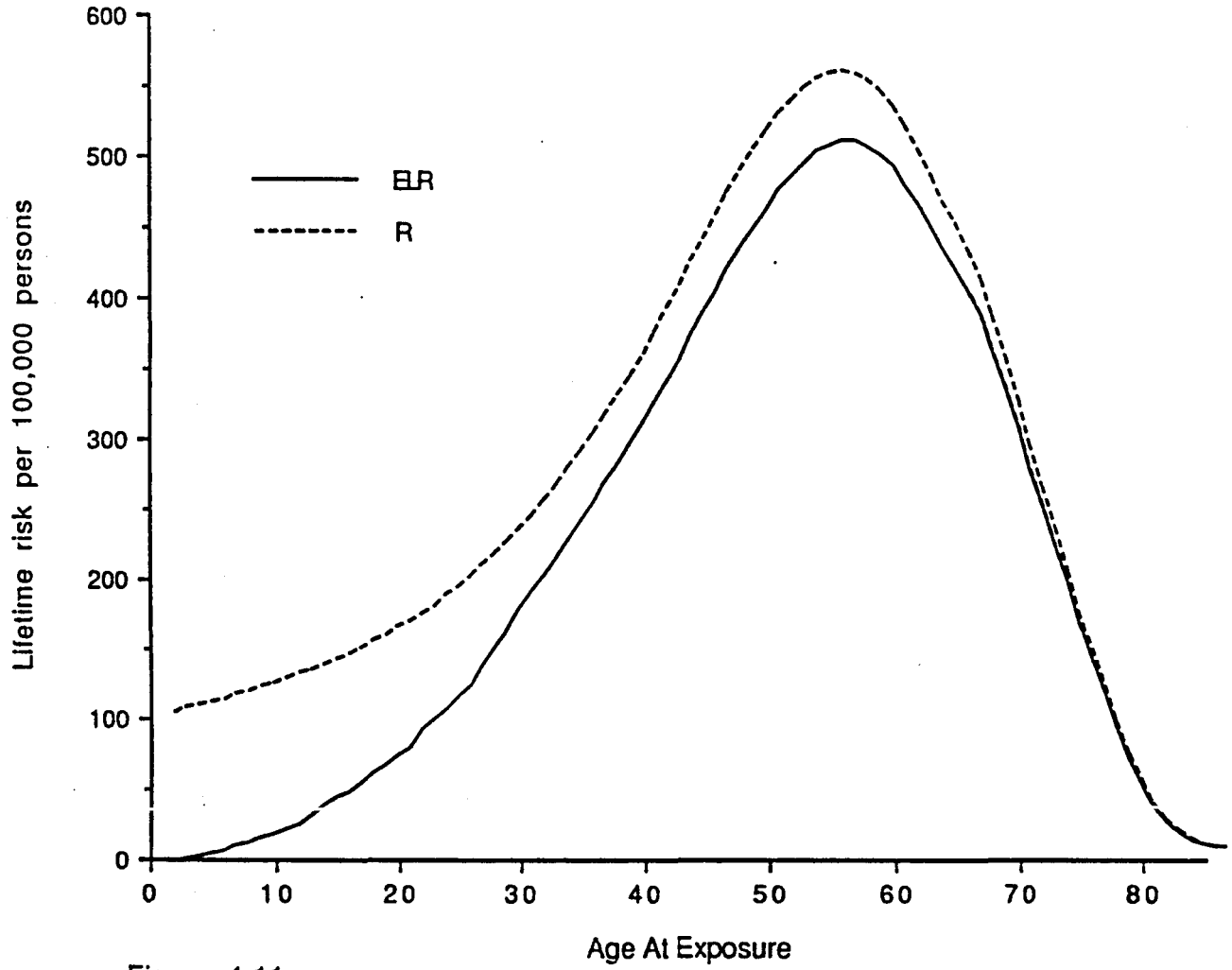



Figure 4.11
Projected excess lifetime risk (ELR) and lifetime risk of fatal cancer (R) for male respiratory cancer by age at exposure resulting from a single whole-body exposure of 0.1 Gy (DDREF=1)

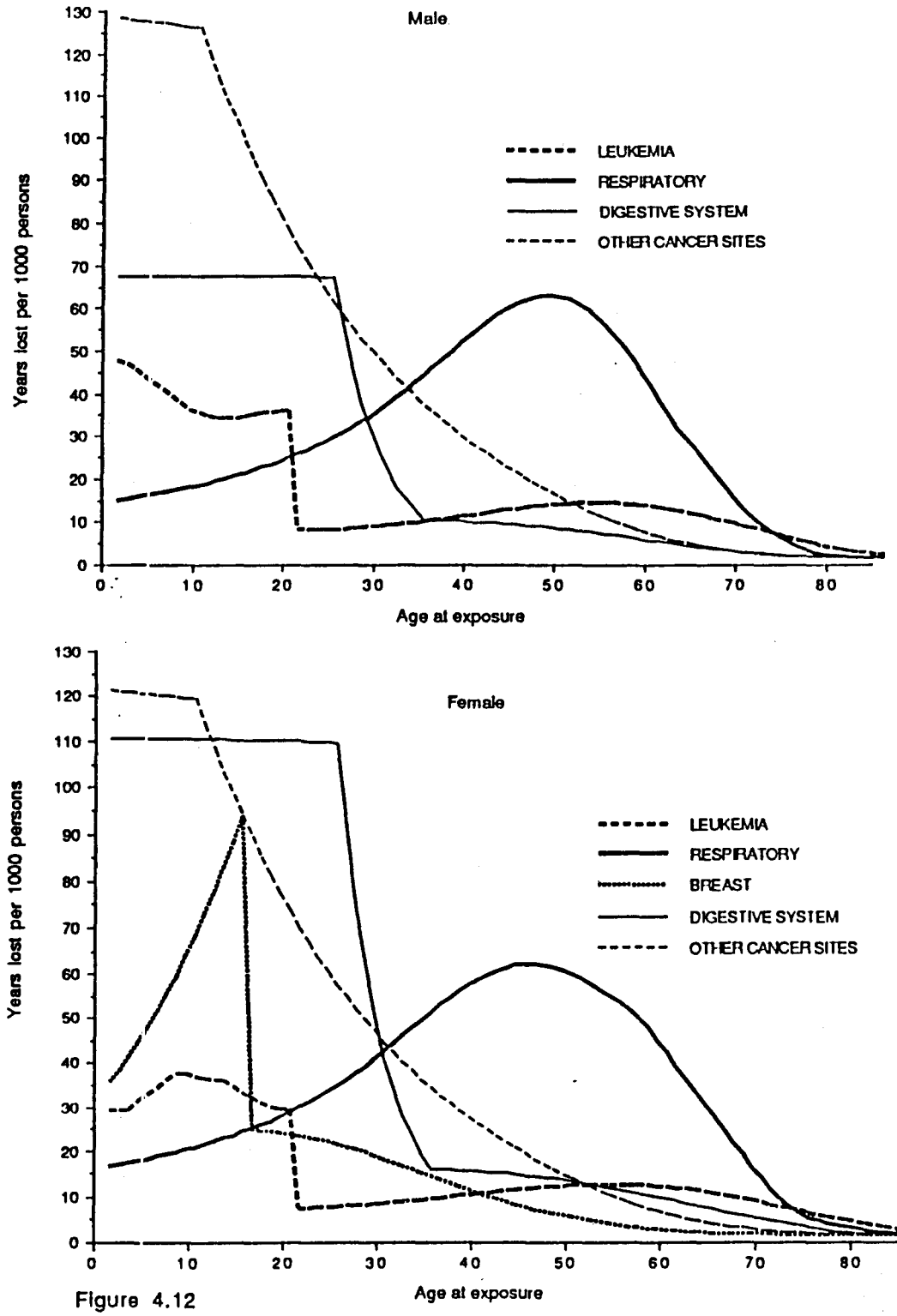
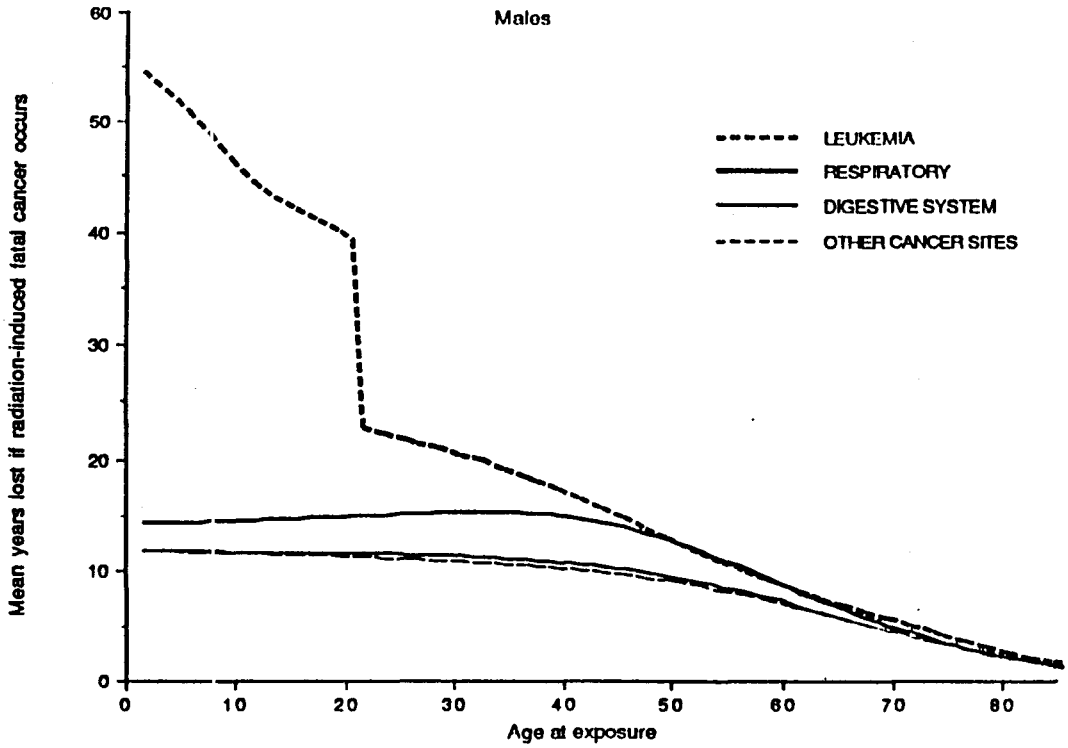



Figure 4.12
Projected loss of life expectancy by age, sex, and cancer group resulting from a single whole-body exposure of 0.1 Gy (DDREF =1)



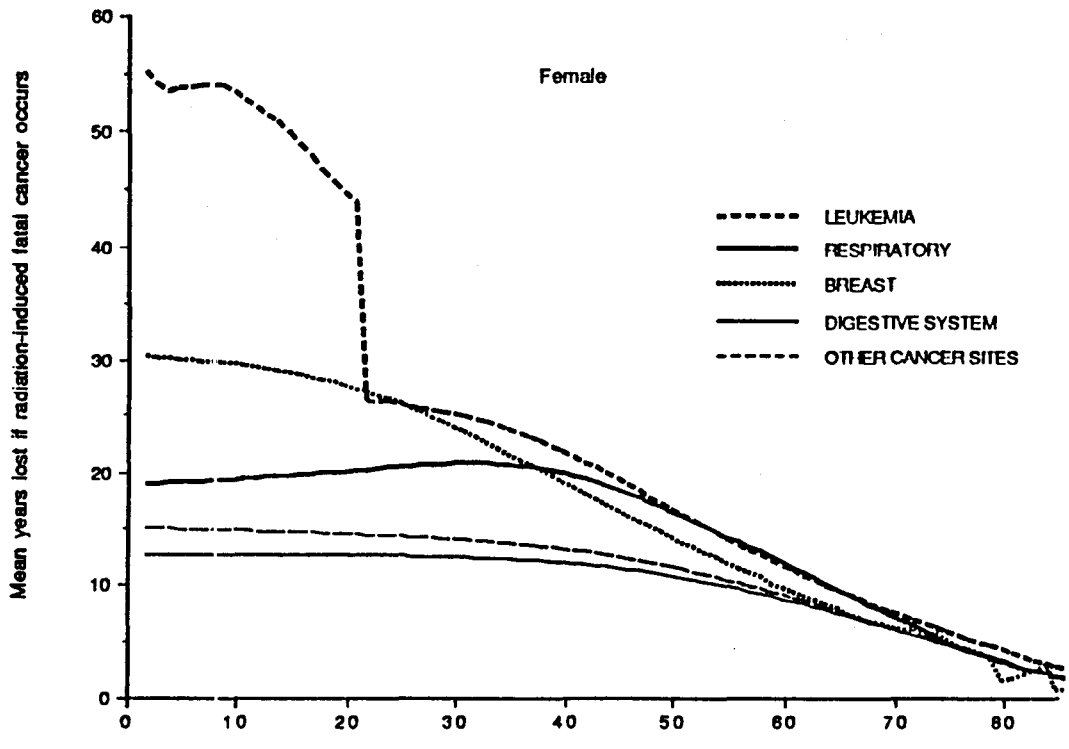
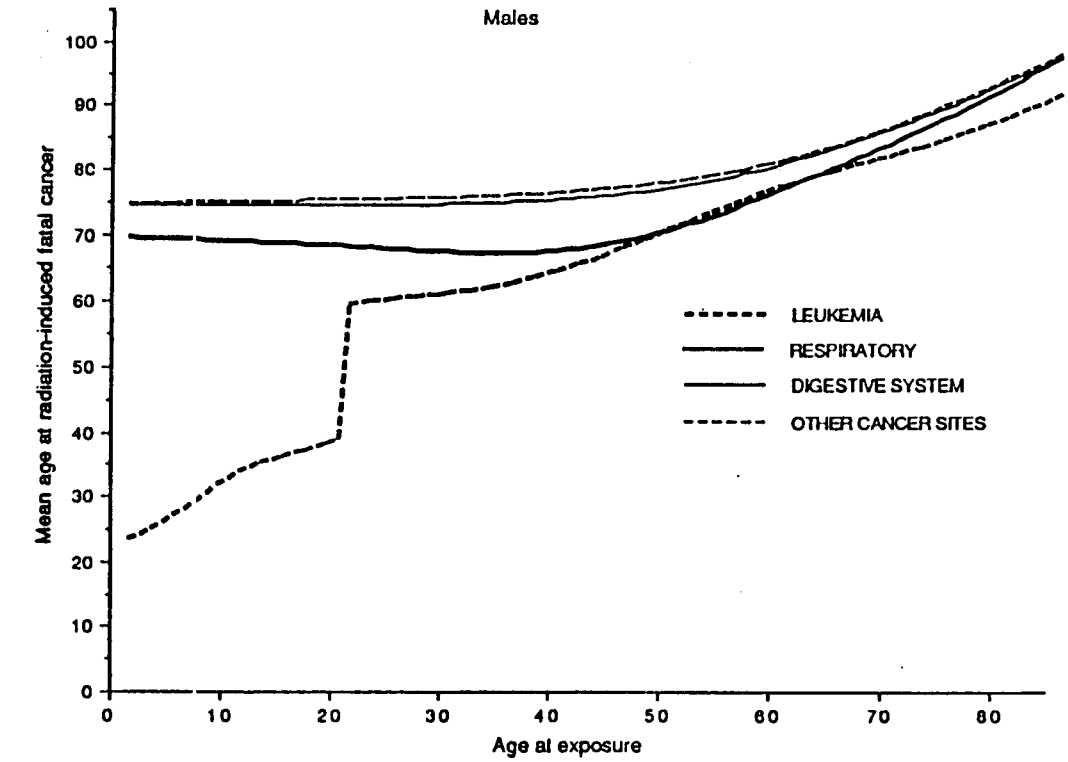




Figure 4.13
Average years lost per fatal cancer by age, sex, and cancer group resulting from a single whole-body exposure of 0.1 Gy (DDREF=1)



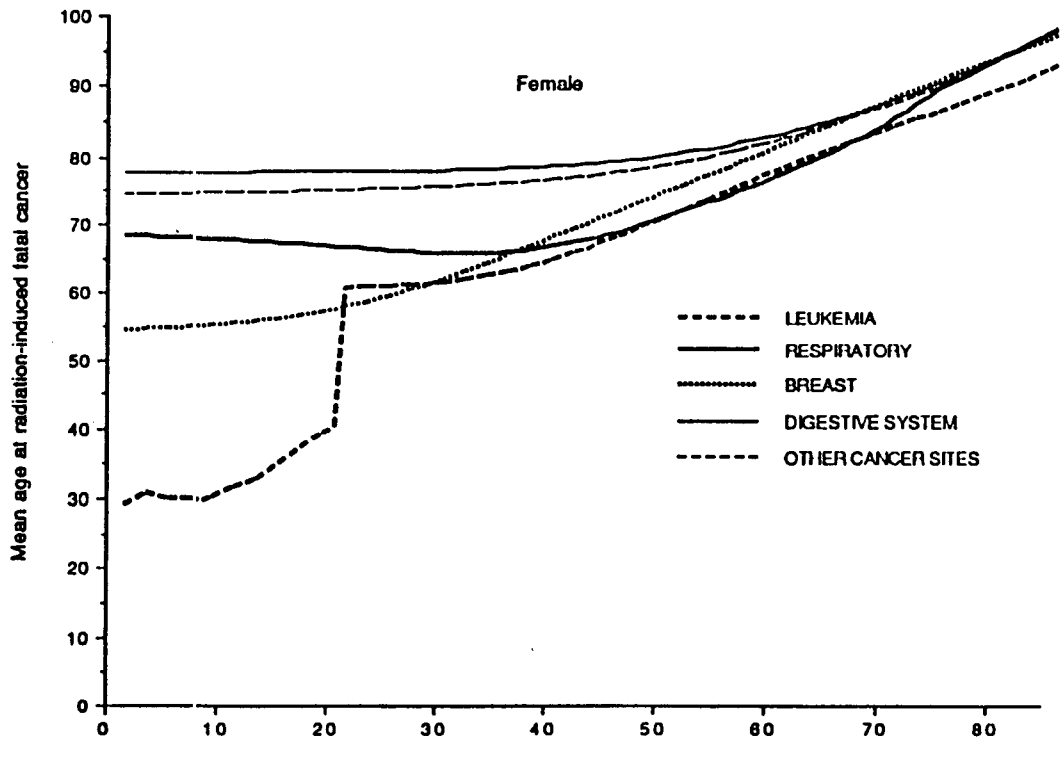




Figure 4.14
Projected mean age at radiation-induced cancer death by age, sex, and cancer group resulting from a single whole-body exposure of 0.1 Gy (DDREF=1)

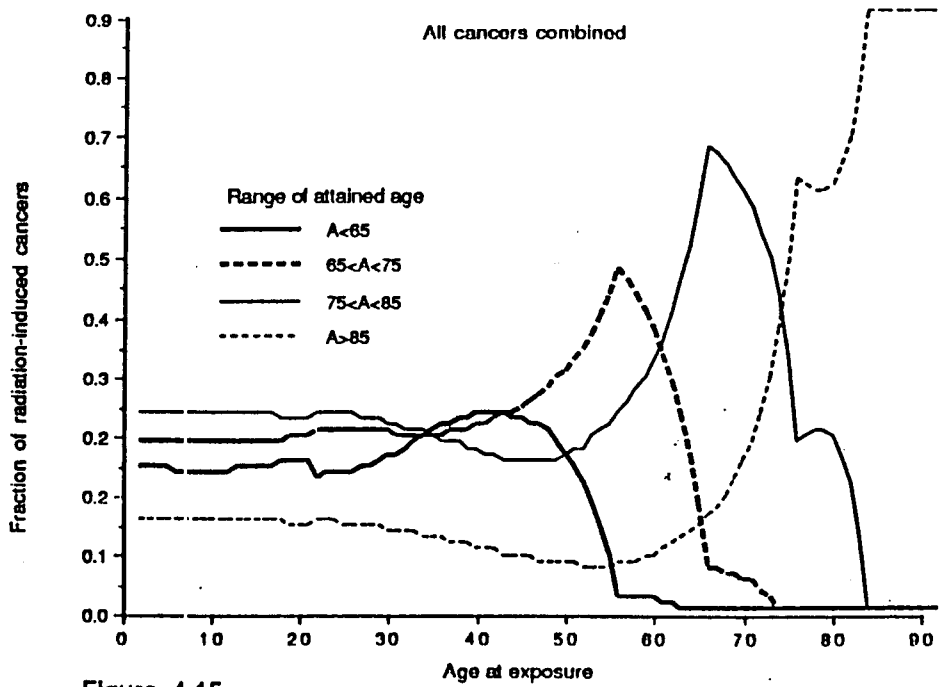



Figure 4.15
Projected fraction of the lifetime number of radiation-induced cancers all combined occurring at different ranges of attained age resulting from a single whole-body exposure of 0.1 Gy (DDREF=1) (1988 Canadian male population)

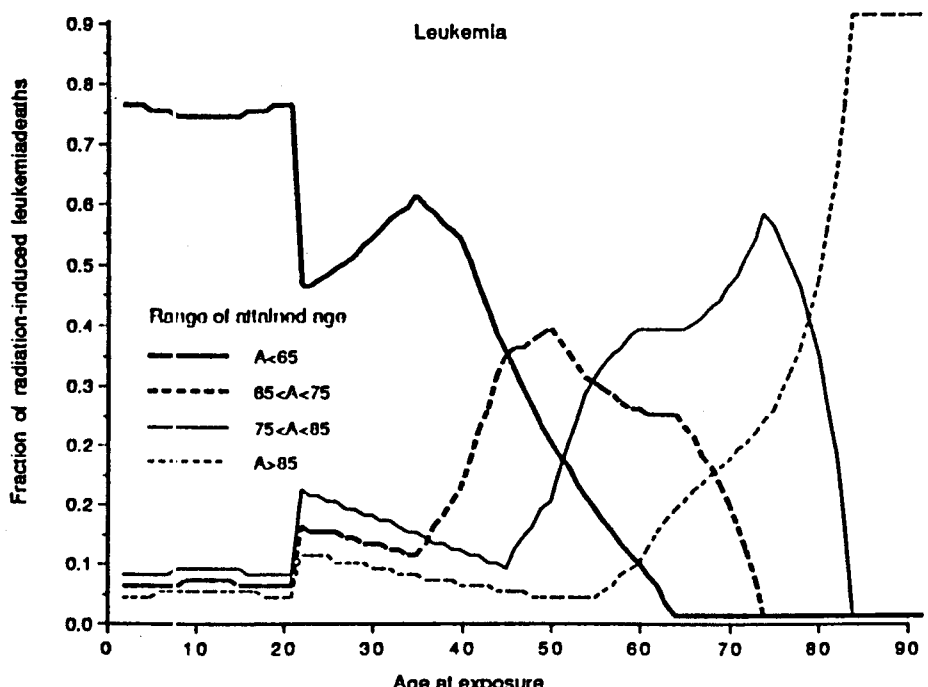



Figure 4.16
Projected fraction of the lifetime number of radiation-induced leukemia deaths occuring at different ranges of attained age resulting from a single whole-body exposure of 0.1 Gy (DDREF=1) (1988 Canadian male population)

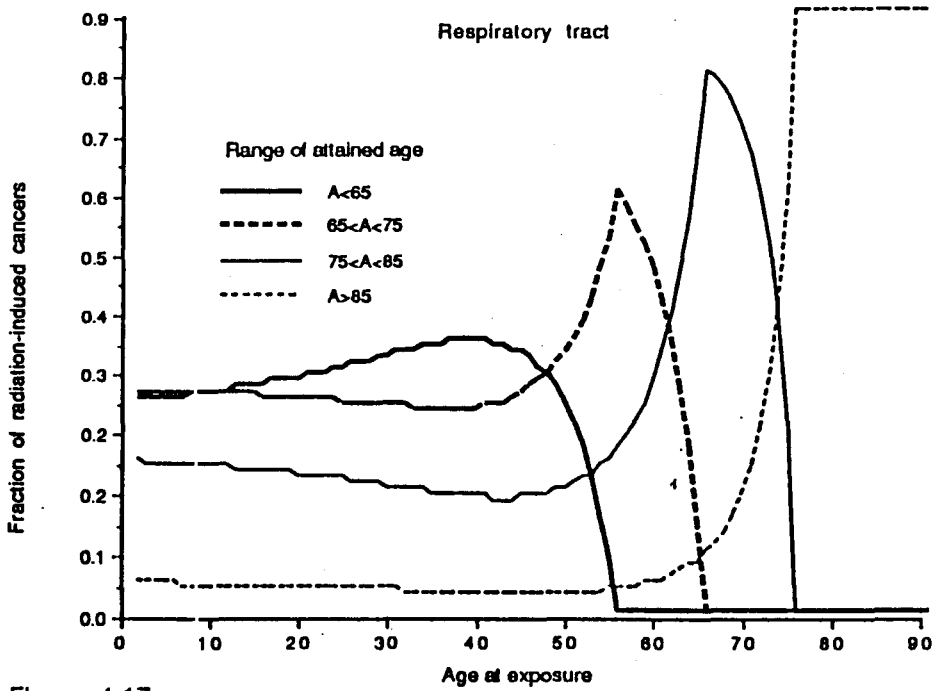



Figure 4.17
Projected fraction of the lifetime number of radiation-induced respiratory cancers occurring at different ranges of attained age resulting from a single whole-body exposure of 0.1 Gy (DDREF=1) (1988 Canadian male population)

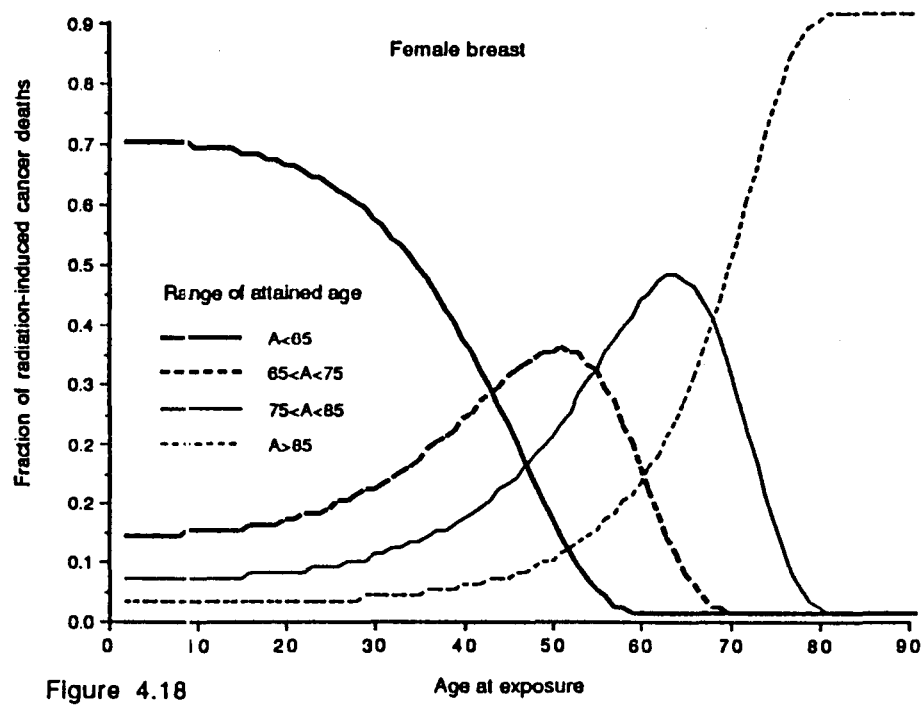



Figure 4.18
Projected fraction of the lifetime number of radiation-induced breast cancers occurring at different ranges of attained age resulting from a single whole-body exposure of 0.1 Gy (DDREF=1) (1988 Canadian female population)

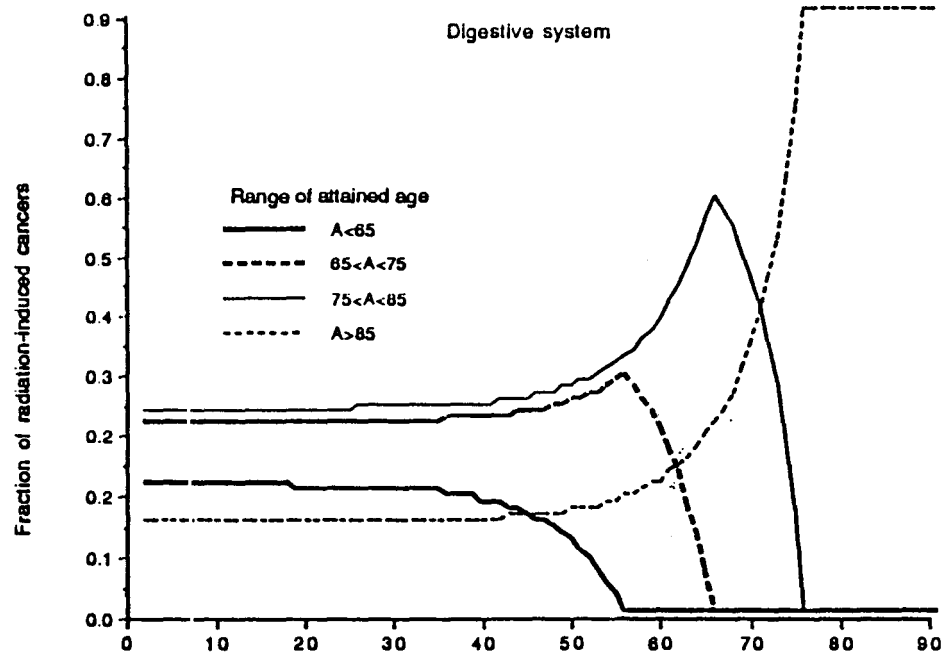



Figure 4.19
Projected fraction of the lifetime number of radiation-induced digestive cancers occurring at different ranges of attained age resulting from a single whole-body exposure of 0.1 Gy (DDREF=1) (1988 Canadian male population)

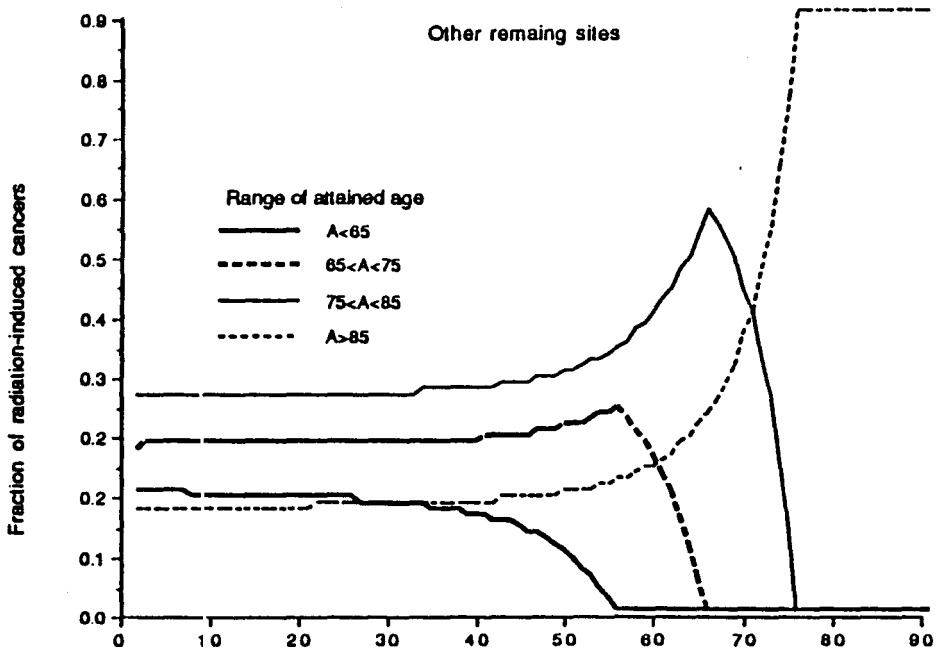



Figure 4.20
Projected fraction of the lifetime number of radiation-induced cancers at other sites occurring at different ranges of attained age resulting from a single whole-body exposure of 0.1 Gy (DDREF=1) (1988 Canadian male population)

# 5.0 Effect on Lifetime Risk Projections of the Choice of Method for Transferring Excess Risk Coefficients Between Populations

## 5.1 Introduction

Section 3.7 discussed the issue of using excess cancer risk coefficients derived from the Life Span Study of the atomic bomb survivors to project the potential lifetime cancer risks in other populations where baseline cancer rates are substantially different from those in Japan. It was explained that either an additive method (which transfers the absolute excess risk) or multiplicative method (which transfers the excess relative risk) was plausibly for transferring risks. Presently there is no general agreement as to what the best method is. This chapter examines the effect the choice of transfer method will have on the projected lifetime risk of fatal cancer (R) and excess lifetime risk (ELR) resulting from a single hypothetical whole-body exposure of 0.1 Gy in the general populations of

1. 1988 Canada,
2. 1984 Japan,
3. 1982 Canada, and
4. 1980 U.S..

The 1984 Japanese baseline cancer rates are used to compute

absolute excess risks for the additive transfer method.

Differences in sex-specific lifetime cancer risks are also compared as well as differences between two measures of the cancer risk: the lifetime R and the ELR.

## 5.2 Materials and Methods

### 5.2.1 Data Sources for baseline rates

Baseline age- and sex-specific cancer rates and overall mortality rates for the 1984 Japanese population were supplied by Dale Preston of the Radiation Effects Research Foundation and those for the U.S. by David Noel of the U.S. National Institute of Health. Baseline cancer rates for 1982 Canada were taken from 1982 Canadian cancer statistics (StatsCan 1985a) and overall mortality rates from the 1980-82 Canadian life-tables (StatsCan 1985b). The 1988 Canadian rates were calculated using data from causes of death tables and population estimates for 1988 supplied by Statistics Canada[36] (StatsCan 1990a, 1990b). The baseline rates by sex, age, and cancer site are given in appendix B.

### 5.2.2 Lifetime risk projections

Lifetime risk projections are carried out using the BEIR V preferred relative risk models and the life-table methods described in chapter 4.0. Absolute excess risk coefficients are taken to be the conditional risk coefficient

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[36] See appendix A for details of calculations.

computed using the 1984 Japanese baseline cancer rates. Projections are performed assuming a single hypothetical whole-body exposure of 0.1 Gy to populations, no DDREF[37], life-table age distribution in populations, and equal numbers of males and females. The term "population risk" will be used to refer to the age-weighted averaged projected risk for a population. The term "North American" will be used to denote averages over 1988 Canada, 1982 Canada, and 1980 U.S. populations.

Ninety percent confidence intervals (CIs) for projections are approximated using the ratio of the upper and lower 90% CIs to the ELR point estimates given in the BEIR V Report (see section 4.7). Any differences in population projected risks are considered statistically significant if the 90% confidence intervals do not overlap.

## 5.3 Differences in Baseline Mortality Rates

### 5.3.1 Cancer baseline rates

Table 5.1 shows the standardized[38] sex- and site-specific cancer mortality rates of the 1988 Canada, 1982

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[37] Note that, except for leukemia, use of a DDREF of 1 may overestimate the lifetime cancer risks at low doses and low dose rates by a factor of 2 or more (see section 3.5). The purpose of this chapter is to compare the relative differences between population risks rather than the absolute magnitude. Chapters 6.0 and 7.0 examine the projected lifetime cancer risks at low doses.

[38] Standardized to the age distribution of the 1988 Canadian population

Canada, 1980 U.S., and 1984 Japanese populations. Among North American (N.A.) males, "other" and respiratory cancers are the most prevalent, representing 85% of the total cancer rate. Among females, cancers of the breast, digestive system, and "others" dominate, accounting for 85% of the total. Except for breast cancer, cancer rates in males are higher than for females by factors from 1.6 (digestive cancers) up to 3.2 (respiratory cancers).

In Japan, anatomical differences between sexes are similar to the N.A. populations, but the pattern and magnitude among cancers are substantially different. Digestive cancer is the most prominent cancer. It represents about 60% of the total baseline rate and is about a 1.5 to 2 times greater than rates for North American male and females, respectively. Rates for other individual cancers are significantly lower in Japan by factors of 1.2 to 4, depending on the sex and cancer. Interestingly, the higher rate of digestive cancer in Japan compensates the lower rates for other cancers so that overall, the total standardized rate for all cancers combined does not differ greatly between Japan and N.A. populations (10% difference between males and 25% between females).

5.3.2 Baseline rates for all causes of death

Figure 5.1 plots the age-and sex-specific mortality rates from all causes of death for the four populations. Japan has the lowest level of mortality, the U.S. the highest, and

Canada is intermediate. Males, on average, have about twice the risk of death than do females. The figure shows that for Canada, the minimum risk of death occurs at about age 10 where the risk is about 1 in ten thousand. The risk of death increases sharply thereafter until age 25 where it levels off and remains constant at about 1 in a thousand for males and 0.5 in a thousand for females until ages 35-40. Above this age, the risk increase log-linearly. The risk of death at age 65 is about 1 in a hundred and at ages greater than 85, it is more than 1 in ten.

The level of mortality in the populations can be compared by computing the average life expectancy at birth. Table 5.2 gives the average life expectancies for males and females in the four populations[39]. Because of its lower mortality rates, Japan has a longer life expectancy by 3-4 years compared to North American populations. In all populations the life expectancies for females are about 6-7 years longer than males.

## 5.4 Results

Tables 5.3 and 5.4 gives an overall detailed summary of the projected lifetime R and ELR by population, sex, radiation-related cancer, and transfer method. Differences between population projections are presented in table 5.5.

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[39] Computed as the expected remaining lifetime at birth using the computer code "Radrisk" (see chapter 4.0)

Table 5.6 shows the variation in North American projected risks caused by the choice of transfer method, table 5.7 the variation by sex, and table 5.8 shows the differences between projections for the lifetime R and ELR.

Figures 5.2 to 5.7 plots the variation of the projected lifetime R with population, sex, and transfer method. Table 5.9 summarizes the effect the choice of transfer method on North American projected lifetime R. Projected risks for leukemia and for all cancers combined are fairly comparable between populations and transfer methods, but differ significantly at other individual sites. In North American populations, lifetime risks projected by the two transfer methods varied by average factors of 1.12, 1.4, 1.7, 3.5, 1.7, and 2.5 for all cancers combined, leukemia, cancers of the respiratory tract, female breast, digestive system, and other remaining, respectively. Most of these differences are comparable to the uncertainty caused by statistical errors in the risk models.

Sex differences in site-specific lifetime risks of fatal cancer projected for North American populations were small compared to the uncertainties caused by the choice of transfer method and statistical errors (see table 5.7). In general, males had higher increased lifetime risks of fatal cancer for leukemia, respiratory and other cancers by factors of 1.3, 1.4, and 1.25, respectively, while females had a

higher estimated increased risk of digestive cancer by a factor of 1.3. The higher risk for digestive cancers plus the additional risk of breast cancer for females, tended to compensate for other lower projected risks. As a result when the lifetime risk for all cancers is combined, there is no real difference in projections between females and males.

Projections for the lifetime risk of fatal cancer using the additive transfer method provides an opportunity to evaluate the influence that the risk from other causes of death have on the value of projected cancer risks. Table 5.5 (bottom table, left column) shows that populations with a higher life expectancy (i.e. lower yearly risk of death) have higher projected risks of radiation-induced cancer because of more people surviving to older ages where the committed, or conditional, radiation-induced cancer risks are greatest. The results suggest that for every year increase in "normal" life expectancy in a population, the projected lifetime risk of radiation-induced cancer mortality should be expected to increase by 3%, everything else being equal. Using this reasoning, one can conclude that females will have a proportionately higher projected lifetime cancer risk due to their higher life expectancy compared to males.

With regard to the differences in the two measures of the cancer risk, the lifetime fatal cancer risk is on average about a factor 1.2 times greater than the ELR. Projections

using the ELR need to be interpreted carefully because the measure does not consider a "premature" radiation-induced cancer as an excess if that cancer is expected to be expressed later in life anyhow. That means populations with higher baseline cancer rates will have a greater likelihood of premature cancer risks of being expressed later in life, and therefore, a smaller fraction of the radiation-induced cancer risk being considered as excess risk. For instance, when the additive transfer method is used to project the lifetime breast cancer risks for Japanese and Canadian female populations, the lifetime R does not differ between populations, but the ELR is 4 times higher in Japan. This could mistakenly be interpreted as suggesting Japanese women are four times more likely to develop radiation-induced breast cancer than Canadian women. In fact, the difference results because the baseline risk for Canadian women is 4 times higher than Japanese women, thus causing a greater subtraction from the total risk of radiation-induced breast cancer mortality of the risk that would be expressed anyhow even if no exposure had occurred.

## 5.5 Conclusions

In view of the difficulty in choosing between transfer methods and the significant effect the choice has on North American site-specific risk projections, it would seem that

the best approach is to carry out lifetime risk projections using both transfer methods and then average the results. With regard to cancer measures, the excess lifetime risk has several undesirable characteristics and is susceptible to producing misleading projections. Therefore, the lifetime risk of fatal cancer following exposure would seem the preferable measure and will be used for the remainder of this report.

