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Thesis

Lung Cancer Risks to Canadians from Residential Radon Exposure

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Abstract

A causal link between radon exposure and lung cancer was previously established through numerous epidemiological studies of miners and residential occupants exposed to radon gas. Although the health detriment to Canadians from residential radon exposure has been estimated in earlier assessments, a comprehensive radon survey by Health Canada in 2011 was the first to sample residences from every health region in the country. Further, this survey yielded higher concentration measurements than previous surveys, with an arithmetic average concentration about twice that of an older survey which was the basis for many of the previous Canadian radon risk assessments. Two exposure–response models from the US NRC were selected for this thesis, along with seasonal adjustment factors for the survey data to compute expected value, rather than conservative, risk estimates. Population-based (population attributable risk, PAR; excess lifetime risk ratio, ELRR; and life-years lost LYL) and individual-based (ELRR and LYL) indices are used to summarize the health detriment. Mean estimates of ELRR, PAR, and LYL for the Canadian population are estimated in the range of 0.31 - 0.48, 0.19 - 0.26, and 0.19 - 0.31, respectively, depending on the model used. Point estimates are also provided for individual provinces and some results are stratified by risk factors, such as smoking history and floor level of residency. A number of sources of uncertainty in the results are identified, and some are incorporated in a two-dimensional uncertainty analysis using Monte Carlo methods.

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Chapter 1

Introduction and Background

Strong evidence links radon exposure to increased lung cancer risk. Epidemiological studies of underground miners exposed to high concentrations of radon demonstrate a higher incidence of lung cancer – an association corroborated by case-control studies and a developing understanding of credible biologic mechanisms. Based on the miner studies, radon has been classified as a human carcinogen [1], and is the second greatest cause of lung cancer after smoking [2], [3]. Radon is also the greatest source of natural radiological exposure to humans, accounting for approximately 50% of the dose received from natural sources [4]. Radon-222, the most significant isotope of radon with respect to lung cancer risk, is a transient constituent of the uranium-238 decay chain. Consequently, radon gas is generally associated with sections of the earth's crust containing uranium. Because it is chemically inert, radon gas can move relatively unimpeded through rocks and soil; a 3.82d half-life provides sufficient time for some of the radon-222 formed near residences to seep through foundation cracks, exposing the occupants. Some building materials may also produce radon gas. If inhaled, radon-222 and its progeny may decay in the lungs, where the resultant radiation can damage the DNA of regenerative lung tissue cells, potentially culminating in lung cancer.

Models have been developed based on the epidemiological studies in an attempt to characterize the relationship between radon exposure and lung cancer. By applying the radon exposures estimated for a population to these models, radon risk assessments estimate the risk or expected health detriment for the

population. These estimates can be used by policy-makers for a number of purposes: to understand how the risk posed by radon exposure compares to other risks facing the population; to determine a maximum acceptable level of exposure; and to form policies or recommendations directed at reducing exposures to acceptable levels.

Atomic Notation

In this thesis, elements are generally referred to in two ways:

1) the element name followed by the atomic mass number (the total number of neutrons and protons in the nucleus) (e.g. radon-222);

2) the element chemical symbol preceded by a superscript indicating the atomic mass number and a subscript indicating the atomic number (the number of protons in the nucleus):

$${}^{A}_{Z}\mathrm{X}$$

Where: Z = Atomic Number (number of protons in the nucleus)

X = Chemical Symbol

A = Atomic Mass Number (total number of neutrons and protons in the nucleus)

The number of neutrons is given by N = A - Z

In the latter format it is common for the atomic number, Z, to be dropped since this is already known from the element symbol (e.g. ${}^{222}_{86}\mathrm{Rn}$ is simplified to ${}^{222}\mathrm{Rn}$).

1.1 Radon and Radon Progeny

Radon-222, which has a half life of 3.8 days, is formed directly from the decay of radium-226. Radium-226 is itself formed from U-238 through a series of decays that have a cumulative half life of billions of years. As shown in Figure 1.1, when Rn-222 decays, its progeny undergo a series of transformations within hours, consisting of two alpha decays and two beta decays, finally resulting in relatively stable lead-210. While the beta emissions of this decay sequence can damage living cells (by ionization), it is the alpha particles, generally 20 times more damaging than beta particles [5], that contribute the most risk to exposed, regenerative cells.

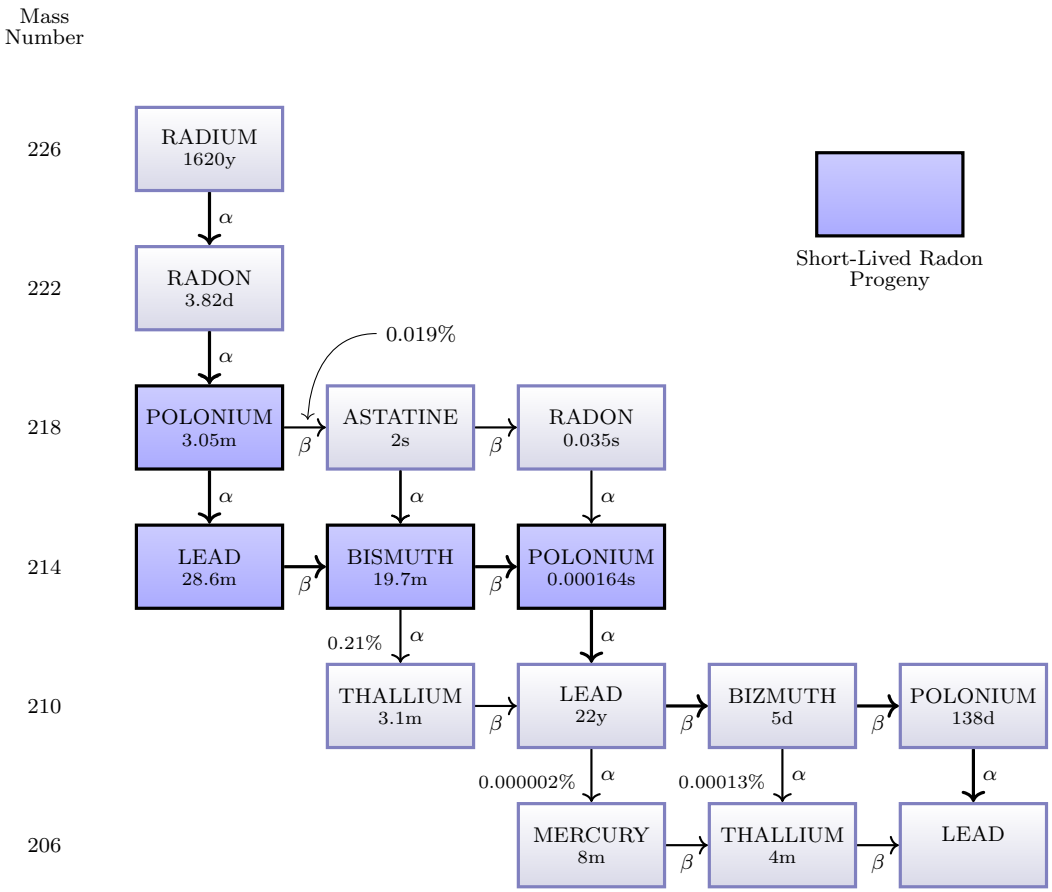

Figure 1.1: The radon decay chain: darker rectangles indicate the short-lived radon progeny which contribute most to lung cancer risk; the thicker arrows show the most probable decay sequence from ^{226}Ra to ^{206}Pb. Source: prepared by author based on charts of nuclides.

Throughout this thesis, the series of short-lived radon progeny, which are highlighted in Figure 1.1, will frequently be referred to as *radon progeny*, whereas the ^{222}Rn will frequently be referred to simply as *radon*.

1.2 Exposure to Radon

The main sources of radon in residences are rocks and soil, with secondary contributions from building materials which happen to contain ^{226}Ra, and well water. Figure 1.2 illustrates the main concepts and sequences of steps associated with the process through which humans receive radiological doses from radon gas in residences. For radon originating from rocks and soil, the main pathway into the residence is through

cracks in the foundation and walls. Outdoor levels of radon gas may also contribute to residential concentrations through windows or air exchange systems, though ventilation is generally beneficial in reducing radon exposure since indoor concentrations are normally higher than outdoors. Once radon enters the residence, the occupants may be exposed to the gas and its short-lived progeny. Residential exposure occurs when the occupants are inside their residences, estimated to be approximately 70% of time for average members of the population [6]. Radon exposures are therefore multiplied by an *Occupancy Factor* to account for the fraction of time exposed.

The properties that allow radon to accumulate in residences (e.g. inert gas, 3.82d half life) also imply that most radon breathed into the lungs is expelled. However, the unstable radon progeny are solids with electrostatic charge and may attach to airborne particulates and aerosols. These may adhere to the surface of the lungs following inhalation, resulting in a reduced chance of being cleared before decaying. Consequentially, it is the progeny, rather than the radon itself, which exert the greatest dose associated with radon exposure. Therefore, for a given radon concentration, it is important to know what concentration of progeny is implied.

These proportions are related by the *Equilibrium Factor*, F. If an enclosed volume was constantly supplied with radon gas, the concentration of the short-lived progeny would increase until they are in secular equilibrium — they are decaying at the same rate that they are created (i.e. the same rate at which the ^{222}Rn is decaying). In such a scenario, the relative contributions to radioactivity from radon and from its progeny at steady-state are known based on the type and energy of the emissions from the radionuclides. In practice, the steady-state proportions are different from the situation described above because radon progeny can be removed from the pool by means other than decay, which includes attachment of the progeny to walls, floors, or other surfaces, as well as deposition of unattached progeny. These phenomena reduce the concentration of radon progeny but not the concentration of radon gas, thereby reducing the equilibrium ratio. The *Equilibrium Factor* is the ratio of the radon progeny activity to radon activity in the scenario of interest. For typical homes, the Equilibrium Factor has been estimated to be approximately 40% by Hopke et al. [7]. Figure 1.2 indicates an indoor concentration of radon gas and radon progeny with some of the radon progeny

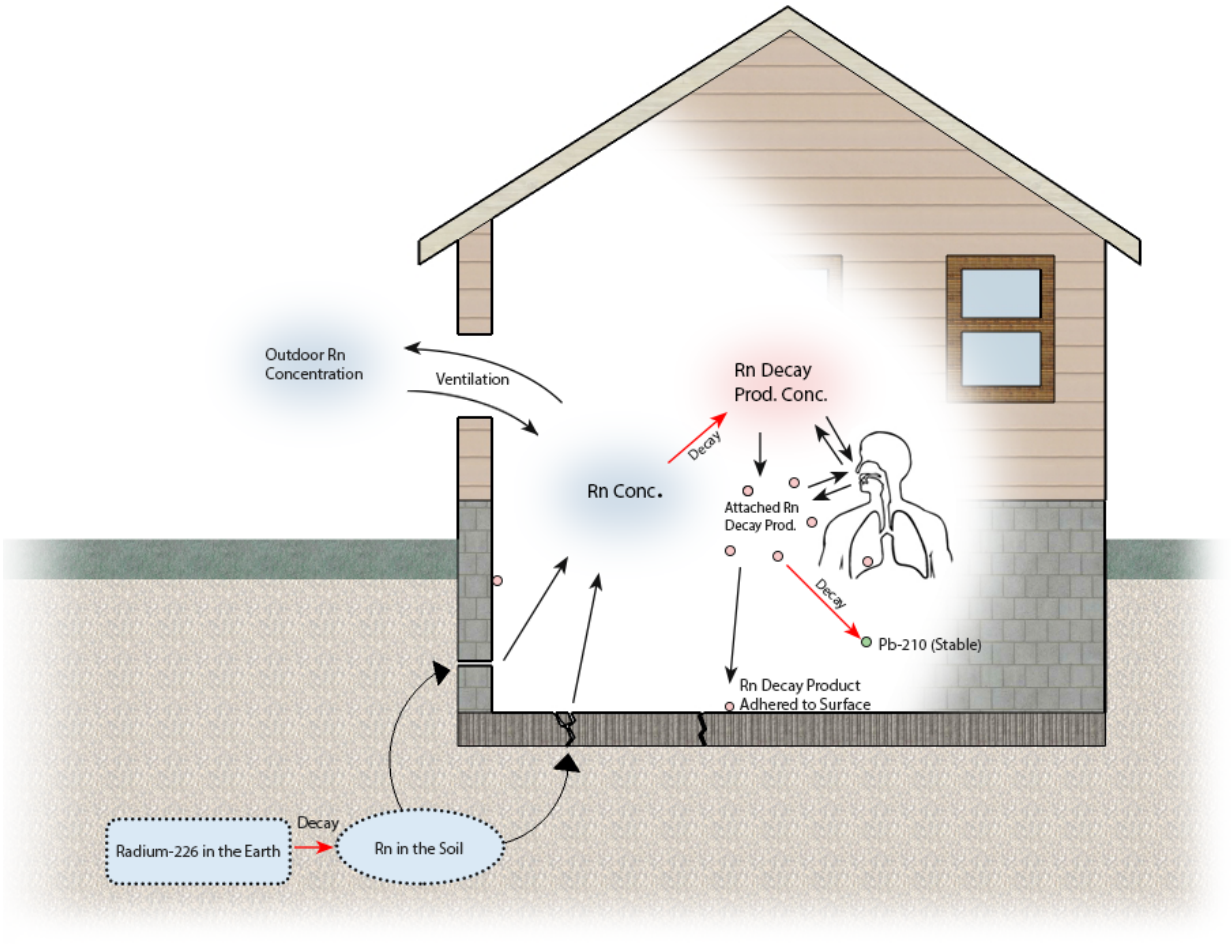

Figure 1.2: Some of the main factors related to residential exposure to radon are shown: 1) ingress of radon gas into the residence; 2) an indoor mixture of radon and radon progeny in equilibrium; 3) attachment of some fraction of the radon progeny to airborne particulates; 4) inhalation and potential adherence to the lungs of radon progeny. Source: prepared by author based on general concepts.

becoming attached to airborne particles and eventually being inhaled into the lungs where they may attach to the lining of the lungs.

1.2.1 Alpha Decay

Alpha decay, the most damaging form of ionizing radiation, is the ejection of an alpha particle from an unstable nucleus. The alpha particle consists of two protons and two neutrons (a helium nucleus). For example, radon-222 alpha decays to polonium-218:

$$^{222}\mathrm{Rn} \quad \rightarrow \quad ^{218}\mathrm{Po} \quad + \quad \alpha$$

Or, equivalently:

$$^{222}\mathrm{Rn} \quad \rightarrow \quad ^{218}\mathrm{Po} \quad + \quad ^{4}_{2}\mathrm{He}$$

Due to their mass and positive electrostatic charge, the trajectory of alpha particles is quickly attenuated by surrounding material. As an example, the dead, outer layer of the human skin epidermis absorbs alpha particles before they reach living tissue, hence alpha particles are only of biological concern when the source of emission is inhaled or otherwise ingested. In this case, the emitted alpha particles can, with low probability, cause large-scale damage to the DNA of a lung tissue cell and the generative cell may pass this damage on to new generations.

1.2.2 Concentration

When referring to the concentration of a radioactive nuclide, it is normally the activity concentration that is of interest, rather than the mass concentration. The SI unit for rate of radioactive decay is the Becquerel, denoted *Bq*, which is one decay per second. Radioactive concentration is measured in $\mathrm{Bq/m^3}$. Clearly, occupants of homes that have higher radon concentrations receive higher doses of radiation from the decay of radon and its progeny, all other factors being equal. Many of the models based on studies of miners express concentration in Working Levels (WL), which is a traditional measure from the mining industry. Since the survey used in this thesis measured concentrations in $\mathrm{Bq/m^3}$, exposure concentration is converted to WLM/y, as detailed in Appendix A, when using these models.

Thoron - A Radon Isotope

The effects of thoron can be considered in studies of lung cancer risk from radon, particularly in measurment of radon concentrations. Thoron (^{220}Rn) is an isotope of radon, but is part of the decay chain originating from thorium, shown in Appendix B. Like radon, thoron is a gaseous nuclide with a series of progeny which are alpha particle emitters. Unlike radon, thoron has a much shorter half-life of 55.6 s, while the second and third nuclides of its decay series have half-lives on the order of hours. These temporal differences (compared to ^{222}Rn) have significant implications when comparing risks from radon and thoron. First, the short half-life of thoron, resulting in a short mean diffusion length in air of 4.4 cm (Meisenberg et al.) [8], precludes ingress from sources other than those in very close proximity to the dwelling, such as building materials or soil and rocks. Second, the concentration profile for thoron would be greatest near the walls or floors of the residence, dropping off steeply with distance from the source, whereas the ^{222}Rn concentration is well-mixed, generally in equilibrium with the air movement patterns of the residence. Third, for equivalent inhaled concentrations of radon and thoron, the thoron progeny commit a smaller dose to the lungs, largely due to the longer half-lives of the progeny — in particular the almost 11-hour half-life of lead-212 — which make them more likely to be cleared from the lungs before decaying (BEIR-VI) [6]. For these reasons, doses from thoron in homes are generally low compared to those from radon, although very high concentrations have been observed in some dwellings which would result in significant dose. Shang et al. [9] found thoron could contribute significantly to the inhaled dose in homes made from materials such as adobe or mudbricks. Building materials used for construction of homes in Canada are unlikely to contribute high levels of thoron. A 2012 Health Canada study based on a survey by Chen et al. [10] of 4000 simultaneous measurements of radon and thoron in Canadian metropolitan homes found that, on average, thoron contributes about 3% of the radiation dose due to indoor radon and thoron exposure. The highest thoron measurement recorded in this survey was 210 Bq/m^3.

Radon and thoron concentrations have been found to be virtually independent parameters with

weak correlations observed [4], [11]. This is intuitive since there is no intersection of the uranium-238 and thoron-232 decay chains from which radon and thoron evolve.

1.3 Radon Concentration Measurements and Estimates

An important feature of radon risk assessments is an accurate assessment of the radon concentrations faced by members of the population of interest (i.e. the radon concentrations in the population's residences). Exposures in residences are typically estimated using contemporary measurements over a period between three and twelve months and then assuming a constant concentration in order to project lifetime lung cancer risks. This is most often accomplished with radon detectors situated in the residence, although some interesting alternative techniques have advanced in recent decades, such as assessing the amount of ^{210}Po – one of the radon progeny – embedded in household glass to infer an exposure history (Lively and Ney) [12]. This method is described in Appendix C.2.2.

1.3.1 Detection and Measurement

Although dose to the lungs is mostly attributed to the radon progeny, radon detectors generally measure the ^{222}Rn gas concentration – one reason being that it is more difficult to measure the radon progeny concentration with passive detectors (Bochicchio et al.) [13]. This emphasizes the importance of using a representative equilibrium fraction, F. For risk assessments, it is important that the detectors either discriminate between radon and thoron measurements or prevent the thoron from being measured. Even if the intention is to estimate the combined risk from radon and thoron, any thoron misinterpreted as radon will overstate the risk since the thoron progeny result in significantly less dose per exposure than the radon progeny. Janik et al. [4] note that although the influence of thoron on radon measurements is usually negligible, in some cases the thoron concentration is much higher than that of radon. Tokonami et al. [14] showed that if the ratio of thoron to radon exceeded 1.0, the radon concentration might be overestimated by a factor of about 4% for detectors that do not discriminate between the two isotopes. A short description of

conventional radon detectors and alternative means of exposure estimation is provided in Appendix C.

Indoor radon concentrations can vary greatly over time and tend to be higher during the winter, especially in colder climates where residents keep the windows closed and homes well sealed for insulation, reducing the exchange of outside air which normally has low background radon concentrations. Because lung cancer is believed to develop from long-term radon exposures, average lifetime exposures are of interest. As a result, 12-month measurements are preferred to shorter measurements to obtain unbiased, annual average radon concentrations.

Seasonal adjustment factors have been studied (Bochicchio et al.) [15] and can be applied to measurements shorter than one year. Krewski et al. [16] validated a seasonal variation model developed by Pinel et al. [17] with Canadian radon measurements and also tested a simple multiplicative model for seasonal variation. Not only do radon concentrations vary from season to season, but the level of seasonal variation may differ significantly from house to house, depending on radon source, building characteristics and region of the country. Because seasonal adjustment factors are based on average seasonal radon variations in residences (for a geographic area), application of seasonal adjustment factors to individual measurements introduces uncertainty (Bochicchio et al.) [15].

1.4 Summary of Risk Estimates of Canadian Studies

Previous studies have attributed approximately 10-15% of lung cancer deaths in Canada to residential radon exposure. This is on the order of 2000 deaths per year (rounding down). Until recently, most Canadian studies were based on data from a Canada-wide survey completed in 1978 by the Department of National Health and Welfare (now Health Canada), which measured an average residential radon concentration of 28.3 Bq/m^3 [18]. This survey, which was carried out in 14 Canadian cities, collected "grab samples" in 9999 homes selected by way of randomization within the cities. The samples were obtained by drawing air from the lowest floor of homes through a filter and scintillator cell in series for five minutes. As such, the samples provide concentration measurements for essentially a point in time, which is not a desirable way

to estimate the long term time integral of a variable parameter. More recent surveys, employing better sampling methods, imply Canadians are exposed to greater residential concentrations. In 2010, Chen et al. [19] estimated the average exposure concentration[1] in Canadian residences to be 45.5 Bq/m^3 based on a number of smaller studies from 4 provinces – a result nearly two-fold higher than the 1978 survey. An extensive survey covering all of Canada's health units and providing a more accurate profile of exposures throughout the country was completed by Health Canada in 2011 and resulted in an estimated average exposure concentration of 96 Bq/m^3 (more than three-fold higher than the 1978 survey). Yet another 2012 Health Canada survey of major metropolitan areas (although not a country-wide survey) returned a higher average exposure of 117.2 Bq/m^3. These most recent surveys generally consisted of 3-month measurements conducted during the winter months. Because residential radon concentrations tend to be higher during the heating season, the results from these surveys, if used to represent average annual exposures, may overstate the exposures to Canadians. Table 1.1 shows a summary of selected Canadian radon risk assessments which estimated lung cancer risks for the entire Canadian population.

Exposure Survey Year (Canada-wide Survey)	Study	Survey Average Concentration (Bq/m^3)	Population Lung Cancer Deaths Caused by Radon
1978	Chen 2005 [20]	28.3	12%*
1978	Brand et al. 2005 [21]	28.3	8%
2011	Chen 2012 [22]	97.0	19.4%*

Table 1.1: Summary of Risk Estimates from Canadian Studies. * Indicates a result that was scaled based on the exposure-response model used to permit more direct comparisons of results.

