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Michael King
AUTEUR DE LA THÈSE / AUTHOR OF THESIS

M.Sc. (Epidemiology)
GRADE / DEGREE

Department of Epidemiology and Community Medicine
FACULTÉ, ÉCOLE, DÉPARTEMENT / FACULTY, SCHOOL, DEPARTMENT

Managing Lung Cancer Risks Associated with Residential Radon Exposure in Canada
TITRE DE LA THÈSE / TITLE OF THESIS

D. Krewski
DIRECTEUR (DIRECTRICE) DE LA THÈSE / THESIS SUPERVISOR

D. Coyle
CO-DIRECTEUR (CO-DIRECTRICE) DE LA THÈSE / THESIS CO-SUPERVISOR

EXAMINATEURS (EXAMINATRICES) DE LA THÈSE / THESIS EXAMINERS

N. Birkett

D. Stieb

Gary W. Slater
LE DOYEN DE LA FACULTÉ DES ÉTUDES SUPÉRIEURES ET POSTDOCTORALES /
DEAN OF THE FACULTY OF GRADUATE AND POSTDOCORAL STUDIES

MANAGING LUNG CANCER RISKS ASSOCIATED WITH RESIDENTIAL RADON EXPOSURE IN CANADA

by

Michael King

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Faculty of Graduate and Post-Doctoral Studies
in partial fulfillment of the

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ABSTRACT

The weight of current evidence supports a small yet significant increase in lung cancer risk associated with exposure to radon in the home. Our study provides an economic evaluation of five national strategies to mitigate residential radon exposure. Analysis is based upon a probabilistic decision model which estimates the time-dependent reductions in age, gender and smoking-specific lung cancer risks following mitigation, and models the impact within simulated Canadian population over 80 years. The estimated costs of the radon programs are compared with these impacts to calculate the cost-effectiveness of residential radon mitigation.

All strategies considered were found to be very expensive, with total costs ranging from about $100 million to several billions of dollars. The more expensive mitigation strategies also tended to be the most cost-effective. In the majority of cases, the strategy with the lowest cost per lung cancer prevented was universal screening of all currently existing dwellings, with mitigation of dwellings above some action level. The cost-effectiveness of radon mitigation was improved by lowering the Canadian radon guideline from its current level of 800 Bqm^{-3}; by maximizing the compliance of homeowners with the recommended action; by maximizing the degree to which radon levels are reduced within mitigated dwellings; and by targeting radon mitigation towards geographic areas with higher average radon concentrations. The implications of our findings are discussed within the context of risk management decision making in Canada.

ACKNOWLEDGEMENTS

A graduate thesis is an enormous undertaking, and virtually impossible to carry out by oneself. Thus, I begin *this* one by acknowledging the numerous individuals and groups who have made significant contributions, directly or indirectly, to the completion of this work.

I would be remiss to start this listing anywhere but with Dr. Daniel Krewski. It was through Dr. Krewski that I was first introduced to population health risk assessment – a scientific discipline which perhaps best suits my broad range of interests and abilities. Since that time, I have learned a great deal from his wealth of knowledge and experience, been inspired by his enthusiasm for science, and have benefited from the numerous opportunities with which he has provided me, including this thesis. For this and much more, I simply cannot thank him enough.

I have benefited greatly from an excellent thesis supervisory committee. I want to express my sincerest gratitude to Dr. Kevin Brand and Mr. Doug Coyle, without whom, respectively, I would not have valid results, and would not know what to do with them anyway. I am also indebted to Dr. Jan Zielinski, whose decade of experience with the radon problem in Canada was a valuable resource from which I could draw. Thank you all so much for your expertise and insight.

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I want to acknowledge my present employer – Health Canada – and particularly Mr. Ken Lee and Dr. Sheila Dubois, who provided me with the opportunity to put my M.Sc. degree to use before it was completed, and displayed great patience as that completion took just a little longer than expected.

My studentship has been generously supported by scholarships from the Department of Epidemiology and Community Medicine, the Faculty of Graduate and Post-Doctoral Studies, and the Ontario Graduate Scholarship Program. I have also benefited from a research assistantship provided by the R. Samuel McLaughlin Centre for Population Health Risk Assessment.

And finally, I end at the place where, in truth, all things in my life both begin and end – with my wife, Kelly-Anne. In the (relatively) short time since this project began, we have together become husband and wife, homeowners, and now, expecting parents. Through these and other distractions that life provides, her unwavering love, support, and occasional ‘kick in the pants’ have been as responsible for the successful completion of this dissertation as anything that I have done. Were I to complete one hundred more, I could never be so proud as I am to share my life with her.

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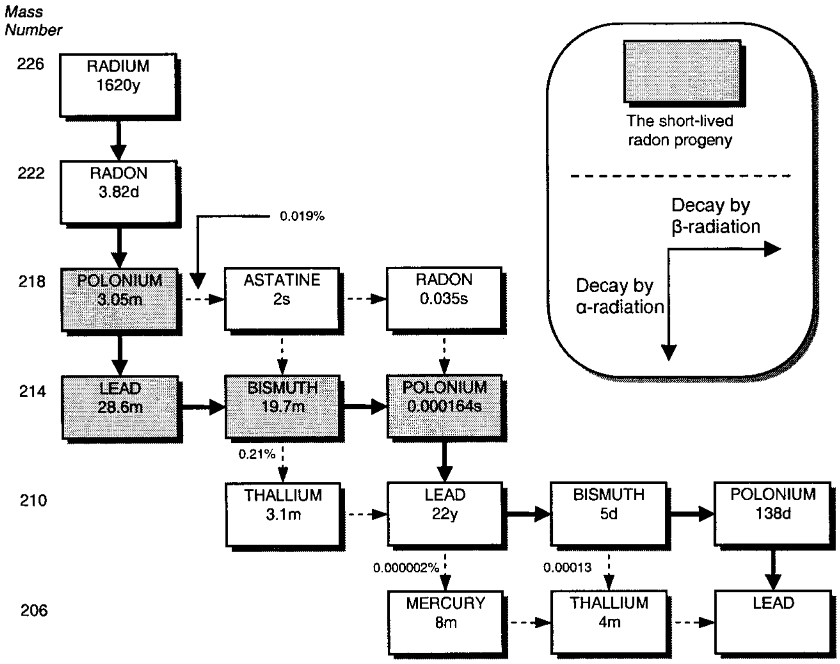
CHAPTER 1
INTRODUCTION AND LITERATURE REVIEW

1.1 BACKGROUND

Radon-222 (herein denoted "radon") is a naturally occurring decay product of uranium-238. It is a chemically inert and electrically uncharged gas, which continuously seeps out of rocks, soil, and ground water, and into the surrounding air. Though outdoor air levels are typically very low, the concentration of radon can increase within enclosed areas from which the gas is not allowed to disperse. For example, high levels of radon can be found in unventilated underground mines, particularly uranium mines. Even the air within a home can contain radon levels that are tens of thousands of times higher than those observed outside [1].

Radon is radioactive. It has a half-life of 3.8 days, and decays naturally to elemental lead through a series of other elements, as illustrated in Figure 1-1.

FIGURE 1-1. The Radon-Decay Chain.
Most decay take place along the unbranched chain marked with thick arrows. The negligible percentage of decay along the dashed arrows is shown at critical points. The end of the chain, lead-206, is stable, not radioactive. Half-lives of each isotope are shown as seconds (s), minutes (m), days (d), or years (y). Adapted from the BEIR VI Report [2].



The intermediate atoms – historically known as radon daughters but now often termed radon-decay products or radon progeny – are electrically charged, and therefore able to attach themselves to particles in the air, such as dust, smoke, and water vapour. If the radon-aerosol complex is inhaled, the continuing radon decay process can expose cells of the lung to alpha, beta and gamma radiation [3]. Of these, alpha radiation (referred to as alpha particles) is of primary concern due to its high *relative biological effectiveness* (RBE), which represents the amount of cell damage caused per unit dose. In particular, alpha-particles released by two radioisotopes in the radon-decay chain, polonium-218 and polonium-214, can deliver sufficient energy to target cells in the respiratory epithelium to cause cancer [4].

Radon has been classified as a known human carcinogen by the International Agency for Research on Cancer [5] – a classification supported by the weight of evidence from a large body of experimental and epidemiologic research, reviewed later in Section 1.6.2. On the basis of this research, action has been taken to reduce elevated radon concentrations found within underground mines. More recently, concern has been raised regarding the possible health effects of the chronic, lower-level residential radon exposures incurred by members of the general population.

1.1.1 Canadian and International Guidelines for Radon in the Home

There are no regulations in Canada governing acceptable levels of radon in Canadian homes. Such decisions regarding risk acceptability are delegated to the individual home owner [6]. Canada does, however, have an advisory exposure guideline [7], introduced in 1988. It reads:

> *"It is recommended that remedial measures be taken where the level of radon in a home is found to exceed* ***800 Bqm^{-3}*** *as the annual average concentration in the normal living area. Because there is some risk at any level of radon exposure, homeowners may wish to reduce levels of radon as low as practicable."*

The Canadian advisory exposure guideline of 800 Bqm^{-3} is the highest of its kind in the world. Figures 1-3(a) and (b), below, compare the Canadian Guideline of 800 Bqm^{-3} with advisory guidelines set by other countries for radon levels in existing and new dwellings, respectively [8].

FIGURE 1-2(a). Government Advisory Guidelines for Radon Levels in Existing Dwellings, by Country

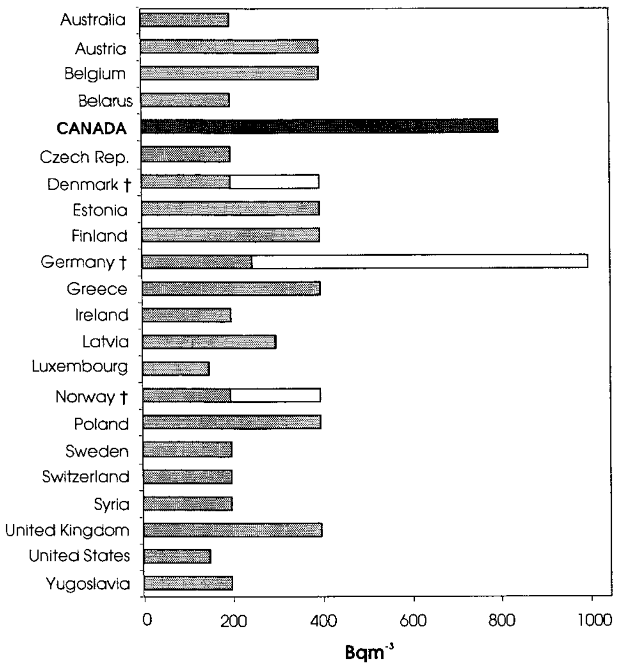
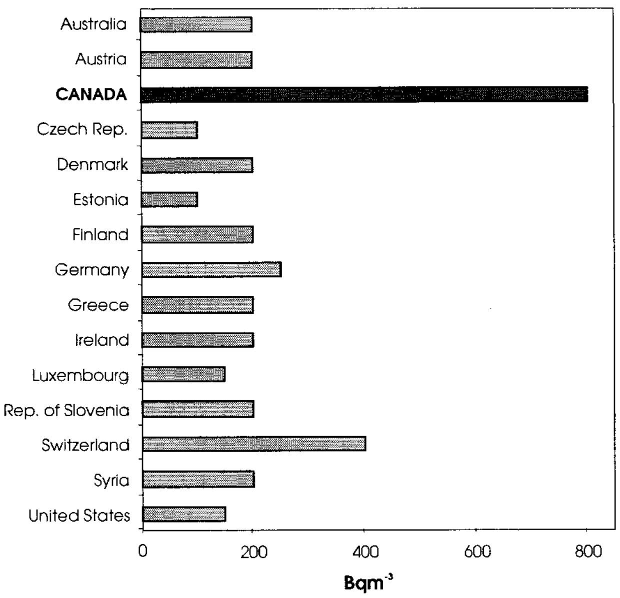


FIGURE 1-2(b). Government Advisory Guidelines for Radon Levels in New Dwellings, by Country



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† 3 Countries (Denmark, Germany and Norway) have more complicated guidelines. Mitigation is recommended at different radon concentrations, depending upon a number of conditions (e.g., cost of mitigation). The multiple guidelines also come with different degrees of recommendation, with mitigation more strongly advised at higher concentrations.

Note that Germany is the only country with an advisory guideline higher than Canada (i.e., mitigation is *strongly* advised at 1000 Bqm^{-3}), although remediation is recommended at a reference level of only 250 Bqm^{-3} when exposure mitigation can be achieved through simple, less expensive measures. All other countries surveyed advise limiting residential radon levels to between 200-400 Bqm^{-3}. The current radon guideline established by the U.S. Environmental Protection Agency is 148 Bqm^{-3} [8, 9]. A number of countries (including Sweden, U.K., Russia, Slovak Republic, Belarus, Czech Republic, Norway, and Poland) *enforce* guidelines as low as 100-200 Bqm^{-3} in some homes (data not shown) [8].

The International Commission on Radiological Protection (ICRP) recommends that a national radon action level should be set within the range of 200-600 Bqm^{-3}, and state that "*it seems clear that some remedial measures against radon in dwellings are almost always justified above a continued annual* [*concentration of 600 Bqm^{-3}*]." [10]

The World Health Organization (WHO) has not established a formal radon guideline in its Air Quality Guidelines for Europe, but did recommend that "simple remedial measures should be considered for buildings with radon progeny concentrations of more than 100 Bqm^{-3} equilibrium equivalent radon as an annual average, with a view to reducing such concentrations wherever possible" [11].

1.2 PURPOSE OF THIS THESIS

The aim of this thesis is to inform risk management decision-making by providing an economic evaluation of five potential national strategies to reduce residential radon exposure within Canada. We estimate the costs, health benefits and cost-effectiveness of all strategies, and consider how these might be affected by changes in risk management criteria (e.g., the strategy, and 'action level' – i.e., the referent radon concentration above which mitigation is recommended) as well as factors such as homeowner compliance with the radon guideline, the % reduction in radon concentrations achieved in mitigated dwellings, the background radon concentration distribution, and the annual discount rate applied to mitigation costs and health benefits.

Previous economic evaluations of residential radon mitigation in Canada, reviewed later in Section 1.6.2, will be enhanced by the use of the BEIR VI model of excess relative risk – the most recent information available regarding the risks of radon exposure by which outcome predictions can be made[†]. Important factors such as population growth and the lag between reduced exposure and reduced lung cancer mortality will be addressed.

Using Monte Carlo simulation, we also present a pilot analysis of the uncertainty within our estimates, demonstrating the potential for these techniques to enhance decision-making.

[†] The most current, and likely the most relevant, information on the association between lung cancer and residential radon exposure comes from a large combined analysis of data from seven North American case control studies of radon exposure within the indoor environment. However, methodological considerations preclude the use of results from case control studies for the estimation of future risks within the population, as required by our methods.

1.3 RADON QUANTITIES, UNITS AND CONVERSIONS

This section provides a brief review of the units by which radon concentrations and exposures are described within this thesis.

1.3.1 Radon Concentration

Amounts of radioactive material can be specified by either mass or activity, although the latter specification is more often used [12]. Activity refers to the rate at which atoms decay radioactively. The concentration of the material is described simply as the measured activity per unit volume. In this thesis, radon concentrations will be expressed in units of *bequerels per cubic meter* (Bqm^{-3}). A *bequerel* (Bq) is an SI unit of radioactivity, representing one nuclear disintegration per second. Thus, 1 Bqm^{-3} is the concentration of radon that produces one 'decay event' (with associated emission of radiation) per second, in one cubic meter of air. The traditional equivalent to the bequerel is the *picocurie*, where 1 PCi = 3.7×10^{-2} Bq. Radon concentrations are often described in the U.S. in terms of picocuries per liter (PCi/L), with 1 PCi/L = 37 Bqm^{-3}.

One can measure radon concentrations in a number of ways. A widely employed dosimeter for monitoring radon radioactivity is based upon a polymer called CR-39 [13, 14]. Alpha particles emitted during the decay process cause visible damage in the form of 'etchings' (often called 'etched-' or 'alpha-tracks') in CR-39 plastic, with a separate track distinguishable for each alpha particle, such that by counting the tracks, one can determine the concentration of alpha-emitters to which the dosimeter was exposed [15]. For further information on CR-39 and all other radon measurement techniques, the reader is referred to reviews published elsewhere [15-17].

1.3.2 Radon Exposure

Cumulative radon exposure is the product of the rate of exposure and time. It is often expressed using a historical unit - the *working level month* (WLM). A *working level* (WL) is a measure of radon concentration – or more specifically of the potential alpha-energy released by a given radon concentration – and is approximately equal to 100 PCi/L or 3700 Bqm^{-3}. The corresponding SI unit is that of energy (joules) per unit volume, J / m^3. The name "working level" historically refers to a referent radon concentration experienced by underground miners – an occupational group that was the principal source of early information on radon. One working level month (WLM) is represents an amount of exposure equivalent to that incurred by being exposed to one working level for one "working month" (170 hours).

The rate of radon exposure is a function of the concentration of radon, modified by a number of additional factors, described below. Conversion of concentrations in Bqm^{-3} to exposures in WLM is described in Section 2.2.2.

1.3.2.1 Equilibrium Factor

When assessing exposure, it is the radioactive radon decay-products – and not radon itself – that are of primary concern. Rather than reporting levels of individual decay products, it is common to report a collective concentration that is normalized to the amount of alpha decay energy that will ultimately result from the mixture of decay products that is present. This is termed the *"equilibrium-equivalent" decay-product concentration* (EEDC), and represents the amount of each radon decay product that would be present at equilibrium in order to collectively produce the measured level of radioactivity [12]. Concentrations of radon progeny are ultimately expressed as the product of the measured radon concentration and an *"equilibrium factor"* – the ratio of EEDC to radon concentration. The equilibrium factor would be equal to 1.0 if radon and all its decay products were in radioactive equilibrium (and therefore had the same radioactivity concentration). Within the indoor environment, the equilibrium factor can range from 0.1 to 0.9, but most are within 30% of the typical value, 0.4 [18]. Note that equilibrium values are somewhat higher in outdoor air, with typical values appearing to range between 0.5-0.7 [18].

1.3.2.2 Residential Occupancy Factor

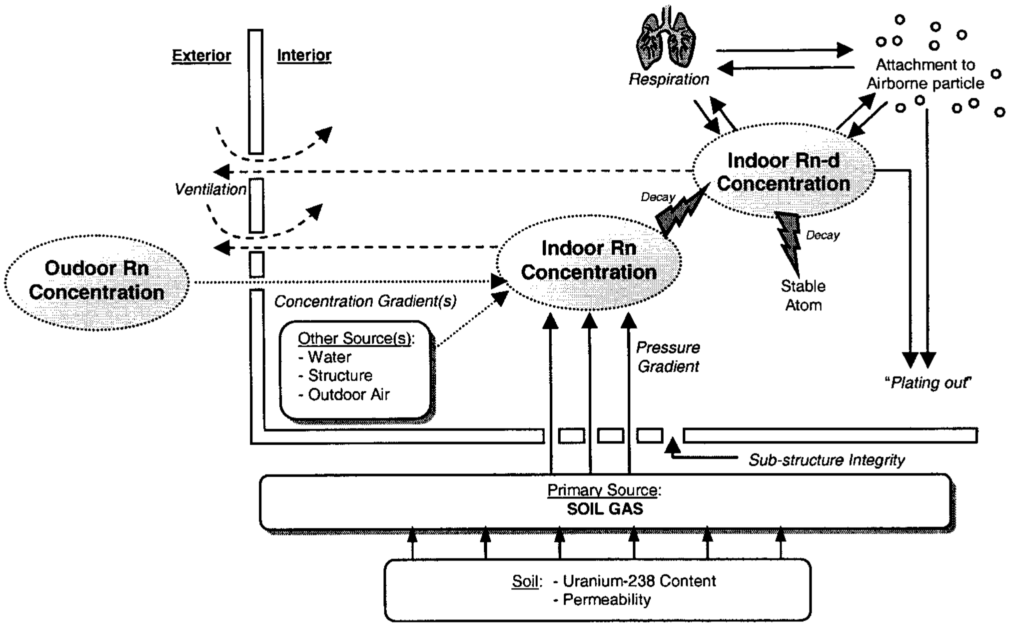
Since the exposure of concern in this thesis is that which occurs within the home, it is necessary to estimate the amount of time spent there. The proportion of time spent in the home within Canada is generally assumed to be approximately 0.70 [19]. This value is consistent with the results of a large survey reporting time-activity patterns of Canadians [20].

1.4 RADON EXPOSURE IN THE GENERAL POPULATION

Because of its natural origins, radon is a ubiquitous indoor air pollutant to which everyone is exposed [2]. Worldwide, it is, on average by far the predominant source of exposure to ionizing radiation among members of the general public [18], and in some populations may represent over 90% of total yearly radiation dose [21].

The principal place of the exposure to radon is in the home, with the primary source of radon entering the home being the soil beneath and around the structure. Under normal conditions of negative indoor air pressure [22], radon-rich soil gas is drawn into the house by advection through a number of entry points, including foundation joints, cracks in floors and walls, drains and piping, electrical penetrations, and cellars with earth floors [23]. Small contributions to indoor radon levels can also be made by the *diffusion* of soil gas [24], radon-contaminated water [25, 26], radium-rich building materials [27], and outdoor air [28]. The contribution to total radon exposure from these other sources is higher in the upper floors of buildings where soil gas cannot reach [28], and also in latitudes where warmer climates preclude the need for tightly sealed homes (thus eliminating the pressure differential with the surrounding soil [18]).

FIGURE 1-3. Factors Influencing the Concentration of Radon Gas (Rn) and its Decay Products (Rn-d) in Indoor Air. Estimates indicate that approximately 95% of the risk from residential radon is due to gas entering the basement along pressure gradients, while only 4% is due the diffusion of outdoor air. The diffusion into indoor air of gas from radon-contaminated drinking water contributes 0.8% of the risk, while consumption of that water adds only 0.1%. Diffusion from building materials is negligible in North America, but is more of a concern in Europe, due to the materials employed. [29]



Radon levels are known to exhibit diurnal and seasonal variation (due to factors such as wind, temperature, and atmospheric pressure) [30], and are affected by characteristics of the soil (such as composition and permeability) [24] as well as those of both the house (e.g., ventilation rate) and homeowner (e.g., sleeping with the window open) [30]. Radon progeny concentrations have an additional level of complexity, as their levels can change rapidly through radioactive decay, chemical reactions, and "plating out" interactions with walls, furnishings and airborne particles [28].

1.4.1 Indoor Radon Levels in Canada

In 1978-1980, McGregor et al. performed a major study of residential radon levels in Canada [31]. The survey involved approximately 19,000 measurements of radon levels in homes in 14 Canadian cities, stratified in such a way as to be representative of the entire housing stock within urban settings. The results of the survey, shown in Table 1-1, below, indicate that levels of radon within Canadian homes are approximately log-normally distributed, with a geometric mean (GM) and geometric standard deviation (GSD) of 11.2 Bqm^{-3} and 3.9, respectively. The corresponding arithmetic mean (AM) is 28.35 Bqm^{-3}. Significant regional variation in radon levels was observed, with the GM in Winnipeg (57 Bqm^{-3}) being more 10 times greater than that of Vancouver (5.2 Bqm^{-3}).

TABLE 1-1. Indoor radon concentrations (Bqm^{-3}), Canada, 1978-80, by city. [31]23

Location	Parameters of log-normal distribution	
	GM[a]	GSD[b]
Calgary	11.5	3.6
Charlottetown	15.2	5.3
Edmonton	17.0	4.5
Fredericton	24.4	4.0
Montreal	10.7	3.3
Quebec	10.4	3.8
Regina	49.2	3.8
Saint John	10.0	5.7
Saskatoon	15.5	4.3
Sherbrooke	13.3	5.4
St. John's	11.1	4.4
Sudbury	21.5	4.0
Thunder Bay	20.0	4.4
Toronto	11.5	2.8
Vancouver	5.2	3.0
Winnipeg	57.0	4.6
CANADA	**11.2**	**3.9**

[a]Geometric mean; [b]Geometric standard deviation

While the shape of the Canadian distribution is consistent with those found in other countries [32], its parameters indicate that average radon concentrations in Canada are relatively low when compared with those same nations (see Table 1-2). The United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) estimated that the worldwide, population-weighted radon distribution had parameters AM=39 Bqm^{-3}, GM=30 Bqm^{-3}, and GSD=2.3 [18], indicating mean radon levels significantly higher than those found in Canada. This difference is likely driven by high mean radon concentrations in Europe, where values of several thousands of Bqm^{-3} have been found in thousands of houses [10]. Even the average radon exposure experienced by the U.S. population is approximately twice that measured for Canada. The dispersion within the Canadian residential radon distribution, as measured by the GSD, appears to be greater than for the distributions reported in other countries.

TABLE 1-2. Results of indoor radon level surveys in Canada and select other countries.

Country	Radon Measurement (Bqm^{-3})				*Ref.*
	AM[a]	GM[b]	Max.	GSD[c]	
Canada	28.35	11.2	1,720	3.9	31
U.S.	46	25	-	3.1	32-34
Germany	50	40	>10,000	1.9	35
Sweden	108	56	85,000	-	36
Switzerland	70	50	10,000	-	37
Norway	73	40	50,000	-	36
Finland	120	84	20,000	2.1	38
U.K.	20	-	10,000	-	39

[a] Arithmetic mean; [b] Geometric mean; [c] Geometric Standard Deviation

It is important to note that the majority of radon measurements in the 1980 Canadian survey were taken in basements – an area of the house home in which the average homeowner will spend a very small proportion of their time. It has been shown that average radon concentrations in the "living areas" of a home are significantly lower than those found in basements (a subsequent survey in Winnipeg [40] revealed concentrations within the living area that were, on average, 33% lower than those found in the basement). The radon measurements based on the survey conducted by McGregor et al. may therefore overestimate the true radon concentrations to which the Canadian population is exposed. This inaccuracy may have been offset somewhat by an additional source or error: radon levels were assessed via a single measurement, during the day and in the summer months – the time and season when radon levels are at their lowest. Despite these limitations, the McGregor et al. survey remains the most comprehensive source of information on the national radon distribution in Canada.

1.5 RADON AND LUNG CANCER

Radon has been classified as a known human carcinogen by the International Agency for Research on Cancer [5]. This classification is supported the large body of evidence from research in several animal species, dosimetric and biological models, molecular biology, and epidemiological studies. The weight of that evidence provides strong support for a linear dose-response relationship between radon and lung cancer – a relationship for which there is no threshold. An important implication of this linear dose-response curve is that there is some degree of risk associated with all levels of radon exposure.

However, the magnitude of that risk can be very small at low radon concentrations. The most comprehensive epidemiological analyses on low-level radon exposure – the pooled analysis of miner data, and the pooled- and meta-analysis of residential studies – provide consistent evidence of a small but significant increasing trend in risk with increasing exposure.

The weight of evidence obtained from each of the multiple sources is reviewed in this section.

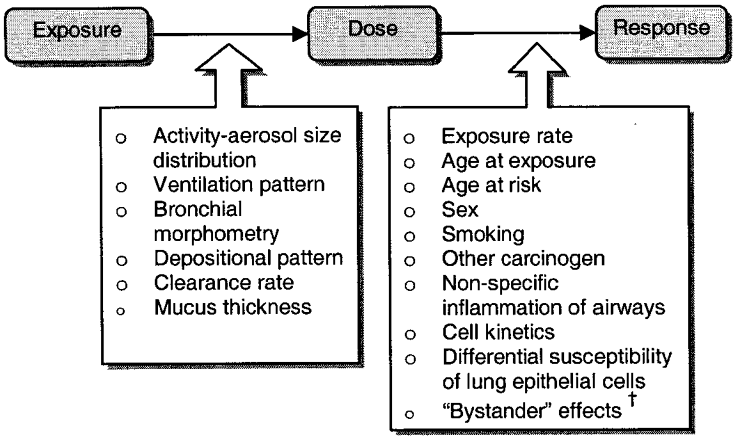
1.5.1 Animal Inhalation Studies

Beginning in the 1950s, radon inhalation studies examined the biological and physical characteristics of radon and progeny [41-43]. It was soon observed that it was radon progeny attached to carrier dust particles, and not radon itself, that was the predominant cause of alpha radiation exposure to the bronchial epithelium of rats and dogs [44]. Experimental data on the carcinogenic potential of radon and its progeny began to appear in the late 1960s and early 1970s. Significant achievements during that time were the confirmation that induction of tumours in rats was due to exposure to radon decay products, and that exposure to mixed aerosols of various contaminants, including radon and its progeny, resulted in a significant lifespan shortening in the beagle (though no effect was observed in the hamster) [45]. Similarly, studies have demonstrated a significant shortening of lifespan in rats receiving exposures above 5,000 WLM [46].

Overall, animal inhalation studies confirm the carcinogenicity of radon and indicate an increase in tumour incidence that is approximately linear with cumulative exposure. However, the capacity of such studies to inform decisions on human health risks is limited by the necessary extrapolations (i) from animal models to humans, and (ii) from high doses (and dose rates) employed in laboratory studies with animals to the low exposures found within the indoor environment, which persist throughout the lifetime of an individual.

Animal studies have provided information on a number of biologic / dosimetric factors that may influence the induction of tumours by exposure to radon (see Figure 1-4). The interested reader is referred to previously published reviews in [2, 46, 47] for additional information related to these factors.

FIGURE 1-4. Factors influencing the relationship between radon exposure, dose, and lung cancer risk. Conversion of exposure to delivered dose is deterministic in nature, dependent on the physical characteristics of the lungs and of radon itself. Conversely, the dose-response relationship is primarily driven by a stochastic event – the probability of an α-particle striking the nucleus of a lung epithelial cell – though what occurs afterwards may be effected by the variables shown. Adapted from [2]



[†] Bystander effects" refers to deleterious changes induced within a group of cells in response to direct radiation damage to only one cell within that group [48].

1.5.2 Cellular / Molecular Research

It has been shown that exposure to ionizing radiation, including alpha-radiation, can produce marked changes in DNA. The most common changes are large *interstitial deletions* (specifically, the removal of DNA from an inner portion of a chromosome between two breaks in the strand, followed by rejoining of the two terminal fragments) and *reciprocal translocations* (the exchange of large DNA segments between two locations, typically on two different chromosomes) [49, 50]. With this is mind, a number of biological mechanisms have been proposed by which radon may interact with lung epithelium at the cellular or molecular level to induce cancer. These were reviewed by the BEIR VI committee [2]. The process very likely results from a combination of the following effects.

1.5.2.1 Oncogene Activation

An oncogene is a segment of DNA that promotes malignancies. Over 100 have been identified in human cancer [2], and have been shown to be activated by gene deletions [51] and reciprocal translocations [52].

1.5.2.2 Tumour-Suppressor Gene Deactivation

There exist within the genome DNA segments known to inhibit malignancies, known as tumour-suppressor genes [53]. Removal of such a gene through radiation-induced DNA deletion is one possible mechanism by which radon might induce cancer (see Kasten et al. [54, 55] and Smith et al. [54, 55] for a well-studied example of this mechanism).

1.5.2.3 Induction of Genomic Instability

The progression from normal cells to a metastatic cancer state is a complex process, likely involving multiple mutations of various types throughout the genome. The means by which a single relatively small dose of radiation could result in the occurrence of a wide variety of genetic anomalies involved in carcinogenesis is unclear. The induction of multiple mutations seems unlikely. A more plausible explanation is that radiation causes broad genomic changes though wide-spread damage to the cell – for example, mutations in a gene responsible for the stability of the genome [2]. The resulting genomic instability could then predispose the cell for the multiple mutations and chromosomal changes involved in carcinogenesis (see, for example, [56-58]).

1.5.2.4 Individual and Genetic Susceptibility

There is substantial evidence that cancer-predisposing genes are present in the human genome, with an estimated prevalence of about 16 per 1,000 live births [59]. A number of studies indicate that ionizing radiation might interact with genetic susceptibility factors to increase the frequency of radiation-induced cancers [60-63], though no strong evidence of this has yet been found in humans [2]. Marked differences in responsiveness to radon exhibited by various species of experimental animals may provide further insight into the role of genetic predisposition to cancer [2].

1.5.2.5 Disruption of Cell Cycle

A normal cell will go through a cycle involving successive stages of growth and metabolism (G_0 and G_1 phase), DNA replication (S phase), preparation for division (G_2 phase), and nuclear and cellular division (M phase, or *mitosis,* and *cytokinesis*). Ionizing radiation, and alpha particles in particular, produce a dose-dependent delay in the progression of a cell through the G_1 and G_2 stages of the cell cycle [54, 64]. It is therefore possible that exposure to radon may disrupt the cell cycle in such a way as to increase the likelihood of cancer – for example, by preventing the cell from entering a phase of the cycle in which it normally would have repaired DNA damage contributing to malignancy [65]. However, cells spend only a small fraction of their time within cell cycles stages that are most sensitive to radiation, indicating that cell-cycle effects likely play only a minor role in the radon-induced carcinogenesis [2].

1.5.2.6 Inhibition of Apoptosis

Apoptosis refers to a 'programmed' cell death in which a damaged cell signals neighbouring cells to destroy it through phagocytosis [66], a common mechanism to remove damaged cells in lung tissue. Disruption of this process by alpha radiation is thus a possible pathway to carcinogenesis for a radiation-damaged cell [67].

1.5.2.7 Perturbation of Cellular Proliferation

Experimental studies have shown that inhalation of radon leads to an increase in the rate of cell turnover in the upper and lower respiratory tract [68], and that these increases are found in the areas with the highest radiation dose [69]. Changes in cell proliferation play an important role in the development of cancer [70], as altered cell kinetics have the potential to increase the clonal expansion of altered or mutated cells. Amplification of genetically altered cells through clonal expansion can greatly increase cancer risk [2].

1.5.3 Biologically-Based Modelling of Human Populations

Radon-related lung-cancer risks have been described using biologically-based risk projection models. These models seek to describe the fundamental biological processes involved in neoplastic transformation of stem cells into malignant cancer cells, including mutation and cell kinetics [71]. Numerous such models have been developed and published in the literature [72-77], and have been recently reviewed [78]. Moolgavkar and Knudson's two-stage clonal expansion model has been shown to adequately describe a number of epidemiologic datasets involving radiation exposure [78-82] and has been used to quantitatively model the interaction of smoking and radon in the causation of lung cancer [83].

A major strength of biologically-based models is that they have a direct biological interpretation, and are flexible to explicitly consider time and age related patterns of exposure [71]. However, current knowledge of the mechanisms involved in radon-induced carcinogenesis is incomplete, especially at the molecular level. Though useful, biologically-based models are currently considered to be an oversimplification of radiation carcinogenesis [2].

1.5.4 Extrapolation from Human Exposures to Gamma–Radiation

Radon risks have also been evaluated through comparative dosimetry. With this approach, a dosimetric model is used to estimate the dose of alpha-radiation that is delivered to the lung [84]. Lung cancer risk is then calculated using a coefficient based on risks in populations exposed to low linear-energy-transfer (low-LET) radiation, such as the Japanese atomic-bomb survivors, and adjusted upwards to account for the greater potency of alpha radiation in causing cancer (a 20-fold increase in risk is generally assumed as a 'quality factor') [2, 85]. The reader is referred to the BEIR IV report [46] for a complete review of dosimetric models developed for extrapolating radon risks from those of gamma radiation.

There is a wealth of epidemiologic data available on the atomic bomb survivors, with well-characterized risks in the cohort in relation to dose. However, this approach is hindered by the many assumptions needed to generate risk estimates for prolonged localized exposure to densely-ionizing alpha radiation, based on risks for acute, whole-body exposure to gamma rays [2].

1.5.5 Epidemiologic Studies in Miners

Radon-222 was the first occupational respiratory carcinogen to be identified. As early as the 1500s, unusually high mortality from respiratory disease was documented among underground metal miners in the Erz Mountains of Eastern Europe [86]. Lung cancer was first linked to radon exposure in the 1920s, based on findings of high levels of radon in mines in that region, and in the nearby mines of Joachimsthal in Czechoslovakia where miners also had high lung-cancer rates [87, 88]. That causal relationship did not gain uniform acceptance until the findings of epidemiologic studies of other groups of radon-exposed underground miners were reported in the 1950s and 1960s [89].

The best current source of evidence on the lung cancer risks associated with occupational radon exposure comes from eleven large epidemiologic cohort studies of underground miners [90-113]. Each of these studies were reanalysed in 1994, in a collaboration of the principal investigators sponsored by the National Cancer Institute (NCI) [114]. The group then combined all eleven datasets to provide a more precise estimate of the risk from high occupational exposures [114] and low level residential exposures [115]. Following updates to four of the miner cohorts, combined analyses of the health effects of exposure to residential radon were again performed by the NCI group in 1997 [116], and in 1999 by the U.S. National Research Council's Committee on the Biological Effects of Ionizing Radiations (BEIR VI) [2]. These combined analyses provide the most comprehensive summary of available data on lung-cancer risks in miners [2], and are thus presented here in lieu of the originally published results.

The NCI analyses of the individual cohorts were based on a linear relative risk model, $RR = 1 + \beta w$, where w represents cumulative exposure in WLM and β is the excess relative risk (ERR) of lung cancer mortality per exposure. Evidence of an exposure-response relationship was found for all cohorts evaluated, and this relationship was adequately described by a linear dose-response function. The overall magnitude of the ERR per unit exposure varied substantially among the cohorts, ranging from 0.2 to 5.1 per 100 WLM. This heterogeneity persisted even after adjustment for potential modifiers [2]. The effects of modifying factors were reasonably consistent among the eleven cohorts [114]. ERR per unit exposure was found to depend on time since exposure and attained age. ERR was also found to depend on exposure rate or, alternatively, exposure duration, with an increase in risk with decreasing exposure rate (or increasing exposure duration). Note that delivering the same cumulative exposure over a longer period of time (i.e., increased exposure duration) decreases the exposure rate. The magnitude of ERR per unit exposure was not found to depend on age at start of exposure [114].

Both the NCI and BEIR VI groups have presented analyses based upon the combined datasets from all eleven occupational cohorts [2, 114-116]. Two models for predicting relative risk were preferred by both groups. Referred to as the *Exposure-Age-Duration* and *Exposure-Age-Concentration* models, they are linear in their dose-response relationship, and contain three categorical covariates (both risk models contain parameters for different categories of attained age and smoking status, while the third parameter denotes either exposure duration or exposure rate). For more details on these radon risk models, the reader is referred to Section 2.4.2.3, or the BEIR VI report [2].

Although the parameter estimates for these models were altered slightly when the updated data were used, the general pattern of effects observed by both groups was comparable [2, 114]. For a given level of exposure, the ERR declined with increased time since exposure and with attained age. Further, lung-cancer risk increased with either lengthening exposure duration or decreasing exposure rate [2].

Of particular interest was the application of the above models to assess the risk associated with exposure to radon among the general public. Analyses indicated significant lifetime relative risks (LRR) for lung cancer mortality at elevated levels of exposure to radon (LRRs of 1.52, 1.58, 1.58, and 1.62 were predicted for lifetime exposure at 150 Bqm^{-3} for male ever- and never-smokers, and female ever- and never- smokers, respectively). Moreover, a marked increase in LRR was predicted for lifetime exposure at increasing radon concentrations. This effect was more pronounced in women and in never-smokers, reflecting their lower baseline lung-cancer mortality rates. Overall, BEIR VI estimated that 10-15% of the approximately 157,400 lung-cancer deaths occurring annually in the U.S. may be attributable to residential radon. As expected, this proportion is greater in never-smokers (19-26%, depending on the risk projection model and assumptions used) than in ever-smokers (9-14%), though due to their baseline rates, the majority of radon-induced lung cancer deaths still occur in smokers [2]. A formal analysis of statistical variation and other sources of uncertainty in BEIR VI suggested between 3,300 to 32,000 lung cancer deaths are attributable to residential radon each year in the U.S. [117].