Table 5.1
National cancer mortality rates by population, sex, and cancer grouping (deaths per 10E05 persons per year) (a)

Male

| Population | Leukemia | Respiratory | Breast | Digestive | Other | Total |
|---|---|---|---|---|---|---|
| 1988 Canada | 5.5 | 73 | -- | 59 | 75 | 213 |
| 1982 Canada | 5.3 | 73 | -- | 62 | 71 | 211 |
| 1981 U.S. | 8.1 | 71 | -- | 54 | 74 | 207 |
| 1984 Japan | 5.0 | 39 | -- | 120 | 28 | 192 |
| N.A. (b) | 6.3 | 72 | -- | 58 | 73 | 210 |
| Ratio N.A./Japan | 1.25 | 1.85 | -- | 0.50 | 2.60 | 1.10 |

Female

| Population | Leukemia | Respiratory | Breast | Digestive | Other | Total |
|---|---|---|---|---|---|---|
| 1988 Canada | 3.2 | 24 | 27 | 34 | 41 | 129 |
| 1982 Canada | 4.0 | 20 | 28 | 39 | 42 | 133 |
| 1981 U.S. | 4.8 | 22 | 26 | 33 | 41 | 127 |
| 1984 Japan | 3.4 | 11 | 7 | 59 | 23 | 103 |
| N.A. (b) | 4.0 | 22 | 27 | 35 | 41 | 130 |
| Ratio N.A./Japan | 1.18 | 2.00 | 3.85 | 0.60 | 1.80 | 1.25 |

(a) Standardized to age distribution of the 1988 Canadian population
(b) Average of Canadian and U.S. populations

Table 5.2
Average life expectancy by population and sex (a)

| Population | Males (yrs) | Females (yrs) | Females - males (yrs) |
|---|---|---|---|
| 1988 Canada | 74 | 80 | 6 |
| 1982 Canada | 72 | 79 | 7 |
| 1981 U.S. | 70 | 77 | 7 |
| 1984 Japan | 76 | 82 | 6 |
| N.A. (a) | 72 | 79 | 7 |
| Difference Japan - N.A. | 4 | 3 | -- |

(a) Values computed by Radrisk computer code

Table 5.3
Projected lifetime risk of fatal cancer (R) (induced cancers per 100,000 persons) by population, sex, cancer group, and transfer method resulting from a single whole-body exposure of 0.1 Gy (a, b)

Additive transfer method

Male

| Population | Leukemia | Respiratory | Breast | Digestive | Other | All Cancers |
|---|---|---|---|---|---|---|
| 1988 Canada | 80 | 155 | -- | 520 | 155 | 910 |
| 1982 Canada | 75 | 150 | -- | 490 | 150 | 865 |
| 1981 U.S. | 75 | 140 | -- | 455 | 135 | 805 |
| 1984 Japan | 80 (30, 205) | 170 (110, 265) | -- | 560 (355, 880) | 170 (110, 265) | 985 (710, 1575) |
| N.A. average (c) | 75 (30, 190) | 150 (95, 235) | -- | 490 (310, 770) | 145 (90, 225) | 860 (620, 1375) |
| Ratio Japan/N.A. | 1.07 | 1.13 | -- | 1.14 | 1.17 | 1.15 |

Female

| Population | Leukemia | Respiratory | Breast | Digestive | Other | All Cancers |
|---|---|---|---|---|---|---|
| 1988 Canada | 60 | 115 | 20 | 600 | 155 | 950 |
| 1982 Canada | 60 | 110 | 20 | 580 | 150 | 920 |
| 1981 U.S. | 60 | 105 | 20 | 545 | 140 | 870 |
| 1984 Japan | 60 (25, 140) | 120 (90, 165) | 20 (15, 30) | 620 (470, 860) | 160 (120, 22) | 980 (745, 1570) |
| N.A. average (c) | 60 (25, 140) | 110 (85, 150) | 20 (15, 30) | 575 (435, 800) | 150 (115, 210) | 915 (695, 1280) |
| Ratio Japan/N.A. | 1.00 | 1.09 | -- | 1.08 | 1.07 | 1.07 |

Multiplicative transfer method

Male

| Population | Leukemia | Respiratory | Breast | Digestive | Other | All Cancers |
|---|---|---|---|---|---|---|
| 1988 Canada | 90 | 285 | -- | 270 | 455 | 1100 |
| 1982 Canada | 85 | 260 | -- | 260 | 390 | 995 |
| 1981 U.S. | 120 | 235 | -- | 205 | 370 | 930 |
| 1984 Japan | 80 (35, 205) | 170 (110, 265) | -- | 560 (355, 880) | 170 (110, 265) | 980 (705, 1570) |
| N.A. average (c) | 100 (45, 255) | 260 (165, 410) | -- | 245 (155, 385) | 405 (260, 635) | 1010 (725, 1615) |
| Ratio N.A./Japan | 1.25 | 1.53 | -- | 0.44 | 2.38 | 1.03 |

Female

| Population | Leukemia | Respiratory | Breast | Digestive | Other | All Cancers |
|---|---|---|---|---|---|---|
| 1988 Canada | 65 | 220 | 70 | 390 | 310 | 1055 |
| 1982 Canada | 75 | 155 | 75 | 410 | 290 | 1005 |
| 1981 U.S. | 90 | 170 | 70 | 320 | 265 | 915 |
| 1984 Japan | 60 (25 140) | 120 (90, 165) | 20 (15, 30) | 620 (470, 860) | 160 (120, 220) | 980 (745, 1370) |
| N.A. average (c) | 75 (30 180) | 180 (140, 250) | 70 (55, 100) | 375 (285, 520) | 290 (220, 400) | 990 (715, 1315) |
| Ratio N.A./Japan | 1.25 | 1.50 | 3.50 | 0.60 | 1.81 | 1.01 |

(a) Weighted average for respective life-table age distributions of 1988 Canada, 1984 Japan, 1982 Canada, and 1981 U.S. population
(b) DDREF=1
(c) Average over 1988 Canada, 1982 Canada, and 1981 U.S.
( ) 90 percent confidence interval

Table 5.4
Projected excess lifetime risk (ELR) (excess cancers per 100,000 persons) by population sex, cancer group, and risk transfer method resulting from a single whole-body exposure of 0.1 Gy (a, b)

Additive transfer method

Male

| Population | Leukemia | Respiratory | Breast | Digestive | Other | All Cancers |
|---|---|---|---|---|---|---|
| 1988 Canada | 75 | 115 | -- | 460 | 80 | 730 |
| 1982 Canada | 70 | 110 | -- | 440 | 85 | 705 |
| 1981 U.S. | 70 | 100 | -- | 420 | 80 | 670 |
| 1984 Japan | 80 (30, 200) | 135 (85, 210) | -- | 450 (285, 705) | 95 (60, 150) | 760 (545, 1215) |
| N.A. average (c) | 70 (35, 185) | 110 (70, 175) | -- | 440 (280, 690) | 80 (50, 125) | 700 (500, 1100) |
| Ratio Japan/N.A. | 1.14 | 1.23 | -- | 1.02 | 1.19 | 1.09 |

Female

| Population | Leukemia | Respiratory | Breast | Digestive | Other | All Cancers |
|---|---|---|---|---|---|---|
| 1988 Canada | 55 | 100 | 5 | 550 | 110 | 820 |
| 1982 Canada | 55 | 100 | 5 | 530 | 110 | 800 |
| 1981 U.S. | 55 | 95 | 10 | 510 | 110 | 780 |
| 1984 Japan | 60 (35, 205) | 110 (85, 155) | 20 (15, 30) | 555 (420, 770) | 140 (85, 155) | 885 (670, 1230) |
| N.A. average (c) | 55 (20, 145) | 100 (75, 140) | 10 (7, 15) | 530 (400, 735) | 110 (85, 155) | 805 (615, 1130) |
| Ratio Japan/N.A. | 1.09 | 1.10 | 2.00 | 1.05 | 1.27 | 1.10 |

Multiplicative transfer method

Male

| Population | Leukemia | Respiratory | Breast | Digestive | Other | All Cancers |
|---|---|---|---|---|---|---|
| 1988 Canada | 85 | 225 | -- | 200 | 360 | 870 |
| 1982 Canada | 80 | 205 | -- | 200 | 315 | 800 |
| 1981 U.S. | 110 | 190 | -- | 160 | 305 | 765 |
| 1984 Japan | 80 (35, 205) | 130 (85, 205) | -- | 450 (285, 705) | 145 (90, 230) | 805 (580, 1290) |
| N.A. average (c) | 90 (40, 230) | 205 (130, 320) | -- | 185 (120, 295) | 325 (210, 520) | 805 (585, 1305) |
| Ratio N.A./Japan | 1.13 | 1.58 | -- | 0.41 | 2.24 | 1.00 |

Female

| Population | Leukemia | Respiratory | Breast | Digestive | Other | All Cancers |
|---|---|---|---|---|---|---|
| 1988 Canada | 60 | 200 | 50 | 335 | 260 | 905 |
| 1982 Canada | 70 | 145 | 55 | 355 | 245 | 870 |
| 1981 U.S. | 85 | 155 | 55 | 285 | 230 | 810 |
| 1984 Japan | 60 (25, 140) | 110 (85, 155) | 20 (15, 30) | 555 (420, 770) | 140 (90, 230) | 885 (670, 1235) |
| N.A. average (c) | 70 (25, 165) | 165 (125, 230) | 55 (40, 75) | 325 (245, 450) | 245 (210, 520) | 860 (665, 1150) |
| Ratio N.A./Japan | 1.17 | 1.50 | 2.75 | 0.59 | 1.75 | 0.97 |

(a) Weighted average for respective life-table age distributions of 1988 Canada, 1984 Japan, 1982 Canada, and 1981 U.S. population
(b) DDREF=1
(c) Average over 1988 Canada,1982 Canada, and 1981 U.S.
( ) 90 percent confidence interval
L: leukemia, R: respiratory cancer, B: breast cancer, D: digestive cancer, O: other cancers

Table 5.5
Variation of projected site-specific lifetime cancer risk projections with risk transfer method resulting from a single whole-body exposure of 0.1 Gy (a,b)

Excess lifetime risk (excess cancers per 100,000 persons)

| Cancer | Additive transfer method | | | | Multiplicative transfer method | | | |
|---|---|---|---|---|---|---|---|---|
| | Population Low | Population High | Population Average (c) | Ratio Hi/Lo | Population Low | Population High | Population Average (c) | Ratio Hi/Lo |
| Leukemia | 55 (20, 145) | 80 (30, 200) | 65 (30, 160) | 1.45 | 60 (25, 140) | 110 (50, 280) | 80 (35, 200) | 1.83 |
| Respiratory | 95 (70, 130) | 135 (85, 210) | 110 (75, 160) | 1.42 | 110 (85, 155) | 225 (145, 355) | 170 (120, 250) | 2.05 |
| Breast | 5 (3, 7) | 20 (15, 30) | 10 (7, 15) | 4.00 | 20 ( 15, 30) | 55 (50, 105) | 40 (30, 55) | 2.75 |
| Digestive | 420 (270, 660) | 555 (420, 770) | 490 (340, 725) | 1.32 | 160 (100, 250) | 555 (420, 770) | 315 (220, 470) | 3.47 |
| Other | 80 (50, 125) | 140 (85, 155) | 110 (75, 160) | 1.75 | 140 (90, 230) | 360 (230, 565) | 250 (170, 370) | 2.57 |
| All cancers | 725 (535, 1100) | 885 (670, 1230) | 760 (560, 1155) | 1.22 | 770 (540, 1240) | 905 (695, 1295) | 830 (615, 1260) | 1.18 |

Lifetime risk of fatal cancer (induced cancers per 100,000 persons)

| Cancer | Additive transfer method | | | | Multiplicative transfer method | | | |
|---|---|---|---|---|---|---|---|---|
| | Population Low | Population High | Population Average (c) | Ratio Hi/Lo | Population Low | Population High | Population Average (c) | Ratio Hi/Lo |
| Leukemia | 60 (25, 140) | 80 (30, 205) | 70 (30, 170) | 1.33 | 60 (25, 140) | 120 (55, 305) | 85 (35, 210) | 2.00 |
| Respiratory | 105 (80, 145) | 170 (110, 265) | 135 (95, 200) | 1.62 | 120 (90, 165) | 285 (180, 445) | 210 (150, 310) | 2.38 |
| Breast | 20 (15, 30) | 20 (15, 30) | 20 (15, 30) | 1.00 | 20 (15, 30) | 75 (55, 105) | 60 (40, 90) | 3.75 |
| Digestive | 455 (290, 715) | 620 (470, 860) | 545 (380, 805) | 1.36 | 205 (130, 320) | 620 (470, 860) | 380 (265, 560) | 3.02 |
| Other | 140 (105, 195) | 170 (110, 265) | 150 (105, 220) | 1.21 | 160 (120, 220) | 455 (290, 715) | 300 (210, 445) | 2.84 |
| Total | 840 (620, 1275) | 985 (730, 1500) | 910 (675, 1385) | 1.17 | 915 (705, 1310) | 1100 (790, 1760) | 995 (735, 1510) | 1.20 |

(a) DDREF=1
(b) Averaged over both sexes and all populations
( ) 90 percent confidence interval

Table 5.6
Effect of the choice of risk transfer method on the projected lifetime risk of fatal cancer (induced cancers per 100,000 persons) and relative contributions of cancer groups to the total risk for a North American population exposed to a single whole-body dose of 0.1 Gy (a, b)

Males

| Cancer | Additive | Multiplicative | Ratio Mult/Add | Relative contribution (c) Add | Mult |
|---|---|---|---|---|---|
| Leukemia | 75 (30, 190) | 100 (45, 255) | 1.33 | 0.10 | 0.10 |
| Respiratory | 150 (95, 235) | 260 (165, 410) | 1.73 | 0.15 | 0.25 |
| Breast | -- | -- | -- | -- | -- |
| Digestive | 490 (310, 770) | 245 (155, 385) | 0.50 | 0.55 | 0.25 |
| Other | 145 (90, 225) | 405 (260, 635) | 2.79 | 0.20 | 0.40 |
| All cancers | 860 (620, 1375) | 1010 (725, 1615) | 1.17 | 1.00 | 1.00 |

Females

| Cancer | Additive | Multiplicative | Ratio Mult/Add | Relative contribution (c) Add | Mult |
|---|---|---|---|---|---|
| Leukemia | 60 (25, 140) | 75 (30, 180) | 1.25 | 0.08 | 0.08 |
| Respiratory | 110 (85, 150) | 180 (135, 250) | 1.64 | 0.10 | 0.20 |
| Breast | 20 (15, 30) | 70 (55, 100) | 3.50 | 0.02 | 0.07 |
| Digestive | 575 (435, 800) | 375 (285, 520) | 0.65 | 0.65 | 0.35 |
| Other | 150 (2115, 210) | 290 (220, 400) | 1.93 | 0.15 | 0.30 |
| All cancers | 915 (695, 1280) | 990 (715, 1315) | 1.08 | 1.00 | 1.00 |

(a) Average over 1988 Canada, 1982 Canada, and 1981 U.S. populations
(b) DDREF=1
(c) Relative contribution to the total risk from all cancers
( ) 90 percent confidence interval

Table 5.7
Variation of the projected site-specific lifetime cancer risks by sex in North America resulting from a single whole-body exposure of 0.1 Gy

Excess lifetime risk (excess cancers per 100,000 persons)

| Cancer | Male | Female | Ratio Male/Female |
|---|---|---|---|
| Leukemia | 80 (35, 200) | 65 (25, 155) | 1.23 |
| Respiratory | 160 (100, 250) | 135 (100, 140) | 1.19 |
| Breast | -- | 35 (25, 50) | -- |
| Digestive | 315 (200, 495) | 430 (325, 600) | 0.73 |
| Other | 205 (130, 320) | 175 (130, 245) | 1.17 |
| All cancers | 755 (543, 1210) | 830 (640, 1185) | 0.91 |

Lifetime risk of fatal cancer (induced cancers per 100,000 persons)

| Cancer | Male | Female | Ratio Male/Female |
|---|---|---|---|
| Leukemia | 90 (50, 230) | 70 (30, 180) | 1.29 |
| Respiratory | 205 (130, 320) | 145 (110, 200) | 1.41 |
| Breast | -- | 45 (35, 60) | -- |
| Digestive | 370 (235, 580) | 475 (360, 660) | 0.78 |
| Other | 275 (175, 430) | 220 (165, 305) | 1.25 |
| All cancers | 935 (670, 1495) | 955 (740, 1390) | 0.98 |

(a) Averaged over North American populations and transfer methods
(b) DDREF=1
( ) 90 percent confidence interval

Table 5.8
Effect of the definition of cancer risk (ELR or R) on the projected site-specific lifetime cancer risks resulting from a single whole-body exposure of 0.1 Gy (a, b)

Males

| Cancer | ELR (c) | R (d) | Ratio R/ELR |
|---|---|---|---|
| Leukemia | 80 (35, 200) | 90 (50, 230) | 1.13 |
| Respiratory | 160 (100, 250) | 205 (130, 320) | 1.28 |
| Breast | -- | -- | -- |
| Digestive | 315 (200, 495) | 370 (235, 580) | 1.17 |
| Other | 205 (130, 320) | 275 (175, 430) | 1.34 |
| All cancers | 755 (543, 1210) | 935 (670, 1495) | 1.24 |

Females

| Cancer | ELR (c) | R (d) | Ratio R/ELR |
|---|---|---|---|
| Leukemia | 65 (25, 155) | 70 (30, 180) | 1.08 |
| Respiratory | 135 (100, 140) | 145 (110, 200) | 1.07 |
| Breast | 35 (25, 50) | 45 (35, 60) | 1.29 |
| Digestive | 430 (325, 600) | 475 (360, 660) | 1.10 |
| Other | 175 (130, 245) | 220 (165, 305) | 1.26 |
| All cancers | 830 (640, 1185) | 955 (755, 1365) | 1.15 |

(a) Averaged over all populations and transfer methods (Japan included only once)
(b) DDREF=1
(c) Excess lifetime risk (excess cancers per 100,000 persons)
(d) Lifetime risk of fatal cancer (induced cancers per 100,000 persons)
( ) 90 percent confidence interval

Table 5.9
Uncertainty due to choice of risk transfer method and sampling variation in the projected lifetime risk of fatal cancer (induced cancers per 100,000 persons) for North America resulting from a single whole-body exposure of 0.1 Gy (a, b)

| | Leukemia | Respiratory | Breast | Digestive | Other | All cancers |
|---|---|---|---|---|---|---|
| North America Average (b) | 80 | 175 | 45 | 420 | 250 | 945 |
| Uncertainty due to Transfer method | 65 - 90 | 130 - 220 | 20 - 70 | 310 - 530 | 145 - 350 | 890 - 1000 |
| Uncertainty due to Sampling variation (c) | 35 - 200 | 120 - 260 | 35 - 60 | 295 - 620 | 175 - 370 | 700 - 1435 |

(a) DDREF=1
(b) Averaged over both sexes, North Amerian populations, and transfer methods
(c) 90% confidence interval

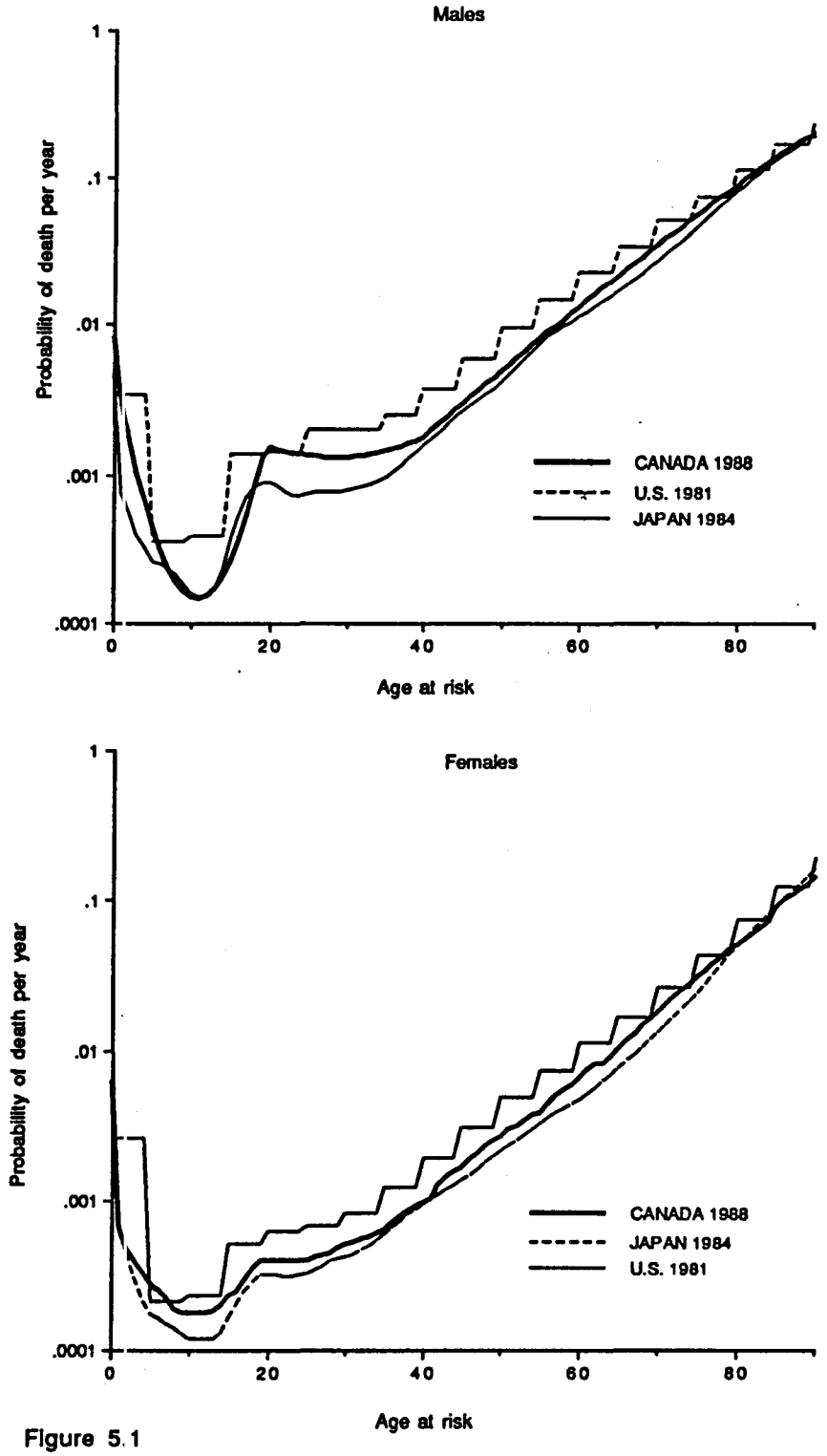


Figure 5.1
National mortality rates for all cause of death by age, sex, and population

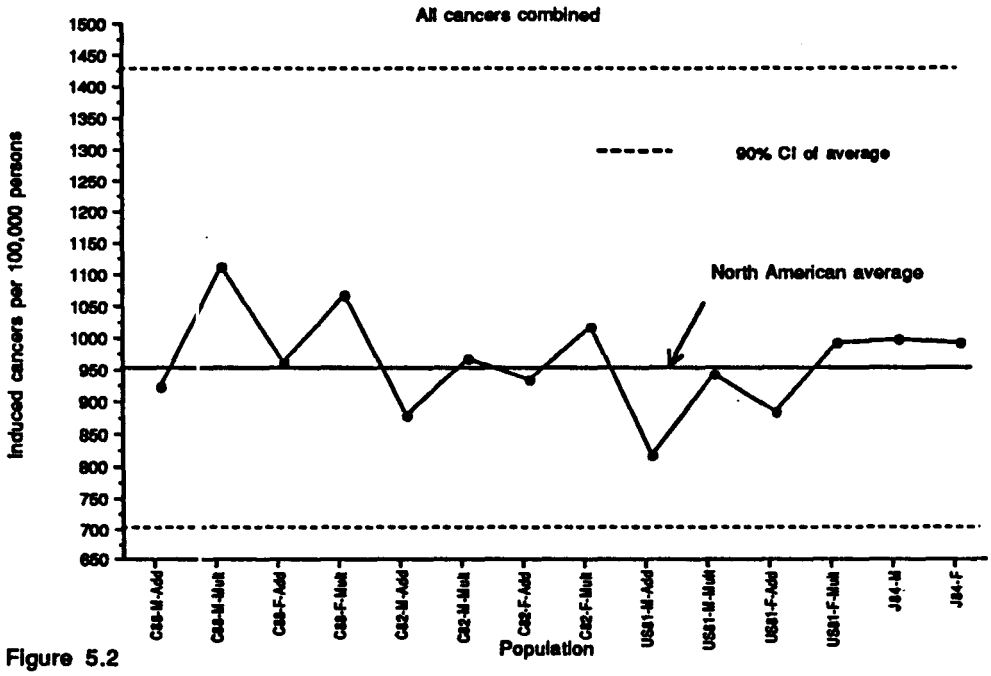


Figure 5.2
Projected lifetime risk of fatal cancer for all cancers combined resulting from a single whole-body exposure of 0.1 Gy (DDREF=1) by population, sex, and transfer method (C: Canada, J: Japan, M: male, F: female, Add: additive method, Mult: multiplicative method)

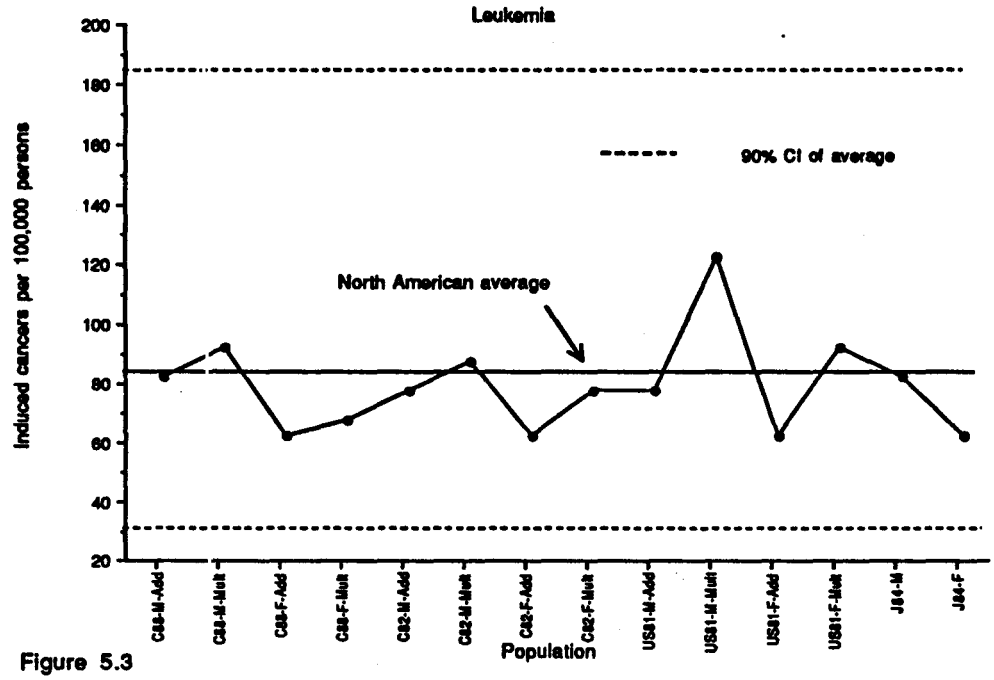


Figure 5.3
Projected lifetime risk of fatal cancer for leukemia resulting from a single whole-body exposure of 0.1 Gy (DDREF=1) by population, sex, and transfer method (C: Canada, J: Japan, M: male, F: female, Add: additive method, Mult: multiplicative method)

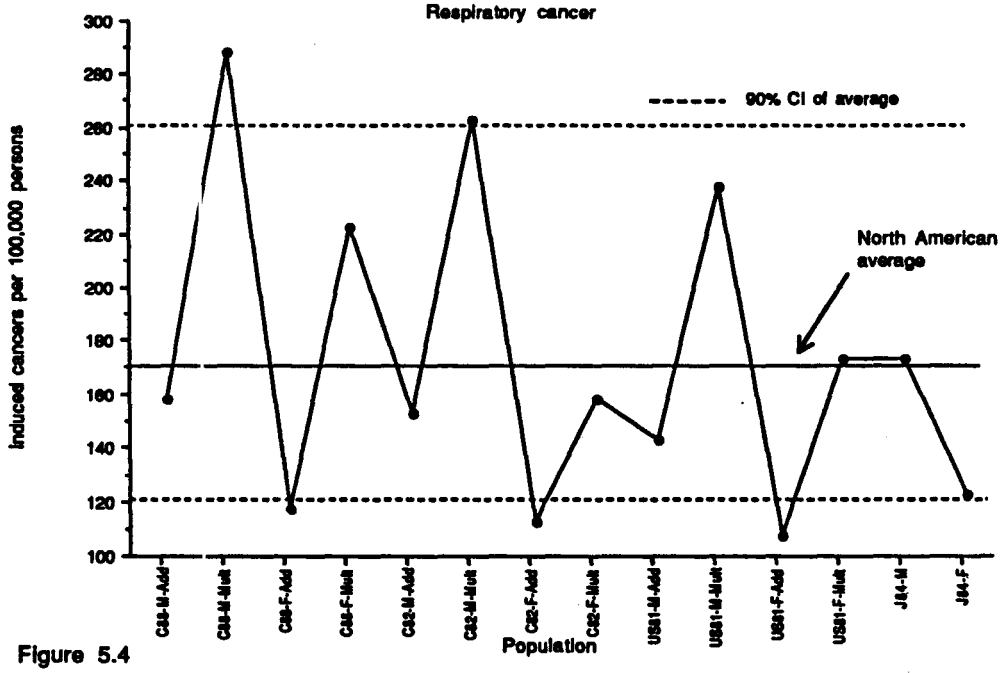


Figure 5.4
Projected lifetime risk of fatal cancer for respiratory cancer resulting from a single whole-body exposure of 0.1 Gy (DDREF=1) by population, sex, and transfer method (C: Canada, J: Japan, M: male, F: female, Add: additive method, Mult: multiplicative method)

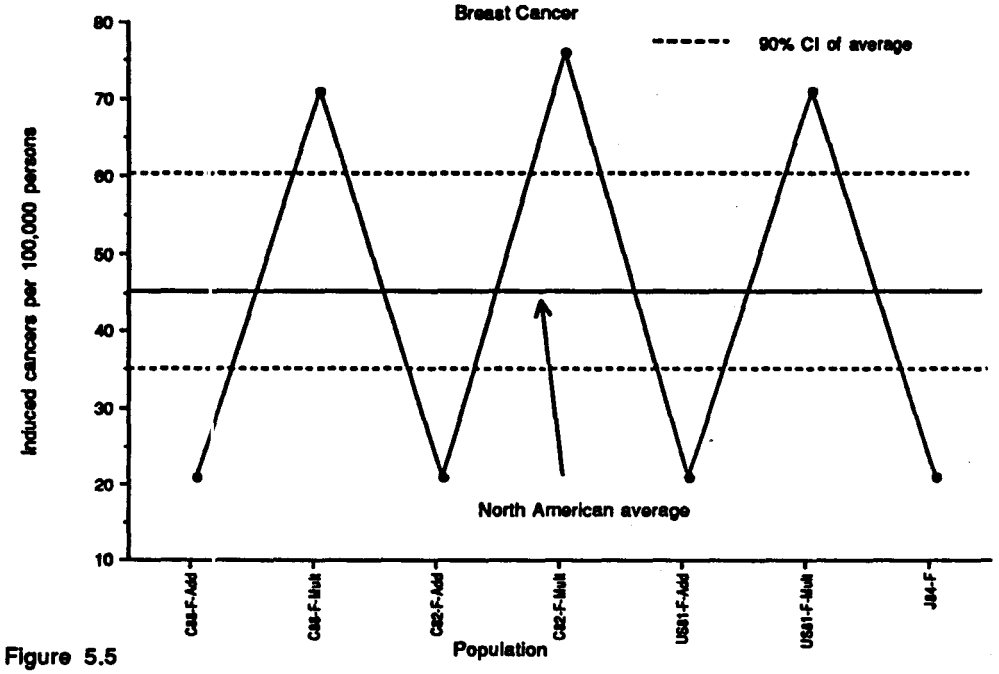


Figure 5.5
Projected lifetime risk of fatal cancer for breast cancer resulting from a single whole-body exposure of 0.1 Gy (DDREF=1) by population, sex, and transfer method (C: Canada, J: Japan, M: male, F: female, Add: additive method, Mult: multiplicative method)

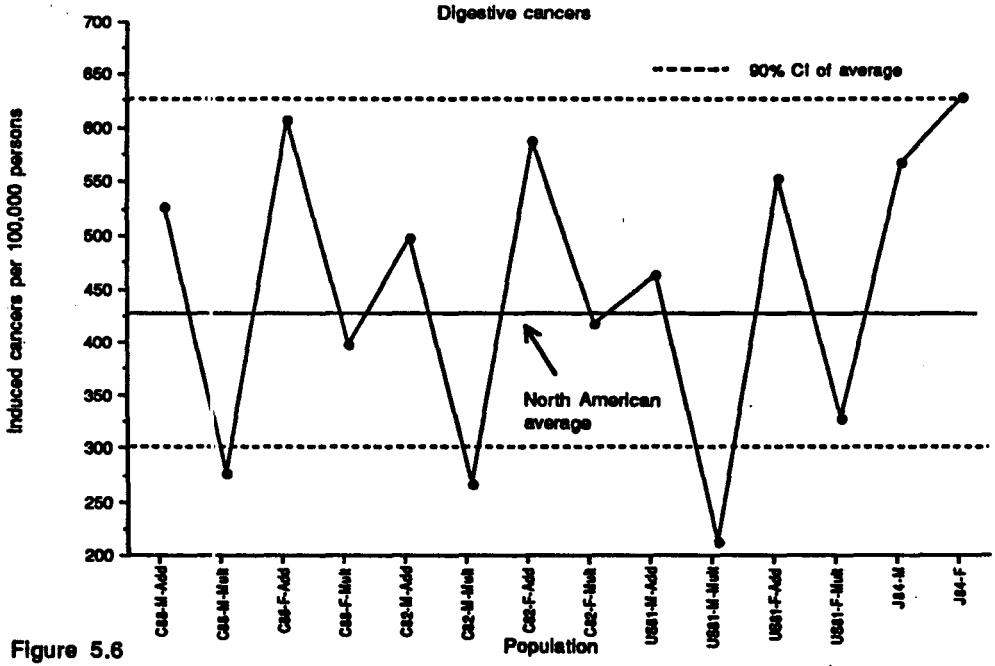


Figure 5.6
Projected lifetime risk of fatal cancer for digestive cancers resulting from a single whole-body exposure of 0.1 Gy (DDREF=1) by population, sex, and transfer method (C: Canada, J: Japan, M: male, F: female, Add: additive method, Mult: multiplicative method)

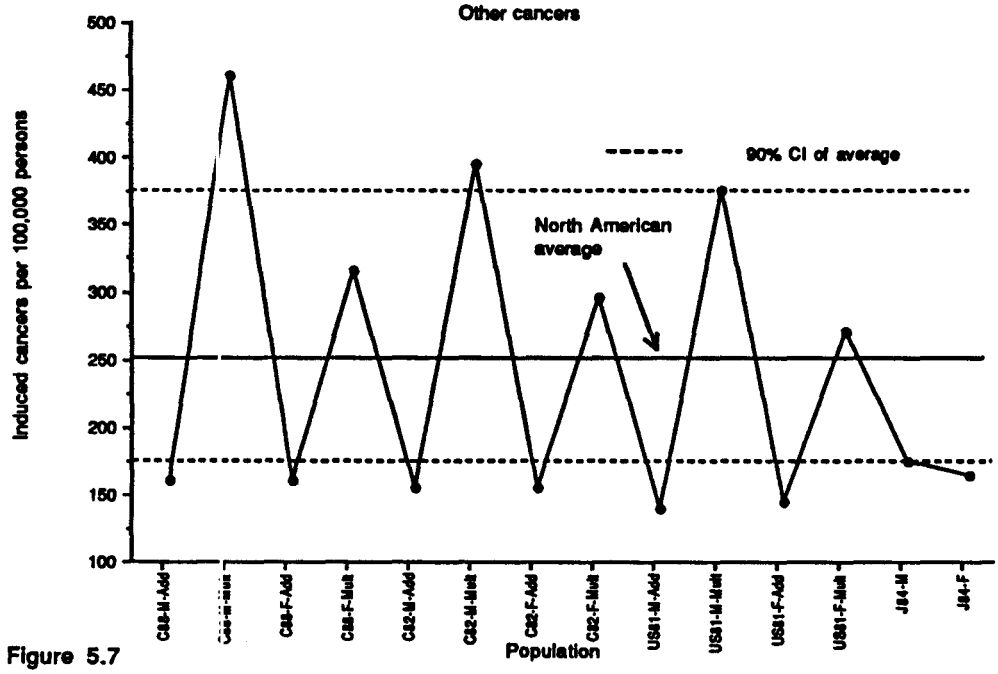


Figure 5.7
Projected lifetime risk of fatal cancer for other cancers combined resulting from a single whole-body exposure of 0.1 Gy (DDREF=1) by population, sex, and transfer method (C: Canada, J: Japan, M: male, F: female, Add: additive method, Mult: multiplicative method)

# 6.0 Effect on Cancer Risk Estimates of Age and Sex Distribution Differences Among Occupations in the Canadian Radiation Workforce

## 6.1 Introduction

Lifetime cancer risk projections for a population are traditionally summarized in terms of the risk to the average "member" of that population. As figure 6.1 shows, the projected cancer risks resulting from single whole-body exposure[50] can vary substantially with sex, age-at-exposure, and cancer site. How one chooses to average over sex and age-at-exposure may have a considerable affect on the projected risk for the average individual. The normal approach taken in reports, such as those by UNSCEAR, BEIR, and the ICRP, is to assume a stationary life-table age distribution with an equal proportion of males and females. However, as this chapter will show, it is not uncommon to have worker age distributions that are skewed with an disproportionate representation of male and female workers.

This chapter examines how differences in the age and sex distribution of workers among occupations in the 1988

[50] Risks projected using Radrisk computer code and averaged over 1982 and 1988 Canadian populations and the additive and multiplicative risk transfer methods

Canadian "radiation" workforce affects the projected lifetime fatal cancer risks per unit dose for the "average" Canadian worker. The affect on the relative contribution of different radiation-related cancers to the total projected risk is also examined.

"Radiation" workers are taken be those workers monitored for radiation exposure in 1988 whose dose records are available at the Canadian National Dose Registry[51]. Age and sex information were available for workers from over 50 job types from six main categories:

1. Administrative,
2. Medical,
3. Industry,
4. Power Stations,
5. Uranium workers, and
6. Miscellaneous

Variations in projected risks are expressed in terms of a "standardized irradiation ratio", or SIR, which expresses the ratio of the occupation- and -sex specific projected risk to that averaged over all occupations and both sex.

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[51] Excluding exposure to radon daughters

## 6.2 Materials and Methods

### 6.2.1 Age- and sex-specific lifetime risk projections

Projections of the age- and sex-specific lifetime risks of fatal cancer are performed using the BEIR V five preferred relative risk models and the life-table methods developed in chapter 4.0. Both the additive and multiplicative method is used to transfer risks to the 1988 and 1982 Canadian life-table populations with resulting projections averaged over population and transfer method. Because projections are for levels of doses and dose rates expected for occupational exposure, a DDREF of 2 is used[52].

Figure 6.1 plots the resulting lifetime fatal cancer risk per unit dose by sex, age-at-exposure, and cancer site or group. The risk to the "average" worker is calculated as the age-and sex- weighted averages of these estimated age- and sex-specific fatal cancer risks.

### 6.2.2 Worker age and sex distributions

Data on the age and number of Canadian male and female workers monitored for radiation exposure in 1988 was supplied by J.P. Ashmore of the Canadian National Dose Registry[53]. A total of 114,219 records were available providing information on worker age and sex in over 50 job types. These job types

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[52] Projections are made for a whole-body dose of 0.1 Gy. Since the dose response for radiation-induced leukemia has an inherent DDREF of 2 at this dose, no DDREF is applied.

[53] National Dose Registry, Bureau of Radiation and Medical Devices Department of National Health and Welfare.

are listed in table 6.1. The category "miscellaneous" refers to records in which no job type was given. A total of 3786 records did not contain age information and were excluded from the analysis. For records for which the sex was unknown but worker age given (2843 records), sex was designated based on the proportion of males and females for that age group and occupation.

Data provided by NDR specified worker age according to age groupings of under 18, age 18-20, 21-25, 26-30, 31-35, 36-40, 41-45, 46-50, 51-55, 56-60, 61-65, and ages over 65. In order to perform age-weighted averages, each age group was designated a single age equal to the midpoint of that age group. That is,

| | |
|---|---|
| 18 -> <18 | 43 -> 41-45 |
| 19 -> 18-20 | 48 -> 46-50 |
| 23 -> 21-25 | 53 -> 51-55 |
| 28 -> 26-30 | 58 -> 56-60 |
| 33 -> 31-35 | 63 -> 61-65 |
| 38 -> 36-40 | 68 -> >65 |

6.2.3 Age- and sex-weighted averages

The projected lifetime risk of fatal cancer per unit dose for cancer site (i) for the "average" worker of sex (s) in occupation (j) is computed as the age-weighted average of the age-specific projected cancer risks in figure 6.1. The average projected worker lifetime risk of cancer i for sex s

in occupation j will be given by

$$R_{i,s,j} = \sum_{T=18}^{T=68} W_{s,j}(T)\ R_{i,s}(T) \qquad (51)$$

where

$R_{c,s,j}$ is the projected lifetime risk of fatal cancer per unit dose for cancer i (leukemia, respiratory cancers, breast cancer, digestive cancers, other cancers, or all cancers combined) for the average worker of sex s in occupation j,

$R_{c,s}(T)$ is the age-specific projected lifetime risk of fatal cancer per unit dose for cancer i, sex s, and exposure age T, and

$W_{s,i}(T)$ fraction of the workers of sex s in occupation j who are of age T

Sex-weighted age averages are used to calculate occupation averages over both sexes.

6.2.4 Standardized Irradiation Ratio

As mentioned in the introduction, the variation of average worker risks with sex and occupation is described using a "standardized irradiation ratio", or SIR. The SIR represents the ratio of the sex- and occupation-specific risk to the weighted average over all occupations and sex. The SIR for breast cancer is computed using only the workforce average for females. However, when the SIR for all cancers as a group averaged over both sexes is computed, the contribution of

breast cancer to the total risk is based on the average between male and female workers, although there is zero risk for males. Since the objective is to compare the projected risk between "average" workers, it did not seem appropriate to take the breast cancer risk to be that of female workers only, particularly if females only accounted for a small proportion of workers in an occupation.

### 6.2.5 Relative cancer weighting factors

The relative contribution of leukemia, respiratory cancers, breast cancers, digestive cancers, and other cancers to the total projected lifetime cancer risk per unit dose are computed for the "average" male and female worker in each occupation. Weighting factors are calculated as the ratio of the cancer-specific risk to the total risk from all cancers combined. Unlike the SIR, the weighting factor for breast cancer is not based on the average over both sexes of the lifetime risk of breast cancer, but only that projected for female workers.

## 6.3 Variation of Worker Age with Sex and Occupation

Table 6.2 shows the number of Canadian workers in 1988 broken down by occupation and sex. In total, the workforce consists of roughly 110,000 workers with an equal proportion of males and females. But as figures 6.2 to 6.5 illustrate, the age distribution among occupations and between gender

differs substantially. The majority of workers (85% of female workers and 50% of male workers) are employed in either medical or miscellaneous occupations (see figure 6.5). Occupations dominated by female workers are administrative, medical, and miscellaneous type jobs while male workers dominate jobs in industry, reactors/power plants, and uranium mining (see figure 6.4).

The most prominent feature in the 1988 workforce is the age difference between male and female workers. Table 6.3 shows the breakdown of the average age of workers by occupation and sex as well as the proportion of workers over and under the age of 35. Female workers make up a relatively young workforce with over 65% of workers under the age of 35. In contrast, male workers are somewhat older with about 55% of workers above the age of 35. The average working age for females ranges from 26 to 46 (mean of 33) and for males, from 33 to 48 (mean of 38).

## 6.4 Results

Tables 6.4 gives the projected lifetime fatal cancer risks per unit dose and relative weighting factors averaged over all occupations and both sex for radiation-induced leukemia, respiratory cancers, female breast cancer, digestive cancers, and other cancers. Tables 6.5 and 6.6 respectively lists the standardized irradiation ratios and weighting

factors by cancer site, occupation, and worker sex. Figures 6.6 to 6.12 shows the distribution of estimated risks for "average" workers among occupations by cancer site. Distributions are normal-like with the female worker distributions being more skewed than males.

The workforce average fatal cancer risk per unit dose for all cancers as a group is about 20% higher for female workers compared to male workers. The higher total risk for female workers is caused almost solely by a greater risk of radiation-induced digestive cancers which represents over half the female total projected cancer risk. In contrast, radiation-induced leukemia and respiratory cancers are greater and of more importance among male workers. For other cancers, projected risks do not differ greatly between the sexes. Site specific results are described in further detail below.

6.4.1 All cancers combined

For all cancers combined, the workforce average fatal cancer risk per unit dose for male and female workers is $425 \times 10^{-4}$ per Sv and $520 \times 10^{-4}$ per Sv, respectively. The age-weighted average over both sexes is $475 \times 10^{-4}$ per Sv. Figure 6.6 shows the distribution of sex-specific worker averages among occupations. About 90 percent of male and 70 percent of female occupations are within 20% of the workforce average over both sex. Occupations in the high and low tails of distributions include:

Male Workers

| "High" per unit dose | "Low" risk per unit dose |
|---|---|
| •therapeutic radiological technicians | •safety officers |
| •well loggers | •therapeutic radiologists |
| •nuclear medicine isotope technicians | •health physicists |
| •industrial radiographers | •diagnostic radiologists |
| •reactor general maintenance workers | •instrument technicians |
| •reactor operation workers | •uranium mill workers |

Female Workers

| "High" per unit dose | "Low" risk per unit dose |
|---|---|
| •reactor control technicians | •uranium mine nurses |
| •gynaecologists | •reactor fuel processors |
| •reactor general maintenance workers | •health physicists |
| •nuclear medicine isotope technicians | •uranium mill workers |
| •reactor chemical and radiation control technicians | •therapeutic radiologists |
| | •diagnostic radiologists |

The range of standardized irradiation ratios among male high "risk" occupations is 0.90 to 1.11 and 1.14 to 1.56 for female workers. Low "risk" SIR ranges for males are 0.78 to 1.14 and for females, 0.66 to 0.86.

6.4.2 Leukemia

The workforce average fatal cancer risk per unit dose for radiation-induced leukemia for male workers is $60 \times 10^{-4}$ per Sv and $40 \times 10^{-4}$ per Sv for female workers. The age-weighted average over both sexes is $50 \times 10^{-4}$ per Sv. The relative weighting factor is 0.15 and 0.10 for the respective sexes and the average is 0.12. The distribution of sex-specific worker averages among occupations is shown is figures 6.6a and 6.6b. Over 95% of male workers are above the overall workforce average while over 90% of female workers are below. However, the majority of both male and female workers are within 25% of the average for the whole workforce.

Male and female occupations in the high and low tail of the distribution of worker averages are the reverse order of those occupations listed for all cancers as a group. The ranges of projected risks among high "risk" occupations for male workers are: SIR= 1.45-1.98, WF[54]= 0.21-0.27, and for female workers: SIR= 0.90-1.31, WF= 0.12-0.19. Low "risk" ranges for males are: SIR= 0.86-1.08, WF= 0.09-0.14, and for females: SIR= 0.55-0.69, WF= 0.04-0.07.

6.4.3 Respiratory tract

The workforce average fatal cancer risk per unit dose for radiation-related respiratory cancers for male workers is $125 \times 10^{-4}$ per Sv and $75 \times 10^{-4}$ per Sv for female

[54] Weighting factor

workers. The average over both sexes is $100x10^{-4}$ per Sv. The relative weighting factor is 0.30 and 0.15 for the respective sexes, with an average of 0.20. The distribution of sex-specific worker averages is nearly identical to that of leukemia (see figures 6.7a and 6.7b). As for leukemia, over 95% of male workers are above and over 90% of female workers are below the overall workforce average. Again, the majority of both sexes are within 25% of the overall average.

Inherent high and low "risk" occupations for radiation-induced respiratory cancers are the same as for leukemia. The range of projected risks among high "risk" occupations for male workers is: SIR= 1.45-1.80, WF= 0.40-0.50, and for female workers: SIR= 0.89-1.11, WF= 0.23-0.40. Low "risk" range for males is: SIR= 0.92-1.11, WF= 0.19-0.26, and for females: SIR= 0.57-0.70, WF= 0.08-0.13.

6.4.4 Female breast

The distribution of the lifetime risk of fatal breast cancer per unit dose among female occupations is highly skewed about the workforce average of $20x10^{-4}$ per Sv. Projected risks per unit dose range from 11 to 25 x $10^{-4}$ per Sv with over 60% of occupations equal or above $20x10^{-4}$ per Sv.

Figure 6.8b plots the distribution of breast cancer weighting factors among occupations when the total cancer risk is calculated using only female worker projections and also when contributions to the total cancer risk from other cancers

are averaged over both sexes. When contribution from other cancer sites are taken to be only for females, the breast cancer weighting factor ranges only from 0.03 to 0.05 with 85% of occupations having the value of 0.04. Occupations in the high and low tail of the distribution of female worker averages are the same as those listed for all cancers combined. When contributions to the total cancer risk are averaged both sexes, the breast cancer weighting ranges from 0.02 to 0.06 with the distribution more skewed towards higher values. While the difference is small in view of the small range of weighting to begin with, it does demonstrate that weighting breast cancer using the sex-average of cancers other than breast cancer will not necessarily cause the weighting factor for the breast to be underestimated.