1.5 Motivation and Research Questions

The greater exposure concentrations measured in more recent, and arguably more reliable, surveys have yielded greater risk estimates. Taking into account previous risk estimates and the different exposure-response model structures, the predicted proportion of lung cancers caused by radon exposure using the

[1]Lung cancer risks were not estimated in this study.

2011 cross-Canada survey can be reasonably anticipated to fall in the range from 20% to 40%. The primary research questions which this thesis attempts to answer are as follows:

1. What is a contemporary estimate of the health detriment to Canadians due to lung cancer risks from residential radon exposures?

2. What is the health detriment to subgroups of the population (e.g. smokers, non-smokers) which face different magnitudes of lung cancer risk from radon exposure?

3. What level of uncertainty is associated with the risk estimates?

This thesis sets out to answer these questions by applying credible dose–response models to the data from the 2011 cross-Canada radon exposure survey [23] and identifying the main sources of uncertainty. A main thrust of this work is to produce true value, rather than conservative, estimates of risk.

In addition to the exposure–response models, this thesis will use conventional methods (e.g. life-table methods) to compute estimates of summary population health impact measures that quantify the detriment of radon exposure to Canadians, such as excess lifetime relative risk (ELRR) and life-years lost (LYL). Monte Carlo methods are also used to characterize the uncertainty inherent in some estimates. The health impact measures, in tandem with the uncertainty attached to them, provide a convenient means of comparison with other risk factors, facilitating high-level, integrated decisions that can allocate resources effectively. Understanding the magnitude of the health burden to a population from radon exposure allows governing bodies to determine the necessity or reasonable extent of interventions.

1.6 Organization of Thesis

Chapter 2 presents an overview of exposure–response models. The studies on which the models are derived are introduced, as well as discussion of the relative merits of each. The chapter closes with a short discussion of uncertainties inherent in the models and their inputs.

The 2011 Health Canada Cross-Canada Survey is summarized in Chapter 3. Generation of health risks based on this survey is one of the main motivations of this thesis. The chapter includes a discussion of

the methodology employed by Health Canada in conducting the survey, as well as graphical and statistical summaries of the survey results.

The risk assessment methodology is documented in Chapter 4, which includes the selection of exposure–response models, the sources and use of inputs, seasonal adjustments for radon measurements, life-table methods, indices of lifetime detriment, and the incorporation of uncertainty.

Chapter 5 presents the results obtained with the stated methodology. Point estimates of risk indices are provided at the provincial and national level, and uncertainties of the risk indices are provided for the national results. The dependence of results on smoking history, residence floor level, and use of seasonal adjustments is explored. The chapter concludes by addressing the thesis research questions.

Chapter 2

Exposure–Response Models

The risk of radon-induced lung cancer has been demonstrated in numerous studies, which generally attempt to construct models that quantify the extra risk of lung cancer death per unit exposure in absolute or relative terms. Absolute excess risk is the additional risk of lung cancer due to exposure. When expressed in terms of absolute excess risk, the total risk of lung cancer is the sum of two terms: the background (unexposed) lung cancer risk and the excess lung cancer risk due to radon exposure. The general form for absolute risk models is as follows:

$$Risk_{Exposed} \quad = \quad Risk_{Background} \quad + \quad Risk_{Excess} \tag{2.1}$$

Relative risk is the ratio of the probability of incurring a consequence in exposed conditions to the risk incurred in unexposed conditions. More specific to this thesis, it is the ratio of the probability of contracting lung cancer for a group exposed to a unit of radon exposure to the probability of contracting lung cancer for an unexposed group. Relative risk models express risk as the product of the background lung cancer risk and the relative lung cancer risk posed by the exposure:

$$Risk_{Exposed} \quad = \quad Risk_{Background} \quad \times \quad RR, \tag{2.2}$$

where RR is the relative risk.

Relative risk models imply that for a given exposure, the increase in risk due to that exposure is proportional to the background risk. Although early models, such as one proposed by Harley [24] in 1981, specified absolute excess risk, it was subsequently determined that models of relative risk were more appropriate for radon exposure [6], [25].

Relative risk models are Linear No-Threshold (LNT) models, which specify that the increase in risk from each additional unit of exposure is the same at all exposure levels from 0 to the limit of the model's applicable range. Regulators and policy-makers responsible for limiting health risks from radiation generally subscribe to the LNT model. Although the incidence of cancers caused by very low doses of radiation is often too small to be of statistical significance in epidemiological studies (and the consequences of exposure difficult to characterize), it is reasoned that the possibility of risk should be guarded against under the assumption of an LNT model for the sake of conservatism and due to the absence of overwhelming and conclusive evidence one way or another [26]. In recent years, various studies have challenged the LNT model for low linear-energy-transfer (low-LET), some claiming that a *decrease* in risk is more likely than an increase in risk from small doses based on indirect evidence [27], [28]. While most of these studies clearly point out that the findings do not apply to high-LET, such as alpha radiation (and therefore radiation from radon), there has been a recent case-control study in the US by Thompson et al. [29] that found a reduction in risk associated with radon exposures of less than 150 Bq/m^3 [1]. Lowered cancer risks due to small doses of radiation is known as *radiation hormesis* and are attributed to an adaptive biological protective response of the cells [30]. The possibility of such a response to radon exposure at low levels broadens the uncertainty of radon risk assessments.

The US NRC's sixth committee on the biological effects of ionizing radiation (BEIR-VI), who developed two prominent models based on occupational studies, makes a mechanistic argument for a LNT radon model based on two important points:

1) At low radon exposures (which includes residential and the lower end of occupational exposures), a lung cell is likely to be traversed by no more than a single alpha particles in a person's lifetime.

[1] It should be noted that this result is from a case-control study (generally ranked as a weaker form of evidence) that involved a relatively small number (i.e. less than 600) subjects.

2) There is good evidence that a single alpha particle can cause substantial change in a cell which may initiate the sequence of events leading to chromosomal aberrations or delayed mutations.

It then follows that as the exposure is further decreased, the damage to any cell traversed by an alpha particle is the same, but the occurrence of such an event decreases proportionally with exposure. The validity of the LNT assumption is of particular importance to the occupational models because they are based primarily on extrapolation from occupational exposures to lower exposures.

Discussion of models here focuses on those derived from occupational studies and residential studies as they are currently the most developed and accepted.

2.1 Models Based on Occupational Studies

Due to the relatively high concentrations of radon in some underground mines, the effects of radon exposure can be more easily observed. Epidemiological studies of miners therefore constitute a practical and important component of research aimed at determining the risks from radon exposure. In this thesis, *occupational studies* refers to the body of epidemiological studies of underground miners.

In the 1950's, various health agencies in the US began collecting data on miners' exposures, smoking history, and mortality due to concerns of health risks associated with mining. At the same time, air sampling programmes were being conducted by the mining companies to measure radon gas levels. These early studies revealed high degrees of temporal and spatial variability of radon concentrations within mines as well as between mines [31]. Over the years, as numerous occupational studies were reported, analysts began to pool collections of studies to increase the statistical power of these studies. These include Thomas et al. [32] (five studies), BEIR-IV [6] (four studies), and Lubin et al. [25] (11 studies). Updates and extensions to the pooled data have occasionally been incorporated in revised studies (e.g. BEIR-VI).

To this day, occupational studies are recognized as the predominant, albeit imperfect, source of evidence for constructing exposure–response models. Occupational studies have been recommended as the preferred source for models in the BEIR-VI report [6], as well as in the International Commission on Radiological

Protection Publication 65 (ICRP65) [33].

One notable issue with the use of occupational studies to develop risk models is the need to extrapolate from the demographics and exposure conditions of the miners to the population inhabiting residences. Differences include significantly higher radon concentrations in mines, airborne particulate characteristics, exposure to other carcinogens, miners being working–age males, breathing rates, etc. A dosimetric modifier, known as the *K–factor*, is intended to account for the differences between the occupational and residential settings that are relevant to dose. Due to the broad range of factors accounting for the differences in exposure–response between the two settings, a significant degree of uncertainty is associated with the K–factor.

2.1.1 Prominent Models Based on Occupational Studies

BEIR-VI Exposure-Age-Duration and Exposure-Age-Concentration Models

The BEIR-VI committee developed two models, referred to as the exposure-age-concentration (EAC) and exposure-age-duration (EAD) models, which are named after their main risk factors. Both present excess relative risk (ERR) per unit exposure and thus fall within the relative risk class of models. The form of the EAC and EAD models is

$$ERR = \beta(\omega_{5-14} + \theta_{15-24}\omega_{15-24} + \theta_{25+}\omega_{25+})\phi_{age}\gamma_z, \tag{2.3}$$

where ERR is the ratio of lung cancer risk (exposed divided by unexposed) minus 1, β is the slope of the exposure–response relationship; ω is the exposure in the periods 5-14, 15-24, and 25+ years prior to the age at which the risk is being calculated; and θ_{15-24} and θ_{25+} are values that modify the relative risk due to exposures at the different exposure periods. The modifier ϕ_{age} reflects changes in the exposure–response relationship for different attained ages. Finally, γ_z is a modifier accounting for the effect of exposure concentration, in the case of the EAC model, or exposure duration, in the case of the EAD model. As a result, the ERR is a function of age and the sequence of computed ERR values for each age category constitutes

an age-specific ERR lifetime profile. Figure 2.2 shows the ERR calculated using the EAC model for an individual subjected to a constant lifetime exposure of 45 Bq/m^3.

The form of Equation 2.3 assumes the dose uptake to a human for a given exposure is the same in homes as it is in mines. In other words, the K-factor is 1, implying the net effect on the dose-exposure relationship from all differences between occupational and residential scenarios is null. Indeed, numerous studies have found that the K-factor is approximately equal to 1 (BEIR-VI, Krewski et al.) [6], [34].

The BEIR-VI committee analyzed limited available data on smoking status from the occupational studies in an attempt to examine and quantify the potential for a synergistic effect from smoking and radon exposure occurring in combination. The outcome of this investigation revealed a sub-multiplicative effect on ERR from smoking and radon exposure together (i.e., the relative risk from both was greater than the sum of, but somewhat less than the product of, the relative risk from each alone). This submultiplicative effect on the ERR is accounted for with the use of modifying factors of 0.9 for ever-smokers , and 1.9 for never-smokers (i.e., the ERR is multiplied by these factors to calculate the ERR for ever-smokers and never-smokers). Although the excess relative risk is greater for never-smokers than for ever-smokers, this factor is relative to the baseline lung cancer risk, which is approximately 10 times lower for never-smokers. Therefore in absolute terms, the increase in risk from radon exposure is significantly greater for ever-smokers.

EPA Model

When the US Environmental Protection Agency assessed the risks from radon in homes for the US in 2004 [35], they built a hybrid of the BEIR-VI models that produces risk estimates between the values projected by the two models. This was achieved by implementing the following steps:

1) calculating the risk per WLM (RWLM) for the EAC (Exposure-Age-Concentration) and EAD (Exposure-Age-Duration) models;

2) calculating the geometric mean of the two results from step 1; and

3) finding a value for β^* that, when substituted into the EAC model produces a risk per WLM equal to the geometric mean from step 2.

The EPA model then has a form similar to the EAC model but with an adjusted risk coefficient, β^*:

$$ERR/WLM = \beta^*(\omega_{5-14} + \theta_{15-24}\omega_{15-24} + \theta_{25+}\omega_{25+})\phi_{age}\gamma_z, \tag{2.4}$$

where $\beta^* = \beta \times \left(\frac{RWLM_{EAD}}{RWLM_{EAC}}\right)^{\frac{1}{2}} = 0.0634$. The risks per WLM for the EAC, EAD, and EPA models are shown in Figure 2.1, which helps illustrate the construction of the EPA model. The EPA also made further

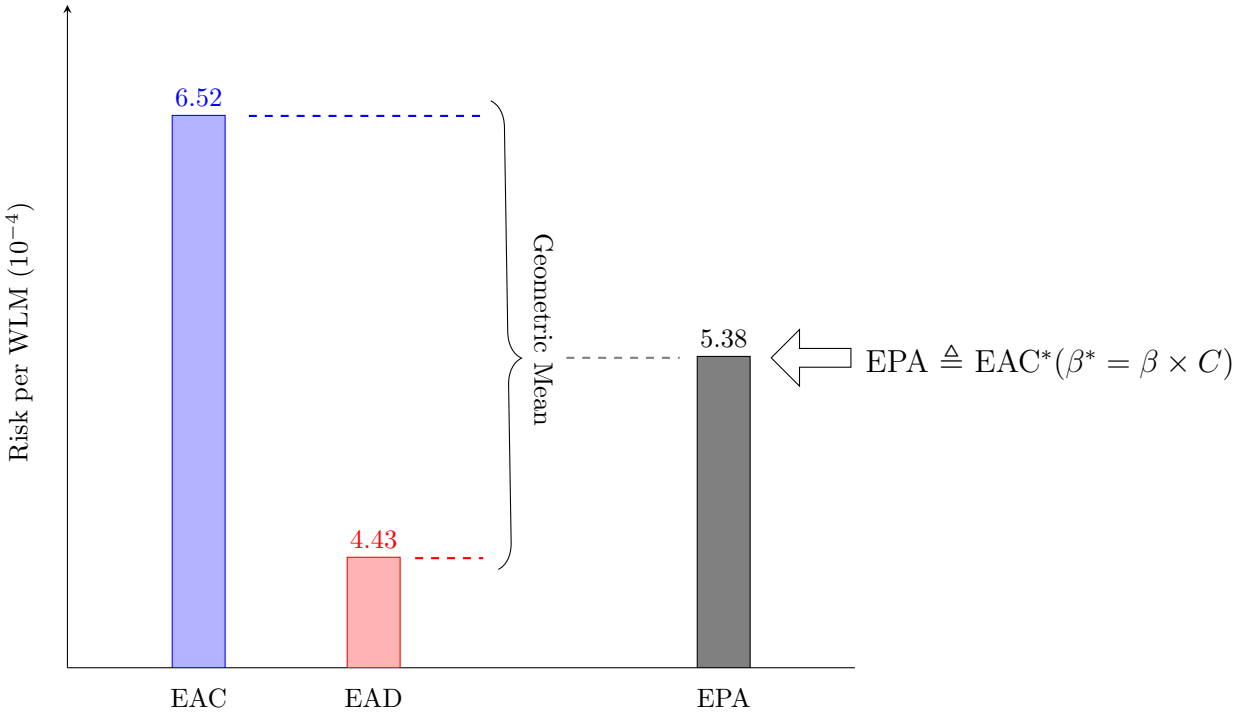

Figure 2.1: The EPA (Environmental Protection Agency) used the BEIR-VI EAC (Exposure-Age-Concentration) model with a modified potency factor, β^*, to estimate residential radon risks for the US population [35]. The potency factor, $\beta^* = 0.0634$, was determined by: 1) calculating the risk per WLM (Working Level Months) for the EAC (Exposure-Age-Concentration) and EAD (Exposure-Age-Duration) models; 2) calculating the geometric mean of the two results from step 1; 3) finding a value for β^* that, when substituted into the EAC model produces a risk per WLM equal to the geometric mean from step 2. Source: prepared by author based on EPA methodology in [35].

adjustments to the model to avoid what they describe as biologically implausible discontinuities introduced by the attained age component, ϕ, at ages 55, 65, and 75. The influence of this parameter on the ERR can be seen in Figure 2.2. Up to age 55, the relative risks increase because cumulative exposures increase

with age. The excess ERR then drops discontinuously at ages 55, 65, and 75. The EPA smoothed the ERR (eliminating the discontinuities) by using monotonic splines with nodes at 40y, 50y, 55y, 65y, 75y, 80y, and 90y, while preserving the integral of ϕ for the intervals 50-80y, 55-65y, and 65-75y. The resulting ERR can be seen in Figure 2.3, which shows a comparison of the ERR profiles generated from the discussed models for an individual exposed constantly to 45 Bq/m^3.

Risks per WLM estimated using the EAC and EAD models are dependent on exposure concentration, lung cancer and overall mortality rates, and smoking prevalence. The EPA model potency factor, $\beta^* = 0.0634$, which is derived from these risks, was therefore uniquely determined from the population data used in their calculation. As a result, the EPA model, as defined, applies only to the population used in its derivation.[2]

If we apply the same process using the Canadian population data (which is described in Chapter 4), a potency factor $\beta^* = 0.0603$ results. This is only approximately 5% lower than the EPA potency factor, however it is noted that general use of the EPA model, if not adjusted for the specific population and period to which it is applied, will not result in a geometric mean risk per WLM of the EAC and EAD models as it was originally constructed to.

[2]This population is actually a synthetic, stationary population whose members exhibit the characteristics of the population data used by the EPA at all ages. The EPA strategy can be tailored to a different scenario by using data for the population and period of interest (e.g. the Canadian population data during the years 2009-2011).

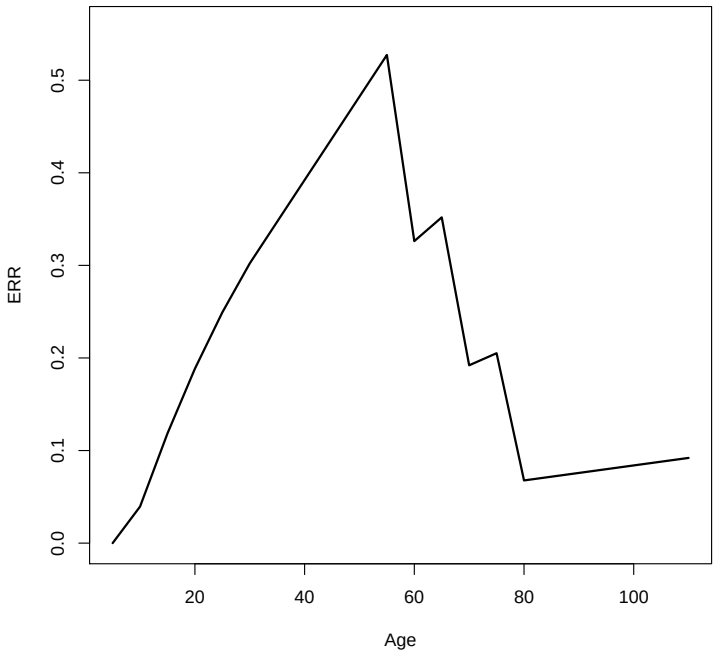

Figure 2.2: Plot of the ERR (Excess Relative Risk) as a function of age, calculated using the EAC (Exposure-Age-Concentration) model [6] and a constant lifetime exposure of 45 Bq/m^3

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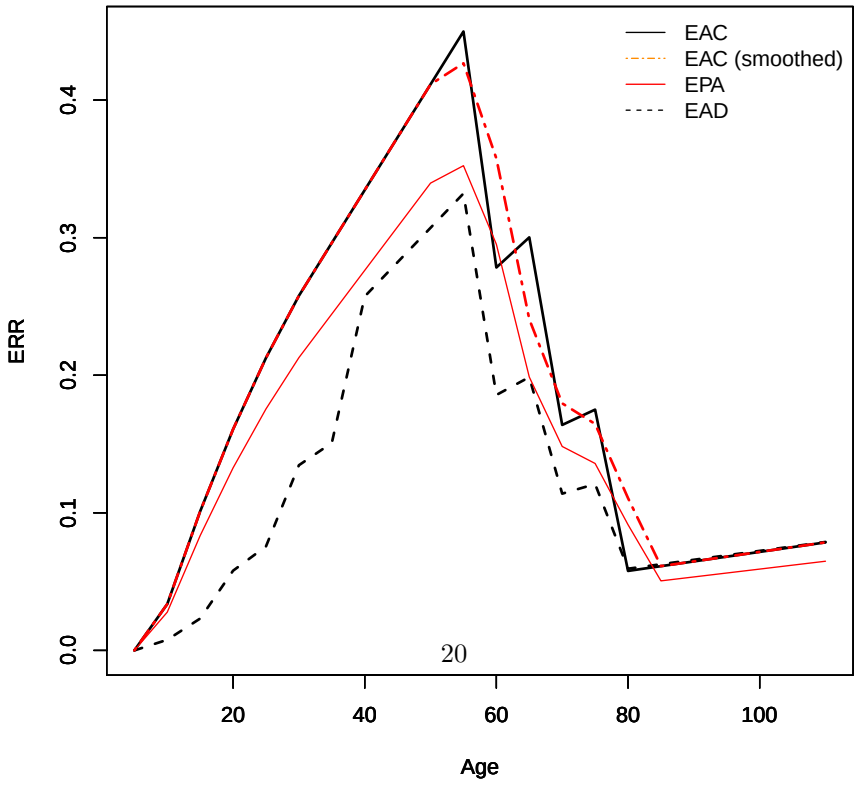

Figure 2.3: Comparison of excess relative risk (ERR) for the EAC (Exposure-Age-Concentration) [6], EAD

2.2 Models from Residential Studies

Another method of studying the exposure–response relationship of radon is to assess the increased lung cancer incidence among people exposed to radon in their residences. A strength of *residential studies* is that they observe the effects of radon in subjects and conditions that more closely represent the conditions of interest to residential radon risk assessments (versus data from underground minor exposures). This includes similarities in the level of exposure, demographics, other carcinogens, etc. These similar conditions lessen the issue of extrapolation in comparison to the occupational studies. Because residential exposures are generally low, the incidence of lung cancer due to the exposures is anticipated to be low. As a consequence, residential studies must use large numbers of subjects in order to identify the level of correlation between radon and lung cancer with a sufficient degree of confidence. Residential radon studies to date have employed two design strategies: *case-control* studies and *cohort* studies.

Case-control studies investigate the relationship between an exposure and a disease or effect by observing two groups: one, referred to as the *case group*, consists of patients who have the disease of interest; the individuals of the other group, referred to as the *control group*, do not have the disease. The relative levels of exposure for the groups are then assessed and compared to infer a statistical association [36]. Case-control studies are considered retrospective because subjects are categorized by current lung cancer status and an attempt is made to define previous exposures for the subjects. This poses the challenge of retrospectively estimating the exposures of individuals from residential histories and living habits. Matching cases and controls for factors other than radon may also be a difficult but important task to avoid selection bias.