From a risk management perspective, it is important to note that only one-third of the radon-induced deaths were found to arise from exposures above 148 Bqm^{-3} – the level at which radon mitigation is advised by the U.S. Environmental Protection Agency [2]. Hence, the committee estimated that completely eliminating all exposures above this guideline would reduce total lung cancer mortality by only 4.2% [2].

A recent study by Brand et al. [118] adapts the BEIR VI analysis to the Canadian situation. Using Canadian data for smoking, mortality and indoor radon concentrations, the study estimates a population attributable risk (PAR) proportion for lung cancer due to residential radon exposure of about 8% for Canadian homeowners. Analysis of the uncertainty in this estimate leads to an uncertainty distribution with 5th and 95th percentiles of 4% and 14%, respectively. Stratifying the calculation of PAR by smoking status yields mean estimates of 7% for ever-smokers, and 13.5% for never-smokers, in Canada.

1.5.6 Epidemiologic Studies of Radon in the Indoor Environment

While results of miner studies are unambiguous in demonstrating an excess risk from radon exposure, significant differences between miners and the overall population (including age and sex distributions; smoking patterns; breathing characteristics; short-term, intermittent, high dose occupational exposure versus long-term, chronic exposure at low residential levels; exposure to other occupational carcinogens; different physical characteristics of radon decay products in mines, including the equilibrium factor, and unattached fraction) [119] may call into question the appropriateness of extrapolating risks from miner cohorts to the general public. Accordingly, attempts have been made to study the association between residential radon and lung cancer directly within the indoor environment. These are reviewed below.

1.5.6.1 Ecologic Studies

Soon after the potential hazard of indoor radon was first identified, a number of ecologic studies were performed and reported. They were very diverse in their approach, including comparison studies [120-130] (in which disease rates and mortality are compared in two or more groups), and regression studies [131-138] (in which the outcome measure is modeled as a function of exposure). Groups were generally defined on the basis of geography. Exposure to radon was estimated through a wide variety of means (e.g., geological or soil characteristics of the area; proximity to particular types of mines; radium/radon concentration in well-water; background gamma-radiation levels; and aggregates of indoor radon readings of individual homes). The outcome measure in all studies was age-adjusted lung cancer incidence or mortality. The extent to which potential confounding factors (sex; smoking; ethnicity; socioeconomic factors; urbanization; and mobility) were adjusted for was variable.

The findings of the studies varied widely. Nine of the studies report a positive and statistically significant association between radon and lung cancer [120-122, 124-126, 129, 131, 132, 139]; three studies demonstrate non-significant associations [127, 133, 140]; three studies report no association [123, 128, 130]; and one study shows a significant increase for one histological type of lung cancer (i.e., small-cell carcinoma) in women, but no overall association with radon [137]. Two groups reported a negative association [134, 138] . One of these in particular – the work of Cohen [138], reporting a strong negative correlation between age-adjusted lung cancer mortality rates and average radon concentrations in 1600 U.S. counties – has sparked considerable debate within the radiation protection community [141-150]. Criticism centres primarily on what many consider to be inadequate adjustment for the confounding effects of smoking. An interesting contribution to the debate has recently been made by Puskin [151], who showed an apparent negative ecologic regression of all five principal smoking-related cancers (specifically, cancers of the. lung, oral, larynx, esophagus, and nasopharynx) with radon exposure, even after inclusion of Cohen's indicator variable for smoking prevalence. This is consistent with Cohen's results for lung cancer, but (Puskin contends) impossible to explain for the other four cancers, as they are unlikely be affected by radon exposure, since there is minimal deposition of radon at those sites. Puskin therefore concludes that Cohen's smoking measurements are biased, and his conclusions invalid. Cohen has replied, and the debate continues.

The ecologic study design has some well-documented methodologic limitations, particularly in the investigation of exposure-risk relationships at the individual level [141, 152-154]. In their review of 15 ecologic studies, Stidley and Samet [155] specifically evaluated each study with respect to 14 potential limitations. All studies were found to have multiple limitations. The general consensus is that ecologic studies are not appropriate for quantitatively estimating the risk associated with radon exposure [2]. Given this conclusion, and the plentiful evidence of radon carcinogenicity from both controlled laboratory experiments and epidemiologic studies at the individual level, it is most likely that Cohen's negative ecologic regression coefficients are artefacts of an inadequate adjustment for smoking, and may be disregarded.

1.5.6.2 Case-Control Studies Using Surrogate Measures of Exposure

Many early attempts [156-163] to quantify the effects of residential radon exposure did not measure radon directly, but rather inferred exposure levels from surrogate measures such as the characteristics of the local geology, housing style (e.g., the presence or absence of a basement) or the type of construction materials. In general, these exposure "classifications" have been found to be positively correlated with measured levels of radon within the home [2].

Though the results of these studies vary, the overall pattern of ORs suggests a positive association between the surrogate measure of radon concentration and lung-cancer risk, with an OR for the high-radon houses about twice that for the low-radon houses [2]. The results do not appear to be materially affected by controlling for smoking, when sufficient data on tobacco consumption was available to make this adjustment.

Given the advances in methodologies to directly measure radon concentrations in homes and the availability of data based on such dosimetric methods, research employing surrogate measures of radon exposure are no longer particularly relevant to the radon debate.

1.5.6.3 Case-Control Studies Using Direct Radon Measurements

To date, there have been at least 18 case-control studies of radon and lung cancer published employing direct radon measurements in the home. These include seven in North America [164-171], nine in Europe [172-180], and two in China [181, 182].

The results of these case-control studies have been much more heterogeneous than were the occupational cohort studies. Three studies [167, 176, 181] found no association with radon exposure whatsoever. The remaining studies appear to weakly support a positive association, but inconsistencies in their results warrant caution in this interpretation. For example, a study in Finland [175] reported ORs exceeding 1.0 for all categories of radon exposure, but there was no significant trend with increasing radon concentration, and the highest category had a low OR. In New Jersey [164-166], there was a significant trend detected, but it appears to be strongly influenced by the highest exposure category (OR=8.7, compared to ORs of 1.0, 1.2 and 1.3 for lower exposures), which contained only 5 cases and 1 control. Further, there was no positive trend with increasing radon concentration among never-smokers, a positive trend in light smokers (<25 cigarettes/d), and a negative trend in heavy smokers (>25 cigarettes/d). While a Swedish study [173] that revealed a positive trend in lung cancer risk with increasing radon concentration was affected by the manner in which exposure was defined (continuous Bqm^{-3}, versus exposure categories defined by the mean concentration), this effect disappeared after adjustment for home occupancy, or the time-weighting of exposures as was preferred by the BEIR IV and VI committees.

Such heterogeneity is not entirely unexpected. While direct measurement of radon is preferable to surrogate measures, these all face considerable challenges common to case-control studies. For example, exposure assessment is particularly difficult, as researchers must try to reconstruct exposure profiles for at least the previous 30 years, which includes the period 5-30 years prior to case ascertainment considered most relevant for radon-induced lung cancer. Non-differential misclassification of exposure will likely bias the slope of the exposure-response relationship towards a null association [183, 184]. Population mobility can also contribute to difficulties in exposure measurement, and will also reduce the range of radon exposures among the study subjects. This decreases the study's statistical power to detect an association between radon and lung cancer [185-187]. Further, controlling for confounding and modifying factors is a challenge, with a sub-multiplicative interaction between radon and smoking hypothesized [2]. Because of this joint effect, it may be very difficult to detect an association between radon and lung cancer in mixed smoking and non-smoking populations [188]. Finally, misclassification of cases can occur with lung cancer, which – like errors in exposure measurement – may bias results towards the null.

Attempts have been made to counteract such effects through various means. Independent histological confirmation of lung cancer has been done in many studies to minimize outcome misclassification. Field et al. [189] employed improved retrospective radon exposure measurement, apparently increasing their ability to detect an association between radon and lung cancer. Other studies were restricted to populations of only women [165, 172] (since females (i) have lower baseline lung cancer rates, (ii) have higher average occupancy rates, and therefore higher average residential exposures, and (iii) are less likely to be occupationally exposed to radon), non-smokers [174], or both [168]. These efforts generally failed to produce evidence of a significant association, due to sample size limitations.

Indeed, recognizing that residential radon exposures are usually low and their anticipated effect is quite small, large sample sizes will most likely be required both for the demonstration of a statistically significant excess risk and the evaluation of subtle patterns of variation in risk [19]. The sample sizes required may be impractical for any individual study of radon in the indoor environment. A more feasible approach to obtaining a sufficiently large sample is through the combined analysis of multiple smaller studies, either through meta-analysis of their summary results (relative risks or odds ratios) or through re-analysis of the pooled microdata. The results from a number of such efforts are described below.

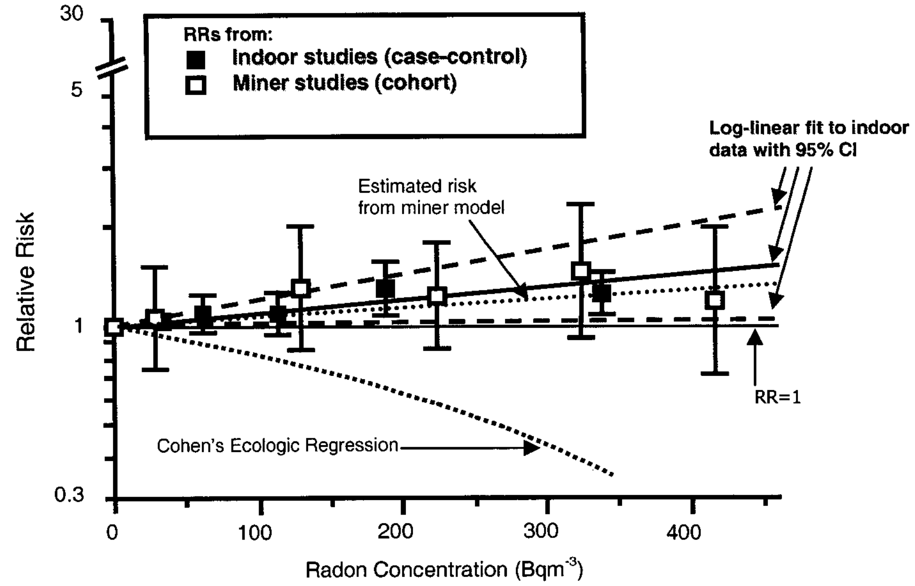
1.5.6.4 Meta-Analysis of 8 Case-Control Studies

In 1997, Lubin and Boyce [190] conducted a meta-analysis of eight case-control studies of indoor radon [165, 167, 168, 172, 173, 175, 181, 191]. In total, 4,262 lung-cancer cases and 6,612 controls contributed to the meta-analysis. A log-linear model, adjusted to pass through the quantitative value for the baseline exposure category, was found to provide good fits to the OR from 7 of the 8 individual studies (a significant departure from linearity was found only for the study conducted by Auvinen et al. [176]). Study specific ORs at 150 Bqm^{-3} differed significantly, ranging from 0.8 to 1.8. The inclusion of a number of variables based on study characteristics (including overall mean radon concentration; % of cases who smoke), both individually and jointly, could not explain the heterogeneity in the study specific exposure response estimates [190].

The meta-analysis of all 8 studies revealed a significant exposure-response relationship, with an overall OR of 1.14 (95% C.I. 1.0-1.3) based on all studies combined. An influence analysis indicated that the overall estimates changed very little when any single study was omitted [190].

Meta-analyses are known to have numerous limitations, including an inability to explore the consistency of results within and between studies, to control for confounding factors, and to evaluate subtle effects [2, 192]. However, as shown in Figure 1-5, these results generally confirm the miner-based extrapolations of risk, which are consistent with a small effect on lung cancer associated with exposure to indoor radon.

FIGURE 1-5. Comparison of risk estimates for low-level radon exposure in the occupational and residential environments. Shown are the summary relative risks (OR) from meta-analysis of 8 case-control studies in homes [190] and the RRs from pooled analysis of data on miners with exposures of less than 50 WLM [116]. Trend lines are included for a log-linear model of the case-control data (with 95% CI), a linear model of the miner data, and for Cohen's negative ecologic regression coefficient [138]. Taken from the BEIR VI Report [2]



1.5.6.5 Analyses of pooled data of Residential Studies

A 1994 analysis [193] of pooled primary data from 3 residential case control studies in New Jersey, Shenyang and Stockholm included almost 1,000 cases of lung cancer. It concluded that there were no significant differences between the 3 studies. Fitted linear excess-OR models yielded study specific estimates of OR (and 95% CI) at 150 Bqm^{-3} of 1.7 (0.8-3.8), 0.9 (0.0-1.2), and 1.2 (0.8-2.4), respectively. The combined analysis showed no trend with increasing radon concentration, with a pooled OR estimate of 1.0 (0.9-1.3) at 150 Bqm^{-3} [193]. These results are consistent with no effect of exposure, but are also consistent with extrapolation of RRs from the miner studies [2].

An analysis of the pooled data from all North American residential case-control studies has recently been completed [19]. This analysis involved 4,081 lung-cancer cases and 5,281 controls. Study-specific results exhibited slight numeric deviations from those of the originally published papers, but did not change the general conclusions reached [19]. Estimates of the excess odds ratio (EOR) at 100 Bqm^{-3} ranged from 0.01 to 0.558. Though some variability was observed among individual studies, differences in radon risk estimates were not statistically significant. ORs increased significantly across radon exposure categories for two studies, while all other studies showed results consistent with no increased risk from residential radon exposure. None of the studies showed a significant decrease in lung cancer risk in relation to indoor radon exposure [19].

Pooling all data available from the studies provided limited evidence of increasing ORs with increasing radon concentration. Based on a linear OR model, the EOR at 100 Bqm^{-3} was 0.096 with 95% CI of (-0.01, 0.26). A linear model was found to adequately describe the exposure-response relationship. There was no significant heterogeneity in the EOR estimates by any of the covariates examined, including age, sex, education level, type of respondent (direct verses proxy), smoking status, number of cigarettes smoked per day, duration of cigarette smoking, time since quitting smoking, and histological type of cancer [19].

The authors note that the overall pooled EOR estimate of 0.096 per 100 Bqm^{-3} (95% CI: -0.01, 0.26) is similar to both the BEIR VI estimate of 0.117 (0.02, 0.25) (based on their simple linear ERR model for miners exposed to less than 50 WLM) and to the meta-analytic ERR estimate of 0.12 (0.0, 0.3) [19].

When analysis was restricted to only those subjects with relatively complete radon dosimetry (specifically, at least 20 years of α-track monitoring within the 5-30 year exposure period of interest) and relatively little mobility (no more than 2 residences occupied during the exposure time window), the resulting EORs were uniformly larger than those in the unrestricted data set. The EOR at 100 Bqm^{-3}, 0.176 with 95% CI of (0.02, 0.43), was statistically significant. There was no indication that the increasing EORs with increasing restriction stringency were the result of differences in the relative contributions of the various studies to the combined database [19].

As with meta-analysis, pooled analyses involve challenges due to differences among contributing studies in design, type and method of data collection, source population, quality-control procedures, information on important confounding variables, and time [194]. Recognizing this, the U.S. Department of Energy (DOE) coordinated investigator workshops in 1989, 91, and 95, to harmonize design protocols and facilitate the eventual pooling of data [195-197]. Data pooling efforts have proceeded independently in North America and in Europe since the mid-1990s. The results from analysis of the pooled European studies will soon be published, reporting on over 11,000 cases and 20,000 controls exposed to much higher average radon levels than the North American subjects. Publishing of the pooled results from European date will be followed by a global pooling of data from North America, Europe and Asia. That combined analysis will be the definitive statement on the lung-cancer risks associated with exposure to residential radon.

1.5.7 Radon and Other Health Effects

The majority of published studies of potential adverse health effects of radon exposure focus on lung cancer, with good reason. Dosimetric analysis indicates that the annual radiation dose resulting from exposure to a given concentration of radon that is absorbed by lung tissues is *at least* a full order of magnitude above that received by other adult tissues [2, 198]. Still, concern has been raised in the literature about the non-lung cancer health effects of radon, specifically (i) cancers other than lung cancer in miners and the general population, (ii) non-malignant respiratory diseases in miners, and (iii) adverse reproductive outcomes of pregnancies in the wives of uranium miners and in the general population within uranium mining / processing communities.

1.5.7.1 Radon and Non-Lung Cancers

A 1989 letter to the editor by Lucie [199] described an association between country-level leukemia incidence and average radon concentration. Subsequently, Henshaw et al. [200, 201] reported associations at the ecologic level between levels of radon and the incidence of some cancers other than lung cancer, in several countries. Other ecologic studies have since been published [202-215], with a number finding statistically significant associations. However, the validity of ecological measures of association for the demonstration of risk at the individual level has once again been questioned. The Henshaw et al. study in particular has received a great deal of criticism for this and many other reasons [204, 216-221].

Studies at the individual level appear to support the null hypothesis of no association between radon and other cancers. For instance, of the main case-control studies of radon and leukemia [222-227], only one suggested a weak association [224]. It should be noted that this study involved only 27 cases, the fewest of any study, and used a surrogate measure of exposure. By comparison, Law et al. [227] – who did not find an association between radon and leukemia – recruited 578 cases, and used duplicate 6-month measurements of radon levels in the home.

A number of mining cohorts have been investigated for a link between radon and other cancers [102, 113, 228], and some have returned positive results (statistically-significant excesses of deaths from cancers of the liver, gallbladder, and extra-hepatic bile ducts were noted in the Czech Cohort [228]). Overall, there were no consistent patterns of excess non-lung malignancies across the cohorts. Darby et al [229] performed an analysis based on pooling of data from 11 underground studies. They observed no excess deaths from cancers other than lung cancer (O/E, 1.01; 95% CI, 0.95-1.07).

Upon reviewing the evidence, the NRC BEIR VI Committee concluded that radon and its progeny are not a major cause of non-lung cancers in the general population [2].

1.5.7.2 Radon and Non-Malignancies

The literature pertaining to a possible association between radon and non-malignant lung disease, as well as reproductive outcomes, is very sparse. After reviewing this literature in detail, the BEIR IV and VI committees both concluded there is insufficient evidence to support radon as a causal factor in their occurrence [2, 46].

1.6 ECONOMIC EVALUATION OF RESIDENTIAL RADON MITIGATION

On the basis of the evidence presented above, risk management programs have been implemented to reduce exposure of underground miners to radon. However, the low average radon concentrations in homes; the relatively small absolute risk associated with exposure; the significant costs of locating and mitigating those at elevated levels of risk – and the uncertainties surrounding all of the above – have contributed to persistent questioning of the appropriateness of directing risk-management strategies towards the full range of population exposures [2].

There are a large number of potential strategies that regulators might employ to control indoor radon exposure in Canada. Choices must be made among them. As additional public funds for such endeavours are scarce at best, financial considerations must factor into the decision-making. The implications of each strategy will likely be complicated, and difficult to compare. Decision-makers require a tool to discern which of the possible strategies will likely yield the 'best' result, whatever they deem that to be. Economic evaluation is one such tool.

1.6.1 Introduction to Economic Evaluation

Economic evaluation can be generally defined as *the comparative analysis of alternative courses of action in terms of both their costs and consequences* [230]. Thus, to perform an economic evaluation of potential radon control strategies, one must *identify*, *measure*, *value* and *compare* the costs and consequences of the programs being considered.

Four forms of economic evaluations are usually identified, characterized by the way in which program consequences are defined and compared. Perhaps the most general is **cost-effectiveness analysis (CEA)**, in which costs are related to some effect (e.g., % reduction in exposure; deaths averted; life-years gained) that is common to all alternative programs, but the magnitude of which may differ. Comparisons are made on the basis of cost-effectiveness ratios – the cost per unit effect (e.g., cost per life-year gained), or alternatively, effect per unit cost (e.g., life-years gained per dollar spent). If one performs a CEA to compare alternative programs in which the consequences are equivalent, then the evaluation is essentially a search for the least costly alternative. This is often termed **cost-minimization analysis (CMA).**

Cost-utility analysis (CUA) is another specialized form of CEA, in which consideration of program consequences is extended to include the *value* of those results. The assigned value –

termed a utility – reflects individual or societal preferences for a particular set of health outcomes (e.g., for a given health state, or a profile of states through time). CUA is useful for health research in that it (i) allows for consideration of quality of life that results from program outcomes, and (ii) provides a generic outcome measure (i.e., *quality-adjusted life years, or QALYs*) for comparison of costs and consequences of programs with different effects. QALYs also allow for the simultaneous consideration of multiple program effects (e.g., reduced incidence of heart disease, COPD, and lung cancer from smoking cessation), whereas CEA in general is restricted to consideration of one output at a time.

Program consequences are also valued in **cost-benefit analysis (CBA)**, allowing for comparison of programs with different and perhaps multiple effects, as described for CUA. Rather than utilities, however, CBA expresses all program outcomes in terms of dollars, forcing one to consider what, for many people, are uncomfortable questions, such as: "How much would you pay to live without cancer?" or "What is the value of saving one human life". A number of techniques have been developed in order to assess the dollar value assigned for a particular result [231-235]. Higher dollar amounts represent greater preference for that state. The outcome of CBA is then simply the difference between a program's costs and the assumed monetary value of its benefits, with differing magnitudes of this difference the basis of comparison of multiple programs. Despite some attractive features, CBA remains a challenging type of economic evaluation, primarily due to difficulties in deriving a valid and reliable estimate of the appropriate dollar amount at which to value the prevention of a given state of health (including death).

There is a vast literature on the economic evaluation of health-related programs, full discussion of which is beyond the scope of this thesis. Interested readers are referred to a number of excellent introductory textbooks [230, 236-239] as a starting point for further information on economic analysis in general, or any of the above-mentioned methodologies in particular.

1.6.2 Economic Evaluations of Indoor Radon Control Strategies

To date, there have been at least 29 full or partial economic evaluations of radon remediation within the residential setting, performed in Canada [240-243], the U.S. [244-252], Great Britain [253-266], Spain [267, 268] and other European countries [269-271]. This number does not include any work done to evaluate programs to control radon in drinking water [272], nor those to remediate dwellings contaminated by uranium mining activities [273]. These latter studies will not be reviewed here, as their scope differs significantly from that considered in this work.

A comparison of completed economic evaluations of radon control programs is complicated by the fact that there exists a large degree of heterogeneity in the methods used. For example, a number of studies focus solely on the cost-effectiveness of mitigation technology, but fail to account for the cost of locating homes requiring such measures [240-242, 244, 246, 249, 270]. Others detail the costs and effects of mitigation programs run within schools [260, 262], or workplaces [253], and not in residential dwellings. Still others [231, 233-235, 274] had as their purpose the *valuation* of the health affects achieved (e.g., expressed or revealed "willingness to pay"), which – though informative – is not the focus of the present study. These studies are excluded from this review, with the following exception.

1.6.2.1 Canadian Studies

The earliest economic evaluation of residential radon mitigation in Canada was undertaken in an effort to inform deliberations on radon guidelines for Canadian homes [240, 241]. The study considered the distribution of exposures within Canada, based on the 1978-80 national radon survey, and calculated the savings in dose that would result from reducing all excessive concentrations within 17 cities to a number of proposed standards. A linear risk estimate of 1×10^{-4} lung cancers per WLM per year was applied to calculate expected lung cancers prevented from this reduction. The study did not account for the costs of national screening programs. Results indicated that the total costs of such a program would be very high, while the impact would be very low. It was later determined that the estimated cost of radon mitigation used in the analysis (i.e., $7500) was high [243], which would have negatively impacted results in terms of cost-effectiveness. This study was the only economic evaluation available at the time at which the current Canadian guideline was set. The overestimated cost of mitigation, along with the need to define a significant yet not unmanageable population to mitigate, may partially explain why the Canadian action level was set significantly higher than most others in the world (i.e., 800 Bqm^{-3}).

A subsequent Canadian effort [243] provided a full economic evaluation of 5 comprehensive national strategies to test for and mitigate radon exposure within new and existing homes (see Table 1-3). Results were initially presented in terms of the cost per unit reduction in exposure to radon (in WLM), as shown in the table. A limited number of results were converted into an estimated cost per lung cancer averted by applying the BEIR VI risk model.

TABLE 1-3. Total Cost and Cost Effectiveness Ratios for 5 radon mitigation strategies in Canada, 1992. Taken from Letourneau et al. [243].

Mitigation Option	Exposure Guideline (Bqm^{-3})	Total Cost[a] (millions of $)	C.E. Ratio[a, b] (1000s of $ / WLM)
(1) Mitigate existing homes	800	306	54
	400	401	26
	150	1067	25
(2) Building cost change	800	11,915	75
(3) Retrofit + building code change	800	12,231	74
	400	12,326	71
	200	12,982	64
(4) Mitigation at point of sale	800	350	33
	400	460	16
	150	1222	15
(5) Mitigation at sale + building code change	800	5,406	74
	400	5,445	71
	150	5,712	65

[a] Costs are calculated in 1992 CAN dollars by applying a 5% discount rate
[b] Cost-effectiveness ratios, based upon a 75% exposure reduction.

All mitigation options analysed were judged by the authors to be unreasonably expensive, with the smallest possible expenditure for a national program predicted to be $ 305 million. The most cost-effective option was screening and mitigation of a dwelling at the point at which it is sold, which yields a cost per WLM of reduced exposure of $33,000 at the current Canadian guideline of 800 Bqm^{-3}. Applying the BEIR IV risk model, this translates into a cost per lung cancer averted of $127,000. Note that the cost-effectiveness of the programs was found to increase as the exposure guideline decreased – a function of decreasing costs per 'at-risk' home identified within the population. Similarly, it was found that such programs were much more cost-efficient within populations having relatively high average exposure levels (e.g., Winnipeg – Geometric mean (GM) exposure: 57.0 Bqm^{-3}; cost per lung cancer averted (intervention at 800 Bqm^{-3}): $8,000) than otherwise (e.g., Vancouver – GM exposure: 5.2 Bqm^{-3}; cost per lung cancer averted: $13,690,000). These regional differences in cost-effectiveness depending on residential radon levels suggests focussing radon risk mitigation efforts on such 'high risk' areas, at least initially.

1.6.2.2 U.S. Studies

A number of U.S. studies have also evaluated different radon control strategies. For universal screening at the national level, Marcinowki and Napolitano [248] found that the incremental cost per life saved ranged from $400,000 to $2,400,000 (assuming 100% national compliance with the action level under consideration). Cost-effectiveness was found to decrease with a decreasing action level (contrary to the Canadian results described above), and with decreasing compliance. It was observed that the costs of risk reduction could be significantly reduced by targeting mitigation efforts towards the 12 states with the highest radon levels (i.e., $400,000 to $600,000 per life saved at an action level of 4 picocuries per liter (pCi/L) (i.e., 148 Bqm^{-3}), compared to $700,000 for a national program). A change in the building code was found to be the most cost-effective means of controlling indoor radon exposure (e.g., $337,000 per lung cancer averted when requiring active mitigation systems to all homes within high-risk areas; reducing to $114,000 when adding a requirement for passive mitigation systems within medium risk areas). The most efficient strategy observed in the 1992 Canadian study – mitigation at the point of sale – resulted in an average cost per life saved of $414,000 if restricted to the targeted states, but $733,000 if implemented at the national level.

Ford et al. [251] developed a decision-tree model to compare the cost-effectiveness of various strategies, ranging from no formal intervention program to universal screening and mitigation. The estimated cost per lung cancer prevented under a universal screening program with mitigation at 148 Bqm^{-3} was approximately $3 million, or $480,000 per life-year (LY) saved. Once again, focussing mitigation efforts within areas of relatively high risk was found to yield significant benefits, as the costs dropped to $2 million per life, or $330,000 per life year gained, respectively.

In addition, the authors note that the costs could be reduced to as much as \$520,000 per life (or \$80,000 per life year) gained by adding a requirement for a second, confirmatory test as part of the screening protocol.

Lin et al. [252] constructed a hierarchal Bayesian decision model to compare four strategies to screen and mitigate radon exposure within the U.S. housing stock. The strategies differed in the length of monitoring used (i.e., short term; short term corrected for bias; long term), and also in the breadth of screening (i.e., 'high-risk' targeted versus universal). Their methodology included a model of the probability a homeowner will decide to remediate, conditional upon the radon measurement obtained. Analysis indicated the targeted strategy to be most cost effective (\$87,000 per life saved when the action level is set at 148 Bqm^{-3}), while universal screening programs functioned most efficiently when employing long-term measurements (\$119,000 per life saved, versus \$138,000 and \$228,000 for corrected and uncorrected short terms measurements, respectively). In all cases, cost effectiveness was increased by raising the action level to 8 pCi/L, or 296 Bqm^{-3} (\$42,000 per life saved by the targeted strategy).

1.6.2.3 European Studies

The most substantial body of recent work on the cost-effectiveness of radon control comes from the United Kingdom. An ongoing study involves the analysis of homes in which mitigation has already been performed by a single contractor within an area known to have relatively high average radon concentrations. The authors "simulate" a population-level screening program by calculating the number of dwellings that, when screened at random, would yield a number in excess of the radon guideline that is equivalent to the number of dwellings in which mitigation was actually performed by the contractor, given the known distribution of radon levels within the area. Results have been reported when the pool of remediated dwellings has included 48 homes [254], 62 homes [255], 65 homes [256], 73 homes [257, 258], and 77 homes [264]. The authors compare the recorded pre- and post- mitigation radon measurements within remediated dwellings – corrected for seasonal variation and standard weights for room occupancy – to calculate the average dose savings per occupant. The estimated number of lung cancers likely averted by this savings in population dose is calculated by applying a lifetime risk estimate of 3.4×10^{-4} lung cancers per WLM per year. These results are compared with the total costs from the actual mitigation and the "screening program" to estimate the cost-effectiveness of a population-level residential radon control program.

The most recent estimate predicts an optimal cost per lung cancer averted of about £900,000 (roughly $2 million CDN), obtained through regional screening program and 100% compliance with an action level of 300 Bqm^{-3} [264]. Costs per life year gained have been estimated to be £13,250 ($30,400 CDN) at 11% compliance, decreasing to just over £5,000 ($11,500 CDN) when compliance rose to 30% [255]. An upward trend in costs was observed with an increasing action level (£1.225 million per lung cancer prevented by 100% compliance with an action level of 500 Bqm^{-3}, increasing dramatically to £10 million when compliance was only 10%) [264]. Cost-effectiveness was shown to be substantially better within regions of higher average radon levels [266]. Also, it was shown that mitigation within newly constructed homes was less cost-effective than universal screening and mitigation of existing homes with 20% compliance or better, but was more cost-effective if compliance was only 10% (which surveys indicate more closely resembles the true rate) [257].

Colgan and Gutiérrez [267, 268] examined the cost-effectiveness of mitigating existing homes (a universal screening program) and new homes (based on radon-resistant construction) in Spain. The number of homes affected was predicted through consideration of the national distribution of concentrations, obtained through a survey of the housing stock. They estimated the savings in population dose that could be achieved, and converted this to savings in lung cancer based on a risk estimate of 1.32×10^{6} lung cancers per Bqm^{-3}. Results indicate a cost per lung cancer averted of $165,000 for intervention in existing homes at 400 Bqm^{-3}, and $145,000 for intervention in new dwellings at 200 Bqm^{-3}, assuming 100% compliance in both cases [268]. Cost effectiveness was improved by directing mitigation efforts towards areas with higher background radon levels (the cost per life saved drops from $480,000 to $20,000 for intervention in new homes with areas with average radon concentrations of 250 and 1000 Bqm^{-3}, respectively) [268].

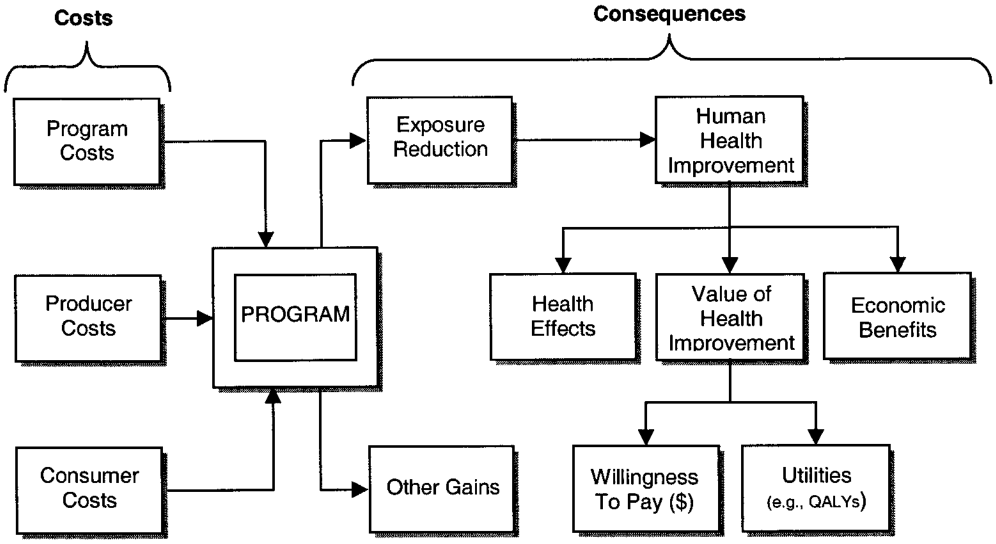
The authors derived an optimize action level for mitigation in existing dwellings of 300 Bqm^{-3}, but noted that the net benefit varied by less than 10% within the 200 to 600 Bqm^{-3} range, and that the optimized value was dependent upon assumptions regarding the costs of remediation and the costs averted (though reduced lung cancers, treatment, etc.) of reduced exposure.

CHAPTER 2
MATERIALS AND METHODS

2.1 STUDY DESIGN: OVERVIEW

This study provides a simulation-based economic evaluation of five potential national strategies to control residential radon exposure within the general population. Analysis follows the decision-oriented framework shown in Figure 2-1, which was developed by Torrance and Krewski [275] for the evaluation of environmental health risk management strategies.

Figure 2-1. A Framework for the Economic Evaluation of Residential Radon Risk Mitigation Strategies. Environmental health risk management strategies are designed to achieve a reduction in exposure to a contaminant – in this case, environmental radon. Reduced exposure is assumed to lead to improved human health, the benefits of which may be captured in terms of (i) observable health effects, and (ii) the value of same, measured both by their economic impacts (e.g., treatment costs averted) and subjective value placed on them by society.



A decision model has been developed which links the implementation of each strategy to its expected resultant changes in radon exposure, lung cancer risks, and ultimately population health impacts. A number of outcome measures – based upon the modelled cost and benefits of the proposed mitigation program – are calculated, thereby allowing for a comparison of the different strategies considered.

2.2 STUDY FRAME

2.2.1 Point Of View

All costs and consequences resulting from the radon control strategies are calculated from a societal point of view. This broad lens is most appropriate when considering public policy interventions affecting the general population [230].

2.2.2 Planning Horizon

The radon control strategies considered here will be implemented over a period of time. Additional time will be required before the benefits of any intervention will be observed, as (1) a minimal latent period of 5 years is hypothesized for radon-induced carcinogenesis; (2) the impact of radon upon lung cancer risk is related to long-term cumulative exposure, with the most relevant exposure window thought to be the previous 5-30 years; and (3) lung cancer is a disease of the aged requiring extended follow-up of the general population to properly assess its occurrence. The combination of these three factors indicates that the most substantial effects of reduced radon exposure will occur within the population of Canadians who are currently young enough to live a substantial period of their lives at these reduced radon levels.

In order to compare the different strategies of interest, it is necessary to specify a planning horizon – a period of observation that is consistent across strategies. An 80-year planning horizon is employed in this analysis. Eighty years is a convenient number, as it approximately equals the current life expectancy of both an average Canadian, and an average Canadian dwelling. Thus, the strategies will be considered over a period that will see a turnover in the majority of the population and the housing stock

2.2.2.1 Adjustment for Time Preference

The gradual implementation of various radon control strategies over time has important implications for economic evaluation. The rate at which homes are affected (i.e., screened, and then mitigated) will differ between strategies, as will the rate at which the costs and benefits of the chosen intervention are incurred (referred to as cost and benefit *streams*). This is significant in light of the existence of *time preference*: individually, and as a society, we prefer to have dollars or resources available now as opposed to later, so we can benefit from them in the interim. Further, more weight is placed upon benefits that occur today rather than in the future [230].

In order to be comparable, program costs and consequences that will be incurred over time must all be measured in present day dollars. Future dollar cost and benefit streams will therefore be discounted to their net present value, to reflect the fact that dollars spent or benefits received in the future do not weight as heavily in program decisions as dollars spent or benefits received today. The required calculation is as follows:

$$C_0 = C_1 + \sum_{t=2}^{k} C_t (1+r)^{-t} , \qquad [1]$$

where C_0 is the total equivalent cost in present day dollars, C_t is the annual cost in year $t = 1,2,\ldots,k$, and r is the annual discount rate. Note that the above formula assumes that all costs are incurred at the beginning of each year, in which case the costs from the first year C_1 are not discounted. In this thesis, a 5% annual discount rate will be used for base results, a rate that is standard for most economic analyses [230]. Alternative discount rates of 0% and 10% will be used to investigate the sensitivity of results to discounting, and the effect these alternative discount rates would have on the residential radon risk management decisions.

2.2.3 Affected Housing Stock

In this thesis, the intervention is assumed to be directed towards dwellings that (1) are private, single family dwellings; (2) exist, or will exist, within Canada over the next 80 years, and (3) have the potential to exhibit elevated radon concentrations. Mitigation programs are assumed to specifically exclude the following dwellings:

1. *Apartments* (except detached duplexes), as the majority of dwellings contained therein will be above the second floor, and therefore will contain relatively low radon levels (see Section 1.3). Note that some dwellings on the first 2 floors may indeed have

elevated radon levels, but mitigating such homes would likely require somewhat different methods than are considered here.

2. *Moveable dwellings*, as the absence of a basement will both reduce radon levels and require different mitigation methods.

3. *Collective dwellings* (e.g., health-related facilities, penitentiaries, and shelters), for reasons similar to those stated for apartments.

The distribution of Canada's 11,562,975 occupied dwellings by structure type in 2001 is shown in Table 2-1.

TABLE 2-1. Occupied Private Housing Stock by Structure Type, Canada, 2001 [276]

Type	No. Dwellings	Affected?
Single, detached	6,615,365	Yes
Semi-detached	561,345	Yes
Row house	615,870	Yes
Apartment, detached duplex	419,755	Yes
Apartment, 5+ stories	1,050,190	No
Apartment, 1-4 stories	2,100,850	No
Other single attached	42,060	Yes
Movable dwelling	157,560	No
TOTAL	**11,562,975**	**8,254,395 (71.23%)**

We assume that this distribution is maintained throughout the planning horizon, for new as well as existing homes, and that it is not associated with any other housing characteristics. Thus, noting the exclusion criteria cited above, the mitigation strategies considered here are assumed to *potentially* affect 71.23% of the Canadian housing stock over the next 80 years.

2.2.4 Affected Population

The benefits of any mitigation strategy can only be expected to occur within that segment of the population residing in dwellings in which the program induces a change in exposure levels. However, the Canadian population is unevenly distributed by housing structure type. In 2001, the distribution of population by dwelling type was as follows:

TABLE 2-2. Population by Structure Type, Canada, 2001 [277]

Type	Population	Affected?
Single, Detached	19,046,200	Yes
Other Radon-Affected	4,395,198[a]	Yes
Apartments	5,716,265[a]	No
Movable Dwelling	364,645	No
Collective Dwelling	479,515	No
TOTAL	**30,001,823**	**23,441,398 (78.12%)**

[a] Estimated by assuming equivalent average household size (1.8) for all apartments

Note that the original data used to generate Table 2-2 counted individuals within semi-detached homes, row houses, apartments (detached duplex), apartments (1-4 stories) and other single-attached dwellings as a single group, comprising a population of 8,176,725 people within a total of 3,739,855 dwellings. As the mitigation programs under consideration will not affect apartments of 1-4 stories, it was necessary to estimate the population residing within these structures. It was assumed that the average size of household living in these apartments would not differ from those living in apartments of five stories or more, for which separate data was reported (i.e., 1,934,735 persons residing in 1,050,195 dwellings, with an average household size of 1.8). Thus, the population living in apartments of less than 5 stories was estimated to be 2,100,850 dwellings (see Table 2-1) X 1.8 persons per dwelling = 3,781,530 persons. The total population residing in apartments is therefore 1,934,735 + 3,781,530 = 5,716,265, while those in mitigation-affected dwellings (other than single detached ones) total 8,176,725 – 3,781,530 = 4,395,195 persons.