6.4.5 Digestive system

The projected lifetime risk of fatality per unit dose of radiation-related digestive cancers for female workers is substantially higher than male workers by factors ranging from 2 to 10. The age-weighted average lifetime risk for the female workforce is $270 \times 10^{-4}$ per Sv and for the male workforce, $130 \times 10^{-4}$ per Sv. The workforce average over both sexes is $195 \times 10^{-4}$ per Sv. The average weighting factor is 0.30 and 0.50 for males and females, respectively, with an average of 0.40. Figures 6.9a and 6.9b illustrates the difference in the distribution of risks between sexes. Over 65% of female

occupations have a higher projected radiation-induced risk than the highest projected value for males ($590 \times 10^{-4}$ per Sv). Over 95% of male averages are below the overall workforce average while 75% percent of female averages are above.

The male and female occupations with high and low risks per unit dose are the same as for total risk from all cancers as a group. The ranges of projected risks among inherently high "risk" operations for male workers are: SIR= 0.71-1.14, WF= 0.33-0.43, and for female workers: SIR= 1.46-2.29, WF= 0.54-0.63. Low "risk" ranges for males are: SIR= 0.23-0.42, WF= 0.12-0.22, and for females: SIR= 0.37-0.65, WF= 0.24-0.0.33.

6.4.6 Other remaining sites

The workforce average fatal cancer risk per unit dose at other remaining cancers for male workers is $110 \times 10^{-4}$ per Sv and $115 \times 10^{-4}$ per Sv for female workers. The age-weighted average over both sexes is $110 \times 10^{-4}$ per Sv. The relative weighting factor is 0.25 and 0.20 for the respective sexes, with an average of 0.25. Worker averages for both male and female occupations are both closely distributed about the overall workforce average (see figures 6.11a and 6.11b). Over 70 percent of worker averages for both male and female occupations are within 20% of the overall average.

Occupations are at the high and low end tails of the distribution of worker averages are again the same as for

total risk from all cancers combined. The ranges of worker averages among males are: SIR= 1.09-1.32, WF= 0.27-0.33, and for female workers: SIR= 1.09-1.37, WF= 0.23-0.26. Low "risk" ranges for males are: SIR= 0.35-0.73, WF= 0.11-0.21, and for females: SIR= 0.34-0.77, WF= 0.12-0.22.

## 6.5 Discussion and Conclusions

Figures 6.6 to 6.12 demonstrate that the differences in the age- and sex-distribution among occupations in the Canadian radiation workforce can cause substantial variation in both estimated lifetime fatal cancer risks per unit dose and the relative cancer weighting factors. Table 6.7 summarizes the standardized irradiation ratios and relative cancer weighting factors for the 6 main occupations by sex and cancer site. There are distinct sex differences in the projected radiation-induced cancer risks projected for the "average" Canadian worker. The lifetime fatal cancer risk per unit dose for all cancers combined is, on average, 20% higher for female than male workers. Radiation-induced digestive cancers dominate the total risk for female workers (average WF= 0.52) and the estimate workforce average is 2 times higher than male workers (270 vs 130 x $10^{-4}$ per Sv). In contrast, the lifetime risks for leukemia and respiratory radiation-induced cancers are more dominant for male workers (average WFs= 0.15 and 0.30, respectively). The risk per Sv for male workers is an average 1.5 times higher for leukemia (60 vs 40 x $10^{-4}$ per

Sv) and 1.7 times higher for respiratory (125 vs 75 x $10^{-4}$) compared to female workers. For "other" remaining cancers, average lifetime risks per unit dose and weighting factors are similar between both sexes (lifetime risk= 110 and 115 x $10^{-4}$ per Sv and WT's= 0.25 and 0.22 for males and females, respectively).

Table 6.8 compares the 1988 Canadian workforce averages to those obtained using the traditional approach of averaging risks over the age distribution of a stationary life-table working population (ages 18-65). Sex differences for the life-table population are similar to that for the 1988 workforce but not as great. For instance, the lifetime risk for digestive cancers is only 1.3 times higher for females than males in the life-table population compared to being 2 times higher in the 1988 Canadian workforce. For "other" remaining cancers, the lifetime risk per unit dose is 1.25 times higher for males than females in the life-table population, but in the Canadian workforce males have a slightly lower average risk than females.

Differences between worker averages computed for the 1988 Canadian workforce and the life-table working population appears to be caused by differences in the age make up between males and female workers. The life-table has a fairly uniform age distribution of male and female workers while in the Canadian workforce the majority of female workers are under

Canadian workforce the majority of female workers are under the age of 35 and the majority of male workers above age 35. As figure 6.1 shows, age-specific lifetime risk projections for radiation-induced cancers of the female breast, digestive system, and "other" remaining sties are significantly higher for exposure ages under 35 while radiation-induced risks are greater for leukemia and respiratory cancers at exposure ages above 35.

It is difficult to make any firm deductions as to whether sex or age differences should be taken into account in the estimated cancer risks or cancer weighting factors. While this chapter shows that differences in age and sex of workers among different occupations in the Canadian workforce does cause variations in the estimates for the average worker, variations are not significant. For all cancer as a group, 80 percent of worker averages (male or female) are within 20% of the overall workforce average. At individual cancer sites, the majority of worker averages (male or female) are within 25 percent of the overall workforce averages. These variations are small compared to those caused by statistical errors in risk models, uncertainty in the choice of risk transfer method, and the uncertainty in how to project future excess cancer risks beyond 40 years following exposure. For the purpose of radiation protection, it would therefore seem reasonable to use only one set of risk estimates which are the

Table 6.1
Occupation and job categories in the Canadian "radiation" workforce

ADMINISTRATIVE
Administrator
Office Staff
Safety Officer

MEDICAL
Chiropractor
Dental Hygienist
Dentist
Gynaecologist
Isotope Tech (NM)
Lab Tech
Medical Physicist
Nurse
Physician
Radiological Tech (D)
Radiological Tech (T)
Radiologist (D)
Radiologist (T)
Veterinarian
Ward Aid/orderly

INDUSTRY
Dial Painter
Fuel Processor
Industrial Radiographer
Instrument Tech
Instructor
Lab Tech
Scientist Engineer (Field)
Scientist/Engineer (Lab)
Well Logger

REACTOR WORKERS
Administration
Chem&Rad Control
Control Techs
Electrical Maintenance
Fuel Handling
General Maintenance
Health Physics
Mechanical Maintenace
Operations
Construction
Scientific/Professional
Training
Visitor

URANIUM MINERS
Underground Miners
Underground Maintenance
Underground Personnel
Support Workers
Surface Miners
Surface Maintenance
Surface Personnel
Surface Support Workers
Mill Workers
Mill Maintenance
Office Staff
Nurses
Visitors

MISCELLANEOUS

Table 6.2
Breakdown of the number of Canadian "radiation" workers in 1988 by occupation, job, and sex

| Occupation & Job Group | Number of workers Male | Female | Total |
|---|---|---|---|
| 1. ADMINISTRATIVE | | | |
| Administrator | 14 | 23 | 37 |
| Office Staff | 182 | 2590 | 2772 |
| Safety Officer | 14 | 5 | 19 |
| TOTAL | 210 | 2618 | 2828 |
| 2. MEDICAL | | | |
| Chiropractor | 691 | 71 | 762 |
| Dental Hygienist | 126 | 5116 | 5242 |
| Dentist | 4486 | 741 | 5227 |
| Gynaecologist | 19 | 9 | 28 |
| Isotope Tech (NM) | 413 | 796 | 1209 |
| Lab Tech | 908 | 2042 | 2950 |
| Medical Physicist | 157 | 42 | 199 |
| Nurse | 153 | 3566 | 3719 |
| Physician | 1581 | 251 | 1832 |
| Radiological Tech (D) | 1607 | 7927 | 9534 |
| Radiological Tech (T) | 88 | 502 | 590 |
| Radiologist (D) | 1271 | 324 | 1595 |
| Radiologist (T) | 103 | 22 | 125 |
| Veterinarian | 1188 | 686 | 1874 |
| Ward Aid/orderly | 491 | 1073 | 1564 |
| TOTAL | 13282 | 23198 | 36480 |
| 3. INDUSTRY | | | |
| Dial Painter | 9 | 0 | 9 |
| Fuel Processor | 54 | 3 | 57 |
| Industrial Radiogragher | 1285 | 157 | 1442 |
| Instrument Tech | 109 | 17 | 126 |
| Instructor | 1018 | 43 | 1061 |
| Lab Tech | 1918 | 1356 | 3274 |
| Scientist Engineer (Field) | 548 | 28 | 576 |
| Scientist/Engineer (Lab) | 2149 | 613 | 2762 |
| Well Logger | 1126 | 6 | 1132 |
| TOTAL | 8216 | 2223 | 10439 |

| Occupation & Job Group | Number of workers Male | Female | Total |
|---|---|---|---|
| 4. REACTOR WORKERS | | | |
| Administration | 1606 | 1089 | 2695 |
| Chem&Rad Control | 382 | 41 | 423 |
| Control Techs | 126 | 2 | 128 |
| Electrical Maintenance | 886 | 13 | 899 |
| Fuel Handling | 68 | 0 | 68 |
| General Maintenance | 2157 | 281 | 2438 |
| Health Physics | 126 | 16 | 142 |
| Mechanical Maintenace | 1182 | 10 | 1192 |
| Operations | 1450 | 28 | 1478 |
| Construction | 1949 | 45 | 1994 |
| Scientific/Professional | 1968 | 150 | 2118 |
| Training | 55 | 8 | 63 |
| Visitor | 108 | 13 | 121 |
| TOTAL | 12063 | 1696 | 13759 |
| 5. URANIUM MINERS | | | |
| underground miners | 130 | 0 | 130 |
| underground maintenance | 6 | 3 | 9 |
| underground personnel | 2131 | 143 | 2274 |
| support workers | 437 | 6 | 443 |
| surface miners | 155 | 3 | 158 |
| surface maintenance | 600 | 6 | 606 |
| surface personnel | 114 | 6 | 120 |
| surface support workers | 176 | 21 | 197 |
| mill workers | 1450 | 26 | 1476 |
| mill maintenace | 207 | 1 | 208 |
| office staff | 144 | 22 | 166 |
| nurses | 1 | 2 | 3 |
| visitors | 26 | 4 | 30 |
| TOTAL | 5407 | 253 | 5660 |
| 6. MISCELLANEOUS | 15871 | 26396 | 42267 |
| TOTAL WORKFORCE | 55049 | 56384 | 111433 |

Table 6.3
Breakdown of the average age of Canadian "radiation" workers in 1988 by occupation, job, and sex

| Occupation & Job Group | Male | | | Female | | | Both sexes | | |
|---|---|---|---|---|---|---|---|---|---|
| | Average Working Age | Proportion (a) Age<35 | Age>35 | Average Working Age | Proportion (a) Age<35 | Age>35 | Average Working Age | Proportion (a) Age<35 | Age>35 |
| 1. ADMINISTRATIVE | | | | | | | | | |
| Administrator | 47 | 0.14 | 0.86 | 33 | 0.74 | 0.26 | 38 | 0.51 | 0.49 |
| Office Staff | 41 | 0.33 | 0.67 | 34 | 0.61 | 0.38 | 34 | 0.59 | 0.40 |
| Safety Officer | 56 | 0.00 | 1.00 | 40 | 0.20 | 0.80 | 52 | 0.05 | 0.95 |
| TOTAL | 43 | 0.35 | 0.65 | 34 | 0.63 | 0.37 | 35 | 0.60 | 0.39 |
| 2. MEDICAL | | | | | | | | | |
| Chiropractor | 43 | 0.29 | 0.71 | 33 | 0.71 | 0.29 | 42 | 0.33 | 0.67 |
| Dental Hygienist | 37 | 0.50 | 0.49 | 31 | 0.79 | 0.22 | 31 | 0.78 | 0.22 |
| Dentist | 42 | 0.30 | 0.71 | 34 | 0.66 | 0.33 | 41 | 0.35 | 0.65 |
| Gynaecologist | 44 | 0.21 | 0.79 | 26 | 1.00 | 0.00 | 39 | 0.46 | 0.54 |
| Isotope Tech (NM) | 33 | 0.66 | 0.34 | 30 | 0.80 | 0.20 | 31 | 0.75 | 0.25 |
| Lab Tech | 35 | 0.55 | 0.45 | 34 | 0.60 | 0.40 | 35 | 0.59 | 0.41 |
| Medical Physicist | 41 | 0.32 | 0.68 | 38 | 0.37 | 0.63 | 41 | 0.33 | 0.67 |
| Nurse | 39 | 0.39 | 0.60 | 38 | 0.44 | 0.56 | 38 | 0.44 | 0.56 |
| Physician | 44 | 0.23 | 0.78 | 38 | 0.48 | 0.53 | 44 | 0.26 | 0.73 |
| Radiological Tech (D) | 33 | 0.45 | 0.55 | 34 | 0.58 | 0.43 | 35 | 0.56 | 0.45 |
| Radiological Tech (T) | 34 | 0.64 | 0.36 | 34 | 0.61 | 0.39 | 34 | 0.61 | 0.39 |
| Radiologist (D) | 47 | 0.23 | 0.77 | 37 | 0.50 | 0.51 | 45 | 0.28 | 0.72 |
| Radiologist (T) | 43 | 0.14 | 0.85 | 39 | 0.41 | 0.59 | 46 | 0.19 | 0.81 |
| Veterinarian | 40 | 0.34 | 0.66 | 31 | 0.78 | 0.22 | 37 | 0.50 | 0.50 |
| Ward Aid/orderly | 39 | 0.46 | 0.55 | 33 | 0.65 | 0.35 | 36 | 0.59 | 0.41 |
| TOTAL | 41 | 0.36 | 0.65 | 33 | 0.65 | 0.35 | 36 | 0.55 | 0.45 |
| 3. INDUSTRY | | | | | | | | | |
| Dial Painter | 38 | 0.67 | 0.33 | -- | 0.00 | 0.00 | 36 | 0.67 | 0.33 |
| Fuel Processor | 44 | 0.33 | 0.66 | 46 | 0.33 | 0.66 | 44 | 0.33 | 0.66 |
| Industrial Radiographer | 35 | 0.59 | 0.41 | 34 | 0.58 | 0.43 | 35 | 0.59 | 0.41 |
| Instrument Tech | 44 | 0.16 | 0.84 | 39 | 0.41 | 0.59 | 44 | 0.20 | 0.80 |
| Instructor | 38 | 0.44 | 0.56 | 33 | 0.69 | 0.31 | 38 | 0.45 | 0.55 |
| Lab Tech | 37 | 0.52 | 0.48 | 33 | 0.69 | 0.31 | 35 | 0.59 | 0.41 |
| Scientist Engineer (Field) | 38 | 0.56 | 0.44 | 32 | 0.64 | 0.36 | 36 | 0.56 | 0.44 |
| Scientist/Engineer (Lab) | 39 | 0.44 | 0.56 | 32 | 0.75 | 0.25 | 37 | 0.51 | 0.49 |
| Well Logger | 30 | 0.84 | 0.16 | 33 | 0.67 | 0.33 | 30 | 0.84 | 0.17 |
| TOTAL | 37 | 0.53 | 0.47 | 33 | 0.66 | 0.34 | 36 | 0.56 | 0.44 |
| 4. REACTOR WORKERS | | | | | | | | | |
| Administration | 39 | 0.40 | 0.61 | 35 | 0.60 | 0.40 | 37 | 0.48 | 0.52 |
| Chem&Rad Control | 36 | 0.38 | 0.62 | 30 | 0.80 | 0.20 | 36 | 0.42 | 0.57 |
| Control Techs | 41 | 0.28 | 0.72 | 26 | 1.00 | 0.00 | 41 | 0.29 | 0.71 |
| Electrical Maintenance | 37 | 0.38 | 0.62 | 29 | 0.80 | 0.20 | 37 | 0.42 | 0.57 |
| Fuel Handling | 36 | 0.38 | 0.61 | -- | 0.00 | 0.00 | 39 | 0.38 | 0.61 |
| General Maintenance | 38 | 0.45 | 0.55 | 29 | 0.76 | 0.24 | 37 | 0.49 | 0.51 |
| Health Physics | 40 | 0.30 | 0.70 | 40 | 0.38 | 0.63 | 40 | 0.31 | 0.69 |
| Mechanical Maintenace | 38 | 0.41 | 0.59 | 32 | 0.90 | 0.10 | 38 | 0.42 | 0.58 |
| Operations | 35 | 0.62 | 0.39 | 31 | 0.82 | 0.18 | 35 | 0.62 | 0.38 |
| Construction | 42 | 0.31 | 0.69 | 34 | 0.60 | 0.40 | 42 | 0.31 | 0.69 |
| Scientific/Professional | 41 | 0.32 | 0.68 | 36 | 0.54 | 0.46 | 40 | 0.33 | 0.67 |
| Training | 42 | 0.20 | 0.80 | 31 | 0.88 | 0.13 | 41 | 0.29 | 0.71 |
| Visitor | 37 | 0.55 | 0.45 | 26 | 0.85 | 0.16 | 36 | 0.58 | 0.42 |
| TOTAL | 39 | 0.43 | 0.57 | 33 | 0.65 | 0.35 | 38 | 0.45 | 0.55 |
| 5. URANIUM MINERS | | | | | | | | | |
| underground miners | 36 | 0.56 | 0.44 | -- | 0.00 | 1.00 | 36 | 0.56 | 0.44 |
| underground maintenance | 33 | 0.83 | 0.17 | 26 | 1.00 | 0.00 | 31 | 0.89 | 0.11 |
| underground personnel | 40 | 0.41 | 0.59 | 34 | 0.64 | 0.36 | 39 | 0.43 | 0.57 |
| support workers | 37 | 0.56 | 0.44 | 31 | 0.67 | 0.33 | 36 | 0.56 | 0.44 |
| surface miners | 39 | 0.45 | 0.55 | 26 | 1.00 | 0.00 | 38 | 0.46 | 0.54 |
| surface maintenance | 37 | 0.51 | 0.49 | 31 | 0.83 | 0.17 | 37 | 0.51 | 0.49 |
| surface personnel | 44 | 0.21 | 0.79 | 34 | 0.67 | 0.33 | 44 | 0.23 | 0.77 |
| surface support workers | 38 | 0.38 | 0.62 | 32 | 1.00 | 0.00 | 38 | 0.38 | 0.62 |
| mill workers | 40 | 0.38 | 0.62 | 30 | 0.21 | 0.79 | 39 | 0.22 | 0.78 |
| mill maintenace | 40 | 0.38 | 0.62 | 26 | 1.00 | 0.00 | 40 | 0.38 | 0.62 |
| office staff | 42 | 0.30 | 0.70 | 31 | 0.82 | 0.18 | 41 | 0.37 | 0.63 |
| nurses | 38 | 0.00 | 1.00 | 46 | 0.00 | 1.00 | 43 | 0.00 | 1.00 |
| visitors | 41 | 0.35 | 0.65 | 52 | 0.00 | 1.00 | 43 | 0.30 | 0.70 |
| TOTAL | 38 | 0.47 | 0.53 | 33 | 0.71 | 0.28 | 38 | 0.48 | 0.52 |
| 6. MISCELLANEOUS | 36 | 0.56 | 0.44 | 32 | 0.72 | 0.28 | 33 | 0.65 | 0.34 |
| TOTAL | 38 | 0.46 | 0.54 | 33 | 0.67 | 0.33 | 36 | 0.56 | 0.44 |

(a) Proportion under and over the age of 35

Table 6.4
Projected lifetime risks of fatal cancer per unit dose and cancer weighting factors for the "average" male and female worker in the 1988 Canadian "radiation" workforce

Lifetime risk of fatal cancer per unit dose (10E-04 per Sv) (a)

| Radiation-related Cancer | Average male worker | Average female worker | Average over both sexes |
|---|---|---|---|
| Leukemia | 60 (44 - 101) | 40 (28 - 72) | 50 |
| Respiratory | 125 (94 - 180) | 75 (58 - 125) | 100 |
| Breast | -- | 20 (11 - 25) | 20 |
| Digestive | 130 (41 - 225) | 270 (76 - 445) | 195 |
| Other | 110 (39 - 136) | 115 (38 - 144) | 110 |
| All cancers | 425 (336 - 502) | 520 (312 - 711) | 475 |

Cancer weighting factor (a)

| Radiation-related Cancer | Average male worker | Average female worker | Average over both sexes |
|---|---|---|---|
| Leukemia | 0.15 (0.09 -0.27) | 0.08 (0.04 - 0.23) | 0.10 |
| Respiratory | 0.30 (0.19 - 0.50) | 0.15 (0.08 - 0.40) | 0.20 |
| Breast | | 0.04 (0.03 - 0.05) | 0.04 |
| Digestive | 0.30 (0.12 - 0.43) | 0.52 (0.23 - 0.63) | 0.41 |
| Other | 0.25 (0.11 - 0.33) | 0.21 (0.16 - 0.26) | 0.25 |
| All cancers | 1.00 | 1.00 | 1.00 |

(a) Age-weighted average over all occupations
( ) Range of prjected averages among occupations

Table 6.5
Standardized Irradiation Ratio by cancer, occupation, and worker sex

Male Workers

| Occupation & Job Group | All Cancers | Leukemia | Respiratory | Breast | Digestive | Other |
|---|---|---|---|---|---|---|
| 1. ADMINISTRATIVE | | | | | | |
| Administrator | 0.80 | 1.59 | 1.59 | | 0.35 | 0.61 |
| Office Staff | 0.88 | 1.33 | 1.35 | | 0.58 | 0.86 |
| Safety Officer | 0.78 | 1.98 | 1.80 | | 0.23 | 0.35 |
| AVERAGE | 0.86 | 1.31 | 1.34 | | 0.55 | 0.86 |
| 2. MEDICAL | | | | | | |
| Chiropractor | 0.86 | 1.29 | 1.33 | | 0.54 | 0.86 |
| Dental Hygienist | 0.89 | 1.12 | 1.17 | | 0.66 | 1.02 |
| Dentist | 0.82 | 1.33 | 1.37 | | 0.45 | 0.79 |
| Gynaecologist | 0.85 | 1.55 | 1.49 | | 0.44 | 0.73 |
| Isotope Tech (NM) | 1.00 | 1.00 | 1.05 | | 0.92 | 1.18 |
| Lab Tech | 0.95 | 1.06 | 1.13 | | 0.81 | 1.09 |
| Medical Physicist | 0.82 | 1.27 | 1.34 | | 0.47 | 0.83 |
| Nurse | 0.85 | 1.18 | 1.26 | | 0.56 | 0.92 |
| Physician | 0.73 | 1.43 | 1.46 | | 0.21 | 0.71 |
| Radiological Tech (D) | 0.94 | 1.18 | 1.22 | | 0.74 | 1.01 |
| Radiological Tech (T) | 1.11 | 1.02 | 1.06 | | 1.14 | 1.23 |
| Radiologist (D) | 0.80 | 1.55 | 1.51 | | 0.38 | 0.65 |
| Radiologist (T) | 0.79 | 1.61 | 1.54 | | 0.35 | 0.60 |
| Veterinarian | 0.82 | 1.24 | 1.30 | | 0.48 | 0.86 |
| Ward Aid/orderly | 0.93 | 1.24 | 1.25 | | 0.72 | 0.97 |
| AVERAGE | 0.86 | 1.29 | 1.33 | | 0.54 | 0.86 |
| 3. INDUSTRY | | | | | | |
| Dial Painter | 0.88 | 1.04 | 1.14 | | 0.64 | 1.05 |
| Fuel Processor | 0.84 | 1.43 | 1.45 | | 0.45 | 0.76 |
| Industrial Radiographer | 0.97 | 1.08 | 1.11 | | 0.83 | 1.12 |
| Instrument Tech | 0.91 | 1.45 | 1.49 | | 0.39 | 0.70 |
| Instructor | 0.98 | 1.16 | 1.24 | | 0.62 | 0.96 |
| Lab Tech | 0.95 | 1.14 | 1.18 | | 0.78 | 1.05 |
| Scientist Engineer (Field) | 0.92 | 1.06 | 1.14 | | 0.74 | 1.06 |
| Scientist/Engineer (Lab) | 0.91 | 1.22 | 1.26 | | 0.67 | 0.95 |
| Well Logger | 1.07 | 0.86 | 0.92 | | 1.10 | 1.32 |
| AVERAGE | 0.94 | 1.12 | 1.18 | | 0.76 | 1.05 |
| 4. REACTOR WORKERS | | | | | | |
| Administration | 0.88 | 1.24 | 1.28 | | 0.59 | 0.92 |
| Chem&Rad Control | 0.84 | 1.22 | 1.28 | | 0.53 | 0.90 |
| Control Techs | 0.79 | 1.25 | 1.35 | | 0.40 | 0.82 |
| Electrical Maintenance | 0.87 | 1.12 | 1.21 | | 0.61 | 0.98 |
| Fuel Handling | 0.82 | 1.22 | 1.26 | | 0.47 | 0.91 |
| General Maintenance | 0.92 | 1.22 | 1.23 | | 0.70 | 1.00 |
| Health Physics | 0.80 | 1.20 | 1.30 | | 0.45 | 0.86 |
| Mechanical Maintenace | 0.88 | 1.18 | 1.25 | | 0.58 | 0.94 |
| Operations | 0.90 | 1.02 | 1.10 | | 0.71 | 1.09 |
| Construction | 0.86 | 1.35 | 1.39 | | 0.52 | 0.82 |
| Scientific/Professional | 0.84 | 1.27 | 1.35 | | 0.50 | 0.85 |
| Training | 0.81 | 1.33 | 1.41 | | 0.42 | 0.77 |
| Visitor | 0.97 | 1.24 | 1.18 | | 0.81 | 1.05 |
| AVERAGE | 0.88 | 1.22 | 1.25 | | 0.61 | 0.95 |
| 5. URANIUM MINERS | | | | | | |
| underground miners | 0.93 | 1.08 | 1.15 | | 0.69 | 1.05 |
| underground maintenance | 0.81 | 0.88 | 1.00 | | 0.56 | 1.14 |
| underground personnel | 0.87 | 1.27 | 1.30 | | 0.56 | 0.90 |
| support workers | 0.93 | 1.12 | 1.17 | | 0.68 | 1.03 |
| surface miners | 0.83 | 1.18 | 1.25 | | 0.58 | 0.94 |
| surface maintenance | 0.83 | 1.12 | 1.19 | | 0.59 | 0.99 |
| surface personnel | 0.81 | 1.43 | 1.47 | | 0.40 | 0.72 |
| surface support workers | 0.85 | 1.24 | 1.29 | | 0.54 | 0.89 |
| mill workers | 0.76 | 1.12 | 1.25 | | 0.39 | 0.90 |
| mill maintenace | 0.84 | 1.22 | 1.29 | | 0.52 | 0.88 |
| office staff | 0.82 | 1.33 | 1.40 | | 0.45 | 0.79 |
| nurses | 0.71 | 1.08 | 1.22 | | 0.30 | 0.88 |
| visitors | 0.90 | 1.31 | 1.35 | | 0.62 | 0.86 |
| TOTAL | 0.87 | 1.18 | 1.24 | | 0.60 | 0.95 |
| 6. MISCELLANEOUS | 0.96 | 1.10 | 1.14 | | 0.81 | 1.08 |
| All groups | 0.90 | 1.20 | 1.24 | | 0.66 | 0.97 |

Table 6.5 cont.
Standardized Irradiation Ratio by cancer, occupation, and worker sex

Female Workers

| Occupation & Job Group | All Cancers | Leukemia | Respiratory | Breast | Digestive | Other |
|---|---|---|---|---|---|---|
| 1. ADMINISTRATIVE | | | | | | |
| Administrator | 1.11 | 0.80 | 0.75 | 1.05 | 1.37 | 1.03 |
| Office Staff | 1.05 | 0.82 | 0.78 | 0.95 | 1.24 | 0.97 |
| Safety Officer | 0.89 | 0.98 | 0.95 | 0.75 | 0.90 | 0.74 |
| AVERAGE | 1.07 | 0.82 | 0.77 | 1.00 | 1.28 | 0.98 |
| 2. MEDICAL | | | | | | |
| Chiropractor | 1.06 | 0.80 | 0.76 | 1.00 | 1.27 | 0.99 |
| Dental Hygienist | 1.16 | 0.75 | 0.71 | 1.10 | 1.49 | 1.10 |
| Dentist | 0.97 | 0.76 | 0.77 | 1.00 | 1.10 | 0.95 |
| Gynaecologist | 1.43 | 0.57 | 0.59 | 1.25 | 2.10 | 1.30 |
| Isotope Tech (NM) | 1.22 | 0.71 | 0.69 | 1.10 | 1.62 | 1.14 |
| Lab Tech | 1.01 | 0.80 | 0.79 | 0.95 | 1.17 | 0.95 |
| Medical Physicist | 0.88 | 0.96 | 0.89 | 0.85 | 0.86 | 0.80 |
| Nurse | 0.88 | 0.90 | 0.87 | 0.85 | 0.87 | 0.81 |
| Physician | 0.79 | 0.90 | 0.87 | 0.85 | 0.69 | 0.77 |
| Radiological Tech (D) | 1.01 | 0.82 | 0.79 | 0.95 | 1.15 | 0.95 |
| Radiological Tech (T) | 1.03 | 0.82 | 0.79 | 0.95 | 1.20 | 0.95 |
| Radiologist (D) | 0.86 | 0.88 | 0.86 | 0.85 | 0.83 | 0.81 |
| Radiologist (T) | 0.82 | 0.90 | 0.87 | 0.80 | 0.76 | 0.77 |
| Veterinarian | 1.05 | 0.69 | 0.72 | 1.05 | 1.27 | 1.05 |
| Ward Aid/orderly | 1.11 | 0.84 | 0.76 | 1.00 | 1.37 | 1.01 |
| AVERAGE | 1.06 | 0.80 | 0.76 | 1.00 | 1.27 | 1.00 |
| 3. INDUSTRY | | | | | | |
| Dial Painter | | | | | | |
| Fuel Processor | 0.69 | 1.20 | 1.07 | 0.60 | 0.44 | 0.52 |
| Industrial Radiographer | 1.00 | 0.80 | 0.80 | 0.95 | 1.13 | 0.94 |
| Instrument Tech | 0.82 | 0.94 | 0.92 | 0.80 | 0.73 | 0.74 |
| Instructor | 1.12 | 0.80 | 0.75 | 1.00 | 1.40 | 1.03 |
| Lab Tech | 1.10 | 0.75 | 0.75 | 1.05 | 1.37 | 1.03 |
| Scientist Engineer (Field) | 1.11 | 0.71 | 0.74 | 1.05 | 1.41 | 1.04 |
| Scientist/Engineer (Lab) | 1.16 | 0.73 | 0.73 | 1.05 | 1.50 | 1.07 |
| Well Logger | 1.05 | 0.75 | 0.76 | 1.00 | 1.27 | 0.99 |
| AVERAGE | 1.09 | 0.76 | 0.75 | 1.00 | 1.35 | 1.01 |
| 4. REACTOR WORKERS | | | | | | |
| Administration | 1.00 | 0.84 | 0.80 | 0.95 | 1.14 | 0.94 |
| Chem&Rad Control | 1.14 | 0.69 | 0.69 | 1.10 | 1.46 | 1.10 |
| Control Techs | 1.51 | 0.55 | 0.57 | 1.25 | 2.29 | 1.34 |
| Electrical Maintenance | 1.25 | 0.83 | 0.85 | 1.15 | 1.71 | 1.18 |
| Fuel Handling | | | | | | |
| General Maintenance | 1.32 | 0.94 | 0.88 | 1.10 | 1.77 | 1.22 |
| Health Physics | 0.76 | 0.98 | 0.96 | 0.75 | 0.59 | 0.68 |
| Mechanical Maintenace | 0.94 | 0.87 | 0.72 | 1.10 | 1.02 | 1.02 |
| Operations | 1.14 | 0.87 | 0.70 | 1.10 | 1.48 | 1.09 |
| Construction | 0.99 | 0.92 | 0.80 | 0.95 | 1.09 | 0.95 |
| Scientific/Professional | 0.95 | 0.86 | 0.82 | 0.90 | 1.02 | 0.89 |
| Training | 0.99 | 0.87 | 0.71 | 1.10 | 1.13 | 1.04 |
| Visitor | 1.56 | 0.98 | 0.81 | 1.20 | 2.27 | 1.37 |
| AVERAGE | 1.08 | 0.86 | 0.76 | 1.00 | 1.30 | 1.01 |
| 5. URANIUM MINERS | | | | | | |
| underground miners | | | | | | |
| underground maintenance | 1.46 | 0.57 | 0.60 | 1.25 | 2.17 | 1.32 |
| underground personnel | 1.04 | 0.86 | 0.77 | 1.00 | 1.21 | 0.98 |
| support workers | 1.05 | 0.87 | 0.72 | 1.05 | 1.27 | 1.05 |
| surface miners | 1.46 | 0.57 | 0.60 | 1.25 | 2.17 | 1.31 |
| surface maintenance | 0.89 | 0.87 | 0.72 | 1.10 | 1.13 | 1.04 |
| surface personnel | 0.87 | 0.75 | 0.77 | 1.00 | 0.87 | 0.92 |
| surface support workers | 1.15 | 0.71 | 0.68 | 1.10 | 1.47 | 1.13 |
| mill workers | 0.79 | 1.31 | 1.05 | 0.55 | 0.65 | 0.52 |
| mill maintenace | 1.21 | 0.59 | 0.62 | 1.20 | 1.63 | 1.19 |
| office staff | 1.05 | 0.69 | 0.72 | 1.05 | 1.26 | 1.05 |
| nurses | 0.66 | 1.14 | 1.11 | 0.55 | 0.39 | 0.49 |
| visitors | 0.67 | 1.41 | 1.23 | 0.40 | 0.37 | 0.34 |
| TOTAL | 1.09 | 0.80 | 0.75 | 1.05 | 1.32 | 1.03 |
| 6. MISCELLANEOUS | 1.19 | 0.78 | 0.73 | 1.05 | 1.54 | 1.09 |
| All groups | 1.10 | 0.80 | 0.75 | 1.00 | 1.36 | 1.03 |

Table 6.5 cont.
Standardized Irradiation Ratio by cancer, occupation, and worker sex

Both Sexes

| Occupation & Job Group | All Cancers | Leukemia | Respiratory | Breast | Digestive | Other |
|---|---|---|---|---|---|---|
| 1. ADMINISTRATIVE | | | | | | |
| Administrator | 0.99 | 1.10 | 1.07 | | 0.98 | 0.87 |
| Office Staff | 1.04 | 0.86 | 0.82 | | 1.19 | 0.96 |
| Safety Officer | 0.81 | 1.73 | 1.58 | | 0.41 | 0.45 |
| AVERAGE | 1.03 | 0.86 | 0.83 | | 1.18 | 0.95 |
| 2. MEDICAL | | | | | | |
| Chiropractor | 0.99 | 0.98 | 0.97 | | 1.01 | 0.95 |
| Dental Hygienist | 1.15 | 0.75 | 0.72 | | 1.47 | 1.10 |
| Dentist | 0.84 | 1.25 | 1.28 | | 0.54 | 0.82 |
| Gynaecologist | 1.03 | 1.24 | 1.21 | | 0.97 | 0.91 |
| Isotope Tech (NM) | 1.14 | 0.80 | 0.80 | | 1.38 | 1.15 |
| Lab Tech | 0.99 | 0.88 | 0.89 | | 1.06 | 0.99 |
| Medical Physicist | 0.83 | 1.22 | 1.25 | | 0.54 | 0.83 |
| Nurse | 0.87 | 0.90 | 0.89 | | 0.86 | 0.81 |
| Physician | 0.79 | 1.35 | 1.38 | | 0.41 | 0.72 |
| Radiological Tech (D) | 0.99 | 0.88 | 0.86 | | 1.08 | 0.95 |
| Radiological Tech (T) | 1.04 | 0.86 | 0.83 | | 1.20 | 0.99 |
| Radiologist (D) | 0.82 | 1.41 | 1.38 | | 0.47 | 0.68 |
| Radiologist (T) | 0.80 | 1.47 | 1.42 | | 0.42 | 0.63 |
| Veterinarian | 0.90 | 1.04 | 1.09 | | 0.77 | 0.93 |
| Ward Aid/orderly | 1.06 | 0.98 | 0.92 | | 1.16 | 1.00 |
| AVERAGE | 0.99 | 0.98 | 0.97 | | 1.01 | 0.95 |
| 3. INDUSTRY | | | | | | |
| Dial Painter | 0.38 | 1.04 | 1.14 | | 0.65 | 1.05 |
| Fuel Processor | 0.33 | 1.41 | 1.43 | | 0.45 | 0.74 |
| Industrial Radiographer | 0.97 | 1.04 | 1.07 | | 0.86 | 1.10 |
| Instrument Tech | 0.90 | 1.37 | 1.41 | | 0.43 | 0.70 |
| Instructor | 0.89 | 1.16 | 1.22 | | 0.65 | 0.96 |
| Lab Tech | 1.01 | 0.98 | 1.00 | | 1.03 | 1.04 |
| Scientist Engineer (Field) | 0.93 | 1.04 | 1.12 | | 0.77 | 1.06 |
| Scientist/Engineer (Lab) | 0.97 | 1.12 | 1.15 | | 0.85 | 0.97 |
| Well Logger | 1.06 | 0.86 | 0.92 | | 1.10 | 1.32 |
| AVERAGE | 0.98 | 1.04 | 1.08 | | 0.90 | 1.04 |
| 4. REACTOR WORKERS | | | | | | |
| Administration | 0.93 | 1.08 | 1.09 | | 0.81 | 0.93 |
| Chem&Rad Control | 0.97 | 1.16 | 1.23 | | 0.62 | 0.92 |
| Control Techs | 0.90 | 1.25 | 1.34 | | 0.42 | 0.83 |
| Electrical Maintenance | 0.98 | 1.12 | 1.20 | | 0.63 | 0.99 |
| Fuel Handling | 0.92 | 1.22 | 1.26 | | 0.47 | 0.91 |
| General Maintenance | 0.97 | 1.18 | 1.16 | | 0.82 | 1.03 |
| Health Physics | 0.90 | 1.18 | 1.26 | | 0.46 | 0.85 |
| Mechanical Maintenace | 0.98 | 1.18 | 1.25 | | 0.58 | 0.94 |
| Operations | 0.90 | 1.00 | 1.09 | | 0.72 | 1.09 |
| Construction | 0.98 | 1.35 | 1.37 | | 0.53 | 0.82 |
| Scientific/Professional | 0.95 | 1.25 | 1.31 | | 0.54 | 0.85 |
| Training | 0.93 | 1.25 | 1.32 | | 0.51 | 0.81 |
| Visitor | 1.04 | 1.22 | 1.12 | | 0.97 | 1.08 |
| AVERAGE | 0.90 | 1.18 | 1.20 | | 0.69 | 0.95 |
| 5. URANIUM MINERS | | | | | | |
| underground miners | 0.90 | 1.08 | 1.15 | | 0.69 | 1.05 |
| underground maintenance | 1.03 | 0.78 | 0.86 | | 1.10 | 1.19 |
| underground personnel | 0.87 | 1.24 | 1.26 | | 0.60 | 0.90 |
| support workers | 0.90 | 1.12 | 1.17 | | 0.69 | 1.03 |
| surface miners | 0.87 | 1.18 | 1.25 | | 0.61 | 0.95 |
| surface maintenance | 0.86 | 1.10 | 1.19 | | 0.60 | 0.99 |
| surface personnel | 0.81 | 1.39 | 1.44 | | 0.42 | 0.73 |
| surface support workers | 0.88 | 1.18 | 1.23 | | 0.64 | 0.92 |
| mill workers | 0.79 | 1.31 | 1.06 | | 0.64 | 0.55 |
| mill maintenace | 0.84 | 1.22 | 1.29 | | 0.52 | 0.89 |
| office staff | 0.85 | 1.24 | 1.31 | | 0.56 | 0.83 |
| nurses | 0.68 | 1.12 | 1.15 | | 0.36 | 0.62 |
| visitors | 0.87 | 1.33 | 1.33 | | 0.59 | 0.79 |
| TOTAL | 0.88 | 1.18 | 1.22 | | 0.63 | 0.95 |
| 6. MISCELLANEOUS | 1.10 | 0.94 | 0.89 | | 1.25 | 1.08 |
| All groups | 1.00 | 1.00 | 1.00 | | 1.00 | 1.00 |

a Proportion under and over the age of 35

Table 6.6
Projected total lifetime fatal cancer risk per unit exposure and the relative contributions of different cancer groups for the "average" worker by occupation and sex

Male Workers

| Occupation & Job Group | All Cancers (a) | Leukemia | Respiratory | Breast | Digestive | Other |
|---|---|---|---|---|---|---|
| 1. ADMINISTRATIVE | | | | | | |
| Administrator | 379 | 0.21 | 0.43 | | 0.18 | 0.18 |
| Office Staff | 413 | 0.16 | 0.33 | | 0.27 | 0.23 |
| Safety Officer | 369 | 0.27 | 0.50 | | 0.12 | 0.11 |
| AVERAGE | 407 | 0.16 | 0.34 | | 0.27 | 0.23 |
| 2. MEDICAL | | | | | | |
| Chiropractor | 403 | 0.16 | 0.34 | | 0.26 | 0.24 |
| Dental Hygienist | 420 | 0.14 | 0.28 | | 0.31 | 0.27 |
| Dentist | 384 | 0.18 | 0.36 | | 0.23 | 0.23 |
| Gynaecologist | 368 | 0.20 | 0.38 | | 0.22 | 0.20 |
| Isotope Tech (NM) | 470 | 0.11 | 0.23 | | 0.39 | 0.28 |
| Lab Tech | 449 | 0.12 | 0.26 | | 0.35 | 0.27 |
| Medical Physicist | 386 | 0.17 | 0.35 | | 0.24 | 0.24 |
| Nurse | 401 | 0.15 | 0.32 | | 0.27 | 0.25 |
| Physician | 342 | 0.21 | 0.44 | | 0.12 | 0.23 |
| Radiological Tech (D) | 442 | 0.14 | 0.28 | | 0.33 | 0.25 |
| Radiological Tech (T) | 521 | 0.10 | 0.21 | | 0.43 | 0.26 |
| Radiologist (D) | 379 | 0.21 | 0.41 | | 0.20 | 0.19 |
| Radiologist (T) | 374 | 0.22 | 0.42 | | 0.18 | 0.18 |
| Veterinarian | 388 | 0.16 | 0.34 | | 0.24 | 0.25 |
| Ward Aid/orderly | 439 | 0.14 | 0.29 | | 0.32 | 0.25 |
| AVERAGE | 403 | 0.16 | 0.34 | | 0.26 | 0.24 |
| 3. INDUSTRY | | | | | | |
| Dial Painter | 413 | 0.13 | 0.28 | | 0.31 | 0.28 |
| Fuel Processor | 394 | 0.19 | 0.38 | | 0.23 | 0.21 |
| Industrial Radiographer | 455 | 0.12 | 0.25 | | 0.36 | 0.27 |
| Instrument Tech | 383 | 0.19 | 0.40 | | 0.20 | 0.21 |
| Instructor | 414 | 0.14 | 0.30 | | 0.29 | 0.26 |
| Lab Tech | 443 | 0.13 | 0.27 | | 0.34 | 0.26 |
| Scientist Engineer (Field) | 433 | 0.12 | 0.27 | | 0.33 | 0.27 |
| Scientist/Engineer (Lab) | 428 | 0.14 | 0.30 | | 0.31 | 0.25 |
| Well Logger | 502 | 0.09 | 0.19 | | 0.43 | 0.29 |
| AVERAGE | 443 | 0.13 | 0.27 | | 0.34 | 0.26 |
| 4. REACTOR WORKERS | | | | | | |
| Administration | 418 | 0.15 | 0.32 | | 0.28 | 0.25 |
| Chem&Rad Control | 397 | 0.16 | 0.33 | | 0.26 | 0.25 |
| Control Techs | 377 | 0.17 | 0.37 | | 0.21 | 0.25 |
| Electrical Maintenance | 410 | 0.14 | 0.30 | | 0.30 | 0.27 |
| Fuel Handling | 384 | 0.16 | 0.34 | | 0.24 | 0.26 |
| General Maintenance | 439 | 0.14 | 0.29 | | 0.31 | 0.26 |
| Health Physics | 378 | 0.16 | 0.35 | | 0.23 | 0.25 |
| Mechanical Maintenace | 407 | 0.15 | 0.31 | | 0.28 | 0.26 |
| Operations | 424 | 0.12 | 0.26 | | 0.33 | 0.29 |
| Construction | 404 | 0.17 | 0.35 | | 0.25 | 0.23 |
| Scientific/Professional | 395 | 0.16 | 0.35 | | 0.25 | 0.24 |
| Training | 381 | 0.18 | 0.38 | | 0.22 | 0.23 |
| Visitor | 456 | 0.14 | 0.26 | | 0.35 | 0.25 |
| AVERAGE | 416 | 0.15 | 0.31 | | 0.29 | 0.25 |
| 5. URANIUM MINERS | | | | | | |
| underground miners | 425 | 0.13 | 0.28 | | 0.32 | 0.28 |
| underground maintenance | 383 | 0.12 | 0.27 | | 0.29 | 0.33 |
| underground personnel | 408 | 0.16 | 0.33 | | 0.27 | 0.25 |
| support workers | 423 | 0.13 | 0.28 | | 0.31 | 0.27 |
| surface miners | 406 | 0.15 | 0.32 | | 0.28 | 0.26 |
| surface maintenance | 405 | 0.14 | 0.30 | | 0.29 | 0.27 |
| surface personnel | 382 | 0.19 | 0.39 | | 0.21 | 0.21 |
| surface support workers | 400 | 0.16 | 0.33 | | 0.27 | 0.25 |
| mill workers | 360 | 0.16 | 0.35 | | 0.21 | 0.28 |
| mill maintenace | 394 | 0.16 | 0.34 | | 0.26 | 0.25 |
| office staff | 388 | 0.18 | 0.37 | | 0.23 | 0.23 |
| nurses | 336 | 0.16 | 0.37 | | 0.18 | 0.29 |
| visitors | 422 | 0.16 | 0.33 | | 0.29 | 0.23 |
| TOTAL | 411 | 0.15 | 0.31 | | 0.29 | 0.26 |
| 6. MISCELLANEOUS | 451 | 0.12 | 0.26 | | 0.35 | 0.27 |
| All groups | 425 | 0.15 | 0.30 | | 0.30 | 0.25 |