Another concern with case-control studies is *recall bias*, which is a propensity that may exist in subjects with lung cancer to overstate past exposure compared to healthy subjects, thereby artificially strengthening the association [37], [38]. Subjects may also exhibit recall bias in overstating histories associated with confounders such as smoking, undermining efforts to control for these co-exposures.

Whereas *case-control studies* begin with a categorization of subjects based on the presence or absence of disease followed by an assessment of past exposure, *cohort studies* begin by categorizing subjects based on prevailing exposure conditions and then observing which subjects contract the disease (e.g., lung cancer)

over time. Radon cohort studies begin by selecting groups that do not have lung cancer and assessing the degree of radon exposure to the individuals. Participants are followed up over time to record the incidence of lung cancer, as well as to record potential changes in exposure, in order to infer the relationship between exposure and lung cancer risk.

Tracking participants through time is an advantage of cohort studies because it provides the opportunity to more accurately characterize exposures. Random selection of participants can help to ensure they are the same with respect to factors other than radon exposure so that differences in observed lung cancer rates can be more reliably attributed to radon exposure. Since the risk of lung cancer from radon exposure is small, residential studies require large numbers of participants observed for many years to adequately assess the exposure–response relationship.

Case-control and cohort studies of residential radon exposure are both referred to as *residential studies* in this thesis. Figure 2.4 depicts the sequence of activities in cohort and case-control studies, which essentially have opposite start and end points. The results derived from residential studies are consistent with excess risks predicted by models based on occupational studies and build on the amassed evidence linking radon exposure to elevated lung cancer risk.

Unlike the occupational models, which provide a method for estimating risk from exposures incurred at any point in the past, the residential models provide risk estimates based on a fixed window of exposure (e.g. the period of time 5 to 30 years prior to the study). The residential models do not provide a means to project lifetime risks from exposures at all ages, since it is not known how to interpret risks from exposures outside the window for which the study assessed subjects exposures.

2.2.1 Case-Control Studies

Since the BEIR-VI committee's report and the ICRP Publication 65 [33], a number of residential case-control studies have been completed, amounting to more than 20 individual case-control studies during the last two decades [39]. As a result of the large pool of available data, The United Nations Scientific Committee on the Effect of Atomic Radiation (UNSCEAR) [40] reported that residential case-control studies are now an

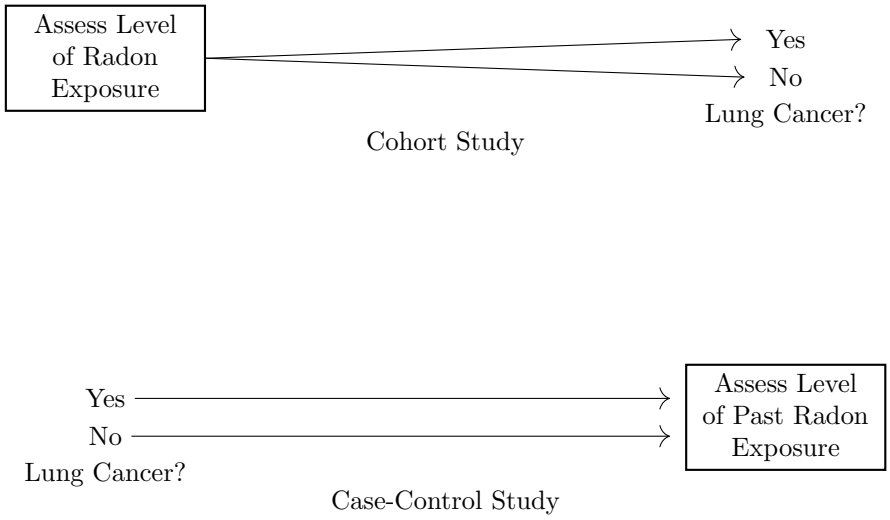

Figure 2.4: Cohort and case-control studies both investigate the relationship between radon exposure and lung cancer risk but have opposite start and end points: cohort studies first assess radon exposure and then record contraction of lung cancer; case-control studies select participants with and without lung cancer and then assess the past exposures.

appropriate basis for exposure–risk assessment. Similar to the occupational studies, a number of residential studies have pooled data from separate studies to improve statistical power.

North American Case-Control Studies

Krewski et al. [41] analyzed a pool of 11 North American residential studies in 2006 and found an odds ratio for lung cancer of 1.10 associated with a radon concentration of 100 Bq/m^3 incurred in the previous 5 to 30 years time-frame. This exposure time window was chosen because the miner studies assessed in the BEIR-VI report [6] indicate this is the period of radon exposure most relevant to radon-induced lung cancer. The exposure–response was expressed as

$$OR = 1 + \beta \cdot X, \tag{2.5}$$

where X is the mean radon exposure in the homes inhabited during the 5- to 30-year period before study

enrollment and β is the slope of the exposure–response.

Heterogeneity with regards to gender (female vs. male), and smoking status (ever vs. never) was explored but not found to be statistically significant.

European Case-Control Studies

Darby et al. studied data pooled from 13 case-control studies spanning nine European countries in 2005 [42]. They found an odds ratio of 1.084 per 100 Bq/m^3 of measured radon exposure in the previous 5- to 30-year period. Their model was also the same form as Equation 2.5. Excess risk did not differ significantly with study, age, gender, or smoking habits.

Chinese Case-Control Studies

Lubin et al. combined two Chinese studies [43]. Also employing the same model as Equation 2.5, they observed an odds ratio for lung cancer of 1.33 per 100 Bq/m^3. As with the pooled North American and European studies, the results were virtually identical for males and females, and for never- and ever-smokers and no significant effects from attained age were observed.

Canadian Geographic Mapping Study

Hystad et al. [44] estimated geographic variation in radon using two mapping methods: 1) average radon concentrations to health regions in Canada using the 14000 measurements from the Health Canada cross-country radon survey by health region; and 2) the radon risk map from Radon Environmental Management Corp., as described in Appendix C.2.1. These maps, which are shown in Figure 2.5, were analyzed in conjunction with lung cancer data from the National Enhanced Cancer Surveillance System (NECSS) [45], a population-based case-control study in eight Canadian provinces using 2390 lung cancer cases and 3507 controls. Ecological measures of radon exposure were developed for the study participants using the two maps and 20-year residential histories of the participants. Based on the ecologically assigned exposures and lung cancer statistics from the NECSS study, Hystad et al. estimated a lung cancer odds ratio of 1.12 (95%

confidence interval: 1.03-1.22) per 50 Bq/m^3 increase in radon concentration [44].

2.2.2 Cohort Studies

To date, two large-scale cohort studies have been conducted for radon exposure risks. In 2011, Turner et al. [46] analyzed the association between radon exposure and lung cancer among the approximately 1.2 million American Cancer Prevention Study-II participants. The Cancer Prevention Study-II is a prospective mortality study administered by the American Cancer Society with the goal of determining factors that cause or prevent cancer. Turner et al. estimated radon exposure history of each of the participants by using the mean residential radon concentrations for the county associated with their ZIP code. Although smoking data were collected for its participants at enrollment in 1982, smoking status was not collected as part of the CPS-II follow-up activities. Consequently, Turner et al. restricted their analysis to data collected during first six years of follow-up following enrollment, since any changes in smoking status of the subjects during this time period are not likely to significantly affect the lung cancer incidence.

A relative lung cancer risk of 1.15 (95% confidence interval: 1.01-1.31) for a 100 Bq/m^3 increase in radon concentration was observed. This study also examined the data to test for heterogeneity associated with smoking status or other risk factors and, like the previously discussed meta-analyses (for the case-control studies), showed no statistically significant heterogeneity.

The second large-scale study was a 2012 study (Brauner et al.) [39] which assessed the association between residential radon and lung cancer by recruiting a Danish cohort of 57,053 individuals during 1993-1997 and tracked them for cancer occurrence until 2006. The subject's residences of occupancy were traced back to 1971, resulting in 35 years of residential history. Radon exposures were estimated for each of the residence addresses using a validated regression model [47], which used databases that included nine explanatory variables, including geographic location, soil type, and house characteristics such as residential type, floor level, basement and building materials. The radon concentrations in Denmark are generally in the lower end of the residential exposure range with a median estimate for radon concentration of 35.8 Bq/m^3. Lung cancer incident rate ratios of 1.04 (95% confidence interval: 0.69-1.56) and 1.67 (95% confidence interval:

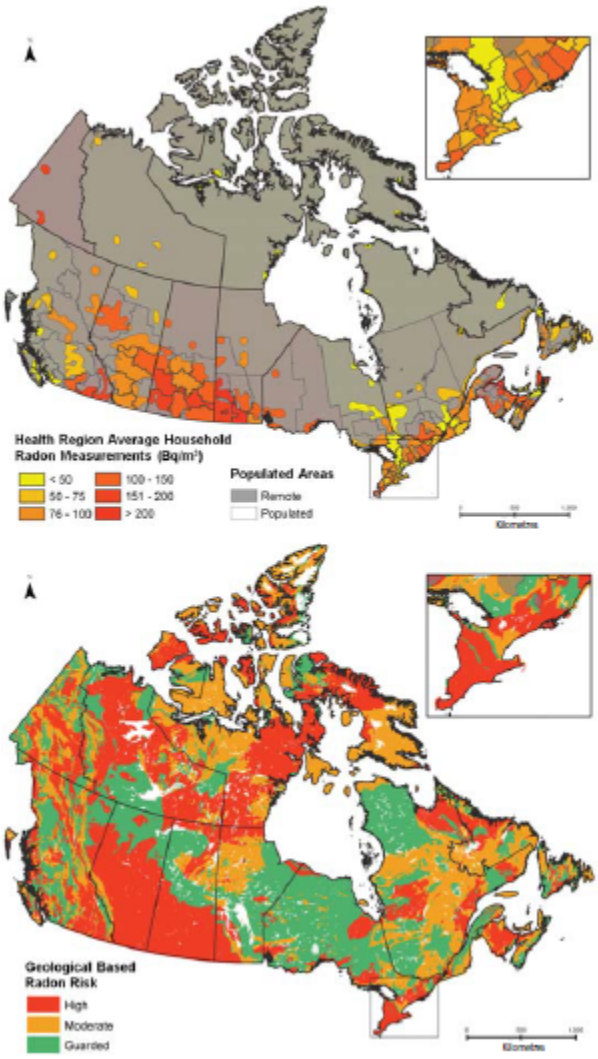

Figure 2.5: Radon concentration estimates (based on surveys and geological factors), shown above, were used in conjunction with lung cancer data to derive ecological radon risk factors by Hystad et al. [44]. The first map assigns the average health region exposures of the 2011 Cross-Canada Radon Survey to each health region. The second map shows the radon risk categories for geological units based on US radon potential classification, uranium geochemistry, and radiometric geophysical response, as determined by Radon Environmental Management Corp. Source: Hystad [44].

0.69-4.04) at 100 Bq/m^3 were found for all participants and non-smokers, respectively, and were invariant by sex.

These cohort studies are notable for predicting radon exposures without direct measurement within the residence. One advantage of this method is the avoidance of participation bias which may affect studies that require consent. A beneficial aspect is that these studies include large numbers of subjects in a cost-effective manner. However, the radon exposure estimates for the subjects are highly uncertain using these methods because studies performed at the *ecological*, or population level, may not reliably link lung cancer incidence rates to radon concentration. Chen and Moir [48] state that the main shortcoming of ecological studies is that exposures characteristic of a region are assigned to individuals, but the average risk determined for the population does not correlate well with the average exposure.

A notable difference between the findings of the occupational and residential studies is the observation of approximate homogeneity of risks across both sexes in the residential studies. To speculate on potential reasons for this, the major differences between occupational residential studies may be considered. For example, the occupational studies assess subjects exposed to significantly higher radon exposures. It may be the case that there exists a threshold below which smoking history does exert an effect on lung cancer risks from radon exposure. Another factor is the number of other types of occupational exposures, such as diesel fumes, facing the miners (in comparison to residents). An unidentified confounder in the occupational setting could result in a misinformed relationship between smoking and radon exposure risks.

2.3 Biological Studies

Biological studies seek to understand the mechanisms of carcinogenesis to assess risk from exposure to radon. The BEIR-VI committee regarded biologically motivated risk models as a desirable goal but dismissed this option given the complexity and current lack of scientific understanding of the biological mechanisms involved in radiation carcinogenesis. UNSCEAR notes that considerable uncertainty exists in the most recent models and that data do not yet provide the basis for the best choices of exposure–response models for each of

the cellular processes [40]. It was noted, however, that when biological mechanisms are better understood, biological-based models may be preferred for characterizing the response to radon exposure [6], [49]. Until that time, insights from biologically motivated models may be useful for confirming or contesting findings of occupational and residential studies. For example, Timarche [50], who estimated mutation rates based on an analysis of French and Czech miner cohorts, observed that an inverse dose-rate effect cannot be excluded for exposure rates greater than 30 WLM/year [40], corroborating findings of an inverse dose-rate effect in the BEIR-VI occupational studies [6]. The inverse dose-rate effect is the notion that lower risks result from a given total exposure if the exposure was incurred over a shorter time (i.e. the dose-rate is higher).

2.4 Uncertainty and Variability

2.4.1 Uncertainty vs. Variability

An uncertainty analysis of the risk estimates provides a quantitative characterization of the distribution of the potential risk outcomes, such as life-years lost. Overall uncertainty in the risk estimate can be attributed to two types of uncertainty: *true uncertainty*, hereafter referred to as *uncertainty*, and *variability*.

Uncertainty exists when a parameter's true value is not known precisely. Often arising from limited availability of empirical information, imperfections in instrumentation, models, or techniques used to develop representations of complex processes [51], it can be described as "the *assessor's* lack of knowledge (level of ignorance) about the parameters" [52]. An example of an uncertain parameter related to radon risk estimates is the measurement of radon concentration, which is subject to error in detection and counting. Residential radon concentrations also vary considerably over time, especially with changing seasons, and short-term measurements incur greater uncertainty as a result.

Broadly speaking, uncertainty can be attributed to lack of information, limitations in understanding of the exposure–response mechanism, and imperfect techniques for assessing exposure.

Variability, on the other hand, arises from heterogeneity of inputs. Height, as an example, is a variable parameter which varies from one individual to the next. This is not due to uncertainty in estimates of height;

rather it is a variable attribute of the populations members. An example of a variable parameter related to the occupational models is the K-factor, which relates the exposure–dose relationship in occupational conditions to residential conditions. This value varies from one exposure (e.g. a person exposed under residential conditions) to the next and is dependent on variables such as aerosol distribution, occupant breathing rates, occupant mass, smoking status, etc. The K-factor is not only variable but is also uncertain, as it is dependent on numerous variables which cannot be known with certainty[3].

2.4.2 Model Uncertainty

Structural Uncertainty

Structural uncertainty exists when we are unsure about the true form of the model (i.e. the exposure–response relationship). We are not certain the output of the model would be correct even if all model parameters and inputs were known with absolute certainty. The development of two models by the BEIR-VI committee for the same data set is a reflection of structural uncertainty. It cannot be said which is the more "correct" model. Draper et al. [53] note that although it is common practice to acknowledge parametric uncertainty about a given model, it is less common to acknowledge structural uncertainty itself. Often, a single "best" choice is made for the model structure and the analysis then proceeds as if the model were known to be correct. Structural uncertainty can be assessed by presenting the results from multiple models or by simulating a combination of the models using a discrete distribution that randomly selects one model or the other for each iteration, as described by Vose [52].

When the distributions of uncertain risk estimates returned by the competing models overlap, decisions based on the risk estimate can be considered robust with respect to structural uncertainty. Conversely, models yielding risk distributions that are mutually exclusive or having little overlap is indicative of significant uncertainty and limited knowledge of the true exposure–response relationship.

[3]Parameters are often both variable and uncertain.

Extrapolation

A common source of uncertainty is *extrapolation*. Extrapolation occurs when a model is used to estimate risks under conditions that extend outside the range of conditions from which the model was derived. Regarding radon studies, an example of extrapolation is the use of models based on occupational studies to predict risk for lifetime residential exposure. The occupational models are based on observations of miners who worked in environments with significantly higher radon concentrations than typical residences. Additionally, a number of other parameters differ between the occupational and residential scenarios include the following:

1. Age at exposure: miners are exposed to radon in the mines when they are of working age.
2. Sex: miners from the occupational studies were males.
3. Smoking status: most of the miners studied were smokers.
4. Duration of exposure: miners were exposed to relatively high radon concentrations during working hours for the length of their careers; the general population is exposed to lower residential levels for the duration of their lives.
5. Differences in radon decay product dosimetry in mines and residences: for a given radon concentration, heavier breathing rates of working miners in combination with different equilibrium fractions, aerosol characteristics, and other differences between mines and residences leads to different lung dosimetry (i.e. the K-factor).

Extrapolation of these parameters were noted by the BEIR-VI committee as sources of uncertainty in their models. Conversely, models derived from residential studies are subject to less extrapolation uncertainty because they are based on conditions which bear greater similarity to radon exposure experienced by the general population.

2.4.3 Input Uncertainty

Inputs may be intrinsic to the structure of the model, such as parameters estimated from miner data, or may be independent of the model's derivation, such as the distribution of radon concentrations across Canadian

residences.

2.5 Chapter Summary

In this chapter, the concepts of absolute risk and relative risk models were introduced. Relative risk models have a LNT exposure–response. This means that the relationship between exposure and risk is constant, and that the relationship holds regardless of the exposure level. Several studies have challenged the LNT model for radiological exposures, and this is a source of uncertainty with use of the existing radon exposure–response models.

The two prominent models based on occupational studies are the BEIR-VI EAC and EAD models, which compute age-dependent excess relative risks for lung cancer due to radon exposure. The study producing these models also found a submultiplicative effect for lung cancer risks from radon exposure and smoking in combination.

A number of residential studies, which can generally be categorized as case-control or cohort studies, were also identified. Unlike the occupational studies, the residential studies found no heterogeneity with respect to smoking history. It was also noted that residential models do not provide a means to project lifetime risks from exposures at all ages, since it is not known how to interpret risks from exposures incurred outside the time period of assessed exposures.

The chapter concluded with a description of uncertainty and variability. Uncertainty may be inherent in the exposure–response models, as well as the inputs to these models.

Chapter 3

Health Canada Cross-Canada Survey of Radon Concentrations in Homes

3.1 Description and Methodology

In 2011, Health Canada completed a country-wide survey that sampled concentrations in approximately 14,000 homes. The survey, as described in the final report from Health Canada [23], was designed to collect samples from all health regions in Canada for a three month period between October and March in order to develop a profile of the prevailing radon levels for all geographic areas of the country during the time of year when indoor concentrations are anticipated to be highest. Depending on the province, each health region has a public health unit or health authority responsible for administering health promotion and disease prevention within the region. As of 2013, there were 141 health regions throughout Canada. According to Health Canada's final report on the Cross-Canada Survey of Radon Concentrations in Homes [23], participants were mailed an alpha track radon detector and asked to position the detector in the lowest portion of their home which was inhabited for four or more hours per day and to leave it positioned for at least three months. Due to the temporal variations in radon concentrations, three months is recommended

by Health Canada as the minimum duration to obtain a representative sample. Homes built on stilts and high-rise condo units above the second floor did not qualify for the survey. Due to the size of the survey, the detectors were mailed in two phases so that participants were measuring radon levels in their homes during the winter months of either 2010 or 2011.

The alpha track counting of the detectors was performed at Health Canada's National Radon Laboratory and was conducted using calibrated equipment. To ensure a high degree of accuracy and precision, the survey used quality control activities including the use of duplicate detectors for approximately 10% of the measurements, as well as blank detectors at a rate of approximately 5%. The blank detectors remain closed and are kept in a low radon environment during the survey. They are opened before shipment with the field detectors to the laboratory and are used to represent the background exposure that may accumulate during shipment and storage of the field detectors.

In the final report, Health Canada calculated that 6.9% (population-weighted) of the population lived in homes in which the radon concentration exceeded the Health Canada guideline of 200 Bq/m^3 — a result which was more than two-fold higher than previous estimates provided by Chen and Moir in 2010 [19], who used recent surveys from 4 provinces, and several times higher than the estimate from Brand et al. in 2005 [21], who used data from the 1978 survey.

3.2 Summary of Measurement Results

3.2.1 Mean, Standard Deviation, and Shape of Survey Measurements

The arithmetic mean radon concentration from the survey data is 97 Bq/m^3, the geometric mean is 49.1 Bq/m^3, and the geometric standard deviation is 3.1. As with many types of exposures, residential radon concentrations generally exhibit log-normal distributions. The resemblance of a log-normal distribution of the survey data can be assessed with a log-normal probability plot. As described in [54], a probability plot is a graphical method for comparing the distribution of empirical data to a theoretical distribution. This is performed by plotting the quantiles of the empirical data against the quantiles of the theoretical distribution.

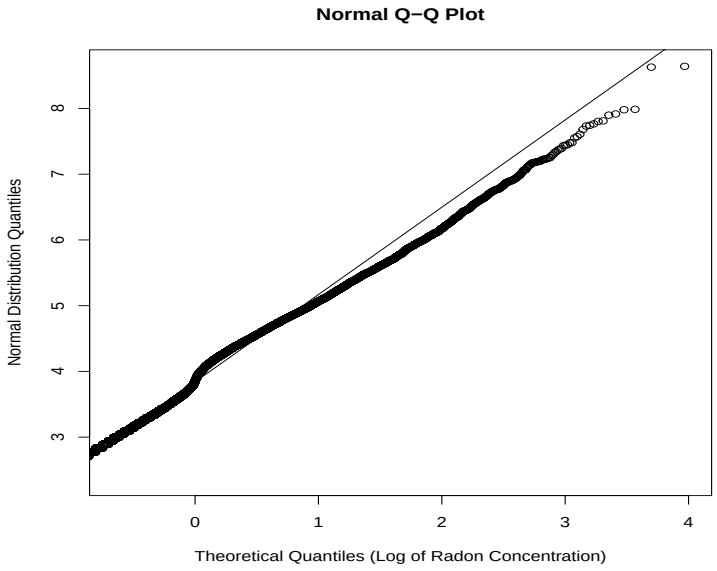

Figure 3.1: A normal probability plot using log of survey measurements is shown. log-normal data would plot along the thin straight line, which represents a plot for a perfect log-normal distribution.