Thus, the radon control programs could potentially affect 78.12% of the Canadian population. As with housing structure type, this distribution of population across housing types is assumed to remain constant over time. For simplicity, our model does not attempt to capture movement of individuals between housing types.

This distribution is assumed to apply to both the current and future Canadian population, as well as immigrants, with one exception. It is unlikely that an immigrant entering Canada will reside in a collective dwelling upon arrival. Assuming that immigrants are distributed throughout the remaining housing stock as per the distribution specified in Table 2-2, it is estimated that radon control programs could potentially affect approximately 80.64% of the immigrant population.

2.3 PROPOSED RADON MITIGATION STRATEGIES

The five national radon control strategies considered in this analysis are based upon those studied by Létourneau *et al*[278]. These options can be summarized as follow.

1. *Mitigation of existing homes.* This option involves screening all currently existing homes, followed by mitigation of radon exposure in those homes where the radon gas concentration exceeds the specified action level.
2. *Building code change.* This option calls for a change in the building code to require the construction of homes in such a way so as to facilitate the installation of radon mitigation equipment, if required, following screening of the newly completed home.
3. *Mitigation of existing homes + building code change.* This option combines features of options (1) and (2) to achieve the maximum possible radon reductions in both existing and new homes.
4. *Mitigation at the point of sale.* Under this option, all homes will be subjected to monitoring and mitigation (if needed) when they are sold.
5. *Mitigation at the point of sale + building code change.* This option calls for both a change to the building code and the screening of existing homes at the point of sale.

Given that the costs and benefits of radon mitigation are viewed in this analysis from the societal point of view, there is no requirement to take into account *how* the above strategies will be funded – i.e., is it a publicly or privately-funded program, or some combination thereof? Note, however, that it is generally assumed here that the individual homeowner would be required to pay for the majority of costs incurred through mitigation (as previous studies have estimated total costs that would place radon mitigation among the most expensive public health programs on record [278]).

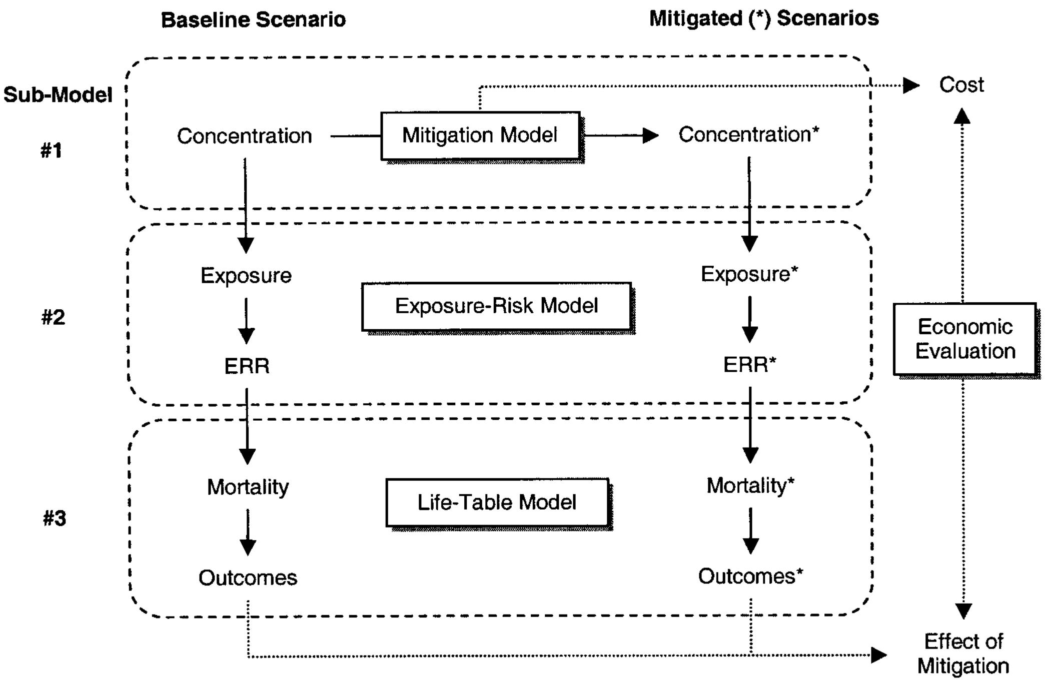
This requirement for the homeowner to pay will act as a disincentive for residential radon mitigation – the effects of which are examined here through analysis of the sensitivity of results to different assumed levels of compliance with the radon guideline.

2.4 DECISION MODEL

A decision model was developed and run using the Microsoft Excel [279] spreadsheet format, with Crystal Ball [280] enhancement to allow for probabilistic simulation. This section describes the model in greater detail.

The decision model is comprised of a large number of interrelated spreadsheets. The spreadsheets may also be grouped into 3 distinct "sub-models", as follows:

FIGURE 2-2. Overview of the Decision Model for Residential Radon Mitigation



As evident from the figure, Sub-Model 1 determines the decrease in radon concentrations that will result from implementation of the mitigation strategy under study. Sub-Model 2 translates this decrease into a change in excess relative risk (ERR) for lung cancer mortality within the population. Sub-Models 1 and 2 are probabilistic in nature: their outputs are the averaged results from repeated simulations (10,000 trials) in which the input parameter values are allowed to vary according to assumed probability distributions

Sub-Model 3 employs life-table methodologies to convert the modelled changes in ERR into population health impacts. In contrast to Sub-Models 1 and 2, Sub-Model 3 is deterministic; calculations contained therein are performed only once, and are based upon averaged outputs from Sub-Model 2, as well as known values of population characteristics (e.g., observed mortality rates).

Note that all three model sub-components were originally intended to be run concurrently. However, the computational requirements for such a simulation exceeded what could feasibly be done within the time frame for this study. Separation of the model into three parts greatly increases its computational efficiency, decreasing the time required for simulation. However, it also requires an important assumption. In Sub-Model 2, we assume that $E(f(c)) = f(E(c))$, where f denotes the function implemented in Sub-Model 2, c represents radon concentration, and E is the expectation operator. Similarly, for Sub-Model 3 (denoted g) we assume $E(g(e_x)) = g(E(e_x))$, where e_x represents age-specific excess relative risk for lung cancer mortality, and E is the expectation operator. The implications of this assumption are explored in the discussion section.

2.4.0.1 Model Validation

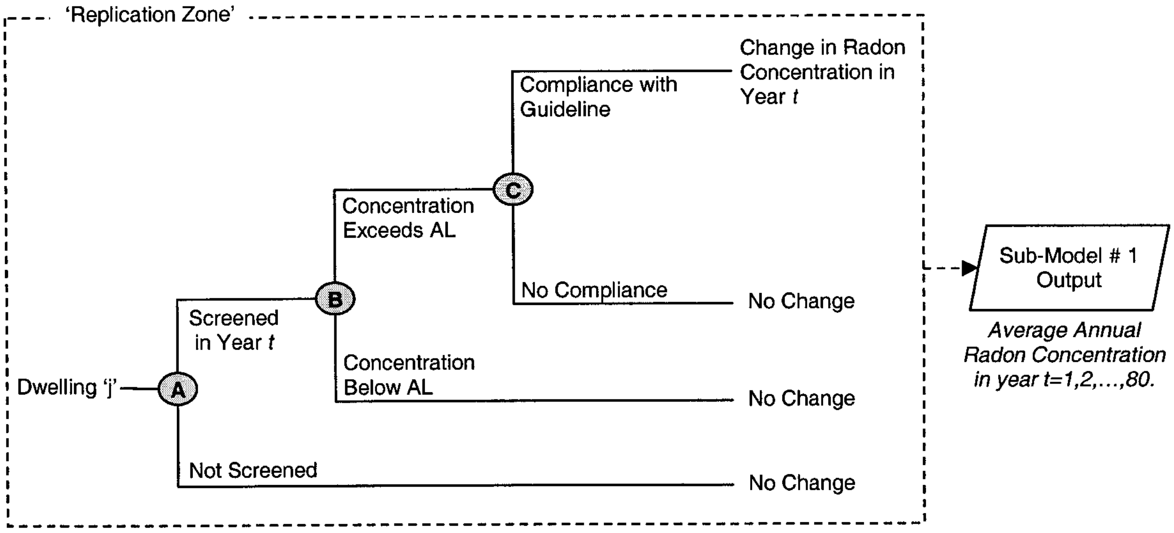
The decision model employed in this analysis was comprised of 31 spreadsheets, housing nearly 65,000 interconnected cells. Significant efforts were therefore made to ensure that the large amount of computer coding required to develop the model was free from errors. Each of the 3 sub-models were validated on an individual basis, ensuring that their outputs were consistent with what would be expected given their inputs. This was also done for the outputs of the decision model as a whole. The results from this validation process are presented in Section 3.1.

2.4.1 SUB-MODEL 1 – Linking Mitigation to Changes in Concentration

A schematic overview of Sub-Model 1 is presented in Figure 2-3, below, showing the link between the mitigation program under study and the resultant changes in average annual radon concentration.

The model starts by randomly sampling hypothetical dwellings from the housing stock that will exist within the 80-year planning horizon, including both current and future dwellings. For each simulated outcome, the model considers up to 2 dwellings (The determination of whether a single dwelling or a pair or dwellings are sampled is described in the following section). This first step involves the determination of whether the characteristics of those dwellings meet the requirements for mitigation by the strategy under study, as described in the next section.

FIGURE 2-3. Overview of Sub-Model 1. A decision-tree is used to model the impact of a particular mitigation strategy upon a series of hypothetical Canadian dwellings. Each 'branch' represents a different path a dwelling might take, ultimately ending at one of two outcomes: mitigation in year t, or no mitigation. The path taken by any given dwelling is determined by a random selection from pre-defined probability distributions for each outcome, depicted in the diagram as nodes (i.e., circles). All steps within the boxed 'replication zone' are repeated for each trial of the Monte Carlo simulation. Each trial yields in a time-dependent concentration profile for a dwelling, estimating post-mitigation radon levels as a % reduction (assumed constant) of the baseline concentration. Averaging these concentration profiles across all trials yields the output for the Sub-Model: a time-dependent profile of average radon concentration within the housing stock.



Probability Distributions
Node A: Housing Stock & Market Characteristics
Node B: Residential Radon Concentrations
Node C: Homeowner Compliance

Decision Variables
Node A: Mitigation Strategy
Node B: Action Level

2.4.1.1 Determining the Characteristics of the Sampled Dwelling(s)

Year of Construction

A number of the mitigation strategies under consideration are directed in whole or in part towards newly constructed homes. For each new home sampled by the model, it is important to determine the year in which it was constructed. New dwellings are assumed to be built for two main reasons: (1) replacement of currently existing dwellings that exceed their lifespan (assumed to be 80 years), or (2) accommodation of increased population size. The model does not allow for the existence of unoccupied homes.

Whether or not an existing dwelling is in need of replacement is assumed to be a function solely of the age of the dwelling age – i.e., the relative state of repair of the dwelling is not taken into account. The number of new dwellings built as replacements within any given year is thus dependent upon the age-distribution of the current national housing stock, as given in Table 2-3.

TABLE 2-3. Housing Stock, by Year of Construction, Canada, 2001[281]

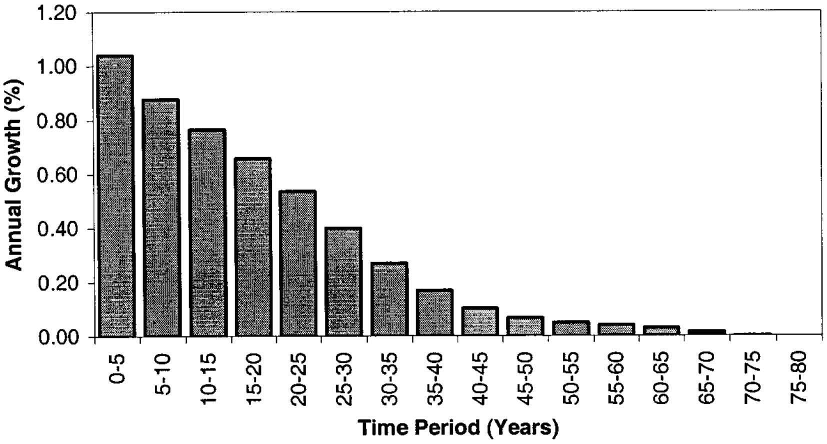
Year of Construction	Homes	%	Age	Remaining Lifespan[a] (years)
1945 or before	1,661,635	14.37	55+	1-24
1946-1960	1,819,730	15.74	40-54	25-39
1961-1970	1,833,290	15.85	30-39	40-49
1971-1980	2,460,455	21.28	20-29	50-59
1981-1985	1,001,665	8.66	15-19	60-64
1986-1990	1,079,075	9.33	10-14	65-69
1991-1995	887,255	7.67	5-9	70-74
1996-2001	819,865	7.09	0-4	75-79
TOTAL	**11,562,970**	**100**		

[a] Assumes a maximum life expectancy for a home of 80 years.

Note that with the assumed maximum lifespan of 80 years, the entire housing stock will be replaced within the planning horizon. We assume the time at which homes are replaced is uniformly distributed within each age interval, leading to an even rate of replacement across the time period of interest. Specifically, replacement homes will be built at a rate of 1,661,635/25 = 66,465 homes per year for the first 25 years, increasing to 1,819,730/15 = 121,315 homes per year for the next 15 years, and so on.

All homes built in addition to those described above are assumed to be necessary to accommodate population growth. A subsequent component of the model involves the prediction of population growth over the 80-year period under baseline and mitigated conditions. Note that as evidence indicates that radon mitigation will have a relatively small impact on the life expectancy of the population as a whole, one can assume that a mitigated population will grow at an average annual rate that is approximately unchanged from baseline conditions. As shown in Figure 2-4, below, our model predicts annual population growth rates ranging from about 1% in the next five years to near zero as the 80-year planning horizon draws to a close.

FIGURE 2-4. Estimated Annual Rate of Population Growth, Canada, By Period

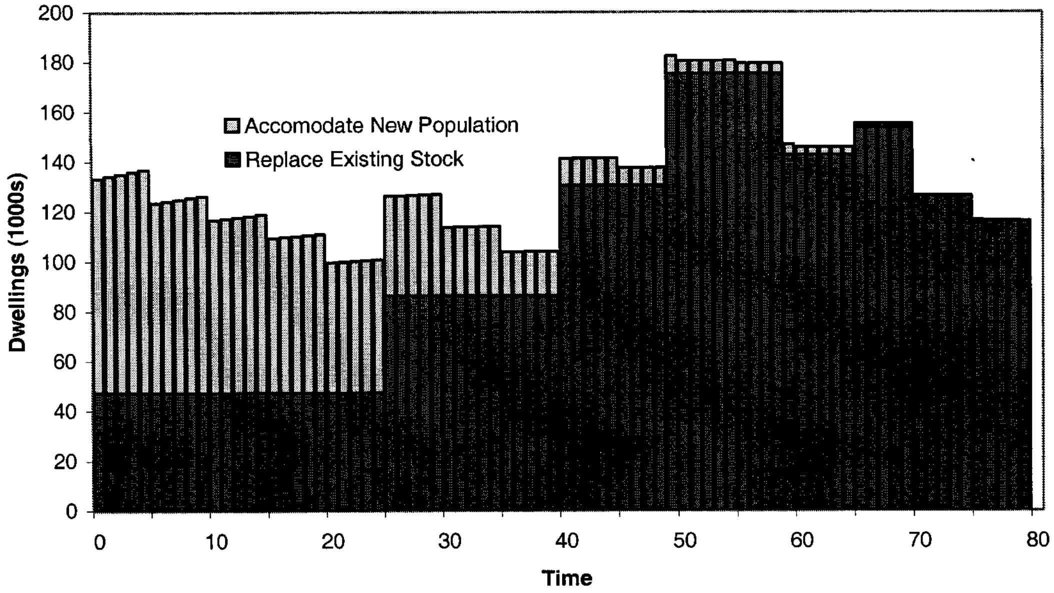


If one assumes a constant household size – currently at 2.34 persons per dwelling [282], then the total size of the housing stock (*H*) would be expected to grow as per the rates specified in Figure 2-2. That is,

$$H_{t+1} = H_t(1 + r_t),$$

where r_t is the rate of growth in year t and H_t denotes the size of the housing stock at time t. Combining this new stock with the replacement stock previously calculated yields the trend in yearly construction rates as shown in Figure 2.5.

FIGURE 2-5. Estimated Number of New Dwellings Constructed Annually, Canada, 2001-2080, by Purpose



The model predicts that 10,596,207 new radon-affected dwellings will be built over the next 80 years, of which 8,246,704 units will be replacements for currently existing stock, with 2,346,263 units required to accommodate the increased population size. Thus, sampling randomly from this pool, the probability that any given simulated new construction will constitute a 'replacement' dwelling is

$$\begin{aligned}\Pr(\text{Replacement}\,|\,\text{New}) &= \frac{\text{No. Replacement Dwellings}}{\text{No. New Dwellings}} \\ &= \frac{8{,}246{,}704}{10{,}596{,}207} \\ &= 0.778.\end{aligned} \qquad [2]$$

The probability that the dwelling is built to accommodate a growing population is therefore 0.222.

When sampling new dwellings in the decision model, we first apply the above probability to determine whether it is a replacement dwelling or one built for population growth. Conditional upon this result, the year of construction is determined in accordance to the probability distributions displayed in Figure 2-5.

Note that if the sampled future dwelling is a replacement, the model considers it as well as the currently existing home it will replace, performing the following steps for each. If the new dwelling is built to accommodate new population, only that home is considered (as it has no precursor).

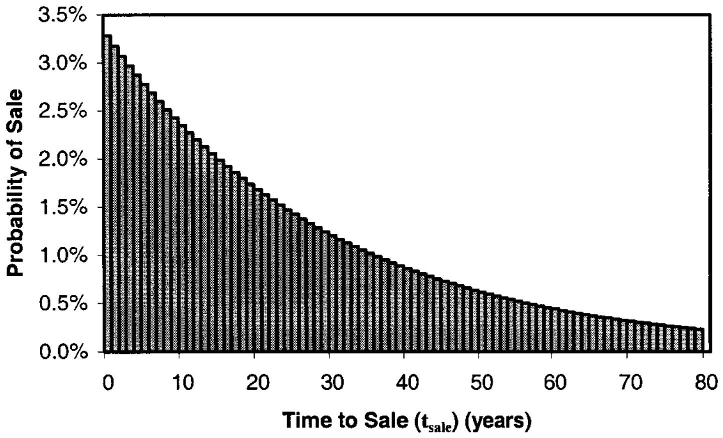
Year of Home Sale

Mitigation strategies 4 and 5 call for homes to be mitigated at the point at which they are sold. The decision model assumes that homes will continue to be sold at a rate equivalent to that observed in 2001, or 3.3% per year [283], and that this rate is independent of all other variables, including the age of the dwelling. Given these assumptions, the probability that a dwelling will be sold within an 80-year period is:

$$\Pr(\textit{Sold in } 80 \textit{ Years}) = 1 - \left(1 - 0.033\right)^{80}. \qquad [3]$$

The above probability is applied in the decision model to determine whether a given home will be sold within 80 years of its entry into the planning horizon (either as an existing home in year t=0, or as a new construction in year t (i.e. the year of its construction)). Given that it is indeed sold, the time until that sale occurs is determined in accordance with the probability distribution shown in Figure 2-6.

FIGURE 2-6. Estimated Time to Sale Canadian Dwellings Sold within 80 Years



This distribution is simply the result for year $t = 1,2,\ldots,80$ of applying the formula

$$\Pr(\text{sale in } t \text{ years}) = 3.3\% \times (1-3.3\%)^{t-1}$$

Note the years shown in the figure represent the 80 year period immediately following entry of the dwelling into the planning horizon. For existing dwellings, these correspond directly with the 80 years of the planning horizon, but for new dwellings they do not. Although this approach is computationally efficient (i.e., it precludes the need to generate 80 different probability distributions for year of sale, conditional upon the age of the home and/or the year of the planning horizon in which the projection is being made), it may yield an invalid result for the year of sale. In some instances, the value returned may indicate a home is to be sold after it has exceeded its normal lifespan (i.e., $Age_{t=0}+ \Delta t_{\text{sale}} > 80$ years), while in others a newly constructed home may be sold after the end of the planning horizon ((i.e., $t_{construction}+ \Delta t_{\text{sale}} > 80$ years). In both cases, the result is disregarded.

Baseline Radon Concentration

A 1978-1980 survey of 14,000 homes in 19 Canadian cities reported radon concentrations which varied according to a lognormal distribution with geometric mean (GM) of 11.2 Bqm^{-3} and geometric standard deviation (GSD) of 3.9 [31]. These parameters are used in this analysis to characterize the current distribution of radon levels in the national housing stock. However, uncertainty regarding the true exposure distribution has been acknowledged. Therefore, following Krewski et al. [117] and Brand et al. [118], a hierarchal structure is imposed upon the distributional assumption, whereby its GM and GSD are themselves modeled as random variables drawn from independent lognormal distributions (see Table 2-4).

TABLE 2-4. Distributional assumptions characterizing uncertainty and variability in the radon concentrations within the current Canadian housing stock. [117, 118]

	Variability	Uncertainty
Radon Concentration	LN ~ (*gm*, *gsd*)	*gm* ~ LN(11.2, 1.12); *gsd* ~ LN(3.9,1.12)

LN: log-norrmal distribution; *gm*: geometric mean; *gsd*: geometric standard deviation

For each home modelled, a random draw from the above distribution provides an indication of its average annual radon concentration. In the next step, the decision model considers how that concentration might be changed by the mitigation strategy under study.

2.4.1.2 Determining the Results of the Mitigation Program

A strategy to control exposure to residential radon within the general population will be comprised of 3 general components, namely:

1. a strategy to screen homes;
2. an 'action level', above which mitigation is recommended; and
3. a means of reducing radon levels in mitigated homes.

In the present step, the decision model determines the answers to a number of questions related to these components to determine the mitigation outcome for each modelled home.

Screening of the Dwellings

One must first determine when (if at all) the sampled dwelling(s) will be screened for radon. This action is dependent upon the characteristics of the home, determined above, and by the mitigation strategy under consideration. For strategy 1, all existing homes are screened at an equal rate over the first five years of the program, while strategy 2 screens all new homes at the time at which they are built. Strategy 3 combines both of these approaches, screening all existing *and* new homes. Under strategy 4, all homes are screened at the time at which they are sold, and strategy 5 combines this approach with that of strategy 2. Note that the model does not allow for the possibility of individual homeowners choosing to screen their homes without the prompting of the radon control program.

Recommendation of Mitigation

Given that a house is screened, the model then considers whether the home's average annual radon concentration ($[\mathrm{Rn}^{222}]_j$) is at or above that at which mitigation is recommended (i.e., the action level – $[\mathrm{Rn}^{222}]_{AL}$). If $[\mathrm{Rn}^{222}]_j \geq [\mathrm{Rn}^{222}]_{AL}$, mitigation is recommended under the guideline.

We model the effects of mitigation at three different action levels – the current Canadian standard, 800 Bq/m^3, as well as more stringent levels of 400 Bq/m^3 and 200 Bq/m^3.

Requirement for a Confirmatory Test

In a joint publication, Health Canada and the Canada Mortgage and Housing Corporation (CMHC) recommend the following procedure be followed when screening a house for radon [6]:

1. Conduct an initial short-term screening;
2. If 150 Bq/m^3 < $[Rn^{222}]_j$ < 800 Bq/m^3, perform a long-term confirmatory test. (If $[Rn^{222}]_j$ > 800 Bq/m^3, a confirmatory test should be performed immediately);
3. If 150 Bq/m^3 > $[Rn^{222}]_j$, no further testing is required.

Given that dwelling j is screened, the decision model determines whether a confirmatory radon test is required as per the above guideline. To simplify the model, it is assumed that the results of the confirmatory test will have no effect on future steps (in particular, we assume the long-term measurement confirms the initial short-term screening, as will most often be the case). However, the model does take into account the costs of the confirmatory test.

Homeowner Compliance

The radon control programs considered in this analysis are advisory in nature. In such cases, mitigation will not occur without homeowner co-operation. Given that dwelling j is both screened and has an average annual radon concentration in excess of the action level, the homeowner's choice to take mitigation action is determined through the application of a simple probability of compliance for the population. This probability is assumed to be independent of all other variables, including the margin by which the measured radon concentration exceeds the action level. The differential effects of 3 compliance levels – 100%, 50%, and 10% - are examined in this thesis, with 50% being the base case from which the other compliance rates are compared.

Note that if all of the above conditions are satisfied, dwelling j is mitigated in year t.

Mitigated Radon Concentration

Following Létourneau et al [243], all mitigation in Canada is assumed to be performed using active sub-slab depressurization (ASD), a highly effective form of residential radon control [242]. The ASD system is assumed to induce a constant percent reduction of pre-mitigation radon concentrations, in all mitigated homes. Initially, a 90% reduction from pre-mitigation levels is assumed. For sensitivity analysis, a 70% reduction factor will also be considered.

Note that in some situations, a 90% reduction of pre-mitigation radon levels could result in a concentration that is still in excess of the action level (e.g., 90% reduction of 2000 Bq/m^3 yields a new concentration of 200 Bq/m^3, which would still warrant mitigation under a radon guideline of 200 Bq/m^3). Although the model does not allow for any such additional reductions, this type of situation is very rare, and will not introduce a significant amount of error into the analysis.

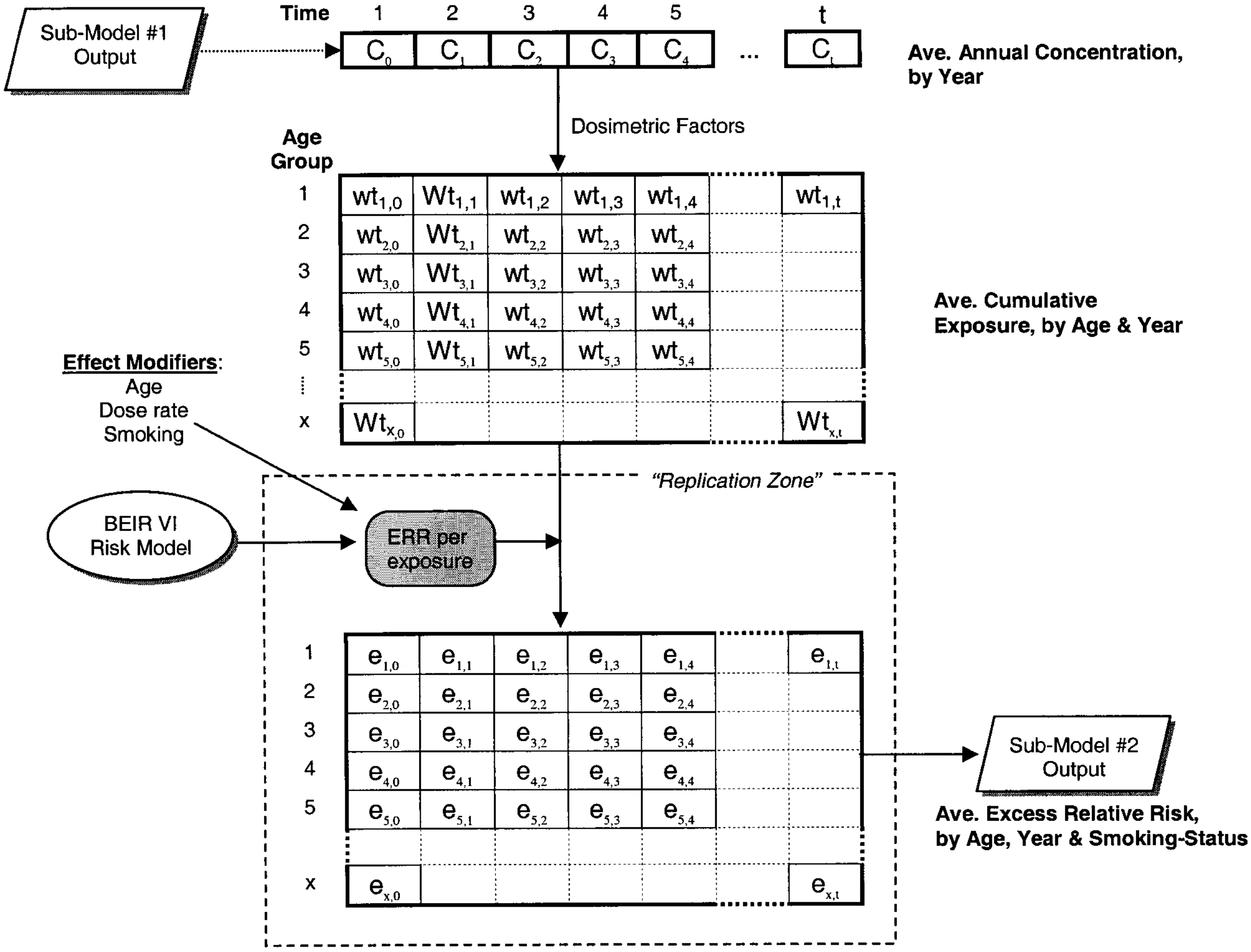
2.4.1.3 OUTPUT: Sub-Model 1 – Time-Dependent Radon Concentration Profiles

For each Monte Carlo replication, Sub-Model 1 returns information on how the average annual radon concentration within a hypothetical Canadian dwelling might change as it ages (and perhaps is eventually replaced), both in the presence and absence of a national radon control strategy. Repeated replications provide many thousands of concentration estimates for every year of the planning horizon, accounting for inter-dwelling variability in concentrations and uncertainty in radon estimates. The results for each year are averaged, yielding a time-dependent average radon concentration profile for the population under both baseline and mitigated scenarios.

2.4.2 SUB-MODEL 2 – Linking Concentration to Excess Relative Risk

Sub-model 2, which is also probabilistic in nature, translates the concentration profiles generated above into age-time-specific estimates of excess relative risk (ERR) for lung cancer mortality. Figure 2-7, below, provides an overview of Sub-Model 2, indicating how the model was implemented in spreadsheet format.

FIGURE 2-7. Overview of Sub-Model 2. A principal feature of this Sub-Model is the conversion of information along a single dimension (i.e., time) into a two-dimensional profile (i.e., age x time), upon which all subsequent calculations are based. This is accomplished by calculating, for 5-year age interval x=1,2,..., x and time t=0,5,..., t, the cumulative exposure previously incurred by an individual within interval x at time t, given the average annual concentration profile output from Sub-Model 1 (repeated for both the unmitigated and mitigated scenarios). The age-time exposure profiles are then translated into excess relative risk (ERR) estimates using the BEIR VI risk model, with parameters sampled randomly from pre-defined uncertainty distributions. As before, the boxed "replication zone" represents the results for a single Monte Carlo trial, with the averaged results from those trials comprising the output of Sub-Model 2.



2.4.2.1 Calculating Cumulative Radon Exposure

The impact of radon upon an individual's risk of dying from lung cancer is primarily a function of cumulative exposure. Cumulative exposure is the product of exposure rate and time, expressed mathematically as

$$w(\Delta t) = \sum_{i=1}^{k} w_i \Delta t_i \qquad [4]$$

for exposure periods i = 1, 2,...,k, where w_i is the average exposure rate during that period (see below) and Δt_i is the duration of that period. The exposure rate is the product of the average annual radon concentration to which an individual is exposed, and a number of dosimetric factors (described earlier in Section 1.2.2). For example, following Brand et al. [118] and Krewski et al. [117], given the assumptions listed in Table 2-5,

TABLE 2-5. Dosimetric assumptions used in the calculation of exposure rate (w) from average annual radon concentration (Bqm^{-3}). [117, 118]

WL and Bq/m^3 at equilibrium	Since 1 WL = 3700 Bq 1 Bq = 0.00027 WL
Equilibrium Factor	= 0.40
Residential Occupancy	= 0.70
"Working Months" per year	$=365.25\frac{\text{days}}{\text{year}}\times 24\frac{\text{hours}}{\text{day}}/170\frac{\text{hours}}{\text{working-month}}$ $=51.6$ working - months per year

exposure to 100 Bq/m^3 yields an average exposure rate of

$$100 \times 0.0027 \times 0.4 \times 0.7 \times 51.6 = 0.39 \text{ WLM/year.}$$

Exposure at that rate for 25 years would therefore yield a total cumulative exposure of approximately 10 WLM.

Radon-related lung cancer risks are determined by lifetime cumulative exposure. For an individual, cumulative exposure at time t is calculated as

$$w(\Delta t)_t = w_0 \times Age_t \qquad [5]$$

if the exposure rate (w_0) has remained constant through throughout their life, and

$$\begin{aligned} w(\Delta \mathrm{t})_t &= \sum_{i=1}^{k} w_i \Delta t_i \\ &= w_1 \Delta t_1^* + \sum_{\mathrm{i}=2}^{\mathrm{k}-1} \left[w_i (\Delta t_i^* - \Delta t_{i-1}^*) \right] + w_k (Age_t - \Delta t_{k-1}^*) \end{aligned} \qquad [6]$$

if he/she has been exposed to different exposure rates w_i, during exposure periods $i = 1,2,\ldots,k$, with $i = 1$ being the most *recent* period, where $w(\Delta t)_t$ is the cumulative exposure incurred up to time t, Δt_i^* is the elapsed time since the start of the ith exposure period, and Age_t is the individual's age at time t.

The above calculation is complicated, however, by the following assumptions made by the U.S. National Research Council's Committee on the Biological Effects of Ionizing Radiation, BEIR VI [2], and adopted in the present analysis:

- there is a 5-year latent period for radon exposure; and
- after that, exposures that occurred 5-14, 15-24, and 25+ years in the past should be given progressively less weight.

Under these assumptions,

$$w(\Delta t)_t = w(\Delta t)_{[5,14]} + \theta_2\, w(\Delta t)_{[15,24]} + \theta_3\, w(\Delta t)_{[25,\infty]} \qquad [7]$$

where $w(\Delta t)_{[a,b]}$ is the cumulative exposure incurred by the individual within the time period between [a] and [b] years in the past, and θ_i are weighting factors ($\theta_2 = 0.77$ for exposures 15-24 years before t, and $\theta_3 = 0.57$ for exposures 25+ years before t).

In the simple case where an individual has been subjected to a single exposure rate throughout their life (w_o), cumulative exposure changes solely as a function of exposure duration, $\Delta t_{[a,b]}$ (in years), calculated as follows:

$$\Delta t_{[a,b]} = \begin{cases} 10 & \text{if } \text{age}_t > b \\ \text{age}_t - a & \text{if } a \le \text{age}_t \le b \\ 0 & \text{Otherwise} \end{cases}$$

By way of example, the weighted cumulative radon exposure attributed to a 50 year-old individual exposed only to w_o would be expressed as

$$
\begin{aligned}
w(\Delta t)_{50} &= w_o \times [\, \Delta t_{[5,14]} + \theta_2 \Delta t_{[15,24]} + \theta_3 \Delta t_{[25,\infty]} \,] \\
&= w_o \times [10 + \theta_2\, 10 + \theta_3\, (50\text{-}25)] \\
&= w_o \times [10 + (0.77) \times 10 + (0.57) \times 25] \\
&\approx w_o \times 31.9 \text{ years}
\end{aligned}
$$

When an individual has been exposed at multiple rates, the calculation of a weighted cumulative exposure is slightly more complicated. One must consider the different exposure rates $w_1, w_2, \ldots, w_k$ to which the individual was exposed *within the relevant time period*, with w_1 being the most *recent* exposure rate within that period, and Δt_i^* again being the elapsed time since the *i*th exposure rate began. If only one exposure rate w_i occurred within the period [a,b] (that is, if $\Delta t_i^* > b$ and $\Delta t^*_{i\text{-}1} < a$), cumulative exposure within that period is simply $w_i \Delta t_{[a,b]}$, with exposure duration $\Delta t_{[a,b]}$ calculated as before.

However, when multiple exposure periods $i = 1,2,\ldots, k$ occur in the [a,b] time interval, then

$$
w(\Delta t)_{[a,b]} = \begin{cases}
w_1(\Delta t_1^* - a) + \sum_{i=2}^{k-1} \left[w_i (\Delta t_i^* - \Delta t_{i-1}^*) \right] + w_k (b - \Delta t_{k-1}^*) & \text{if } age_t > b \\
w_1(\Delta t_1^* - a) + \sum_{i=2}^{k-1} \left[w_i (\Delta t_i^* - \Delta t_{i-1}^*) \right] + w_k (Age_t - \Delta t_{k-1}^*) & \text{if } a \leq age_t \leq b \\
0 & \text{Otherwise.}
\end{cases}
$$

2.4.2.2 Age-Time-Specific Cumulative Exposure Profile

The above method provides a means of calculating the weighted lifetime cumulative exposure incurred by an individual of age x at time t, given previous exposure rates $w_1, w_2, \ldots, w_k$. However, the decision model does not operating at the individual level. Rather, we approximate exposure patterns within the entire population by calculating an age-profile of cumulative exposure at time t

– that is, we simultaneously estimate the exposure incurred by an average member of each 5-year age interval x, given the average concentration profile for the population output from Sub-Model 1. The calculation is based upon a person who is at the start of the age-interval, and is repeated at the start of every 5-year period within the planning horizon. Note that individuals born prior to the start of the planning horizon are assumed to have been exposed during that time at an average rate equal to that present at baseline.

2.4.2.3 Estimating Excess Relative Risk (ERR) of Lung Cancer Mortality

The population profile of age-time-specific cumulative exposure is used to calculate a similar profile representing excess risk of lung cancer mortality resulting from residential radon. This calculation is based upon an extrapolation from data on 11 occupational cohorts of radon exposed miners (described in Section 1.3.3), as was done by the BEIR VI committee [2]. The committee preferred two similar models for predicting the impact of residential radon, referred to as the *exposure-age-concentration* and *exposure-age-duration* models [2]. Since both models give similar predictions of risk, consideration is restricted to the *exposure-age-concentration* model.

The model expresses age-specific excess relative risk of lung cancer mortality at time t, $e_{x,t}$, as a function of cumulative radon exposure $w(\Delta t)_{x,t}$, i.e.,

$$e_{x,t} = \beta \times w(\Delta t)_{x,t} \times \phi_x \times \gamma_w \times \mathrm{K} \times \delta \qquad [8]$$

where β (in WLM^{-1}) is the potency of radon (after controlling for the other modifying factors), K is a dosimetric factor accounting for differences between occupational and environmental exposures, calculated as

$$K = \frac{\mathrm{Dose}_{Home} / \mathrm{Exposure}_{Home}}{\mathrm{Dose}_{Mine} / \mathrm{Exposure}_{Mine}}$$

and δ represents the modifying effect of smoking, where

$$\delta = \begin{cases} 1.92 & \text{for never-smokers} \\ 0.91 & \text{for ever-smokers} \\ 1.00 & \text{for never- and ever-smokers combined.} \end{cases}$$

It is important to note that setting δ = 1.0 yields an estimate of ERR for lung cancer within a population of ever- and never-smokers in which the prevalence of ever-smokers is equal to that found within the occupational cohorts upon which the BEIR VI models are based. This is significant, as applying the models to the general population – which has smoking rates, and hence baseline lung cancer risks, far below those typically found in a cohort of miners – would lead to invalid estimates of radon exposed-modified lung cancer risk.