(a) Lifetime risk of fatal cancer per unit dose (10E-04 per Sv)

Table 6.6 cont.
Projected total lifetime fatal cancer risk per unit exposure and the relative contributions of different cancer groups for the "average" worker by occupation and sex

Female Workers

| Occupation & Job Group | All Cancers (a) | Leukemia | Respiratory | Breast | Digestive | Other |
|---|---|---|---|---|---|---|
| 1. ADMINISTRATIVE | | | | | | |
| Administrator | 522 | 0.08 | 0.15 | 0.04 | 0.52 | 0.22 |
| Office Staff | 493 | 0.09 | 0.16 | 0.04 | 0.49 | 0.22 |
| Safety Officer | 421 | 0.12 | 0.23 | 0.04 | 0.42 | 0.19 |
| AVERAGE | 503 | 0.08 | 0.16 | 0.04 | 0.50 | 0.22 |
| 2. MEDICAL | | | | | | |
| Chiropractor | 501 | 0.08 | 0.16 | 0.04 | 0.50 | 0.22 |
| Dental Hygienist | 547 | 0.07 | 0.13 | 0.04 | 0.54 | 0.22 |
| Dentist | 459 | 0.08 | 0.17 | 0.04 | 0.47 | 0.23 |
| Gynaecologist | 672 | 0.04 | 0.09 | 0.04 | 0.62 | 0.21 |
| Isotope Tech (NM) | 574 | 0.06 | 0.12 | 0.04 | 0.56 | 0.22 |
| Lab Tech | 470 | 0.09 | 0.17 | 0.04 | 0.48 | 0.22 |
| Medical Physicist | 415 | 0.12 | 0.22 | 0.04 | 0.41 | 0.21 |
| Nurse | 413 | 0.11 | 0.22 | 0.04 | 0.41 | 0.22 |
| Physician | 374 | 0.12 | 0.24 | 0.05 | 0.36 | 0.23 |
| Radiological Tech (D) | 474 | 0.09 | 0.17 | 0.04 | 0.48 | 0.22 |
| Radiological Tech (T) | 485 | 0.09 | 0.17 | 0.04 | 0.49 | 0.22 |
| Radiologist (D) | 403 | 0.11 | 0.22 | 0.04 | 0.40 | 0.22 |
| Radiologist (T) | 386 | 0.12 | 0.23 | 0.04 | 0.39 | 0.22 |
| Veterinarian | 496 | 0.07 | 0.15 | 0.04 | 0.51 | 0.23 |
| Ward Aid/orderly | 523 | 0.08 | 0.15 | 0.04 | 0.52 | 0.21 |
| AVERAGE | 501 | 0.08 | 0.16 | 0.04 | 0.50 | 0.22 |
| 3. INDUSTRY | | | | | | |
| Dial Painter | | | | | | |
| Fuel Processor | 326 | 0.19 | 0.33 | 0.04 | 0.26 | 0.18 |
| Industrial Radiographer | 469 | 0.09 | 0.17 | 0.04 | 0.48 | 0.22 |
| Instrument Tech | 384 | 0.13 | 0.24 | 0.04 | 0.38 | 0.21 |
| Instructor | 527 | 0.08 | 0.15 | 0.04 | 0.52 | 0.22 |
| Lab Tech | 519 | 0.07 | 0.15 | 0.04 | 0.52 | 0.22 |
| Scientist Engineer (Field) | 525 | 0.07 | 0.14 | 0.04 | 0.53 | 0.22 |
| Scientist/Engineer (Lab) | 546 | 0.07 | 0.14 | 0.04 | 0.54 | 0.22 |
| Well Logger | 496 | 0.08 | 0.16 | 0.04 | 0.50 | 0.22 |
| AVERAGE | 513 | 0.08 | 0.15 | 0.04 | 0.52 | 0.22 |
| 4. REACTOR WORKERS | | | | | | |
| Administration | 472 | 0.09 | 0.17 | 0.04 | 0.47 | 0.22 |
| Chem&Rad Control | 536 | 0.07 | 0.13 | 0.04 | 0.54 | 0.23 |
| Control Techs | 711 | 0.04 | 0.08 | 0.04 | 0.83 | 0.21 |
| Electrical Maintenance | 589 | 0.05 | 0.11 | 0.04 | 0.57 | 0.22 |
| Fuel Handling | | | | | | |
| General Maintenance | 622 | 0.08 | 0.11 | 0.04 | 0.56 | 0.22 |
| Health Physics | 356 | 0.14 | 0.28 | 0.04 | 0.33 | 0.21 |
| Mechanical Maintenace | 443 | 0.08 | 0.16 | 0.05 | 0.45 | 0.26 |
| Operations | 536 | 0.06 | 0.13 | 0.04 | 0.54 | 0.23 |
| Construction | 468 | 0.10 | 0.18 | 0.04 | 0.46 | 0.23 |
| Scientific/Professional | 448 | 0.10 | 0.19 | 0.04 | 0.45 | 0.22 |
| Training | 465 | 0.07 | 0.15 | 0.05 | 0.48 | 0.25 |
| Visitor | 736 | 0.07 | 0.08 | 0.03 | 0.61 | 0.21 |
| AVERAGE | 510 | 0.09 | 0.15 | 0.04 | 0.50 | 0.22 |
| 5. URANIUM MINERS | | | | | | |
| underground miners | | | | | | |
| underground maintenance | 688 | 0.04 | 0.09 | 0.04 | 0.62 | 0.21 |
| underground personnel | 490 | 0.09 | 0.16 | 0.04 | 0.49 | 0.22 |
| support workers | 494 | 0.07 | 0.15 | 0.04 | 0.51 | 0.23 |
| surface miners | 687 | 0.04 | 0.09 | 0.04 | 0.62 | 0.21 |
| surface maintenance | 467 | 0.07 | 0.16 | 0.05 | 0.48 | 0.25 |
| surface personnel | 411 | 0.09 | 0.19 | 0.05 | 0.42 | 0.25 |
| surface support workers | 542 | 0.07 | 0.13 | 0.04 | 0.54 | 0.23 |
| mill workers | 372 | 0.18 | 0.29 | 0.03 | 0.35 | 0.16 |
| mill maintenace | 570 | 0.05 | 0.11 | 0.04 | 0.56 | 0.23 |
| office staff | 493 | 0.07 | 0.15 | 0.04 | 0.50 | 0.24 |
| nurses | 312 | 0.19 | 0.36 | 0.04 | 0.24 | 0.17 |
| visitors | 315 | 0.23 | 0.40 | 0.03 | 0.23 | 0.12 |
| TOTAL | 512 | 0.08 | 0.15 | 0.04 | 0.51 | 0.22 |
| 6. MISCELLANEOUS | 559 | 0.07 | 0.13 | 0.04 | 0.54 | 0.22 |
| All groups | 520 | 0.08 | 0.15 | 0.04 | 0.52 | 0.21 |

(a) Lifetime risk of fatal cancer per unit dose (10E-04 per Sv)

Table 6.6 cont.
Projected total lifetime fatal cancer risk per unit exposure and the relative contributions of different cancer groups for the "average" worker by occupation and sex

Both Sexes

| Occupation & Job Group | All Cancers (a) | Leukemia | Respiratory | Breast | Digestive | Other |
|---|---|---|---|---|---|---|
| 1. ADMINISTRATIVE | | | | | | |
| Administrator | 476 | 0.12 | 0.23 | 0.04 | 0.41 | 0.20 |
| Office Staff | 489 | 0.09 | 0.17 | 0.04 | 0.48 | 0.22 |
| Safety Officer | 394 | 0.22 | 0.41 | 0.04 | 0.20 | 0.13 |
| AVERAGE | 486 | 0.09 | 0.17 | 0.04 | 0.48 | 0.22 |
| 2. MEDICAL | | | | | | |
| Chiropractor | 473 | 0.11 | 0.21 | 0.04 | 0.42 | 0.22 |
| Dental Hygienist | 544 | 0.07 | 0.13 | 0.04 | 0.53 | 0.22 |
| Dentist | 412 | 0.16 | 0.32 | 0.05 | 0.26 | 0.22 |
| Gynaecologist | 504 | 0.13 | 0.24 | 0.05 | 0.38 | 0.20 |
| Isotope Tech (NM) | 545 | 0.08 | 0.15 | 0.04 | 0.50 | 0.23 |
| Lab Tech | 473 | 0.10 | 0.19 | 0.04 | 0.44 | 0.23 |
| Medical Physicist | 408 | 0.15 | 0.32 | 0.04 | 0.26 | 0.23 |
| Nurse | 413 | 0.11 | 0.22 | 0.04 | 0.41 | 0.22 |
| Physician | 387 | 0.18 | 0.36 | 0.04 | 0.21 | 0.21 |
| Radiological Tech (D) | 471 | 0.10 | 0.19 | 0.04 | 0.45 | 0.23 |
| Radiological Tech (T) | 494 | 0.09 | 0.17 | 0.04 | 0.48 | 0.22 |
| Radiologist (D) | 398 | 0.18 | 0.35 | 0.04 | 0.23 | 0.19 |
| Radiologist (T) | 389 | 0.19 | 0.37 | 0.04 | 0.21 | 0.18 |
| Veterinarian | 439 | 0.12 | 0.25 | 0.05 | 0.34 | 0.23 |
| Ward Aid/orderly | 504 | 0.10 | 0.19 | 0.04 | 0.45 | 0.22 |
| AVERAGE | 473 | 0.11 | 0.21 | 0.04 | 0.42 | 0.22 |
| 3. INDUSTRY | | | | | | |
| Dial Painter | 415 | 0.13 | 0.28 | 0.00 | 0.31 | 0.28 |
| Fuel Processor | 401 | 0.18 | 0.36 | 0.03 | 0.22 | 0.20 |
| Industrial Radiographer | 472 | 0.11 | 0.23 | 0.04 | 0.36 | 0.26 |
| Instrument Tech | 393 | 0.18 | 0.37 | 0.04 | 0.22 | 0.20 |
| Instructor | 438 | 0.13 | 0.26 | 0.05 | 0.29 | 0.24 |
| Lab Tech | 490 | 0.10 | 0.21 | 0.04 | 0.41 | 0.23 |
| Scientist Engineer (Field) | 457 | 0.12 | 0.25 | 0.05 | 0.33 | 0.26 |
| Scientist/Engineer (Lab) | 471 | 0.12 | 0.25 | 0.04 | 0.36 | 0.23 |
| Well Logger | 521 | 0.08 | 0.18 | 0.04 | 0.42 | 0.28 |
| AVERAGE | 475 | 0.11 | 0.23 | 0.04 | 0.37 | 0.24 |
| 4. REACTOR WORKERS | | | | | | |
| Administration | 448 | 0.12 | 0.25 | 0.04 | 0.36 | 0.23 |
| Chem&Rad Control | 430 | 0.14 | 0.29 | 0.05 | 0.28 | 0.24 |
| Control Techs | 401 | 0.16 | 0.34 | 0.06 | 0.21 | 0.23 |
| Electrical Maintenance | 436 | 0.13 | 0.26 | 0.05 | 0.28 | 0.25 |
| Fuel Handling | 384 | 0.16 | 0.34 | 0.00 | 0.24 | 0.26 |
| General Maintenance | 475 | 0.13 | 0.25 | 0.05 | 0.34 | 0.24 |
| Health Physics | 389 | 0.15 | 0.33 | 0.04 | 0.23 | 0.24 |
| Mechanical Maintenace | 428 | 0.14 | 0.30 | 0.05 | 0.27 | 0.24 |
| Operations | 447 | 0.11 | 0.25 | 0.05 | 0.32 | 0.27 |
| Construction | 424 | 0.16 | 0.33 | 0.04 | 0.25 | 0.21 |
| Scientific/Professional | 416 | 0.15 | 0.32 | 0.04 | 0.25 | 0.23 |
| Training | 411 | 0.16 | 0.33 | 0.05 | 0.24 | 0.22 |
| Visitor | 511 | 0.12 | 0.22 | 0.05 | 0.37 | 0.23 |
| AVERAGE | 444 | 0.14 | 0.27 | 0.05 | 0.31 | 0.24 |
| 5. URANIUM MINERS | | | | | | |
| underground miners | 425 | 0.13 | 0.28 | 0.00 | 0.32 | 0.28 |
| underground maintenance | 501 | 0.08 | 0.16 | 0.05 | 0.43 | 0.26 |
| underground personnel | 430 | 0.15 | 0.30 | 0.05 | 0.27 | 0.23 |
| support workers | 446 | 0.13 | 0.27 | 0.05 | 0.30 | 0.26 |
| surface miners | 437 | 0.14 | 0.29 | 0.06 | 0.27 | 0.24 |
| surface maintenance | 427 | 0.13 | 0.28 | 0.05 | 0.28 | 0.26 |
| surface personnel | 402 | 0.18 | 0.37 | 0.05 | 0.21 | 0.20 |
| surface support workers | 435 | 0.14 | 0.29 | 0.05 | 0.29 | 0.23 |
| mill workers | 373 | 0.18 | 0.29 | 0.03 | 0.34 | 0.16 |
| mill maintenace | 420 | 0.15 | 0.31 | 0.06 | 0.25 | 0.24 |
| office staff | 420 | 0.15 | 0.32 | 0.05 | 0.26 | 0.22 |
| nurses | 325 | 0.18 | 0.36 | 0.03 | 0.22 | 0.21 |
| visitors | 416 | 0.16 | 0.33 | 0.02 | 0.28 | 0.21 |
| TOTAL | 436 | 0.14 | 0.28 | 0.05 | 0.29 | 0.24 |
| 6. MISCELLANEOUS | 526 | 0.09 | 0.17 | 0.04 | 0.47 | 0.23 |
| All groups | 481 | 0.11 | 0.21 | 0.04 | 0.41 | 0.23 |

(a) Lifetime risk of fatal cancer per unit dose (10E-04 per Sv)

Table 6.7
Summary of the standardized irradiation ratio and cancer weighting factors for the "average" worker in the 6 main occupation categories

Standized irradiation ratio

| Occupation Category | All cancers | | | Leukemia | | | Respiratory cancers | | | Breast cancers | | | Digestive cancers | | | Other cancers | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Males | Females | Both sexes | Males | Females | Both sexes | Males | Females | Both sexes | Males | Females | Both sexes | Males | Females | Both sexes | Males | Females | Both sexes |
| Administrative | 0.86 | 1.07 | 1.03 | 1.31 | 0.82 | 0.86 | 1.34 | 0.77 | 0.83 | -- | 1.00 | -- | 0.55 | 1.28 | 0.98 | 0.86 | 0.98 | 0.87 |
| Medical | 0.86 | 1.06 | 0.99 | 1.29 | 0.80 | 0.98 | 1.33 | 0.76 | 0.97 | -- | 1.00 | -- | 0.54 | 1.27 | 1.01 | 0.86 | 1.00 | 0.95 |
| Industry | 0.94 | 1.08 | 0.98 | 1.12 | 0.76 | 1.04 | 1.18 | 0.75 | 1.08 | -- | 1.00 | -- | 0.76 | 1.35 | 0.90 | 1.05 | 1.01 | 1.04 |
| Reactor Workers | 0.88 | 1.09 | 0.90 | 1.22 | 0.86 | 1.18 | 1.25 | 0.76 | 1.20 | -- | 1.00 | -- | 0.61 | 1.30 | 0.69 | 0.95 | 1.01 | 0.95 |
| Uranium mines | 0.87 | 1.19 | 0.88 | 1.18 | 0.80 | 1.18 | 1.24 | 0.75 | 1.22 | -- | 1.05 | -- | 0.60 | 1.32 | 0.63 | 0.95 | 1.03 | 0.95 |
| Miscellaneous | 0.96 | 1.19 | 1.10 | 1.10 | 0.78 | 0.94 | 1.14 | 0.73 | 0.89 | -- | 1.05 | -- | 0.81 | 1.54 | 1.25 | 1.08 | 1.09 | 1.08 |
| All occupations | 0.90 | 1.20 | 1.00 | 1.20 | 0.80 | 1.00 | 1.24 | 0.75 | 1.00 | -- | 1.00 | -- | 0.66 | 1.36 | 1.00 | 0.97 | 1.03 | 1.00 |

Cancer weighting factors

| Occupation Category | Leukemia | | | Respiratory cancers | | | Breast cancers | | | Digestive cancers | | | Other cancers | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Males | Females | Both sexes | Males | Females | Both sexes | Males | Females | Both sexes | Males | Females | Both sexes | Males | Females | Both sexes |
| Administrative | 0.16 | 0.08 | 0.09 | 0.34 | 0.16 | 0.17 | -- | 0.04 | 0.04 | 0.27 | 0.50 | 0.48 | 0.23 | 0.22 | 0.22 |
| Medical | 0.16 | 0.08 | 0.11 | 0.34 | 0.16 | 0.21 | -- | 0.04 | 0.04 | 0.26 | 0.50 | 0.42 | 0.24 | 0.22 | 0.22 |
| Industry | 0.13 | 0.08 | 0.11 | 0.27 | 0.15 | 0.23 | -- | 0.04 | 0.04 | 0.34 | 0.52 | 0.37 | 0.26 | 0.22 | 0.24 |
| Reactor Workers | 0.15 | 0.09 | 0.14 | 0.31 | 0.15 | 0.27 | -- | 0.04 | 0.05 | 0.29 | 0.50 | 0.31 | 0.25 | 0.22 | 0.24 |
| Uranium mines | 0.15 | 0.08 | 0.14 | 0.31 | 0.15 | 0.28 | -- | 0.04 | 0.05 | 0.29 | 0.51 | 0.29 | 0.26 | 0.22 | 0.24 |
| Miscellaneous | 0.12 | 0.07 | 0.09 | 0.26 | 0.13 | 0.17 | -- | 0.04 | 0.04 | 0.35 | 0.54 | 0.47 | 0.27 | 0.22 | 0.23 |
| All occupations | 0.14 | 0.08 | 0.11 | 0.30 | 0.15 | 0.21 | -- | 0.04 | 0.04 | 0.31 | 0.52 | 0.41 | 0.25 | 0.22 | 0.23 |

Table 6.8
Comparison of the overall risk of fatal cancer per unit dose for a life-table working population and the 1988 Canadian radiation workforce

Lifetime risk of fatal cancer (10E-04 per Sv)

| Population | All cancers as a group | | | Leukemia | | | Respiratory Tract | | | Female Breast | | | Digestive System | | | Other sites | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Males | Females | M/F | Males | Females | M/F | Males | Females | M/F | Males | Females | M/F | Males | Females | M/F | Males | Females | M/F |
| Life-table Working Population (ages 18-65) | 455 | 440 | 1.0 | 73 | 57 | 1.3 | 135 | 95 | 1.4 | – | 15 | – | 145 | 190 | 0.8 | 103 | 83 | 1.2 |
| 1988 Canadian Radiation Workforce | 425 | 520 | 0.8 | 60 | 40 | 1.5 | 125 | 75 | 1.7 | – | 20 | – | 130 | 270 | 0.5 | 110 | 115 | 1.0 |

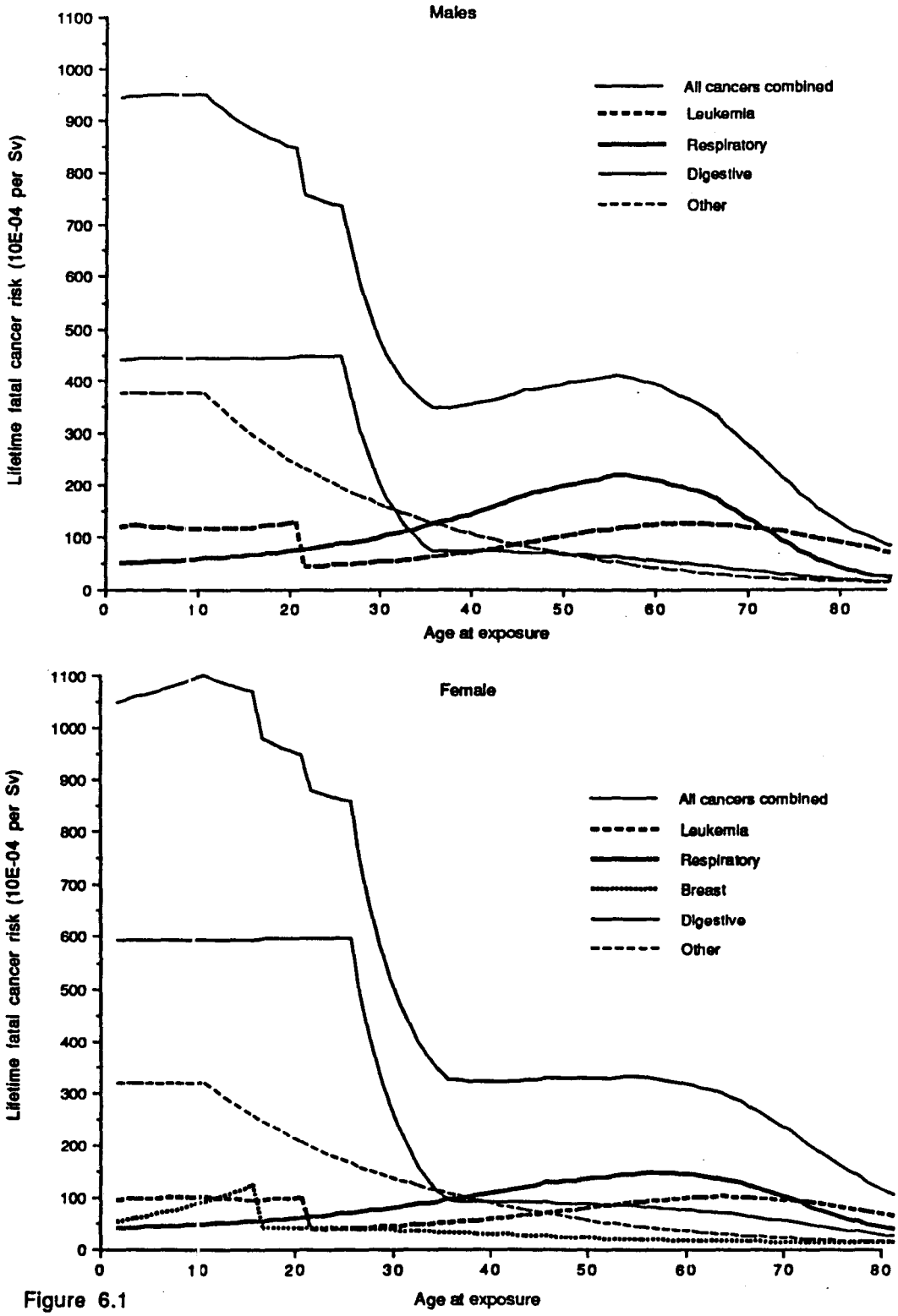


Figure 6.1
Lifetime fatal cancer risk per unit dose by age, sex, and cancer group projected for the Canadian population

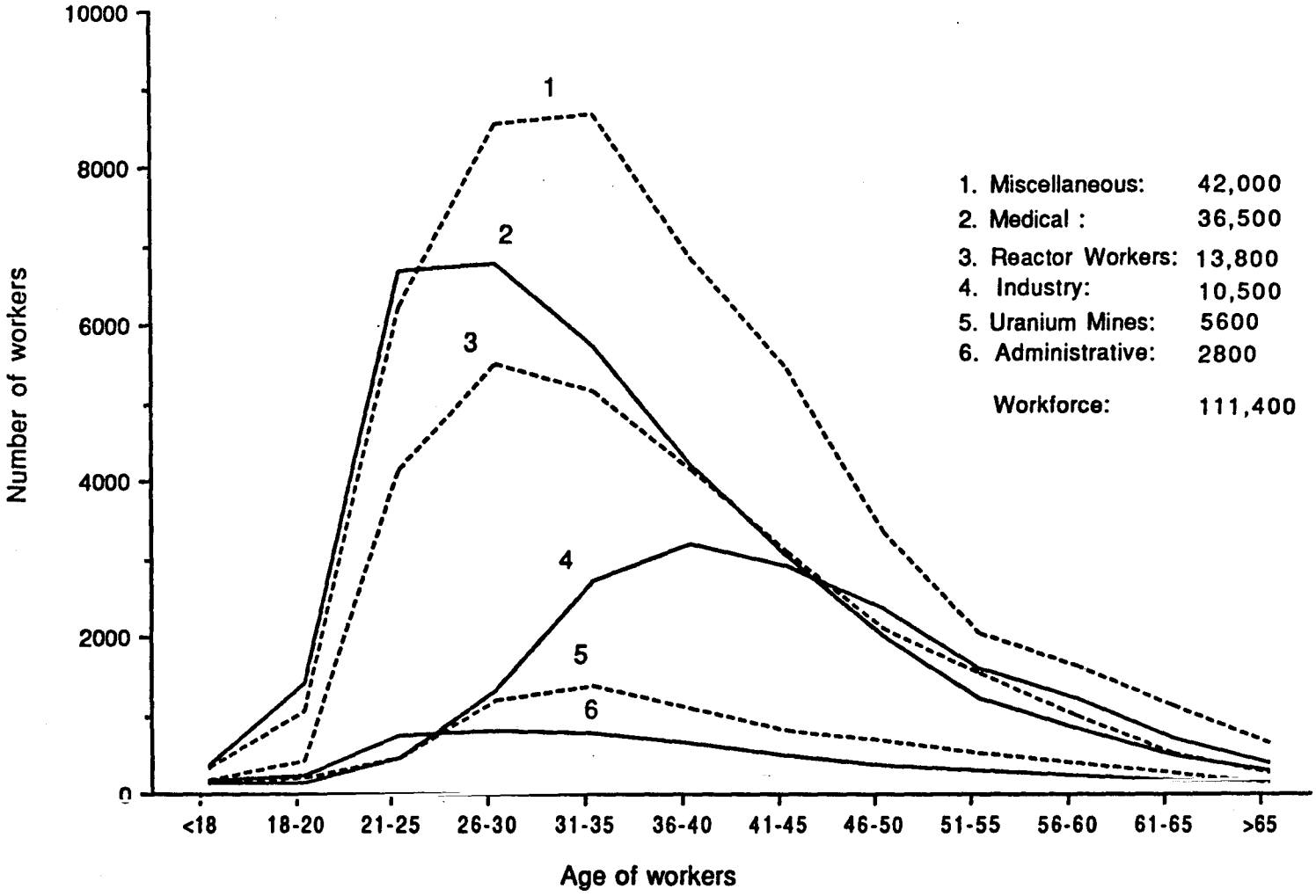


Figure 6.2
Age distribution of the 1988 Canadian "radiation" workforce by occupation

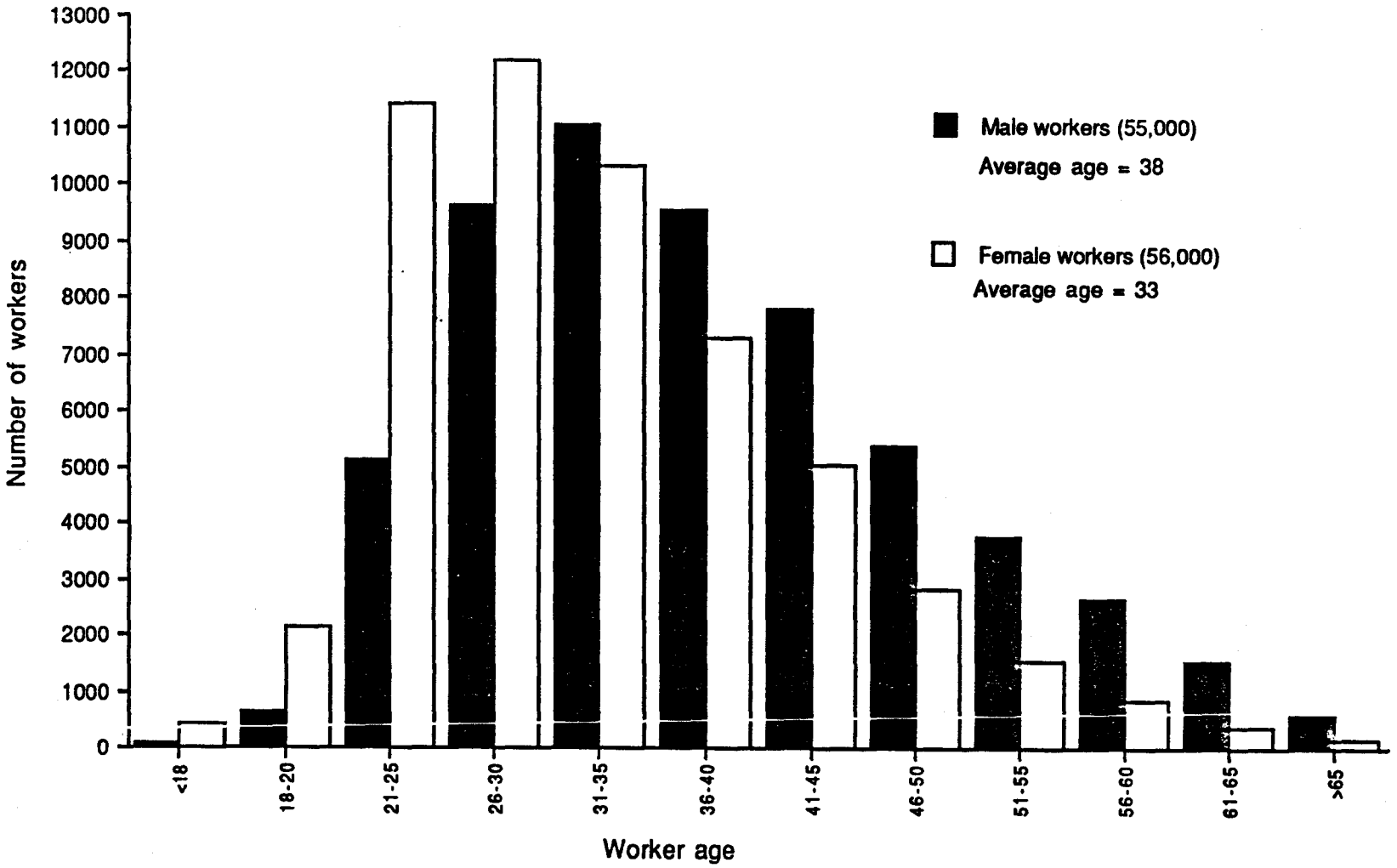


Figure 6.3
Age distribution among Canadian male and female workers

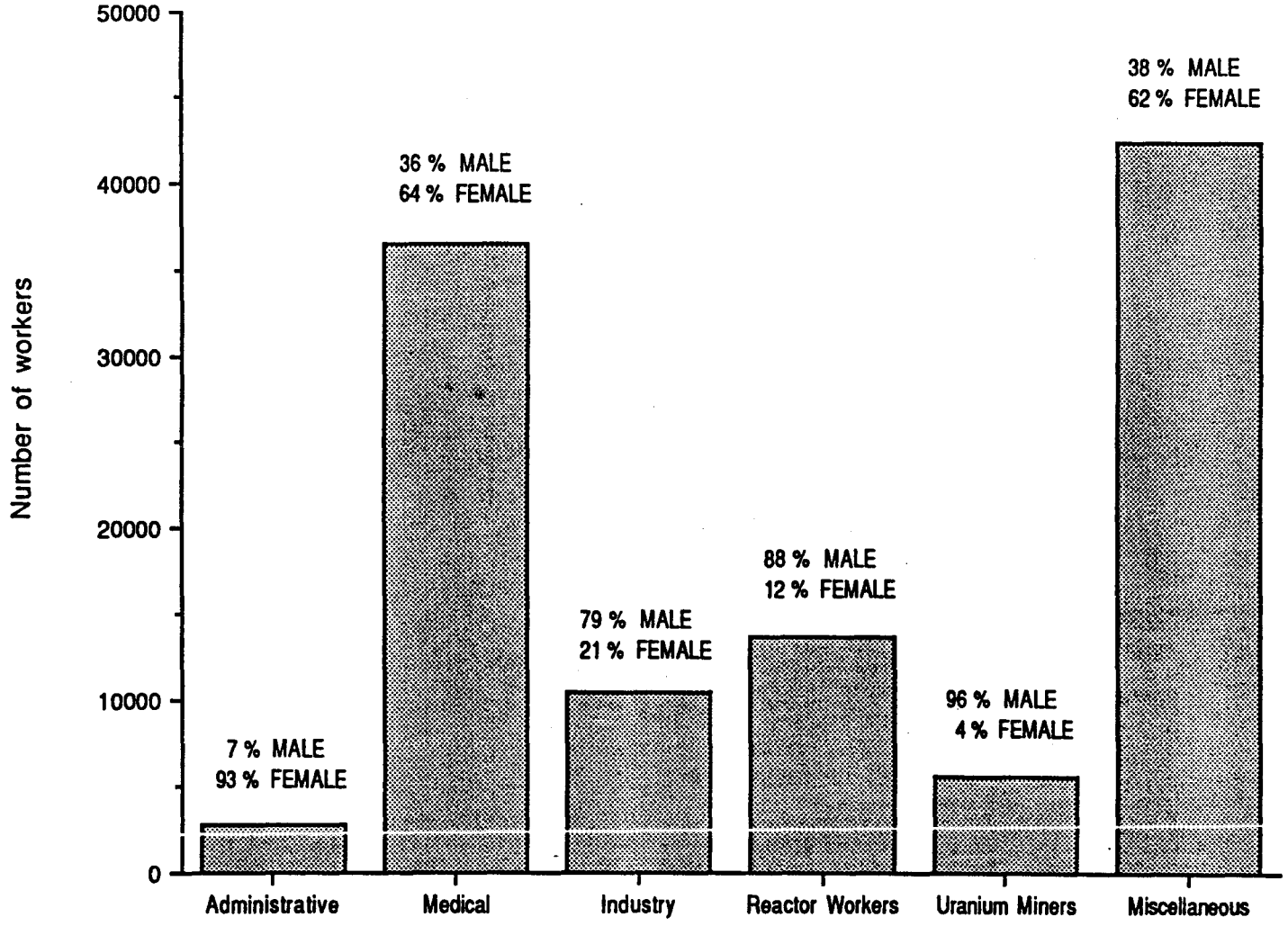


Figure 6.4
Number of workers in the 1988 Canadian "radiation" workforce by occupation and proportion of male and female workers

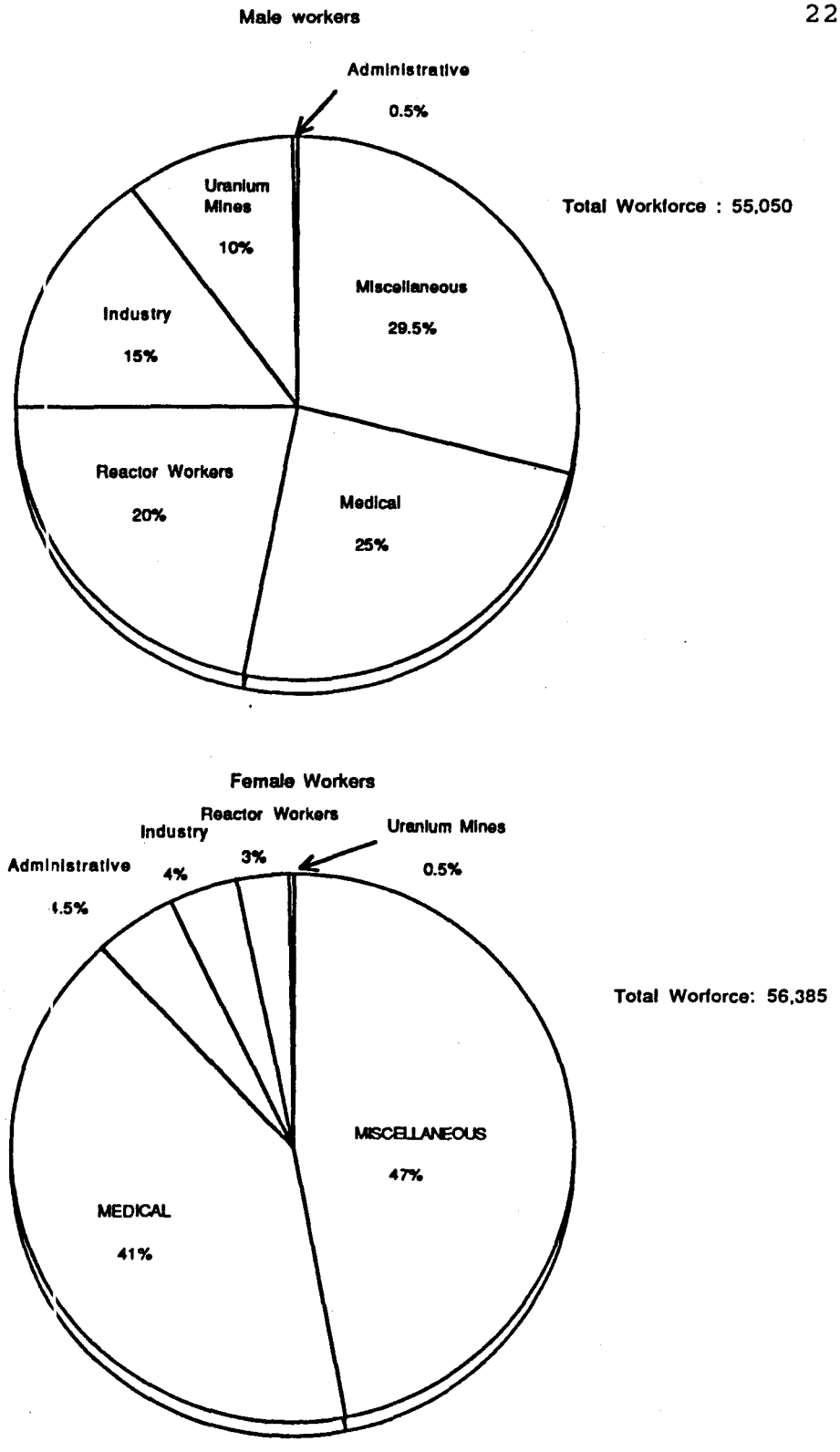


Figure 6.5
Proportional breakdown of 1988 Canadian "radiation" workforce by sex and occupation

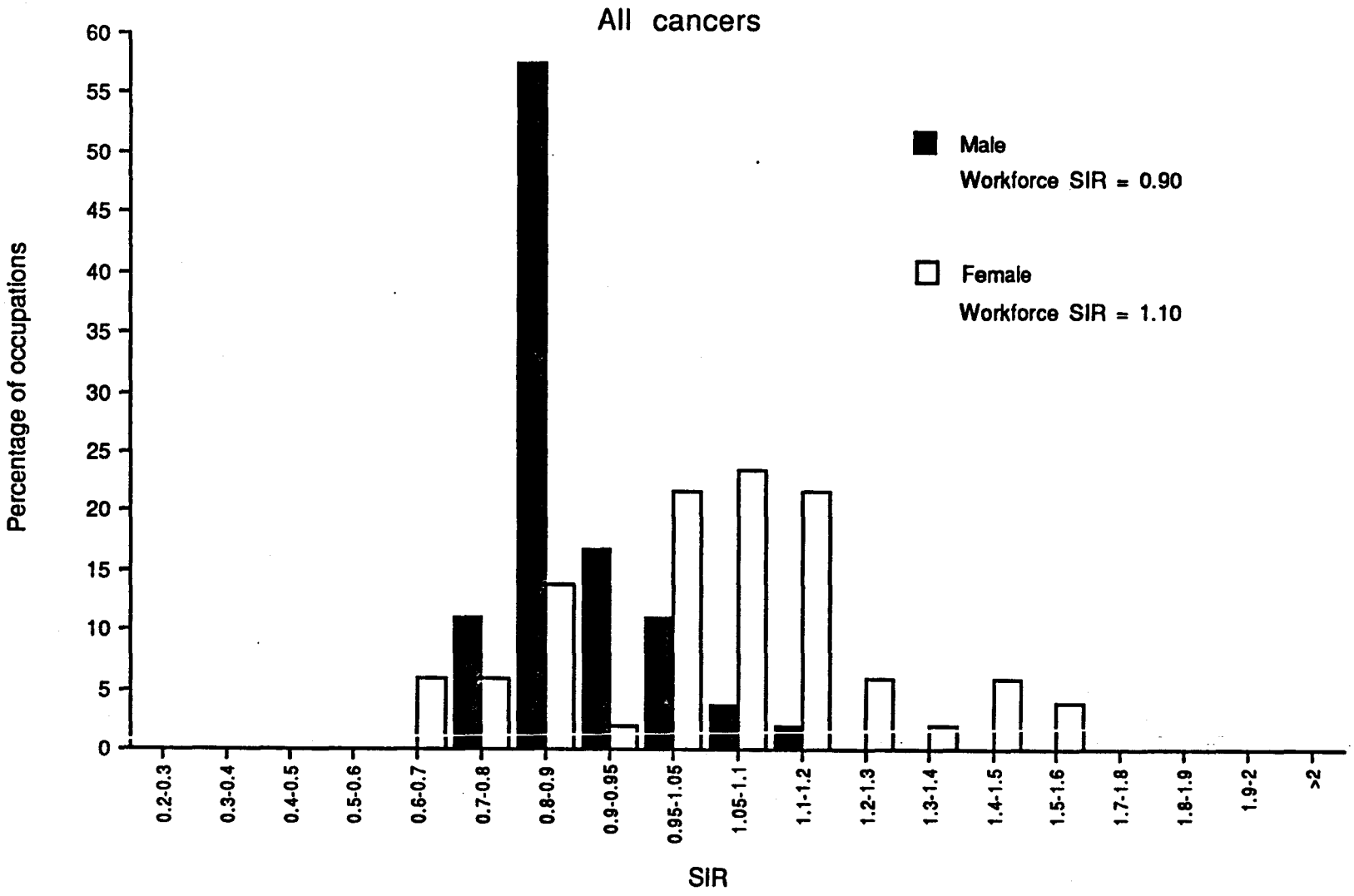


Figure 6.6
Distribution of the "average" worker standardized irradiation ratio for all cancers combined

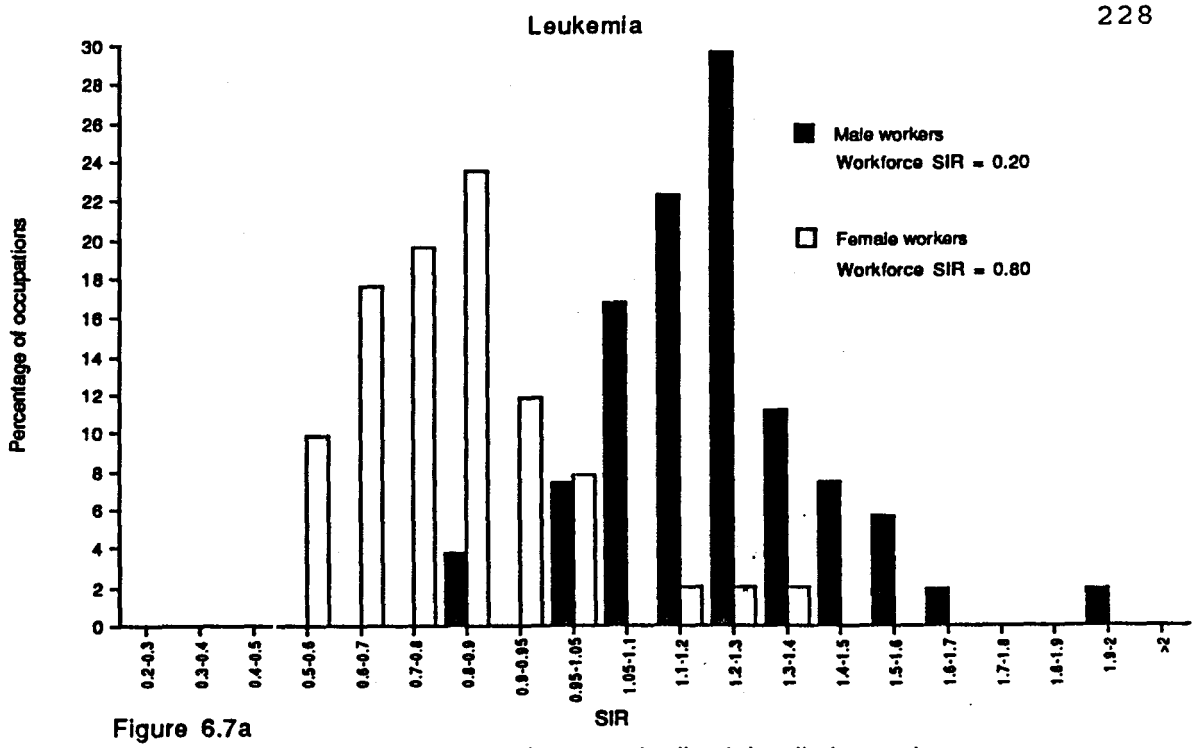


Figure 6.7a
Distribution of the "average" worker standardized irradiation ratio for radiation-induced leukemia