If the plot is a straight line, the distribution of the empirical data matches the theoretical distribution for which it is being assessed. The quantiles of the natural logarithm of the survey concentration measurements are plotted against the quantiles of a normal distribution in Figure 3.1. Log-normally distributed data would fall on a straight line line when plotted this way. Note that survey measurements less than 15 Bq/m^3 are excluded from Figure 3.1 because these measurements were reported as "less than 15 Bq/m^3" in the survey due to the high uncertainty associated with low concentration measurements. In addition to this plot, a cumulative distribution frequency plot of the survey data is shown in Figure 3.2 with the x-axis (radon concentration) in log-scale. The cumulative distribution of a normally distributed random variable (or log-normal distribution on log-scale) would exhibit the classic S-curve. Here we see two main deviations from a log-normal distribution: 1) the step at approximately 10 Bq/m^3 is due to the assignment of all measurements below 15 Bq/m^3 to $15/\sqrt{2}$ Bq/m^3; 2) a flexure centered at approximately 50 Bq/m^3 due to the dip in the number of measurements near this concentration.

This dip is apparent in the histogram of the survey data in Figure 3.2, which reveals the existence of two

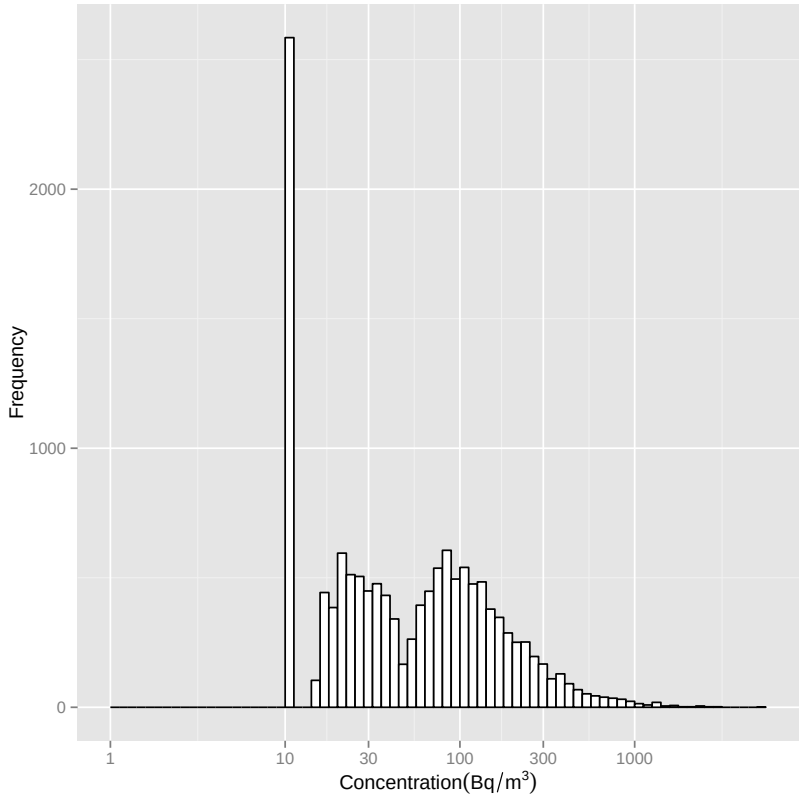

Figure 3.2: A frequency distribution plot for the approximately 14,000 measurements reported by Health Canada from the 2011 Cross-Canada survey data [23] is shown. The distribution clearly reveals the bimodality of measurements. The tall bar at approximately 10 $\mathrm{Bq/m^3}$ results from setting all measurements reported as "below 15 $\mathrm{Bq/m^3}$" measurements to $15/\sqrt{2}$ $\mathrm{Bq/m^3}$.

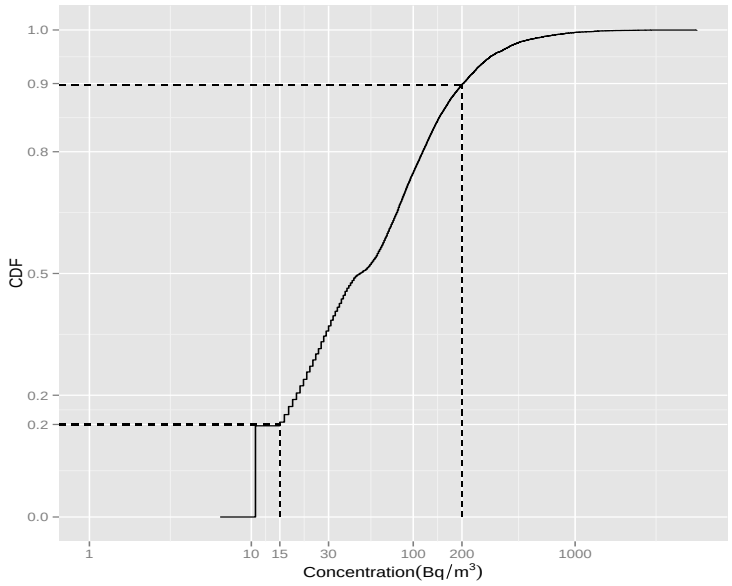

Figure 3.3: Cumulative distribution plot for the 2011 cross-Canada survey [23]: The dotted lines identify the Health Canada recommended action level of 200 Bq/m^3 and the 15 Bq/m^3 threshold below which measurements were recorded as "below 15 Bq/m^3". Slightly more than 11% of the raw measurements (neither seasonally adjusted nor population-weighted) exceeded the recommended action level. The approximately 19% of measurements below 15 Bq/m^3 were reported by Health Canada as simply "less than 15 Bq/m^3". These measurements are counted as $15/\sqrt{2}$ Bq/m^3 in this thesis and generate the step seen in the plot. The flexure at approximately 50 Bq/m^3 indicates a departure from the "classic S-shape" that would be expected if the cross-Canada data followed a log-normal distribution (since the horizontal axis in this figure is log-scale).

modes in the distribution. Conventionally, log-normal distributions are assumed for the residential radon concentrations in radon risk studies, including those by the Health Canada, the US EPA, and the BEIR-VI committee. It's worth noting that bimodality was observed to sustain even when data was stratified by province, health unit, etc. It was not apparently explained by characteristics[1] such as floor level, foundation type, etc., which were collected in the survey. Correlations between radon levels and housing characteristics for the cross-Canada survey were investigated by Jiang [11], but only weak correlations were observed.

Another survey was conducted by Health Canada in 2012 which involved simultaneous measurements of radon and thoron concentrations in about 4000 Canadian homes (less than one third of the size of the 2011 cross-Canada survey). Because this survey also used Health Canada's guide for radon measurements [55], it was of interest to investigate whether the measurements from this survey also exhibited the bimodality. Density log plots of the cross-Canada survey and the 2012 Radon/Thoron survey are shown in Figure 3.4. Unlike the cross-Canada survey, the distribution of the Radon/Thoron survey shows no evidence of bimodality, and reveals a bell shape that would be expected if log-normally distributed data were plotted using a histogram with the horizontal axis in log-scale. A normal quantile-quantile probability plot of the Radon/Thoron survey measurements in Figure 3.5 shows a straight line for the better part of the range of measurements (excluding measurements below 15 Bq/m^3), rendering the cross-Canada distribution even more puzzling.

Residential radon levels have been found in past surveys to show systematically different averages by the level (basement, versus first or second floor) of the home, with the higher levels in the basement and lower floors, as documented by Marcinowski et al. [56]. Results from this study concur with this finding and summary measures for the concentration levels by floor are shown in Table 3.1. Figure 3.7 shows box plots of the concentration by floor and illustrates different concentrations, and, perhaps more significantly, the greater variability of 2nd floor measurements.

[1]These characteristics include the type of residence (e.g. bungalow, two-story), type of room that detector was placed in, age of residence, whether the basement was finished, how many windows are open in the basement, whether an existent sump hole is capped/sealed, the type of foundation, the type of heating, installation of air conditioning, the type of water supply, occurrence of recent renovations.

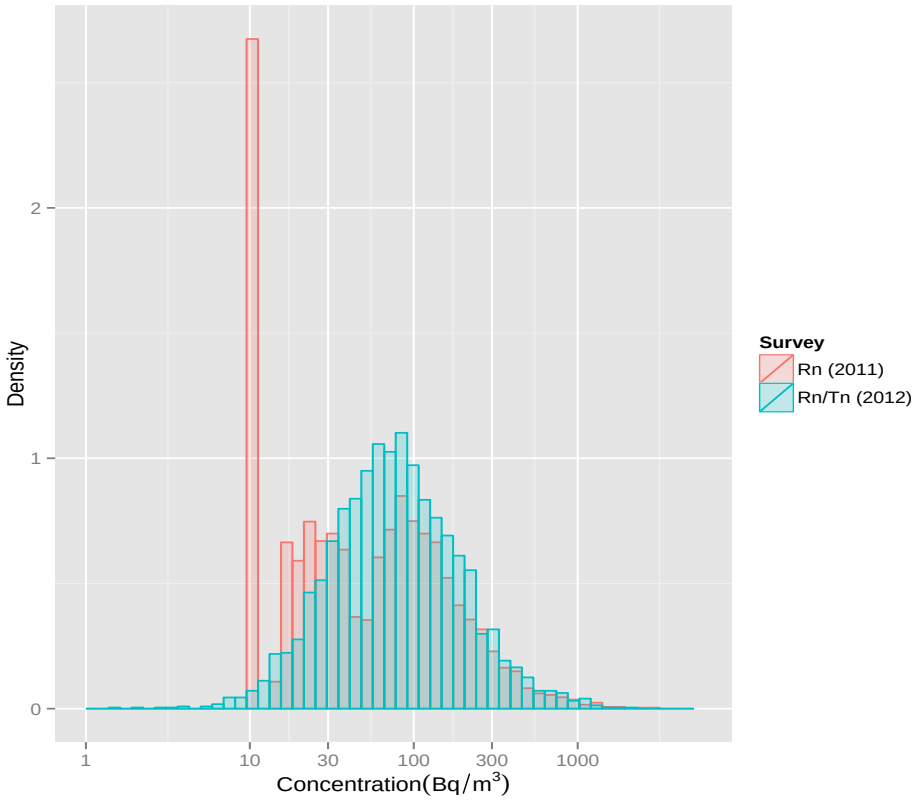

Figure 3.4: Density plots for the Health Canada 2011 cross-Canada and 2012 Radon/Thoron surveys. The overlaying of the two plots contrasts the bimodal distribution of the cross-Canada survey with the approximately log-normal distribution of the Radon/Thoron survey.

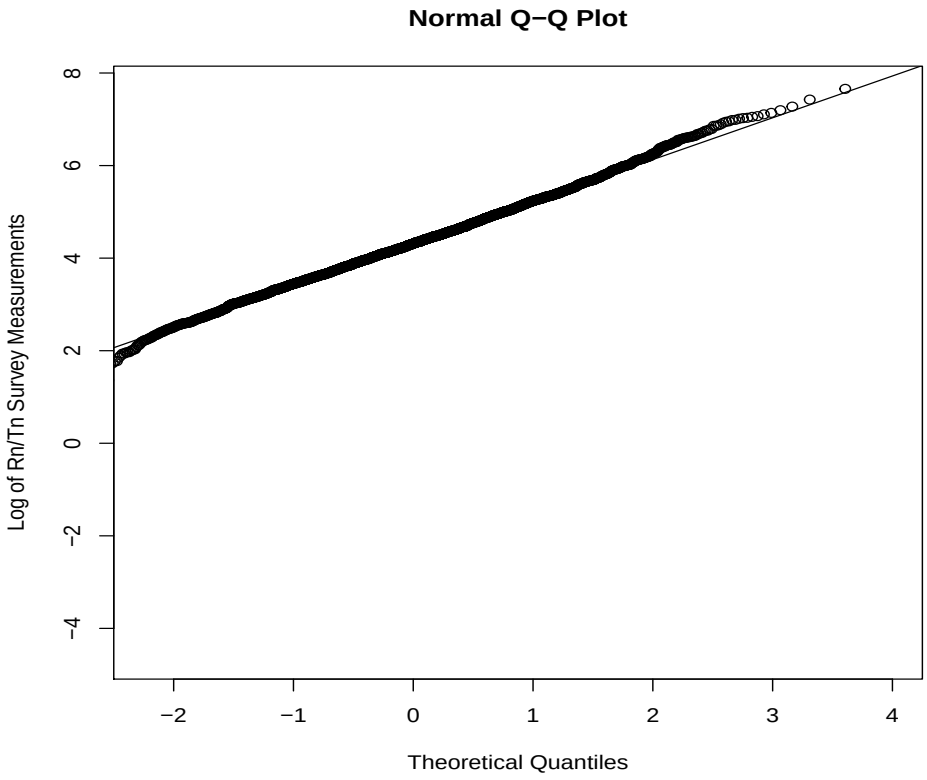

Figure 3.5: The normal probability plot for the 2012 Rn/Tn survey measurements falls along the thin line, which would be expected for log-normally distributed data. Compared to the bimodal distribution of the 2011 cross-Canada distribution, this survey exhibits a clean log-normal distribution.

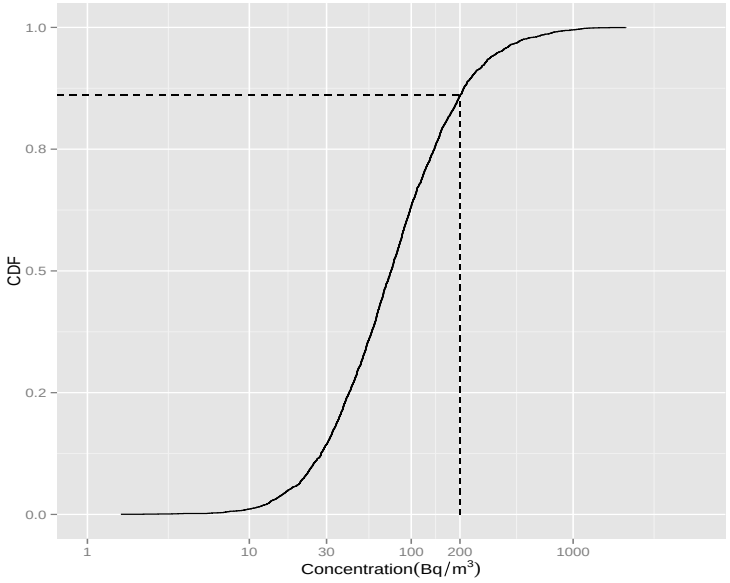

Figure 3.6: The cumulative distribution plot for 2012 Radon/Thoron survey exhibits the classic "S-curve" that would be expected from log-normally distributed data plotted in this fashion (with the horizontal axis plotted on log-scale). The dashed lines indicates the 200 Bq/m^3 threshold recommended by Health Canada for remedial action. Source: HC Radon/Thoron final results.

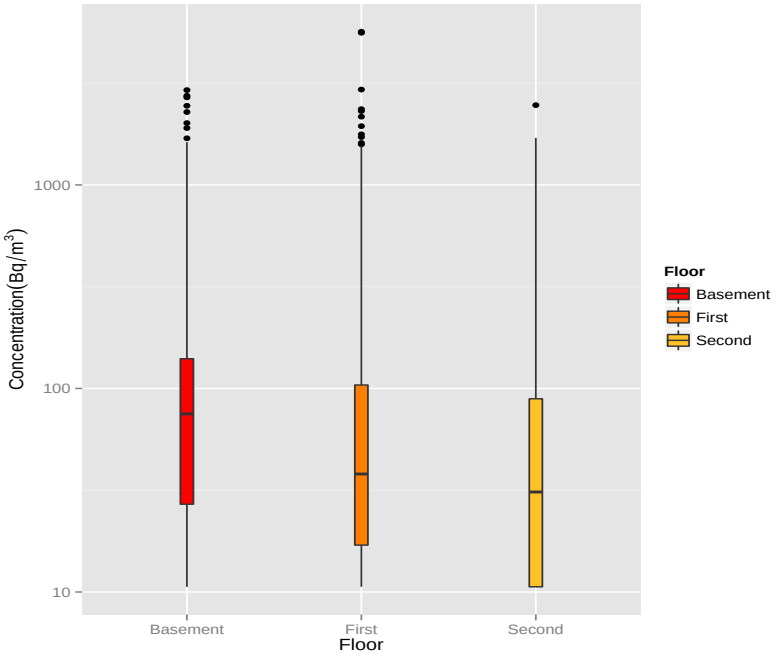

Figure 3.7: Box and whiskers plots by floor for the 2011 cross-Canada radon survey [23]: the boxes enclose 50% of the data while whiskers capture the extent of data falling within 1.5 times the interquartile range between the 1st quartile (lower whisker) and 3rd quartile (higher whisker). Outliers (beyond the 1.5 interquartile range) are shown as dots. Outliers are observed on the high side (with the exception of the 2nd floor) as would be expected for right-skewed data (e.g. log-normally distributed data). It is noted that the interquartile ranges (Q3-Q1) overlap across all three levels. It should also be pointed out that the 2011 cross-Canada survey included no matched data (e.g. basement versus 1st floor measurements, matched by house). The sample sizes are 4013, 8335, and 1162 for the basement, 1st, and 2nd floors, respectively.

	Overall	Basement	First Floor	Second Floor
Arithmetic Mean	97.0	121.0	87.5	79.7
Geometric Mean	49.1	66.1	44.2	38.1
Geometric SD	3.1	3.0	3.1	3.1
Sample Size	13807	4013	8335	1162

Table 3.1: Measurements from the 2011 cross-Canada survey are stratified by floor. Units are in Bq/m^3 except for the GSD, which is unitless. Average basement exposures are seen to be significantly higher than other floor levels.

Arithmetic and geometric mean concentrations for each province are summarized in Table 3.2, revealing an approximately four-fold range in the surveyed concentration means (arithmetic) from the province with the lowest measurements (PE) to the province with the highest (NB).

	AB	BC	MB	NB	NL	NS	ON	PE	QC	SK
Arithmetic Mean	92.0	69.4	150.1	179.4	61.9	93.8	82.9	46.7	81.2	126.6
Geometric Mean	64.8	31.5	88.5	77.8	29.7	36.6	45.7	26.3	39.5	88.7
Geometric Standard Deviation	2.3	3.1	2.9	3.7	2.9	3.3	2.9	2.6	3.1	2.4
Sample Size	1131	1817	1183	830	713	592	3950	113	1784	1206

Table 3.2: Exposure data from the 2011 cross-Canada survey is stratified by province. Units are Bq/m^3, except for GSD which is unitless.

3.2.2 Population-Weighting

Summary survey statistics can be calculated so that average exposures of geographic regions are weighted according to the population sizes inhabiting those regions. In this thesis, the regions selected for this purpose are the health regions of Canada, along with their 2013 populations[2]. For a given summary measure, X (e.g. average exposure, ILD, etc.), the population-weighted average is calculated as follows.

$$\overline{X} = \sum_{hr} (X_{hr} * Pop_{hr})/Pop_{Can} \tag{3.1}$$

[2]Between completion of the cross-Canada survey and 2013, a number of health units have been amalgamated. For these cases, the survey data from the health units were combined so that the measurements and population data were attributed to the same region.

Where hr connotes health region, Pop_{hr} is the population size of a health region and Pop_{Can} is the Canadian population size. Table 3.3 shows the population-weighted arithmetic and geometric means for the cross-Canada survey, stratified by floor.

	Overall	Basement	First Floor	Second Floor
Arithmetic Mean	72.12	86.39	65.78	56.52
Geometric Mean	43.67	55.00	39.52	37.71

Table 3.3: Population-weighted mean estimates are shown by floor. Units are in Bq/m^3.

Chen et al. [22] also estimated a population-weighted geometric mean and geometric standard deviation for the 2011 cross-Canada survey. They calculated values of 41.9 Bq/m^3 and 2.8 Bq/m^3 for these summary values, respectively. These estimates are in close accordance with those calculated in Table 3.3. The slight discrepancy in the geometric mean could be attributed to differences in how the measurements below 15 Bq/m^3 were treated, or due to different population size statistics.

3.2.3 Seasonal Adjustments to Measurements

Short-term radon measurements (e.g. three-month measurements) may not accurately represent the average long-term radon concentration due to seasonal variations in residential radon concentrations. An attempt to compensate for seasonal variation captured by a short-term measurement can be made by adopting seasonal correction factors.

As discussed further in Chapter 4, seasonal adjustment factors can be applied to each survey measurement depending on which months of the year it obtained. Applying the seasonal adjustment factors to the cross-Canada survey measurements reduces the average exposure. This is due to the survey measurements being taken during the winter season, for which the seasonal correction factors are less than unity. Table 3.4 shows the geometric means by floor for measurements that are both corrected with seasonal adjustment factors and population-weighted.

	Overall	Basement	First Floor	Second Floor
Arithmetic Mean	60.0	72.0	54.6	46.6
Geometric Mean	36.3	45.6	32.8	30.9

Table 3.4: Seasonally adjusted population weighted exposure estimates by floor in Bq/m^3. In this assessment, both seasonal adjustments and population weighting result in reduced average concentrations.

Arithmetic and geometric means of the seasonally-adjusted exposures were also calculated for each province and are presented in Table 3.5.

	AB	BC	MB	NB	NL	NS	ON	PE	QC	SK	Can
Arithmetic Mean	76.0	56.2	126.1	148.8	50.3	76.0	67.6	39.8	69.1	104.8	80.2
Geometric Mean	53.4	25.3	73.9	64.5	23.9	29.6	37.2	22.6	33.5	73.4	40.4

Table 3.5: Seasonally adjusted provincial survey measurement summaries in Bq/m^3.

Basically, the seasonal adjustments reduce the average of the 2011 cross-Canada survey measurements by almost 20%.

The cumulative distribution frequency is plotted again with the seasonally adjusted measurements and is shown in Figure 3.8. The approximately 8.2% of adjusted measurements exceeding the 200 Bq/m^3 concentration is identified on the plot.