The categorical variables ϕ_x and γ_w (both unitless) represent the modifying effect of attained age and exposure-rate, respectively, where

$$\phi_x = \begin{cases} \phi_1 = 1 & \text{if } Age_t < 55 \\ \phi_2 & \text{if } 55 \le Age_t \le 64 \\ \phi_3 & \text{if } 65 \le Age_t \le 74 \\ \phi_4 & \text{if } Age_t > 75 \end{cases} \quad \text{and} \quad \gamma_w = \begin{cases} \gamma_1 = 1 & \text{If } \overline{w}_t < 0.5 \\ \gamma_2 & \text{If } 0.5 \le \overline{w}_t \le 1.0 \\ \gamma_3 & \text{If } 1.0 \le \overline{w}_t \le 3.0 \\ \gamma_4 & \text{If } 3.0 \le \overline{w}_t \le 5.0 \\ \gamma_5 & \text{If } 5.0 \le \overline{w}_t \le 15 \\ \gamma_6 & \text{If } \overline{w}_t \ge 15. \end{cases}$$

To simplify the analysis, the modifying effect of exposure rate is calculated based upon the estimated average rate of exposure through an individual's lifetime (less the 5 year latent period), $\overline{w}$, calculated simply as by dividing cumulative exposure at time *t* by Age$_t$, i.e.,

$$\overline{w}_t = \frac{w_1(\Delta t_1^* - 5) + \sum_{i=2}^{k-1}\left[w_i(\Delta t_i^* - \Delta t_{i-1}^*)\right] + w_k(Age_t - \Delta t_{k-1}^*)}{Age_t - 5} \qquad [9]$$

2.4.2.4 Uncertainty Distributions for Model Input Parameters

Following Krewski et al. [117] and Brand et al. [118], a measure of the uncertainty and/or variability within each of the parameters in the BEIR VI model is characterized by some basic distributional assumptions, described in Table 2-6. These assumptions are guided by both empirical data and the expert judgment of the BEIR VI committee.

Note that the inclusion of uncertainty and variability distributions for model input parameters was originally intended to facilitate a full uncertainty analysis in which uncertainty distributions were described for all key results. However, logistical considerations (i.e. computer processing requirements) resulted in the uncertainty analysis being scaled down to a pilot-level analysis only, presented later in Section 4.3.3.

While the carcinogenic potency of radon is assumed not to vary among individuals, reflecting constant susceptibility within the general population, the uncertainty in this estimate is characterized by a log-normal distribution with known geometric mean and geometric standard deviation. Similarly, the modifying effect of age is assumed not to vary between individuals in a given age group, but uncertainty is described via assumed log-normal distributions for the non-referent ϕ's. Inter-individual variability in the K-factor is characterized using a lognormal distribution; uncertainty in the GSD of this distribution is described by a log-uniform distribution. Finally, no uncertainty is assumed to exist regarding the differential effects of radon upon the baseline lung cancer risks of ever- and never-smokers [117, 118].

TABLE 2-6. Distributional Assumptions for Parameters of the BEIR VI Exposure-Age-Concentration Model. [117]

Risk Factor	Variability	Uncertainty
Potency (β)	Constant	~ LN (gm=0.08, gsd=1.36)
Age (ϕ_x)	Constant	
Age ≤ 54 Years		~ LN(1.0, 1.10)
55 ≤ Age ≤ 64		~ LN(0.57, 1.27)
65 ≤ Age ≤ 74		~ LN(0.29,1.39)
Age ≥ 75		~ LN(0.09, 2.55)
"K" Factor	~ LN (gm, gsd)	gm = 1.00; gsd ~ LU(1.2, 2.2)

[a] Log-Normal distribution; [b] Geometric Mean; [c] Geometric Standard Deviation; [d] Log-Uniform distribution

Note that Sub-Model 2 does not take into account variability in radon concentrations within the housing stock. This variability is modeled only within Sub-Model 1, but is "averaged out" before concentration is used as an input into Sub-Model 2.

The central estimates each of the parameters within the models are provided in Table 2-7. The parameters in the model are assumed to vary together in accordance with the covariance matrix in the table [117, 118].

2.4.2.5 OUTPUT: Sub-Model 2 – Age-Time-Specific ERR Profile

Each Monte Carlo trial returns a unique combination of parameters for the BEIR VI ERR model, in accordance with the above probability distributions. Applying these to the age-time-dependent cumulative exposure estimates obtained earlier in Sub-Model 2 – and averaging the result across all trials – yields an age-, time- and smoking-status (ever vs. never) specific profile of excess relative risk for lung cancer. A separate profile is produced for both ever- and never-smokers, under baseline as well as mitigated conditions.

TABLE 2-7. Estimated Values and Covariance of Parameters of the BEIR VI Exposure-Age-Concentration Model. [117]

	I. Estimated Values of Parameters[a]										
	β	θ2	θ3	φ2	φ3	φ4	γ2	γ3	γ4	γ5	γ6
	-2.57	0.77	0.51	-0.56	-1.23	-2.38	-0.78	-0.98	-1.13	-1.8	-2.21
	II. Covariance Matrix[b]										
β	9.47										
θ2	-0.36	0.77									
θ3	-0.04	0.14	0.42								
φ2	-2.87	-0.10	-0.50	5.71							
φ3	-3.18	-0.17	-0.33	2.85	10.87						
φ4	-3.44	-0.19	-0.54	2.90	3.20	87.65					
γ2	-5.57	-0.10	-0.02	0.14	0.42	0.83	8.14				
γ3	-6.36	-0.12	-0.11	0.50	0.53	0.97	5.88	6.93			
γ4	-6.58	-0.16	-0.10	0.18	0.59	1.08	5.83	6.69	7.30		
γ5	-6.90	-0.05	-0.09	0.26	0.61	0.81	5.69	6.51	6.67	7.84	
γ6	-7.04	-0.02	-0.08	0.27	0.54	0.50	5.63	6.44	6.64	7.33	8.59

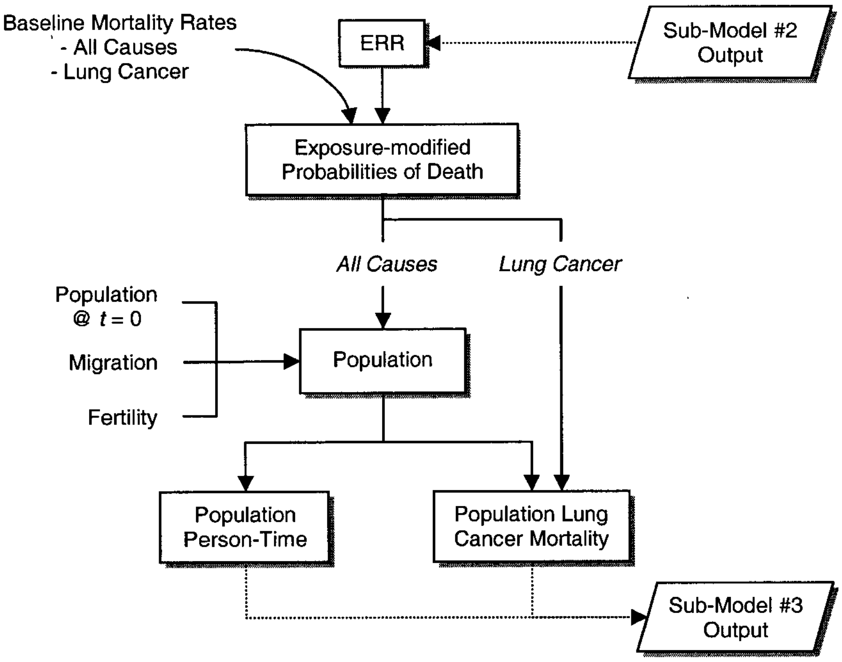
[a] Except for θ2 and θ3 estimates are in log(e) scale.

[b] Except for θ2 and θ3 estimates are in log(e) scale. All values multiplied by 100.

2.4.3 SUB-MODEL 3 – Linking Changes in Risk to Population Health Impacts

In this Sub-Model, which is deterministic rather than probabilistic, all-cause and lung cancer mortality for a radon-exposed individual within age interval x at time t are modelled as a function of their observed mortality, and the excess relative risk (ERR) calculated as above. The age-time mortality profiles are then used to model population health outcomes across the observation period. We follow the multiple decrement and cause-modified [284] (a generalization of cause-elimination) life-table methods used by both BEIR VI [2] and Brand et al. [118]. An overview of Sub-Model 3 is provided in Figure 2-8, below.

FIGURE 2-8. Overview of Sub-Model 3. Life-table methods are used to model changes in all-cause and cause-specific mortality as a function of baseline mortality and excess relative risk (ERR). Population survivorship, and hence population person-time lived, is determined by the all-cause mortality estimates. Lung cancer mortality is calculated as the product of population and cause-specific probability of death. As per the output of Sub-Model 2, rectangular boxes shown in the diagram represent age-time profiles for the variables contained therein. As before, all calculations are repeated to capture outcomes under unmitigated and mitigated scenarios.



2.4.3.1 Estimating Baseline Smoking-Specific Mortality Rates

Age-specific death (D_x) [285] and census counts (N_x) [286] were obtained for the 2001 Canadian population, and are used to estimate average age-specific mortality rates:

$$M_x = \frac{D_x}{N_x \cdot T} \qquad [10]$$

where T is the period over which the deaths occur – 5 years in this analysis. As in standard period life-table analysis, these mortality rates are assumed to remain constant over time. Mortality rates for ever-smokers (ES) and never-smokers (NS) at each age can be estimated by noting that, at each age, the rate for the population as a whole is simply a weighted average of the smoking-specific rates [118, 287, 288], i.e.,

$$M_x = \Pr_x(NS) \cdot M_x^{NS} + \Pr_x(ES) \cdot M_x^{ES} \qquad [11]$$

where $\Pr_x$(NS) and $\Pr_x$(ES) are the prevalence of never- and ever-smokers respectively, shown in Figure 2-9.

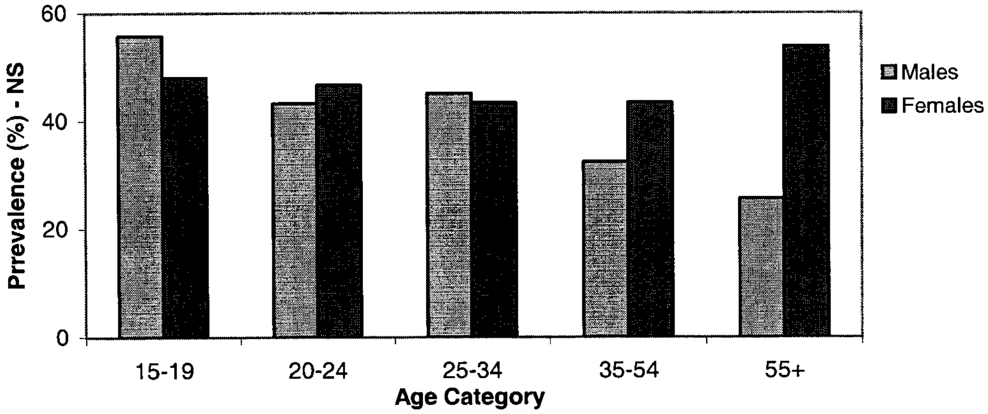
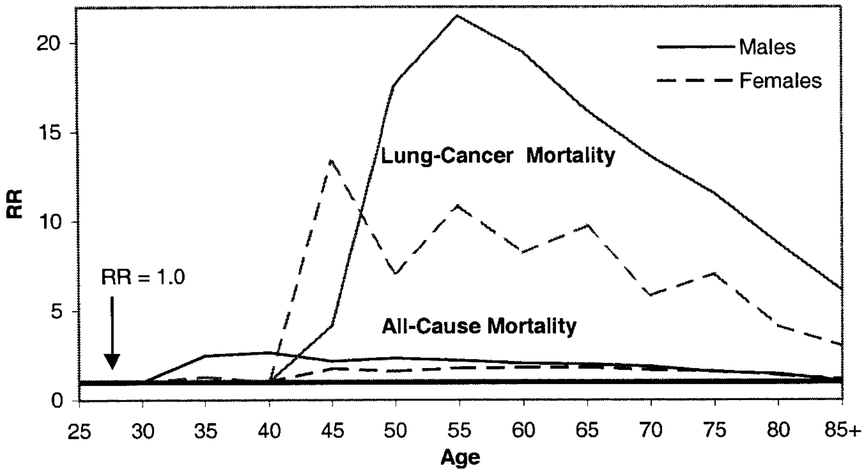


FIGURE 2-9. Prevalence of Never-Smokers, by Age, Canada, 1996 [289]

Note that M_x^{ES} may be expressed as $M_x^{ES} = M_{xn}^{S}$ x RR_x^{ES}, where RR_x^{ES} is the relative risk of death associated with being an ever-smoker (shown in Figure 2-10).

FIGURE 2-10. Relative Risk (RR) of Death from All Causes and Lung Cancer, by Age, Sex and Smoking-Status [290]



Using this relation, equation [10] can be modified to solve for M_x^{NS}, i.e.,

$$M_x^{NS} = \frac{M_x}{\Pr_x(NS) + \Pr_x(ES) \cdot RR_x^{ES}} \tag{12}$$

The estimated baseline 5-year all-cause and cause-specific mortality rates by age, sex and smoking-status (ever vs. never) are shown in Figures 2-11(a) and (b), below.

FIGURE 2-11(a). Estimated Mortality Rates (All Causes), by Age, Sex and Smoking Status

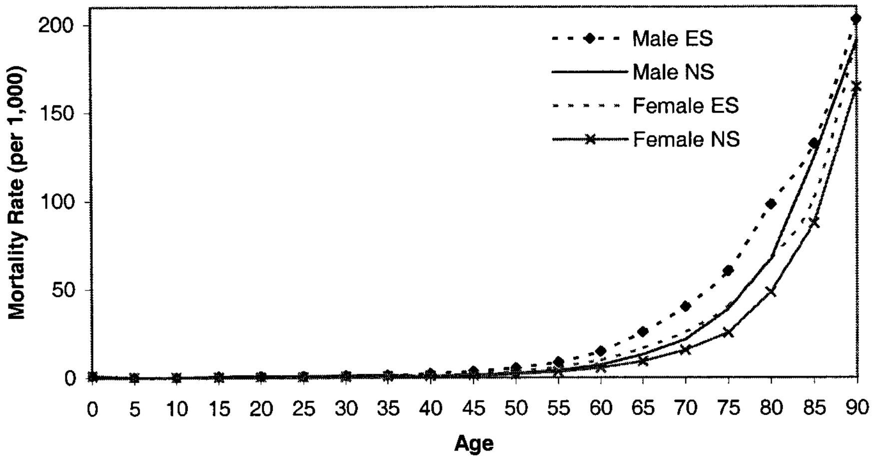
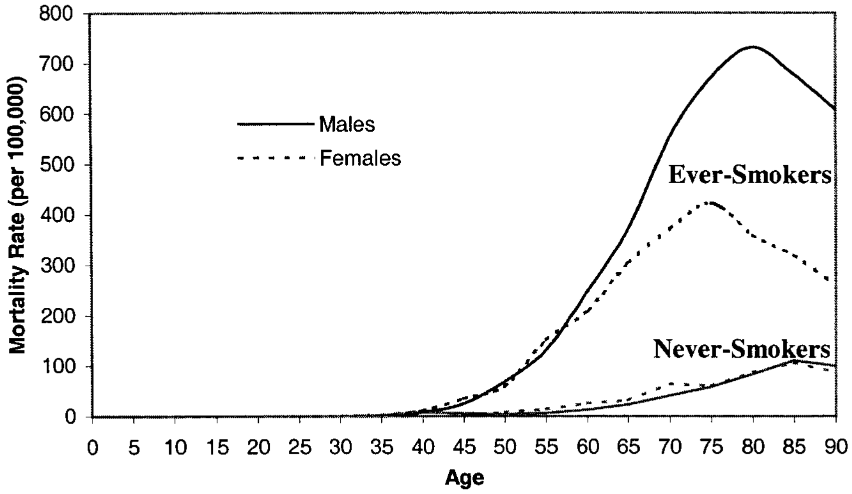


FIGURE 2-11(b). Estimated Mortality Rates (Lung Cancer), by Age, Sex and Smoking Status



2.4.3.2 Calculating All-Cause and Cause-Specific Probabilities of Death

At any given point in time, the population is subject to a 'force of mortality', denoted μ, which is the instantaneous hazard rate of the occurrence of death, defined mathematically as

$$\mu(t) = \lim_{\Delta t \to 0} \frac{1}{\Delta t} \Pr(\text{die by time } t + \Delta t \mid \text{alive at } t). \qquad [13]$$

From survival analysis theory, we know that the probability of surviving a 5-year interval $[t, t+5]$ (conditional upon being alive at t) may be expressed as

$$S_{[t,t+5]} = \exp\left(- \int_t^{t+5} \mu(t)dt \right). \qquad [14]$$

If one assumes the force of mortality to be constant within each age interval $x = 1,2,\ldots, x$, this reduces to $S_x = S_{[t,\, t+5]} = \exp(-5\mu_x)$. In this case, $\mu_x = M_x$, calculated above, and the equation may be re-expressed as $S_x = \exp(-5M_x)$. Note that the probability of dying within the same interval is the complement, i.e., $q_x = 1 - S_x = 1 - \exp(-5M_x)$.

A number of authors have proposed alternatives to the above expressions [291-294]. The current analysis follows the work of Chiang, in which age-specific 5-year probability of death (q_x) is expressed as:

$$q_x = \frac{5M_x}{1+(1-\alpha_x)\cdot 5M_x} \qquad [15]$$

where α_x is the mean fraction of the xth interval lived by those individuals who died during that interval [294]. Following actuarial methods, α_x is assumed to be 0.5 for all age intervals except the first (i.e., comprising ages 0-4) which, based upon an analysis of the 1990 Canadian population, is estimated to be 0.254 [118].

Following Chiang's proportionate hazards assumption, the ratio of cause-specific [295] (superscript L) to all-cause mortality (often termed the 'intensity ratio), denoted $\psi_x = \frac{\mu_x^L}{\mu_x} = \frac{M_x^L}{M_x}$, is assumed to be constant within each age interval [296]. Thus, the age-specific probability of lung cancer death is calculated as $q_x^L = q_x \times \psi_x$.

2.4.3.3 Radon-Modified Probabilities of Death

Sub-Model 2 yields estimates of age-time-specific excess relative risk (ERR) for lung cancer death, denoted here as $e_{x,t}$, for both ever- and never-smokers. As described by Brand et al. [118], lung cancer mortality under exposed conditions (denoted by the subscript E) may be expressed as ${}_E\mu_{x,t}^L = \mu_x^L(1+e_{x,t})$, where μ_x^L represents baseline cause-specific mortality. From the previous section, we know that $\mu_x^L = \psi_x \mu_x$, allowing for re-expression of the above as ${}_E\mu_{x,t}^L = \mu_x \psi_x (1+e_{x,t})$. Note that all-cause mortality is also modified by exposure, since an increase in lung cancer adds to overall mortality. Thus, ${}_E\mu_{x,t} = \mu_x(1+\psi_x e_{x,t})$.

Substituting these expressions for modified mortality into the survival analysis calculations above, we see that the probability of surviving a 5-year interval when subjected to radon exposure is $S_x = \exp(-5\mu_x(1+e_{x,t}))$. The exposure-modified version of Chiang's expression for probability of death is therefore [284]

$$ {}_E q_{x,t} = 1 - \left(1 - \frac{5M_x}{1+(1-\alpha_x)\cdot 5M_x}\right)^{(1+\psi_x e_{x,t})} . \qquad [16] $$

The modified intensity ratio is [284]

$$ {}_E \psi'_{x,t} = \frac{{}_E \mu^L_{x,t}}{{}_E \mu_{x,t}} = \frac{\psi_x(1+e_{x,t})}{1+\psi_x e_{x,t}} , \qquad [17] $$

while the radon-modified probability of lung cancer death is the product ${}_E q_{x,t} \times {}_E \psi_{x,t}$.

Repeating these calculations for age intervals x = 1,2,.., x at times t = 0,5,..., t results in an age-time profile describing how age-specific probability of death from both lung cancer and all causes will change over the planning horizon given the mitigation scenario under consideration (including no mitigation). Using the baseline mortality rates displayed in Figures 2-11(a) and (b), one such profile is generated for each of 4 groups, defined by their gender and smoking-status (ever vs. never).

2.4.3.4 Modelling Population Survival

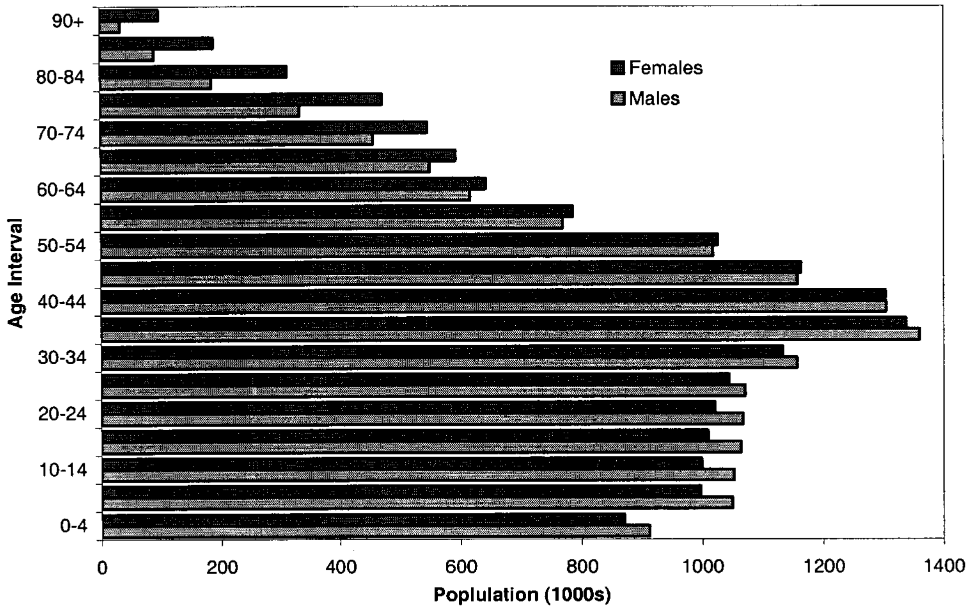
Initially, the population is assumed to have the age-sex-composition detailed by the 2001 Census, shown in Figure 2-12, below. As time progresses, the population within each age-sex group will survive (i.e., advance into older age groups) at different rates, changing this distribution. Tracking this progression is relatively straightforward. The calculations for males are shown below. Females – treated as a separate cohort – are modelled in an identical fashion.

Let $N^m_{x,t}$ be the number of males within the xth 5-year age interval at time t, the sum of male ever smokers (ES) and never smokers (NS), i.e.,

$$N_{x,t}^{m} = N_{x,t}^{m,ES} + N_{x,t}^{m,NS} \qquad [18]$$
$$= N_{x,t}^{m}(1 - \Pr(NS \mid m)) + N_{x,t}^{m} \Pr(NS \mid m)),$$

where $\Pr(NS \mid m)$ is the age-gender-specific prevalence of never-smoking, shown in Figure 2-9. Note that this prevalence is assumed to remain constant throughout the planning horizon.

FIGURE 2-12. Age-Sex Composition of the Canadian Population, 2001



It follows that $N_{x+1,t+5}^{m}$ – the number of these same people who will be alive in interval x+1 at time t+5 – is:

$$N_{x+1,t+5}^{m} = N_{x,t}^{m,ES} \cdot S_{x,t}^{m,ES} + N_{x,t}^{m,NS} \cdot S_{x,t}^{m,NS} \qquad [19]$$
$$= N_{x,t}^{m,ES} \cdot (1 - q_{x,t}^{m,ES}) + N_{x,t}^{m,NS} \cdot (1 - q_{x,t}^{m,NS}),$$

where $S_{x,t}^{m}$ and $q_{x,t}^{m}$ are the 5-year probabilities of survival and death, respectively, for males in age interval x at time t, calculated separately for ever-smokers (*ES*) and never-smokers (*NS*). (Note as the entire Canadian population is exposed to radon at some level, the exposure-modified probability of death ${}_{E}q_{x,t}$ will herein be denoted simply as $q_{x,t}$). These probabilities are assumed to be dependent solely on age and on the excess risk from radon exposure.

It is important to note that $S_{x,t}$ is not truly the 5-year survival probability for any individual within the xth age interval. Rather, it is probability that an individual will survive the xth 5-year age interval, given that they have reached the beginning of it. Thus, the population is modelled as if the age intervals were aligned with the 5-year period divisions of the planning horizon – i.e., it assumes that all individuals within age interval x are at the start of that interval when time period t begins.

The above formula holds true for all age intervals, save the last one. The final interval, k, comprises all individuals above the age of 90 years. It has no upper limit, which is reflected in the calculations as follows (shown only for ever-smokers, for simplicity):

$$N_{k,t+5}^{m,ES} = N_{k-1,t}^{m,ES} \cdot S_{k-1,t}^{m,ES} + N_{k,t}^{m,ES} \cdot S_{k,t}^{m,ES}. \qquad [20]$$

Thus, the current population in the final interval is equal to the sum of survivors from that interval, as well as those from the interval preceding it.

2.4.3.5 Modelling Population Growth

If defined solely by the calculations described above, our modelled population can only decrease in size as members die. This is obviously not the case in reality, as the size of a true population in year $t+\Delta t$ can be described by the function

$$N_{t+\Delta t} = N_t + \Delta N_{[t,t+\Delta t]}, \qquad [21]$$

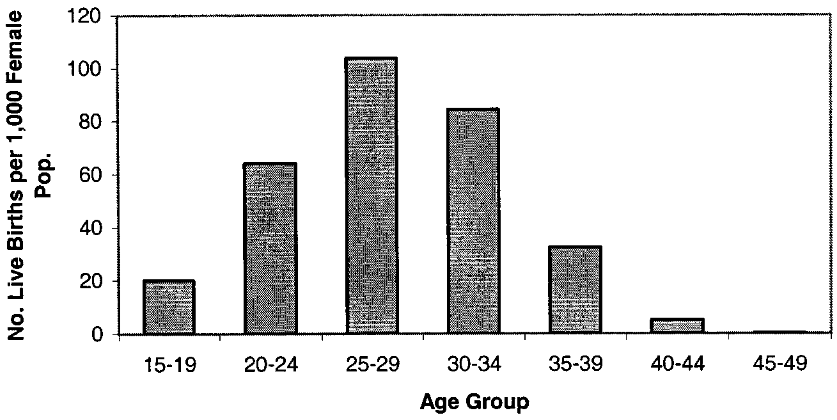
where N_t is the starting population, and $\Delta N_{[t,t+\Delta t]}$ represents net population growth in the interval $[t+\Delta t]$, or the sum of births and immigration, less deaths and emigration. Attrition due to death was described in the previous section. The three remaining components of population growth are considered here.

The population within the first age interval – comprising ages 0-4 – is primarily dependent upon the number of births that occurred in the previous 5 years. Births, in turn, are calculated as a function of the number of females (N^f) within the population, as follows:

$$\begin{aligned} N_{1,t+5} &= B^m_{[t,t+5]} + B^f_{[t,t+5]} \\ &= 5\sum_{x=1}^{k}\left(N^f_{x,t}\cdot F_x\right)\cdot \Pr(\mathrm{B}^m|B) + 5\sum_{x=1}^{k}\left(N^f_{x,t}\cdot F_x\right)\cdot(1-\Pr(\mathrm{B}^m|B)), \end{aligned} \qquad [22]$$

where $B^m_{[t,t+5]}$ and $B^f_{[t,t+5]}$ represent the number of male and female births within time interval [*t*,*t*+5], respectively, $N^f_{x,t}$ is the number of females in age interval x at time *t*, F_x is the age-specific annual fertility rate (i.e., the average number of children born per year to females of age x), and $\Pr(\mathrm{B}^m|B)$ is the probability the newborn is male. Age-specific fertility rates, for which the estimates shown in Figure 2-13 (below) were obtained for 1997 Canadian female population [297], are assumed to be constant over time, and to depend solely on age. Women are assumed to be fertile from age 15-49 – fertility rates for all other ages are set to 0.

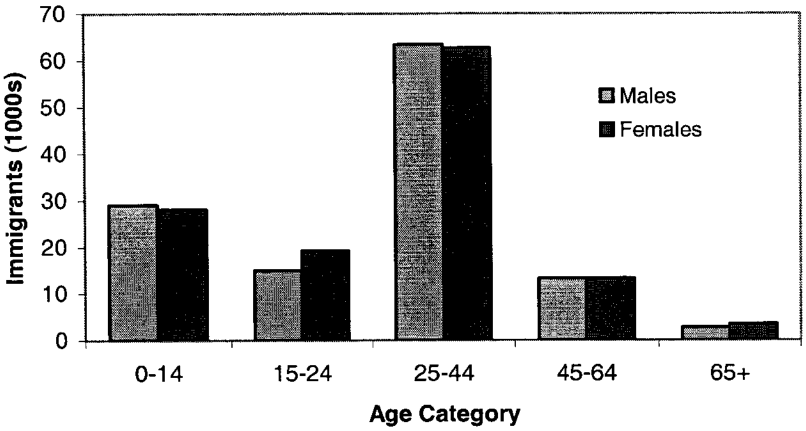
FIGURE 2-13. Annual Fertility Rate, by Age, Canada, 1997 [298]



The probability that a given child will be male, $\Pr(\mathrm{B}^m|B)$, is estimated from births in the year 2001 to be 0.512 [299]. This gives a sex ratio at birth of 1.05 – a figure that appears consistent with the population distribution for younger ages evident in Figure 2-12. This probability is also assumed to be constant over time.

In 2001, 250,352 immigrants arrived in Canada, having an age-sex distribution as shown in Figure 2-14.

FIGURE 2-14. Immigration by Age and Sex, Canada, 2001 [300].



We assume that this number of immigrants will continue to be added to the Canadian population each year, with an identical age and sex distribution as in 2001. The assumption of constant immigration levels avoids the need to make uncertain projections about future immigration rates. Note that the estimates shown are sub-divided by age intervals that are wider than 5-years. Lacking further information, we assume the immigrant population to be uniformly distributed within those broader age intervals. Thus, of the 29,082 male immigrants who were 0-14 in 2001, we estimate that an equal number (i.e., one third – 9,694) fell within each of the 0-4, 5-9. 10-14 age intervals. It was arbitrarily assumed that no immigrants exceeded the age of 75, and that all immigrants survive until the end of the 5-year period in which they arrive. Further, the age-gender-specific prevalence of never-smokers within the immigrant population is assumed to be the same as the rest of the Canadian population. Finally, all immigrants are assumed to have incurred a cumulative radon exposure equivalent to that which they would have received over their lifetime had they lived in Canada.

In 2001, 37,640 individuals emigrated from Canada permanently [301]. This represents approximately 0.2% of the Canadian population not residing in collective dwellings (Note: as with immigrants, it is assumed that no emigrants will come from collective dwellings such as health care facilities or penitentiaries). As neither an age nor gender distribution of emigrants were available, future emigration from Canada is modelled by applying this rate to all age groups under 75 years, including newborns. The rate of emigration is assumed to be constant over time.

Thus, equation [19] may be extended to provide the estimated male population within interval x+1 at time t+5 within an open population model, given the above assumptions, as

$$\begin{aligned} N_{x+1,t+5}^{m} &= \left(N_{x,t}^{m,ES} \cdot S_{x,t}^{m,ES} + N_{x,t}^{m,NS} \cdot S_{x,t}^{m,NS}\right) + I_{x}^{m} - E_{x}^{m} \\ &= \left(N_{x,t}^{m,ES} \cdot S_{x,t}^{m,ES} + N_{x,t}^{m,NS} \cdot S_{x,t}^{m,NS}\right) + I_{x}^{m} - 0.2 \cdot N_{x,t}^{m} \end{aligned} \qquad [23]$$

for age intervals 5-9 through 70-74, where I_x^m is the number of males immigrants within that age interval annually, while E_x^m is the number of male emigrants, calculated as a constant proportion of the population at time t. Again, the same calculations are performed for females.

2.5 ANALYSIS

The analytical framework presented in Figure 2-1 provides the basis for determining which model outputs are useful in an economic evaluation of the radon control strategies under study.

2.5.1 Net Total Cost

2.5.1.1 Total Gross Costs Incurred

Section 2.4.1.2 detailed the decision model's consideration of the activities that would occur under each mitigation strategy. As per the analytical framework detailed in Figure 2.1, these activities will come at some cost to administrators of the radon program, to producers, and to consumers. Note that in the case of radon control within dwellings, the majority of costs are borne by the consumer (i.e., the homeowner), with no anticipated costs to producers.

The volume of a particular activity (e.g., number of ASD systems installed) occurring in year t is calculated by tracking the proportion of simulated dwellings undergoing the activity in the relevant year, and multiplying by the total number of dwellings the strategy is anticipated to effect. Total cost for that activity in year t is simply the annual volume multiplied by the unit cost for the activity. Annual costs are then discounted to their present-day value, and summed to estimate the total cost for the activity over the planning horizon. Total cost of the mitigation program as a whole is equal to the sum of the discounted costs for each activity, less the direct cost savings received by reducing the number of lung cancers with which the population is burdened.

Unit costs for the activities are estimated as follows.

Screening Costs

Initial screening tests are assumed be short-term tests using charcoal canisters, costing $36.93[†] (1997 estimate [6]: $35). Confirmatory and follow-up tests are assumed to be long-term integrated alpha-track monitoring, at an estimated cost of $131.04 [†] (1997 estimate 6: $125) per unit.

Mitigation Costs

All remediation is assumed to be achieved using active sub-slab depressurization (ASD). 1997 cost estimates for retrofitting an existing Canadian dwelling with ASD ranged from $800-3000 [6]. The estimate used here is $1900 (1997), which in year 2000 dollars is $2,048.20 [†]. This is approximately equal to the estimated average cost of ASD in the U.S.

Mitigation of new dwellings at the time of construction is less expensive, as new homes can be built in such a way as to facilitate the implementation of the ASD system, if required. 1992 estimates of this cost were $1,200 [243], which in year 2000 dollars is $1,312.80 [‡].

[†] Inflated by Consumer Price Index (CPI) for *Home Maintenance and Repair* (1.078) [302]

[‡] Inflated by Consumer Price Index (CPI) for *Home Maintenance and Repair* (1.094) [302]

Upkeep Costs

The installation of an ASD system will entail additional costs related to maintenance and electricity requirements. These were estimated to be $120 per year in 1992 [243], or $131.04 [§] in year 2000 dollars.

Program Administration Costs

The majority of work carried out during any national program to control radon exposure will be performed by private, radon contractors hired by the individual homeowner. There will also be a requirement for some co-ordinating / promotion work, presumably by the federal and/or provincial government. It was arbitrarily assumed that all strategies would require the same program administration effort: 10 full time equivalents (FTE) per year [303] split in some manner between the federal and provincial level. Average annual costs per FTE were estimated to be $81,000, comprised of a base salary of $60,000, 20% benefits ($12,000), and 13.5% accommodation ($8,100), which are standard estimates for staff-related costs within public programs of this nature [304]. Thus, total program administration costs were estimated to be $810,000 per year. All other costs that may arise over the course of program administration were excluded from this analysis.

The above assumptions likely introduce some error into our analysis. For example, administration costs are very likely to vary, depending on the type of radon program that is implemented. As well, the restriction of administration costs to only those related to staffing will likely improve the apparent cost-effectiveness of all strategies relative to what would be seen in reality, should other types of costs be incurred. However, given the relatively small proportion of total costs that program administration costs represent (see Figure 3-8 in the results), the error introduced by both these factors is likely to be small.

[§] Inflated by Consumer Price Index (CPI) for *Electricity* – i.e. the dominant component of upkeep costs (1.092) [302]

2.5.1.2 Treatment Costs Averted

A reduction in the number of lung cancers in the affected population (as approximated by the reduction in lung cancer mortality, calculated below in Section 2.4.5.2) would yield cost savings to the health care system in the form of reduced treatment costs. This economic benefit must be considered in the analysis.

Evans et al. [305] estimated the average 5-year costs of lung cancer treatment in Canada to be $21,000 (1995 dollars). Further, they determined that approximately 82% of that cost was incurred within the first year of treatment. To estimate cost savings from reduced lung cancer, this 5-year cost was first discounted into an equivalent 1-year cost by assuming that the remaining 18% of cost was incurred uniformly over the final 4 years of treatment. Discounting at 5% yields an average equivalent 1-year cost in CAN$(2000) of $22,869 [††].

As with program activities, treatment costs averted (i.e., lung cancers prevented X $22,869) are estimated for each year, discounted and summed to yield total equivalent present-day costs. These are then subtracted from total program activities costs to estimate the total net cost of the strategy under consideration.

Inclusion of health care cost savings from reduced lung cancer incidence leads to a parallel justification for inclusion of the costs associated with subsequent health events now arising within those individuals in whom the lung cancer was prevented. This is not taken into account in our analysis, due to the significant increase in model complexity required to incorporate such alternative health endpoints. Note, however, that as a result of the prolonged induction period needed for the health effects of radon exposure reduction to be expressed (particularly when that reduction is implemented gradually over time, as in many of our mitigation strategies), a large portion of these subsequent health events would occur after the end of the 80-year planning horizon, and wouldn't be captured even if they were modeled.

[††] Inflated by Consumer Price Index (CPI) (1.089) [302]

2.5.2 Population Health Benefits

2.5.2.1 Lung Cancer Deaths Prevented

A reduction in radon exposure levels within the general population is expected to result in a decrease in radon-related lung cancer incidence, and hence mortality. We estimate lung cancer deaths only, as the BEIR VI risk models describe lung cancer mortality, not incidence. Total lung cancers prevented (ΔD^L) are estimated separately for male and female ever- and never-smokers, discounted to the present value, and summed to calculate the effect for the total population. The required calculation for one group (e.g., male ever-smokers) is

$$\begin{aligned}\Delta D^L{}_{[0,80]} &= \sum_{t=0}^{80} \Delta D_t^L \cdot (1+r)^{-(2.5+5(t-1))} \\ &= \sum_{t=0}^{80} ({}_B D_t^L - {}_M D_t^L)(1+r)^{-(2.5+5(t-1))} \\ &= \sum_{t=0}^{80} \left(\sum_{x=1}^{k} {}_B N_{x,t} \cdot {}_B q_{x,t}^L - \sum_{x=1}^{k} {}_M N_{x,t} \cdot {}_M q_{x,t}^L \right) (1+r)^{-(2.5+5(t-1))}, \end{aligned} \qquad [24]$$

for 5-year age intervals x=1,2,...x, and 5-year time periods t=0,5,...80, where 2.5+5(t-1) defines the period's midpoint (in years since t=0). The quantities ${}_B D_t^L$ and ${}_M D_t^L$ (the number of lung cancers occurring within the population at time t), ${}_B N_{x,t}$ and ${}_M N_{x,t}$ (the population within age interval x at time t), and ${}_B q_{x,t}^L$ and ${}_S q_{x,t}^L$ (the age-specific, radon-modified 5-year probability of lung cancer death) are calculated under baseline (subscript B) and mitigated (subscript M) exposure conditions, respectively. The discounting factor $(1+r)^{-t}$ is described in Section 2.2.2.1.

2.5.2.2 Population Life Years Gained

The decrease in the radon-related burden of lung cancer mortality achieved through mitigation efforts can also be gauged by calculating the number of life-years gained by the population. Population life-years, or person-time, is the sum of the number of years lived by each individual within the planning horizon. The number of discounted life-years gained over the planning horizon ($\Delta LY_{[0,80]}$) is calculated in a way similar to lung cancers prevented, i.e.,

$$\begin{aligned}\Delta LY_{[0,80]} &= \sum_{t=1}^{80} \Delta LY_t \cdot (1+r)^{-(2.5+5(t-1))} \\ &= \sum_{t=1}^{80} ({}_B LY_t - {}_M LY_t)(1+r)^{-(2.5+5(t-1))} \\ &= \sum_{t=1}^{80} \left(\sum_{x=1}^{k} {}_B LY_{x,t} - \sum_{x=1}^{k} {}_M LY_{x,t} \right) (1+r)^{-(2.5+5(t-1))} \end{aligned} \quad [25]$$

for each gender-smoking group. Within a closed population, the number of life years lived by individuals in age interval x during time period $[t, t{+}5]$ would be calculated as

$$LY_{x,[t,t+5]} = 5 \cdot N_{x,t}(1-\alpha_x \cdot q_{x,t}) \quad [26]$$

where $N_{x,t}$ is the population at the start of age interval x at time t, $q_{x,t}$ is the radon-modified age-specific 5-year probability of death at time t, and α_x is the average proportion of the period $[t, t{+}5]$ lived by those who die within it. Note that the life-year estimates calculated at time *t* capture person-time lived within the previous 5-years.