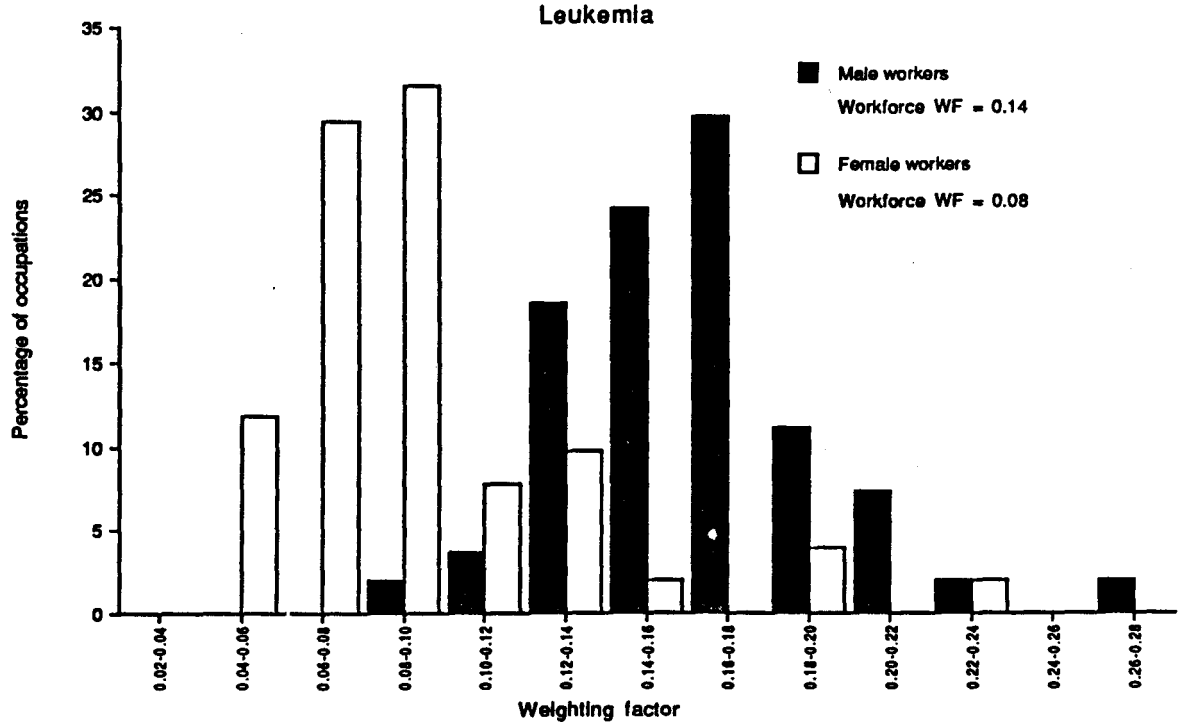


Figure 6.7b
Distribution of the "average" worker weighting factor for radiation-induced leukemia

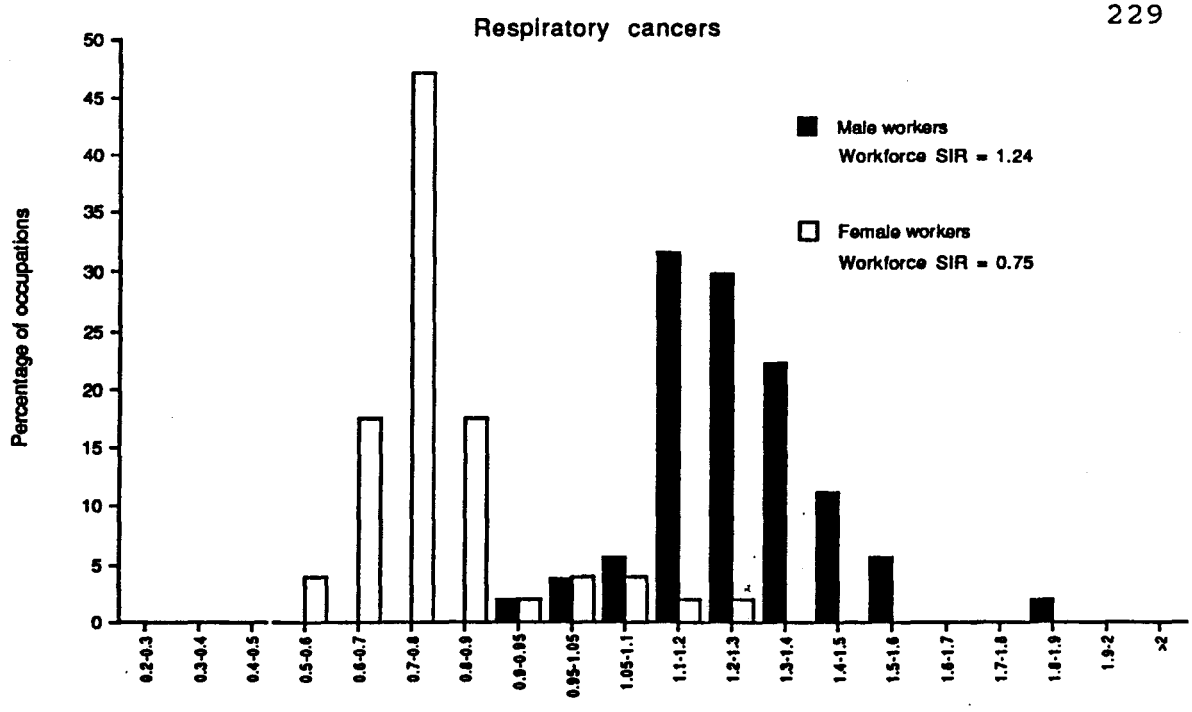


Figure 6.8a
Distribution of the "average" worker standardized irradiation ratio for radiation-induced respiratory cancers

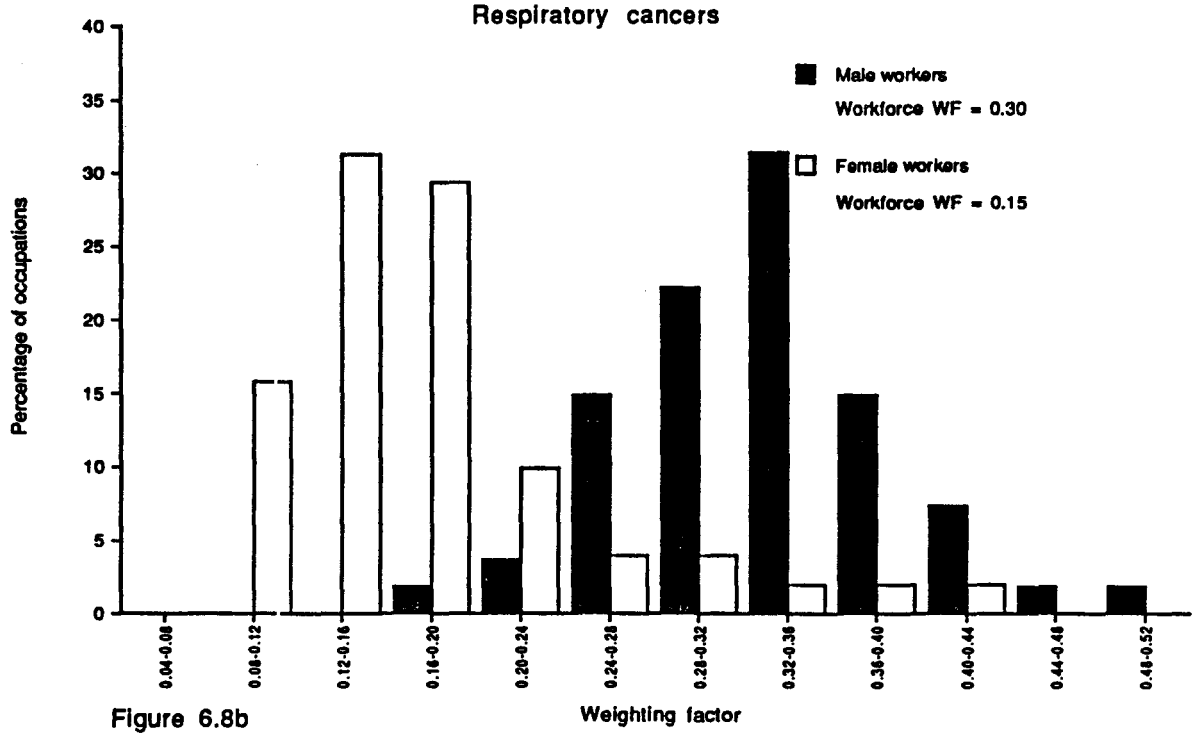


Figure 6.8b
Distribution of the "average" worker weighting factor for radiation-induced respiratory cancers

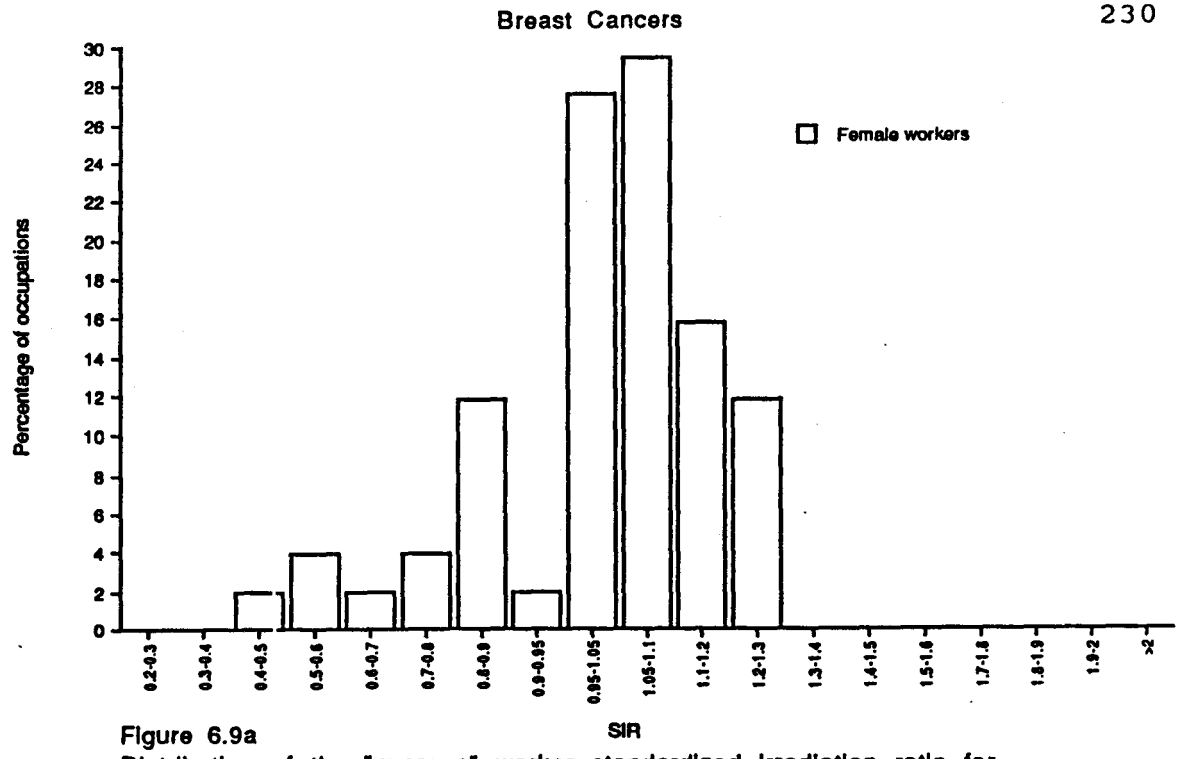


Figure 6.9a
Distribution of the "average" worker standardized irradiation ratio for radiation-induced breast cancer

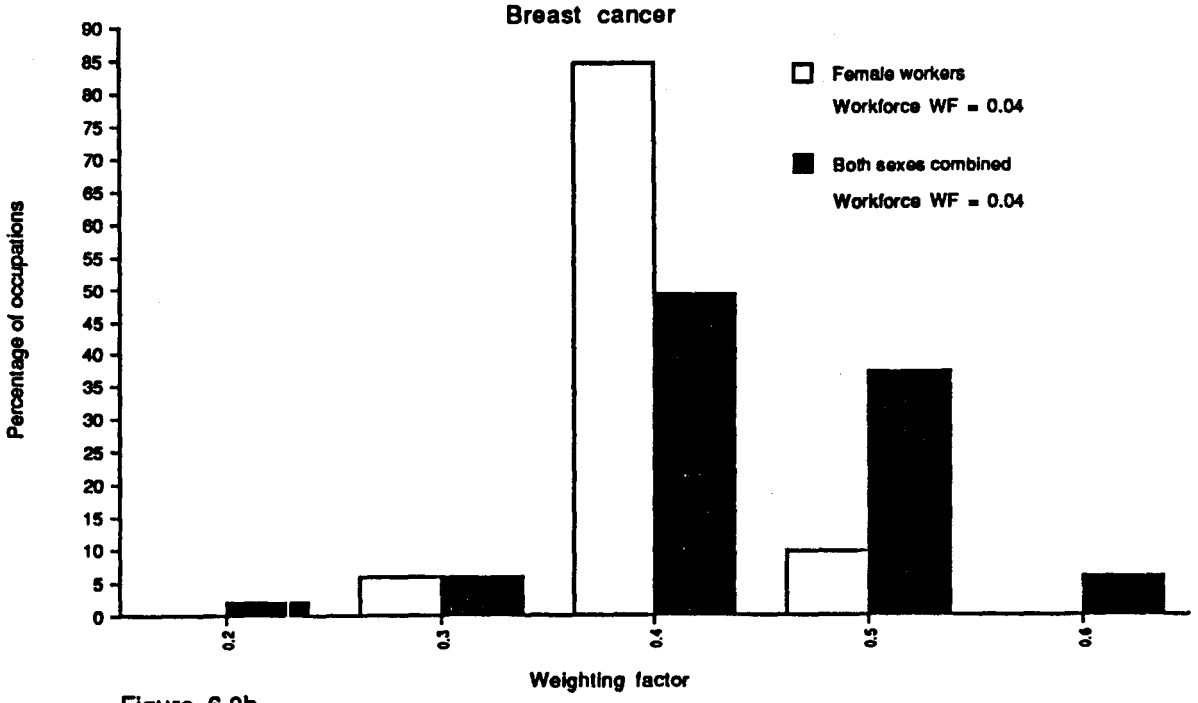


Figure 6.9b
Distribution of the "average" worker weighting factor for radiation-induced breast cancer

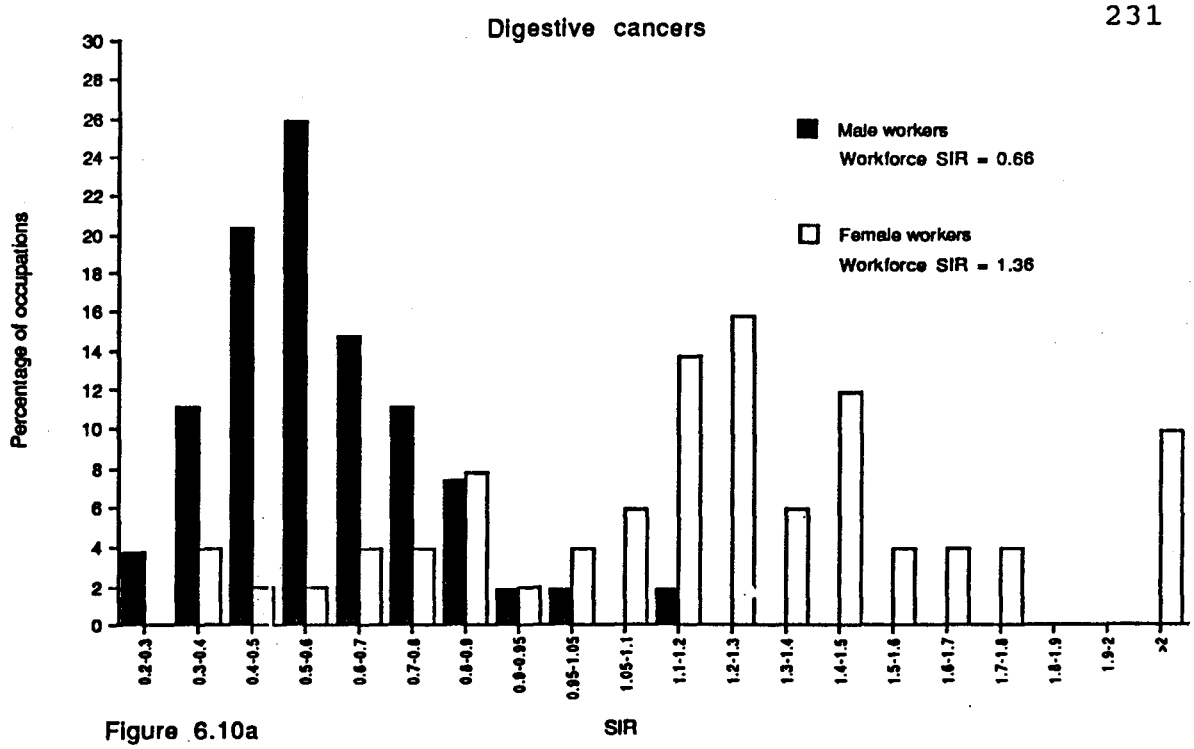


Figure 6.10a
Distribution of the "average" worker standardized irradiation ratio for radiation-induced digestive cancers

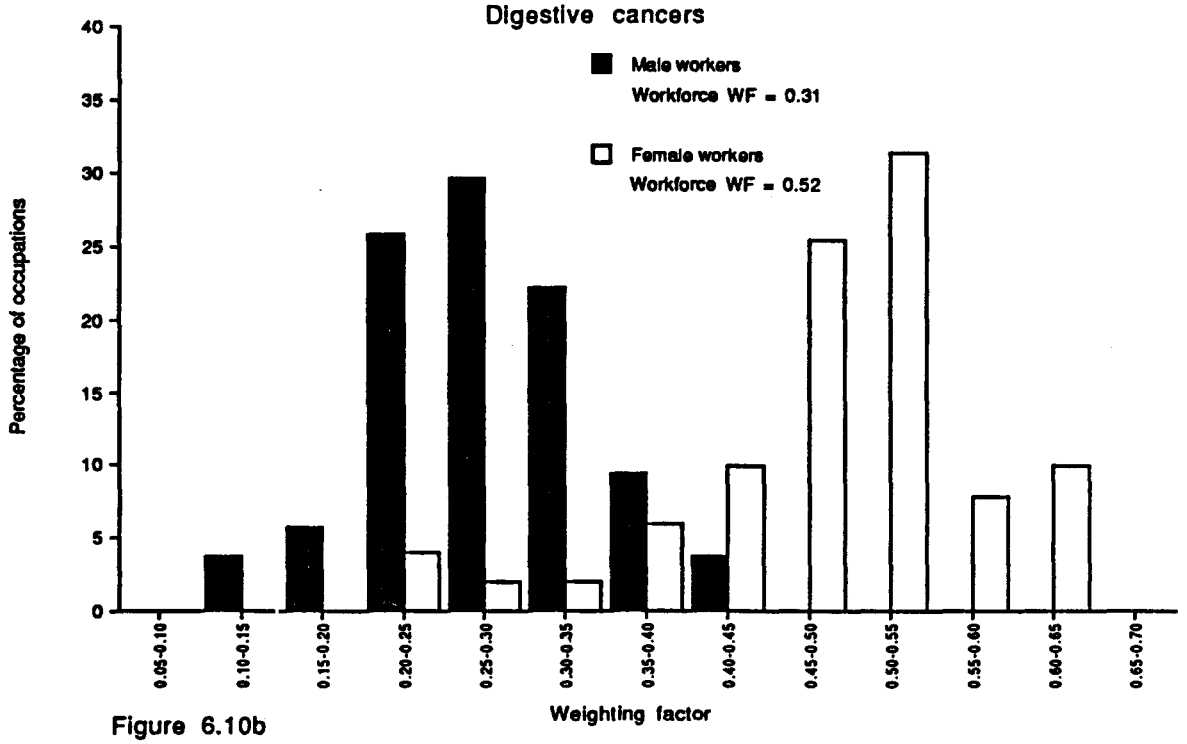


Figure 6.10b
Distribution of the "average" worker weighting factor for radiation-induced digestive cancers

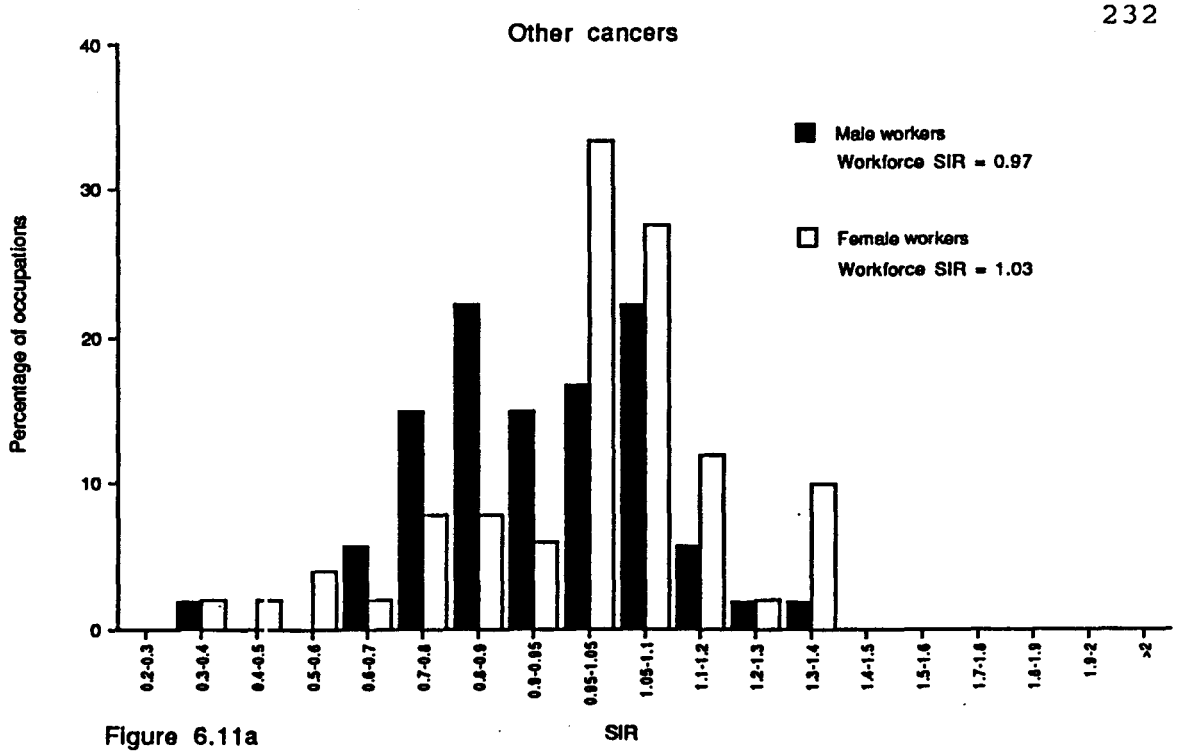


Figure 6.11a
Distribution of the "average" worker standardized irradiation ratio for radiation-induced other cancers

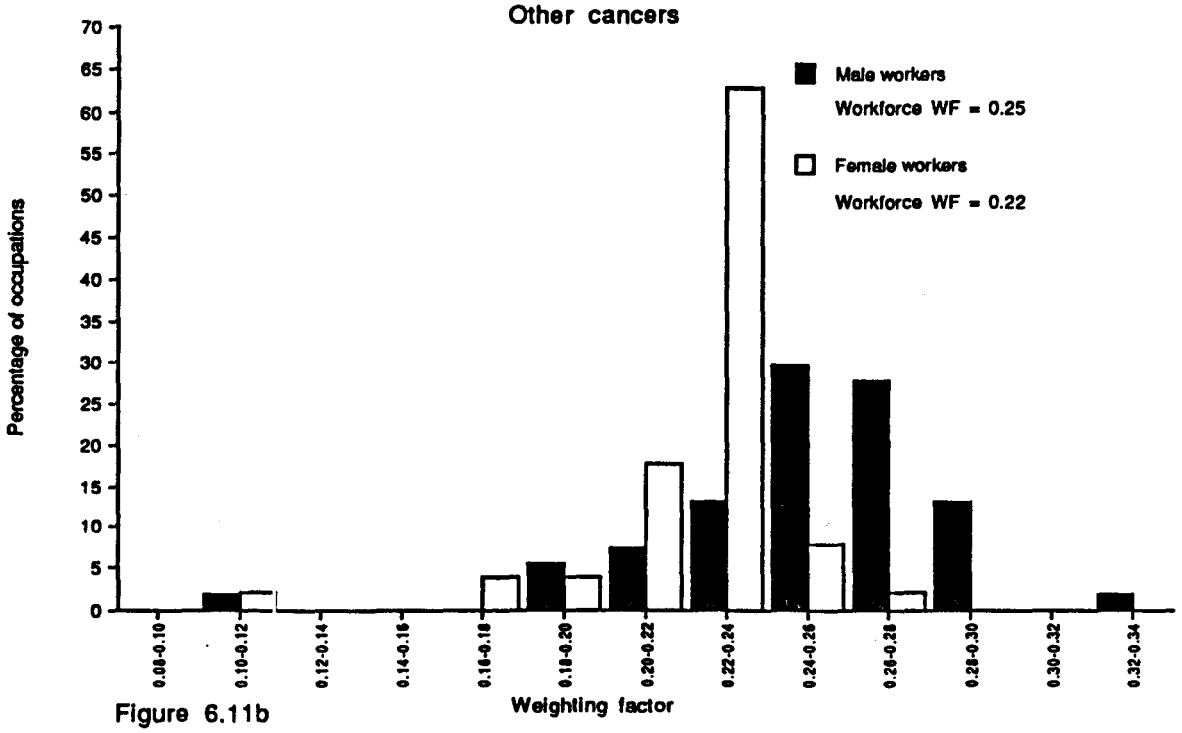


Figure 6.11b
Distribution of the "average" worker weighting factor for other radiation-induced cancers

## 7.0 Assessment of the Applicability of the ICRP-60 Risk Estimates to the Canadian Population

### 7.1 Introduction

The International Commission on Radiological Protection recently published new recommendations on radiation protection in ICRP publication 60 (ICRP 1991). The recommendations includes a new set of nominal fatal cancer risk estimates and tissue weighting factors ($w_T$s). The recommended fatal cancer risk factor for the average member of the "world" population for a single whole-body exposure of low-level radiation increased from the previously recommended value of 125 x $10^{-4}$ per Sv (ICRP 1977) to 5.0 x $10^{-4}$ per Sv for a general population and to 4.0 x $10^{-4}$ per Sv for a working population. The number of individual organ and tissue risk estimates and $w_T$s rose from six to thirteen with a different make up of remainder tissues. Public and occupation effective dose[40] limits were revised to reflect the higher estimated cancer risk. The recommended public limit decreased from 5 mSv in a year to 1 mSv in a year[41] and the occupation limit from 50 mSv per year to 20 mSv per year averaged over defined

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[40] ICRP 60 uses the term "effective dose" to denote the effective dose equivalent.

[41] In special circumstances, the Commission recommends a higher value of effective dose could be allowed in a single year, provided that the average over 5 years does not exceed 1 mSv per year

periods of 5 years[42].

As discussed in chapter 1.0, it is not clear whether the new ICRP risk factors and $w_T$s are directly applicable to the Canadian population. The assessment by the ICRP used the site-specific excess relative risk coefficients given by the RERF analysis of cancer mortality among atomic bomb survivors (Shimizu et al. 1988). The A-bomb survivor coefficients were used to project fatal cancer risks at 9 cancer sites: leukemia, lung, female breast, stomach, colon, oesophagus, ovary, bladder, and remainder. The Commission also recommended fatal risk estimates for cancers of the bone surface, liver, thyroid, and skin using results taken from other reports.

The analysis of cancer mortality data from the LSS cohort by the BEIR V Committee found that the excess number cancer deaths were not sufficient to produce stable risk models for colon, oesophagus, ovary, or bladder[43]. The Committee instead chose to group these cancers under the general categories of either "digestive system" or "other". Because the Commission chose not to use the BEIR V risk models in its assessment, it is reasonable to question the reliability of the lifetime risk estimates given in ICRP 60

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[42] With the further provision that the effective dose should not exceed 50 mSv in any single year.

[43] The number of observed and expected cancers for survivors with kerma doses greater than 0.5 Gy were 32 and 21 for the colon; 24 and 17 for the oesophagus; 13 and 8 for the ovary; 20 and 9.5 for the bladder (see table 2.3 in chapter 2.0)

for these four cancer sites. For cancers of the bone surface and liver, the ICRP low-LET radiation risk estimates were based on studies of internal alpha-irradiation of these tissues. And the fatal cancer risk estimates for the thyroid and skin were based on risk estimates of the increased incidence of these cancers.

This chapter examines whether the ICRP nominal fatal cancer risks, tissue weighting factors, and risk projections for prolonged exposure are suitable for the planning and regulation of radiation protection in Canada and whether the ICRP more detailed site-specific estimates are consistent with those made for the Canadian general and working population using the BEIR V five preferred relative risk models. For completeness, the suitability of the excess lifetime risk estimates given in the BEIR V report for risk assessment in Canada is examined as well.

## 7.2 ICRP 60 Lifetime Risk Projections

### 7.2.1 Fatal cancer risk factors

Site-specific lifetime fatal cancer risk projections were carried out for most cancers by the ICRP using the excess relative risk coefficients given in the most recent report of atomic bomb survivor cancer mortality by the Radiation Effects Research Foundation (Shimizu et al. 1988). The RERF report provided primary risk coefficients for the oesophagus,

stomach, colon, lung, breast, ovary, bladder, bone marrow, and all cancers except leukemia as a group. Coefficients were given by sex and age at time of bombing for 10 year age subsets for all separate sites except for the oesophagus, ovary, and bladder cancer. No coefficients were given for remainder cancers. For cancers of the oesophagus, ovary, and bladder, the excess risk coefficients given in the RERF report did not give sufficient age-specific information to perform reliable lifetime risk projections (Land and Sinclair 1991). The Commission chose to derive their own coefficients using organ-dose-specific data from RERF Table 4 and kerma-specific data from RERF Table 12 and RERF Appendix Tables 2-6, 2-19, 2-21, 2-26, and 2-27 given in Shimizu et al. (1988). Excess risk coefficients for remainder tissues were derived by subtracting the absolute risk coefficients of the individual non-leukemia sites from the coefficients for non-leukemia cancers analyzed as a group. The absolute risks were then converted to relative risk coefficients using Japanese national rates and a Japanese life-table for 1986-87 (Land and Sinclair 1991).

Projections were made by the ICRP for average members in five different national populations (Japan, United States, Puerto Rico, the United Kingdom, and China) within age groups of 0-90, 0-19, 20-64, and 65-90. Each age group was assumed to have life-table age distributions and equal numbers of males and females. Age constant relative risk projections models

were used for cancers except for leukemia, for which an age-constant absolute model was used. A minimum latency period of 2 years was assumed for leukemia and for other cancers 10 years, with a plateau period of 40 years taken for leukemia and infinity for nonleukemia cancers. Coefficients were transferred between populations using two transfer methods. The first was the straight multiplicative method (see section 3.7 and chapter 5.0) and the second, the additive type method used in the preparation of the U.S. National Institutes of Health 1985 probability of causation tables (NIH 1985). For cancers other than leukemia, the NIH method transfers the excess absolute risk observed in the first 40 years of A-bomb survivor follow-up to the population of interest. It is then converted to a relative risk using the baseline rates and life-table of the population of interest and projections beyond 40 years carried out using the age-constant relative risk model. The results of these projections are given in ICRP publication 60 (ICRP 1991) and Land and Sinclair (1991).

The Commission's assessment found that factors such as age, sex, transfer method, and population characteristics can cause substantial variation in site-specific projections for the average member in a population. It was concluded that since "some of these factors, such as the choice of model (method) for transfer between populations, involves uncertainties simply not resolvable at this time ... since the

total risk does not differ greatly between males and females ... and since other factors involved, broadly speaking, cause greater variations than those attributable to sex", averaging over age, sex, transfer methods, and national populations was as good a method as any other for deriving nominal risk factors for radiation protection purposes (Land and Sinclair 1991, ICRP 1991).

Risk estimates were also provided by the ICRP for cancers of the thyroid, bone, skin, and liver using estimates from other reports. The risk of fatal thyroid cancer was based on the incidence estimates presented in NCRP Report 80 (NCRP 1985), the risk of bone cancer mortality from incidence risk estimates risk for Ra-226 intake given in the BEIR IV report (NRC 1988), the risk of fatal skin cancer from the report of the ICRP Task Group on the Skin[44], and the risk of liver cancer was taken from estimates based on data for chronic alpha-irradiation by internally deposited Th-232 from Thorotrast studies in Germany, Portugal, Japan, and Denmark quoted in the BEIR V Report (NRC 1990). These additional fatal risk estimates were subtracted from the risk for remainder tissues estimated from the A-bomb survivor data.

The final nominal fatal cancer risk factors recommended in ICRP 60 are shown in table 7.1. Estimates include a dose and dose rate effectiveness factor of 2.

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[44] The Task Group's report was still in preparation and unavailable for review at the time this project report was being prepared.

7.2.2 Tissue Weighting Factors

An aggregative method was used by the ICRP to derive values of tissue weighting factors relating the relative contribution of specific organs to the total detriment resulting from uniform whole-body irradiation. In ICRP publication 26 (ICRP 1977), the Commission defined detriment using the probability of a radiation-induced health effect weighted by a factor representing the severity of the effect. The weighting factor was taken as 1 for the death of individuals and for severe hereditary effects. Smaller weighting factors for less severe effects were implied, but not specified. In ICRP 60, a similar definition is used but a broader approach is used to weight for the severity of an effect. The ICRP 60 severity weight takes into account that not all radiation-induced cancers are fatal and that there are differences in the expected years of life lost for radiation-induced cancers of different organs.

The first step the Commission took was to determine the total risk of both fatal and non-fatal radiation-induced cancers. The non-fatal risk was estimated indirectly using the estimated fatal cancer risk and a lethality fraction. The incidence rate for a radiation-induced cancer, $R_{INC}$, was approximated by dividing the fatal cancer risk, R, by the lethality fraction, k. This allowed the non-fatal risk, $R_{NF}$, to be written as

$$\begin{aligned} R_{NF} &= R_{INC} - R \\ &= \frac{R}{k} - R \\ &= \frac{R}{k}(1 - k) \end{aligned} \tag{48}$$

The total cancer risk, $R_{TOT}$, was taken to be the addition of the fatal and non-fatal cancer risks but with a greater weighting given to the fatal cancer risk in order to take into account that cancers with a higher rate of fatality are usually associated with a lower quality of life for those who survive. It was decided to weight the non-fatal cancer risk by the lethality fraction. This resulted in the total weighted cancer risk to be given as

$$\begin{aligned} R_{TOT} &= R + k\, R_{NF} \\ &= R + k\left(\frac{R}{k}(1 - k)\right) \\ &= R\,(2-k) \end{aligned} \tag{49}$$

Allowance for differences in expected years of life lost for different cancers was made by weighting the above expression by the relative expected life lost per fatal cancer. The relative life lost per fatal cancer being the ratio of the average life lost per fatal cancer for a cancer site, Y, to the average life lost per fatal cancer for all cancers combined, $Y_T$. This gave a final expression for the potential detriment caused by radiation-induced cancer i of the form

$$D_i = R_i \frac{Y_i}{Y_T} (2-k_i) \qquad (50)$$

where

$D_i$ is the detriment due to radiation-induced cancer at site i;

$R_i$ is the risk of fatal cancer at site i;

$Y_i$ is the mean years of life lost if radiation-induced death occurs at cancer site i;

$Y_T$ is the mean years of life lost if radiation-induced death occurs for all cancer sites combined; and

$k_i$ is the lethality fraction for cancer i

The product $Y/Y_{TOT}$ (2-k) may be thought as representing the weighting factor for severity. Unlike the severity weight in ICRP 26, the new weight can possibly take on any value.

The Commission based values of lethality fractions on data from the National Cancer Institute of the United States (U.S. DHHS, 1989)[45] for lethality in five years following diagnosis (data from years 1980-85) and 20 years following diagnosis (data from years 1950-70). The five year lethality rates were considered too low for full expression of lethality and the 20 year lethality fractions too high for present day standards of treatment. The Commission therefore chose to estimate lethality fractions using the average of these two

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[45] Number were derived from tables and graphical data of that report by F.A. Mettler and W.K. Sinclair

sets and judgement to reflect improved treatment of some types of cancers. The U.S. 5 year, 20 year, and ICRP 60 recommended lethality fractions are shown in table 7.2.

Table 7.3 summarizes the various components of the detriment, the total detriment, and relative contribution of organs to the total detriment computed in the Commission's assessment.

7.2.3 Continuous annual exposures

Projections were also carried out by the Commission examining the potential detriment following prolonged exposures from birth over a lifetime and from occupational exposure from age 18 to 65. Four different attributes were computed to describe the risk. These were:

(a) lifetime risk of fatal cancer (R);

(b) mean years lost per fatal cancer (Y);

(c) loss of life expectancy (LLE); and

(d) the average age of projected radiation-induced death.

Chapter 4.0 defines and describes these quantities.

Lifetime risk projection were made by the Commission for a life-table population with baseline cancer rates of the 1986 Japanese population and the mortality rates from all causes of the 1986 Swedish population. An age-constant relative risk model was used to project the lifetime cancer risks for all cancers as a group using the primary risk coefficients from the A-bomb survivor study for leukemia and

for all cancers other than leukemia as a group[46].

Table 7.4 summarizes the results of the ICRP's projections for continuous lifetime low dose exposures (ages 0-90) of 1, 2, 3, and 5 mSv per year and for continuous annual occupational exposures (ages 18 to 65) of 10, 20, 30, and 50 mSv.

## 7.3 Canadian Lifetime Risk Projections

### 7.3.1 Fatal cancer risk factors

Lifetime risk projections for the Canadian population are performed using the excess relative risk coefficients of the BEIR V preferred modified risk models for leukemia, cancers of the respiratory tract, female breast, digestive system, and other remaining organs and tissues. The models, and projection methodology, are described in chapter 4.0. Cancer-specific lifetime fatal risks per unit dose are projected for the average member in the 1982 and 1988 Canadian population for exposure ages between 0 and 85. Risk coefficients are transferred to the two Canadian populations using the additive and multiplicative transfer method and then averaged over both populations. Projections are for a single whole-body exposure of 0.1 Sv. A dose and dose rate

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[46] Coefficients given for males and females and grouped in exposure ages of 0-9, 10-19, 20-29, 30-39, and above 40

effectiveness of 2 is assumed for nonleukemia cancers[47]. Results are given in terms of risk per Sv.

Table 7.5 gives the lifetime risks of fatal cancer per unit dose by cancer site for the average member in the 1982 and 1988 Canadian general (ages 0-85) and working (18-65) life-table populations. In addition the projected mean years of life lost per fatal cancer , loss of life expectancy, and mean age at radiation-induced death are also presented.

7.3.2 Tissue weighting factors

Tissue weighting factors are computed in the same manner as done by the ICRP except the cancer groupings are those given by the BEIR V risk models. The risk of genetic effects for all generations following gonadal dose is taken to be the ICRP 60 recommended value of $100 \times 10^{-4}$ per Sv.

There is no apparent reason as to why the lethality fractions recommended in ICRP 60 should not be used to compute the "radiation" detriment in Canada as well. Table 7.2 shows five years adult lethality fractions in Saskatchewan for the years 1970-86 by sex and selected cancer sites (NCIC 1990). Lethality fractions are similar between sexes[48] and are consistent with the U.S. 5 year lethality fractions. The ICRP 60 recommended lethality fractions have therefore been used

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[47] The dose response for the BEIR V model for radiation-induced leukemia has an inherent DDREF of 2 at 0.1 Sv.

[48] Except for cancers which are unique to a particular sex, e.g., cancers of the breast, cervix, uterus, ovary, and prostate.

here. The lethality fraction for the BEIR V grouping of "digestive" cancers was computed as the weighted average of the ICRP fractions for cancers of the colon, liver, oesophagus, and stomach, where weighting is by the ICRP site-specific fatal cancer risk factors. A similar averaging was performed for the lethality fraction for the BEIR V grouping of "other" cancers using the ICRP fractions for cancers of the bladder, bone surface, ovary, skin, thyroid, and remaining sites.

The projected components of the detriment and the relative contribution of BEIR V cancer groupings to the total detriment for the Canadian population is given in table 7.6 for both the case when the additive and multiplicative methods are used to transfer risk coefficients.

### 7.3.3 Continuous annual exposures

Projections for prolonged exposures were made for each of the BEIR V groupings and then summed to obtain the risk from all cancers as a group. Table 7.7 shows the results of projections of the same annual doses used in the ICRP assessment. Projections have been averaged over sex, 1982 and 1988 Canadian populations, and transfer methods.

## 7.4 Comparison of ICRP 60 and Canada Lifetime Risk Projections

In order to compare ICRP and Canada nominal risk factors and tissue weighting factors, the ICRP site-specific

values have been combined to give the same cancer groupings used in the BEIR V models. The corresponding groupings are:

| BEIR V | ICRP 60 |
|---|---|
| Leukemia | •Bone marrow |
| Respiratory | •Lung |
| Breast | •Breast |
| Digestive | •Stomach, colon, liver, and oesophagus |
| Other | •Bladder, bone surface, ovary, skin, thyroid, and remainder |

Because of the uncertainty in the choice of method for transferring excess risk coefficients between populations, the results for Canada are presented as the range of values given by the two plausible methods.

### 7.4.1 Fatal cancer risk factors

Table 7.8 shows the site-specific nominal fatal cancer risk factors derived in ICRP 60 and the range of Canadian site-specific values projected by the additive and multiplicative transfer methods. The nominal risk for all cancers as a group projected for the Canadian general population ranged from 495 to 575 x $10^{-4}$ per Sv, depending on the transfer method. This is in fair agreement with the ICRP 60 recommended value of 500 x $10^{-4}$ per Sv. For a working population, the Canadian risk factor ranges from 410 to 505 x $10^{-4}$ per Sv, the ICRP recommended value is 400 x $10^{-4}$ per Sv. For specific cancers groups except leukemia, the ICRP nominal

risks for a general population fall roughly within the middle of the range predicted for Canada. For leukemia, the ICRP estimate is slightly lower than that for Canada, a value of 50 x $10^{-4}$ per Sv compared to the Canadian range of 70 to 80 x $10^{-4}$ per Sv. However, in view of the large statistical error in modelling excess leukemia mortality, the estimates are in fair agreement (lower and upper 90% confidence intervals for the Canadian leukemia estimates are approximately 25 and 150 x $10^{-4}$ per Sv)[49].

7.4.2 Tissue weighting factors

Table 7.7 presents the ICRP 60 and Canadian tissue weighting factors. As was the case for the nominal risk factors, there is generally good agreement between the Canadian and ICRP derived values. Of particular interest is the consistency of the $w_T$s for the digestive system and other remaining sites. For the digestive system the Canadian factor ranges from 0.39 and 0.20, ICRP's value is 0.35. For "other" sites the "Canadian" $w_T$ ranges from 0.12 to 0.25 compared to the ICRP value of 0.16. This gives some confidence that ICRP more detailed breakdown of these cancer groups individual sites provides reasonable weighting factors for these tissues.

7.4.3 Continuous annual exposures

Tables 7.4 and 7.7 summarise the various risk

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[49] 90% CI calculated indirectly using the ratio of the upper and low 90% CI given to the ELR point estimates given in the BEIR V Report (see section 4.7).

attributes resulting from various levels of annual exposures from birth over a lifetime and exposures over a working lifetime projected by the ICRP and for Canada, respectively. Figure 7.1 plots the distribution of the probability per year of radiation-induced cancer mortality with attained age averaged over males and females projected for a 1 mSv per year exposure from birth over a lifetime (the ICRP recommended public effective dose limit) and for a 20 mSv per year exposure from ages 18 to 65 (the ICRP recommended occupation effective dose limit).

The total lifetime probability of fatal cancer for a 1 mSv per year lifetime exposure is the same for both ICRP and Canadian projections, a predicted 0.4% lifetime probability. The Canadian projections, however, predict that cancers will be expressed at slightly earlier ages. The "Canadian" average age at radiation-induced death is 71 compared to the ICRP value of 79 and the "Canadian" average years lost per fatal cancer is 14.6 compared to the ICRP 13.4 years. For a 20 mSv per year exposure over a working lifetime, the Canadian projected lifetime probability of fatal cancer is 3.95%, slightly higher than the ICRP 60 value of 3.6%. The distribution of risk with attained age (figure 7.1) differs somewhat as well. The risk per year rises more quickly for attained ages between 55 and 75 for Canadian projections. The average Canadian age at radiation-induced cancer death is 73

while ICRP predicts an age of 79. The Canadian average years of life lost per fatal cancer is 13.5 years and the ICRP value 12.7 years.

## 7.5 Suitability of excess lifetime risk estimates in the BEIR V Report

Since Canadian lifetime risk projections are performed using the preferred relative risk models developed by the BEIR V Committee, one might expect the excess lifetime risk projections given in the BEIR V report could be suitable for predicting the potential radiogenic risks in the Canadian population. However, this is not necessarily so. There are a number of issues associated with the Committee's approach to lifetime risk projections that make it questionable whether the Committee's risk estimates should be used in Canada.