3.2.4 Proportion Exceeding the Recommended Action Level of 200 Bq/m^3

Health Canada recommends taking efforts to reduce radon concentration levels in homes that exceed 200 Bq/m^3. A population-weighted estimate of the proportion of Canadians exposed to a radon concentration of more than 200 Bq/m^3 is calculated by estimating the proportion of the population exceeding the level in each health region and then calculating the national proportion by weighting the health region proportions according to their population sizes in 2013. This was done by assuming the proportion of the population exceeding 200 Bq/m^3 for each health region was equal to the proportion of survey measurements exceeding 200 Bq/m^3

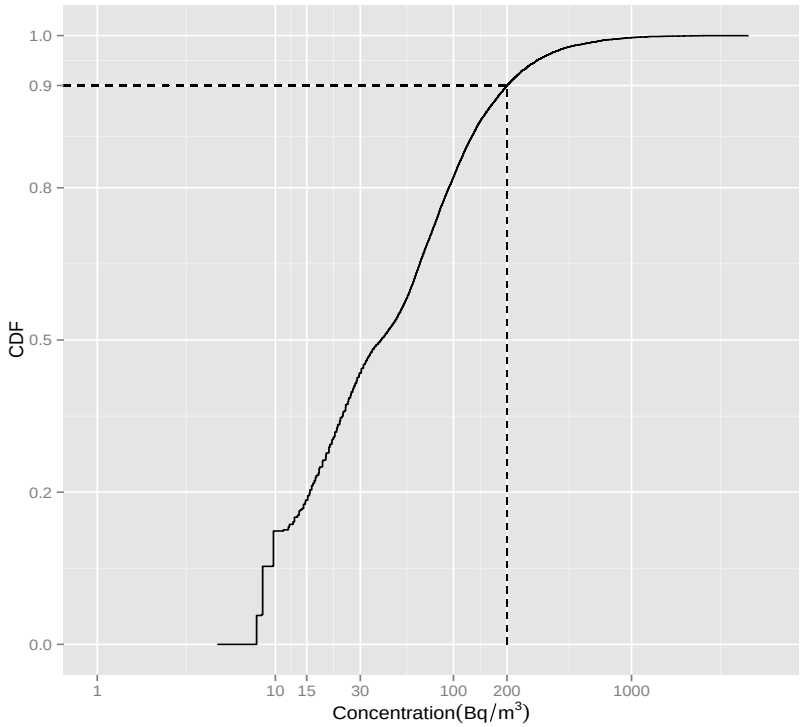

Figure 3.8: Cumulative Distribution Plot of the Seasonally-Adjusted Measurements: The dotted line identifies the Health Canada recommended action level of 200 Bq/m^3. Approximately 8.2% of the adjusted measurements are greater than the action level. This value does not represent a population-weighted percentage.

in that region. This resulted a nation-wide population-weighted estimate of 6.8%[3]. Accounting for seasonal adjustment factors, the estimated proportion exceeding the action level becomes 5%. If the distribution of radon concentrations in each health region is assumed to be log-normal and the geometric means and geometric standard deviations were calculated using the survey data from each region, the calculated Canada-wide proportion exceeding the 200 Bq/m^3 action level is 6.5% and 4.7% based on the unadjusted and seasonally adjusted measurements, respectively. For the sake of comparison, the same methods were applied to the 2013 Radon/Thoron survey. Recall that the geometric mean of this survey was higher than the Canada-wide survey - 73.8 Bq/m^3 compared to 49.1 Bq/m^3. Because the 2013 survey was conducted in the 33 census metropolitan areas of Canada, the population weighting is based on the population sizes of these areas (using 2014 census data), rather than health regions. The population-weighted estimate based on the log-normal parameters of the survey (i.e. the geometric mean and geometric standard deviation) is better suited to the Radon/Thoron survey as this survey exhibited a cleaner log-normal distribution. The population-weighted estimate of the proportion of Canadians exceeding 200 Bq/m^3 is 8.5%. Re-calculating, using the proportions of the survey measurements exceeding the action level for each of the census metropolitan regions (instead of the geometric mean and standard deviation), the population-weighted estimate is 6.4%. Note that these proportions estimated for the Radon/Thoron survey reflect measurements which are not seasonally adjusted, and are therefore a point of comparison with the proportions estimated using the unadjusted (for season) cross-Canada survey measurements presented above.

3.3 Chapter Summary

This chapter described Health Canada's 2011 Canada-wide radon survey. This survey sampled residential radon concentrations from all health units in Canada, resulting in approximately 14,000 radon measurements. Health Canada's survey protocol required participants to place a radon detector in the lowest floor level of their residence in which they spent four or more hours per day in. The three-month measurements were taken during the heating season (i.e. between October and March). Quality control efforts involved the use

[3]Recall that this value was calculated by Chen et al. [22] as 6.9%.

of duplicate and blank detectors.

The aithmetric mean of the survey was 97 Bq/m^3. The geometric mean was 49.1 Bq/m^3, and the geometric standard deviation was 3.1. Visual inspection of the survey measurements distribution reveals a bimodal distribution, the source of which was not identified. A log-normal plot further reveals the departure from a log-normal distribution. This is somewhat surprising, since the distribution of measurements from a 2012 Health Canada radon survey (of 4000 Canadian homes) which used the same protocol, strongly resembles a log-normal distribution. The 2011 survey concurs with previous surveys, in finding that radon concentrations in basements are higher than those on the first floors, which are in turn higher than concentrations on second floors.

Health Canada recommends homeowners take remedial action when residential radon concentrations exceed 200 Bq/m^3. The percentage of raw survey measurements exceeding this threshold is 8.2%. The population-weighted estimated of Canadian residences exceeding the threshold is 6.8%. If seasonal adjustment factors are applied to the survey measurements, as well as population-weighting, the figure is 5%.

Chapter 4

Methods

This chapter outlines the risk assessment methodology used to answer the research questions from Chapter 1. The main steps of the risk assessment are as follows:

1. Choose the exposure–response models to use in the assessment.
2. Calculate the model inputs as necessary.
3. Compute the ERR using the exposure–response models and exposure data.
4. Compute ILDs from the ERR.

4.1 Choice of Exposure–Response Models

The BEIR-VI models, which are based on occupational studies, continue to be well-recognized due to the depth of analysis and significant pooling of data on which they were built. The BEIR-VI committee presented both the EAC and EAD models as a means to assess risk from exposure, and stated that neither model was preferred over the other. As a result, both models will be included in the analysis of this thesis.

The EPA model is based directly on the two BEIR-VI models and thus does not offer significantly different or deeper insight into the exposure–response relationship. Results from the EPA model will therefore not be included.

Residential studies are more directly relevant to lung cancer risks from residential radon exposure than underground miner studies. However, residential models have not previously been used to project lifetime risk and there is no clear method for doing so based on the documented studies. Unlike the occupational models, which provide a method for estimating risk from exposures incurred at any point in life, the residential models provide risk estimates based on a fixed window of exposure (e.g. the period of time 5 to 30 years prior to the study), and so it is not known how to interpret lifetime risks from exposures outside of this window.

With these considerations, the analysis will include the BEIR-VI EAC and EAD models.

4.2 Inputs

4.2.1 Demographic Data

Various demographic data are necessary for coupling the ERR to base lung cancer rates. These include estimates of population sizes, all-cause death counts, lung cancer death counts, and smoking prevalence (estimates of the proportions of Canadians, stratified by age and province, who have ever smoked and never smoked). Data were collected for the most recent year(s) available.

Population

Statistics Canada conducts censuses, which attempt to enumerate the entire population of each province and territory and include Canadian citizens, landed immigrants, and non-permanent residents, as well as family members living with them. Censuses are conducted every five years with the most recent completed in 2011. Population sizes for years between censuses are estimated using the most recent census data and subsequent births, deaths, and international and inter-provincial migration. Canadian population size estimates for the years 2009-2011 are used in this thesis. These data were available from Statistics Canada in 5-year age categories up to 90 years of age. Counts for people older than 90 years were combined in a 90+ category. Gender proportions for each age category are calculated directly from the population size estimates (which

include the population size for each gender).

Death Counts

Provincial and territorial Vital Statistics Acts require all deaths to be registered in their respective Vital Statistics Registries. These death registrations, which include the medical certificate of cause of death, are sent to Statistics Canada.

As with the population counts, death counts were available from Statistics Canada in 5-year age categories for each both sexes for the 2009-2011 calendar years.

4.2.2 Smoking Prevalence

Smoking prevalence data were available from Health Canada for the 2012 calendar year. At the national level, smoking prevalence was published for males and females in 5-year age categories from ages 15 to 54, followed by a 55+ category. At the provincial level, smoking prevalence was published for 15-24 and 25+ age categories. The data for both levels (national and provincial) were collected as national and provincial risks will be calculated.

4.2.3 Radon Concentration Measurements

Survey Measurements below 15 Bq/m^3

As discussed in Chapter 3, the 2011 cross-Canada radon survey data-set only reported concentrations of 15 Bq/m^3 or more with other measurements reported as "<15 Bq/m^3". This analysis assumes a concentration of $15/\sqrt{2}$ Bq/m^3 for these measurements.

4.2.4 Seasonal Adjustments to Exposure Measurements

The radon concentration measurements from the cross-Canada survey were taken over a period of three months during the colder months (i.e., between the months of October and March), when residential concentrations are generally higher than the annual average. To compensate for this potential bias, seasonal

adjustment factors are applied to each measurement using values derived by Pinel et al. [17]. Their derivation used data from a survey of more than 2000 measurements from residences in England with a statistical model to derive seasonal correction factors for three-month and measurements. The correction factors are assigned to measurements based on the starting month and length of the measurement. This method was validated for Canadian measurements by Krewski et al. [16] by taking two consecutive six-month measurements (with different detectors) in over 4500 residences in Winnipeg, Canada. Reasonable agreement was found between integrated annual exposures measured by the two six-month measurement periods and the annual exposures predicted by applying the correction factors to the six-month measurements.

Since the measurements from the Health Canada survey were generally taken between November and March, average annual concentrations predicted by incorporating the seasonal correction factors will be lower than the reported values. Correction factors from Pinel et al. [17] are used in this thesis because they derived factors for three-month measurements (the same duration as the cross-Canada survey measurements), whereas Krewski et al. derived factors for six-month measurements in their study [16]. The correction factors from Pinel et al. [17] are shown in Table 4.1. Each correction factor is ascribed to a month; this is the factor that should be applied for a three-month measurement that begins in that month. For example, a three-month measurement beginning at the start of January and ending at the end of March should be multiplied by the value associated with January (0.74) to adjust for the seasonal variation associated this period.

Seasonal Adjustment Factors	
Month in which measurement commenced	Correction factor for three-month measurement
January	0.74
February	0.79
March	0.91
April	1.10
May	1.34
June	1.55
July	1.56
August	1.36
September	1.12
October	0.92
November	0.80
December	0.74

Table 4.1: Seasonal correction factors from Pinel et al. [17] are used to adjust the three-month measurements from the cross-Canada radon survey. The correction factor assigned to each month is the value that a three-month measurement beginning in that month is to be multiplied by. (e.g. a three-month measurement spanning January-March should be multiplied by 0.74)

4.3 Computing Excess Relative Risks (ERR) with the Models

The chosen models, which were introduced in Chapter 2, express risk per unit exposure and therefore must be multiplied by an exposure concentration to produce the corresponding ERR values. For practicality, it is assumed that individuals are exposed to a constant radon concentration for the duration of their lives (adjusted for the average fraction of time spent inside residences) as would transpire if they resided in the same house throughout their life, provided the measured radon exposure served as a reasonable proxy of the annual average characterizing that house. It should be noted that the models include factors dependent on attained age and time since exposure, and therefore may result in a unique ERR at each age. This age-specific ERR profile can be "collapsed" into single lifetime risk values using life-table methods, as will be demonstrated in section 4.5.

The remainder of this section provides detailed information necessary for implementation each of the selected models.

4.3.1 BEIR-VI Models

The BEIR-VI models are of the form:

$$ERR = \beta(\omega_{5-14} + \theta_{15-24}\,\omega_{15-24} + \theta_{25+}\,\omega_{25+})\phi_{age}\gamma_z \tag{4.1}$$

These models express ERR per unit exposure rate, where the exposure rate is in units WLM/y. Since the survey used in this thesis measured concentrations in Bq/m^3, exposure concentrations are converted to the equivalent WLM/y when using the BEIR-VI models. This conversion is described in Appendix A.

The parameters of the BEIR-VI models were described in Chapter 2. In their report, the BEIR-VI committee provided information on the uncertainty and variability of the model parameters, as well as the covariance among the parameters. This information will be used to calculate the uncertainty associated with risk estimates, as described in greater detail in Chapter 3. The central parameter values, as well as the covariance matrix for the EAC and EAD models, are shown in Tables 4.2 and 4.4, respectively. As discussed in Chapter 2, risk modifiers are used to account for a sub-multiplicative effect of smoking and radon exposure in combination. The ERR is adjusted by a factor of 0.9 for ever-smokers and 1.9 for never-smokers for both the EAC and EAD models.

BEIR-VI Exposure-Age-Concentration

Central Values (in Log Space) of Parameters[1]											
	β	θ_2	θ_3	ϕ_2	ϕ_3	ϕ_4	γ_2	γ_3	γ_4	γ_5	γ_6
	-2.5	0.8	0.6	-0.6	-1.2	-2.4	-0.7	-1.0	-1.1	-1.8	-2.2
Covariance Matrix[2]											
β	9.5										
θ_2	-0.4	0.8									
θ_3	0.0	0.2	0.4								
ϕ_2	-2.9	-0.1	-0.2	5.7							
ϕ_3	-3.2	-0.2	-0.3	2.8	10.9						
ϕ_4	-3.4	-0.2	-0.5	2.9	3.2	87					
γ_2	-5.6	-0.1	0.0	0.1	0.4	0.8	8.2				
γ_3	-6.4	-0.1	-0.1	0.2	0.5	0.9	5.9	6.9			
γ_4	-6.6	-0.2	-0.1	0.2	0.6	0.0	5.8	6.7	7.3		
γ_5	-6.9	0.0	-0.1	0.3	0.6	0.8	5.7	6.5	6.7	7.8	
γ_6	-7.0	0.0	-0.1	0.3	0.5	0.5	5.6	6.4	6.6	7.3	8.6

Table 4.2: Central estimates and covariance terms corresponding to an overall fit of the Exposure-Age-Concentration (EAC) model [6]. [1]Except for θ_2 and θ_3, estimates are in $log_e()$ scale. [2]Except for θ_2 and θ_3, estimates are in $log_e()$ scale. All values are multiplied by 100.

In the case of the EAC model, γ is a modifier dependent on exposure concentration, modifying the risk per exposure downward for greater exposure concentrations. The γ values are provided in Table 4.3 for each of the partitioned exposure ranges.

γ Modifiers[3]	
Exposure (WL)	γ_z
$< .5$	$\gamma_1 = 0.0$
0.5-1.0	$\gamma_2 = -0.7$
1.0-3.0	$\gamma_3 = -1.0$
3.0-5.0	$\gamma_4 = -1.1$
5.0-15.0	$\gamma_5 = -1.8$
15.0+	$\gamma_6 = -2.2$

Table 4.3: γ modifiers for the EAC (Exposure-Age-Concentration) model [6]. [3]Estimates are in $log_e()$ scale. The values repeat those shown in Table 4.2

BEIR-VI Exposure-Age-Duration

Central Values (in Log Space) of Parameters[1]										
	β	θ_2	θ_3	ϕ_2	ϕ_3	ϕ_4	γ_2	γ_3	γ_4	γ_5
	−5.2	0.7	0.4	−0.6	-1.3	-2.0	1.0	1.5	1.9	2.3
Covariance Matrix[2]										
β	8.0									
θ_2	−0.3	1.0								
θ_3	0.0	0.2	0.4							
ϕ_2	−2.1	−0.1	−0.2	4.3						
ϕ_3	−2.2	−0.2	−0.4	2.1	9.6					
ϕ_4	−2.4	−0.2	−0.6	2.1	2.4	95.4				
γ_2	−5.1	−0.2	−0.1	0.3	0.4	0.5	4.6			
γ_3	−5.6	−0.4	−0.2	0.3	0.5	0.9	4.7	5.9		
γ_4	−5.7	−0.4	−0.2	0.2	0.4	0.8	4.7	5.6	6.8	
γ_5	−5.7	−0.4	−0.1	−0.2	0.2	0.6	4.8	5.6	6.0	7.3

Table 4.4: Central estimates corresponding to an overall fit of the EAD (Exposure-Age-Duration model) [6]. [1]Except for θ_2 and θ_3, estimates are in $log_e()$ scale. [2]Except for θ_2 and θ_3, estimates are in $log_e()$ scale. All values are multiplied by 100.

For the EAD model, the γ modifier is based on the duration of the exposure. For a given total exposure, the model assumes that risk increases when the exposure is incurred over a longer duration. Table 4.5 shows the γ values, which are greatest for durations longer than 35 years.

γ Modifiers[3]	
Exposure Duration (yrs)	γ_z
< 5	$\gamma_1 = 0.0$
5-14	$\gamma_2 = 1.0$
15-24	$\gamma_3 = 1.5$
25-34	$\gamma_4 = 1.9$
35+	$\gamma_5 = 2.3$

Table 4.5: γ modifiers for the EAD (Exposure-Age-Duration) model [6]. [3]Estimates are in $log_e()$ scale. The values repeat those shown in Table 4.4

As with the EAC model, ERR risks are adjusted by a factor of 0.9 for ever-smokers and 1.9 for never-smokers.

4.4 Applying the Models

In this thesis, two types of risk estimates are calculated: 1) estimates that are based on point estimates of model parameters and exposure data, and 2) estimates that incorporate the uncertainty or variability of these estimates. In the case of the former, arithmetic means of the uncertain or variable parameters are used as the point estimates. Since the BEIR-VI model parameters are lognormal distributions presented in terms of geometric mean and geometric standard deviation, the arithmetic means were calculated as follows:

$$AM = exp\left(ln(GM) + \frac{ln(GSD)^2}{2}\right). \tag{4.2}$$

The arithmetic mean estimates calculated for the EAC and EAD models are shown in Tables 4.6 and 4.7, respectively.

Arithmetic Mean Values of EAC Parameters										
β	θ_2	θ_3	ϕ_2	ϕ_3	ϕ_4	γ_2	γ_3	γ_4	γ_5	γ_6
0.1	0.8	0.6	0.6	0.3	0.1	0.5	0.4	0.3	0.2	0.1

Table 4.6: Central estimates using arithmetic means corresponding to an overall fit of the EAD (Exposure-Age-Duration) model [6].

Arithmetic Mean Values of EAD Parameters									
β	θ_2	θ_3	ϕ_2	ϕ_3	ϕ_4	γ_2	γ_3	γ_4	γ_5
0.01	0.72	0.44	0.52	0.28	0.13	2.78	4.42	6.62	10.2

Table 4.7: Central estimates using arithmetic means corresponding to an overall fit of the EAD (Exposure-Age-Duration) model [6].

The arithmetic means calculated for the dosimetric modifier, K (for both BEIR-VI models), is $K = 1.151$

4.5 Life-Table Calculations

Life-table methods, as described in Chiang [57], Preston et al. [58], and Brand [59] can be used to summarize mortality patterns with a single index of lifetime health detriment (ILD). Life-tables characterize the expected

mortality experience of a populations members and are generally categorized as one of two types: *cohort* or *current* life-tables.

In this thesis, a *current* (or *period*) life-table is used, which is constructed using the age-specific mortality rates of a population during a recent period of time, often the most recent year for which data have been collected. This life-table is based on a hypothetical cohort who, at all ages of their lives, would be subjected to the corresponding mortality rates recorded during the period. The period is often chosen to span several years (as opposed to a single year) in order to "average out" random fluctuations in mortality rates. In this thesis, the period used for life-table construction is the three calendar years from 2009 through 2011, as data for these years were the most recent publicly available from Statistics Canada on the CANSIM website.

Another characteristic of the life-table is the discretization of age. A life-table constructed using single-year age categories is commonly referred to as a *complete* life-table. Alternatively, an *abridged* life-table uses age intervals greater than one year and may offer several advantages over complete life-tables (Chiang) [60], one being that data for single year intervals are often unavailable. The Statistics Canada data available on the CANSIM website is discretized in five-year intervals, therefore an abridged life-table with 22 five-year intervals from 0 to 110 years of age is used. The use of abridged tables also necessitates a further assumption of constant mortality rate, not only across the period, but also across age interval. However, errors introduced by this assumption are less significant when life-tables are used to calculate indices of change, as is the case in this thesis (i.e. errors are offset somewhat when the difference or ratio of indices are calculated for unexposed and exposed conditions).

4.5.1 Period Age-Specific Mortality Rates

Generally, a population faces an instantaneous and varying death rate. This is referred to as the *mortality rate* and is analogous to the concept of *hazard rate*, commonly used in reliability engineering. The mortality rate, $\mu(t)$, is not observable and in practice is estimated from the death rates observed for each age category over the period for which the life-table is constructed. The estimated death rates are assumed to remain constant within each age category and can be calculated as

$$M_i = \frac{Number\ of\ deaths\ over\ duration\ t\ in\ the\ category\ beginning\ at\ age\ i}{Number\ of\ person-years\ lived\ over\ duration\ t\ in\ category\ beginning\ at\ age\ i}, \tag{4.3}$$

where t is the length of the period and a person-year is one year lived by one person. Person-years for the period is estimated using the mid-period population sizes for each age category (i.e. the population sizes for each age category on July 1, 2010).

The calculated Canadian mortality rates for lung cancer and all causes using the 2009-2011 data are shown in Figure 4.1. These mortality rates, based on observed statistics, represent the *radon-exposed* mortality rates. However, based on the analysis by Nelson et al. [61], this thesis assumes the observed mortality rate as the baseline, *unexposed* rate, which is affected by radon exposure (according to the ERR). This analysis observed that the slope of the exposure–response (β), derived in [6] was underestimated because the miners' residential exposures were not taken into account. It was further determined that if the residential exposures of the population being assessed are similar to those of the miners, then the underestimate of β is approximately offset by proceeding as if the observed (exposed) mortality rates were actually the *unexposed* baseline rates.

4.5.2 Life Expectancy and Lifetime Risk

The life-table results used in this study can be derived from two primary indices, as indicated by Brand et al. [21]: Life Expectancy (LE) and Lifetime Risk (LR). Life expectancy is the most frequently used summary index of mortality and is easy to interpret by anyone. Lifetime risk represents the chance an individual has of dying from lung cancer, given the existing conditions. Lifetime risk provides a measure of the incidence and case-fatality of a disease.