However, the population modelled here is not closed. International migrants contribute to the life-years lived by the population. In all cases, it is assumed that immigration and emigration occur at a uniform rate over the time period. Thus, an average immigrant or emigrant will live in Canada for half of the time interval in which they arrive. Migration may therefore be incorporated into Equation [26] as follows:

$$LY_{x,[t,t+5]} = 5 \cdot N_{x,t}(1-\alpha_x \cdot q_{x,t}) + 5(0.5)I_x - 5(0.5)E_x \quad [27]$$

Recall that the all members of the immigrant population are assumed to survive until the end of the interval during which they entered Canada.

In our analysis, all calculations are performed at the start of each 5-year time period, at which time the above equation is used to estimate population life-years lived in the previous period. Given the assumption described previously regarding the "alignment" of 5-year age intervals with time periods, individuals within the first age interval (comprising ages 0-4) do not contribute any person time the population life-year estimates (as they were not born within the period to which the life-year estimates apply).

2.5.2.3 Quality-Adjusted Life Years (QALYs) Gained

Recently, economic and other types of analyses have attempted to move beyond simply measuring the number of life-years lived, giving consideration the *value* of each particular life year. Value may be assigned to life-years in a number of ways: in dollars (based on expressed or revealed willingness to pay), or in *utilities*, as is done here. The *Health Utility index* (HUI) provides a utility weight based upon an individual's functional capacity, defined by 8 attributes: vision, hearing, speech, mobility, dexterity, cognition, emotion, and pain and discomfort [306]. The resulting index provides a single numerical value between 0 and 1, with 1 representing full health and 0 representing death.

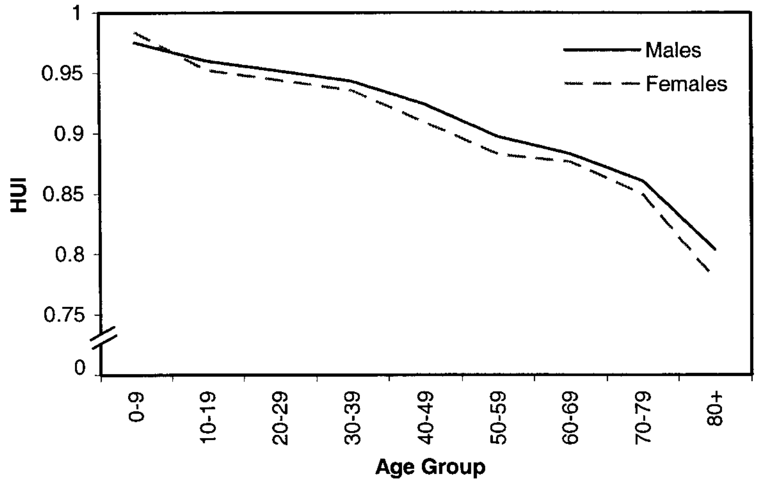
Quality-adjusted life-years (QALYs) are simply the product of the number of life-years lived at each age and the utility by which a year of life at that age is weighted, i.e.,

$$QALYs_x = LY_x \times U_x . \qquad [28]$$

In this thesis, QALYs are calculated as above using the average age-sex-specific HUI scores for the Canadian population, estimated from the 1996-97 National Population Health Survey (NPHS). The HUI scores are shown in Figure 2-15, below.

These utility scores are assumed to remain constant throughout the planning horizon. Note that it was not possible to incorporate lung-cancer specific utilities into the analysis, as this would require data on both lung cancer incidence and mortality; the BEIR VI risk model used in this analysis is based on lung cancer mortality, not incidence.

FIGURE 2-15. Average HUI Score, Canada, 1997, by Age and Gender



2.5.3 Incremental Cost-Effectiveness

The strategies will also be compared on the basis of their incremental cost-effectiveness, calculated as

$$CE_{S_1}^{S_2} = \frac{\Delta C}{\Delta E} = \frac{C_r^{S_2} - C_r^{S_1}}{E_r^{S_2} - E_r^{S_1}}, \qquad [29]$$

where $CE_{S_1}^{S_2}$ is the incremental cost-effectiveness of moving from strategy S_1 to strategy S_2, where S_2 may be no intervention, $C_R^{S_1}$ and $C_R^{S_2}$ are the cost estimate for strategies S_1 and S_2, respectively, and $E_R^{S_1}$ and $E_R^{S_2}$ are estimated effects of strategies S_1 and S_2. It is important to note that this is the ratio of the mean incremental costs and incremental benefits; not the mean ratio [307, 308].

In studies such as this which multiple interventions strategies are being compared, cost-effectiveness ratios are often calculated based upon the cost and effect estimated for each strategy, relative to no intervention. This produces what are termed mean cost-effectiveness ratios, which are often quite informative. However, for decision-making purposes, the most appropriate use of cost-effectiveness analysis is to estimate the cost per additional health unit (e.g., QALY) gained when moving from the most cost-effective strategy to the second most cost-effective, and then from the second to the third, and so on [309].

Strategies will be compared on the basis of their incremental cost per lung cancer prevented, as well as their cost per life year and QALY gained.

2.5.4 Sensitivity Analysis

Sensitivity analysis is performed to consider how the health benefits, total costs and cost-effectiveness of each strategy might change with changes in model parameters, including:

- the action level (i.e., 800, 400 and 200 Bq/m^3) at which mitigation is recommended;
- the rate of homeowner compliance with the radon guideline (0%, 50%, 100%);
- the percent reduction of radon concentrations achieved in mitigated dwellings (70%, 90%);
- the annual discount rate applied to the cost and health benefits of mitigation (0%, 5%, 10%); and
- the distribution of baseline radon concentrations, examining the impact of targeting intervention towards geographic regions with higher average radon concentrations. Specifically, the concentration distributions observed for the housing stock in Sudbury and Winnipeg are used in this analysis. Winnipeg was chosen for this analysis because it has the highest average radon levels within Canada. Sudbury, which has a mean concentration between that observed for Winnipeg and Canada, was selected rather than a city (e.g., Vancouver) with radon levels below the national average. This choice was made because the computer memory required to obtain numerically-stable estimates for Vancouver was prohibitive (e.g., a single dwelling with radon concentrations in excess of 800 Bqm^{-3} was modelled after 25,000 Monte Carlo trials).

CHAPTER 3
RESULTS

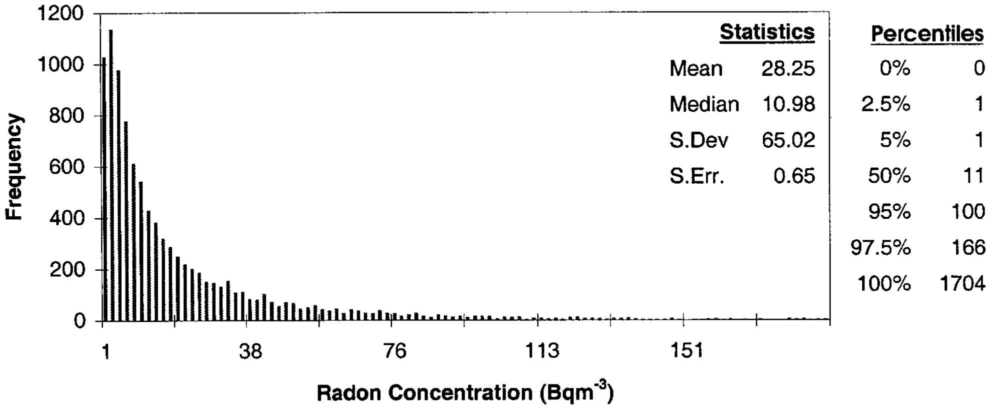
3.1 MODEL VALIDATION

This section describes the results of significant efforts made to ensure the validity of outputs of each of the decision-model subcomponents.

3.1.1 Sub-Model 1 Validation

Sub-Model 1 modelled the change in average annual radon concentrations within Canada that would result from implementation of a national residential radon mitigation program. The current concentration distribution for indoor radon in Canada was modelled as a log-normal distribution with parameters as defined by survey data reported by MacGregor et al.[31]: geometric mean (GM) 11.2 Bqm^{-3} and geometric standard deviation (GSD) 3.9. The corresponding arithmetic mean was (AM) of 28.35 Bqm^{-3}. Following 10,000 Monte Carlo trials, the distribution of radon levels output by the decision model was very consistent with these values, as shown in Figure 3-1.

FIGURE 3-1. Indoor Radon Concentration Distribution, Canada, Baseline



Statistics	
Mean	28.25
Median	10.98
S.Dev	65.02
S.Err.	0.65

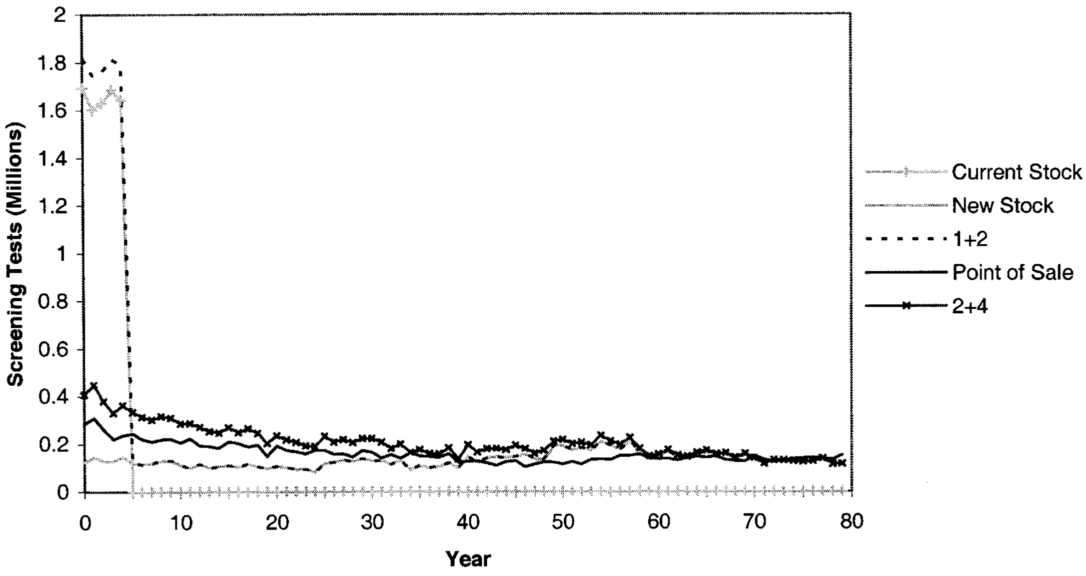
Percentiles	
0%	0
2.5%	1
5%	1
50%	11
95%	100
97.5%	166
100%	1704

Note: Displayed range is 0–187 Bqm^{-3}. Actual range is 0 –1794 Bqm^{-3}.

Model outputs for the distribution of radon mitigation-related activities over time (shown in Figure 3.2, below) are exactly as would be expected, given the mitigation strategy under consideration. Strategy 1 (screen all 8.3 million current dwellings over 5 years) involves the screening of about 1.65 million dwellings per year for 5 years, with no screening tests performed after that time.

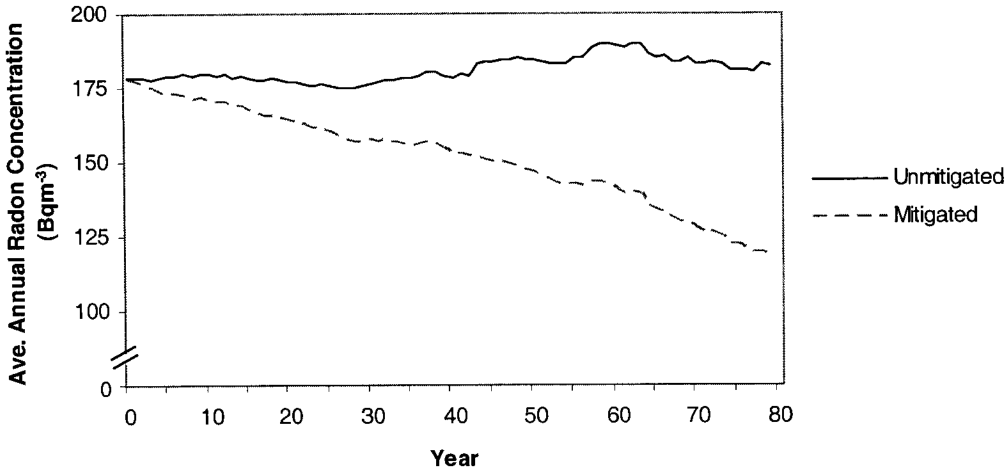
Conversely, strategies 2 (screen all newly constructed dwellings), 4 (screen all homes when they are sold) and 5 (a combination of 2 and 4) display relatively constant rates of screening over the 80 year observation period, as expected. Further, strategy 3 (screen all current and new dwellings) initially follows a pattern very similar to those of strategy 1 (with slightly more tests performed, since it captures new dwellings as well, and then involves screening at a rate identical to strategy 2.

FIGURE 3-2. Number of Screening Tests Performed, by Mitigation Strategy and Year.



Implementation of a population-level radon mitigation strategy is expected to lower radon concentrations with Canadian homes. This effect is shown in Figure 3-3, documenting a modelled decrease in the average radon concentration within Canadian dwellings under Strategy 2 – screening all newly-constructed dwellings and mitigating as required. As expected under this strategy, the decrease in average radon concentration becomes more pronounced as time progresses and a greater proportion of the housing stock is affected by the radon mitigation program.

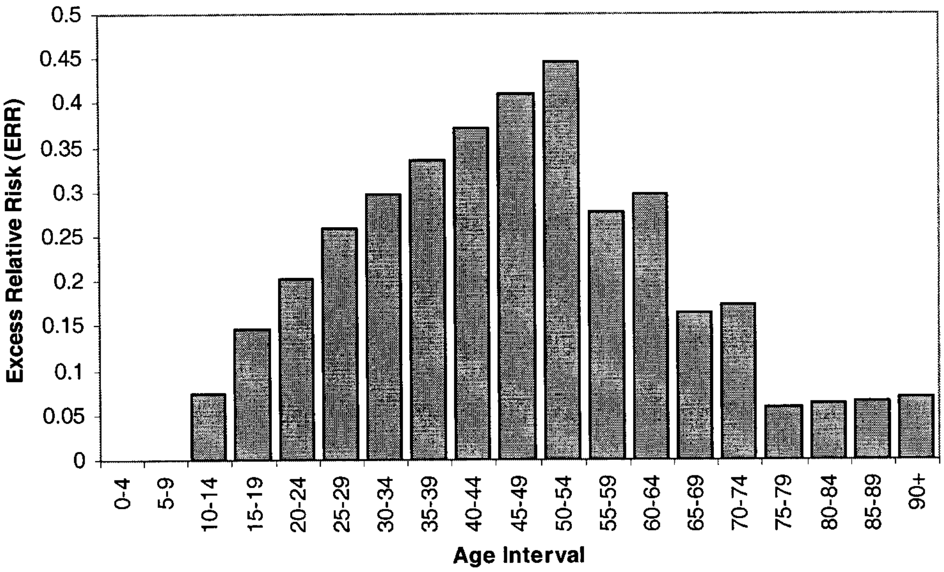
FIGURE 3-3. Modelled Average Annual Radon Concentration, Winnipeg, by Year, Unmitigated vs. Mitigation (Strategy 2, 200 Bqm-3)



3.1.2 Sub-Model 2 Validation

Sub-Model 2 modelled the time-dependant change in age-specific, smoking specific excess relative risk (ERR) that would result from a decreasing average annual radon concentration within Canadian dwellings. Assuming a constant rate of radon exposure, Sub-Model 2 estimates a profile of age-specific ERR similar to that shown below in Figure 3-3.

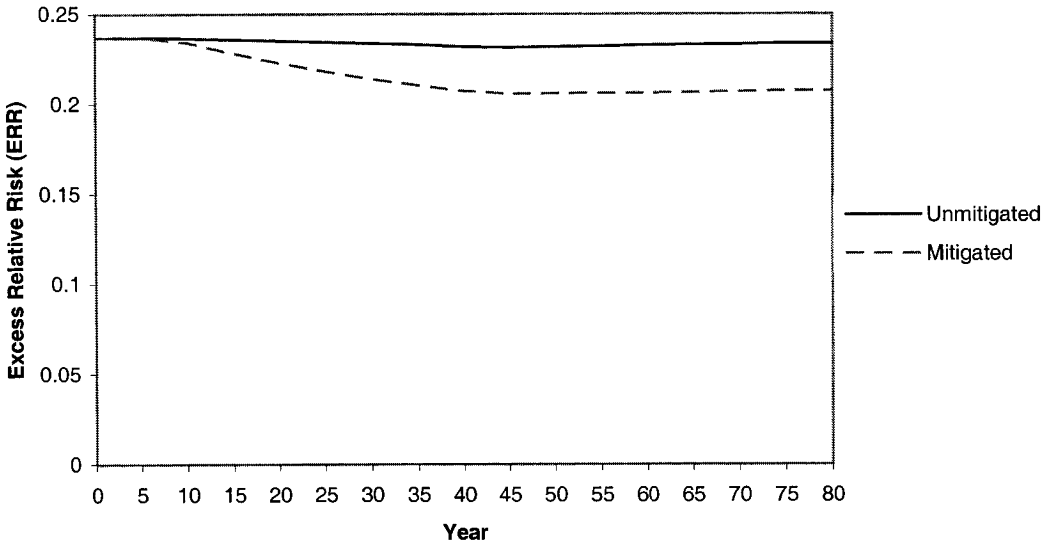
FIGURE 3-4. Excess Relative Risk (ERR) for Lung Cancer Mortality, Baseline Exposure, by Age.



As described by Brand et al. [118], this ERR profile is a characteristic output of the BEIR VI risk model, and therefore provides evidence in support of the validity of Sub-Model 2. Further, a comparison of Sub-Model 2 with the equivalent component of the model used by Brand et al. [118] showed identical output values of ERR when using the same inputs.

With mitigation, age-specific ERR should decrease with decreasing average radon concentrations over time. Figure 3-5 shows an example of this decrease, as modelled for ever-smokers within the 40-44 year age group following implementation of strategy 3.

FIGURE 3-5. Impact of Radon Mitigation on Excess Relative Risk (ERR) for Lung Cancer Mortality, Ages 40-44, by Year (Stategy 3; Action Level: 200 Bqm^{-3}).



By way of confirming that the trend shown in Figure 3-5 is as expected, note that Strategy 3 (screen all current and new dwellings) is expected to quickly decrease radon levels within the population (over 5 years) and then keep them relatively stable at some lower average concentration. Accordingly, as shown in the figure, the age-specific ERR of the 40-44 year age group gradually decreases (following the assumed 5 year latency period – see Section 2.4.2.1) as, over time, individuals within that group have lived a progressively larger proportion of their lifetime at the lower exposure rates. As of year 45, and in all subsequent years, members of this group will have lived their entire lives at the lower, relatively constant level of radon exposure, and their age-specific ERR becomes relatively stable once again.

3.1.3 Sub-Model 3 Validation

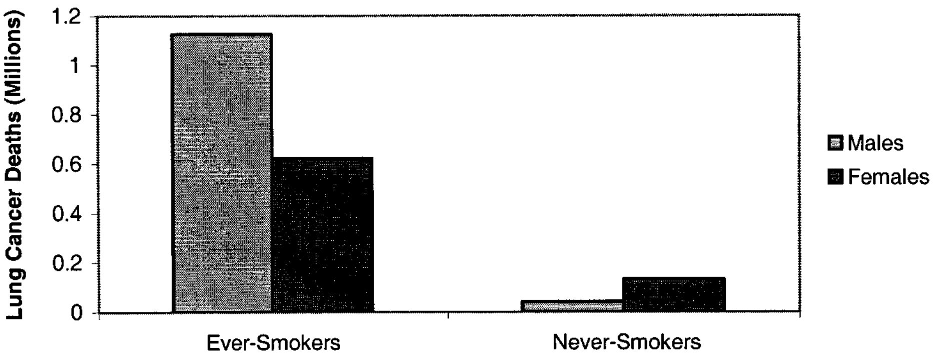
In Sub-Model 3, we model the impact of the decrease in excess relative risk (ERR) for lung cancer that results from radon mitigation upon lung cancer mortality within the population over 80 years.

3.1.3.2 Lung Cancer Mortality

As with the validation of our calculations of age-specific ERR (Sub-Model 2), we had the advantage of being able to benchmark our calculations of all-cause and cause-specific (i.e. lung cancer) mortality against those of Brand et al. [118]. As before, given identical inputs in terms of age-specific ERR, the two models produce identical values for age-specific probabilities for all-cause and lung cancer mortality. (Note that methodological differences between our model and that used by Brand et al. preclude any benchmarking of results beyond the calculation of age-specific ERR and probabilities of death based upon a constant radon exposure rate).

Under baseline exposure conditions, the decision-model simulates approximately 1.92 million lung cancer deaths over the 80 year study period. Of these, approximately 91% occur in ever-smokers, and 61% occur in males (Figure 3-6).

FIGURE 3-6. Total Lung Cancer Mortality, Baseline Exposure, 80 Years, by Sex and Smoking-Status



The modelled lung cancer mortality is consistent with current rates (i.e. about 16,000 lung cancer deaths in 2001), taking into account an increase in lung cancer mortality that will result from a progressively larger proportion of the population occupying older age strata. The increased incidence of lung cancer mortality the male and ever-smoking populations is as expected, given the higher risks of lung cancer associated with those two groups (note that the increased risk is males is due in large part to an increased prevalence of ever-smoking).

For implementation of the most stringent mitigation option considered here – screen all current and new dwellings (strategy 3); action level: 200 Bqm^{-3}; 100% compliance; and 90% reduction of radon levels – we estimate a total reduction in lung cancer mortality by 27,312 deaths (undiscounted) over 80 years (see Table A-29 in the Appendix). Assuming that the proportion of lung cancer cases attributable to radon exposure would remain constant at 8% under baseline conditions [118], 27,312 deaths prevented represents about 18% of the 153,000 lung cancer deaths (i.e. 1.92 million X 8%) that would have occurred due to residential radon. This mitigation effect is larger than that estimated by Brand et al., who show a 17% decrease in radon-attributable lung cancer mortality when all radon concentrations are reduced to below 123 Bqm^{-3}, but is still reasonably consistent. It is also quite consistent with expectations which follow from the BEIR VI report, which concluded that reducing all indoor radon concentrations in excess of the U.S. action level of 148 Bqm^{-3} would decrease radon-attributable lung cancer mortality within that country by about 30%. Our number is consistent with that estimate, given that (1) the action level we simulate (200 Bqm^{-3}) is 52 Bqm^{-3-} higher than that used in the BEIR VI model – a range of concentration that is likely responsible for a significant burden of radon-induced lung cancer mortality; and (2) average radon concentrations are generally higher within the U.S., which would lead to a larger impact from mitigation.

3.2 RESULTS – REFERENT CASE

This section presents the results for the modelled implementation of the five national radon mitigation strategies described in Section 2.3, assuming 50% homeowner compliance and 90%

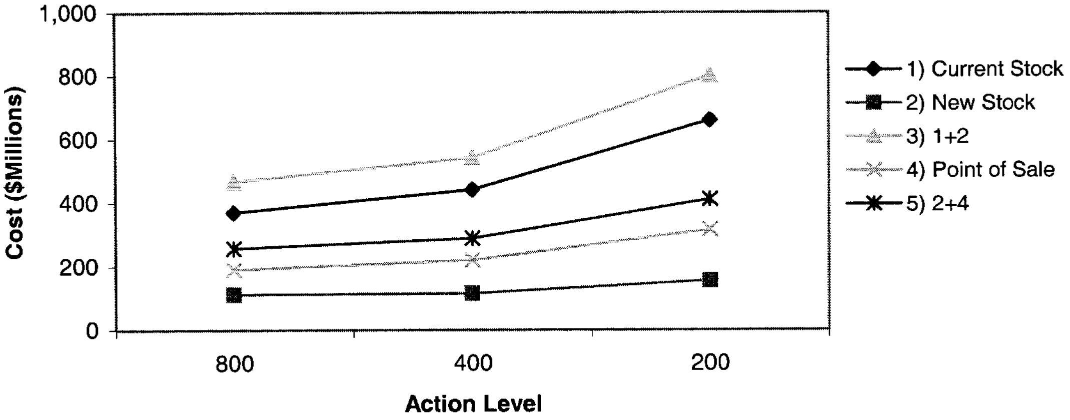
mitigation effectiveness. A 5% discount rate is applied to calculate the present value of future costs and benefits.

Note that the results presented in this section were selected in order to demonstrate key comparisons and/or trends, while maintaining brevity. For the reader's reference, this manuscript includes a large Appendix containing data tables in which are found all of the outputs of the decision model used in this thesis.

3.2.1 Total Cost

The estimated total discounted cost of each mitigation strategy is shown in Figure 3-7, by action level. All strategies are likely to be very expensive, with estimated total net costs ranging from \$112 million to \$467 million for mitigation at 800 Bqm^{-3}.

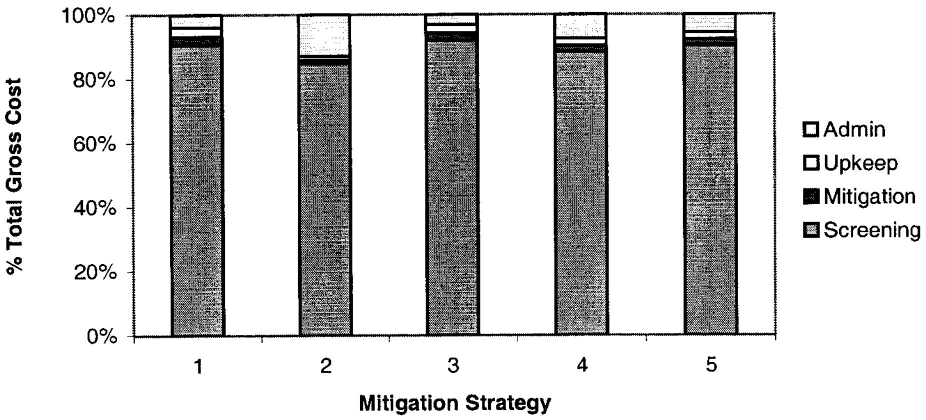
FIGURE 3-7. Net Total Cost, by Mitigation Strategy and Action Level



The most comprehensive approach to radon risk mitigation – strategy 3, which involves screening and mitigation all current dwellings, as well as newly-constructed dwellings – is the most expensive option. Note, however, that its cost is dominated by the cost of intervention within the currently existing housing stock (strategy 1). Mitigation within newly constructed dwellings (strategy 2) is the least expensive option if implemented alone. All strategies become more expensive as the level at which mitigation is recommended (the action level) decreases, with a large difference between the strategies in the degree to which costs increase (ranging from 39% to 79%) with the decreasing action level.

The costs shown in Figure 3-7 are comprised of 5 major components: screening costs, mitigation costs, upkeep costs, program administration costs, and treatment cost savings due to decreased lung cancer mortality. The sum of costs in the first four categories is the total gross cost, and is broken down as follows.

FIGURE 3-8(a). Percent Total Gross Cost, by Cost Component and Mitigation Strategy (Action Level: 800 Bqm^{-3})



It is evident that for a mitigation program run at the current Canadian guideline of 800 Bqm^{-3}, screening would be the dominant cost (ranging from 85-92% of the total gross cost). Lowering the action level has a significant effect on the distribution of costs among the four cost components, with the costs of screening comprising a progressively smaller proportion of total gross costs (Figure 3-8(b)).

FIGURE 3-8(b). Percent Total Gross Cost, by Cost Component and Action Level (Strategy 3)

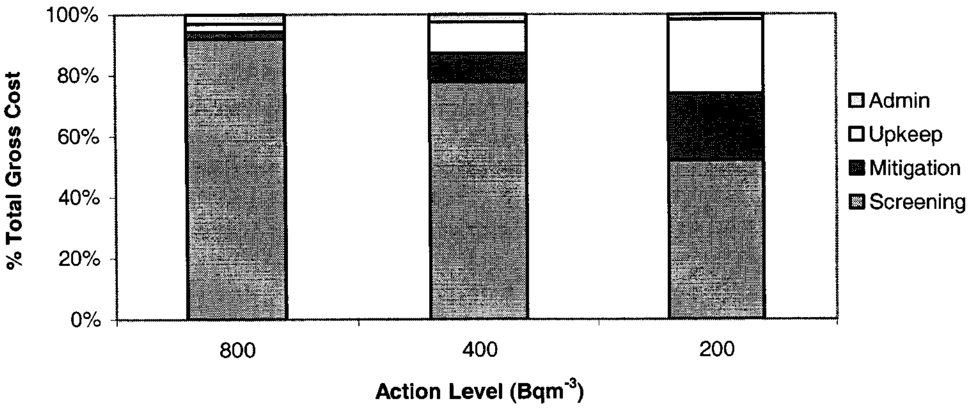
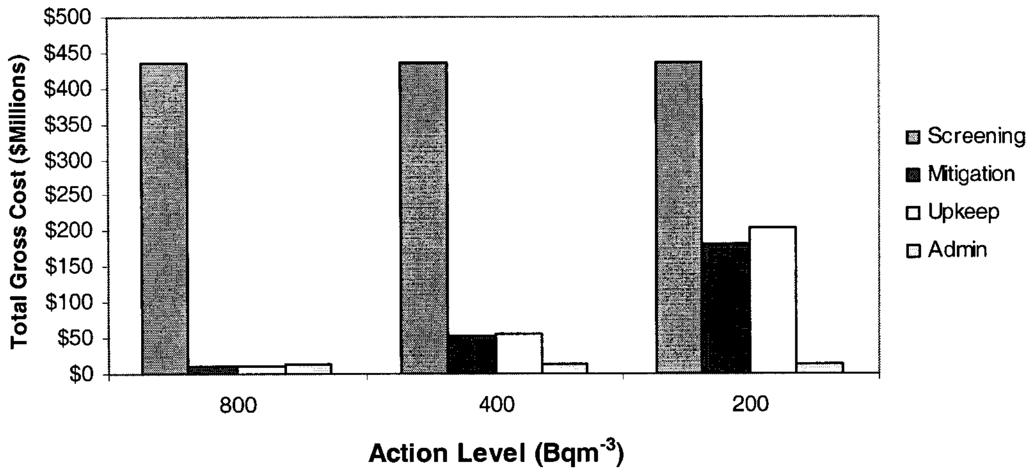


Figure 3-8(c) confirms that this changing distribution is a function of increasing mitigation-related costs, rather than a decrease in the screening costs themselves.

FIGURE 3-8(c). Total Gross Cost, by Cost Component and Action Level (Strategy 3)



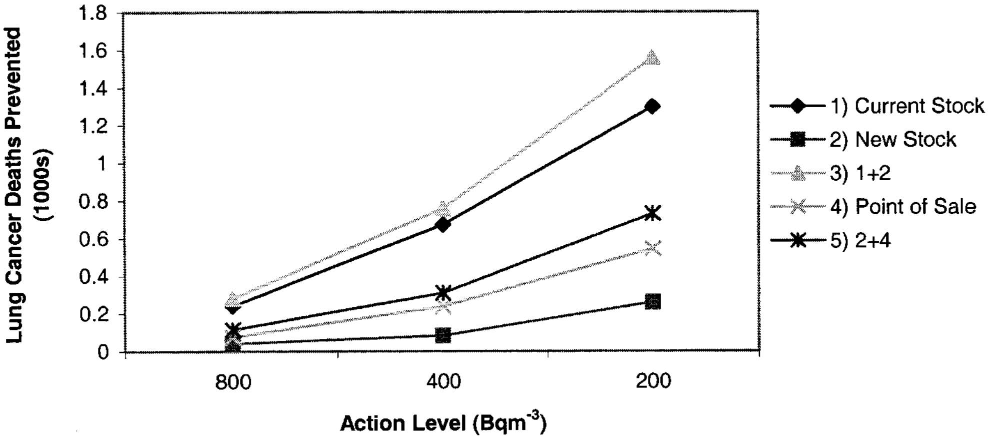
3.2.2 Health Benefits

3.2.2.1 Lung Cancer Deaths Prevented

Reducing residential radon exposure will have an impact upon the burden of lung cancer mortality within the affected population, preventing between 641 and 2,427 deaths over an 80-year period. However, as a significant proportion of these deaths occur well into the future, discounting at an annual rate of 5% yields an equivalent present value far below that number – between 35 and 238 lung cancer deaths prevented. As seen in Figure 3-9, below, the impact of radon mitigation on lung cancer mortality seems to vary directly with costs, with the most expensive mitigation option, strategy 3, having the largest effect (again dominated by the effect of strategy 1); the least effective option was also the least expensive – strategy 2.

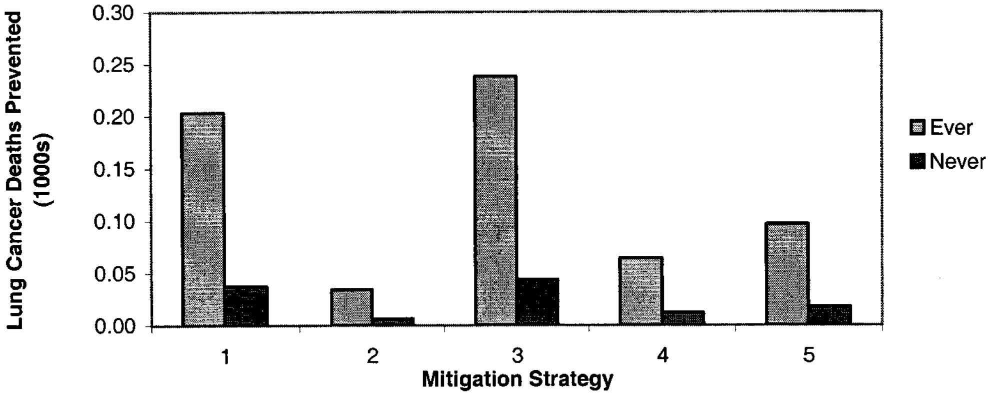
Decreasing the action level results in a 4- to 6-fold increase in the number of lung cancers deaths prevented, as well as an increase in the difference in impact achieved by the most- and least-effective strategies.

FIGURE 3-9. Lung Cancer Deaths Prevented, by Mitigation Strategy and Action Level



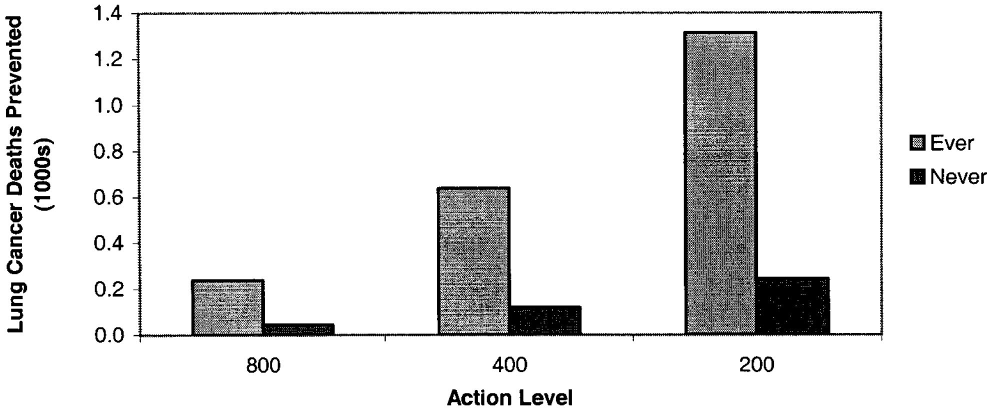
For all strategies, the majority (roughly 84%) of lung cancer deaths prevented would have occurred in ever-smokers, as illustrated in Figure 3-10(a).

FIGURE 3-10(a). Lung Cancer Deaths Prevented, Ever- vs. Never-Smokers, by Mitigation Strategy (Action Level: 800 Bqm^{-3})



Further, the increase in mitigation effectiveness with a decreasing action level is primarily the result of an increased number of deaths prevented in ever-smokers, as illustrated in Figure 3-10(b), below.

FIGURE 3-10(b). Lung Cancer Deaths Prevented, Ever- vs. Never-Smokers, by Action Level (Strategy 3)

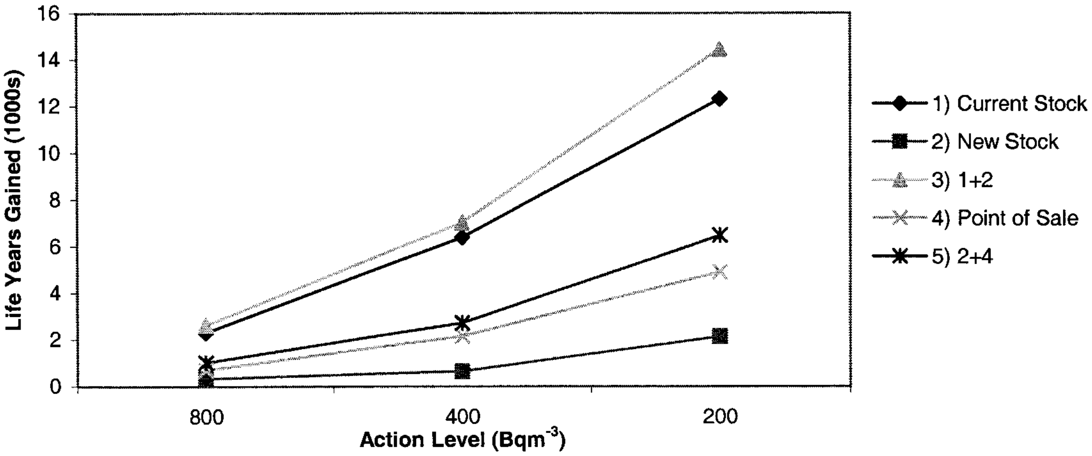


Approximately 84% of lung cancer deaths prevented would have occurred in ever-smokers, regardless of the action level. The results shown in Figure 3-11(b) are for strategy 3, but are representative of the results seen with all mitigation strategies.

3.2.2.2 Life-Years Gained

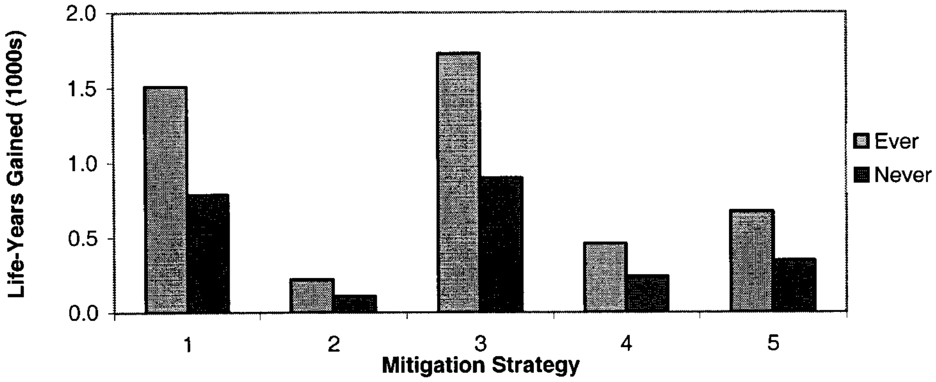
A reduction in lung cancer mortality will add between 329 and 2,627 discounted life-years to the affected population at an action level of 800 Bqm^{-3}, with impacts again rising as the action level is lowered (Figure 3-11).