First, the BEIR V committee carried out lifetime risk projections using 1980 U.S. national baseline cancer rates and a U.S. life-table population. Baseline rates for cancers other than leukemia are 25 to 35% lower in the U.S. than Canada while in Canada, the average life expectancy is 2 to 4 years higher than in the U.S. (see tables 5.1 and 5.2 in chapter 5.0). Table 7.8 compares the excess lifetime cancer risk projected for a 1988 Canadian population and that by the BEIR V Committee following a single whole-body exposure of 0.1 Sv to 100,000 males and females at exposure ages between 1 and

85. As a result of the lower baseline rates and smaller life expectancy in the U.S., U.S. excess lifetime risks, except for leukemia, are about 20% lower than similar projections for Canada. The ELR for radiation-induced leukemia, however, is 30% higher for the U.S., a consequence of the higher baseline rates. It is unclear why leukemia baseline rates would be higher in the U.S. while other cancer rates are lower than rates in Canada. It may be the case that the Committee included the baseline rate for chronic lymphatic leukemia.

A second difficulty with BEIR V risk estimates is that no dose and dose rate effectiveness factor has been applied to excess lifetime risks for non-leukemia cancers. A linear quadratic dose response was used for radiation-induced leukemia which has an inherent dose and dose rate effectiveness factor (DDREF) of 2 at a dose of 0.1 Sv. However, a linear dose response was used for non-leukemia cancers with no DDREF applied. As discussed in section 3.5, radiobiological theory and animal studies strongly indicate the ability of low-LET radiation to induce cancer is reduced at low doses and low dose rates, by a factor ranging from 2 to 10.

A third problem with the Committee's projections is that only the multiplicative method is used to transfer excess risk coefficients to the U.S. population. As discussed in section 3.7, there is no general agreement on which, if

either, of the two plausible transfer models, additive or multiplicative, is more appropriate. Chapter 5.0 showed that the choice can significantly effect the risk projected at specific cancer sites. BEIR V committee chose to use only the multiplicative method on the basis of its analyses of the incidence of breast cancer and thyroid cancer among different population cohorts. However, the interpretation of the results in these cohort studies have been inconsistent, changing as follow-up has been extended and new statistical methods developed. The BEIR V report even acknowledges that "it is not clear whether cancers risks derived in one population are applicable to the another, and if so, whether relative or absolute risks should be used..." and "... it may be that neither absolute nor relative risks can be extrapolated with assurance" (page 218, NRC 1990). Therefore, the Committee's decision to use only the multiplicative transfer method appears not to be clearly justified.

And finally, the Committee choice to use the excess lifetime risk (ELR) as the measure of the radiation-induced cancer risk. As discussed in section 4.5.2 and chapter 5.0, the ELR is difficult to interpret and has several undesirable characteristics because a cancer risk caused by radiation is not considered an excess if it would have been expressed later in life anyhow, even if there were no exposure. Projections using the ELR are on average a factor of 1.2 times lower than

equivalent projections that use the lifetime risk of fatal cancer. The lifetime risk of fatal cancer seems to be the more preferable cancer measure for radiation protection purposes.

## 7.6 Discussion and Conclusions

Tables 7.8, 7.9, and figure 7.1 give some assurance that the ICRP 60 recommended nominal fatal cancer risk factors, tissue weighting factors, and lifetime risk projections for prolonged exposure are reasonable and suitable for the planning and regulation of radiation protection in Canada.

The agreement between the Canadian projections for cancers of the digestive system and other remaining cancers and the ICRP projections for the same groupings is encouraging. It indicates that the ICRP breakdown of individual organ sites in these cancer groupings is reasonable and that the individual site estimates are probably the best available at this time. The agreement between the Canadian and ICRP projection risks for respiratory and female breast cancer is interesting since the BEIR V risk models have the relative risk decreasing with time-since-exposure while ICRP used an age-constant relative risk model. This might suggest that for the present risk models, projections that are averaged over all ages and both sexes in a population, the assumption of a constant relative risk may not be too unreasonable.

Table 7.1
Fatal cancer risk factors and tissue weighting factors derived by the ICRP in 1977 and 1990

| Site | ICRP 1977 | | ICRP 1990 | |
|---|---|---|---|---|
| | Fatal cancer (10E-04 per Sv) | Tissue weighting factor | Fatal cancer (10E-04 per Sv) | Tissue weighting factor |
| Bladder | | | 30 | 0.05 |
| Bone marrow | 20 | 0.12 | 50 | 0.12 |
| Bone surface | 5 | 0.03 | 5 | 0.01 |
| Breast | 25 | 0.15 | 20 | 0.05 |
| Colon | | | 85 | 0.12 |
| Liver | 20 | 0.12 | 15 | 0.05 |
| Lung | | | 85 | 0.12 |
| Oesophagus | | | 30 | 0.05 |
| Ovary | | | 10 | (c) |
| Skin | | | 2 | 0.01 |
| Stomach | | | 110 | 0.12 |
| Thyroid | 5 | 0.03 | 8 | 0.05 |
| Remainder | 50 | 0.30 | 50 | 0.05 |
| All sites | 125 | -- | 500 | -- |
| Gonads | 40 (a) | 0.25 | 100 (b) | 0.2 |

(a) Genetic risk: first two generations only
(b) Genetic risk: all generations
(c) weighting factor included in the gonads

Table 7.2
Lethality fractions for cancer in U.S. and Canadian adults and those recommended in ICRP 60

| Cancer | Canada (1970-86) (a) 5 year lethality Male | Female | U.S. (b) 5 year lethality (1980-85) | 20 year lethality (1950-70) | ICRP 60 recommended lethality fraction k |
|---|---|---|---|---|---|
| Bladder | 0.20 | 0.22 | 0.22 | 0.58 | 0.50 |
| Bone | – | – | – | 0.72 | 0.70 |
| Brain | 0.75 | 0.75 | 0.75 | 0.84 | 0.80 |
| Breast | – | 0.27 | 0.24 | 0.62 | 0.50 |
| Cervix | – | 0.34 | 0.33 | 0.50 | 0.45 |
| Colon | 0.48 | 0.48 | 0.45 | 0.62 | 0.55 |
| Kidney | 0.53 | 0.50 | 0.48 | 0.78 | 0.65 |
| Leukemia (acute) | – | – | 0.98 | 0.99 | 0.99 |
| Liver | – | – | 0.95 | 0.98 | 0.95 |
| Lung | 0.89 | 0.83 | 0.87 | 0.96 | 0.95 |
| Oesophagus | – | – | 0.92 | 0.97 | 0.95 |
| Ovary | – | 0.60 | 0.62 | 0.74 | 0.70 |
| Pancreas | 0.96 | 0.97 | 0.97 | 0.99 | 0.99 |
| Prostate | 0.37 | – | 0.26 | 0.84 | 0.55 |
| Skin | – | – | – | – | 0.002 |
| Stomach | 0.83 | 0.86 | 0.85 | 0.90 | 0.90 |
| Thyroid | – | – | 0.06 | 0.15 | 0.10 |
| Uterus | – | 0.16 | 0.17 | 0.35 | 0.30 |

(a) Based on data in table 10, "Five year survival rates for selected cancer sites in Saskatchewan" from NCIC (1991)
(b) Numbers derived from tables and graphical data of U.S. DHHS (1989) by F.A. Mettler and W.K. Sinclair

Table 7.3
ICRP derived detriment and tissue weighting factors associated with low-dose radiation exposure (a)

| Cancer | Lifetime R (10E-04 per Sv) | Genetic effects (10E-04 per Sv) | Y (yrs/death) | Y/Y(total) | k | 2 - k | Detriment R Y/Y(total) (2-k) (10E-04 per Sv) | Relative Contribution |
|---|---|---|---|---|---|---|---|---|
| Bladder | 30 | | 9.8 | 0.65 | 0.50 | 1.50 | 29.4 | 0.040 |
| Bone marrow | 50 | | 30.9 | 2.06 | 0.99 | 1.01 | 104.0 | 0.143 |
| Bone surface | 5 | | 15.7 | 1.00 | 0.70 | 1.30 | 6.5 | 0.009 |
| Breast | 20 | | 18.2 | 1.21 | 0.50 | 1.50 | 36.4 | 0.050 |
| Colon | 85 | | 12.5 | 0.83 | 0.55 | 1.45 | 102.7 | 0.141 |
| Liver | 15 | | 15.7 | 1.00 | 0.95 | 1.05 | 15.8 | 0.022 |
| Lung | 85 | | 13.5 | 0.90 | 0.95 | 1.05 | 80.3 | 0.111 |
| Oesophagus | 30 | | 11.5 | 0.77 | 0.95 | 1.05 | 24.2 | 0.034 |
| Ovary (b) | 10 | | 16.8 | 1.12 | 0.70 | 1.30 | 14.6 | 0.020 |
| Skin | 2 | | 15.7 | 1.00 | 0.00 | 2.00 | 4.0 | 0.006 |
| Stomach | 110 | | 12.5 | 0.83 | 0.90 | 1.10 | 100.0 | 0.139 |
| Thyroid | 8 | | 15.7 | 1.00 | 0.10 | 1.90 | 15.2 | 0.021 |
| Remainder | 50 | | 13.6 | 0.91 | 0.71 | 1.29 | 58.9 | 0.081 |
| Gonads (b) | | 100 | 20.0 | 1.33 | -- | -- | 133.3 | 0.183 |
| Total | 500 | | 15.0 | | | | 725.3 | 1.000 |

(a) Taken from table B-20, ICRP 1991
(b) Gonads (including the ovary)
R: Lifetime risk of fatal cancer
Y: Years of life lost if radiation-induced cancer death occurs
k: Lethality fraction

Table 7.4
ICRP 60 derived risk attributes for all cancers combined associated with continuous annual exposures from birth over lifetime and from age 18 to 65 (a)

Exposure from birth over lifetime

| Annual Dose (mSv) | R (10 E-02) | Y (yrs/death) | LLE (yrs/1000) | A (yrs) |
|---|---|---|---|---|
| 1 | 0.40 | 13.4 | 0.05 | 79 |
| 2 | 0.80 | 13.4 | 0.11 | 79 |
| 3 | 1.12 | 13.4 | 0.16 | 79 |
| 4 | -- | -- | -- | -- |
| 5 | 1.99 | 13.4 | 0.27 | 79 |

Exposure from age 18 to 65

| Annual Dose (mSv) | R (10 E-02) | Y (yrs/death) | LLE (yrs) | A (yrs) |
|---|---|---|---|---|
| 10 | 1.80 | 12.7 | 0.23 | 78 |
| 20 | 3.60 | 12.7 | 0.46 | 78 |
| 30 | 5.30 | 12.8 | 0.68 | 77 |
| 40 | -- | -- | -- | -- |
| 50 | 8.55 | 13.0 | 1.11 | 77 |

(a) Based on table 5, C-5, and C-6 from ICRP 1991
R: lifetime risk of fatal cancer;
Y: mean years lost if radiation induced cancer occurs
LLE: loss of life expectancy;
A: average age at radiation-induced death

Table 7.5
Canadian derived fatal cancer risk factors and other attributes associated with a single whole-body low-dose exposure (a)

Average member in the general population (ages 0-85)

| Cancer | R (10 E-04 per Sv) | Y (yrs/death) | LLE (yrs per Sv) | A |
|---|---|---|---|---|
| Leukemia | 75 | 26.7 | 0.20 | 57 |
| Respiratory | 90 | 13.3 | 0.12 | 72 |
| Breast | 25 | 19.2 | 0.06 | 68 |
| Digestive | 220 | 12.3 | 0.27 | 77 |
| Other | 130 | 12.9 | 0.16 | 77 |
| All cancers | 540 | 13.8 | 0.76 | 73 |

Average member in the working population (ages 18-65)

| Cancer | R (10 E-04 per Sv) | Y (yrs/death) | LLE (yrs per Sv) | A |
|---|---|---|---|---|
| Leukemia | 65 | 18.4 | 0.12 | 62 |
| Respiratory | 115 | 13.9 | 0.16 | 70 |
| Breast | 15 | 19.2 | 0.05 | 68 |
| Digestive | 170 | 11.8 | 0.20 | 76 |
| Other | 90 | 12.2 | 0.11 | 76 |
| All cancers | 455 | 12.7 | 0.59 | 72 |

(a) Risks average over sex, age, and transfer method in a 1982 and 1988 Canadian life-table population
R: lifetime risk of fatal cancer;
Y: mean years lost if radiation induced cancer occurs
LLE: loss of life expectancy;
A: average age at radiation-induced death

Table 7.6
Canadian derived detriment and tissue weighting factors associated with low-dose radiation exposure (a)

Additive transfer method

| Cancer | Lifetime R (10E-04 per Sv) | Genetic effects (10E-04 per Sv) | Y (yrs/death) | Y/Y(total) | k | 2 - k | Detriment R Y/Y(total) (2-k) (10E-04 per Sv) | Weighting Factor |
|---|---|---|---|---|---|---|---|---|
| Leukemia | 70 | – | 32.1 | 2.14 | 0.99 | 1.01 | 151.3 | 0.209 |
| Respiratory | 65 | – | 11.9 | 0.79 | 0.95 | 1.05 | 54.1 | 0.075 |
| Breast | 10 | – | 20.3 | 1.35 | 0.50 | 1.50 | 20.3 | 0.028 |
| Digestive | 275 | – | 12.7 | 0.85 | 0.79 (c) | 1.21 | 281.7 | 0.388 |
| Other | 75 | – | 12.9 | 0.86 | 0.68 (d) | 1.32 | 85.1 | 0.117 |
| Gonads | | 100 (b) | 20.0 | 1.33 | - | - | 133.0 | 0.183 |
| Total | 495 | | 15.0 | | | | 725.6 | 1.000 |

Multiplicative transfer method

| Cancer | Lifetime R (10E-04 per Sv) | Genetic effects (10E-04 per Sv) | Y (yrs/death) | Y/Y(total) | k | 2 - k | Detriment R Y/Y(total) (2-k) (10E-04 per Sv) | Weighting Factor |
|---|---|---|---|---|---|---|---|---|
| Leukemia | 80 | – | 24.1 | 1.79 | 0.99 | 1.01 | 144.2 | 0.165 |
| Respiratory | 115 | – | 14.3 | 1.06 | 0.95 | 1.05 | 127.9 | 0.146 |
| Breast | 35 | – | 18.0 | 1.33 | 0.50 | 1.50 | 70.0 | 0.080 |
| Digestive | 165 | – | 11.5 | 0.85 | 0.79 (c) | 1.21 | 170.1 | 0.194 |
| Other | 180 | – | 12.2 | 0.90 | 0.68 (d) | 1.32 | 214.7 | 0.245 |
| Gonads | | 100 (b) | 20.0 | 1.48 | - | - | 148.0 | 0.169 |
| Total | 575 | | 13.5 | | | | 874.9 | 1.000 |

(a) Averaged over both sexes (ages 1-85) in a 1982 and 1988 Canadian stationary life-table population
(b) Genetic risk taken from table B-19, ICRP 1991
(c) The lethality fraction is the weighted average over the ICRP 60 fractions lethality fractions for cancers of the colon, liver, oesophagus and stomach, where weighting is by the ICRP 60 fatal cancer risk factors
(d) The lethality fraction is the weighted average over the lethality fractions for cancers of the bladder, bone surface, ovary, skin, thyroid, and remaining sites, where weighting is by the ICRP 60 fatal cancer risk factors
R: Lifetime risk of fatal cancer
Y: Years of life lost if radiation-induced cancer death occurs
k: Lethality fraction

Table 7.7
Canadian derived risk attributes for all cancers combined associated with continuous annual exposures from birth over lifetime and from age 18 to 65 (a)

Exposure from birth over lifetime

| Annual Dose (mSv) | R (10 E-02) | Y (yrs/death) | LLE (yrs) | A (yrs) |
|---|---|---|---|---|
| 1 | 0.40 | 14.6 | 0.06 | 71 |
| 2 | 0.75 | 14.6 | 0.11 | 71 |
| 3 | 1.15 | 14.6 | 0.17 | 71 |
| 4 | 1.55 | 14.6 | 0.23 | 71 |
| 5 | 1.90 | 14.6 | 0.28 | 71 |

Exposure from age 18 to 65

| Annual Dose (mSv) | R (10 E-02) | Y (yrs/death) | LLE (yrs) | A (yrs) |
|---|---|---|---|---|
| 10 | 2.00 | 13.5 | 0.27 | 73 |
| 20 | 3.95 | 13.5 | 0.54 | 73 |
| 30 | 5.90 | 13.7 | 0.81 | 73 |
| 40 | 7.75 | 13.9 | 1.07 | 72 |
| 50 | 9.55 | 13.9 | 1.33 | 72 |

(a) Risks averaged over sex and transfer method in a 1982 and 1988 Canadian life-table population
R: lifetime risk of fatal cancer;
Y: mean years lost if radiation induced cancer occurs
LLE: loss of life expectancy;
A: average age at radiation-induced death

Table 7.8
Comparison of ICRP 60 and Canada derived nominal fatal cancer risk factors

Average member in a general population
(10E-04 per Sv)

| Cancer | Canada (a) Add(b) - Mult(c) | ICRP 60 (d) |
|---|---|---|
| Leukemia | 70 - 80 | 50 |
| Respiratory | 65 - 115 | 85 |
| Breast | 10 - 35 | 20 |
| Digestive | 275 - 165 | 240 (e) |
| Other | 75 - 180 | 105 (f) |
| All cancers | 495 - 575 | 500 |

Average member in a working population
(10E-04 per Sv)

| Cancer | Canada (a) AR (b) - RR (c) | ICRP 60 (d) |
|---|---|---|
| Leukemia | 60 - 70 | -- |
| Respiratory | 80 - 150 | -- |
| Breast | 5 - 25 | -- |
| Digestive | 210 - 130 | -- |
| Other | 55 - 130 | -- |
| All cancers | 410 - 505 | 400 |

(a) Averaged over sex and life-table age distribution (ages 1-85) in a 1988 and 1982 Candian populations
(b) Risk projected using additive transfer method
(c) Risk projected using multiplicative transfer method
(d) Taken from table B-17, ICRP 1990 (averaged over both sexes (ages 1-90) in five national populations (Japan, U.S., Puerto Rico, U.K., and China, and both transfer methods)
(e) Combined ICRP risk coefficients for stomach, colin, liver, and oesophagus
(f) Combined ICRP risk coefficients for bladder, bone surface, ovary, skin, thyroid, and remainder organs

Table 7.9
Comparison of ICRP 60 and Canada derived tissue weighting factors

| Site | Tissue weighting factors | |
|---|---|---|
| | Canada (a) Add (b) - Mult (c) | ICRP 60 (d) |
| Gonads | 0.17 - 0.15 | 0.20 |
| Leukemia | 0.21 - 0.15 | 0.12 |
| Respiratory | 0.08 - 0.15 | 0.12 |
| Breast | 0.03 - 0.08 | 0.05 |
| Digestive | 0.39 - 0.20 | 0.35 (e) |
| Other | 0.12 - 0.25 | 0.16 (f) |

(a) Averaged over sex and life-table age distribution (ages 1-85) in a 1988 and 1982 Candian populations
(b) Risk projected using additive transfer method
(c) Risk projected using multiplicative transfer method
(d) Taken from table 2, ICRP 1990 (averaged over both sexes (ages 1-90) in five national populations (Japan, U.S., Puerto Rico, U.K., and China, and both transfer methods)
(e) Combined ICRP risk coefficients for stomach, colin, liver, and oesophagus
(f) Combined ICRP risk coefficients for bladder, bone surface, ovary, skin, thyroid, and remainder organs

Table 7.10
Comparison of the excess lifetime risk (excess cancers per 100,000 persons) projected for a 1988 Canadian population and that given in the BEIR V report result ing from a single whole-body dose of 0.1 Sv (a, b, c)

Males

| Age at exposure | All cancers | | | Leukemia | | | Respiratory | | | Breast | | | Digestive | | | Other | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Canada | BEIR V | Can/BEIR | Canada | BEIR V | Can/BEIR | Canada | BEIR V | Can/BEIR | Canada | BEIR V | Can/BEIR | Canada | BEIR V | Can/BEIR | Canada | BEIR V | Can/BEIR |
| 5 | 1523 | 1276 | 1.19 | 70 | 111 | 0.63 | – | 17 | – | | – | | 477 | 361 | 1.32 | 976 | 787 | 1.24 |
| 15 | 1360 | 1144 | 1.19 | 71 | 109 | 0.65 | 41 | 54 | 0.76 | | – | | 491 | 369 | 1.33 | 756 | 612 | 1.24 |
| 25 | 1107 | 921 | 1.20 | 24 | 36 | 0.67 | 115 | 124 | 0.93 | | – | | 518 | 389 | 1.33 | 451 | 372 | 1.21 |
| 35 | 615 | 566 | 1.09 | 43 | 62 | 0.69 | 247 | 243 | 1.02 | | – | | 33 | 28 | 1.18 | 292 | 233 | 1.25 |
| 45 | 646 | 600 | 1.08 | 76 | 108 | 0.70 | 396 | 353 | 1.12 | | – | | 27 | 22 | 1.23 | 147 | 117 | 1.26 |
| 55 | 649 | 616 | 1.05 | 128 | 166 | 0.77 | 501 | 393 | 1.27 | | – | | 16 | 15 | 1.07 | 48 | 42 | 1.14 |
| 65 | 570 | 481 | 1.19 | 159 | 191 | 0.83 | 397 | 272 | 1.46 | | – | | 11 | 11 | 1.00 | 3 | 7 | 0.43 |
| 75 | 284 | 258 | 1.10 | 147 | 165 | 0.89 | 135 | 90 | 1.50 | | – | | 7 | 5 | 1.40 | – | – | – |
| 85 | 94 | 110 | 0.85 | 98 | 96 | 1.02 | 2 | 17 | 0.12 | | – | | – | – | – | – | – | – |
| Average (c) | 870 | 770 | 1.13 | 85 | 110 | 0.77 | 220 | 190 | 1.16 | | – | | 200 | 170 | 1.18 | 360 | 300 | 1.20 |

Females

| Age at exposure | All cancers | | | Leukemia | | | Respiratory | | | Breast | | | Digestive | | | Other | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Canada | BEIR V | Can/BEIR | Canada | BEIR V | Can/BEIR | Canada | BEIR V | Can/BEIR | Canada | BEIR V | Can/BEIR | Canada | BEIR V | Can/BEIR | Canada | BEIR V | Can/BEIR |
| 5 | 1754 | 1532 | 1.14 | 51 | 75 | 0.68 | 52 | 24 | 2.17 | 112 | 129 | 0.87 | 801 | 655 | 1.22 | 737 | 625 | 1.18 |
| 15 | 1771 | 1566 | 1.13 | 58 | 72 | 0.81 | 77 | 70 | 1.10 | 281 | 295 | 0.95 | 798 | 653 | 1.22 | 558 | 476 | 1.17 |
| 25 | 1373 | 1178 | 1.17 | 19 | 29 | 0.66 | 138 | 125 | 1.10 | 45 | 52 | 0.87 | 831 | 679 | 1.22 | 340 | 293 | 1.16 |
| 35 | 619 | 557 | 1.11 | 33 | 46 | 0.72 | 235 | 208 | 1.13 | 45 | 43 | 1.05 | 87 | 73 | 1.19 | 220 | 187 | 1.18 |
| 45 | 610 | 541 | 1.13 | 53 | 73 | 0.73 | 333 | 277 | 1.20 | 23 | 20 | 1.15 | 84 | 71 | 1.18 | 117 | 100 | 1.17 |
| 55 | 603 | 505 | 1.19 | 84 | 117 | 0.72 | 385 | 273 | 1.41 | 6 | 6 | 1.00 | 76 | 64 | 1.19 | 52 | 45 | 1.16 |
| 65 | 493 | 386 | 1.28 | 108 | 146 | 0.74 | 306 | 172 | 1.78 | – | – | – | 63 | 52 | 1.21 | 17 | 16 | 1.06 |
| 75 | 240 | 227 | 1.06 | 99 | 127 | 0.78 | 97 | 72 | 1.35 | – | – | – | 39 | 26 | 1.50 | 6 | 3 | 2.00 |
| 85 | 92 | 90 | 1.02 | 65 | 73 | 0.89 | 23 | 15 | 1.53 | – | – | – | 5 | 4 | 1.25 | – | – | – |
| Average (c) | 905 | 810 | 1.12 | 60 | 80 | 0.75 | 200 | 150 | 1.33 | 50 | 70 | 0.71 | 335 | 290 | 1.16 | 260 | 220 | 1.18 |

(a) Risk transferred using multiplicative method
(b) DDREF=1
(c) BEIR V values taken from table 4-3, NRC 1990
(d) Averaged over the life-table age distribution of a 1988 Canada and 1980 U.S. populations, repectively

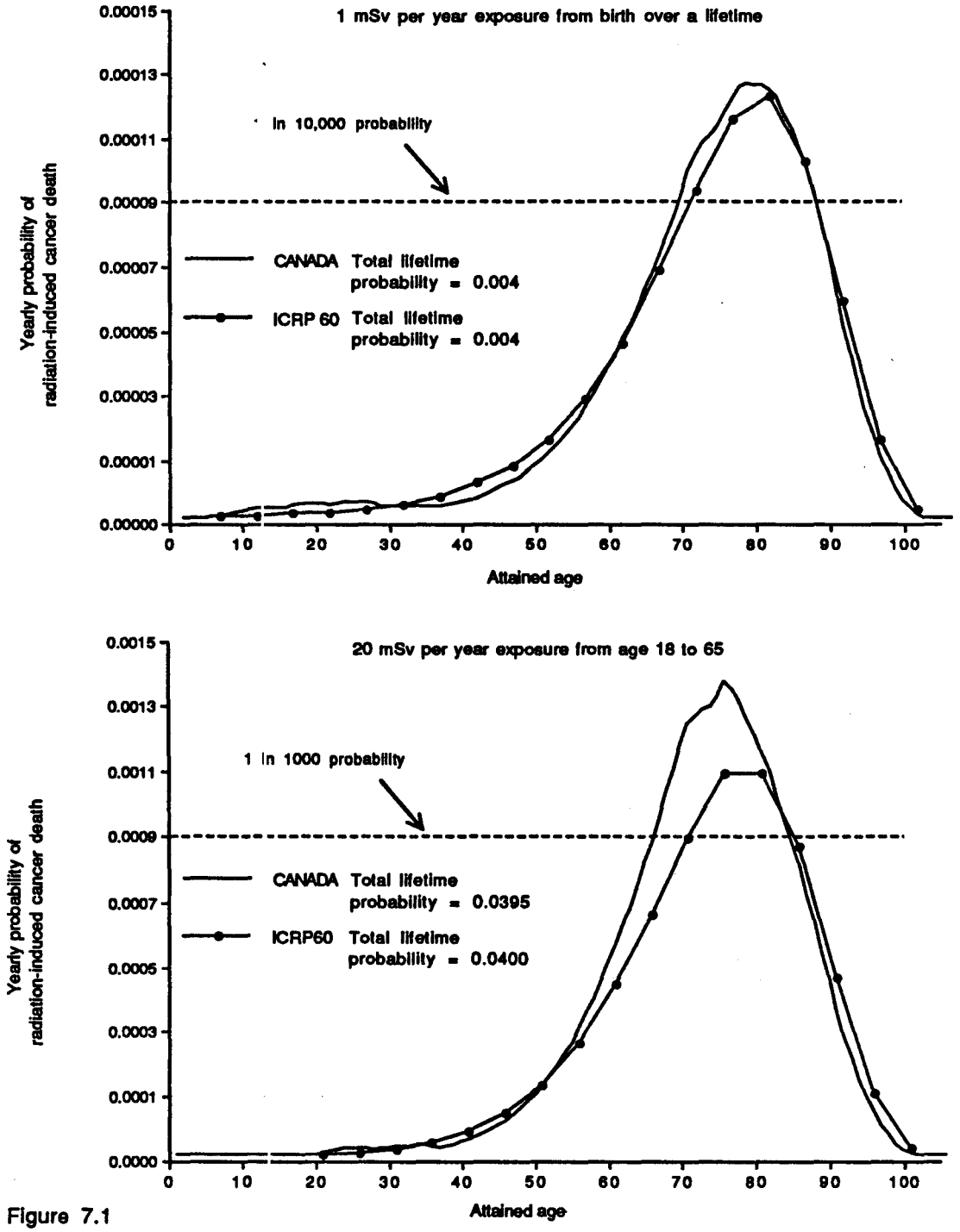



Figure 7.1
ICRP 60 and Canada projected distributions of radiation-induced cancers with attained age resulting from a continuous exposure of 1 mSv/yr from birth over a lifetime and 20 mSv/yr from age 18 to 65

## 8.0 Summary and Conclusions

Statistical and design limitations restrict the ability of epidemiological studies to measure increased risks of cancer at low dose levels. As a consequence, estimates of the carcinogenic risk have relied on the extrapolation of radiation effects observed from studies of populations receiving high radiation doses delivered at high dose rates. These high dose studies show that radiation increases the risk of cancer at most cancer sites. However, they provide limited information for the quantitative cancer risk at specific sites and on the influence of (a) sex, (b) age at exposure, (c) time since exposure, (d) fractionation of exposure, (e) dose rate, (f) radiation quality, (g) internal irradiation of organs, (h) differences in population baseline rates, (i) genetic, physiological and environmental factors, and (g) exposure to other carcinogens. Risk models presently used to analyze epidemiological data have little underlying relevance to the biological mechanisms of cancer induction. While biophysical models are being developed, no model yet exists that offers any advantage over the epidemiological approach. Until understanding of the carcinogenic process improves considerably, the knowledge of the carcinogenic risk of low-

level radiation exposure will continue to be based on human epidemiological and animal experimental studies.

The human epidemiological studies reviewed in chapter 2.0 demonstrate that high doses of low-LET ionizing radiation delivered at high dose rates can cause the subsequent radiation risk to be increased significantly for all types of leukemia excluding chronic lymphatic leukemia (CLL), cancers of the thyroid, female breast, lung, stomach, colon, oesophagus, bladder, ovary, salivary glands, rectum, brain and nervous system, kidney, body of the uterus, bone and connective tissue, and also multiple myeloma and non-Hodgkin's lymphoma. At other individual cancer sites, there is no clear evidence that radiation exposure increases the subsequent cancer risk. However, this may be due to the lack of available data involving sufficiently high irradiation of the appropriate organs to cause measurable increases (Darby 1991).

This report has derived fatal cancer risk estimates for the Canadian population using standard life-table techniques and the BEIR V preferred relative risk models for leukemia, cancers of the respiratory tract, female breast, digestive system, and "other" remaining sites.

Table 8.1 shows the variation of the derived lifetime fatal cancer risk factors derived for the Canadian general population caused by different sources of uncertainty. These sources include:

(1) sampling variation;

(2) extrapolation to low doses and low dose rates;

(3) projection of excess cancer risks beyond the current period of follow-up of atomic bomb survivors (i.e. beyond 40 years since exposure);

(4) the choice of method for transferring excess risk coefficients at individual cancer sites or groups of sites where baseline cancer mortality rates are substantially different between Japan and Canada; and

(5) differences in worker age and sex distributions among occupations in the Canadian "radiation" workforce.

Included in the table are the corresponding risk factors recommended in ICRP publication 60.

The relative importance of the different sources of uncertainty varies somewhat depending on the individual cancer site or group of sites. For radiation-induced leukemia, statistical errors in risk models causes the greatest source of uncertainty in the lifetime fatal cancer risk, with risks ranging from about 30 to 185 x $10^{-4}$ per Sv. For respiratory cancer, both sampling variation and uncertainty in the choice of risk transfer method causes substantial variations in the projected risk, with projections ranging from 60 to 135 x $10^{-4}$ per Sv. The choice of transfer method is the greatest source of uncertainty for female breast mortality. The estimated risk ranges from 10 to 35 x $10^{-4}$ per Sv, depending on whether an

1additive or multiplicative transfer method is used. The risk models for leukemia and cancers of the respiratory tract and female breast include time dependent terms that have the relative risk decreasing after a period of time following exposure. As a result lifetime projections are not significantly affected by projecting risks beyond 40 years since exposure.

For digestive and "other" cancers, age-constant relative risk are used which assumes the relative risk remains elevated over a lifetime. As a consequence, the projection of risks beyond 40 years represents a major source of uncertainty. On average, over 80% of the lifetime cancer risk of fatal cancer is projected beyond 40 years for these two cancer groups. The choice of risk transfer method is an additional source of substantial uncertainty. For digestive cancers, the additive transfer method results in an average fatal cancer risk of 275 x $10^{-4}$ per Sv compared to 165 x $10^{-4}$ per Sv projected by the multiplicative method. For other remaining cancers, the average projected risk is 75 and 180 x $10^{-4}$ per Sv for the additive and multiplicative method, respectively.

Variations are seen in the site-specific lifetime fatal cancer risk factors projected for the "average" male and female worker in the Canadian population. Site-specific worker averages differ by as much as a factor 3. The average

female worker risk factor for digestive cancer is over twice as high compared to average male worker. In contrast, male workers tend to have higher risk factors for leukemia and respiratory cancers by factors of about 1.5 and 1.7, respectively. The sex difference appears to be partly due to female workers being a younger workforce than male workers. At exposure ages under 35, age-specific lifetime cancer risks for digestive and "other" are substantially higher compared to older ages, while leukemia and respiratory age-specific risks are lower. Overall however, the majority of worker risk factors are within 25% of the site-specific projections for the workforce as a whole.

It difficult to interpret the significance of the variation of site-specific lifetime fatal cancer risks with age-at-exposure since most of the lifetime risk at younger ages is projected risk beyond the current follow-up of the Life Span Study. As table 8.2 and figure 8.1 shows, the projected risk beyond 40 years represents a significant proportion of the total lifetime risk of fatal cancer following exposure. For all cancers combined, over half of the average fatal cancer risk is projected beyond forty years. For digestive and "other" cancers, projections beyond 40 years for exposure ages under 20 represent over 90% of the lifetime projected cancer risk. For leukemia and cancers of respiratory tract and breast cancer, the proportion of the lifetime risks

projected beyond 40 years is not as significant.

Convolved into all the uncertainties at the different sites discussed above, is the uncertainty caused by extrapolating risks to low doses and low dose rates. The ICRP concludes that sufficient evidence exists to justify applying a DDREF of 2 to risk estimates derived from high dose and high dose rate data. Animal studies suggest the DDREF could range up to a value of 10, with a single best estimate of 5 (NRC 1990). Table 8.1 shows the range of site-specific estimates when a DDREF between 1 and 5 is assumed.

In view of the range of uncertainty in lifetime cancer risk projections, the ICRP 60 recommended nominal fatal cancer risk estimates, tissue weighting factors, and lifetime risk projections for prolonged exposures are in good agreement with risks derived for the Canadian population. The agreement is encouraging, particularly since Canadian projections were performed using the BEIR V relative risk models, while the ICRP estimates were not. Rather, the ICRP projections were based almost entirely on risk coefficients given in the RERF study of A-bomb cancer mortality and on the use of age-constant relative risk models. The good agreement between risk estimates suggest the more site-specific detailed nominal fatal cancer risk factors and $W_T$s recommended in ICRP 60 are as good any presently available. The results in this report supports the use of the ICRP values for the planning and

regulation of radiation protection in Canada.

It must be emphasized that the ICRP risks estimates and $w_T$s are "nominal", and are not appropriate for assessing the increased risk of cancer mortality for specific exposed individuals. There are however, occasions where over-exposures of individuals do occur, and where the estimation of the resulting potential risk to the individual may be useful. ICRP 60 suggests that it would be better to assess possible risks using (a) the organ absorbed dose, (b) specific data relating to the relative biological effectiveness of the radiations concern, and (c) the age-, sex-, and site-specific fatal cancer risks per unit dose factors derived for the population that the exposed individuals are from. Table 8.3 gives the age- and sex-specific lifetime fatal cancer risk per unit dose by cancer site derived in this report for the Canadian population. These estimates might be considered the best available for the risk assessment of exposed individuals in Canada. For cancers of the thyroid, skin, and bone surface, the risk estimates given in ICRP 60 are probably the best presently available.

It must always kept in mind that the age- and sex-specific fatal cancer risk factors represent only the average predicted risk expressed over lifetime for a cohort of individuals in a hypothetical life-table population. In view of the large uncertainties, any individual risk assessment

needs to interpreted cautiously.

Table 8.1
Variation of the derived lifetime risk of fatal cancer per unit dose for the Canadian population caused by different sources of uncertainty

| Source of Uncertainty | Liftime risk of fatal cancer (10E-04 per Sv) All cancers combined | Leukemia | Respiratory Tract | Female Breast | Digestive System | Other remaining cancers |
|---|---|---|---|---|---|---|
| Derived estimate for Canadian general population (a) | 530 | 75 | 90 | 25 | 220 | 130 |
| ICRP-60 recommended estimates | 500 | 50 | 85 | 20 | 240 | 105 |
| Sampling Variation (b) | 390 - 805 | 30 - 185 | 60 - 135 | 20 - 35 | 155 - 325 | 90 - 190 |
| Extrapolation to low doses and dose rates (c) | 230 - 1160 | 30 - 150 | 40 - 180 | 10 - 50 | 90 - 440 | 50 - 260 |
| Projecting excess risks beyond 40 years (d) | 240 - 530 | 68 - 75 | 73 - 90 | 15 - 25 | 45 - 220 | 30 - 130 |
| Transfer of risks between populations (e) | 495 - 575 | 70 - 80 | 65 - 115 | 10 - 35 | 275 - 165 | 75 - 180 |
| Differences in age and sex distributions (f) | 310 - 710 | 30 - 100 | 60 - 180 | 10 - 25 | 40 - 445 | 40 - 135 |

(a) Averaged over sex, age distribution of 1982 and 1988 Canadian life-tables (ages 1-85), and risk transfer methods (additive and multiplicative). Estimates include a DDREF= 2
(b) 90% confidence intervals of derived estimates - approximated using the ratio of the BEIR V upper and lower 90% CI to the excess lifetime risk point estimates given in the BEIR V Report (see section 4.7)
(c) Variation of the derived estimates assuming the DDREF could range from 1 to 5
(d) Derived estimates when projections are up to 40 years and over a lifetime (40 years and beyond)
(e) Variations of the derived estimates when an additive and multiplicative method is used to transfer risk coefficients of the BEIR V models to the Canadian general population
(f) Top and bottom of the range of lifetime cancer risks per unit dose projected for female and male workers in the Canadian "radiation" workforce (see chapter 6.0)

Table 8.2
Projected lifetime cancer risk per unit dose over a lifetime (40 years and beyond) and over only 40 years for a 1988 Canadian population resulting from low-dose radiation exposure (a, b, c)

Lifetime risk of fatal cancer
(10E-04 per Sv)

| Cancer site | Males | | | Females | | |
|---|---|---|---|---|---|---|
| | Lifetime | 40 years | Ratio Lifetime/40years | Lifetime | 40 years | Ratio Lifetime/40years |
| Leukemia | 92 | 84 | 1.10 | 66 | 60 | 1.10 |
| Respiratory tract | 142 | 115 | 1.23 | 109 | 89 | 1.22 |
| Breast | -- | -- | -- | 36 | 21 | 1.70 |
| Digestive system | 135 | 28 | 4.80 | 195 | 36 | 5.40 |
| Other | 229 | 51 | 4.50 | 155 | 36 | 4.30 |
| All cancers combined | 598 | 278 | 2.15 | 562 | 242 | 2.30 |

(a) Projections performed using the BEIR V relative risk models and the multiplicative risk transfer method
(b) DDREF= 2
(c) Averaged over a 1988 Canadian life-table age distribution

Table 8.3
Derived age- and sex-specific lifetime fatal cancer risk factors for Canadian risk assessments following low-dose radiation exposure (a, b)

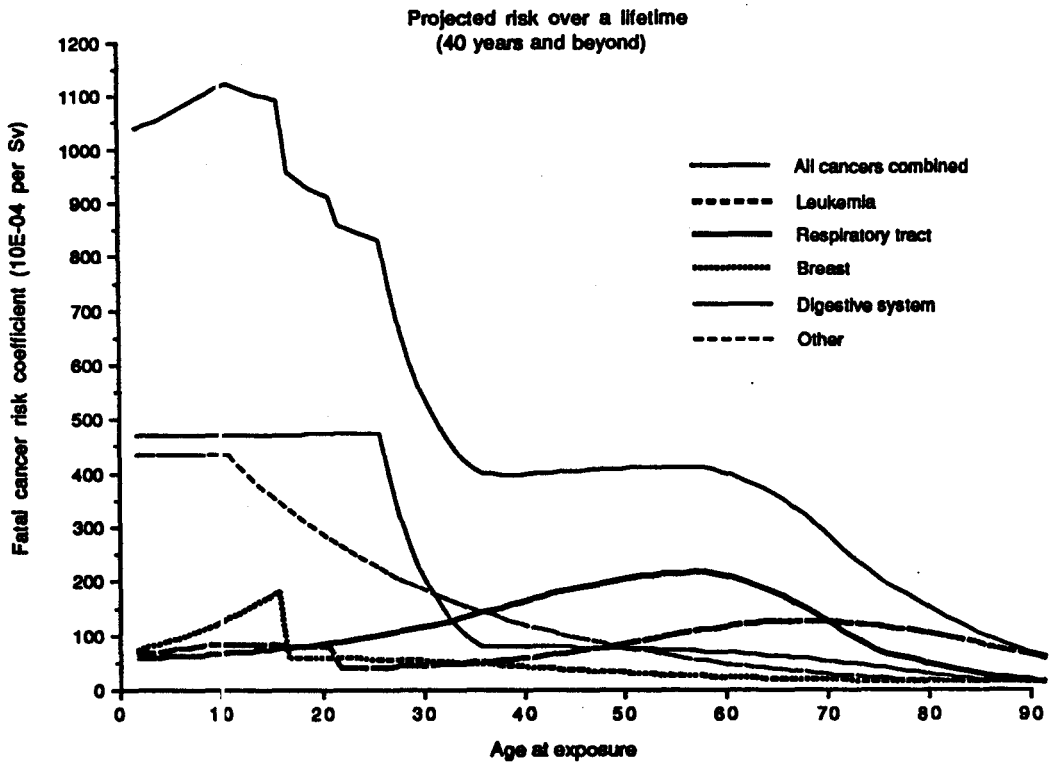
Males
(10E-04 per Sv)

| Age at exposure | Leukemia | Respiratory | Breast | Digestive | Other | All cancers |
|---|---|---|---|---|---|---|
| 5 | 106 | 40 | - | 429 | 362 | 937 |
| 10 | 103 | 45 | - | 430 | 361 | 939 |
| 15 | 104 | 52 | - | 430 | 286 | 873 |
| 20 | 115 | 61 | - | 432 | 227 | 835 |
| 25 | 34 | 73 | - | 434 | 181 | 723 |
| 30 | 41 | 89 | - | 162 | 144 | 435 |
| 35 | 49 | 111 | - | 60 | 114 | 333 |
| 40 | 60 | 135 | - | 59 | 89 | 342 |
| 45 | 74 | 167 | - | 57 | 69 | 366 |
| 50 | 89 | 187 | - | 53 | 52 | 382 |
| 55 | 103 | 206 | - | 49 | 39 | 397 |
| 60 | 111 | 195 | - | 41 | 27 | 374 |
| 65 | 111 | 169 | - | 33 | 19 | 332 |
| 70 | 104 | 115 | - | 23 | 11 | 252 |
| 75 | 91 | 60 | - | 13 | 6 | 169 |
| 80 | 74 | 25 | - | 6 | 2 | 107 |
| 85 | 56 | 9 | - | 2 | 1 | 68 |

Females
(10E-04 per Sv)

| Age at exposure | Leukemia | Respiratory | Breast | Digestive | Other | All cancers |
|---|---|---|---|---|---|---|
| 5 | 85 | 31 | 55 | 580 | 306 | 1057 |
| 10 | 87 | 35 | 79 | 581 | 306 | 1087 |
| 15 | 82 | 40 | 111 | 581 | 242 | 1057 |
| 20 | 87 | 48 | 27 | 582 | 192 | 935 |
| 25 | 28 | 57 | 26 | 582 | 152 | 844 |
| 30 | 32 | 68 | 23 | 216 | 120 | 458 |
| 35 | 38 | 83 | 19 | 80 | 94 | 313 |
| 40 | 45 | 97 | 15 | 78 | 73 | 309 |
| 45 | 56 | 114 | 12 | 77 | 56 | 314 |
| 50 | 68 | 124 | 8 | 74 | 41 | 316 |
| 55 | 79 | 134 | 6 | 69 | 30 | 319 |
| 60 | 86 | 130 | 4 | 62 | 21 | 303 |
| 65 | 88 | 114 | 2 | 53 | 14 | 271 |
| 70 | 80 | 84 | 1 | 40 | 8 | 214 |
| 75 | 68 | 49 | 1 | 26 | 4 | 149 |
| 80 | 54 | 28 | 0 | 13 | 2 | 96 |
| 85 | 39 | 12 | 0 | 5 | 0 | 56 |

(a) Averaged projected lifetime risk of fatal cancer for 1982 and 1988 Canadian life-table populations. Projected risks averaged over both the additive and multiplicative risk transfer methods
(b) A dose and dose rate effectiveness factor of 2 has been applied to estimates



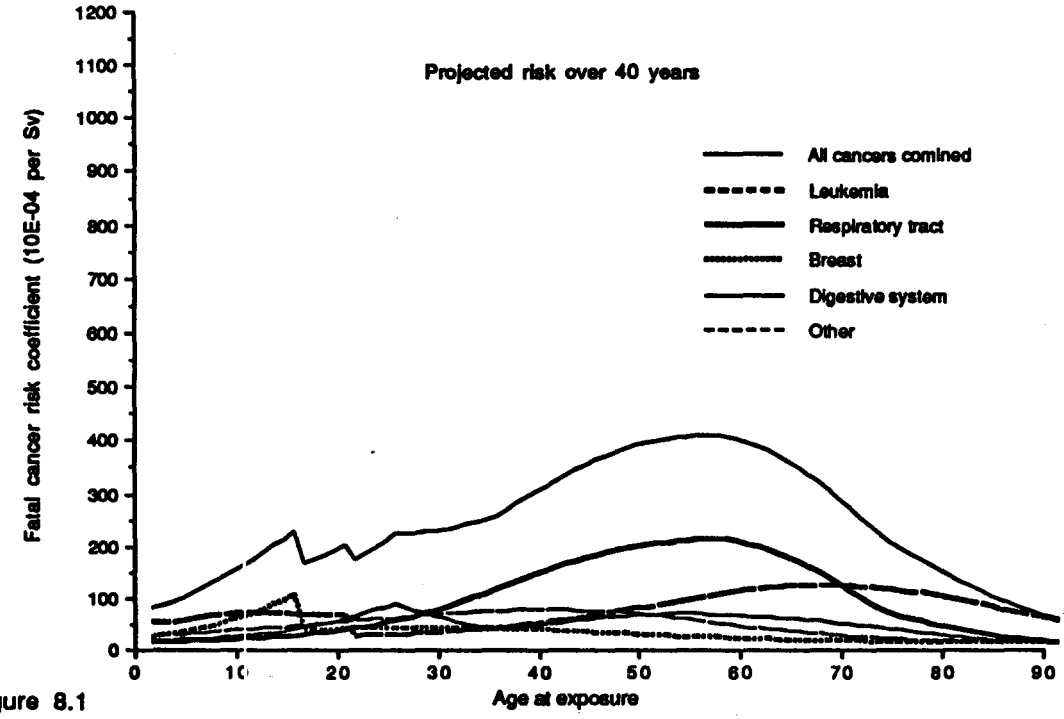




Figure 8.1
Projected risk of fatal cancer per unit dose over an entire lifetime and over only 40 years following exposure by cancer site (Projections for a 1988 Canadian female population using the multiplicative transfer method and a DDREF=2)

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## Appendix A: Calculation of 1988 Canadian Cancer Mortality and Mortality From All Causes Rates

Statistics Canada ceased publishing baseline cancer mortality rates for Canada in 1985, the last report being for cancer in Canada in 1982 (StatsCan 1985). In the desire to have the most up-to-date baseline rates for conducting lifetime risk projections for the Canadian population, it was decided to calculate baseline rates for Canada in 1988 using data from causes of death tables and population estimates obtained from Statistics Canada (StatsCan 1990a, 1990b). Death tables listed categories of causes of deaths according to the International Classification of Disease (ICD) codes (World Health Organization). Data was given by sex and for the age intervals: under age 1, 1-4, five year intervals up to age 85, and 85 and above. Population estimates were given by sex and single age up to age 85.