LE and LR both require S_i, the probability of surviving from birth up to the beginning of the *ith* age interval, for all *i*. When subjected to the mortality rate, the probability of surviving an interval $[x, x + \Delta x]$, assuming survival to the start of the interval, is [58]

$$S_{[x,x+\Delta x]} = exp\bigg(- \int_x^{x+\Delta x} \mu(t)dt \bigg). \tag{4.4}$$

For convenience, the probability of survival from birth to the beginning of the *ith* age interval is denoted $S_i = S_{[0,i]}$. Substituting the approximation $\mu_i = M_i$,

$$S_i = exp\Big(-\sum_{k=1}^{i-1} 5M_k \Big) \tag{4.5}$$

is obtained.

The factor of 5 in the summation of equation 4.5 accounts for the cumulative exposure to the mortality rate one faces throughout the five-year duration of each age category.

The probability of dying in the *ith* interval, assuming survival to the beginning of the interval, is denoted q_i:

$$q_i = 1 - exp(-5M_i) \tag{4.6}$$

Life expectancy is

$$LE = 5\sum_{i=1}^{21} S_i - 5\sum_{i=1}^{21} S_i q_i (1 - \alpha_i) \tag{4.7}$$

The first term in 4.7 represents the life expectancy if everyone died at the end of the last age interval they lived to. Because deaths will occur somewhere *within* the last age interval survived to, the second term in 4.7 must be subtracted to ensure only the fraction of the last age interval lived is included in the total. The average fraction of the *ith* age interval lived by those who die in that interval is α_i. This analysis uses α_i=0.5 for all i, which is common practice, according to Chiang [60].

The lifetime risk (LR) risk is the probability of dying from lung cancer:

$$LR = \sum_{i=1}^{22} S_i q_i \psi_i. \tag{4.8}$$

Here, we once again use S_i and q_i, as defined earlier. ψ_i is the *intensity ratio* defined as $\frac{\mu_i^L}{\mu_i}$, where μ_i^L and μ_i are the mortality rates for lung cancer and all causes, respectively. ψ_i is estimated as the ratio of the lung cancer death counts and all-cause death counts in the *ith* age interval, $\psi_i = \frac{D_i^L}{D_i}$. The product $S_i \times q_i \times \psi_i$ is the probability of surviving to the *ith* interval and then dying from lung cancer in that interval. Equation

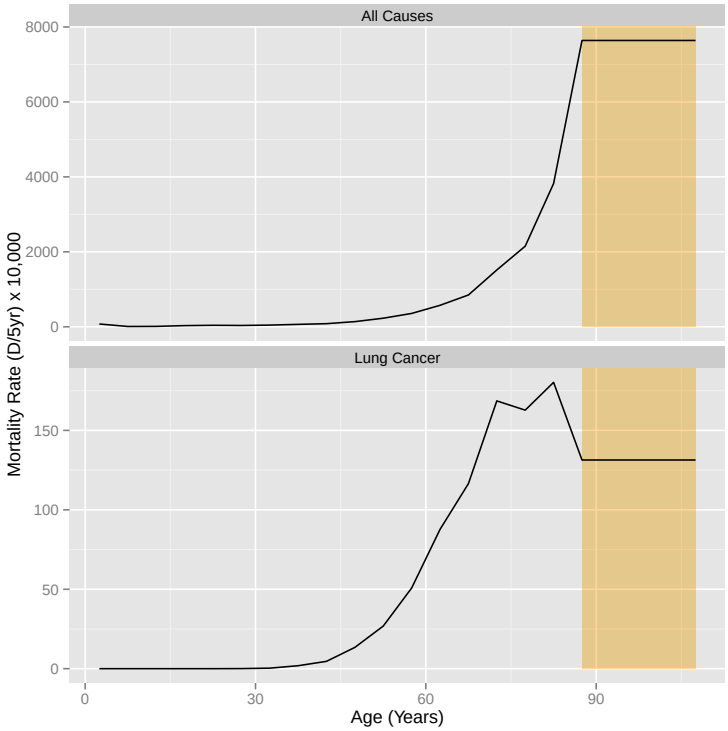

Figure 4.1: Age-specific mortality rates estimated for the Canadian population, averaging data across the years 2009 - 2011. The orange shaded region corresponds to the open "90+" age category. Mortality rates are assumed to be constant throughout the "90+" category and are set equal to the rate calculated for the "85-90" age category. Note that graph captures mid-age values so that the plot starts at age 2.5, rather than age 0. The dip in mortality rates between 75 and 80 years of age may be due to an effect from cohort patterns (related to smoking habits) affecting trends in age-specific death rates, as explored by Ouellette et al. [62].

4.8 is the summation of the probabilities of dying from lung cancer at each age category, and is the lifetime probability of dying from lung cancer.

4.5.3 Incorporating Radon Exposure into the Life-Table Analysis

In order to infer the health detriment from radon exposure, the period life-table can be re-constructed to reflect the added impact of radon exposure (as mediated through an increment in lung cancer rates).

As described by Brand et al. [21], the increase in lung cancer mortality of an individual exposed to radon is $\mu_i^L \varepsilon_i$, where ε_i is the age-specific ERR. The modified lung cancer mortality rate of an *exposed* individual is

then the sum of the baseline mortality and exposure-related mortality, $\mu_{Ei}^{L} = \mu_i^L + \mu_i^L \varepsilon_i$, where the subscript E denotes an exposed individual. In terms of the intensity ratio (ψ_i), $\mu_{Ei}^{L} = \mu_i \psi_i (1 + \varepsilon_i)$. The resultant all-cause mortality for an individual exposed to radon is now

$$\mu_{Ei} = \mu_i + \mu_i^L \varepsilon_i = \mu_i + \psi_i \mu_i \varepsilon_i = \mu_i (1 + \psi_i \varepsilon_i). \tag{4.9}$$

Approximating $M_i = \mu_i$, the above expressions for mortality can be substituted into 4.5 and 4.6 to yield

$$S_{E_i} = exp\left(-5 \sum_{k=1}^{i-1} M_k (1 + \psi_k \varepsilon_k) \right). \tag{4.10}$$

and

$$q_{E_i} = 1 - exp(-5 M_i (1 + \psi_i \varepsilon_i)), \tag{4.11}$$

The intensity ratio for an exposed individual is

$$\psi_{E_i} = \frac{\psi_i (1 + \varepsilon_i)}{1 + \psi_i \varepsilon_i}. \tag{4.12}$$

For the exposed population, the lifetime risk and life expectancy (LR_E and LE_E, respectively) can be calculated substituting equations 4.10, 4.11, and 4.12 into equations 4.7 and 4.8.

Once the radon-modified LE and LR indices are found, the health detriment can be summarized by calculating ILDs, such as ELRR or LYL, which reflect the change from unexposed to exposed conditions. ILDs such as these, which reflect change, are one of the very useful applications of life-tables as they convey the assessment of health detriment with a single number.

4.5.4 Life-Years Lost (LYL)

A summary which concisely captures the comparison of LE and LE_E is *life-years lost*. LYL is the difference in life expectancy between exposed and baseline conditions and represents the shortening of expected lifespan due to exposure.

$$LYL = \Delta LE = LE - LE_E. \tag{4.13}$$

4.5.5 Excess Lifetime Risk Ratio (ELRR)

The *excess lifetime risk ratio* conveys the relative impact from exposure on lifetime risk with a single value. The lifetime risk ratio for lung cancer is the ratio of the lifetime risk under exposed conditions divided by the the lifetime risk without exposure:

$$LRR = \frac{LR_E}{LR} \tag{4.14}$$

ELRR is simply the this ratio minus one:

$$ELRR = \frac{LR_E}{LR} - 1 \tag{4.15}$$

ELRR is thus the risk increase one faces from the exposure, as a proportion of their unexposed risk. As an example, if the lifetime risk of dying from lung cancer is 8% for someone not exposed to residential radon and 9% for someone who is exposed, the ELRR is 0.125.

4.5.6 Population Attributable Risk (PAR)

The *population attributable risk* is defined as the fraction of the cause-specific mortality that could be avoided if the exposure were eliminated (Lubin and Boice) [63]. It captures the the fraction of incidence among the general population (including those exposed) that is in excess of what it would be in the absence of exposure (i.e. the proportion of lung cancer deaths caused by radon exposure). For example, if 20,000 Canadian deaths in a given year were caused by lung cancer, and the lung cancer PAR for radon was 10%, 2000 of these deaths are attributed to radon exposure.

The PAR is calculated as

$$PAR = \frac{\overline{LR}_E - LR}{\overline{LR}_E} = 1 - \frac{1}{\overline{ELRR} + 1}, \tag{4.16}$$

where $\overline{LR}_E$ and $\overline{ELRR}_E$ are the exposed, population-averaged values and LR is the baseline lifetime risk of lung cancer for unexposed conditions. PAR thus averages out variability (due to parameters that vary from individual to individual, such as exposure concentration and the K-factor) and is only subject to uncertainty.

4.5.7 Lung Cancer Force of Mortality for Never-Smokers and Ever-Smokers

Risks predicted by the BEIR-VI models are dependent on smoking history. In deriving the models, the BEIR-VI committee exposure-response slopes for miners who were never-smokers and ever-smokers. Consequently, the ILDs for ever-smokers and never-smokers are computed separately in this thesis. Calculation of the implied lung cancer and all-cause mortality rates for ever- and never-smokers, requires an estimate of the age-dependent smoking prevalence. The estimated ever-smoker proportions for the Canadian population in 2012 are displayed in Table 4.8.

Age	Females	Males
15-19	11.4	14.3
20-24	23.2	29.3
25-29	34.4	44.9
30-34	34.4	44.9
35-39	33.5	45.5
40-44	33.5	45.5
45-49	45.7	51.4
50-54	45.7	51.4
55+	46.9	69.8

Table 4.8: Estimated Canadian ever-smoker proportions (%).

The lung cancer mortality rates for ever- and never-smokers are calculated for each age category using

$$\mu^L_{pop} = (1-p)\cdot\mu^L_{NS} + p\cdot\mu^L_{ES}, \tag{4.17}$$

where μ^L_{pop} is the overall lung cancer mortality rate for the population, μ^L_{NS} and μ^L_{ES} are the lung cancer mortality rates for never-smokers and ever-smokers, and p is the ever-smoker prevalence. The relative risk of lung cancer (in each age category) due to smoking (RR), can be used to substitute for μ^L_{ES}:

$$\mu^L_{pop} = (1-p) \cdot \mu^L_{NS} + p \cdot RR \cdot \mu^L_{NS}. \tag{4.18}$$

RR is the relative risk of dying from lung cancer for ever-smokers, relative to never-smokers. A RR of 13.2 for males and 7.6 for females was calculated[1] as follows:

1) Relative risks for current and former smokers from a 2012 study (Pesch et al.) [64], based on data from the American Cancer Society Cancer Prevention Study 1 and 2 were used;

2) The ever-smoker RR was calculated for Canadians as a weighted average of the current and former smoker RRs based on 2012 Canadian proportions published by Health Canada [23].

Returning to Equation 4.18, the left side is observed and two of three terms on the right side are known (p, RR). The equation can thus be rearranged to solve for the unknown μ^L_{NS}:

$$\mu^L_{NS} = \frac{\mu^L_{pop}}{(1-p) + p \cdot RR} \tag{4.19}$$

The ES mortality rate can then be calculated as:

$$\mu^L_{ES} = RR \cdot \mu^L_{NS}. \tag{4.20}$$

This process can be applied to all age categories, resulting in the never- and ever-smoker lung cancer mortality rates for all ages. Figure 4.2 shows the calculated never- and ever-smoker lung cancer mortality rates as a function of age.

[1]The relative risks for current and former smokers from Pesch et al. [64] used in this calculation applied to all ages. Accordingly, this thesis calculates a single RR for each sex, which does not vary with age.

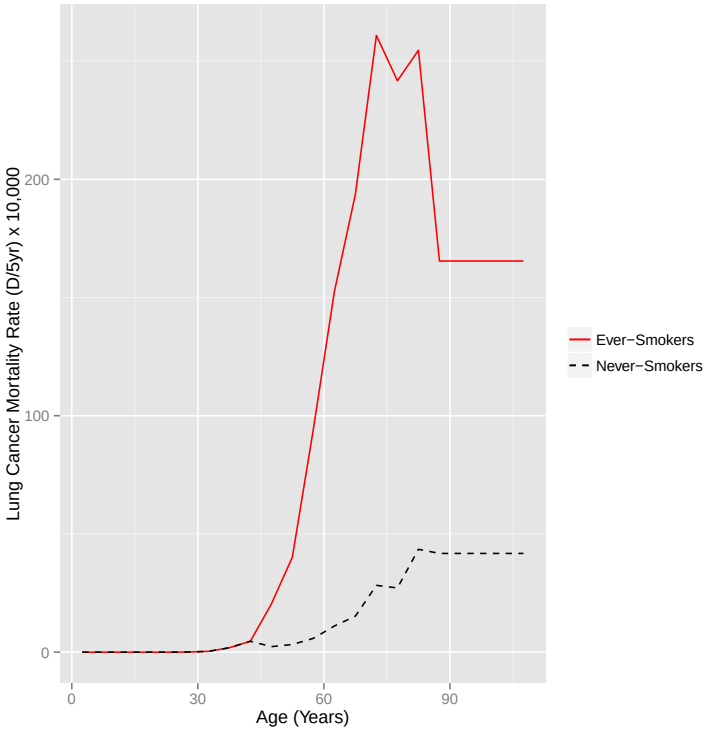

Figure 4.2: The calculated mortality rates for never- and ever-smoker Canadians are shown. These rates were calculated from 2012 smoking prevalence data from Health Canada, relative lung cancer risks for current and former Canadian smokers for 2012 from Health Canada [23], and relative lung cancer risks for males relative to females from Pesch et al. [64].

4.6 Uncertainty and Variability

The risk values calculated in this thesis are subject to numerous sources of uncertainty and variability. The main sources, including those discussed in earlier sections, are identified in this section.

4.6.1 Radon Survey Data

The survey measurements were taken over three-months, which is relatively short because radon concentrations often vary from one season to the next. Detectors could be mishandled or not installed according to the survey protocol. It may be natural to place the detectors in corners of rooms or near walls, where concentrations may be higher (from building materials or entry through cracks) so that they are not obtrusive.

Measurements in such a scenario may not be representative of exposures, even those incurred in the same room as the detector. Additionally, the use of seasonal adjustments is used to compensate for a potential biases in the measured values due to the season they are obtained, but uncertainty is inherent in the use of these adjustments, which are based on average seasonal variations. However, positive and negative errors from using seasonal measurements are expected to somewhat offset each other in the overall calculations of population-level risks.

The survey is subject to sampling error, since measurements were obtained for a fraction of the Canadian residences. As with all surveys, there are also effects associated with the design of the survey, which would introduce uncertainty.

Finally, there are relatively smaller uncertainties associated with determination alpha track counts (and inferred radon concentration) from the detector material in the laboratory. Alpha track radon measurements are subject to calibration errors and measurement errors, as discussed in Appendix C. Low residential radon concentrations may be below a level at which the concentration can be measured reliably - the minimum detectable level - and are subject to greater error. For this reason the, measurements below 15 Bq/m^3 in the 2011 cross-Canada survey were identified as"$<$ 15 Bq/m^3". Assigning a value of half the limit of detection (LOD) for contaminant concentrations below the LOD is common, according to [65]. However, assigning a value of the LOD divided by the square root of two, as was done in this thesis, has been assessed to incur less error [65], [66]. The error incurred by measurements below the LOD is anticipated to have a very small effect on the risk results since the majority of the measurements are well above the 15 Bq/m^3 threshold and small exposures contribute only slightly to the overall population risks.

4.6.2 Demographic Data and Calculations

Demographic data subject to uncertainty include data such as death counts, population, smoking prevalence, gender proportions, and relative risks of smoking-induced lung cancer for males and females.

The smoking exposure data are from a survey (i.e., the Canadian Tobacco use Monitoring Survey) and are disposed to sampling error. The 2012 smoking prevalence from this survey for all Canadians aged 15

or more was 16%. Another survey, the Canadian Community Health Survey, found smoking prevalence among all Canadians aged 12 or older in 2012 was 20.3%. Importantly, smoking prevalence has been steadily decreasing in Canada and is expected to be lower today and in the future, resulting in lower lung cancer risks from radon exposure. Sensitivity of LYL to smoking prevalence is explored in Chapter 5 (see Figure 5.3).

Overall population ILDs are generated as a weighted average of male and female ever- and never-smokers. In additions to gender proportions, this calculation requires an uncertain estimate of the ever-smoker proportion of the male and female populations. The calculation of the lung cancer force of mortality for ever-smokers and never-smokers, as described in section 5.1, is also based on the ever-smoker proportion, as well as uncertain relative risks of lung cancer for ever- and never-smokers.

4.6.3 Residential Occupancy

The occupancy factor (assumed to be 0.7) is variable from one individual to the next. Use of the occupancy factor may also bias total radon-induced lung cancer risks downward since individuals may receive radon exposures when not inside their residences. Although outdoor radon levels are generally low (i.e., less than 15 Bq/m^3) and are not practically avertable, the time spent outside of the residence may involve exposures indoors (e.g. at a workplace) which may be similar to the concentrations in residences. Excluding radon exposures incurred outside of the residences provides results which are specifically attributed to residential radon exposures (the goal of this thesis), rather than all radon exposures.

A further source of uncertainty related to occupancy is the distribution of time individuals spend on different floor levels in multi-level residences. Health Canada's protocol for radon measurements requires homeowners to place the detector in the lowest floor level at which the resident spends at least four hours per day. As shown in Chapter 3, lower floor levels generally have higher radon concentrations. As a result, representing occupants' average exposures with a measurement from the prescribed floor level is likely to overestimate their actual exposures incurred from occupancy habits which may actually be distributed over two or more floors. An occupancy factor of 0.7 equates to spending 17 hours per day in the residence.

Placing the detector on the lowest floor in which one spends at least four hours implies that as much as two thirds of the time could have been spent on a higher floor (with typically lower radon concentrations).

4.6.4 Equilibrium Factor

Use of the occupational models requires converting survey measurements, in Bq/m^3, to WLM/y. This conversion is dependent on the equilibrium factor, F, which is variable from one residence to the next. A value of 0.4, which is normally assumed for indoor settings, is used in this thesis. Variability in this factor was assessed in [7] and it was found that F was within 30% of the assumed value of 0.4 for most residences in which measurements (of the equilibrium factor) were taken.

4.6.5 Threshold

The presence of a threshold, or even radiation hormesis, may introduce significant uncertainty. Although studies which identify a threshold for low radiation levels generally don't apply to alpha radiation, a recent case-control study in the US by Thompson et al. that found a reduction in risk associated with radon exposures of less than 150 Bq/m^3 [29]. It should be noted that this analysis was a case-control study with less than 600 subjects. However, if such a threshold exists, risk from radon exposure would be significantly lower than those predicted by the LNT models. A threshold sensitivity analysis is performed in Chapter 5.

4.6.6 BEIR-VI Exposure–Response Models

Structural uncertainty exists due the lack of understanding of the true form of the exposure–response. Structural uncertainty is accounted for to some degree by the use of two models (the EAC and EAD) derived by the BERI-VI committee from the miner data.

The K-factor accounts for diverse environmental and physiological factors associated with extrapolation from the occupational setting to the residential setting and is therefore subject to substantial uncertainty and variability.

The BEIR-VI committee acknowledged significant uncertainty in the effect of smoking on modifying risks from radon exposure. Sparce information on smoking (limited information on smoking habits was available

for 6 of the 11 study cohorts) limits the BEIR-VI committee's characterization of the combined effects from smoking and radon exposure. Consequently, the study considered only whether subjects were ever-smokers or never-smokers. Moreover, since nearly all of the miners studied were ever-smokers, the results for never-smokers are more uncertain than those for ever-smokers.

The BEIR-VI committee attempted to quantify the uncertainty (and variability) for a number of the significant inputs used in their models (with the notable exception of the modifier for smoking history, δ). These quantified uncertainties will be incorporated in the results from these models in Chapter 5.

4.6.7 Incorporating Uncertainty

Uncertainty and variability in the BEIR-VI model parameters was quantified by the BEIR-VI committee in their analysis, and is readily incorporated in this thesis. Variability in the radon survey is also approximated by the distribution of the survey measurement results.

Uncertainty and variability can be estimated for the outputs of mathematical operations (e.g. exposure–response models and life-table methods) using Monte Carlo analysis, a numerical simulation technique. Uncertainty in model inputs and parameters is transmitted to the model outputs by randomly drawing sets of values from the uncertain input distributions and computing an output value from each set. When the number of such iterations, U, is large enough so that the distribution of output values is stable with respect to changes in U, the set of output values characterize the corresponding uncertainty of the output. This set of output values is essentially a simulated set of sample values for the joint distribution of all of the random variable model inputs [51]. The process of uncertainty propagation using Monte Carlo methods is illustrated in Figure 4.3, where U represents the number of draws necessary to achieve the necessary stability [2] of the output distribution.

[2]Theoretically, this is an infinite number of draws. The simulation is considered stable when the number of draws achieves a distribution that will not change significantly, with respect to the goals or desired outputs of the simulation, with further sampling.

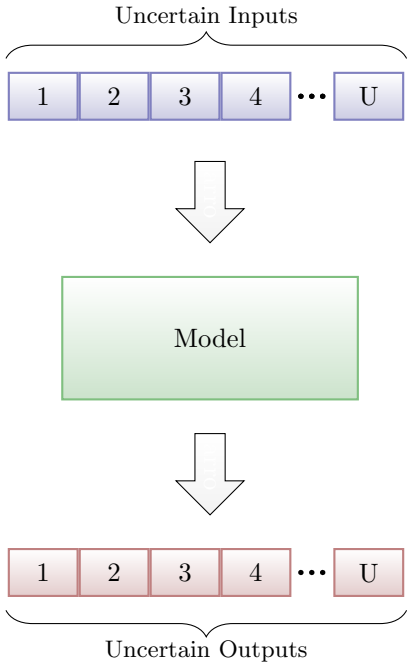

Figure 4.3: Propagation of uncertainty: U sets of inputs are applied to the model and produce U outputs. Each of the U sets of inputs represents the full set of model parameters, where the values for the parameters were drawn from their respective uncertainty distributions.

Variability can be propagated in combination with uncertainty by structuring the parameter draws as a two-dimensional array of size $U \times V$, reserving one dimension for uncertainty and one for variability.

In the case of the BEIR-VI models, the distribution for the K-factor is itself uncertain, adding an extra dimension of uncertainty. A three-dimensional array of size $U \times V \times H$ can be used to incorporate the hyperparameter uncertainty. Propagation of these three dimensions through a model is illustrated in Figure 4.4.