FIGURE 3-11. Life-Years Gained, by Mitigation Strategy and Action Level



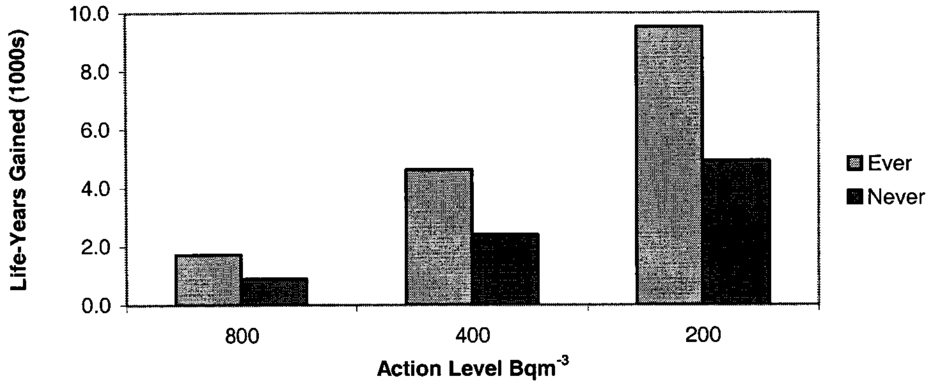
As with decreased mortality, the majority of the life-years gained by radon mitigation are observed within ever-smokers. Note, however, that never-smokers account for a much greater proportion of the total life-years gained than they did lung cancers prevented – a fact that can be explained by an increased number of offspring (from individuals who now survive to parenthood) and the low prevalence of smoking within younger age groups.

FIGURE 3-12(a). Life-Years Gained, Ever- vs. Never-Smokers, by Mitigation Strategy (Action Level: 800 Bq^{-3})



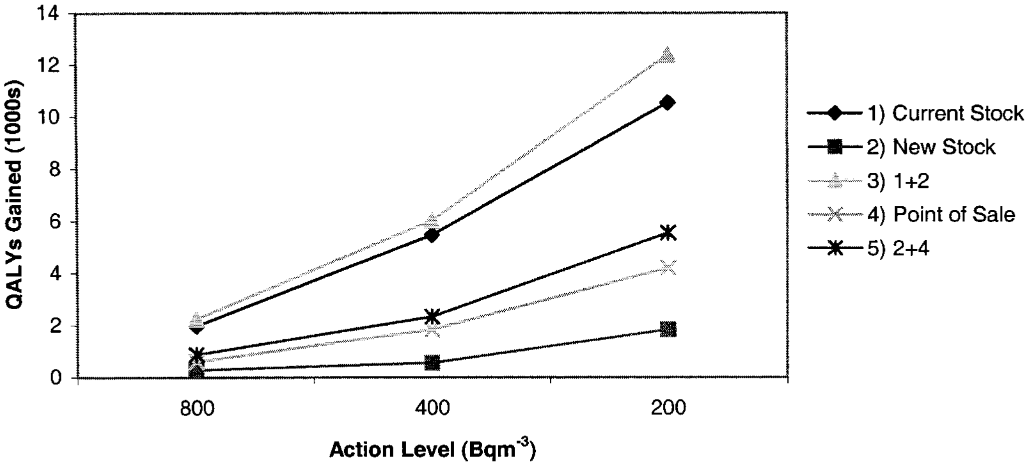
This remains true at action levels below 800 Bqm^{-3}, as shown in Figure 3-12(b). Although results are shown only for strategy 3, results for other radon mitigation strategies are similar.

FIGURE 3-12(b). Life-Years Gained, Ever- vs. Never-Smokers, by Mitigation Strategy (Action Level: 800 Bqm^{-3})



Adjustment of life-years gained for quality of life has a similar effect on the effectiveness of all of the mitigation strategies considered here, decreasing the estimated gains in life expectancy by about 14% for all strategies.

FIGURE 3-13. QALYs Gained, by Mitigation Strategy and Action Level

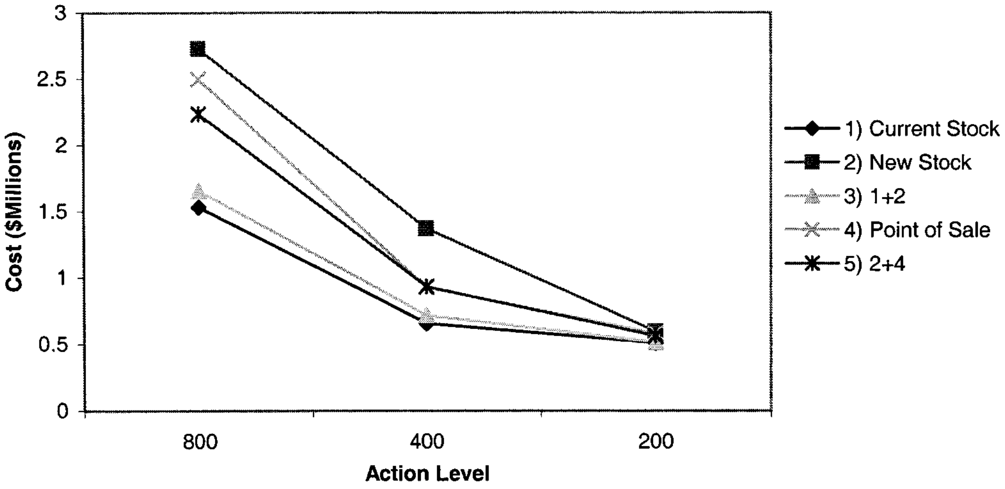


The patterns seen when stratifying QALYs gained by smoking-status are as seen previously. Radon mitigation had a greater impact in terms of QALYs gained when the action level was lowered from 800 Bqm^{-3}, and ever-smokers accounted for the large majority of QALYs gained.

3.2.3 Incremental Cost-Effectiveness

We report the estimated cost per lung cancer death (LCD) prevented by strategies 1 through 5, repeated for each action level, in Figure 3-14.

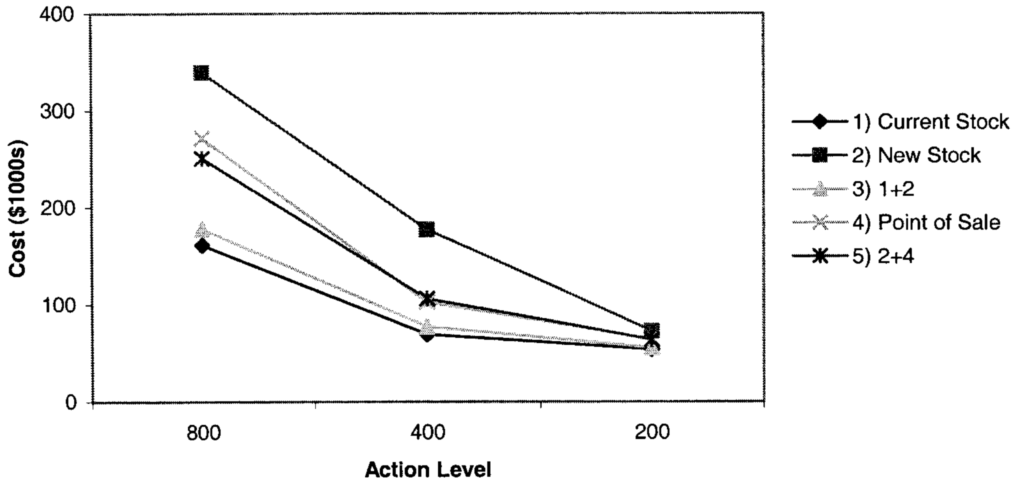
FIGURE 3-14. Cost per Lung Cancer Death Prevented, by Mitigation Strategy and Action Level



The strategies with the lowest average cost-effectiveness ratios are those found earlier to be the highest in cost but also the most effective – strategies 1 and 3 – exhibiting a cost per lung cancer prevented of $1.5 and $1.6 million at 800 Bqm^{-3}, respectively. This translates into a cost per life-year gained (Figure 3-16) of roughly $161,000 to $178,000, with adjustment for quality of life increasing these estimates by about 17%. Strategy 2, the least costly, also has the highest cost-effective ratios ($2.7 million per LCD prevented; $339,000 per life-year gained) due to its relatively small impact on lung cancer mortality.

For all strategies, decreasing the action level from 800 Bqm^{-3} results in a significant reduction in the cost per life saved. It also greatly decreases the difference in average cost effectiveness between the most and least cost-effective strategies to just over $87,000 per LCD prevented at the lowest action level of 200 Bqm^{-3}.

FIGURE 3-15. Cost per Life-Year Gained, by Mitigation Strategy and Action Level



While the above figures are informative, decision-making is perhaps better informed by considering the total costs and health benefits of mitigation simultaneously. In Figure 3-16, we plot the cost of each strategy-action level combination by its effectiveness in terms of QALYs gained. The slope from one data point to another represents the incremental cost-effectiveness of changing between the strategies they represent. The line shown in the Figure depicts the most incrementally cost-effective mitigation options, with cost-effectiveness ratios shown in Table 3-1.

FIGURE 3-16. Cost-Effectiveness Plane, by Mitigation Strategy (S) and Action Level

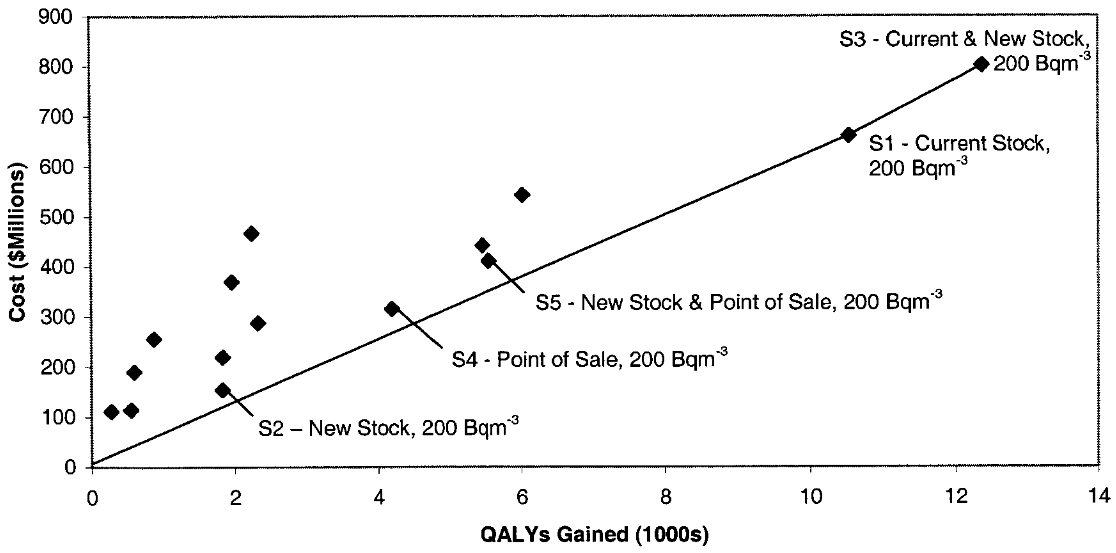


TABLE 3-1. Incremental Cost-Effectiveness Analysis, 50% Compliance

Rank[a] (C.E.)	Strategy	AL[b] (Bqm^{-3})	Cost ($Milions)	Health Effects: LCD[c] Prevented	Health Effects: QALYs[d] Gained	Incremental Cost per: LCD Prevented	Incremental Cost per: QALY Gained
1	Current Stock	200	$661	1,298	10,546	$509,082	$62.636
2	Current & New Stock	200	$801	1,557	12,379	$540,820	$76,615

[a] Rank in terms of Cost-Effectiveness (Note that only those options that are the most incrementally cost-effective means of increasing the health impact of radon mitigation are presented).
[b] Action Level; [c] Lung Cancer Death; [d] Quality-adjusted Life-Years

From Figure 3-16 and Table 3-1, it is clear that the most cost-effective mitigation option is to screen all currently existing dwellings (strategy 1), and mitigate those above 200 Bqm^{-3}. This would cost about $62,636 per QALY gained. Further, the most cost-effective way to increase the impact of radon mitigation above that achieved by strategy 1 would be to add to it a program to screen and mitigate all newly constructed dwellings (strategy 3), again with an action level of 200 Bqm^{-3}. This change would cost $76,615 for every additional QALY gained.

3.3 SENSITIVITY ANALYSIS

In this section, we consider the impact that changes to a number of variables will have upon the costs, benefits, and cost-effectiveness of the five national radon mitigation strategies considered here. For brevity, only results for ever- and never-smokers combined are presented.

3.3.1 Effect of Compliance with Radon Guideline

The rate at which homeowners comply with the set action level is an important characteristic of any program to reduce radon levels within Canada. While increased compliance results in relatively minor changes in the net total cost of mitigation by any strategy at 800 Bqm^{-3} (see Figure 3-17), the change in the number of lung cancer deaths prevented is far more pronounced, as shown in Figure 3-18.

FIGURE 3-17. Net Total Cost, by Mitigation Strategy and Compliance Rate (Action Level: 800 Bqm^{-3})

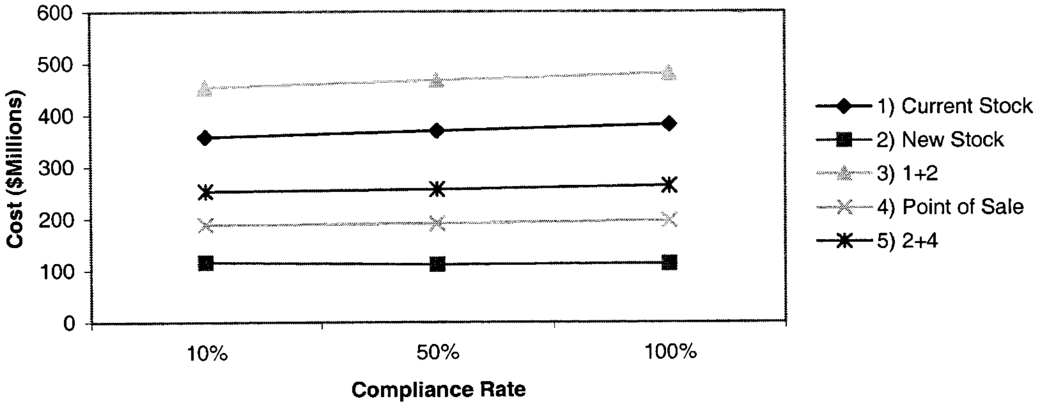
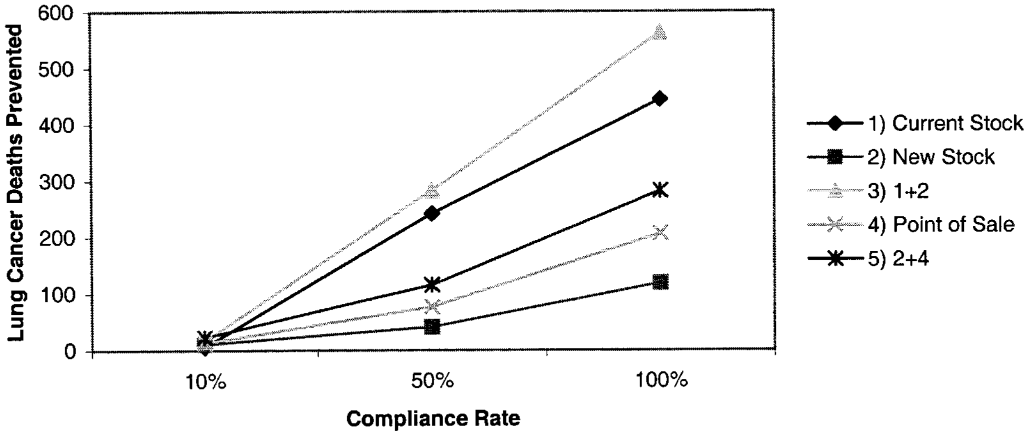


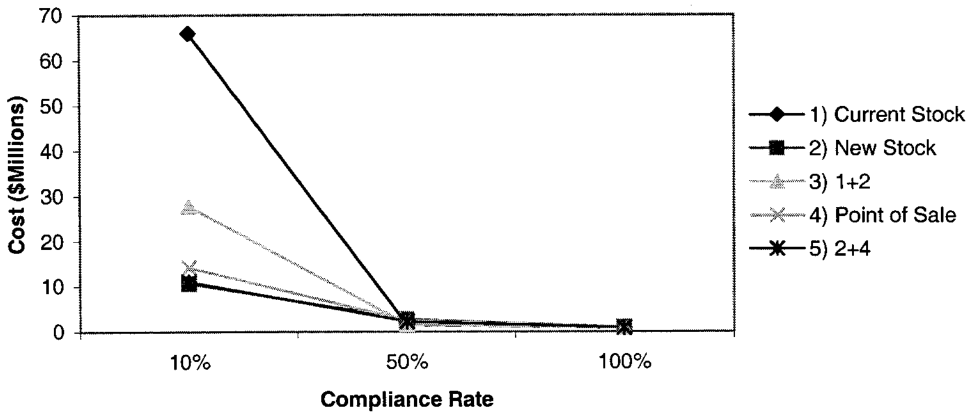
FIGURE 3-18. Lung Cancer Deaths Prevented, by Mitigation Strategy and Compliance Rate (Action Level: 800 Bqm^{-3}).



This is especially true with those strategies that prevent the greatest number of lung cancer deaths. For example, with strategy 1, an increase in compliance from 10% to 50% results in an estimated 40-fold increase in lung cancer deaths prevented. Raising compliance to 100% further doubles the impact of strategy 1.

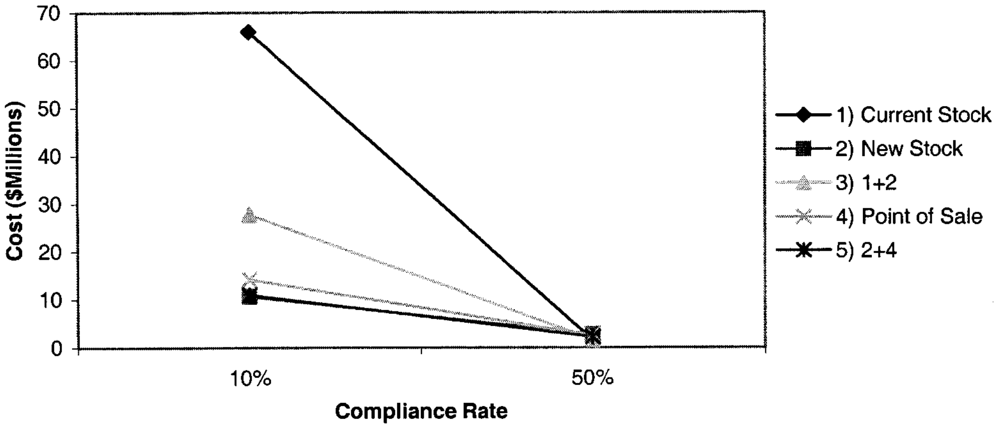
The combination of the above results leads to a significant impact upon the cost-effectiveness of the strategies with increasing compliance, as demonstrated in Figure 3-19(a) and (b).

FIGURE 3-19(a). Cost per Lung Cancer Death Prevented, by Mitigation Strategy and Annual Discount Rate (Action Level: 800 Bqm^{-3})



Removing the case of 10% compliance from Figure 3-19(a) allows for a more clear illustration of the effect of moving from 50% to 100%, as shown in Figure 3-19(b).

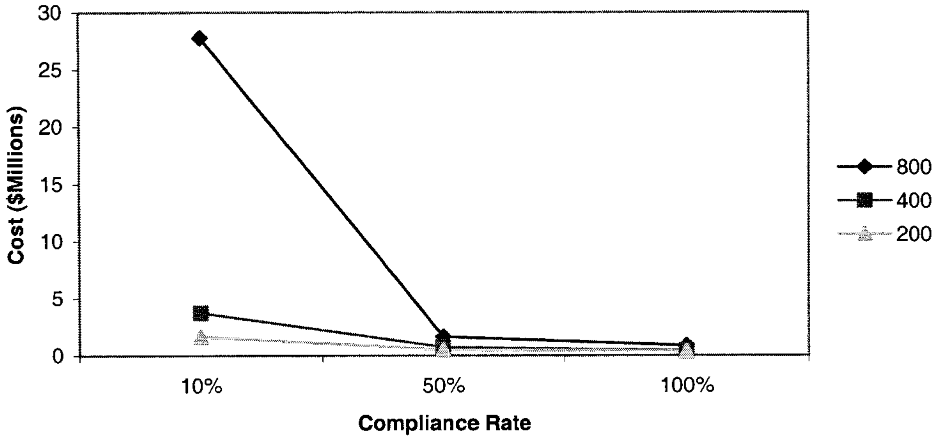
FIGURE 3-19(b). Cost per Lung Cancer Death Prevented, by Mitigation Strategy and Annual Discount Rate (Action Level: 800 Bqm^{-3})



Thus, increasing compliance both decreases the cost per lung cancer prevented, and also the differences in that cost among the five mitigation strategies. There exists a $1.5 million difference per LCD prevented between the most and least cost-effective strategies at 50% compliance, which reduces to about $100,000 at 100% compliance.

Figure 3-20 indicates that as the action level is lowered from 800 Bqm^{-3}, the importance of the compliance rate in the determination of the cost-effectiveness is diminished,

FIGURE 3-20. Cost per Lung Cancer Death Prevented, by Action Level and Compliance Rate (Strategy 3)



Figures 3-21(a) and (b) plot the net total cost of each strategy-action level combination against its anticipated effects, for 10% and 100% compliance respectively. Table 3-2 provides the incremental cost-effectiveness of the most cost-effective intervention strategies, which are again joined by solid lines with the figures.

FIGURE 3-21(a). Cost-Effectiveness Plane, by Mitigation Strategy (S) and Action Level (10% Compliance)

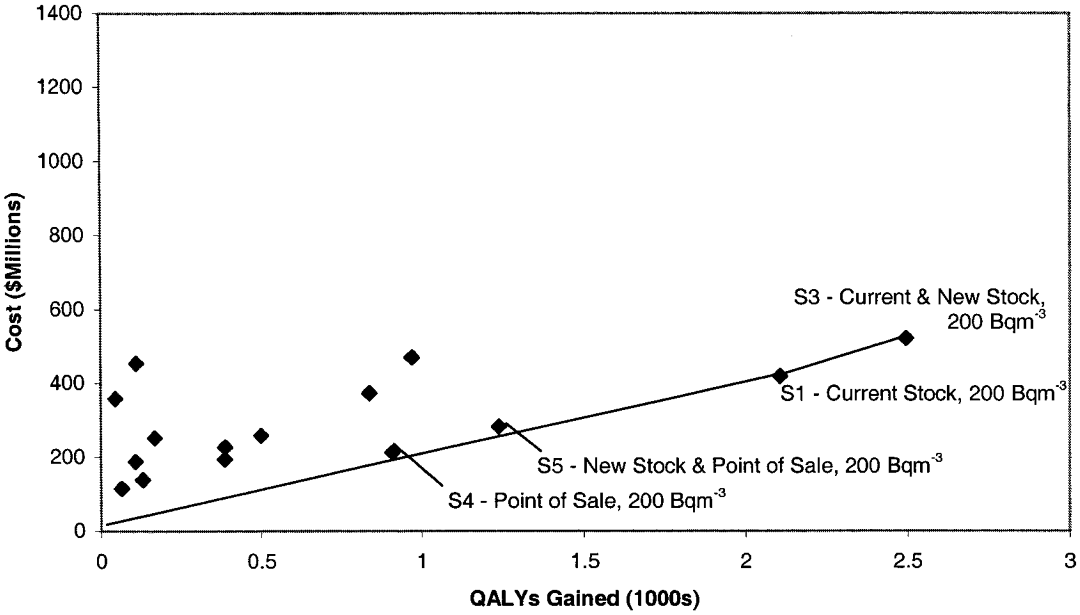


FIGURE 3-21(b). Cost-Effectiveness Plane, by Mitigation Strategy (S) and Action Level (100% Compliance)

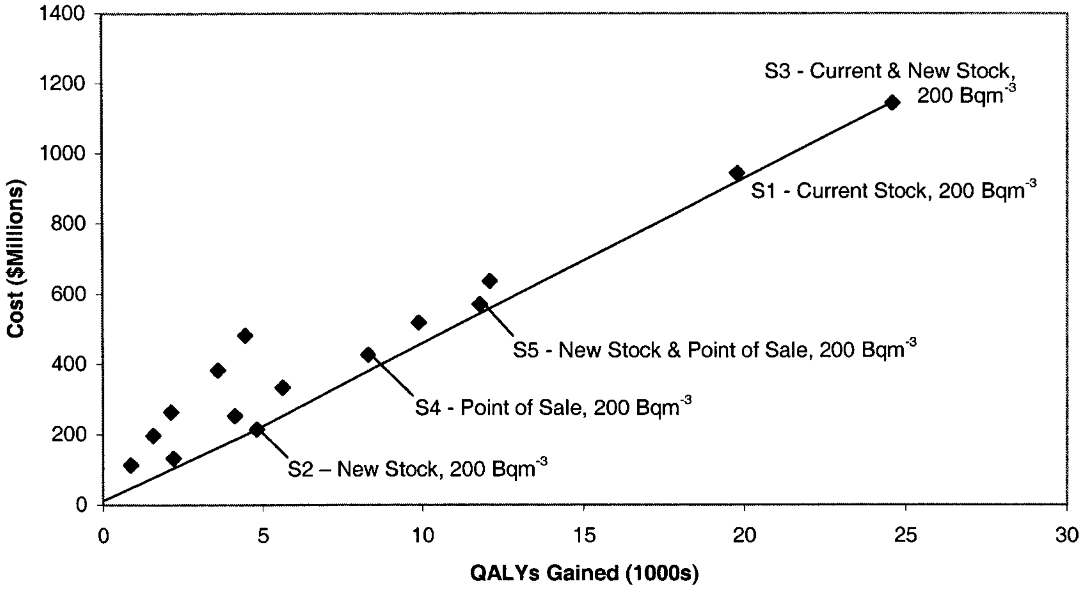


TABLE 3-2. Incremental Cost-Effectiveness Analysis, by Rate of Homeowner Compliance

Rank [a] (C.E.)	Strategy	AL[a] (Bqm^{-3})	Cost ($Milions)	Health Effects		Incremental Cost per	
				LCD[a] Prevented	QALYs [b] Gained	LCD Prevented	QALY Gained
			10% Compliance				
1	Current Stock	200	$418	259	2,107	$1,618,284	$198,578
2	Current & New Stock	200	$521	317	2,495	$1,767,537	$264,337
			50% Compliance				
1	Current Stock	200	$661	1,298	10,546	$509,082	$62,636
2	Current & New Stock	200	$801	1,557	12,379	$540,820	$76,615
			100% Compliance				
1	New Stock	200	$214	661	4,813	$323,764	$44,491
2	Current & New Stock	200	$1,143	3,097	24,601	$381,488	$46,959

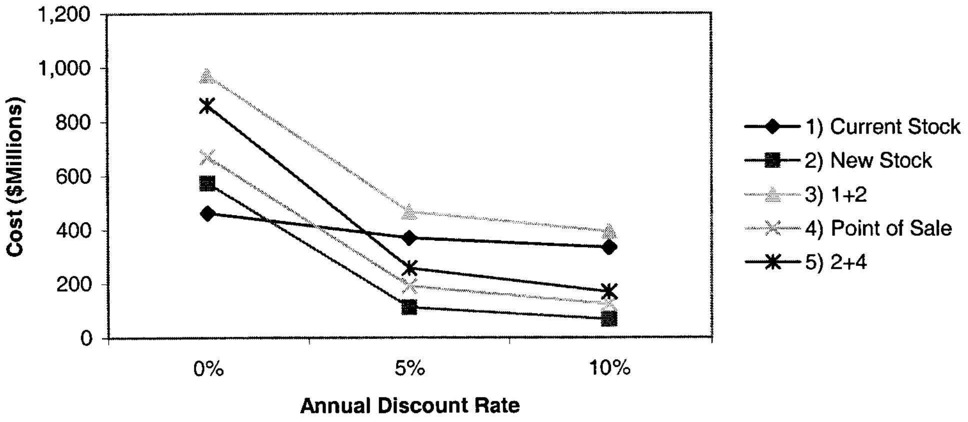
[a] Rank in terms of Cost-Effectiveness (Note that only those options that are the most incrementally cost-effective means of increasing the health impact of radon mitigation are presented).
[b] Action Level; [c] Lung Cancer Death; [d] Quality-adjusted Life-Years

Increasing compliance clearly has a significant impact upon the incremental cost-effectiveness of residential radon mitigation, such that full compliance changes the rank order of the strategies, with strategy 2 becoming the most cost-effective option.

3.3.2 Effect of Discounting

The degree to which decision-makers place more emphasis on costs and benefits occurring earlier as compared to later – represented by the annual discount rate applied in economic analyses – can significantly change the attractiveness of residential radon mitigation strategies. This is evident in Figure 3-22, which describes the effect of the choice of discount rate on the estimated total net cost of mitigation at 800 Bqm^{-3}.

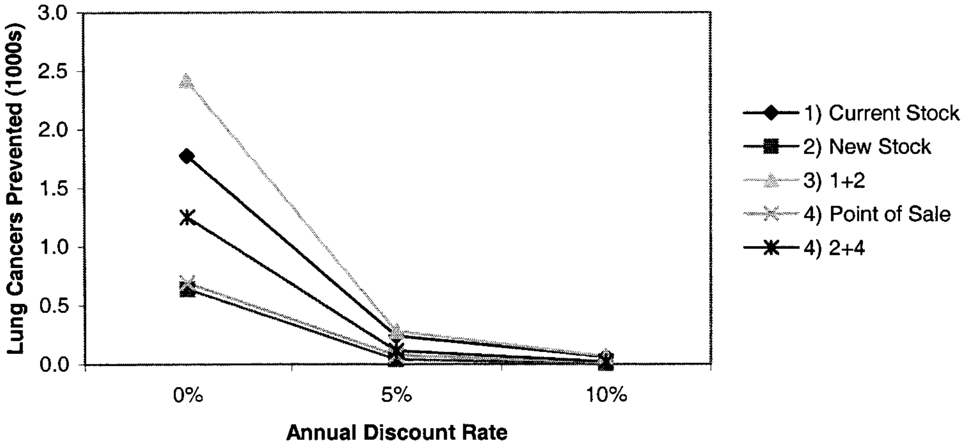
FIGURE 3-22. Net Total Cost, by Mitigation Strategy and Annual Discount Rate (Action Level: 800 Bqm^{-3})



Discounting has a marked impact on the estimated costs of those strategies implemented over a extended period of time. For example, the cost of strategy 2 decreases by 88% when a discount rate of 10% is used, rather than a rate of 0%. In contrast, the relative insensitivity of strategy 1 (a 28% decrease), for which the majority of costs are incurred within the first 5 years of the planning horizon, results in it being very nearly the most expensive choice when any discounting is applied, but the least expensive otherwise. Generally, costs appear to be more sensitive to the difference between the presence and absence of discounting than they do between the choice of the discount rate.

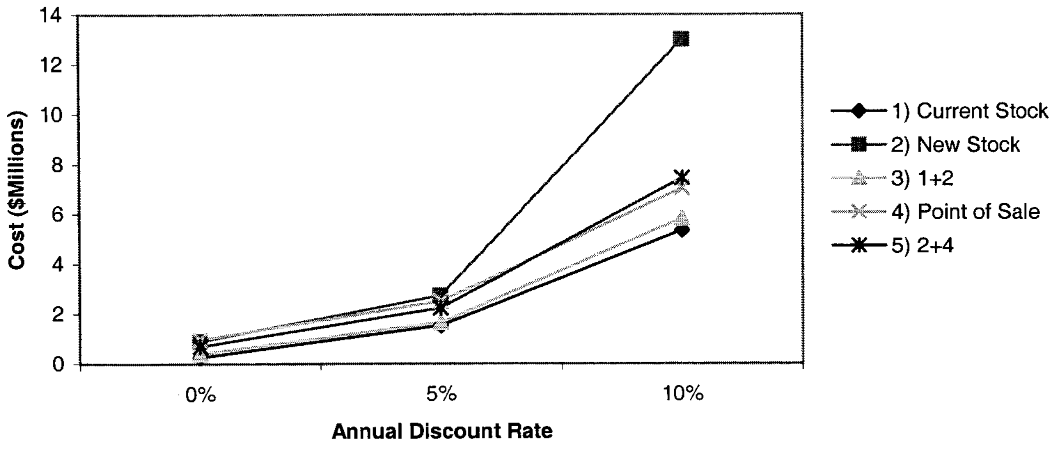
As described earlier, discounting has a notable impact on the predicted health benefits of radon mitigation. As shown in Figure 3-23, moving from 0% to 5% discounting decreases the estimated number of lung cancers prevented by between 85% and 95% (similar decreases are observed in life-years gained). Increasing the discount rate also decreases the difference between the most and least effective strategies in the number of lung cancer deaths prevented, from 1,776 deaths at 0% to just 57 at 10%.

FIGURE 3-23. Lung Cancer Deaths Prevented, by Mitigation Strategy and Annual Discount Rate (Action Level: 800 Bqm^{-3})



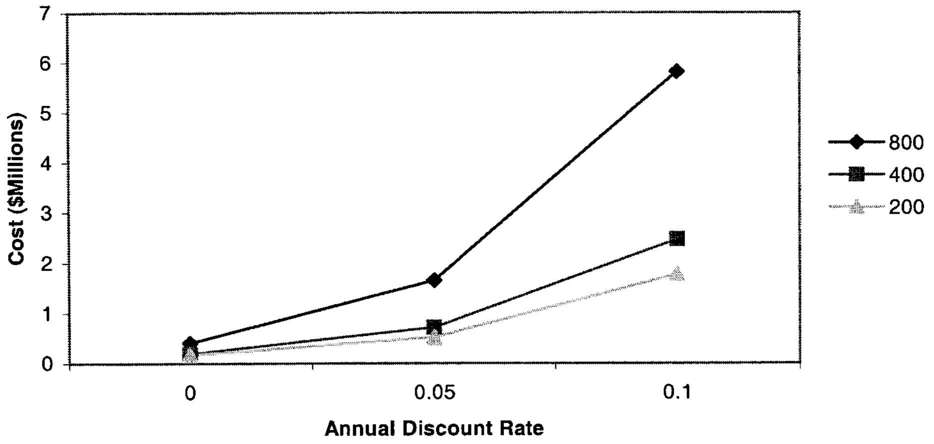
Comparing Figure 3-23 with Figure 3-24, it is apparent that the benefits of radon mitigation are more sensitive to discounting than are costs. As a results, the cost effectiveness of all strategies decreases dramatically with an increasing discount rate, with anywhere from a 6 to 20-fold increase in the cost to prevent a lung cancer death (Figure 3-25, below). The strategies with higher cost-effective ratios are generally more sensitive to discounting, increasing the differences between them and the more cost-effective options with increasing discount rate (from $700,000 at 0% to $7.6 million at 10%). Interestingly, the average cost-effectiveness ratios appear to be more sensitive to the difference between discounting at 5% and 10%, rather than to changing between 0% and 5% as is seen for both costs and benefits.

FIGURE 3-24. Cost per Lung Cancer Death Prevented, by Mitigation Strategy and Annual Discount Rate (Action Level: 800 Bqm^{-3})



As demonstrated in Figure 3-26, below, sensitivity of the cost-effectiveness ratios to discounting decreases with a decreasing action level.

FIGURE 3-25. Cost per Lung Cancer Death Prevented, by Action Level and Annual Discount Rate (Strategy 3)



The choice of discount rate may impact the attractiveness of radon mitigation to decision-makers. To illustrate this, Figure 3-26 presents the cost-effectiveness plane for a 0% discount rate with 50% compliance, while Table 3-3 considers the most incrementally cost-effective options at each of the 3 discount rates. The cost-effectiveness plane with 10% discounting was similar to that seen with 5%, shown in Figure 3-16, and hence is not presented.

FIGURE 3-26. Cost-Effectiveness Plane, Mitigation Strategy (S) by Action Level (50% Compliance; 0% Discounting)

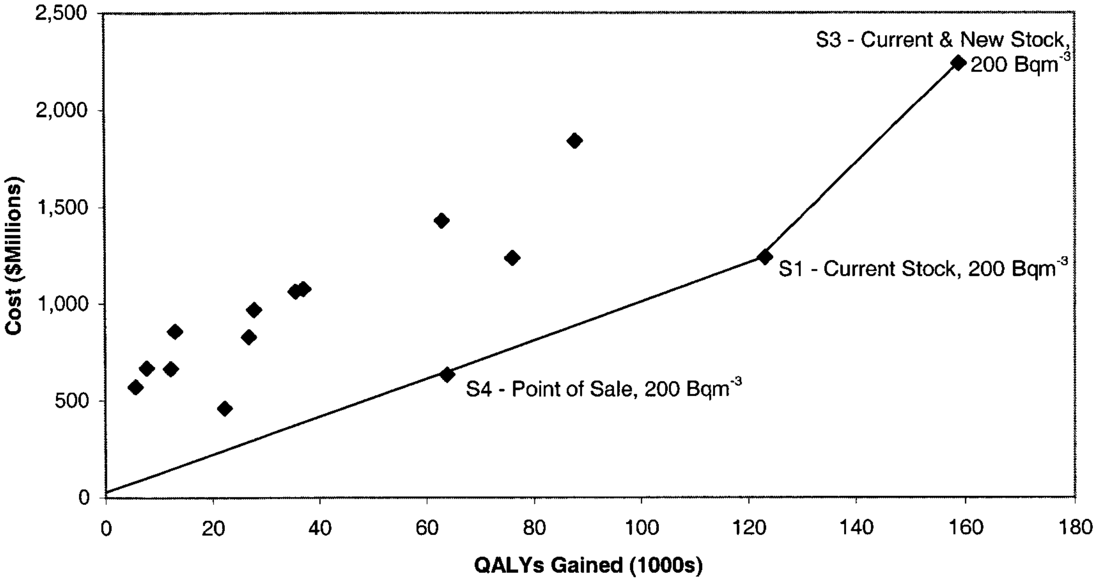


TABLE 3-3. Incremental Cost-Effectiveness Analysis, by Discount Rate (50% Compliance)

Rank[a] (C.E.)	Strategy	AL[a] (Bqm^{-3})	Cost ($Milions)	Health Effects		Incremental Cost per	
				LCD[a] Prevented	QALYs[b] Gained	LCD Prevented	QALY Gained
				0% Discount			
1	Current Stock	400	$633	5,111	63,884	$123,952	$9,916
2	Current Stock	200	$1,240	9,797	123,171	$129,456	$10,233
3	Current & New Stock	200	$2,241	11,516	158,902	$582,049	$27,998
				5% Discount			
1	Current Stock	200	$661	1,298	10,546	$509,082	$62,636
2	Current & New Stock	200	$801	1,557	12,379	$540,820	$76,615
				10% Discount			
1	New Stock	200	$556	324	1,712	$1,716,303	$325,123
2	Current & New Stock	200	$636	358	1,882	$2,357,034	$467,519

[a] Rank in terms of Cost-Effectiveness (Note that only those options that are the most incrementally cost-effective means of increasing the health impact of radon mitigation are presented).
[b] Action Level; [c] Lung Cancer Death; [d] Quality-adjusted Life-Years

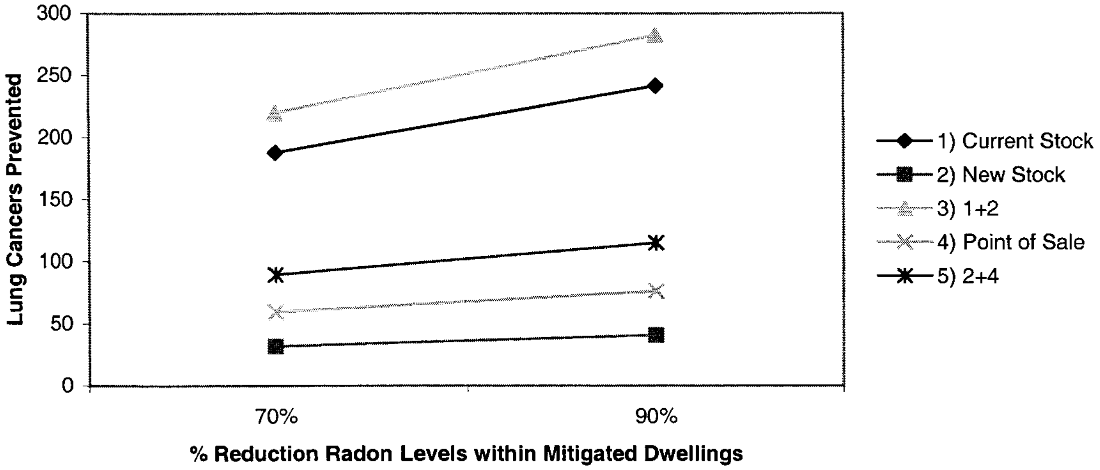
As previously observed, increasing the discount rate decreases the apparent cost-effectiveness of radon mitigation. It is interesting to note that with 0% discounting, implementing strategy 1 with an action level of 400 Bqm^{-3} is the most cost-effective option – the first time a higher radon guideline has provided the most attractive mitigation option. After this, the most incrementally cost-effective way of improving the impact with 0% discounting is to lower the guideline to 200 Bqm^{-3}. Note also that when discounting is increased to 10%, the most cost-effective option is strategy 2 at 200 Bqm^{-3} – the same as was observed when compliance was raised to 100%.