This appendix describes the methodology used to calculate cancer mortality and all causes of death rates using the death and demographic data.

### A.1 Conditional Probability of Death (Mortality Rate)

Mortality rates used in this report are conditional probability of death rates. Normally, mortality rates

represent a central rate indicating the relative frequency of death. However, it does not precisely describe the risk of dying for a life table cohort. The conditional probability of death rate on the other hand expresses the chance of death in an interval of time for persons alive at the beginning of the interval (Shyrock and Siegel 1973). The mortality rate at age u, m(u,t), during the time interval (t,t+dt) is defined as the ratio of the average death rate in the interval to the number of person alive at the beginning of the interval, l'(u,t). That is,

$$m(u,t) = \frac{1}{l'(u,t)} \frac{dd(u,t)}{dt}$$

where

m(u,t) is the age-specific mortality rate or the time interval (t,t+dt);

u is the age of population at time t;

d d(u,t)/dt is the average death rate for age u in the interval (t,t+dt); and

l'(u,t) is number of persons aged u alive at time t

If d t is taken as one year, then the above expression may be rewritten as

$$m(u) = \frac{d(u)}{PY(u)}$$

where

m(u) is the age-specific mortality rate for the

year;

d(u) is the total number of deaths in the year for age u; and

PY(u) is the number of persons of age u living at the beginning of the year

Because demographic tables report the midyear population as an approximation of the average population, it is necessary to approximate the number of persons of age u living at the beginning of the year (Shyrock and Siegel 1973). If it is assumed that deaths will be uniformly distributed throughout the year, half of the deaths will occur in the first half of the year and the number of person living at the beginning will be given by

$$PY(u) = l(u) + \frac{1}{2}d(u) \qquad (A1)$$

where

l(u) is the midyear population of persons aged u

The age-specific mortality rate may be rewritten as

$$m(u) = \frac{d(u)}{l(u) + \frac{1}{2}d(u)} \qquad (A2)$$

Similarly, the age-specific cancer mortality rate for cancer of type i, $\lambda_i(u)$, is given by

$$\lambda_i(u) = \frac{d_i(u)}{l(u) + \frac{1}{2}d(u)} \qquad \text{(A3)}$$

where

$d_i(u)$ is the number of cancer deaths of cancer type i in the year; and

$d(u)$ is the total number of deaths in the year from all causes

Deaths of interest in this report are all causes combined and by those cancers in the BEIR V risk model groupings of sites:

| | |
|---|---|
| Leukemia | (ICD 204-207), |
| Respiratory cancer | (ICD 160-163), |
| Female breast cancer | (ICD 174), |
| Digestive cancer | (ICD 150-159), and |
| Other cancers | (ICD 140-209 less those listed above) |

## A.2 Calculation of Age-specific Mortality Rates

Death data obtained from Statistics Canada were given in terms of ages under 1, 1-4, five year age intervals up to age 85 and ages 85 and above grouped together. Most Cancer statistics are given in this manner and radiological reports that perform risk projections, such as BEIR V and ICRP 60, leave them as five year interval age rates. For this report, it was decided to interpolate rates to single years and

extrapolate rates to ages beyond 85. This was done in three steps:

1. interpolating Canadian death data to single year values and extrapolating death data beyond age 85 and population data beyond 90;
2. calculating the mortality rates for both sexes and each age up to 105 using equations A2 and A3; and
3. fitting a smooth line through computed points using the AMFIT Poisson regression program.

In retrospect, interpolating death to single years before computing mortality rates with equations A2 and A3 proved to be a more difficult task in contrast to first computing five year interval age rates and then interpolating to single ages. However, it did allow rates to be extrapolated beyond age 85. Each of the above steps will briefly be described.

Step 1

Age grouped death data for ages under 85 were interpolated to single years assuming an uniform distribution of deaths within an age group. Single age deaths were computed so that the sum of single age values within an age group equalled the total number of deaths as a group.

For the open ended age group of 85 and above, extrapolation of deaths at older ages were made using two assumptions. First that the maximum age of the population is

105 and second, the variation of deaths with age would be described by a second degree polynomial. Single age deaths above age 85 were computed so that the sum of single age deaths equalled that for age 85 and above as a group and so that there would be no deaths beyond age 105.

Statistics Canada population estimates were given in single ages up to age 90 and ages above 90 as a group. Data was extrapolated to ages u beyond age 90 using the function

$$F(u) = e^{(\alpha_0+\alpha_1 u+\alpha_2 u^2)}$$

The parameters $\alpha_0$, $\alpha_1$, and $\alpha_2$ were determined by using the program AMFIT to fit the function to population data at ages between 75 and 90.

Step 2

Age-specific mortality rates were calculated using equations A3 and A3 and the single death and population data produced in step 1.

Step 3

It is a normal procedure to graduate, or smooth, rates that are computed in the manner done in step 1 and step 2 (Shyrock and Siegel 1973). This was done by using the AMFIT Poisson regression program. The program was used to fit data to the function[1]

[1] This form of function was used because mortality rates tend to follow a log-normal distribution, particularly between the ages of 35 and 85 (conservation with Pierce 1990).

$$d(u) = PY(u)\, e^{(\alpha_0 + \alpha_1 u + \alpha_2 u^2)}$$

where

d(u) is the number of cancer deaths at single ages u; and

PY(u) is the person-years at risk at age u as given by equation A1.

Various models with different values $\alpha_0$, $\alpha_1$, and $\alpha_2$ of were used to fit different age portions to produce the best overall fitting curve.

No gradation was performed for age-specific cancer mortality rates above the age of 85. Any discontinuities or irregularities in the curve were corrected manually. For mortality from all causes, interpolated and extrapolated rates peaked at about age 90 and then deceased thereafter. Mortality rates in life tables must continue to increase and reach a value of 100% probability of death at the last age in the population. Because of this, rates after the peak age, $u_{peak}$, were extrapolated to the age 105 using the function

$$m(u) = m(u_{peak})\ e^{\beta (u - u_{peak})}$$

where $u_{peak} \leq u \leq 105$

The parameter $\beta$ was chosen so that the probability of death at age 105 is one.

## A.3 Results of Calculations

Figures A1 to A6 plots the calculated age-specific

mortality rates by sex and cause when data was not interpolated or extrapolated (five year interval age rates) and when data were interpolated and extrapolated (single age rates). Overall, the single age rates provide a good fit to the five year interval age rates. The extrapolation procedure produced rates that are consistent with what would be expected - peaking at an older and then declining thereafter. For female respiratory cancer, however, rates were seen to peak at about age 80, decline, begin to increase again at age 85, reach a second peak at age 95, and decline thereafter (see figure A2). This double peaking was most likely an artifact of the extrapolation procedure.

When lifetime risk projections were perform separately using the both five year interval rates[2] and single age cancer mortality rates, resulting cancer risk estimates did not differ greatly (within ± 1-3% for most exposure ages). However, not interpolating mortality rates for all causes of death did have somewhat an effect, with projections using interpolated and non-interpolated rates differing by 5-15% at some cancer sites for exposures at younger ages. It appears that it is sufficient to use five year interval age cancer mortality rates for lifetime risk projection, while rates for all causes of death should be single age specific rates if

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[2] Single age rates within an age group taken to be the five year interval rate and rates at single above age 85 assumed to be constant and equal to the above 85 group rate.

possible. If rates are to be interpolated to single ages, it is not recommended that the approach used in this appendix be used. An alternative, and much easier approach, would be to calculated five year interval age rates first, and then interpolate linearly to single ages.

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StatsCan 1990a. *"Causes of death tables: Canada 1988"*. Statistics Canada (1990).

StatsCan 1990b. *"1988 Canadian Population Estimates"*. Statistics Canada (1990).

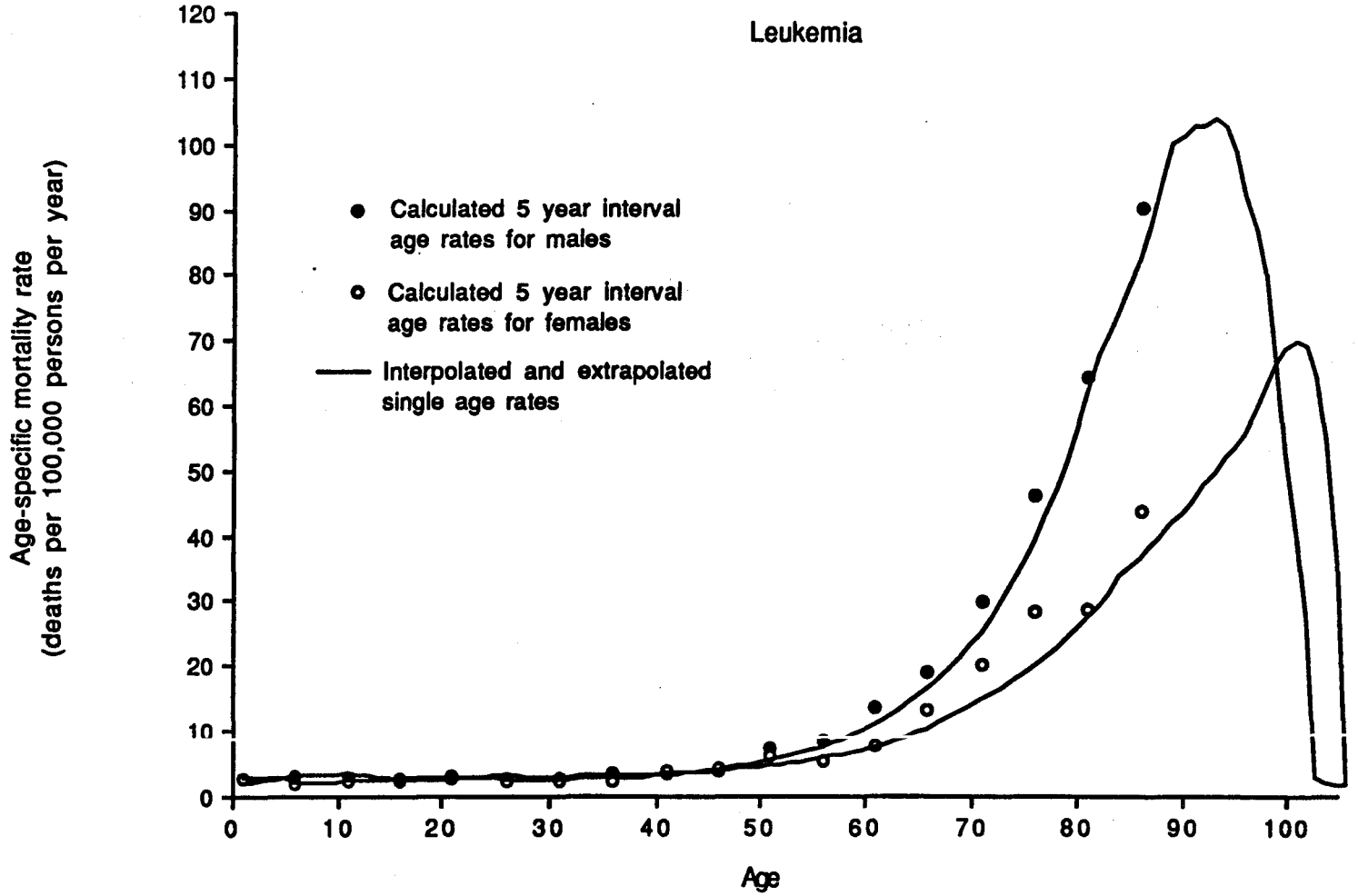


Figure A1
Calculated 1988 Canada age-specific mortality rates for leukemia

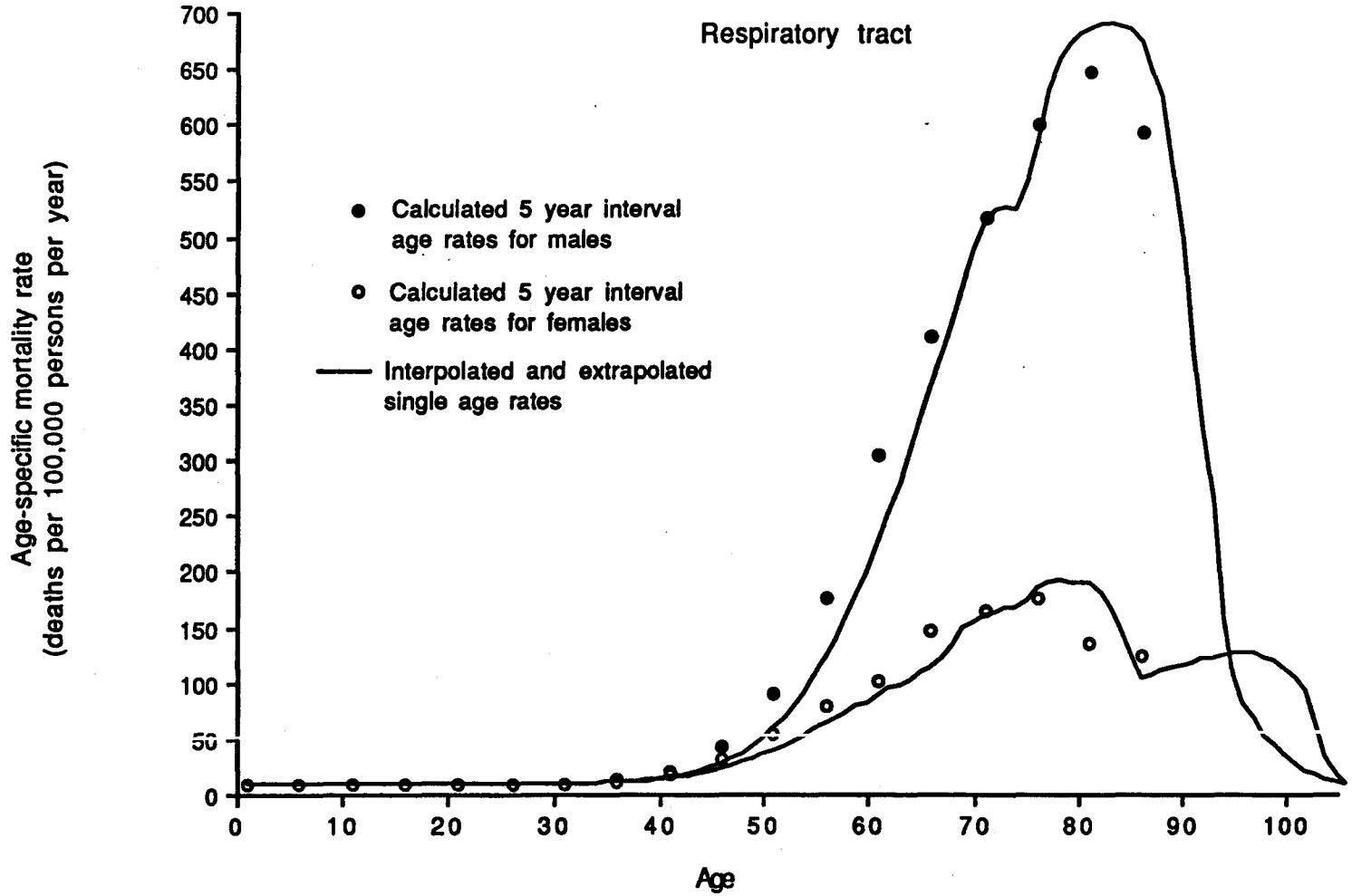


Figure A2
Calculated 1988 Canada age-specific mortality rates for respiratory tract

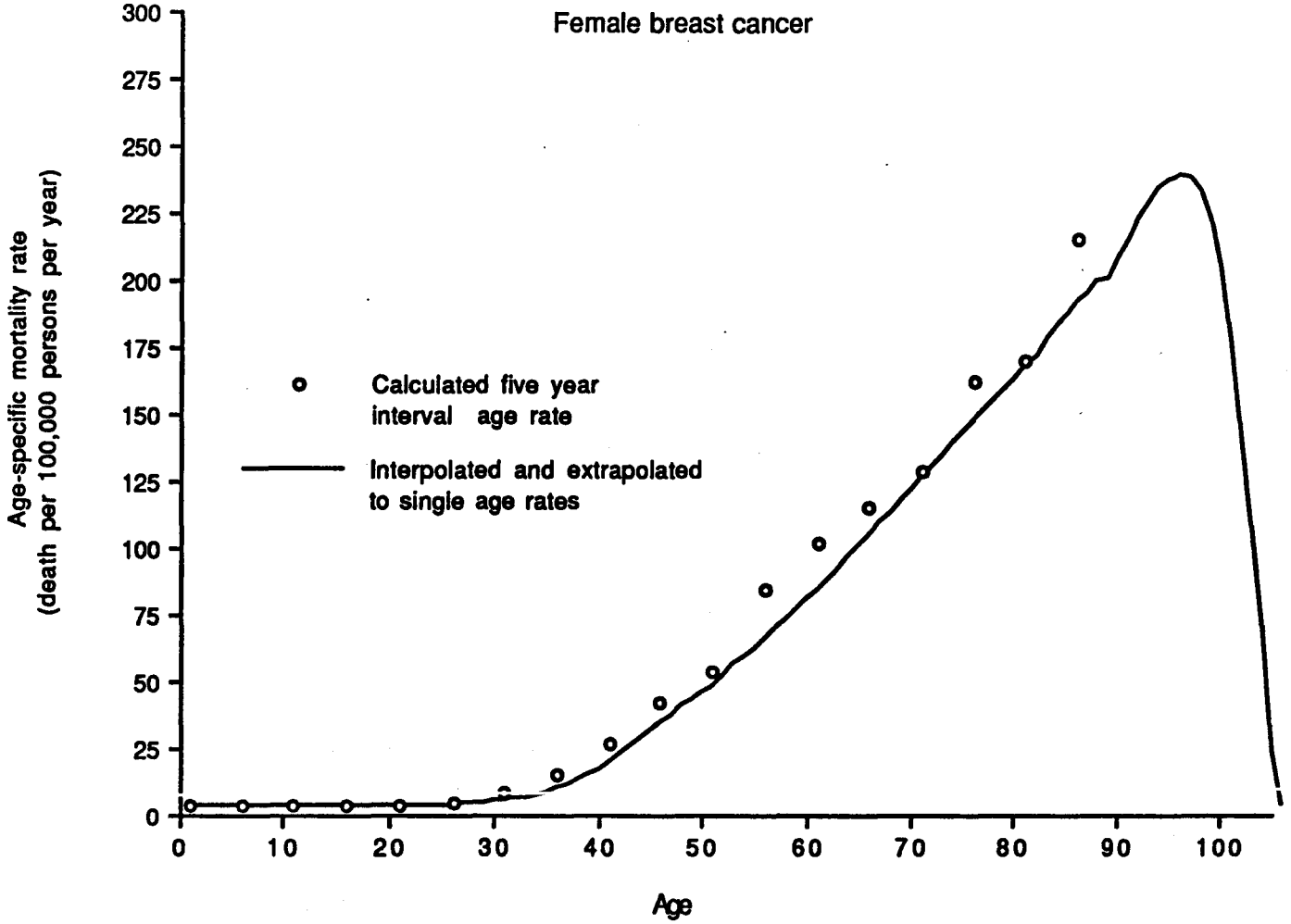


Figure A3
Calculated 1988 Canada age-specific mortality rates for female breast cancer

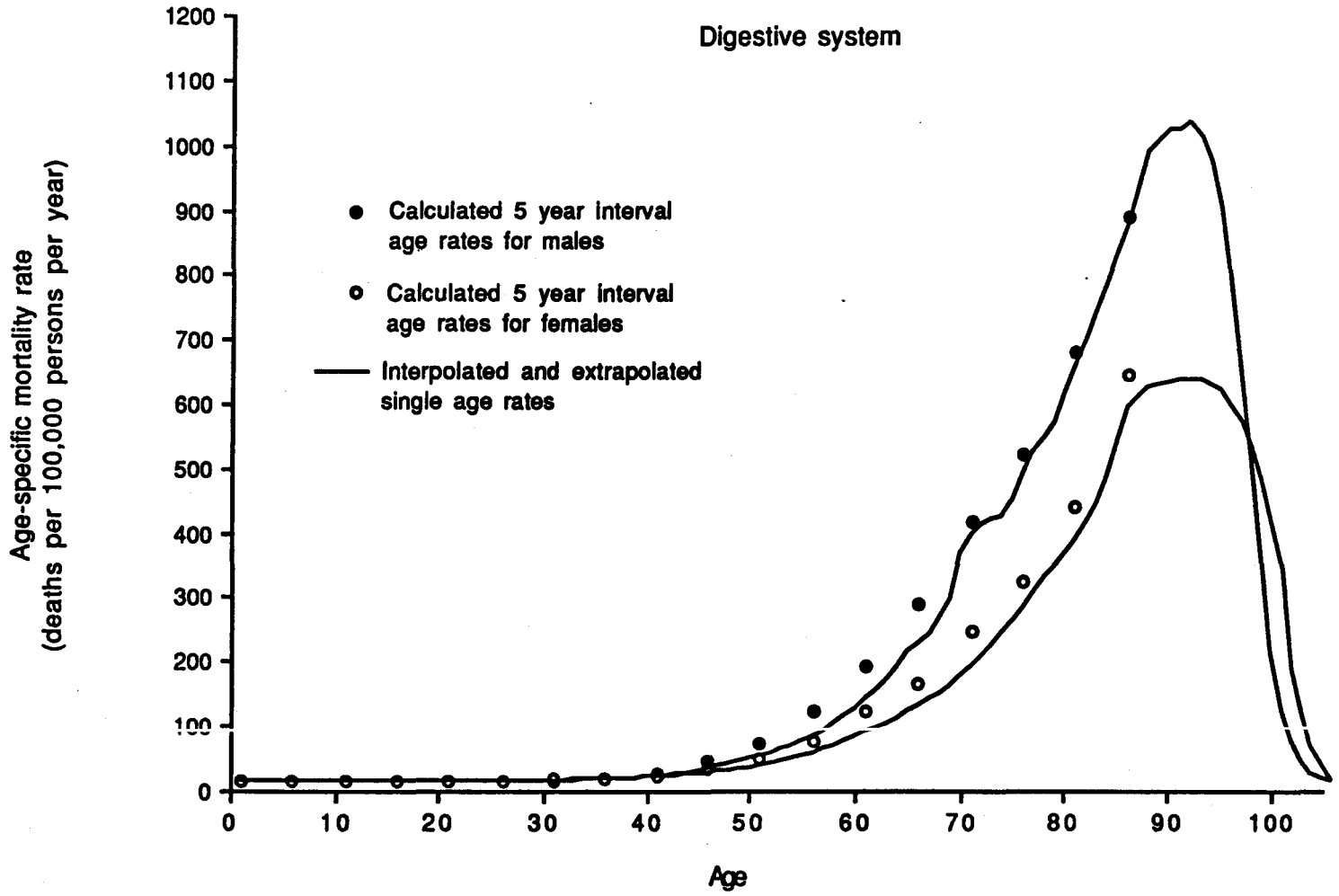


Figure A4
Calculated 1988 Canada age-specific mortality rates for digestive system

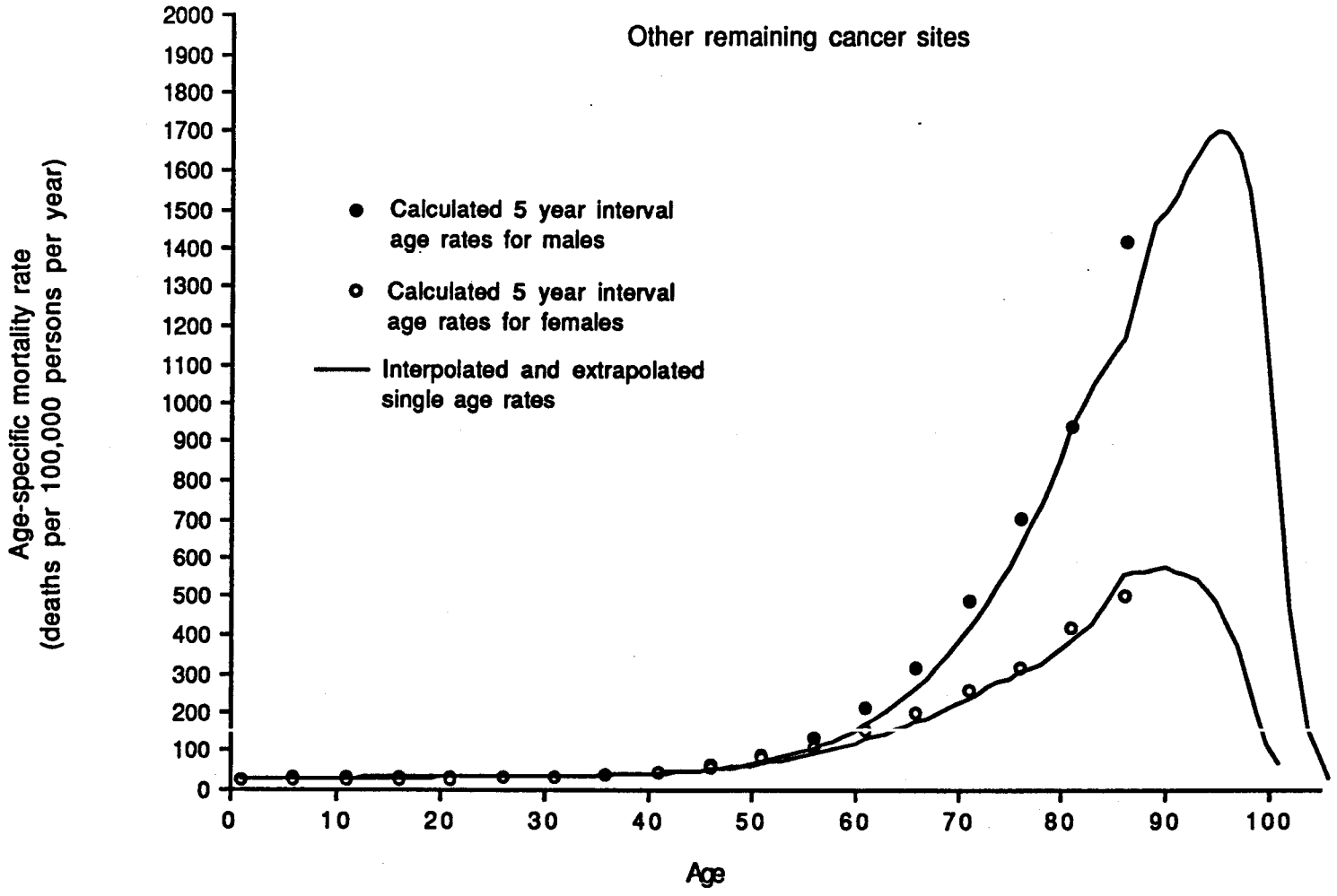


Figure A5
Calculated 1988 Canada age-specific mortality rates for other remaining cancers sites

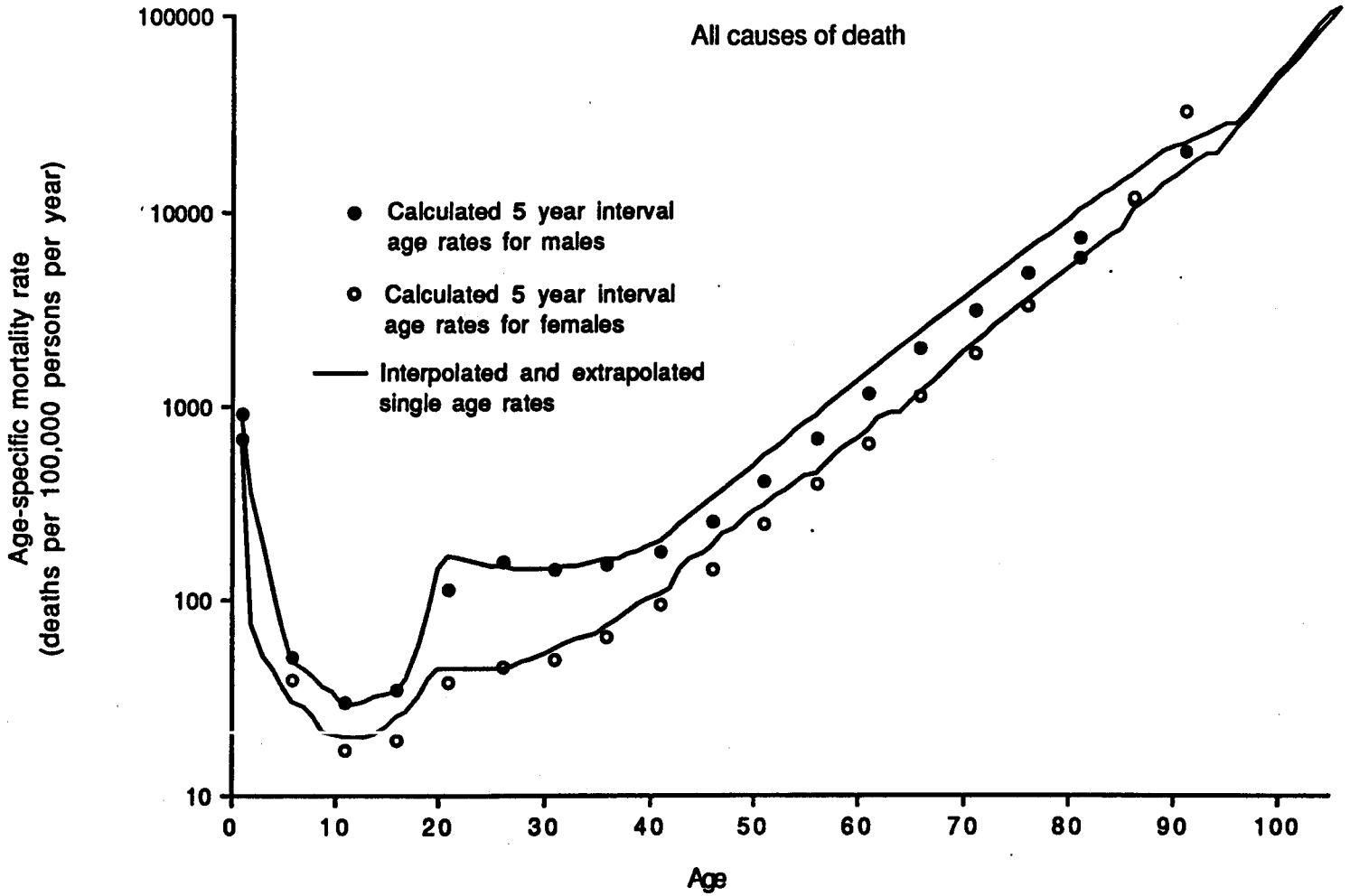


Figure A6
Calculated 1988 Canada age-specific mortality rates for all causes of death

## Appendix B: Age- and Sex Specific Cancer Mortality and Mortality From All Causes Rates for 1988 Canada, 1982 Canada, 1984 Japan, and 1980 U.S.

This appendix gives in tabular form the baseline age- and sex-specific mortality rate data for all causes of death combined and the for the cancer sites or groups of sites used in the BEIR V models. Rates for the 1984 Japanese population were supplied by Dale Preston of the Radiation Effects Research Foundation and those for the 1980 U.S. population by David Noel of the U.S. National Institute of Health. Baseline cancer rates for 1982 Canada were taken from 1982 Canadian cancer statistics (StatsCan 1985a) and overall mortality rates from the 1980-82 Canadian life-tables (StatsCan 1985b). The 1988 Canadian rates were calculated using data from causes of death tables and population estimates for 1988 supplied by Statistics Canada (StatsCan 1990a, 1990b). See appendix A for details on calculations.

Table B1 gives the baseline age- and sex-specific mortality rates for cancer and table B2 for all causes of death. Cancer mortality rate for Canada in 1988 are presented as five year interval age rates, but the interpolated and extrapolated single age rates calculated in appendix A are used for lifetime risk projections. For rates in the three

other populations, single age rates within an age group were assumed to be constant and equal to the five year interval rate and rates at single ages above age 85 were assumed to be the rate for the above 85 age group.

References

StatsCan 1990a. *"Causes of death tables: Canada 1988"*. Statistics Canada (1990).

StatsCan 1990b. *"1988 Canadian Population Estimates"*. Statistics Canada (1990).

StatsCan 1985a. *"1982 Canada Cancer Rates"*. Statistics Canada (1985).

StatsCan 1985b. *"Canada 1980-82 current life tables"*. Statistics Canada (1985).

Table B1
Age-specific cancer mortality rates (deaths per 100,000 per year)

1988 Canada

| Age | All cancers | | Leukemia | | Respiratory | | Breast | | Digestive | | Other | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Male | Female | Male | Female | Male | Female | Male | Female | Male | Female | Male | Female |
| 0-4 | 4.0 | 4.0 | 1.0 | 1.0 | 0.0 | 0.0 | — | 0.0 | 0.0 | 0.2 | 3.1 | 2.8 |
| 5-9 | 5.2 | 3.3 | 1.6 | 0.2 | 0.1 | 0.1 | — | 0.0 | 0.1 | 0.1 | 3.4 | 2.8 |
| 10-14 | 4.6 | 2.7 | 1.2 | 0.8 | 0.0 | 0.0 | — | 0.0 | 0.1 | 0.0 | 3.3 | 1.9 |
| 15-19 | 4.6 | 4.0 | 0.9 | 1.3 | 0.0 | 0.0 | — | 0.0 | 0.2 | 0.0 | 3.5 | 2.7 |
| 20-24 | 6.9 | 3.6 | 1.4 | 1.0 | 0.3 | 0.1 | — | 0.0 | 0.2 | 0.2 | 5.1 | 2.4 |
| 25-29 | 8.3 | 7.7 | 1.0 | 0.7 | 0.3 | 0.3 | — | 0.9 | 0.6 | 0.3 | 6.4 | 5.5 |
| 30-34 | 11.8 | 17.2 | 1.1 | 0.7 | 0.7 | 0.9 | — | 5.0 | 1.9 | 2.3 | 8.1 | 8.2 |
| 35-39 | 22.1 | 30.4 | 1.8 | 0.7 | 3.5 | 3.3 | — | 11.4 | 5.0 | 2.3 | 11.9 | 12.7 |
| 40-44 | 46.1 | 64.4 | 2.0 | 2.5 | 11.2 | 8.9 | — | 22.6 | 13.5 | 9.1 | 19.4 | 21.2 |
| 45-49 | 102.3 | 114.1 | 2.5 | 2.7 | 34.0 | 21.9 | — | 38.1 | 29.8 | 19.3 | 36.0 | 32.2 |
| 50-54 | 207.4 | 190.9 | 5.7 | 4.5 | 81.4 | 44.5 | — | 50.3 | 57.7 | 36.5 | 62.6 | 55.0 |
| 55-59 | 391.2 | 301.4 | 6.9 | 3.9 | 167.7 | 69.1 | — | 80.3 | 109.3 | 61.4 | 107.3 | 86.8 |
| 60-64 | 672.2 | 437.3 | 11.9 | 6.3 | 296.5 | 93.2 | — | 98.3 | 176.4 | 107.2 | 187.5 | 132.2 |
| 65-69 | 984.5 | 583.7 | 17.5 | 11.6 | 401.2 | 137.3 | — | 111.2 | 274.7 | 149.1 | 291.1 | 174.6 |
| 70-74 | 1405.5 | 764.8 | 28.2 | 18.6 | 507.0 | 156.7 | — | 125.1 | 404.3 | 230.9 | 466.0 | 233.5 |
| 75-79 | 1821.8 | 953.0 | 44.4 | 26.6 | 592.2 | 167.3 | — | 159.1 | 507.6 | 308.6 | 677.6 | 291.3 |
| 80-84 | 2281.3 | 1139.1 | 62.9 | 27.1 | 639.3 | 127.1 | — | 166.1 | 666.1 | 424.5 | 913.0 | 394.2 |
| 85+ | 2942.2 | 1473.0 | 88.5 | 42.2 | 584.3 | 114.2 | — | 211.7 | 875.3 | 629.6 | 1394.1 | 475.3 |

1982 Canada

| Age | All cancers | | Leukemia | | Respiratory | | Breast | | Digestive | | Other | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Male | Female | Male | Female | Male | Female | Male | Female | Male | Female | Male | Female |
| 0-24 | 6.1 | 4.1 | 1.6 | 1.1 | 0.0 | 0 | - | 0 | 0.1 | 0.1 | 4.4 | 2.9 |
| 25-34 | 12.5 | 12 | 1.5 | 0.8 | 0.5 | 0.6 | - | 2.3 | 1.8 | 1.4 | 8.7 | 6.9 |
| 35-44 | 37.5 | 49.5 | 2.3 | 2.2 | 9.9 | 9.2 | - | 16.4 | 9.7 | 8 | 15.6 | 13.7 |
| 45-54 | 160.2 | 151.9 | 3.1 | 3.1 | 62.4 | 27.2 | - | 46.7 | 48.3 | 29 | 46.4 | 45.9 |
| 55-64 | 497.3 | 345.8 | 8.7 | 7 | 215.2 | 64.2 | - | 83.9 | 136.8 | 85.9 | 136.6 | 104.8 |
| 65-74 | 1106.1 | 633.7 | 20.1 | 18.3 | 434.3 | 102 | - | 114.6 | 331.7 | 198.9 | 320.0 | 199.9 |
| 75-84 | 1935.2 | 963.2 | 45.8 | 29.2 | 595.3 | 98.6 | - | 146.7 | 587.9 | 376.1 | 706.2 | 312.6 |
| 85+ | 2737.8 | 1451.8 | 73.2 | 38.2 | 487.8 | 83.3 | - | 208.2 | 885.6 | 652.3 | 1291.2 | 469.8 |

Table B1 cont.
Age-specific cancer mortality rates (deaths per 100,000 per year)

1984 Japan

| Age | All cancers | | Leukemia | | Respiratory | | Breast | | Digestive | | Other | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Male | Female | Male | Female | Male | Female | Male | Female | Male | Female | Male | Female |
| 0-4 | 4.6 | 4.3 | 1.9 | 1.5 | 0.1 | 0 | 0 | 0 | 0.4 | 0.2 | 2.2 | 2.6 |
| 5-9 | 4.4 | 3.3 | 2.3 | 1.8 | 0.1 | 0 | 0 | 0 | 0.1 | 0.1 | 1.9 | 1.4 |
| 10-14 | 4.1 | 3.2 | 2.1 | 1.8 | 0 | 0 | 0 | 0 | 0.2 | 0.2 | 1.8 | 1.2 |
| 15-19 | 5.6 | 4.1 | 2 | 1.9 | 0.2 | 0.1 | 0 | 0 | 0.6 | 0.4 | 2.8 | 1.7 |
| 20-24 | 6.4 | 5.7 | 2 | 1.8 | 0.4 | 0.1 | 0 | 0.2 | 1.2 | 1.3 | 2.8 | 2.3 |
| 25-29 | 9.1 | 9.3 | 2 | 1.2 | 0.6 | 0.4 | 0 | 0.8 | 3.7 | 4.4 | 2.8 | 2.5 |
| 30-34 | 17.2 | 20.7 | 2.7 | 2.4 | 1.5 | 1.3 | 0 | 2.8 | 8.8 | 9.9 | 4.2 | 4.3 |
| 35-39 | 29.3 | 33.1 | 3.1 | 2.3 | 3.1 | 1.7 | 0 | 5.4 | 18.4 | 16.5 | 4.7 | 7.2 |
| 40-44 | 51.7 | 54.3 | 4.1 | 2.9 | 6.2 | 3.5 | 0 | 9.3 | 34 | 26.4 | 7.4 | 12.2 |
| 45-49 | 104.8 | 86.6 | 4.8 | 3.6 | 12.4 | 6.6 | 0 | 13.8 | 73 | 42.1 | 14.6 | 20.5 |
| 50-54 | 219.8 | 131.5 | 7.7 | 4.5 | 30.1 | 10.8 | 0.1 | 18.5 | 156.8 | 64.9 | 25.1 | 32.8 |
| 55-59 | 361.4 | 192.2 | 8.2 | 6 | 59.5 | 17.6 | 0 | 21.9 | 251.5 | 97.6 | 42.2 | 49.1 |
| 60-64 | 550.4 | 271.4 | 10.5 | 5.6 | 110.7 | 28.1 | 0.3 | 19.3 | 362.4 | 156.4 | 66.5 | 62 |
| 65-69 | 854.6 | 401.5 | 14.4 | 9.1 | 192.4 | 47.7 | 0.3 | 18 | 540.4 | 238.9 | 107.1 | 87.8 |
| 70-74 | 1218.5 | 569 | 20.4 | 11.6 | 295 | 71.9 | 0.4 | 18.2 | 740.5 | 347.1 | 162.2 | 120.2 |
| 75-79 | 1672.1 | 799.8 | 22.5 | 13 | 410.5 | 101.4 | 0.8 | 18.6 | 999.8 | 506.9 | 238.5 | 159.9 |
| 80-84 | 2049.9 | 1011.6 | 22.9 | 13.6 | 442.2 | 122.2 | 0.8 | 20.4 | 1258.8 | 659.5 | 325.2 | 195.9 |
| 85+ | 2024.8 | 1068 | 22.6 | 12.2 | 382.1 | 115.8 | 0.4 | 24.7 | 1242.7 | 711.8 | 377 | 203.5 |

1960 U.S.

| Age | All cancers | | Leukemia | | Respiratory | | Breast | | Digestive | | Other | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Male | Female | Male | Female | Male | Female | Male | Female | Male | Female | Male | Female |
| 0-4 | 4.8 | 4 | 1.5 | 1.4 | 0.2 | 0.2 | — | 0 | 0.6 | 0.4 | 2.5 | 2 |
| 5-9 | 4.9 | 3.7 | 2.3 | 1.5 | 0.0 | 0 | — | 0 | 0.0 | 0.1 | 2.6 | 2.1 |
| 10-14 | 4.1 | 3.3 | 1.7 | 1.2 | 0.0 | 0 | — | 0 | 0.1 | 0.1 | 2.3 | 2 |
| 15-19 | 5.9 | 4 | 1.9 | 1.2 | 0.1 | 0.1 | — | 0 | 0.3 | 0.1 | 3.6 | 2.6 |
| 20-24 | 7.4 | 4.5 | 1.7 | 1 | 0.2 | 0.1 | — | 0.1 | 0.5 | 0.4 | 5.0 | 2.9 |
| 25-29 | 10.5 | 7.8 | 1.9 | 1.2 | 0.4 | 0.2 | — | 1.2 | 1.5 | 0.8 | 6.7 | 4.4 |
| 30-34 | 14.9 | 16.1 | 2.1 | 1.6 | 1.4 | 0.9 | — | 5.6 | 2.6 | 1.7 | 8.8 | 6.3 |
| 35-39 | 28.6 | 33.2 | 2.7 | 2.1 | 6.3 | 3.8 | — | 12.5 | 6.0 | 4.2 | 13.6 | 10.6 |
| 40-44 | 60.5 | 64.1 | 3.2 | 2.7 | 19.8 | 9.9 | — | 23.2 | 13.6 | 9 | 23.9 | 19.3 |
| 45-49 | 127.7 | 119.1 | 5.0 | 3.7 | 51.5 | 24.4 | — | 37.7 | 30.5 | 18.9 | 40.7 | 34.4 |
| 50-54 | 244.8 | 203.2 | 6.5 | 5.2 | 108.2 | 43.9 | — | 58 | 56.3 | 36.2 | 73.8 | 59.9 |
| 55-59 | 413.9 | 300.3 | 11.7 | 6.8 | 180.8 | 68.1 | — | 73.9 | 102.2 | 61.8 | 119.2 | 89.7 |
| 60-64 | 641.9 | 408.3 | 18.3 | 10.4 | 271.0 | 89.8 | — | 84.8 | 160.4 | 92.7 | 192.2 | 130.6 |
| 65-69 | 939.7 | 541.1 | 28.6 | 14.4 | 384.5 | 110.2 | — | 100.7 | 239.2 | 139.8 | 287.4 | 176 |
| 70-74 | 1283.9 | 662.3 | 43.9 | 22 | 468.4 | 112.8 | — | 109 | 342.4 | 201.6 | 429.2 | 216.9 |
| 75-79 | 1624.5 | 814.8 | 62.0 | 33 | 515.6 | 104.1 | — | 119.4 | 441 | 278.9 | 605.9 | 279.4 |
| 80-84 | 2006.3 | 1009.3 | 90.9 | 45.8 | 499.3 | 96.8 | — | 137.7 | 559.5 | 386.1 | 856.6 | 342.9 |
| 85+ | 2350.7 | 1225.1 | 104.4 | 59.4 | 413.5 | 91.4 | — | 171.8 | 683.7 | 496.7 | 1149.1 | 405.8 |