In this thesis, each MC iteration draws model parameters from the multivariate probability model, which are shown in Tables 4.2 through 4.5. Canadian exposure concentration variability is represented by randomly sampling, with replacement, from the cross-Canada survey measurements (adjusted for season). In each iteration, an ERR is generated from these parameters for each age category using the exposure-response model. The ILDs are calculated from this ERR and the mortality data (i.e. overall and lung cancer mortality

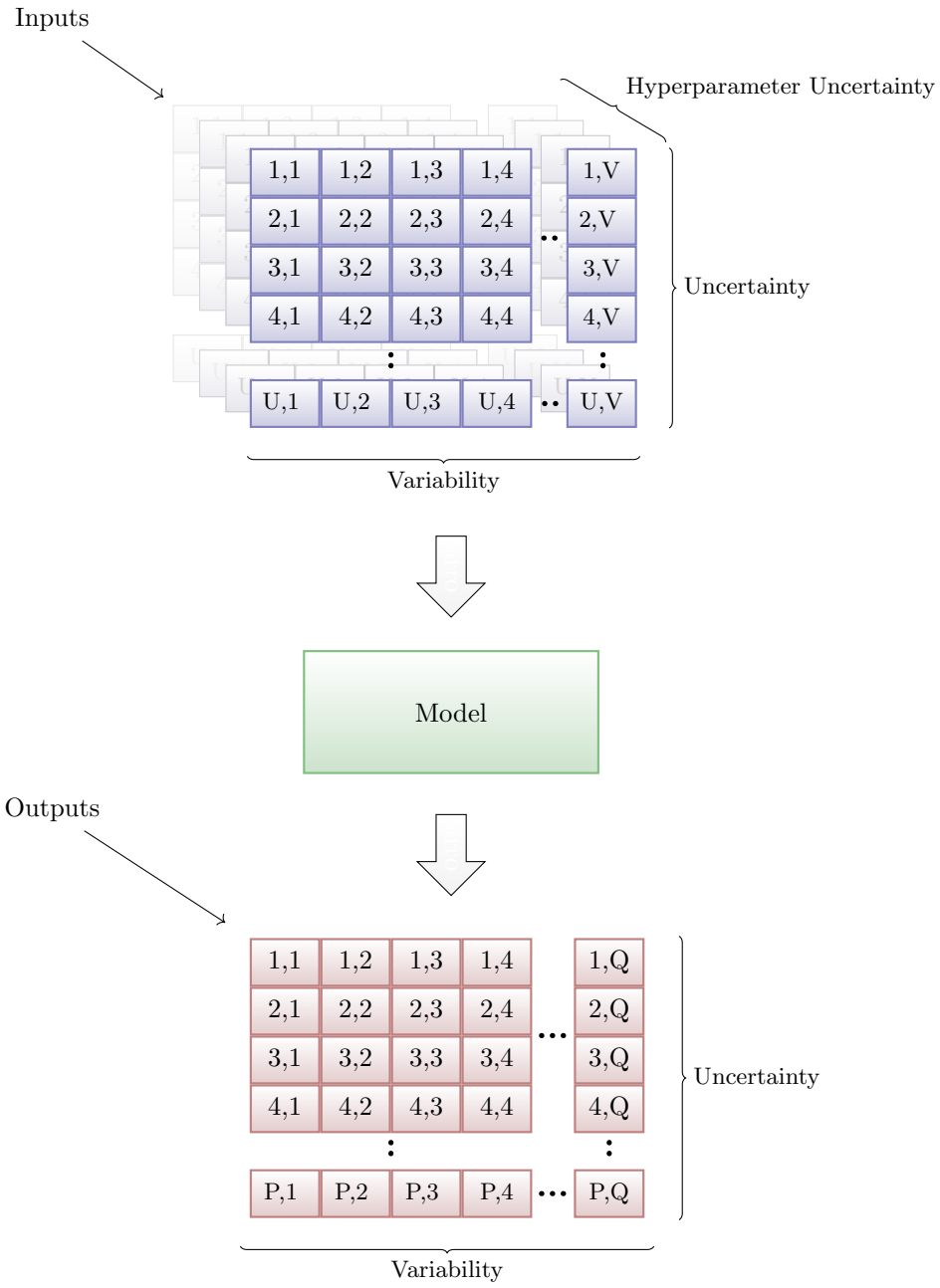

Figure 4.4: Propagation of uncertainty, variability, and hyperparameter uncertainty through a model. UxVxH Monte Carlo simulations are performed, incorporating uncertainty, hyperparameter uncertainty and variability. Each node represents a set of model input values, randomly drawn from the distributions for each input, that would be used for a single simulation. The simulations result in outputs that are both uncertain and variable.

rates under exposed and unexposed conditions) using the life-table calculations shown in Section 4.5. This process is repeated until stable distributions are obtained for female never-smokers, female ever-smokers, male never-smokers, and male ever-smokers separately. To combine the four separate sets and obtain overall population results, each of the four sets is resampled so that the relative number of samples in each of the categories matches the proportions of the Canadian population which fall in each of the categories. The resampled sets are then combined into a single collection, representing the overall population distribution for the ILDs.

Although the uncertainty associated with the possibility of a threshold is not quantified, a sensitivity analysis is conducted by calculating LYL under the assumption that a threshold exists at three levels: 50 Bq/m^3, 100 Bq/m^3, and 150 Bq/m^3. This is achieved for each of the three cases by setting survey measurements below the threshold level to 0, effectively associating no risk to these exposures.

The structural uncertainty represented by the two BEIR-VI models is also incorporated by computing results with both models (EAC and EAD).

4.7 Chapter Summary

The main steps of the methodology were to select the exposure–response models, calculate the inputs for the model, compute the ERR using the models and inputs, and calculate the ILDs from the ERR values and mortality data.

The BEIR-VI EAC and EAD models were selected for use because they are based on significant pooling of data from occupational studies and continue to be regarded as two of the preeminent exposure–response models.

Inputs for the exposure–response models and life-table methods include demographic data (e.g. lung cancer mortality data, all cause mortality data, smoking prevalence, gender proportions), radon survey measurements, and seasonal adjustment factors.

Age-specific ERRs computed from the exposure–response models were used to calculate mortality rates for the radon exposure conditions in Canada. Differences in mortality experiences caused by radon exposure

were summarized with ILDs using life-table methods. ELRR, LYL, and PAR were calculated for never-smoker and ever-smoker males and females and were combined to form overall ILDs for the Canadian population based on smoking prevalence and gender proportions.

Uncertainty and variability in the exposure–response models and radon survey were incorporated using Monte Carlo simulation. The effect of a potential threshold in the exposure–response was investigated with a sensitivity analysis by replacing survey measurements below candidate threshold levels of 50, 100, and 150 Bq/m^3 to zero. Structural uncertainty was incorporated by calculating results from the EAC and EAD models.

Chapter 5

Results

Results are presented in terms of the ILDs described in Chapter 4: Excess Lifetime Relative Risk (ELRR), Population Attributable Risk (PAR), and Life-years Lost (LYL). Unless otherwise stated, the results are computed using the Canadian population characteristics (e.g. mortality rates, etc.) as described in Chapter 3. The ELRR and LYL values reported pertain to the approximately 80% of Canadians exposed to residential radon. The calculated PAR takes this fraction into account[1].

Plots of the ELRR vs. exposure concentration for the selected models is shown in Figure 5.1. The slopes of the plots are dependent on a number of factors, including the model parameters, such as β, and characteristics of the population, such as the mortality rates, or smoking prevalence. The plotted range of 0 - 1000 Bq/m^3 encompasses approximately 99.7% of the seasonally-adjusted survey measurements. This plot provides two main insights: first, there is a significant difference in health detriment predicted by the different models, even between the EAC and EAD models, which were derived from the same data; second, the ELRR is approximately linearly proportional to the exposure concentration for all models in the plotted range.

[1]The value of 80% represents the fraction of Canadians who live on the 2nd or lower level of a residence. Here, it is assumed to be constant for all regions of Canada.

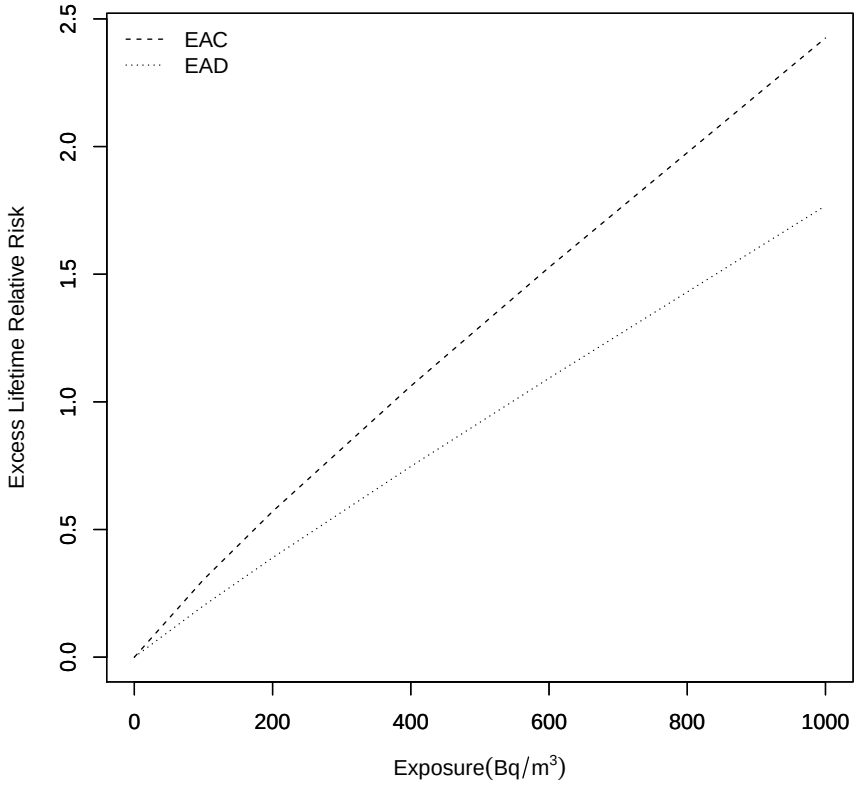

Figure 5.1: ELRR (Excess Lifetime Relative Risk) for the Canadian population is plotted against exposure for the EAC (Exposure-Age-Concentration) and EAD (Exposure-Age-Duration) models [6]. Note that within this range, none of the concentration modifiers of the EAC model (γ) come into play, hence the ELRR plot for the EAC model does not exhibit any of the effects (e.g. drops in ELRR) that would occur from these modifiers.

Figure 5.2 shows a density plot of the seasonally-adjusted survey measurements superimposed on the plot of ELRR vs. exposure for the estimated 96% of Canadians (population-weighted and seasonally adjusted value) exposed to average concentrations less than 200 Bq/m^3. It can be seen from this plot that the health detriment is significantly greater for the highest exposed individuals than for the majority of the population.

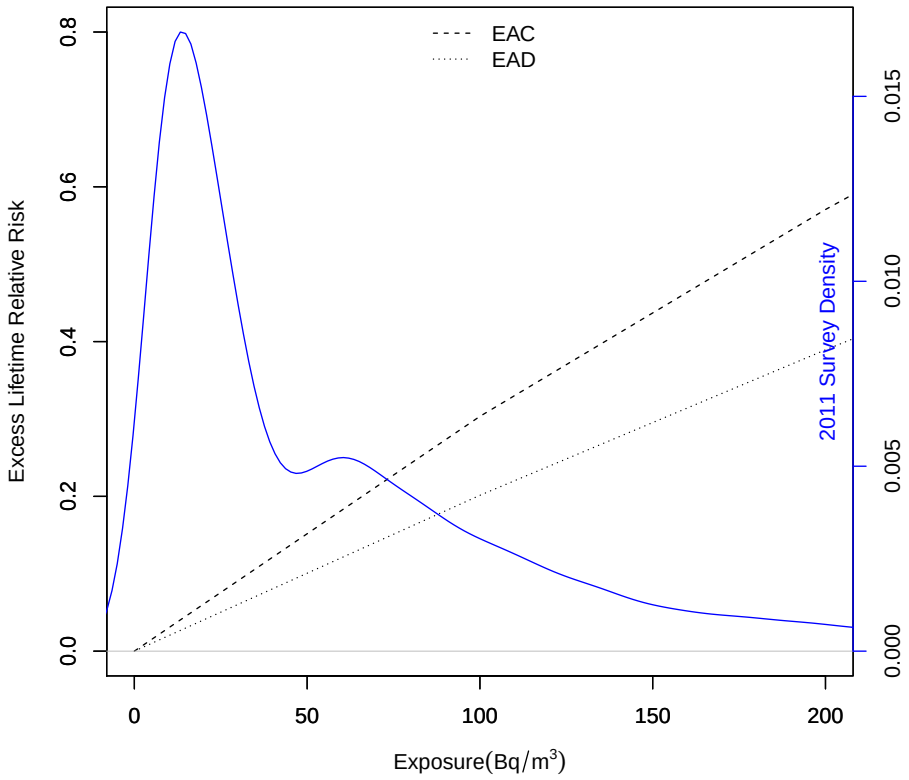

Figure 5.2: ELRR (Excess Lifetime Relative Risk) is plotted against Exposure for the EAC (Exposure-Age-Concentration) and EAD (Exposure-Age-Duration) models [6] in the 0-200 Bq/m^3 using the seasonally-adjusted measurements. An estimated 96% of Canadians are exposed to concentrations within this range. A smoothed density plot for the 2011 Health Canada radon survey measurements is also shown in blue. Note that the non-zero plot below 0 Bq/m^3 is misinformative and results from smoothing of the density plot.

5.1 Effect of Seasonal Adjustments on ILDs

The seasonal adjustments modify the magnitude of the exposure concentration. As can be seen in Figures 5.1 and 5.2, the computed ELRR is proportional to the exposure concentration for all models in the plotted range. Consequently, a change in estimated exposure magnitude (such as that imposed by a seasonal adjustment) results in a proportional change in ELRR. Brand et al. [21] found this proportionality also holds for LYL in

their exploration of heuristics relating the BEIR-VI EAC inputs to the ILDs [21]. For ELRR and LYL, results computed using unadjusted exposures can therefore be quickly converted to reflect results for seasonally-adjusted exposures by multiplying the ILD by the seasonal adjustment correction factor and vice-versa.

The average conversion factors used for all measurements Canada-wide, as well as for each province, are shown in Table 5.1. Results are presented hereafter using only the seasonally-adjusted measurements.

	AB	BC	MB	NB	NL	NS	ON	PE	QC	SK	CAN
Ave Correction Factor	0.83	0.81	0.84	0.83	0.81	0.81	0.82	0.85	0.85	0.83	0.83

Table 5.1: Average seasonal correction factors applied to the cross-Canada survey measurements for Canada and Provinces. Note that the different average correction factors among the provinces is a function of the different temporal distributions of the radon measurements from one province to the next (e.g. the average measurement start date may be earlier or later depending on the province).

5.2 Point Estimates

Distributions reflecting uncertainty or variability of model parameters were estimated by BEIR-VI for the EAC and EAD models and described in terms of the geometric means and geometric standard deviations. Point estimates of ELRR, PAR, and LYL resulting from the EAC and EAD models are computed using the average values for exposure and model parameters, which are documented in Tables 5.2 and 5.3, respectively.

5.2.1 Canadian and Provincial Estimates

The results from the EAC model are shown at the national and provincial level in Table 5.2. The ELRR and LYL results are applicable to the approximately 80% of Canadians considered to be exposed (i.e. those inhabiting the basement, 1st, and 2nd housing levels).

	AB	BC	MB	NB	NL	NS	ON	PE	QC	SK	CAN
Arith. Mean	75.99	56.15	126.07	148.79	50.34	75.96	67.56	39.79	69.06	104.81	80.23
ELRR	0.45	0.34	0.75	0.88	0.30	0.45	0.40	0.24	0.41	0.63	0.48
PAR	0.24	0.20	0.33	0.37	0.18	0.24	0.23	0.15	0.23	0.30	0.26
LYL	0.28	0.21	0.46	0.55	0.19	0.28	0.25	0.15	0.26	0.39	0.31

Table 5.2: ILDs are shown for the EAC model [6] and the 2011 cross-Canada radon survey using point estimates. The arithmetic mean concentration units are Bq/m^3, LYL is years, and ELRR and PAR are dimensionless. The ELRR and LYL results apply to the 80 percent of Canadians considered exposed (this value is assumed to be constant across all regions of the country).

Recall that Brand et al. [21] assessed radon lung cancer risks using the EAC model in 2005. ELRR, PAR, and LYL estimates from that assessment were 0.08, 0.08, and 0.10 years, respectively. As displayed in Table 5.2, results from this assessment are 0.48, 0.26, 0.31, respectively for the same indices. The main factor causing the higher risk results here are the significantly greater measured concentrations from the 2011 cross-Canada radon survey (arithmetic mean of 97 Bq/m^3), in comparison to the 1978 survey (arithmetic mean of 28.3 Bq/m^3) used by Brand et al. [21]. Other factors that are different between these assessments are the demographic data, as well as the use of seasonal correctional factors in this thesis.

The results from the EAD model are shown in Table 5.3. As with earlier studies, the EAD model predicts lower ILDs compared to the EAC.

	AB	BC	MB	NB	NL	NS	ON	PE	QC	SK	CAN
Arith. Mean	75.99	56.15	126.07	148.79	50.34	75.96	67.56	39.79	69.06	104.81	80.23
ELRR	0.30	0.22	0.49	0.58	0.20	0.30	0.26	0.16	0.27	0.41	0.31
PAR	0.18	0.14	0.26	0.29	0.13	0.18	0.17	0.11	0.17	0.23	0.19
LYL	0.18	0.13	0.30	0.35	0.12	0.18	0.16	0.09	0.16	0.25	0.19

Table 5.3: ILDs are shown for EAD model [6] and the 2011 cross-Canada radon survey using point estimates. The arithmetic mean concentration units are Bq/m^3, LYL is years, and ELRR and PAR are dimensionless. The ELRR and LYL results apply to the 80 percent of Canadians considered exposed (this percentage is assume to be constant across all regions of the country).

5.2.2 Effects of Smoking

The BEIR-VI committee found a sub-multiplicative synergy for smoking and radon exposure in combination, accounted for by a factor, δ, which is equal to 0.9 for ever-smokers and 1.9 for never-smokers. Moreover, the radon-induced lung cancer risk is relative to the baseline lung cancer risk, which is significantly higher for ever-smokers, as was estimated and displayed in Figure 4.2. Due to these factors, the EAC and EAD models predict a greater health detriment from radon exposure for ever-smokers. Population risk summaries are therefore highly dependent on smoking prevalence, in addition to radon concentration. Table 5.4 shows the LYL for ever-smokers and never-smokers at the national average exposure of 80.2 Bq/m^3. At this exposure, the models based on occupational studies predict a health detriment approximately three times higher for ever-smokers than for never-smokers.

Figure 5.3 shows a plot of LYL versus Canadian smoking prevalence assuming the seasonally-adjusted national average exposure of 80.2 Bq/m^3. The linearity revealed by this figure is not surprising: in this thesis, ILDs are computed for never-smokers and ever-smokers separately before being combined as a weighted average of the never-smoker and ever-smoker proportions of the population. The population-based ILD is therefore a linear combination of the never-smoker and ever-smoker ILDs:

$$LYL_{POP} = p \times LYL_{ES} + (1 - p) \times LYL_{NS} \tag{5.1}$$

where LYL_{ES} and LYL_{NS} are the LYL values calculated for ever-smokers and never-smokers, respectively, and p is the estimated ever-smoker prevalence. Substituting $p = 0$ provides the estimated average LYL for a never-smoking population or, more practically, a never-smoker exposed to the average Canadian residential radon concentration. Never-smoking Canadians exposed to the national average exposure are expected to incur an average LYL detriment of approximately one to two months using any of the models, as observed in Figure 5.3 and Table 5.4. For the results of Monte Carlo simulations presented in Section 5.2.4, the never-smoker and ever-smoker LYLs are combined as described in Section 4.6.7.

Model	ES	NS
EAC	0.54	0.16
EAD	0.31	0.11

Table 5.4: Average LYL (Life-Years Lost) for ES (Ever-Smoker) and NS (Never-Smoker) Canadians calculated with the EAC (Exposure-Age-Concentration) and EAD (Exposure-Age-Duration) models [6]. LYL is seen to be approximately three times greater for ever-smokers using either model.

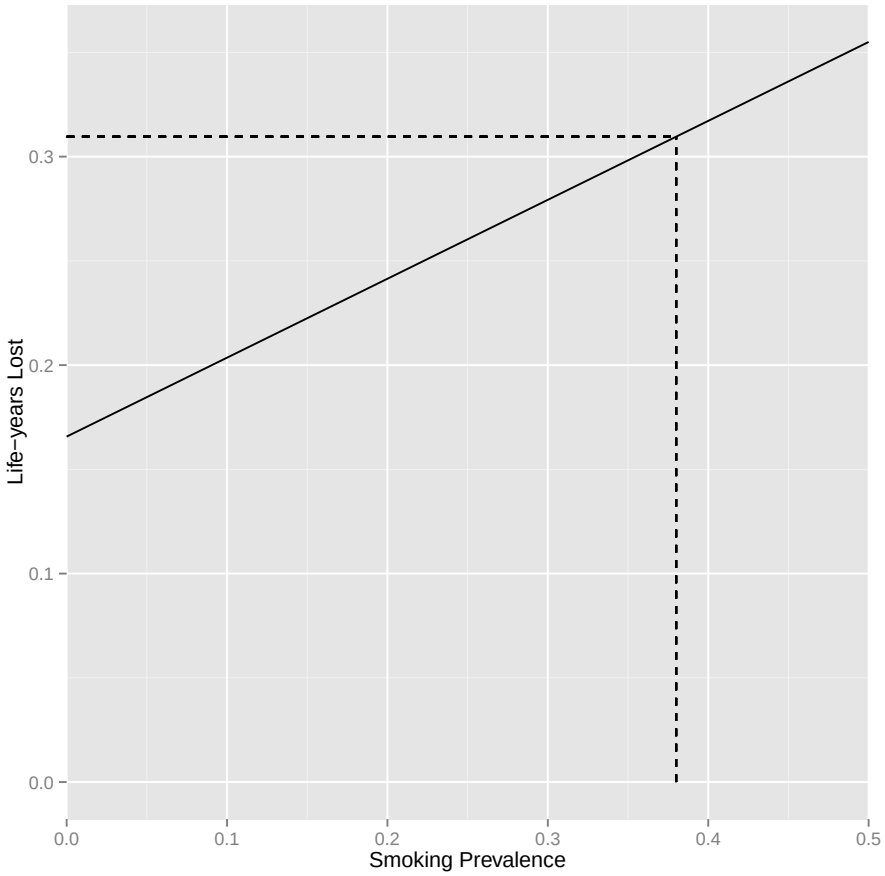

Figure 5.3: Life-Years Lost vs. Smoking Prevalence as predicted by the EAC (Exposure-Age-Concentration) model [6] at the average season-adjusted Canadian exposure of 80.2 Bq/m^3. The dotted line indicates the estimated proportion of ever-smokers in Canada (females and males) in 2013 of 0.38.