For brevity, we do not present tables detailing the how the results in Table 3-3 change with a changing rate of homeowner compliance. Analysis indicates that compliance does not change which strategy-action level combinations are most cost-effective at each level of discounting, with two exceptions: (1) for 5% discounting, as previously seen in Table 3-2; and (2) when discounting at 0%, moving from 50% to 10% compliance eliminates strategy 1 at 400 Bqm^{-3} from among the optimal choices. As observed before, increasing compliance improves the cost-effectiveness of intervention at all levels of discounting. The degree of increase observed is greater when a smaller annual discount rate is applied.

3.3.3 Effect of the % Reduction in Radon Levels Achieved in Mitigated Dwellings

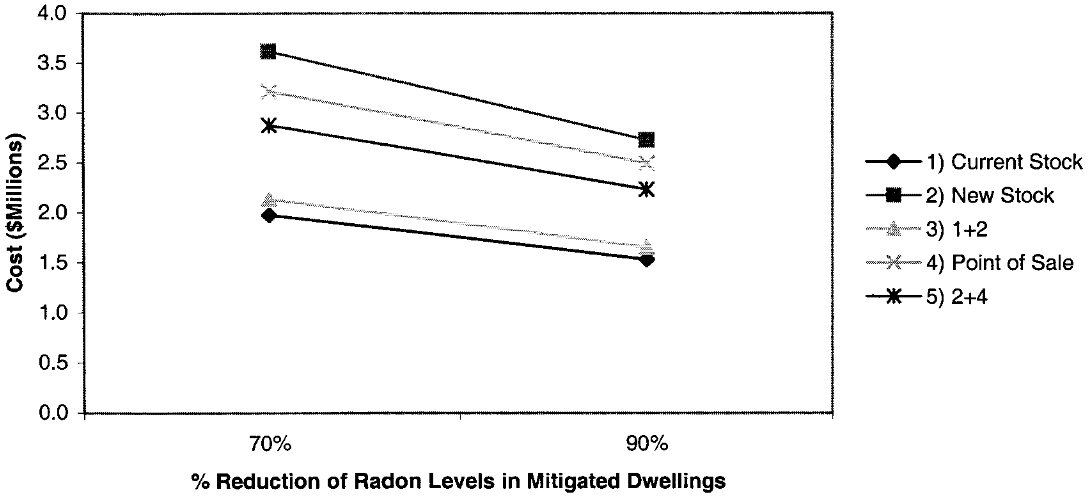
With total costs dominated by those associated with screening dwellings as previously observed, the net total cost of each strategy is highly insensitive to the effectiveness of radon reduction within dwellings demonstrating radon concentrations at or above 800 Bqm^{-3} (Small decreases in cost ($0.3-3.0 million) are observed for all strategies, resulting from treatment cost savings due to reduced lung cancer mortality). However, the number of lung cancer deaths prevented by each strategy is quite sensitive to the percent reduction in radon concentrations than are costs, as shown in Figure 3-27, below. A decrease in the % reduction of radon concentrations achieved by mitigation from 90% to 70% lowers the number of lung cancer deaths prevented by all strategies by 28%, regardless of the action level.

FIGURE 3-27. Lung Cancer Deaths Prevented, by Mitigation Strategy and % Reduction in Radon Levels Achieved (Action Level: 800 Bqm^{-3})



It follows, then, that an increase in cost-effectiveness is expected when moving from 70% to 90% reduction from pre-mitigation radon levels for all strategies. This is shown in Figure 3-28.

FIGURE 3-28. Cost per Lung Cancer Death Prevented, by Mitigation Strategy and % Reduction in Radon Levels Achieved (Action Level: 800 Bqm^{-3})



Sensitivity of the cost-effectiveness ratios to the percent reduction in radon levels becomes less pronounced as the action level is lowered from 800 Bqm^{-3}. However, as seen in Figure 3-28, below, strategy 2 retains more sensitivity (due to its lower total cost) than do the other options.

FIGURE 3-29. Cost per Lung Cancer Death Prevented, by Mitigation Strategy and % Reduction in Radon Levels Achieved (Action Level: 200 Bqm^{-3})

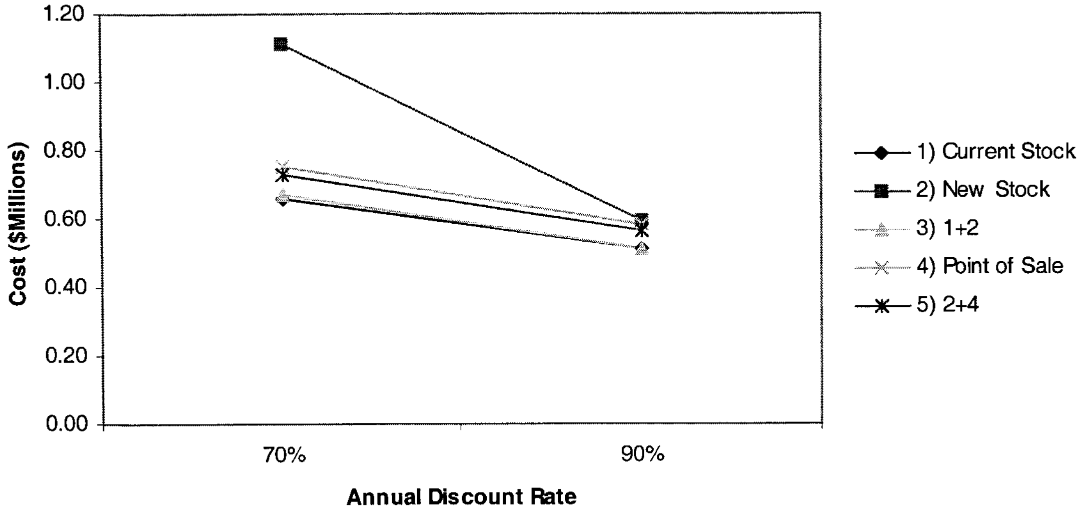


Table 3-4 presents the results from the incremental cost-effectiveness analysis for the optimal strategy-action level combinations with 50% compliance. A figure detailing the cost-effectiveness plane is not shown, as in all cases the optimal strategies followed the same pattern as was seen before in Figure 3-17.

TABLE 3-4. Incremental Cost-Effectiveness Analysis, by % Reduction In Radon Levels (50% Compliance)

Rank [a] (C.E.)	Strategy	AL[a] (Bqm^{-3})	Cost ($Milions)	Health Effects		Incremental Cost per...	
				LCD[a] Prevented	QALYs [b] Gained	LCD Prevented	QALY Gained
			70% Reduction				
1	Current Stock	200	$667	1,009	8,202	$660,958	$81,322
2	Current & New Stock	200	$809	1,211	9,628	$701,811	$99,419
			90% Reduction				
1	Current Stock	200	$661	1,298	10,546	$509,082	$62.636
2	Current & New Stock	200	$801	1,557	12,379	$540,820	$76,615

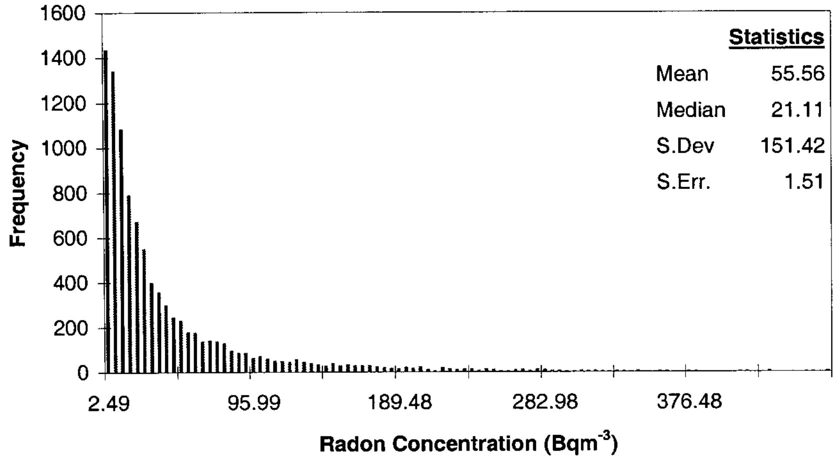
[a] Rank in terms of Cost-Effectiveness (Note that only those options that are the most incrementally cost-effective means of increasing the health impact of radon mitigation are presented).
[b] Action Level; [c] Lung Cancer Death; [d] Quality-adjusted Life-Years

Table 3-4 demonstrates that while the percent reduction in radon levels has very little impact on total cost, its effect on the health impacts of mitigation is such that it has a significant effect on the incremental cost-effectiveness of intervention. Since similar impacts are seen at all levels of compliance, results stratified by compliance rate are not presented here.

3.3.4 Effect of Background Radon Concentration Distribution

After 10,000 Monte Carlo trials, the baseline indoor radon concentration distributions for Sudbury and Winnipeg were modelled as follows.

FIGURE 3-30(a). Indoor Radon Concentration Distribution, Sudbury, Baseline

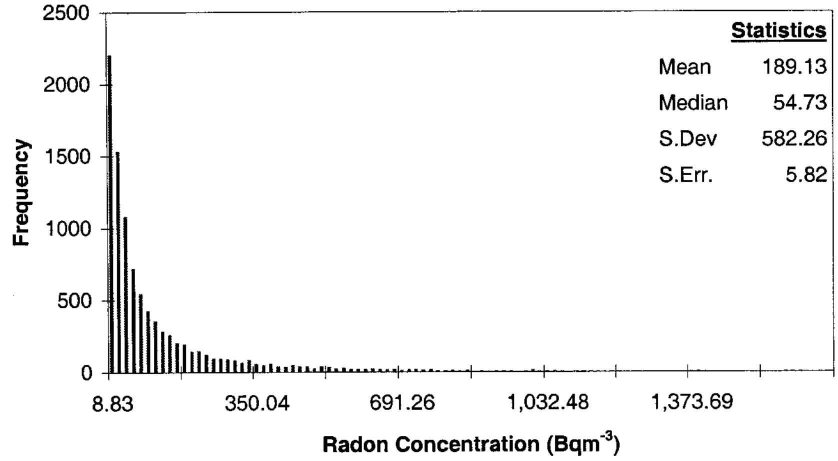


Statistics	
Mean	55.56
Median	21.11
S.Dev	151.42
S.Err.	1.51

Percentiles	
0%	0.2
2.5%	1.4
5%	2.3
50%	21.1
95%	203.9
97.5%	313.1
100%	8,284.1

Note: Displayed range is from 0.15 to 467.64 Bqm^{-3}. Actual range is from 0.15 to 8,284.10 Bqm^{-3}

FIGURE 3-30(b). Indoor Radon Concentration Distribution, Winnipeg, Baseline



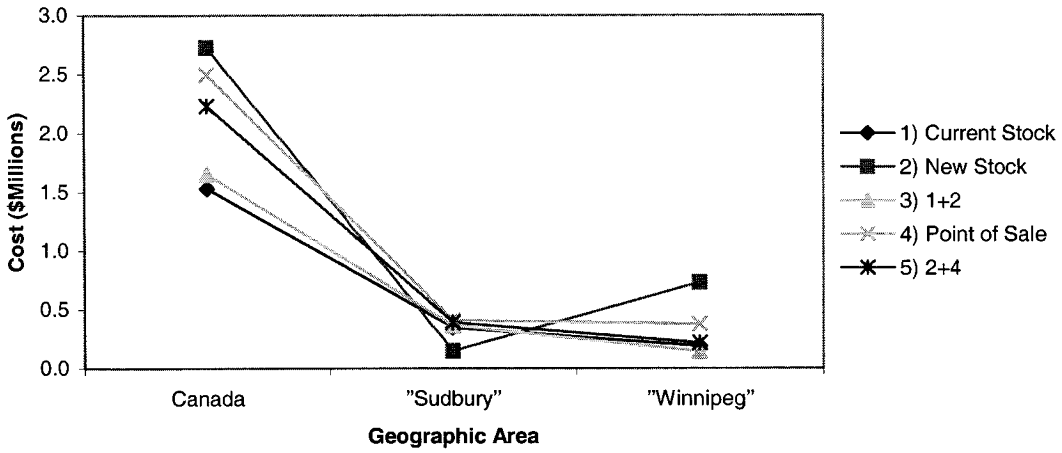
Statistics	
Mean	189.13
Median	54.73
S.Dev	582.26
S.Err.	5.82

Percentiles	
0%	0.3
2.5%	2.9
5%	4.7
50%	54.7
95%	707.0
97.5%	1,136.7
100%	18,549.4

Note: Displayed range is from 0.30 to 1,706.38 Bqm^{-3}. Actual range is from 0.27 to 18,549.36 Bqm^{-3}

The effect on cost-effectiveness of a change in the indoor radon concentration distribution to which radon reduction strategies are applied can be seen in Figure 3-31. Recall that the arithmetic means of the distributions for Sudbury (55.6 Bqm^{-3}) and Winnipeg (189.1 Bqm^{-3}) are substantially higher than that for Canada as a whole (i.e. 28.5 Bqm^{-3}).

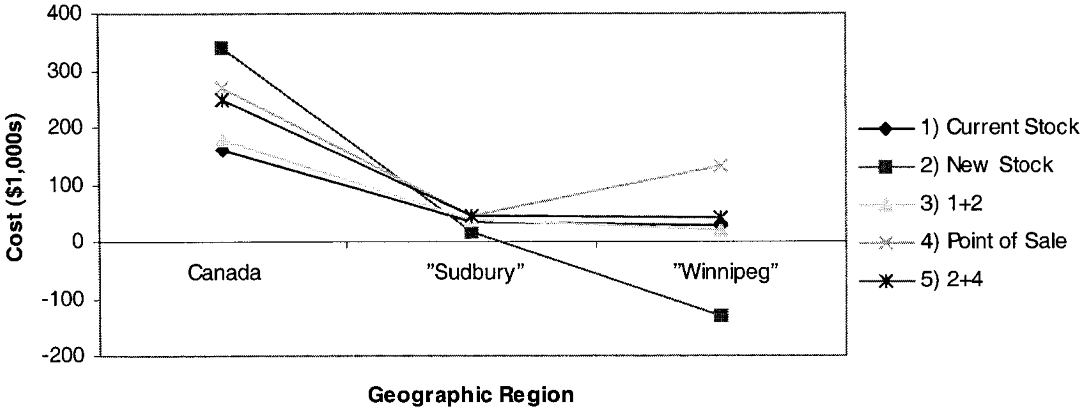
FIGURE 3-31. Cost per Lung Cancer Prevented, by Mitigation Strategy and Mean Radon Concentration (Action Level: 800 Bqm^{-3})



Two results are evident from Figure 3-31: (1) in general, the cost per lung cancer prevented by radon mitigation appears to decrease substantially when risk reduction efforts are applied to an area with higher average indoor radon concentrations; and (2) the change in cost-effectiveness modelled for strategy 2 is significantly different than observed with the other 4 strategies; for strategy two, an increase in average background radon concentrations from those seen in Sudbury those to seen in Winnipeg increased the average cost per lung cancer prevented.

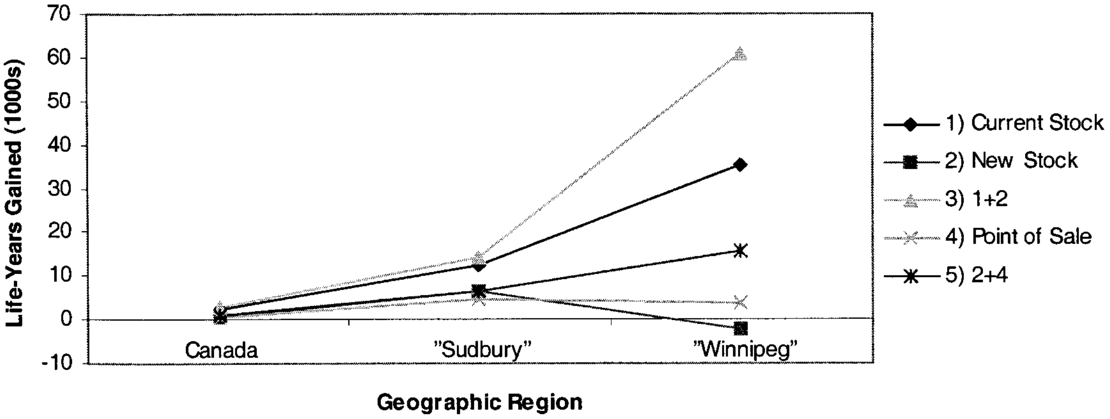
It is plausible that a change in background radon distribution might have a different effect on the cost-effectiveness of strategy 2 than is observed for the other mitigation options. However, measuring cost-effectiveness in terms of cost per life-years gained yields evidence that the validity of the decision model for the prediction of health effects with changing radon concentration distributions may be called into question (Figure 3-32).

FIGURE 3-32. Cost per Life-Year Gained, by Mitigation Strategy and Mean Radon Concentration (Action Level: 800 Bqm^{3-})



As evident from the figure, strategy 2 exhibits a negative cost per life-year gained under the Winnipeg scenario. A negative cost-effectiveness ratio would result from either negative costs (i.e., cost savings) or negative effects (i.e., *decreased* number of life-years lived under mitigated conditions). The results in Figure 3-33 indicate that the latter explanation holds.

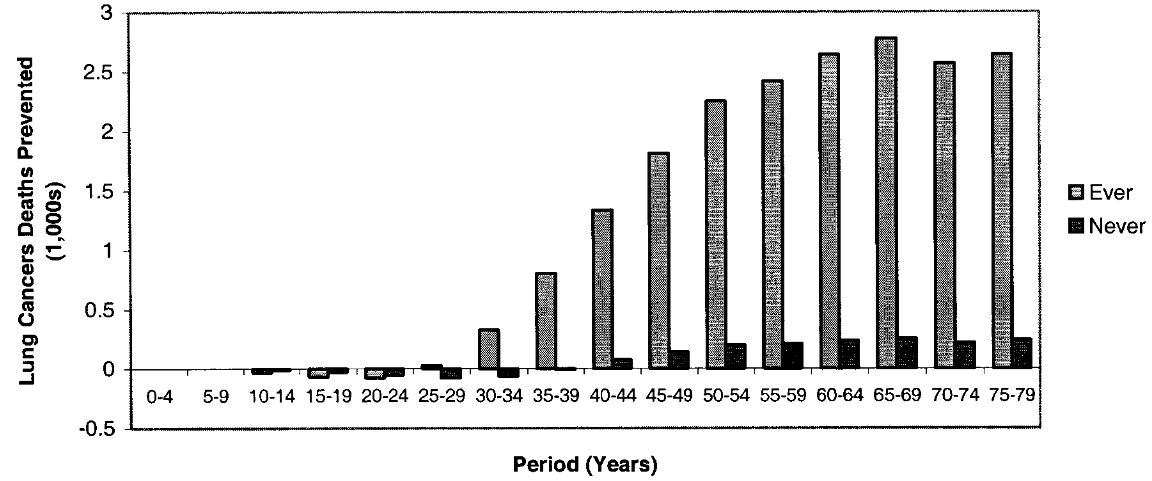
FIGURE 3-33. Life-Years Gained, by Mitigation Strategy and Mean Radon Concentration (Action Level: 800 Bqm^{-3})



A reduced number of life-years lived is indicative of an increased number of lung cancer deaths. A closer look at the distribution of lung cancer deaths prevented by the modelled radon mitigation reveals an interesting pattern.

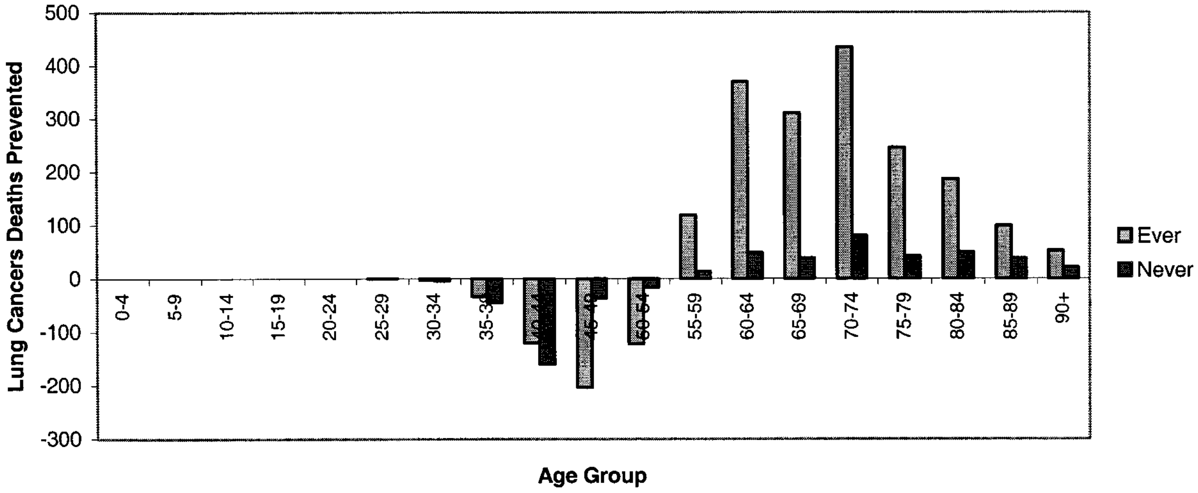
Figure 3-34 presents the decision model's output for lung cancer deaths prevented by year in the Winnipeg scenario. One can observe a small number of periods in which there is a net increase in lung cancer deaths resulting from a reduction in indoor radon concentrations.

FIGURE 3-34. Lung Cancer Deaths Prevented, by Year and Smoking Status, "Winnipeg", (Strategy 2, 800 Bqm^{-3})



Within those periods showing a net decrease in lung cancer deaths, there are a number of age intervals in which there is a large increase. Although the example shown in Figure 3-35 is for the period 40-44 years into the planning horizon, similar effects are seen for almost all years.

FIGURE 3-35. Lung Cancer Deaths Prevented, Years 40-44, By Age and Smoking Status, "Winnipeg", (Strategy 2, 800 Bqm^{-3})



The above effects are not isolated to strategy 2, but are seen to a lesser degree within all strategies.

Note that the occurrence of negative values for lung cancer deaths prevented are not observed under any circumstances when the decision model is implemented with indoor radon concentrations distributions as specified for either Canada or Sudbury. This includes all simulations reported for the sensitivity analyses within this section.

Analysis of the implementation of the decision model in the Winnipeg scenario reveals that the above phenomenon can be attributed to two parameters of the BEIR VI risk model employed in Sub-Model 2. One of these parameters describes the inverse dose-rate effect of radon exposure – a factor the BEIR VI committee deem not relevant at the relatively low radon concentrations found within the indoor environment. As such, no further results from the sensitivity analysis for background radon concentration will be presented. Full consideration of will be given to this issue in the discussion section.

CHAPTER 4
DISCUSSION

4.1 SUMMARY OF KEY FINDINGS

In Chapter 1, we summarized a large body of previously published evidence characterizing a possible human health risk from exposure to radon gas and its decay products within the general population. Due to its radioactive nature and presence within the indoor environment, radon-222 constitutes an environmental health hazard of concern. The log-normal distribution of residential radon concentrations within Canadian homes (GM: 11.2 Bqm^{-3}, GSD: 3.9) indicates that a large majority of the population lives in homes with very low concentrations of radon gas, although select homes demonstrate relatively high radon concentrations. The weight of evidence from numerous sources – including animal inhalation studies, molecular biology, comparative dosimetry, as well as epidemiologic investigations within both occupational cohorts and the general population – was shown to be consistent with a small but measurable excess risk for lung cancer resulting from indoor radon exposure. The best risk estimates currently available suggest that approximately 8% of lung cancer mortality within Canadian homeowners is attributable to radon exposure, accounting for about 7% of the burden in ever-smokers and roughly 13.5% in never-smokers [118]. Analysis of the uncertainty in this estimate indicates a possible range of roughly 4% - 14% of the approximately 16,000 lung cancer deaths per year in Canada.

We noted that Canada's current recommended action level for radon mitigation, 800 Bqm^{-3}, is the highest of its kind in the world. Most countries that have established radon guidelines, or international bodies such as the WHO, recommend some form of radon control when concentrations exceed 200-400 Bqm^{-3}. In addition to having a larger impact upon the health of the mitigated population, evidence appears to indicate that intervention at a lower action level comes at a smaller cost per life saved. The evidence from economic evaluations of residential radon mitigation was reviewed in Section 1.6.2, describing the results for studies from Canada, the U.S., and abroad. The current study seeks to build upon this body of evidence, and estimate the costs, health effects, and cost-effectiveness of controlling indoor radon exposure within Canada.

Chapter 2 described the probabilistic decision model upon which our economic evaluation was based. The model consisted of three parts, describing (1) the changes that implementation of a particular mitigation strategy will induce in the average annual radon concentration to which the Canadian population is exposed; (2) how this change in concentration will affect the population's age-specific excess relative risk of lung cancer mortality; and (3) how this change in relative risk will impact the population as it progresses through the planning horizon, specifically with regards to the lung cancer mortality with which it is burdened. Using this decision model, we can estimate both the cost and cost-effectiveness of the radon mitigation programs under study.

In Chapter 3, we presented results from the economic evaluation of 5 comprehensive national strategies to reduce residential radon exposure among members of the general Canadian population, under numerous conditions. The programs were found to be very expensive, with a total net cost ranging between $112 million and $481 million at the current Canadian action level of 800 $Bqm^{-3.}$ Cost estimates were dominated by the cost of locating individual dwellings that exceed the action level (i.e., screening costs), which comprised over 85% of the total net cost.

Lowering the action level from 800 Bqm^{-3} was found to increase both the total cost and cost-effectiveness of radon mitigation. At low to moderate levels of homeowner compliance, the most cost-effective mitigation strategies involved screening all currently existing dwellings within five years and mitigating those exceeding 200 Bqm^{-3}, or the combination of this strategy with the screening and mitigating of all newly constructed homes. With 50% compliance, we estimate that radon mitigation could add one quality-adjusted life year (QALY) to the population's life expectancy for $62,636 (which equates to approximately $509,000 per lung cancer death prevented). As compliance increases to 100%, mitigation of newly constructed dwellings becomes the most cost-effective option, reducing the cost per QALY gained and lung cancer death prevented to $44,491 and $323,764 respectively.

In the majority of circumstances, the mitigation options with the lowest cost per lung cancer death prevented are also the most expensive, with total costs often exceeding $500 million. A number of less costly options do exist, though at a higher cost per lung cancer death prevented. Whether any mitigation strategy is worthwhile, and if so which is the best, is dependent upon how much one is willing to spend and the rate of compliance achieved, and can involve those described above or screening and mitigating dwellings at the point at which they are sold. In the vast majority of cases, the most cost-effective intervention options include an action level of 200 Bqm^{-3}.

A number of factors can influence the attractiveness of residential radon mitigation, in addition to the strategy, action level, and compliance rate as described above. The degree to which society prefers to benefit from monetary and health gains now, rather than later, is one such factor. Applying a higher annual discount rate to the costs and benefits in economic analysis markedly decreases the estimated cost-effectiveness of radon mitigation. As well, lowering the degree to which indoor radon levels are reduced in mitigated dwellings lowers the cost-effectiveness, as the health benefits of mitigation are reduced while costs are remain relatively stable.

The distribution of indoor radon levels within the housing stock will also affect the cost-effectiveness of intervention. Results appear to support the findings of other authors, detailing an increase in cost-effectiveness when risk management efforts are directed towards areas with higher mean radon levels. However, an interesting and unexpected output of the BEIR VI excess relative risk (ERR) model used in the analysis – resulting in a modelled increase in lung cancer mortality in some age groups following mitigation – speaks to the need for caution in the interpretation of these results.

4.2 ANALYSIS AND INTERPRETATION OF RESULTS

4.2.1 Comparison of Results with Previous Findings

As described in Section 1.5.2.1, Létourneau et al. [243] previously provided an economic evaluation of the five national mitigation strategies studied here. They found that the most cost-effective option was to screen and mitigate homes at the point at which they are sold (strategy 4), with a universal screening program for the current housing stock (strategy 1) being the second best option. This contrasts with our findings, which places the universal screening program as the most cost-effective option under most circumstances. Note also that the previous study assumed 100% compliance with the radon guideline. With 100% compliance, we find that a program to screen and mitigate newly constructed dwellings (strategy 3) to be the best intervention option; this was by far the least cost-effective option in the 1992 analysis.

It is likely that a number of differences between the two studies may account for the discrepancy between the results. Four important differences exist.

1. Rate of Sale for Canadian Dwellings. The authors assumed a constant rate of sale for Canadian dwellings of 10% per year, and that the entire existing housing stock would therefore change hands (and hence be screened under strategy 4) within 10 years. This rate of sale is much higher than that modelled here (i.e., 3.3%). Thus, strategy 4 would be expected to reduce average radon concentrations much more rapidly, yielding significantly more time within the planning horizon during which the population would live at lower levels of radon exposure. This would increase the cost-effectiveness of mitigation. A re-analysis of our results for strategy 4 shows a decrease in the cost per QALY gained with an increasing rate of home sale – from $75,160 assuming 50% compliance with an action level of 200 Bqm^{-3} and a 3.3% rate of sale, down to $73,591 per QALY gained when the rate of sale increases to 6%. Note, however, that raising the rate of home sale in our model to even 10% does not allow strategy 4 to approach being the most cost-effective mitigation option.

2. Costs of Mitigating New Dwellings. With 100% compliance, we found the mitigation of newly constructed dwellings to be the most cost-effective option, whereas Létourneau et al. concluded it was the least cost-effective. This difference is explained by a large difference in the estimated cost to mitigate new dwellings. Létourneau et al. assumed strategy 2 would involve mandatory installation of an active sub-slab depressurization (ASD) system within all new dwellings, regardless of the concentration of radon that would eventually be found in its absence. This is an extremely expensive proposition, resulting in estimated total costs on the order of $12 billion dollars. Our approach was to assume homes were constructed such that radon mitigation equipment could later be installed, if screening tests indicate the completed dwelling has an elevated radon concentration. The necessary modifications have a negligible impact upon the costs of construction, and are consistent with current radon-resistant building practices. Our estimated cost of strategy 2 with 100% compliance at 200 Bqm^{-3} is therefore only $214 million, dramatically increasing the cost-effectiveness of this program.

3. Discounting of Health Benefits. In the previous study, the impact of intervention was measured in terms of the reduction in population cumulative exposure, in WLM, which was then converted to lung cancers prevented using a simple linear risk coefficient. Though costs were discounted at 5% per year, the outcomes of mitigation were not adjusted for any time delay in their occurrence. Indeed, the methodology employed appears not to have allowed for any estimation of the time profile of lung cancers prevented. Currently, the majority of authors writing on the economic evaluation of health-related interventions support the idea of health as a resource from which individuals can benefit, and thus one that must be discounted in analyses [230]. Further, Kennedy et al. have shown that adopting different benefit stream profiles results in a range of cost-effectiveness ratios from £14,912 to £52,416 per life-year gained by radon mitigation [265]. Given the significant time delay in the impact of radon remediation upon lung cancer mortality observed in our study, failure to discount those health effects would result in a significant improvement in cost-effectiveness.

4. Length of Planning Horizon. We modelled the impact of radon mitigation for a full 80 years, whereas the planning horizon used in the previous Canadian study was 40 years. While an increased length of follow-up allows for more costs to be incurred, the impact upon lung cancer mortality (i.e., increasing the number of deaths prevented) is more substantial due to the latent period for health effects following a reduction in radon exposure. However, it is difficult to say if, and to what degree, this would have increased the cost-effectiveness of mitigation; with discounting, the additional lung cancers occurring in the latter half of the study period (i.e., 40-80 years from the start of the study) will have a much smaller impact upon the cost-effectiveness ratios than they would in its absence (e.g., 1000 lung cancers deaths prevented in year 79 is equivalent to 26 lung cancers deaths when discounted at 5%).

A number of other studies have provided analyses of the most cost-effective approaches to controlling indoor radon exposure. In the U.S., Marcinowki and Napolitano [248] found a change in the building code to be most efficient option, concurring with our results for 100% compliance. Colgan and Gutiérrez also found the cost per lung cancer prevented to be lower when risk management efforts were directed towards new homes, rather than existing ones (though a higher action level, 400 Bqm^{-3}, was assumed for existing dwellings than for new ones, for which an action level of 200 Bqm^{-3} was used).

Denman et al. [257] found the mitigation of new dwellings to be optimal when compliance was low (i.e., 10%), but with compliance rates of 20% or greater a universal screening program was the better choice. This contradicts the results presented in Table 3-3. However, as with the earlier Canadian study, numerous differences in methodology limit comparability with our results. These limits include: (1) higher background radon concentrations in the area in which mitigation was performed, which are likely to increase the measured cost-effectiveness of all strategies; (2) higher annual discount rates, shown here to decrease the cost-effectiveness of all strategies, but in particular intervention within new homes as seen in **Figure** 3-22; (3) a different methodology of radon-resistant construction than was studied in this thesis, employing the installation of a low-cost membrane within the sub-structure of the dwelling. Numerous studies have shown active sub-soil depressurization (ASD) to be the most cost-effective radon reduction technology (see, for example, Proskiw [242]).

The majority of previous studies support our finding of increased cost-effectiveness with a decreased action level, and with increased homeowner compliance. Most also found an improvement of cost-effectiveness when risk management efforts were directed towards areas with higher average radon concentrations. We attempted a similar analysis, modelling the change in cost-effectiveness of mitigation if Canada had a radon distribution with higher average levels, as observed in Sudbury and Winnipeg (the latter containing some of the highest levels in

North America). Our results generally appear to concur with the finding of increased cost-effectiveness. This is not unexpected, as mitigation within areas with high radon concentrations can lead to notable decreases in radon (as shown in Figure 4-1), greatly increasing the health impact of mitigation relative to the increase in costs.

However, as described in Section 3.3.4, our decision model predicts an increase in lung cancer mortality within select age-period groups following mitigation within Winnipeg, relative to what would have occurred in the absence of mitigation. This gives cause for concern regarding the validity of our model for this specific analysis.

In our model, implementation of a mitigation program reduces the average radon concentration within the housing stock, and thus the average rate of radon exposure within the population (see Figure 3.3). A reduced rate of exposure will impact age-specific ERR for lung cancer mortality, as calculated by the BEIR VI *exposure-age-concentration* model (i.e. SubModel 2), via two parameters (see equation 8 in Section 2.4.2.3). One of these, $w(\Delta t)_{x,t}$, accounts for cumulative exposure (i.e. the product of exposure rate and time – see Section 2.4.2.1), while the other, γ_w, models the inverse-dose rate effect. In the BEIR VI model, lower dose-rates are associated with increased risk. This latter parameter is the primary driver of the modelled increase in lung cancer mortality following mitigation in the modelled 'Winnipeg'.

Consider a hypothetical cohort '*c*' comprised of individuals in Winnipeg who are within any 5-year age-group x at time t. As the average radon concentration within Winnipeg begins to fall following implementation of the radon control program, the average cumulative exposure that has been incurred by this group at time t is less than what it would have been in the absence of mitigation. That is, $w(\Delta t)_{x,t}^{mitigated} < w(\Delta t)_{x,t}^{unmitigated}$. As dose-rate is modelled in Sub-Model 2 as simply the average exposure rate that has been experienced by cohort '*c*' – estimated by dividing cumulative exposure by age (see Section 2.4.2.3) – it follows that the estimated average dose-rate for the group is also less under mitigated conditions at any time '*t*'. But whereas decreasing cumulative exposure $w(\Delta t)_{x,t}$ results in a proportionate decrease in ERR, decreasing the dose-rate can result in an increase in γ_w, and hence ERR, that is not linear [118].

Our analysis indicates that the modelled age-period-specific increases in lung cancer mortality within the mitigated scenario (relative to the unmitigated state) results when, for that particular age-period group, the decrease in cumulative exposure between the unmitigated and mitigated scenarios is less than the increase in the γ_w parameter that results from the difference in lifetime average exposure rates. The product of the two parameters – and hence ERR – is therefore larger in the mitigated scenario. Note that re-running the simulation without the inverse dose-rate effect (i.e. $\gamma_w = 1$) eliminates any modelled increase in lung cancer mortality following mitigation.

The BEIR VI committee concluded that the inverse dose-rate effect is important at the high average exposure rates present in mining cohorts, but of little importance at the relatively low radon concentrations observed within the general population. The reason was that biological evidence indicates that the effect results from multiple alpha particles traversing the nucleus of the same lung cell – a rare occurrence at low environmental radon levels.

Though the average radon concentrations within Winnipeg are significantly higher than within the majority of dwellings in Canada, and thus more closely resemble those observed within the occupational environment, the conclusions of the BEIR VI report regarding the absence of protraction-enhancement effects in the general population provides sufficient reason to question the validity of our model in the specific 'Winnipeg' analysis. Still, the general trends we observed are consistent with the findings of numerous other authors – targeting radon mitigation strategies towards areas of high risk appears to decrease the cost per life saved.

4.2.2 Informing Risk Management Decision-Making

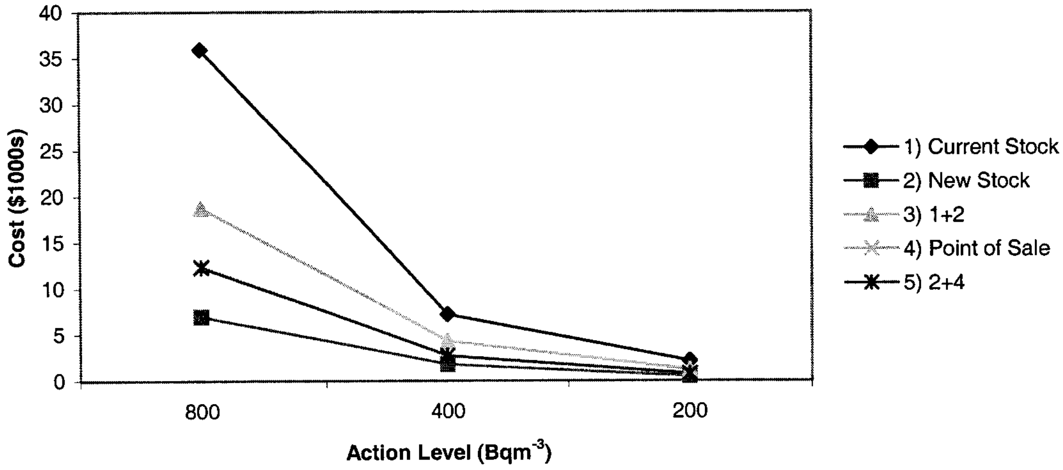
The weight of evidence suggests that radon is an important public health issue, possibly responsible for some 1,500 deaths in Canada each year. Although one cannot ever completely eliminate radon exposure in homes, the technology currently exists to reduce the level of that exposure to a minimum. But is this worthwhile? The best information currently available suggests that there is no level of radon that is "safe". Indeed, the BEIR VI committee found that over two-thirds of radon-induced lung cancer mortality was likely attributable to prolonged exposure at levels below 148 Bqm^{-3}[2].