Table B2
Age-specifi mortality rates for all causes of death combined

Males
(deaths per 100,000 persons per year)

| Age | 1988 Canada | 1982 Canada | 1984 Japan | 1980 U.S. |
|---|---|---|---|---|
| 0 | 813 | 1092 | 482 | 336 |
| 1 | 311 | 81 | 75 | 336 |
| 2 | 172 | 63 | 55 | 336 |
| 3 | 101 | 48 | 40 | 336 |
| 4 | 63 | 47 | 31 | 336 |
| 5 | 42 | 39 | 26 | 35 |
| 6 | 30 | 30 | 25 | 35 |
| 7 | 23 | 22 | 23 | 35 |
| 8 | 19 | 19 | 21 | 35 |
| 9 | 16 | 19 | 18 | 35 |
| 10 | 15 | 22 | 16 | 38 |
| 11 | 15 | 27 | 15 | 38 |
| 12 | 15 | 35 | 15 | 38 |
| 13 | 17 | 49 | 17 | 38 |
| 14 | 20 | 69 | 23 | 38 |
| 15 | 26 | 92 | 36 | 134 |
| 16 | 35 | 112 | 52 | 134 |
| 17 | 51 | 128 | 69 | 134 |
| 18 | 79 | 139 | 81 | 134 |
| 19 | 129 | 147 | 87 | 134 |
| 20 | 150 | 153 | 87 | 140 |
| 21 | 144 | 157 | 83 | 140 |
| 22 | 140 | 158 | 77 | 140 |
| 23 | 136 | 157 | 73 | 140 |
| 24 | 134 | 153 | 72 | 140 |
| 25 | 131 | 148 | 74 | 194 |
| 26 | 130 | 143 | 76 | 194 |
| 27 | 129 | 139 | 76 | 194 |
| 28 | 128 | 136 | 76 | 194 |
| 29 | 129 | 134 | 76 | 194 |
| 30 | 129 | 132 | 78 | 196 |
| 31 | 131 | 132 | 80 | 196 |
| 32 | 133 | 134 | 82 | 196 |
| 33 | 135 | 139 | 86 | 196 |
| 34 | 139 | 145 | 91 | 196 |
| 35 | 143 | 153 | 97 | 246 |
| 36 | 147 | 163 | 105 | 246 |
| 37 | 153 | 175 | 115 | 246 |
| 38 | 160 | 189 | 127 | 246 |
| 39 | 167 | 205 | 140 | 246 |
| 40 | 176 | 223 | 155 | 367 |
| 41 | 197 | 245 | 172 | 367 |
| 42 | 218 | 271 | 192 | 367 |
| 43 | 241 | 301 | 216 | 367 |
| 44 | 267 | 334 | 240 | 367 |
| 45 | 295 | 372 | 264 | 585 |
| 46 | 326 | 414 | 286 | 585 |
| 47 | 361 | 461 | 308 | 585 |
| 48 | 399 | 512 | 336 | 585 |
| 49 | 441 | 567 | 372 | 585 |

| Age | 1988 Canada | 1982 Canada | 1984 Japan | 1980 U.S. |
|---|---|---|---|---|
| 50 | 487 | 628 | 416 | 937 |
| 51 | 538 | 694 | 464 | 937 |
| 52 | 594 | 768 | 518 | 937 |
| 53 | 656 | 848 | 579 | 937 |
| 54 | 724 | 933 | 648 | 937 |
| 55 | 799 | 1026 | 727 | 1446 |
| 56 | 882 | 1127 | 808 | 1446 |
| 57 | 973 | 1239 | 888 | 1446 |
| 58 | 1074 | 1360 | 963 | 1446 |
| 59 | 1185 | 1488 | 1034 | 1446 |
| 60 | 1306 | 1628 | 1108 | 2243 |
| 61 | 1441 | 1781 | 1190 | 2243 |
| 62 | 1588 | 1951 | 1286 | 2243 |
| 63 | 1751 | 2138 | 1397 | 2243 |
| 64 | 1929 | 2339 | 1518 | 2243 |
| 65 | 2126 | 2556 | 1654 | 3373 |
| 66 | 2342 | 2790 | 1803 | 3373 |
| 67 | 2579 | 3046 | 1977 | 3373 |
| 68 | 2841 | 3317 | 2176 | 3373 |
| 69 | 3128 | 3601 | 2410 | 3373 |
| 70 | 3443 | 3907 | 2675 | 5060 |
| 71 | 3790 | 4243 | 2975 | 5090 |
| 72 | 4171 | 4617 | 3308 | 5090 |
| 73 | 4589 | 5024 | 3673 | 5090 |
| 74 | 5048 | 5460 | 4067 | 5090 |
| 75 | 5552 | 5930 | 4527 | 7495 |
| 76 | 6106 | 6442 | 5071 | 7495 |
| 77 | 6713 | 7002 | 5698 | 7495 |
| 78 | 7380 | 7607 | 6400 | 7495 |
| 79 | 8111 | 8251 | 7189 | 7495 |
| 80 | 8913 | 8941 | 7987 | 11343 |
| 81 | 9792 | 9683 | 8861 | 11343 |
| 82 | 10757 | 10483 | 9805 | 11343 |
| 83 | 11645 | 11338 | 10824 | 11343 |
| 84 | 12556 | 12243 | 11922 | 11343 |
| 85 | 13697 | 13203 | 13106 | 16922 |
| 86 | 15004 | 14227 | 14379 | 16922 |
| 87 | 16481 | 15319 | 15747 | 16922 |
| 88 | 18074 | 16475 | 17214 | 16922 |
| 89 | 19000 | 17692 | 18787 | 16922 |
| 90 | 19664 | 18975 | 20468 | 24429 |
| 91 | 20924 | 20332 | 22264 | 24429 |
| 92 | 22172 | 21767 | 24176 | 24429 |
| 93 | 23345 | 22325 | 26210 | 24429 |
| 94 | 24352 | 22003 | 28366 | 24429 |
| 95 | 25060 | 22234 | 28400 | 33688 |
| 96 | 29038 | 24450 | 31800 | 33688 |
| 97 | 33648 | 30086 | 35700 | 33688 |
| 98 | 38989 | 41245 | 40000 | 33688 |
| 99 | 45179 | 56973 | 44900 | 33688 |
| 100 | 52352 | 74112 | 50300 | 33688 |
| 101 | 60663 | 89506 | 56400 | |
| 102 | 70293 | 100000 | 63200 | |
| 103 | 81452 | | 70900 | |
| 104 | 94383 | | 89200 | |
| 105 | 100000 | | 100000 | |

Table B2 cont.
Age-specifi mortality rates for all causes of death combined

Females
(deaths per 100,000 persons per year)

| Age | 1988 Canada | 1982 Canada | 1984 Japan | 1980 U.S. |
|---|---|---|---|---|
| 0 | 640 | 843 | 425 | 261 |
| 1 | 66 | 66 | 62 | 261 |
| 2 | 46 | 46 | 44 | 261 |
| 3 | 39 | 39 | 30 | 261 |
| 4 | 32 | 32 | 21 | 261 |
| 5 | 27 | 27 | 17 | 21 |
| 6 | 25 | 23 | 16 | 21 |
| 7 | 22 | 20 | 15 | 21 |
| 8 | 19 | 19 | 14 | 21 |
| 9 | 18 | 18 | 13 | 21 |
| 10 | 18 | 18 | 12 | 23 |
| 11 | 18 | 19 | 12 | 23 |
| 12 | 18 | 22 | 12 | 23 |
| 13 | 18 | 25 | 12 | 23 |
| 14 | 20 | 30 | 13 | 23 |
| 15 | 23 | 36 | 16 | 50 |
| 16 | 24 | 41 | 20 | 50 |
| 17 | 29 | 45 | 24 | 50 |
| 18 | 35 | 46 | 28 | 50 |
| 19 | 39 | 47 | 31 | 50 |
| 20 | 39 | 47 | 31 | 61 |
| 21 | 39 | 47 | 31 | 61 |
| 22 | 39 | 47 | 30 | 61 |
| 23 | 39 | 48 | 30 | 61 |
| 24 | 39 | 49 | 31 | 61 |
| 25 | 40 | 50 | 32 | 67 |
| 26 | 41 | 52 | 34 | 67 |
| 27 | 43 | 53 | 36 | 67 |
| 28 | 44 | 55 | 39 | 67 |
| 29 | 47 | 56 | 40 | 67 |
| 30 | 50 | 57 | 42 | 82 |
| 31 | 52 | 60 | 43 | 82 |
| 32 | 56 | 63 | 46 | 82 |
| 33 | 57 | 69 | 49 | 82 |
| 34 | 60 | 75 | 54 | 82 |
| 35 | 64 | 82 | 59 | 123 |
| 36 | 72 | 90 | 65 | 123 |
| 37 | 78 | 99 | 72 | 123 |
| 38 | 85 | 100 | 79 | 123 |
| 39 | 91 | 120 | 87 | 123 |
| 40 | 96 | 132 | 95 | 195 |
| 41 | 102 | 145 | 102 | 195 |
| 42 | 128 | 160 | 110 | 195 |
| 43 | 145 | 170 | 118 | 195 |
| 44 | 156 | 192 | 127 | 195 |
| 45 | 168 | 210 | 138 | 312 |
| 46 | 193 | 23[illegible] | 149 | 312 |
| 47 | 210 | 254 | 163 | 312 |
| 48 | 233 | 280 | 180 | 312 |
| 49 | 252 | 300 | 199 | 312 |

| Age | 1988 Canada | 1982 Canada | 1984 Japan | 1980 U.S. |
|---|---|---|---|---|
| 50 | 273 | 338 | 217 | 487 |
| 51 | 302 | 371 | 235 | 487 |
| 52 | 322 | 407 | 253 | 487 |
| 53 | 353 | 445 | 275 | 487 |
| 54 | 385 | 485 | 301 | 487 |
| 55 | 394 | 528 | 329 | 734 |
| 56 | 449 | 575 | 358 | 734 |
| 57 | 499 | 627 | 386 | 734 |
| 58 | 556 | 682 | 416 | 734 |
| 59 | 606 | 740 | 448 | 734 |
| 60 | 664 | 804 | 483 | 1130 |
| 61 | 758 | 875 | 525 | 1130 |
| 62 | 819 | 957 | 573 | 1130 |
| 63 | 821 | 1050 | 629 | 1130 |
| 64 | 926 | 1150 | 695 | 1130 |
| 65 | 1040 | 1260 | 773 | 1690 |
| 66 | 1170 | 1380 | 859 | 1690 |
| 67 | 1310 | 1510 | 957 | 1690 |
| 68 | 1470 | 1660 | 1070 | 1690 |
| 69 | 1640 | 1810 | 1200 | 1690 |
| 70 | 1840 | 1980 | 1340 | 2640 |
| 71 | 2050 | 2180 | 1500 | 2640 |
| 72 | 2280 | 2400 | 1690 | 2640 |
| 73 | 2530 | 2650 | 1900 | 2640 |
| 74 | 2800 | 2910 | 2140 | 2640 |
| 75 | 3100 | 3210 | 2430 | 4220 |
| 76 | 3430 | 3550 | 2770 | 4220 |
| 77 | 3790 | 3940 | 3180 | 4220 |
| 78 | 4170 | 4380 | 3660 | 4220 |
| 79 | 4590 | 4870 | 4230 | 4220 |
| 80 | 5040 | 5400 | 4860 | 7270 |
| 81 | 5520 | 6000 | 5530 | 7270 |
| 82 | 6050 | 6660 | 6270 | 7270 |
| 83 | 6610 | 7380 | 7100 | 7270 |
| 84 | 7210 | 8160 | 8020 | 7270 |
| 85 | 9000 | 9000 | 9040 | 12100 |
| 86 | 9910 | 9910 | 10200 | 12100 |
| 87 | 10900 | 10900 | 11400 | 12100 |
| 88 | 12000 | 12000 | 12800 | 12100 |
| 89 | 13100 | 13100 | 14300 | 12100 |
| 90 | 14400 | 14400 | 16000 | 19200 |
| 91 | 15700 | 15700 | 17900 | 19200 |
| 92 | 17100 | 17100 | 19900 | 19200 |
| 93 | 17500 | 17500 | 22100 | 19200 |
| 94 | 20300 | 17100 | 24500 | 19200 |
| 95 | 23500 | 17200 | 27800 | 27800 |
| 96 | 27100 | 19500 | 31600 | 27800 |
| 97 | 31300 | 25500 | 35900 | 27800 |
| 98 | 36200 | 37300 | 40800 | 27800 |
| 99 | 41900 | 54100 | 46400 | 27800 |
| 100 | 48400 | 72400 | 52700 | 27800 |
| 101 | 56000 | 88900 | 59900 | |
| 102 | 64700 | 100000 | 68100 | |
| 103 | 74800 | | 77400 | |
| 104 | 86500 | | 88000 | |
| 105 | 100000 | | 100000 | |

## Appendix C: "Radrisk" Computer Code For Projecting Lifetime Cancer Risks For Populations Exposed to Ionizing Radiation

This appendix presents the code for the computer program "Radrisk" that was written to calculate the lifetime risk projections performed in this report. Because it was impractical to present the entire code[1], only those sections of the code which are associated with the actual use of the BEIR V models and life-table techniques described in chapter 4.0 are given. Sections of code presented are:

1. Program menu;
2. Construction of life-table;
3. Calculation of conditional risk coefficients using the BEIR V preferred relative risk models;
4. Calculation of lifetime cancer risk attributes;
5. Calculation of population averages; and
6. Menu for printing results to screen and to data files

[1] Code involving reading of data files, storing of data, screen display, and writing of results to file and screen are not given due to impracticality, in terms of length and quantity, of including it in the appendix.

```
print "*************************************************************************"
print "**                    RADRISK (version 1.0)                            **"
print "**                                                                     **"
print "**      PROJECTION OF LIFETIME CANCER RISKS TO A POPULATION            **"
print "**                    EXPOSED TO IONIZING RADIATION                    **"
print "**                                                                     **"
print "**       L.R. Rasmussen, September, 1990 (all rights reserved)         **"
print "*************************************************************************"
print " "
COLOR 14: print "     MENU:"
print "          (1) CONTINUOUS ANNUAL EXPOSURE"
print "          (2) SINGLE INSTANTANEOUS EXPOSURE"
PRINT "          (3) ACCIDENTAL OCCUPATION EXPOSURE
print "          (4) NOT TO BE SELECTED UNDER ANY CIRCUMSTANCES
PRINT "          (5) ANALYSIS
PRINT "          (6) QUIT
'ARRAYS:
'
'                  DOSE(PERSON,SEX,ATB): dose record
'          EO(PERSON,PERSON2,SEX,CANCER): reduction of life expectancy
'         ELR(PERSON,PERSON2,SEX,CANCER): excess lifetime risk
'            R(PERSN,PERSON2,SEX,CANCER): lifetime attributable probability of de
'           Y(PERSON,PERSON2,SEX,CANCER): years of life lost
'      YMEAN(PERSON,PERSON2,SEX,CANCER): years of life lost per excess death
'               LLE(PERSON,PERSON2,SEX): loss of life expectancy
'        R65(PERSON,PERSON2,SEX,CANCER): age 0-65
'       R6575(PERSON,PERSON2,SEX,CANCER): age 65-75   proportion of risk
'       R7585(PERSON,PERSON2,SEX,CANCER): age 75-85   with repect to age
'        R85A(PERSON,PERSON2,SEX,CANCER): age 85+
'                        Q(TBL,SEX,AGE): age-specific conditional probability of
'                        D(TBL,SEX,AGE): deaths at specific age
'                       L1(TBL,SEX,AGE): person living at specific age
'                       L2(TBL,SEX,106): person  years lived at specific age
'                        T(TBL,SEX,AGE): remaining person years to live
'                        P(TBL,SEX,AGE): probability of surviving the year at sp
'                    S(TBL,TBL,SEX,AGE): probability of surviving to specific ag
'                        E(TBL,SEX,AGE): remaining life expectancy
'                         LOSS(SEX,AGE): years of life lost if radiation death o
'                  RCAN(SEX,AGE,CANCER): age-specific conditional probability of
'                         QRAD(SEX,AGE): age-specific conditional probablity of
'                      SQRAD(TBL,SEX,AGE): age-specific unconditional probability
'                 CANCER(SEX,AGE,CANCER): Canadian background age-specific cancer
'                   BEIR(SEX,AGE,CANCER): BEIRV radiation risk factors
' VARIABLES:
'
'                                  PERSON: labels each exposure history
'                                 PERSON2: labels age-at-exposure for instaneous e
'                                     SEX: 1-male
'                                          2-female
'                                          3-both
'                                  CANCER: 1-leukemia
'                                          2-repiratory
'                                          3-breast cancer
'                                          4-digestive cancer
'                                          5-other
'                                          6-total
'                                          7-nonleukemia
'                                     ATB: age-at-exposure
'                                     AGE: 0-105
'                                     TBL: labels lifetable
'
'                                          1-reference lifetable
'                                          2-radiation lifetable
```

```
*****************************************************************************
*                                                                           *
*                CONSTRUCTION OF LIFETABLE                                  *
*                                                                           *
*                                                                           *
*****************************************************************************
' 1. LIFETABLE
' *************
LIFETAB:
'This section calculates the various components of the life table. Quanties
'are:
'
' Q(TAB,SEX,AGE): Probability of death at particular age
' P(TAB,SEX,AGE): Probability of surviving at particular age
' D(TAB,SEX,AGE): Deaths at particular age
'L1(TAB,SEX,AGE): No. persons alive at particular age
'L2(TAB,SEX,AGE): Person years lived at particular age
' T(TAB,SEX,AGE): Person years remaining to live
' E(TAB,SEX,AGE): Life expectancy for particular age
' S(TAB,SEX,AGE): Probability of surviving to a particular age
' SQRAD(SEX,AGE): Person years exposed at particular age
'where
'       TAB: 1=reference table; 2=radiation table
'       SEX: 1=male; 2=female; 3=both
'       AGE: Age at risk
'       ATB: Age-at-exposure
L1!(TBL,SEX,MIN)=100000
D!(TBL,SEX,MIN)= Q!(TBL,SEX,MIN)*L1!(TBL,SEX,MIN)
P!(TBL,SEX,MIN)= 1-Q!(TBL,SEX,MIN)
S!(TBL,SEX,MIN)= 1
IF TBL=2 THEN SQRAD!(2,SEX,MIN)=S!(TBL,SEX,MIN)*RCAN!(SEX,MIN,6)

FOR AGE=MIN+1 TO 105
'LOCATE 15,1: PRINT "AGE: " AGE-1
'PRINT "P!: " P!(TBL,SEX,AGE-1)
    L1!(TBL,SEX,AGE)=    L1!(TBL,SEX,AGE-1)*P!(TBL,SEX,AGE-1)
    L2!(TBL,SEX,AGE-1)= (L1!(TBL,SEX,AGE-1)+L1!(TBL,SEX,AGE))/2
'    D!(TBL,SEX,AGE)=     Q!(TBL,SEX,AGE)*L1!(TBL,SEX,AGE)
    P!(TBL,SEX,AGE)=     1-Q!(TBL,SEX,AGE)
    S!(TBL,SEX,AGE)=     S!(TBL,SEX,AGE-1)*P!(TBL,SEX,AGE-1)
    IF TBL=2 THEN SQRAD!(2,SEX,AGE)=S!(TBL,SEX,AGE)*RCAN!(SEX,AGE,6)
    IF Q!(TBL,SEX,AGE)>1 THEN P!(TBL,SEX,AGE)=0
NEXT AGE
L2!(TBL,SEX,105)=(L1!(TBL,SEX,104)-D!(TBL,SEX,104))/2
IF TBL=2 THEN SQRAD!(2,SEX,105)=S!(TBL,SEX,105)*RCAN!(SEX,105,6)
FOR AGE=MIN TO 105
    FOR I=AGE TO 105
        T!(TBL,SEX,AGE)=T!(TBL,SEX,AGE)+L2!(TBL,SEX,I)
    NEXT I
NEXT AGE
FOR AGE=MIN TO 105
    IF L1!(TBL,SEX,AGE)<=0 THEN E!(TBL,SEX,AGE)=0: GOTO N33
    E!(TBL,SEX,AGE)=T!(TBL,SEX,AGE)/L1!(TBL,SEX,AGE)
NEXT AGE
N33:
   RETURN
   FOR SEX=1 TO 2
       FOR AGE=MIN TO 105
           D!(TBL,SEX,ATB)=0
       NEXT AGE
```

```
*************************************************************************
*                                                                       *
*                                                                       *
*            CALCULATION OF CONDITIONAL RISK COEFFICIENTS               *
*                                                                       *
*************************************************************************

' 2. RADRISK
' **********
RADRISK:
'This routine calculates probability of death at a particular age due natural
'causes and radiation exposure. Quantities used are:
'
'          Q(TBL,SEX,AGE): total probability of death at a particular age
'           QRAD(SEX,AGE): probabilility of radiation-induced death at age
'    RCAN(SEX,AGE,CANCER): probability of radiation-induced cancer
'  CANCER(SEX,AGE,CANCER): Canadian background cancer rates
'                    DREF: dose rate effect factor
'
'           RELATIVE RISK = 1 + f(d) g(B)
IF DREF!=0 THEN DREF!=1
IF MENU=2 OR MENU7=2 THEN GOTO RISK11

FOR ATB=MIN TO 105
    DE!=DOSE!(PERSON,SEX,ATB)/1000
    ON CANCER GOSUB LEUKEMIA, RESPIRATORY, BREAST, DIGESTIVE, OTHER ,CAN6, CAN7
NEXT ATB
GOTO RISK20:

RISK11:
   DE!=DOSE!(PERSON,SEX,AGE1)/1000
   ATB=AGE1
   ON CANCER GOSUB LEUKEMIA, RESPIRATORY, BREAST, DIGESTIVE, OTHER ,CAN6, CAN7
RISK20:
   FOR AGE=0 TO 105
       FOR CNCER=1 TO 5
           BEIR!(SEX,AGE,6)= BEIR!(SEX,AGE,6)+BEIR!(SEX,AGE,CNCER)
           RCAN!(SEX,AGE,6)= RCAN!(SEX,AGE,6)+RCAN!(SEX,AGE,CNCER)
       NEXT CNCER
       Q!(2,SEX,AGE)=Q!(1,SEX,AGE)+RCAN!(SEX,AGE,6)
   NEXT AGE
   RETURN
CAN6: '******ALL CANCERS******
   FOR CNCER=1 TO 5
       ON CNCER GOSUB LEUKEMIA, RESPIRATORY, BREAST, DIGESTIVE, OTHER
   NEXT CNCER
   RETURN
CAN7: '*******NON-LEUKEMIA*******
   FOR CNCER=1 TO 4
       ON CNCER GOSUB RESPIRATORY, BREAST, DIGESTIVE, OTHER
   NEXT CNCER
   RETURN
LEUKEMIA: '******LEUKEMIA******
   A1!=0.243: A2!=0.271
   B1!=4.885: B2!=3.380: B3!=2.367: B4!=1.638
   F!=A1!*DE!+A2!*DE!^2
   LOCATE 22,1: PRINT "CALCULATING LEUKEMIA
   FOR AGE=ATB+2 TO 105
       TS=AGE-ATB
       IF ATB>20 THEN GOTO PROMPT200
```

```
      IF TS<=15            THEN G!=EXP(B1!): GOTO PROMPT210
      IF TS>15 AND TS<=25 THEN G!=EXP(B2!): GOTO PROMPT210
      G!=1: GOTO PROMPT210
   PROMPT200:                          'age > 20
      IF TS<=25            THEN G!=EXP(B3!): GOTO PROMPT210
      IF TS>25 AND TS<=30 THEN G!=EXP(B4!): GOTO PROMPT210
      G!=1
   PROMPT210:
         BEIR!(SEX,AGE,1)=BEIR!(SEX,AGE,1)+F!*G!
         RCAN!(SEX,AGE,1)=RCAN!(SEX,AGE,1)+CANCER!(SEX,AGE,1)*(F!*G!)
   NEXT AGE
   RETURN
RESPIRATORY: '******RESPIRATORY CANCER******
   A1!=0.636
   B1!=-1.437: B2!=0.711
   F!=A1!*DE!
   LOCATE 22,1: PRINT "CALCULATING          RESPIRATORY
   IF ATB+10>105 THEN RETURN
   FOR AGE=ATB+10 TO 105
      TS=AGE-ATB
      IF SEX=1 THEN G!=EXP(B1!*LOG(TS/20))_
               ELSE G!=EXP(B1!*LOG(TS/20)+B2!)
      BEIR!(SEX,AGE,2)=BEIR!(SEX,AGE,2)+F!*G!/DREF!
      RCAN!(SEX,AGE,2)=RCAN!(SEX,AGE,2)+CANCER!(SEX,AGE,2)*(F!*G!/DREF!)
   NEXT AGE
   RETURN
BREAST: '******BREAST CANCER******
   A1!=1.220
   B1!=1.385: B2!=-0.104: B3!=-2.212: B4!=-0.0628
   F!=A1!*DE!
   IF SEX=1 THEN RETURN
   LOCATE 22,1: PRINT "CALCULATING                          BREAST       "
   FOR AGE=ATB+1 TO 105
      TS=AGE-ATB
      IF ATB<=15  THEN G!=EXP(B1!+B2!*LOG(TS/20)+B3!*(LOG(TS/20))^2)
      IF ATB>15 THEN G!=EXP(B2!*LOG(TS/20)+B3!*(LOG(TS/20))^2+B4!*(ATB-15))
      BEIR!(SEX,AGE,3)= BEIR!(SEX,AGE,3)+F!*G!/DREF!
      RCAN!(SEX,AGE,3)  = RCAN!(SEX,AGE,3)+CANCER!(SEX,AGE,3)*(F!*G!/DREF!)
   NEXT AGE
   RETURN
DIGESTIVE: '******DIGESTIVE CANCER******
   A1!=0.809
   B1!=0.553: B2!=-0.198
   F!=A1!*DE!
   LOCATE 22,1: PRINT "CALCULATING                                  DIGESTIVE
   IF ATB+10>105 THEN RETURN
   IF SEX=1 THEN GOTO MALE ELSE GOTO FEMALE
MALE:
   FOR AGE=ATB+10 TO 105
      IF ATB<=25             THEN G!=1: GOTO PRMT10
      IF ATB>25 AND ATB<=35 THEN G!=EXP(B2!*(ATB-25)): GOTO PRMT10
      IF ATB>35              THEN G!=EXP(B2!*10): GOTO PRMT10
      COLOR 12: PRINT "ERROR IN DIGESTIVE CANCER": CALL CONTINUE
    PRMT10:
      BEIR!(1,AGE,4)=BEIR!(1,AGE,4)+F!*G!/DREF!
      RCAN!(1,AGE,4)=RCAN!(1,AGE,4)+CANCER!(1,AGE,4)*(F!*G!/DREF!)
    NEXT AGE
RETURN
FEMALE:
    FOR AGE=ATB+10 TO 105
```

```
        IF ATB<=25              THEN G!=EXP(B1!): GOTO PRMT15
        IF ATB>25 AND ATB<=35 THEN G!=EXP(B1!+B2!*(ATB-25)): GOTO PRMT15
        IF ATB>35               THEN G!=EXP(B1!+B2!*10): GOTO PRMT15
        COLOR 12: PRINT "ERROR IN DIGESTIVE CANCER": CALL CONTINUE
     PRMT15:
        BEIR!(2,AGE,4)=BEIR!(2,AGE,4)+F!*G!/DREF!
        RCAN!(2,AGE,4)=RCAN!(2,AGE,4)+CANCER!(2,AGE,4)*(F!*G!/DREF!)
     NEXT AGE
     RETURN
OTHER: '******OTHER CANCERS******
   A1!=1.220: B1!=-0.0464
   F!=A1!*DE!
   LOCATE 22,1: PRINT "CALCULATING                                                    OTHER
   IF ATB+10>105 THEN RETURN
   FOR AGE=ATB+10 TO 105
        IF ATB<=10 THEN G!=1: GOTO PRMT20
        IF ATB>10  THEN G!=EXP(B1!*(ATB-10)): GOTO PRMT20
        COLOR 12: PRINT "ERROR IN OTHER CANCERS": CALL CONTINUE
   PRMT20:
        BEIR!(SEX,AGE,5)=BEIR!(SEX,AGE,5)+F!*G!
        RCAN!(SEX,AGE,5)=RCAN!(SEX,AGE,5)+CANCER!(SEX,AGE,5)*(F!*G!/DREF!)
   NEXT AGE
   LOCATE 22,1: PRINT "CALCULATING
   RETURN
```

```
*************************************************************************************
*                                                                                   *
*                                                                                   *
*           CALCULATION OF LIFETIME RISK ATTRIBUTES                                 *
*                                                                                   *
*************************************************************************************

CONTDOSE: '******ANNUAL CONTINUOUS EXPOSURE********
CLS: COLOR 15: PRINT "                    ANNUAL CONTINUOUS EXPOSUE
     COLOR 14: PRINT
               PRINT "                    EXCESS LIFETIME RISK  (ELR)
               PRINT "                    LIFETIME ATTRIBUTABE PROBABILITY OF DEATH
               PRINT "                    YEARS OF LIFELOST (Y)
               PRINT "                    YEARS OF LIFE LOST PER EXCESS DEATH
               PRINT "                    LOSS OF LIFE EXPECTANCY  (LLE)
PRINT
PRINT
'CALL WHATCANCER
CANCER=6
PRINT
COLOR 15: INPUT "DREF: ", DREF!
RANGE2=1
DIM DYNAMIC ELR!(RANGE,RANGE2,1:2,1:6), R!(RANGE,RANGE2,1:2,1:6), Y!(RANGE,RANGE
           YMEAN!(RANGE,RANGE2,1:2,1:6), LLE!(RANGE,RANGE2,1:2),_
           AGEMEAN!(RANGE,RANGE2,1:2)
 FOR PERSON=1 TO RANGE
    CLS: LOCATE 13,25: COLOR 14: PRINT "CALCULATING EXCESS RISKS"
    GOSUB NULLBEIR
    GOSUB NULLLIFETAB
    GOSUB NULLCANCER
    FOR SEX=SEXMIN TO SEXMAX
        DTOT!=0
        LOCATE 1,1: PRINT "DOSE, SEX: " PERSON SEX
        GOSUB RADRISK
        TBL=1: GOSUB LIFETAB
        TBL=2: GOSUB LIFETAB

        FOR AGE2=ATBMIN TO 105
            FOR CNCER=1 TO 5
                S1!=S!(1,SEX,AGE2)/S!(1,SEX,ATBMIN)
                S2!=S!(2,SEX,AGE2)/S!(2,SEX,ATBMIN)
                RISKa!=S2!*RCAN!(SEX,AGE2,CNCER)
                RISKb!=RISKa!*E!(2,SEX,AGE2)
                CORRTERMa!=CANCER!(SEX,AGE2,CNCER)*(S1!-S2!)
                CORRTERMb!=CANCER!(SEX,AGE2,CNCER)*(S1!*E!(1,SEX,AGE2)-S2!*E!(2,
                ELR!(PERSON,PERSON2,SEX,6)=      ELR!(PERSON,PERSON2,SEX,6)+(RISK
                R!(PERSON,PERSON2,SEX,CNCER)=    R!(PERSON,PERSON2,SEX,CNCER)+RIS
                R!(PERSON,PERSON2,SEX,6)=        R!(PERSON,PERSON2,SEX,6)+RISKa!
                Y!(PERSON,PERSON2,SEX,CNCER)=    Y!(PERSON,PERSON2,SEX,CNCER)+(RI
                DD!(SEX,AGE2)=DD!(SEX,AGE2)+RISKa!
               DTOT!=DTOT!+RISKa!
             NEXT CNCER
              DD!(SEX,AGE2)=DD!(SEX,AGE2)+E!(2,SEX,AGE2)/100000
        NEXT AGE2
        LLE!(PERSON,PERSON2,SEX)= E!(1,SEX,ATBMIN)-E!(2,SEX,ATBMIN)
        Y!(PERSON,PERSON2,SEX,6)= E!(1,SEX,ATBMIN)-E!(2,SEX,ATBMIN)
        FOR CNCER=1 TO 6
            IF R!(PERSON,PERSON2,SEX,CNCER) > 0 THEN YMEAN!(PERSON,PERSON2,SEX,C
        NEXT CNCER
```

```
FOR AGE2=ATBMIN TO 105
    AGEMEAN!(PERSON,PERSON2,SEX)=AGEMEAN!(PERSON,PERSON2,SEX)+AGE2*DD!(1,AGE2)/D
NEXT AGE2
'EMEAN!=E!(2,SEX,AGEMEAN!)
'LOCATE 17,1: PRINT "MEAN AGE: " AGEMEAN!
'PRINT "AGEMEAN2: " E!(2,SEX,0)
'PRINT "YMEAN: " YMEAN!(PERSON,PERSON2,SEX,6)
'PRINT "EMEAN: " EMEAN!
'CALL CONTINUE3
    NEXT SEX
    SEX=SEXMIN
NEXT PERSON

CLS
RETURN

INSTDOSE: '******INSTANTANEOUS EXPOSURE******
CLS: COLOR 15: PRINT "                    SINGLE INSTANAEOUS EXPOSUE
     COLOR 14: PRINT
               PRINT "                    EXCESS LIFETIME RISK  (ELR)
               PRINT "                    LIFETIME ATTRIBUTABE PROBABILITY OF DEATH
               PRINT "                    YEARS OF LIFELOST (Y)
               PRINT "                    YEARS OF LIFE LOST PER EXCESS DEATH
               PRINT "                    LOSS OF LIFE EXPECTANCY  (LLE)
CLS: CALL WHATCANCER
PRINT
PRINT
COLOR 15: INPUT "DREF: ", DREF!
DIM DYNAMIC ELR!(RANGE,RANGE2,1:2,1:6), R!(RANGE,RANGE2,1:2,1:6), Y!(RANGE,RANGE
            YMEAN!(RANGE,RANGE2,1:2,1:6), LLE!(RANGE,RANGE2,1:2),_
            AGEMEAN!(RANGE,RANGE2,1:2)

IF RANGE2>50 THEN RANGE=1
FOR PERSON=1 TO RANGE
    CLS: LOCATE 13,25: COLOR 15: PRINT "CALCULATING EXCESS RISKS"
    COLOR 14
    AGE1=ATBMIN
    FOR PERSON2=1 TO RANGE2
        GOSUB NULLBEIR
        GOSUB NULLLIFETAB
        GOSUB NULLCANCER
        FOR SEX=SEXMIN TO SEXMAX
        DTOT!=0
            LOCATE 1,1: PRINT "DOSE, AGE, SEX: " PERSON AGE1 SEX
            GOSUB RADRISK
            TBL=1: GOSUB LIFETAB
            TBL=2: GOSUB LIFETAB
            IF RANGE2>50 THEN PERSON=0
            FOR AGE2=AGE1 TO 105
                FOR CNCER=1 TO 5
                    IF S!(1,SEX,AGE1)<>0 THEN S1!=S!(1,SEX,AGE2)/S!(1,SEX,AGE1):
                                              S2!=S!(2,SEX,AGE2)/S!(2,SEX,AGE1)
                    RISKa!=S2!*RCAN!(SEX,AGE2,CNCER)
                    RISKb!=RISKa!*E!(2,SEX,AGE2)
                    CORRTERMa!=CANCER!(SEX,AGE2,CNCER)*(S1!-S2!)
                    CORRTERMb!=CANCER!(SEX,AGE2,CNCER)*(S1!*E!(1,SEX,AGE2)-S2!*E
                    IF (RISKa!-CORRTERMa!)<0 THEN GOTO BA10
                    ELR!(PERSON,PERSON2,SEX,CNCER)= ELR!(PERSON,PERSON2,SEX,CNCE
                    ELR!(PERSON,PERSON2,SEX,6)=     ELR!(PERSON,PERSON2,SEX,6)+(
                    R!(PERSON,PERSON2,SEX,CNCER)=   R!(PERSON,PERSON2,SEX,CNCER)
```

```
                    R!(PERSON,PERSON2,SEX,6)=       R!(PERSON,PERSON2,SEX,6)+RIS
                    Y!(PERSON,PERSON2,SEX,CNCER)=   Y!(PERSON,PERSON2,SEX,CNCER)
                    DD!(SEX,AGE2)=DD!(SEX,AGE2)+RISKa!
                    DTOT!=DTOT!+RISKa!
'BA10:
          NEXT CNCER
'LOCATE 18,1: PRINT 'AGE: " AGE2
'LOCATE 19,1: PRINT 'RCAN: " RCAN!(SEX,AGE2,6);: PRINT "CANCER: " 6
'LOCATE 20,1: PRINT 'RISK: " RISKa!;: PRINT "CORRTERM: " CORRTERMa!
'LOCATE 21,1: PRINT 'ELR!: " ELR!(PERSON,PERSON2,SEX,6)
'CALL CONTINUE3
          NEXT AGE2
          LLE!(PERSON,PERSON2,SEX)= E!(1,SEX,ATBMIN)-E!(2,SEX,ATBMIN)
          Y!(PERSON,PERSON2,SEX,6)= E!(1,SEX,ATBMIN)-E!(2,SEX,ATBMIN)
          FOR CNCER=1 TO 6
              IF R!(PERSON,PERSON2,SEX,CNCER) > 0 THEN YMEAN!(PERSON,PERSON2,S
              IF Y!(PERSON,PERSON2,SEX,CNCER)<0 THEN YMEAN!(PERSON,PERSON2,SEX
          NEXT CNCER

'LOCATE 15,1: PRINT 'PERSON2: " PERSON2
'PRINT "YMEAN: " YMEAN!(PERSON,PERSON2,SEX,6)
'CALL CONTINUE3
FOR AGE2=ATBMIN TO 105
    IF DTOT!<>0 THEN AGEMEAN!(PERSON,PERSON2,SEX)=AGEMEAN!(PERSON,PERSON2,SEX)+A
NEXT AGE2

          IF RANGE2>50 THEN PERSON=1
       NEXT SEX
       AGE1=AGE1+1
       SEX=SEXMIN
    NEXT PERSON2
NEXT PERSON
CLS
RETURN
```

```
*************************************************************************
*                                                                       *
*                  CALULATION OF POPULATION AVERAGE                     *
*                                                                       *
*                                                                       *
*************************************************************************
AVERAGE:
'IF HAVEAVERAGE<>1 THEN GOTO EXCESS
CLS: COLOR 15: PRINT "POPULATION-WEIGHTED AVERAGE"
PRINT
PRINT
COLOR 14: PRINT "POPULATION TYPE:
PRINT
PRINT "       (1) PUBLIC  STATIONARY POPULATION (ALL AGES)
PRINT "       (2) WORKING STATIONARY POPULATION (18-65 yrs)
PRINT "       (3) USER DEFINED POPULATION
PRINT
COLOR 15: INPUT "ENTER SELECTION BY NUMBER: ", MENU20
IF MENU20<0 OR MENU20>3 THEN GOTO AVERAGE

ERASE POP!, TOTPOP!, AVELR!, AVR!, AVY!, AVYMEAN!, AVLLE!, AVEAGE!
DIM DYNAMIC POP!(1:2,106), TOTPOP!(1:2), AVELR!(1:2,1:6), AVR!(1:2,1:6),_
            AVY!(1:2,1:6), AVYMEAN!(1:2,1:6), AVLLE!(1:2), AVEAGE!(1:2)

ON MENU20 GOSUB PUBLIC, WORKING, USER2
IF RANGE2>50 THEN RANGE=1
PERSON=0
    FOR SEX=SEXMIN TO SEXMAX
        AGE1=ATBMIN
        FOR PERSON2=1 TO RANGE2
            FOR CNCER=1 TO 6
                AVELR!(SEX,CNCER)=AVELR!(SEX,CNCER)+ELR!(PERSON,PERSON2,SEX,CNCE
                AVR!(SEX,CNCER)=AVR!(SEX,CNCER)+R!(PERSON,PERSON2,SEX,CNCER)*POP
                AVY!(SEX,CNCER)=AVY!(SEX,CNCER)+Y!(PERSON,PERSON2,SEX,CNCER)*POP
                AVYMEAN!(SEX,CNCER)=AVYMEAN!(SEX,CNCER)+YMEAN!(PERSON,PERSON2,SE
            NEXT CNCER
            AVLLE!(SEX)= AVLLE!(SEX)+LLE!(PERSON,PERSON2,SEX)*POP!(SEX,AGE1)/TOT
            AVEAGE!(SEX)=AVEAGE!(SEX)+AGEMEAN!(PERSON,PERSON2,SEX)*POP!(SEX,AGE1
            AGE1=AGE1+1
        NEXT PERSON2
LOCATE 15,1:
'PRINT "AGE: " AGE1 SEX
'PRINT "POPULATION: " POP!(SEX,AGE1)
'PRINT "TOTAL POP:  " TOTPOP!(SEX)
'PRINT "ELR: " ELR!(PERSON,PERSON2,SEX,6)
'PRINT "AVERAGE ELR: " AVELR!(SEX,6)
'CALL CONTINUE3
     NEXT SEX
     SEX=SEXMIN
GOTO PRNTEXCESS

PUBLIC: '******PUBLIC STATIONARY POPULATION******
    FOR SEX=SEXMIN TO SEXMAX
        FOR AGE=1 TO 85
            POP!(SEX,AGE)=    L1!(1,SEX,AGE)
            TOTPOP!(SEX)= TOTPOP!(SEX)+L1!(1,SEX,AGE)
       NEXT AGE
    NEXT SEX
RETURN
```

```
WORKING: '******WORKING STATIONARY POPULATION******
    FOR SEX=SEXMIN TO SEXMAX
        FOR AGE=18 TO 65
            POP!(SEX,AGE)=     L1!(1,SEX,AGE)
            TOTPOP!(SEX)= TOTPOP!(SEX)+L1!(1,SEX,AGE)
        NEXT AGE
    NEXT SEX
RETURN

USER2: '******USER DEFINED POPULATION******'
    CLS: COLOR 15: PRINT "USER DEFINED POPULATION
    COLOR 14: PRINT "POPULATION MUST BE READ FROM INPUT FILE
    COLOR 15: PRINT "REFER TO USER MANUAL FOR DETAILS ON FORMAT
    CALL CONTINUE3
    PRINT
    PRINT
    COLOR 15: INPUT "INPUT FILE:", FILE$
    OPEN "I",#1,FILE$
'     INPUT# 1, AGERANGE
    FOR I=1 TO 105
        INPUT# 1, AGE, POP!(1,AGE), POP!(2,AGE)
        TOTPOP!(1)=TOTPOP!(1)+POP!(1,AGE)
        TOTPOP!(2)=TOTPOP!(2)+POP!(2,AGE)
    NEXT I
CLOSE #1
RETURN
```

```
'       ************************************************************************"
'       **                                                                    **"
'       **                FILEPRINT1: RADRISK (version 1.1)                  **"
'       **                                                                    **"
'       **         CONTAINS ROUTINES FOR PRINTING TO SCREEN,                  **"
'       *          PRINTING TO PRINTER, AND WRITING TO FILE                   **"
'       **                                                                    **"
'       **                     L. RASMUSSEN 90.06.25                          **"
'       ************************************************************************"

' 1. PRINT EXCESS RISKS
' *********************
PRNTEXCESS:
CLS: LOCATE 5,1: COLOR 14: PRINT "OPTIONS:
COLOR 15
PRINT "   (1) ALL RISK ATTRIBUTES (BY SEX AND ATB)
PRINT "   (2) AGE-DISTRIBUTION OF DEATH
PRINT "   (3) EXCESS LIFETIME RISK (BY SEX, ATB, AND CANCER SITE)
PRINT "   (4) LIFETIME ATTRIBUTABLE PROBABILITY OF DEATH (BY SEX, ATB, AND CANCER
PRINT "   (5) YEARS OF LIFE LOST (BY SEX, ATB, AND CANCER SITE)
PRINT "   (6) YEARS OF LIFE LOST PER EXCESS DEATH
PRINT "   (7) RETURN TO ANALYSIS
PRINT "   (8) RETURN TO MENU
COLOR 14: INPUT "ENTER OPTION BY NUMBER:", MENU15
```