5.2.3 Estimates for Floor Levels

Recall from Chapter 3 that radon concentrations in lower levels are systematically higher, as documented by Marcinowski et al. [56]. Analysis of the survey data in Chapter 3 confirms this phenomenon. This translates to greater average health detriment from radon exposure for occupants of lower residential levels. Table 5.5 provides the average Canadian LYL stratified by floor, as predicted by the BEIR-VI EAC model. The average concentration for basements was approximately 50% higher than the 1st floors, while the 1st floors were only about 10% greater than the 2nd floors.

	Basement	1st Floor	2nd Floor
LYL	0.212	0.170	0.160

Table 5.5: Life-years Lost (in years) by residence level using the EAC (Exposure-Age-Concentration) model. [6]

5.2.4 Monte Carlo Analysis

Monte Carlo simulation was performed for the BEIR-VI EAC and EAD models using the uncertainty and variability estimated for model parameters, as provided in the BEIR-VI report [6]. For each simulation, exposure concentration was drawn randomly from the seasonally-adjusted survey results, with replacement.

The ELRR and LYL results include both uncertainty and variability. Constructed using the population-averaged lifetime risk of lung cancer for exposed individuals, PAR is subject only to uncertainty.

Results using the EAC and EAD models are displayed in Tables 5.6 and 5.7, respectively.

	2.5%	5%	50%	95%	97.5%	Mean
ELRR	0.020	0.026	0.215	1.939	2.899	0.506
PAR	0.136	0.149	0.257	0.409	0.439	0.263
LYL	0.009	0.012	0.117	1.237	1.868	0.314

Table 5.6: Quantiles and (arithmetic) means (in Bq/m^3) summarize the uncertainty distributions for the ELRR (Excess Lifetime Relative Risk), PAR (Population Attributable Risk), and LYL (Life-years Lost) computed with the EAC (Exposure-Age-Concentration) model [6]. The results reveal a significant range (more than two orders of magnitude) for the 95 percent confidence intevals of the ELRR and LYL, while the PAR exhibit a fairly narrow spread. Note that the distribution of PAR values reflects uncertainty alone since variability across the population has been averaged in it's calculation.

	2.5%	5%	50%	95%	97.5%	Mean
ELRR	0.013	0.017	0.144	1.264	1.940	0.335
PAR	0.103	0.111	0.188	0.322	0.362	0.197
LYL	0.006	0.008	0.074	0.763	1.148	0.198

Table 5.7: Quantiles and (arithmetic) means (in Bq/m^3) summarize the uncertainty distributions for the ELRR (Excess Lifetime Relative Risk), PAR (Population Attributable Risk), and LYL (Life-years Lost) computed with the EAD model [6]. Note that the distribution of PAR values reflects uncertainty alone since variability across the population has been averaged in it's calculation.

Figure 5.4 shows density plots of the ELRR results from both the EAC and EAD models. The distorted shapes of the distributions (in comparison to a log-normal distribution) are due to the unusual, bimodal distribution of the survey data, as discussed in Chapter 3. The outcome for residential radon concentrations that follow a perfect log-normal distribution was investigated by repeating the analysis but with exposure values drawn from a log-normal distribution defined with a geometric mean and geometric standard deviation calculated from the survey results.

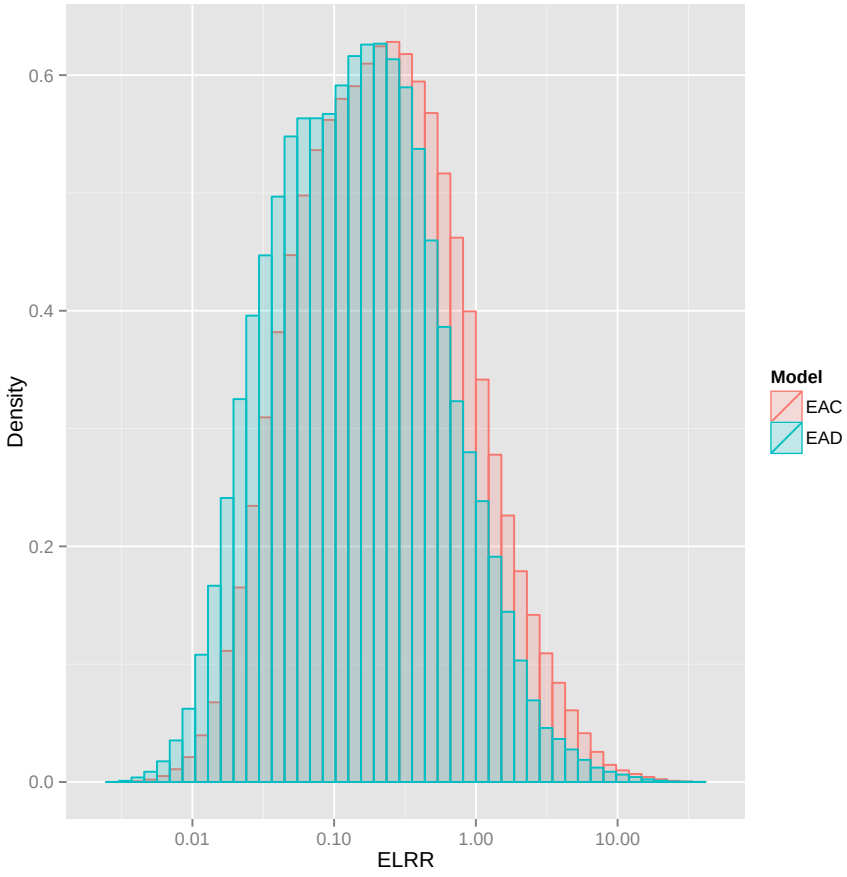

Figure 5.4: Distribution plots of the Monte Carlo ELRR (Excess Lifetime Relative Risk) results generated using the EAC (Exposure-Age-Concentration) and EAD (Exposure-Age-Duration) models [6]. A high degree of overlap exists in the results of the two models, indicating a relatively low level of structural uncertainty.

Combined EAC and EAD Results

The ILD results generated by Monte Carlo analysis using the EAC and EAD models are combined here by weighting the results from both models equally. By combining results, (see Table 5.8), the structural uncertainty, reflected by the two competing models, is now accounted for in addition to the uncertainty and variability of the model parameters and inputs. The models were weighted equally since neither model is preferred over the other.

	2.5%	5%	50%	95%	97.5%	Mean
ELRR	0.015	0.021	0.176	1.622	2.456	0.445
PAR	0.110	0.119	0.219	0.383	0.418	0.231
LYL	0.007	0.010	0.093	1.011	1.539	0.261

Table 5.8: Quantiles and (arithmetic) means (in Bq/m^3) summarize the uncertainty distributions for the ELRR (Excess Lifetime Relative Risk), PAR (Population Attributable Risk), and LYL (Life-years Lost) of the EAC (Exposure-Age-Concentration) and EAD (Exposure-Age-Duration) [6] combined results. Note that the distribution of PAR values reflects uncertainty alone since variability across the population has been averaged in it's calculation.

The mean PAR value of 0.231 implies that approximately 20% of the lung cancer burden in the Canadian population is attributed to radon exposure. In 2013, this would have amounted to 4040 Canadian deaths, based on lung cancer mortality estimates for that year. The quantiles of Table 5.8 also imply that the PAR may be as high as 38.3% at the 95% upper confidence limit. This would translate to 7737 Canadian deaths from radon exposure each year, based on 2013 lung cancer death estimates.

5.2.5 Sensitivity to Threshold

If an exposure threshold for health effects does exist, the accuracy of models based on occupational studies would be most affected (compared to residential models) since the occupational models rely on the linear extrapolation of risk per concentration from occupational to residential concentrations. The sensitivity of LYL to thresholds at 50 Bq/m^3, 100 Bq/m^3, and 150 Bq/m^3 was considered. This was done for each postulated threshold for the EAC model by setting all survey measurements (seasonally adjusted) which were below the thresholds to zero. Computationally, this achieves the requirement for the model to translate these concentrations to zero risk. Average LYL for Canadians, assuming thresholds at 50 Bq/m^3, 100 Bq/m^3, and 150 Bq/m^3 are 0.266, 0.205, and 0.157, which are a reduction of the values obtained assuming a LNT exposure–response by 13.2%, 33%, and 48.7%, respectively. Note that 150 Bq/m^3 is the estimated threshold in the study by Thompson et al. [29].

5.3 Chapter Summary

Applying seasonal adjustment factors to the survey measurements was found to reduce the values to 83% of the measured value, on average.

Point estimates of the ILDs for the Canadian population using the EAC and EAD models are shown Table 5.9.

	EAC	EAD
ELRR	0.48	0.31
PAR	0.26	0.19
LYL	0.31	0.19

Table 5.9: ILDs calculated for the Canadian population using the BEIR-VI EAC (Exposure-Age-Concentration), and EAD (Exposure-Age-Duration) models.

Canada-wide point estimates for never- and ever-smokers are shown in Table 5.4.

Exposure incurred at basement levels were also found to have higher average annual concentrations and therefore higher health detriment. LYL calculated using the EAC model for basement, 1st floor, and 2nd floor levels were calculated to be 0.212, 0.17, and 0.16 years, respectively.

Uncertainty and variability in ILDs was also computed for the EAC and EAD models using Monte Carlo simulation. Mean values for ELRR, PAR, and LYL from the results of the two models combined were 0.44, 0.23, and 0.26, respectively. The geometric standard deviations for ELRR, PAR, and LYL were 3.88, 1.43, and 4.12, respectively.

Considering the existence of a threshold, the sensitivity analysis showed that the average LYL for Canadians would be reduced by 13.2%, 33%, and 48.7%, respectively.

Chapter 6

Conclusion

The lung cancer risk from radon exposure for Canadians was assessed in this thesis using the most recent radon exposure and mortality data available. The methodology was based on occupational studies in conjunction with Health Canada's 2011 cross-Canada radon survey, and used abridged life-table methods to produce results in the form of indices of lifetime detriment. The methodology strives to generate average, rather than conservative, risk estimates through the inclusion of alternate models and use of seasonal correction factors.

The thesis research questions are repeated here:

1. What is a contemporary estimate of the health detriment to Canadians due to lung cancer risks from residential radon exposures?

2. What is the health detriment to subgroups (e.g. smokers, non-smokers) of the population which face different magnitudes of lung cancer risk from radon exposure?

3. What level of uncertainty is associated with the risk estimates?

With respect to question 1, point estimates for life-years lost (LYL) are 0.31 and 0.19 years for the EAC and EAD models (Tables 5.6 and 5.7), respectively. Combined results from these models results in an estimated mean health detriment of approximately 0.26 life-years lost (LYL), an excess lifetime risk ratio (ELRR) of 0.44, and a population attributable risk (PAR) of 23% (see Table 5.8).

In response to question 2, the effects of smoking in combination with radon exposure were also assessed

for the EAC and EAD models. LYL for ever-smokers was estimated at approximately three times than that for never-smokers, as shown in Table 5.4.

Regarding question 3, a significant number of sources of uncertainty were identified in Chapter 4, which limits the robustness of the calculated results. For those which have been quantified, Monte Carlo simulations were performed for the occupational models using the distributions for uncertainty and variability from the BEIR-VI report [6], revealing a significant range of uncertainty and variability in the risk estimates (e.g. the 95% confidence interval for LYL ranges from 0.007 to 1.539 for the combined EAC and EAD results). In addition, the sensitivity of the EAC model to thresholds at 50, 100, and 150 Bq/m^3 was tested and shown to result in reductions to the Canada-wide LYL of 13.2%, 33%, and 48.7%, respectively.

The main limitations of this thesis are listed here:

1. Abundant sources of uncertainty lead to results which lack robustness.

2. The preferred exposure–response models are limited because they are based on the occupational setting and must be extrapolated significantly for use in the residential setting (i.e. extrapolation from the occupational setting).

3. The numbers of survey measurements in the territories were too small to produce reliable results for those regions.

Further residential studies should be undertaken to characterize the exposure–response relationship in the residential setting with greater certainty, including the effects of potential modifying factors, such as smoking history. In order to develop a more accurate representation of risks facing Canadians, future surveys may benefit from protocols which attempt to measure expected, rather than conservative measurements for residential exposures. Representative, rather than conservative, risk values are of value to policy-makers who strive to direct resources towards areas in which the marginal risk reductions per expenditure are maximal. With this information in hand, policy-makers may still have latitude to embed conservativeness in their policies, should it be appropriate.

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Appendices

Appendix A

Converting Bq/m^3 to WLM

The radon survey used in this thesis recorded radon concentrations in Bq/m^3, the SI unit for radioactivity concentration. The BEIR-VI exposure–response models express ERR in terms of exposure concentrations in historical units, Working Level Months per Year (WLM/y). A Working Level (WL) is defined as 100 pCiL^{-1}. Ci, a *Curie* is a non-SI unit of activity, representing 3.7x10^7 decays per second. It follows that 1 WL is equal to 3700 Bq/m^3 from radon. The total radon concentration in the residential setting equivalent to 1 WL is then

$$1WL = \frac{3700Bq/m^3}{F}, \tag{A.1}$$

where division by the Equilibrium Factor, F, accounts for WLs being a measure of the radon decay products only. Assuming F=0.4, we have

$$1WL = \frac{3700Bq/m^3}{0.4} = 9250Bq/m^3, \tag{A.2}$$

which is the conversion assumed in this thesis to indicate the equivalent exposure concentration *in the residential setting.*

A WLM is an exposure concentration of 1 WL for 170 h (the nominal number of hours worked by a miner in the early days of uranium mining). At an exposure concentration of 1 WL, the number of WLM per year is 365.25x24/170 = 51.6 WLM/y. To convert an exposure concentration in Bq/m^3 in the residential setting to an approximately equivalent WLM/y in the occupational setting (thereby producing an exposure

rate compatible with exposure–response models based on occupational studies), we must account for the Equilibrium Fraction, as well as the Occupancy Factor, which are assumed to be 0.4 and 0.7, respectively. Converting a residential average exposure concentration in Bq/m^3 to WLM/y therefore uses the following product:

$$\underbrace{WL/Bq/m^3}_{\text{equiv. WL per } Bq/m^3} \times \underbrace{WLM/y/WL}_{\text{WLM in a year @ 1 WL}} \times \underbrace{F}_{\text{equilib. fract.}} \times \underbrace{Residential\,Occupancy\,Factor}_{\text{fraction time exposed to residential radon}}$$

Which gives

$$1\,Bq/m^3 \quad \Rightarrow 0.00027 \times 51.6 \times 0.4 \times 0.7 = 0.0039\;WLM/y.$$

Appendix B

Thoron

Figure B.1 shows the thoron decay chain.

B.1 Dosimetric Studies

Dosimetric models incorporate data on deposition of radon and it's decay products onto the respiratory tract. Vulnerability of key cells in the respiratory tract to emissions from radionuclides is affected by a variety of factors, including aerosol size distribution, as well as physiological considerations, such as breathing rate and mucous clearance rate. As a result, for a given exposure the dose committed to a subject will vary depending on the radionuclide in combination with the other exposure conditions. To account for this, a dosimetric model assigns a dose conversion factor for radon exposure under assumed conditions. Such a dose conversion factor is akin to the K-factor used in occupational studies, which adjusts for different conditions between mines and residences. As noted by the BEIR-VI committee, conversion factors for dosimetric models are often based on studies of atomic bomb survivors. Although this approach includes cohorts of males and females of all ages, covering the range of subjects exposed to radon, the BEIR-VI committee rejected this approach due to the significant uncertainty inherent to the act of extrapolating exposure risks from large, acute gamma-ray doses to small, alpha particle doses accumulated over long periods of time.

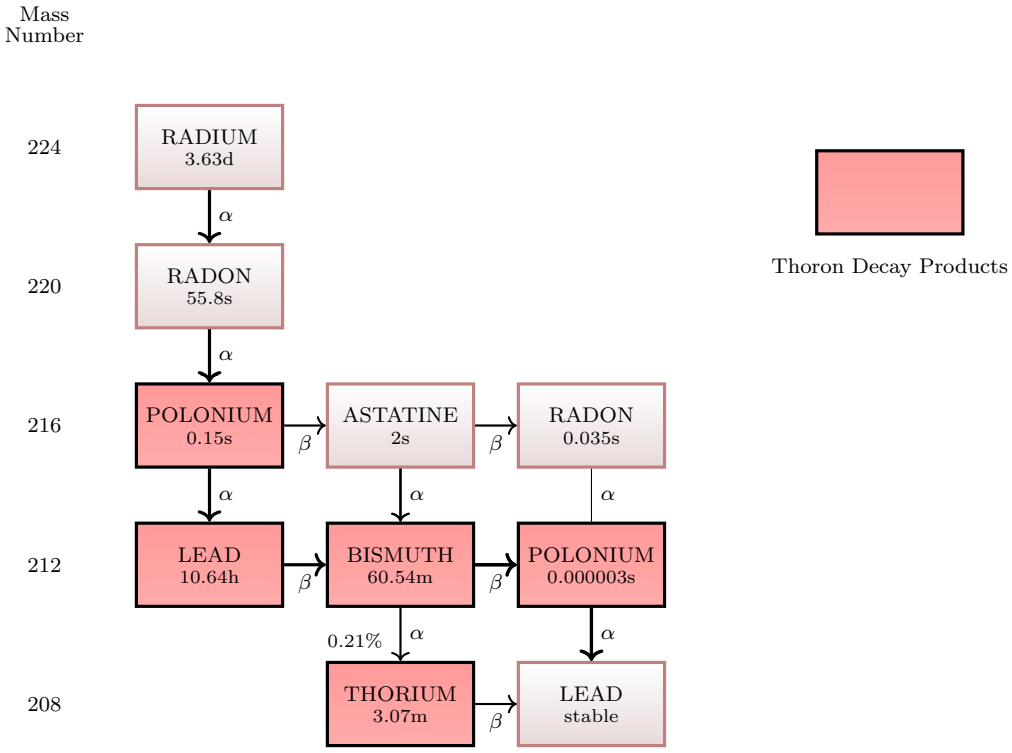

Figure B.1: The Thoron Decay Chain

Appendix C

Radon Exposure Measurement and Estimates

C.1 Types of Detectors

There are a variety of detectors that measure radon concentrations. Detectors are generally of the *open* type or *closed* type design. Open detectors are exposed to alpha particles from any proximal alpha emitters, including radon, thoron, and their progeny. Closed detectors use a barrier to prevent the entry of progeny so that only alpha emissions from radon are detected.

Continuous monitors provide real-time continuous measurements of radon gas concentrations and may consist of an ionization chamber or scintillation cell, which deduce radon concentrations by generating small electric currents proportional to the radon concentration. In the case of the ionization chamber, the electric signal is generated by the formation of ion pairs created from incident ionizing radiation. The ion pairs are attracted to electrodes of opposite charge, thus drawing a current across the electrodes. Detectors using scintillator cells generate a proportional electric current by converting light pulses — generated when emitted particles transfer energy to the scintillator material — to electrical current by means with a photomultiplier tube.

Alpha track detectors use materials such as poly-allyl-diglycol-carbonate (PADC) (also known by its commercial name, CR-39), cellulose nitrate (CN) film, or polycarbonate (PC) that can be damaged by alpha

particles, leaving a track along the particles path. After the exposure period, latent tracks in the detector material can be made visible by chemical etching processes, allowing them to be counted. Counting can be done manually (by eye, aided with a microscope), or by computers, as is typical. The detector material must be sent to a laboratory following the exposure period for this counting process. Alpha track detectors are commonly used for large radon surveys owing to their practical advantages: durability, low cost, and small size [67]. With respect to detection, Zhukovsky et al. [68] divide sources of uncertainty for alpha track radon measurements into two main groups: calibration errors and measurement errors. Calibration errors include systematic bias of reference equipment (e.g. radioactive source, measurement equipment used to establish the establish the reference radon concentration for calibration purposes), and random errors. Measurement errors include calibration bias, random errors, and residual systemic bias of the measurement. They found the total uncertainty was 30-40% with a confidence level of 95%.

C.2 Alternative Methods of Estimating Radon Exposure

C.2.1 Geographic Estimates of Residential Radon Concentration

An alternative to direct radon measurements is to estimate radon concentrations based on geographic location. Because average residential radon concentrations are correlated to the presence of uranium in the earth, the geology of a region is an indicator of radon concentration and recorded radon measurements can be used to estimate radon concentrations en masse in residences based on geographic location.

Researchers at the Lawrence Berkeley Laboratory coupled short-term and long-term radon monitoring data with a collection of geological, soil, meteorological, and housing data to predict average annual residential radon concentrations.

Radon Management Corp. [69] has created a *radon risk map* that ranks *geological units* of Canada into one of three radon risk categories. The geological units were allocated to the risk categories based on data sets from a number of previous surveys. Rock information from the data sets was evaluated based on the U.S. radon potential classification, uranium geochemistry, and its radiometric geophysical response to categorize the radon risk for each geological unit.

C.2.2 Exposure Estimates from Embedded ^{210}Po Activity in Glass Objects

The concept of assessing indoor radon exposure, retrospectively, based on measurements of embedded ^{210}Po, was proposed by Lively [12], refined by Samuelsson [70] and has been used over the past decade or more to determine historic levels of radon in residences [71]. The recoil energy associated with most alpha-decay is of sufficient energy to implant the progeny ^{222}Po in the surface of glass on which it is attached. Measuring the ^{222}Po provides an estimate of the radon concentrations in the vicinity of the glass.