Canada's approach to managing the risks from radon exposure is currently being reviewed by the federal department of health [310]. This includes a review of the government's recommended action level for radon mitigation, currently set at 800 Bqm^{-3}. The current analysis provides decision-makers with information that could aid in this process. Importantly, this information aims to: (1) illustrate the most cost-effective strategy to reduce indoor radon exposure; (2) demonstrate factors with the potential to increase or decrease that cost-effectiveness; (3) facilitate consideration of whether indoor radon mitigation should be undertaken at all, given (a) the amount society is willing to spend to increase health, and (b) alternative means of doing so.

4.2.2.1 Comparing Radon Mitigation Strategies

From the results presented in Chapter 3, it seems clear that lowering the Canadian recommended action level from 800 Bqm^{-3} would enhance the impact of any resources expended on managing the risks from residential radon. All things being equal, lowering the guideline would result in a larger number of homes being mitigated for the same amount of money spent on screening – the predominant cost component of any risk mitigation strategy. This is illustrated in Figure 4-2.

FIGURE 4-2. Initial Screening Cost per "At-Risk" Home Identified, by Mitigation Strategy



Results also indicate that compliance with the guideline will be a key factor in determining the success of any risk management strategy – one that may present quite a challenge. A survey of radon reduction behaviour in the United Kingdom indicates that the rate of citizen compliance with the government recommended action level is generally around 11% [311]. Such compliance levels would severely compromise the cost-effectiveness of radon mitigation within Canada, as shown in Table 3-4. Yet current rates of compliance within this country are likely to be similar to that figure – a fact supported by Spiegel and Krewski's [234] finding that a compliance rate of 50% could be achieved only when the homes to be mitigated had an average annual radon concentration in excess of 1,200 Bqm^{-3}. Thus, any intervention strategy employing a voluntary radon guideline should include an informational / educational component, focusing on improving the rate at which Canadians follow that guideline.

It is important that efforts to remediate dwellings with elevated radon levels focus on maximizing the reduction in concentration achieved. The majority of analyses presented here assumed a reduction of pre-mitigation radon levels by 90% – a reduction that is quite achievable with the installation of an active sub-slab depressurization (ASD) system. It was shown in Chapter 3 that decreasing the effectiveness of radon reduction technologies has important detrimental effects upon the cost-effectiveness of the intervention.

A key finding from Chapter 3 was that, in most cases, the most cost-effective means of controlling the health risks from residential radon was also the most costly. For example, with 50% compliance, the most cost-effective mitigation strategy was universal screening of the existing housing stock with an action level of 200 Bqm^{-3}, at a total cost of $611 million. Though the majority of that cost would likely be borne by the individual homeowner, this total cost figure may appear excessive to decision-makers.

Our results can help guide choices of radon control strategies when it is deemed necessary to restrict total costs. For example, Table 4-1 identifies the most cost effective strategies with total costs under $500 million, and under $250 million.

TABLE 4-1. Incremental Cost-Effectiveness Analysis, 50% Compliance, with Cost Restrictions

Rank[a] (C.E.)	Strategy	AL[a] (Bqm^{-3})	Cost ($Milions)	Health Effects		Incremental Cost per...	
				LCD[a] Prevented	QALYs[b] Gained	LCD Prevented	QALY Gained
1	1	200	$661	1,298	10,546	$509,082	$62.636
2	3	200	$801	1,557	12,379	$540,820	$76,615
			A) Restriction of Total Cost to < $500 Million				
1	5	200	$411	730	5,549	$563,371	$74,122
			B) Restriction of Total Cost to < $250 Million				
1	2	200	$155	260	1,832	$596,632	$84,516

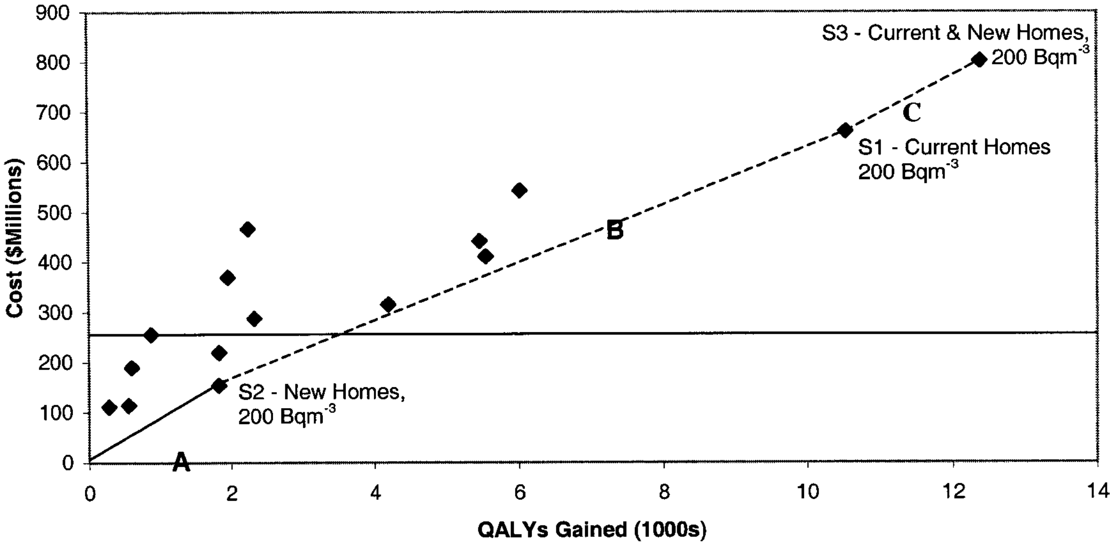
[a] Rank in terms of Cost-Effectiveness; Note that only those options that are (1) most cost-effective, or (2) more effective than the most cost-effective option are presented.
[b] Action Level; [c] Lung Cancer Death; [d] Quality-adjusted Life-Years

Thus, if one wished to restrict costs to below $500 million, the most cost-effective option would be strategy 5 (i.e., screen all homes at the point of sale, and screen all new constructions), again with the action level being 200 Bqm^{-3}. This option would cost approximately $74,122 per QALY gained, which is less cost-effective than the $62,636 per QALY that could be achieved with no cost restrictions. However, this option saves $250 million in total costs. Note that the most cost-effective radon program with total net costs of less than $250 million is strategy 2 (i.e., new constructions only) at 200 Bqm^{-3}, with average costs of $84,516 per QALY gained.

As resources are scarce, decision-makers must factor total costs into their deliberations regarding indoor radon control, as well as consider how best to maximize the impact of the any dollars spent. However, it is important that they realize that restriction of the total costs of the program carries with it an implicit, seemingly counter-intuitive choice – described below by examining the incremental cost-effectiveness of moving to more costly alternatives.

Figure 4-3 again presents the cost-effectiveness plan for mitigation with 50% compliance, with a horizontal line indicating that costs are restricted to below $250 million.

FIGURE 4-3. Cost-Effectiveness Plane, 50% Compliance, With Cost Restrictions



Slope A represents the cost effectiveness of the optimal choice under those conditions (i.e., strategy 2 at 200 Bqm^{-3}), while slopes B and C represent the incremental cost-effectiveness of choosing instead to move to more costly (and more cost-effective) alternatives. The results from the analysis are shown in Table 4-2.

TABLE 4-2. Incremental Cost-Effectiveness of Removing Cost Restriction of $250 Million, 50% Compliance

Increment	Strategy	AL[a] (Bqm^{-3})	Cost ($Milions)	Health Effects		Incremental Cost per...	
				LCD[b] Prevented	QALYs [c] Gained	LCD Prevented	QALY Gained
A	5	200	$155	260	1832	$596,632	$84,516
B	1	200	$661	1298	10546	$487,188	$58,034
C	3	200	$801	1557	12379	$540,820	$76,615

[a] Action Level; [b] Lung Cancer Death; [c] Quality-adjusted Life-Years

Thus, in choosing to restrict costs to under $250 million and implement strategy 5 with an action level of 200 Bqm^{-3}, decision-makers would implicitly be stating that spending $84,516 to gain one QALY is an acceptable investment. If that is indeed the case, it may be difficult to justify not instead employing strategy 1, which would require only $58,034 for every additional QALY gained – a significantly better return on investment.

4.2.2.2 Deciding to Implement a Radon Mitigation Program

If the object of mitigation is to maximize the net health benefit from a given investment, incremental cost-effectiveness analysis plainly shows the optimal choice is strategy 1 at 200 Bqm^{-3}, assuming 50% compliance. The cost of this option is $62,636 per QALY gained. The question now becomes: *is $62,626 per QALY gained 'cost-effective'?* Clearly, this figure is *more* cost-effective than alternative mitigation strategies under identical conditions. However, the usefulness of this result to decision-makers who are trying to decide whether or not they should implement such a program is precluded in the absence of some framework in which to interpret it. That is, one must determine what society is willing to pay for a QALY, and if it is more than the $62,626 cost associated with this risk management option.

Such decisions go beyond the scope of this thesis. However, we might move closer to an answer through examination of work published elsewhere. Laupacis et al. [312] describe 5 *'grades of recommendation'* for the adoption and utilization of new health-related technologies. Of most interest are their grades B through D, concerning interventions that are both more costly and more effective than current practice. They put forth the idea that a cost-effectiveness ratio of less than $20,000 per QALY gained provides *strong* evidence for adoption and appropriate utilization, while a range of $20,000 - $100,000 per QALY gained offers *moderate* evidence in support of same; a ratio in excess of $100,000 per QALY is said to provide *weak* evidence. Under this classification, the results reported above can be thought of as moderate evidence for implementation of the program. Though again, the usefulness of this declaration is questionable.

It is possible to gain insight into the value society places on a QALY by examining the cost-effectiveness of other life-saving interventions that have been adopted. There is enormous variation in the cost-effectiveness ratios observed, from less than $0 (i.e., interventions that both save money and are more effective, such as childhood immunization) to many millions of dollars per life-year gained [313, 314]. Our estimated ratio of roughly $62,000 per QALY gained is approximately equal to that those found previously for annual screening for cervical cancer using pap smears in women aged 65+ [314], or for the addition of passenger-side airbags (in addition to the driver's-side) to automobiles [315]. It is significant to note that a review of 124 studies detailing the cost-effectiveness of programs to control environmental toxins found a median cost per life-year saved of $4.2 million [316]. Clearly, compared to other environmental remediation programs, the control of indoor radon is remarkably cost-effective.

The definition of a value of a QALY is a question yet to be answered, but without this one cannot state for certain if $62,636 per QALY gained is an appropriate investment in and of itself. One is limited to comparing the cost-effectiveness of different interventions, as is done above, and from there attempting to decide the most appropriate 'suite' of interventions in which to invest available resources. As radon mitigation seeks to reduce lung cancer mortality, such an analysis would not be complete without consideration of smoking cessation. Recall a key finding from Chapter 3: that the majority (84%) of reduced lung cancer mortality will likely occur within the ever-smoking population, regardless of the strategy or action level employed.

Smoking is clearly the most important source of lung cancer risk, with relative risks of ever-smoking ranging from 3 to 20 (see Figure 2-10), compared to RRs on the order of 1.12 for radon exposure [118]. Efforts to decrease the prevalence of tobacco use therefore have the potential for a far greater impact upon lung cancer mortality than radon remediation. By way of example, in Table 3-3 we reported that given 100% compliance with the most comprehensive radon mitigation strategy (i.e., screen and mitigate all existing and newly constructed dwellings with a 200 Bqm^{-3} action level), one could reduce lung cancer mortality by 3,097 deaths (discounted at 5%) over 80 years, and gain 24,601 QALYs in the process. Reconfiguring our decision model to incorporate a 5% reduction in the prevalence of ever-smoking at all ages yields an estimate discounted savings in lung cancer mortality of 25,398 deaths (undiscounted: 107,631 deaths) over the same 80-year period, yielding 643,829 additional discounted QALYs.

It is important to note that as radon-related lung cancer risks are modelled here as a function of baseline risk, a reduction in smoking would eliminate a proportion of the lung cancer burden that would have been attributable to radon exposure.

Further, unlike radon, tobacco use is associated with many cancers in addition to lung cancer, and also makes significant contributions to the burden of cardiovascular and respiratory disease within the population. Thus, a decrease in the prevalence of smoking will lead to substantial population health benefits in addition to decreased lung cancer mortality, as modelled in our brief example, above. This will further increase the cost-effectiveness of anti-smoking programs compared to radon mitigation. Note that a review of 6 economic evaluations of smoking cessation programs found cost-effectiveness ratios within the range of ≤$0 to $13,000 per life-year gained [317] – far more cost-effective than we have estimated for even the most efficient radon reduction programs (e.g. $46,959 per QALY).

4.3 REVIEW AND CRITIQUE OF METHODS

4.3.1 Strengths and Value-Added Contributions

The methods used in this thesis represent a significant improvement in the means by which an economic evaluation of residential radon mitigation in Canada may be performed. Specific strengths of our methodology include the following.

4.3.1.1 BEIR VI Model of Excess Relative Risk

We calculate excess risks of lung cancer mortality on an age-specific basis using a model preferred by the BEIR VI committee – the first economic analysis of radon mitigation to do so. This model, derived from the combined analysis of 11 seven large occupational cohort studies capturing 2,674 lung cancer deaths and 888,906 exposed person-years – represents the best epidemiologic information currently available for the prediction of risks associated with a given level of radon exposure.

4.3.1.2 Uncertainty Distributions for Model Input Parameters

Excess risk calculations are not based upon point estimates for each model parameter, but rather upon probability distributions representing the range and likelihood of all plausible values the parameters might take, as defined by the BEIR VI Committee. A similar distribution is defined to represent uncertainty and variability within indoor radon concentrations in Canada. Monte Carlo simulation may be used to propagate this uncertainty through to model outputs, as is done in our pilot uncertainty analysis in Section 4.4.3, below. These techniques may improve risk management decision making, as any decision based the probabilistic results can take into account the resident uncertainty inherent within our risk estimates.

4.3.1.3 Stratification by Age, Period, Gender, and Smoking Status

The majority of previous economic analyses of radon mitigation estimate a savings in total population radon dose achieved by the intervention, and then translate this dose savings into a number of lung cancer deaths prevented using a simple linear dose-response coefficient (e.g., 10^{-4} lung cancers per WLM). We have employed a much more detailed approach to modelling the impacts of intervention on risk, by (1) tracking the impact of reduced radon concentrations upon the average cumulative exposure incurred by individuals within different age groups; (This is significant, as the importance of reduced exposure rates in the determination of risk is likely diminished within older individuals whom have already received a large proportion of their lifetime cumulative radon exposure at the higher rates.); (2) tracking the gradual reduction of average indoor concentrations over time, and the associated gradual reduction in excess risk; (The observable impact of mitigation on health will change over time on account of an increasing number of mitigated homes and the latent period for the induction of lung cancer by radon. Modelling these time-related benefit streams avoids the need to assume some distribution of lung cancers prevented over time, as is done in other economic analyses [265]); (3) modelling the Canadian population as an age-period-gender-smoking cohort; (Health impacts are estimated based upon data regarding the age-, gender- and smoking-distribution of the Canadian population, its fertility and net migration rates, as well as the group- and period-specific mortality rates calculated as a function of declining radon levels. The housing stock, and thus costs of mitigation, is modelled with consideration of the rate at which the population will grow). As a consequence of these enhancements, our analysis likely provides a much closer representation of the expected impacts of mitigation within Canada than could be obtained via methods used in previous analyses.

4.3.2 Limitations

The decision model used in this analysis, as with any model, is a simplified representation of a complex reality. One cannot possibly capture the intricate interaction of all variables within a free-standing population upon which the outcome of interest is dependent. Choices must be made. Thus, it is important to give consideration to the following assumptions upon which the results of this analysis depend to varying degrees.

4.3.2.1 BEIR VI Assumptions

Calculations of excess risk from radon exposure are based upon a risk model derived from epidemiologic analysis of miner cohorts that differed substantially from the general population in many respects.

Levels of Exposure. The miner cohorts were generally subject to significantly higher doses, at significantly higher dose rates, than is seen in the population at large. Extrapolation of risks downward from occupational to environmental radon levels therefore requires an assumption about the shape of the dose-response curve as one approaches zero exposure. The BEIR VI risk model assumes the curve to be linear at low doses, with no threshold

Given that different shaped risk models have been shown to result in widely varying estimates of risk [318], and also that nearly the entire population is subjected to exposure within the low, linear range, any deviation of the true dose-response curve from the linear hypothesis could result in under- or over-estimation of population risk, depending of the direction of the deviation (i.e., a true sub-linear model would lead to an over-estimation, and vice versa). Note, however, that any difference between the assumed slope of the linear dose-response curve and the true one, assuming that it is in fact linear, would not introduce a significant amount of error into this analysis.

The existence of a threshold would lead to over-estimation of risk, the size of which is dependent on the threshold concentration. This has been clearly demonstrated by Brand et al.,[118] who estimate a 15% reduction in population lung cancer risks associated with residential radon exposure given a threshold average concentration of 13 Bqm^{-3}, but an 80% reduction if that threshold was as high as 128 Bqm^{-3}.

Note the linear no-threshold assumption for the radon dose-response curve is supported by consideration of the biological mechanism by which radon exposure causes cancer. Note also that the results of BEIR VI analyses restricted to miners with the lowest levels of cumulative exposure are consistent with the linear-no threshold hypothesis [2]. Further, the results of the combined analysis of residential case control studies are also consistent with this linear extrapolation of the miner data to residential exposure levels [19].

Age and Gender. Occupational miner cohorts are typically younger than the general population, and were predominantly male. One must therefore assume when using the BEIR VI model that the ERR per exposure does not vary by age at exposure, nor with gender. A gender-effect is possible, given differences in breathing rates and volume between the sexes, which might lead to a larger radiation dose per exposure in males. As the large majority of miner cohorts were male, such an effect may have lead us to over-estimate the risk of radon exposure within the general population. The absence of an effect with age at exposure contrasts with the results of studies for other types of radiation and cancers (e.g., atomic bomb cohorts), though the differences between alpha particle and gamma radiation may explain this difference [2]. The BEIR VI analysis did not reveal any effect with age at exposure, though information was limited for persons less than 20 years old. Clearly, an existing interaction between ERR and these variables could bias our results in any number of ways.

Interaction between Radon and Smoking. The BEIR VI Committee observed a sub-multiplicative interaction between tobacco use and radon exposure in the determination of lung cancer risks, with the ratio of ERR to exposure in never-smokers approximately twice that estimated for ever-smokers. Our model reflects this observation. The committee acknowledges data limitations in making this assumption [2]. Any difference in the true nature of the joint effect from that assumed here would impact the validity of our results. If the relation is, in fact, additive, we will have under-estimated ERR in ever-smokers, and thus also likely under-estimated the impact of radon mitigation on population lung cancer mortality. In contrast a fully multiplicative joint effect will have lead to an upward bias in our estimates of health impacts.

Lung Dosimetry. The risk model assumes there to be little difference in the deposition of radon and its progeny within the lung between miners and the population (i.e., K=1.0), though there may be differences in breathing patterns or the equilibrium fraction of radon with its progeny. Higher rates of respiration (by miners) would increase their risk, and thus lead us to over-estimate the risks within the general population. The same effect would be seen with an increased fraction of radon progeny within mines. Note that uncertainty in the "K-factor" is acknowledged within the model during the Monte Carlo simulation, diminishing the impact of this variable upon the validity of our results.

Other Differences. There are a number of other differences between the indoor and mining environments (e.g., exposure to other carcinogens, such as diesel fumes and silica). The BEIR VI Committee assumed there to be no interactive relationship between these other differences and the estimated ERR per WLM of radon exposure. If there was in fact a synergistic relationship with some mine exposure, our model would over-estimate the risks associated with radon in the general population and would bias the estimated cost-effectiveness ratios downwards (i.e., making them appear more attractive).

4.3.2.2 Modelling Population Averages Rather Than Individuals

We did not attempt to model individuals within the population, but focused rather on population averages. Excess risk calculations were based upon the average radon concentration within the modelled housing stock, by year. This requires the assumption that distribution of exposure rates in the population is equal to that seen in Canadian dwellings (i.e., the population is randomly allocated to the existing housing stock). We then estimated ERR for an average member of each age group on a smoking-specific basis, and assumed that all members within that group have the same baseline risk of mortality. These concessions make the modelling process much simpler. However, they also involved the assumption that $E(f(c))=f(E(c))$, where f denotes the function implemented in ERR model implemented in Sub-Model 2, c represents radon concentration, and E is the expectation operator. Analysis indicates that age-specific ERR estimates based upon an averaged population radon exposure, $f(E(c))$, are approximately 26% higher than those based calculated when concentration is allowed to vary among individuals, $E(f(c))$. Thus, we have likely overestimated the impact of radon mitigation upon lung cancer mortality within Canada by some degree, though the 'cohort' nature of the decision model makes it difficult to estimate precisely how much error has been introduced. The consistency of our results in terms of health impact of mitigation with other estimates (e.g. BEIR VI [2] and Brand et al. [118]) indicate that this error may be relatively small.

A similar assumption required to implement Sub-Model 3 (i.e. $E(g(e_x)) = g(E(e_x))$, where g represents the function estimating the health impacts of reduced age-specific ERR for lung cancer mortality, e_x, and E is again the expectation operator) was shown to introduce a negligible amount of error into the model (e.g. on the order of a 1% difference).

4.3.2.3 Constancy of Rates within the Population and Housing Stock

We have assumed that a number of rates pertaining to both the population (e.g., baseline mortality rates; fertility rates; number of immigrants; emigration rate; prevalence of never-smoking; age and gender-specific quality of life; health impacts of lung cancer, and the cost to treat them) and the housing stock (i.e., rate of sale) will remain constant throughout the planning horizon. Small fluctuations in most rates are likely to occur, introducing a small amount of error into our analysis. As was indicated in Section 4.2.1, a change in the assumed rate of sale for dwellings from 3.3% to 10% had very little impact on the estimated cost-effectiveness of mitigation, and would not change the decisions made regarding choice of strategy in any way.

Perhaps the greatest source of potential error from these assumptions is with regards to smoking. Age and gender-specific rates of smoking prevalence are assumed to be constant within the Canadian population. However, recent trends indicate a decline in tobacco use within

Canada [319]. As described previously, even a small decrease in smoking rates over the planning horizon would have important impacts upon lung cancer mortality. Declining baseline risks of lung cancer could significantly decrease the impact – and hence the cost-effectiveness – of radon mitigation, leading us to have over-estimated the attractiveness of these programs.

Given the amount of evidence currently available regarding observed temporal trends in smoking and related trends in lung cancer incidence and mortality, it likely would have been possible to incorporate these trends in the decision model with a fair degree of accuracy in the short term. However, the prolonged nature of the planning horizon used in this analysis, and the increasing uncertainty that would surround smoking and lung cancer projections that approach 80-years, lead to the possibility that such projections might introduce a larger degree of error than has come from the assumption of constancy. This, combined with the relative computational simplicity of the later assumption, made not including temporal trends in smoking and cancer the appropriate choice for this analysis.

4.3.2.4 Choice of Discount Rates for Cost and Health Benefits

In this analysis, we discounted the costs associated with radon mitigation, as well as its associated population health benefits (measured in terms of either lung cancer deaths prevented or QALYs gained), at an equal, constant discount rate of 5% per year. Discounting is a well established practice within health economics, generally supported by two principle arguments: (1) *Opportunity Costs* – i.e., deferring expenditure (or conversely, advancing income) provides the opportunity to use resources in some other productive way; and (2) *Time Preference* – i.e., individually and as a society, we generally prefer to delay expenditures, and to incur benefits earlier, whenever possible [320].

Our use of the annual discount rate of 5% is intended to reflect the actual positive time preference expressed by society. Though a constant 5% rate is currently the most widely employed discounting method in health economics, it may not be an accurate representation of societal preferences. Any change in this rate (e.g., a move to the rate of 6% currently recommended by the U.K. Government) will impact the attractiveness of mitigation, with an increase in discount rate decreasing the apparent cost-effectiveness, as seen in our results. Use of a more complex, time-dependent discount rate could have a number of different impacts on our results, though society is far less likely to express a non-constant discount rate than are individuals [321].

The choice to discount health benefits at a rate that is equal to that applied to cost streams is somewhat controversial in nature, though this (followed by sensitivity analysis) is the practice most often recommended by authors [230]. Again, this may not accurately reflect societal time preference for costs and health benefits. A number of authors have proposed that health benefits are more appropriately discounted at a rate that is less than costs, or even at a rate of 0% (i.e., no discounting) [321-323]. They make a number of arguments, perhaps the most important of which, for our purposes, is the significant bias that using equivalent discount rates for costs and benefits introduces against prevention programs, such as residential radon mitigation [320]. An interesting counter argument has been put forth by Keeler and Cretin, showing that the practice of discounting benefits at a rate less than that applied to costs results in the ability to *always* improve the cost-effectiveness of intervention by delaying implementation by one year [324].

The choice of an appropriate discount rate discount rate which accurately reflects societal preferences for time-dependent for costs and health benefits will depend upon a number of variables, including (1) the degree to which health is viewed as a "tradeable good" (i.e., as merely a means to gain other benefits, such as the ability to work and gain financial rewards); (2) the uncertainty surrounding future expenditures and benefits, and the individual's aversion to risk; and (3) the extent to which the marginal utility of future monetary and health benefits decline at different rates with increasing wealth and population health, respectively [325].

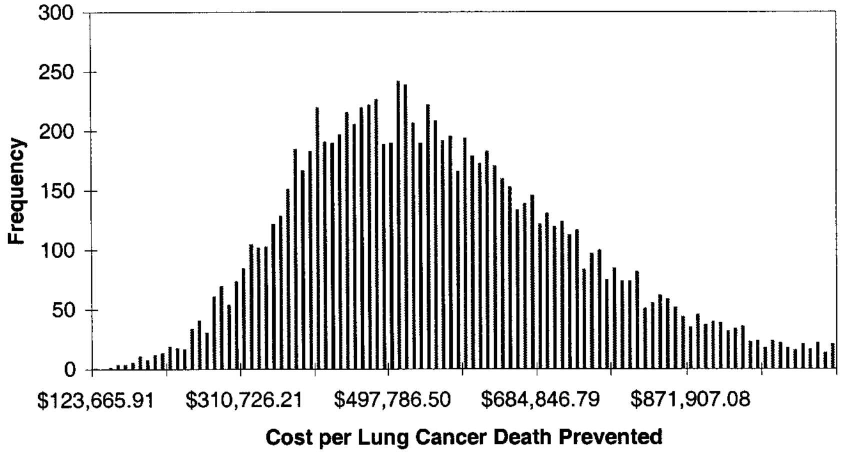
While not fully explored in our sensitivity analysis, it is clear that any change in our methodology that reduces the annual discount rate applied to health benefits below that used for cost streams would greatly improve the cost-effectiveness ratios within our results. .

4.3.3 The Next Step – Probabilistic Analysis of Uncertainty

The present analysis provides point estimates for the cost-effectiveness of residential radon mitigation within Canada. However, the probabilistic techniques used in our decision model allow for the generation of uncertainty distributions for those estimates. This type of analysis can greatly inform risk management decision-making by providing not only the most likely value, but also the range of possible values with an estimate of the probability of the occurrence of each. We present an illustration below for each of the 5 mitigation strategies with an action level of 200 Bqm^{-3}, assuming 50% homeowner compliance, 90% reduction in radon dwellings, and using a 5% annual discount rate for costs and health benefits.

Figure 4-4, below, presents the range in possible values in the cost per lung cancer death prevented by mitigation strategy 1 (i.e., screen all current homes) under these conditions, following 10,000 Monte Carlo trials.

FIGURE 4-4. Uncertainty Distribution, Cost per Lung Cancer Death Prevented, (Strategy 1; Action Level: 200 Bqm^{-3}; 10,000 Trials)



From the figure, it is evident that there is a large degree of uncertainty within the decision-model's estimate for the cost per lung cancer death prevented under these conditions, and that the uncertainty is distributed approximately symmetrically around the central value of about $130,000. The result is a range of possible values for the cost-effectiveness ratio, from about -$10,000 to about $558,000 (0% and 100% percentiles, respectively – not displayed in the

Figure), though the range in which the bulk of the probability density function falls is much more narrow (e.g., 5% and 95% percentiles are $58,000 and $270,000, respectively).

A similar degree of uncertainty is evident for the four other strategies, as seen in Figures 4-5 through 4-8, below.

FIGURE 4-5. Uncertainty Distribution, Cost per Lung Cancer Death Prevented, (Strategy 2; Action Level: 200 Bqm^{-3}; 10,000 Trials)

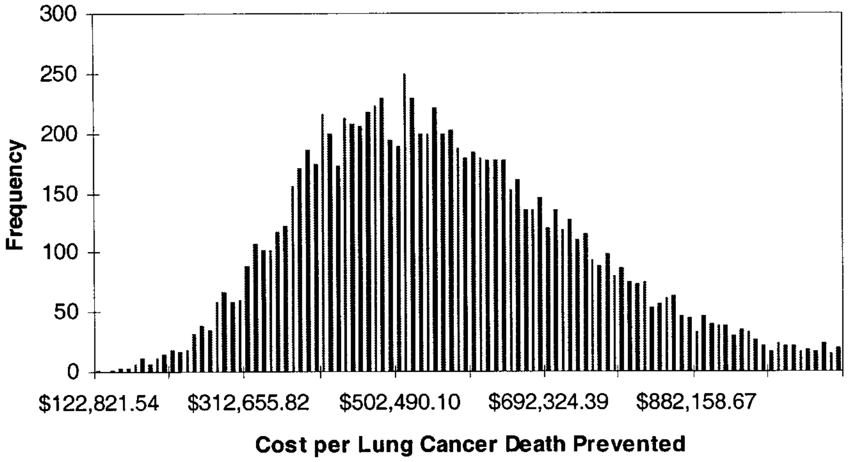


FIGURE 4-6. Uncertainty Distribution, Cost per Lung Cancer Death Prevented, (Strategy 3; Action Level: 200 Bqm^{-3}; 10,000 Trials)

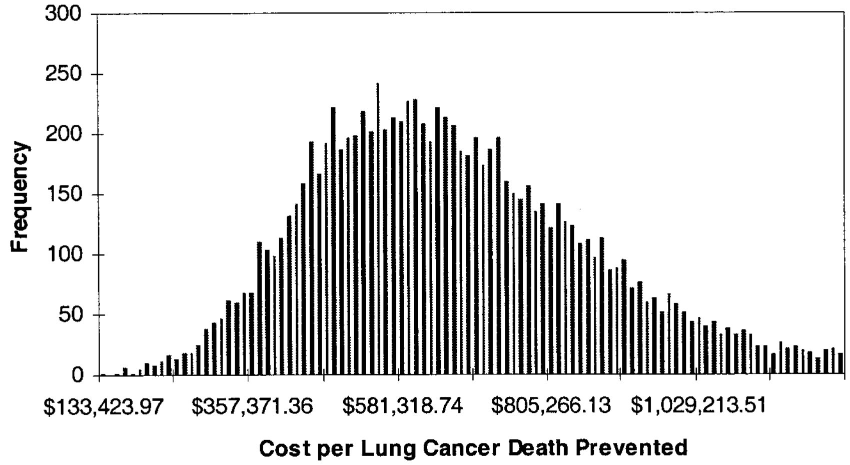


FIGURE 4-7. Uncertainty Distribution, Cost per Lung Cancer Death Prevented, (Strategy 4; Action Level: 200 Bqm^{-3}; 10,000 Trials)

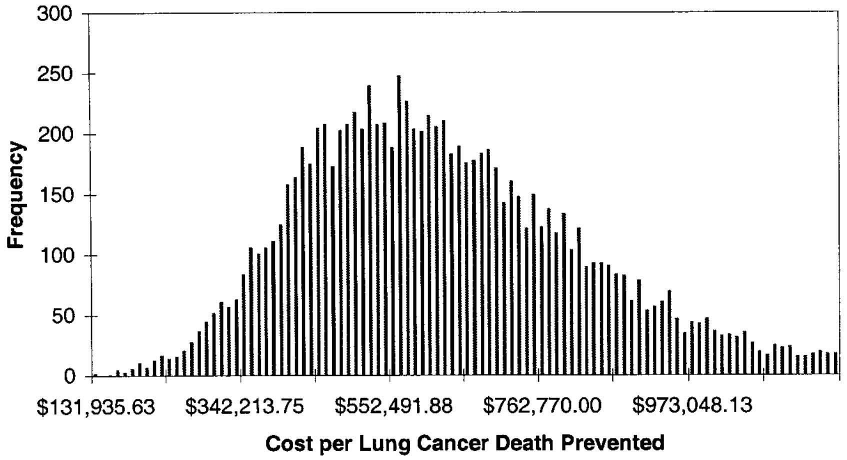
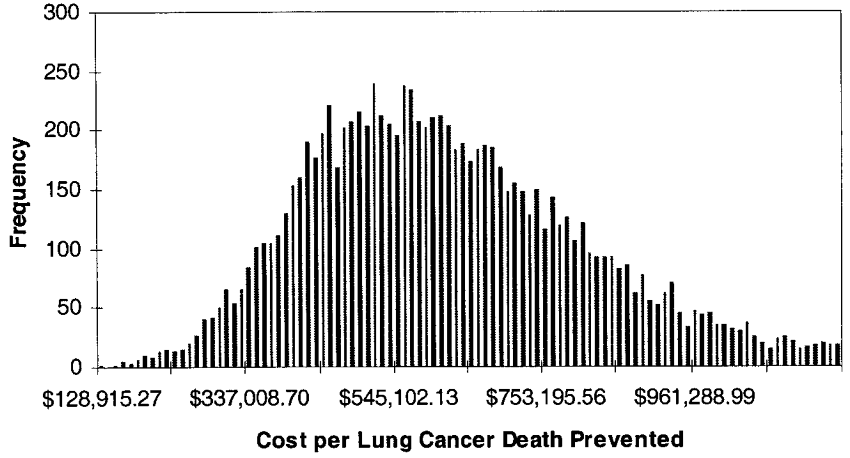


FIGURE 4-8. Uncertainty Distribution, Cost per Lung Cancer Death Prevented, (Strategy 5; Action Level: 200 Bqm^{-3}; 10,000 Trials)



Tables 4-2 and 4-3, present percentiles describing the uncertainty distributions for the cost per lung cancer death prevented and cost per QALY gained, respectively, for each of the 5 radon mitigation strategies considered in this thesis.

TABLE 4-3. Percentiles of Uncertainty Distributions, Cost per Lung Cancer Death Prevented, by Mitigation Strategy (Action Level: 200 Bqm^{-3})

Mitigation Strategy	Distribution Percentiles ($1,000s per Lung Cancer Death Prevented)					Mean
	2.5%	5%	50%	95%	97.5%	
1) Current Stock	$296	$337	$596	$1,009	$1,113	$566
2) New Stock	$315	$359	$637	$1,082	$1,192	$667
3) 1 + 2	$274	$310	$547	$925	$1,021	$573
4) Point of Sale	$301	$342	$603	$1,021	$1,125	$632
5) 2+4	$296	$337	$596	$1,009	$1,113	$624

TABLE 4-4. Percentiles of Uncertainty Distributions, Cost per Quality-Adjusted Life Year Gained, by Mitigation Strategy (Action Level: 200 Bqm^{-3})

Mitigation Strategy	Distribution Percentiles ($1,000s per Lung Cancer Death Prevented)					Mean
	2.5%	5%	50%	95%	97.5%	
1) Current Stock	$36	$39	$66	$108	$118	$68
2) New Stock	$48	$53	$89	$146	$161	$93
3) 1 + 2	$37	$41	$68	$111	$122	$71
4) Point of Sale	$42	$46	$77	$127	$139	$80
5) 2+4	$42	$46	$77	$127	$139	$80

(Note that the appearance of identical values for Mitigation Strategies 4 and 5 in Table 4-4 is an artefact of the rounding used in presenting the table).

Two important observations can be made from Tables 4-2 and 4-3. First, there is clearly substantial uncertainty in the decision-model estimates of cost-effectiveness for each of the five mitigation strategies, limiting the ability of the model to accurately predict what the true cost per health benefit derived from residential radon control would be. Second, Tables 4-2 and 4-3 clearly demonstrate a significant overlap of the uncertainty distributions for the estimated cost-effectiveness ratios. Thus, under this particular set of conditions, the decision model has very little power to differentiate between the 5 radon mitigation strategies in terms of cost-effectiveness, given the uncertainty in the estimates.

This degree of overlap in the uncertainty distributions is not unexpected given that Figure 3-16 (p. 93 in the Results section) demonstrated very little difference in the associated point estimates between the five mitigation strategies. The uncertainty distributions would have to be very 'tight' to allow for statistical discrimination between the strategies at 200 Bqm^{-3}. Note that Figure 3-16 shows considerably greater difference between the average estimates for cost per lung cancer death prevented with an action level of 800 Bqm^{-3}. It is therefore possible that the model would have a greater ability to discriminate between the five mitigation strategies at this action level, even when acknowledging uncertainty within those estimates.

Characterization of even a fairly wide range of uncertainty within cost-effectiveness estimates can be very useful to risk management decision-makers, especially when compared to the utility of point estimates alone. Table 4-2 can tell us a number of things about mitigation Strategy 1 that could help when making a decision with regards to its implementation. For example, the uncertainty distribution indicates that there is only slightly more than a 5% probability of the costs per lung cancer prevented by strategy exceeding $1 million. Note that it is equally unlikely that the costs will be as low as $337,000. Further, there is an equal chance that the cost per lung cancer prevented will be above or below $596,000 (i.e., the 50th percentile).

It would be desirable to decrease the amount of uncertainty within the above cost-effectiveness estimates, wherever possible. To reduce the uncertainty in model outputs, one must reduce uncertainty in the inputs by providing additional and/or improved information. With limited funds available, one might have to make a choice regarding the specific set of model inputs towards which new research will be directed. Note that a formal, 2-dimensional (i.e., inter-individual variability vs. true knowledge uncertainty) analysis of uncertainty in our estimates could identify those uncertain model input(s) having the greatest impact upon the uncertainty within the model outputs, and thus help target research funds towards those areas with the potential to have the greatest impact in terms of reduction of uncertainty within the results.

4.3.4 Other Future Considerations

Our research is limited to consideration of five radon mitigation strategies and three action levels. There are very likely means by which to augment the impact of a national radon program not considered here. These might include the study of possible linkages of radon remediation with smoking cessation programs; analysis of the likely effects, and cost-effectiveness, of various types of educational campaigns aimed at increasing population awareness and action related to the radon problem; consideration of different types of policy mechanisms (e.g., tax deduction of a portion of mitigative work) by which the government might lesson the cost burden on homeowners and increase compliance with the guideline; the derivation of an optimal action level which maximizes the cost-effectiveness of mitigation, which is likely not 200 Bqm^{-3}. Colgan and Guiterrez derived an optimal action level of be 300 Bqm^{-3} in Spain [267]. A similar analysis would be useful to decision-makers in this country.

For simplicity, the present study was limited to the consideration of direct costs, both in terms of costs incurred (e.g., mitigation costs) and averted (e.g., treatment costs). There are additional, indirect costs to society of radon mitigation; for example, an individual may have to stay home from work while mitigation is taking place, resulting in a loss of free time, and possibly wages. Similarly, there are additional financial benefits to society of preventing lung cancers, beyond those gained by averting treatment costs. Further, the prevention of a lung cancer today is likely to general health care costs for the treatment of some later cause of death, be it lung cancer, heart disease, or any other. Future economic analyses of radon mitigation in Canada might consider such costs of a more indirect nature, to the extent possible.

4.4 CONCLUSIONS

The weight of evidence suggests that radon is an important public health issue in Canada – and one that can be addressed. Technology exists to greatly reduce radon concentrations within residences, thus limiting the exposures received by the majority of the population. Of the mitigation strategies considered here, the most cost-effective is the universal screening of the existing housing stock, with the recommendation of mitigation within all dwellings with an average annual concentration exceeding 200 Bqm^{-3}.

The cost-effectiveness of intervention is enhanced by raising the rate of homeowner compliance with the guideline, and also the percent reduction is radon levels achieved within mitigated dwellings. Any radon strategy should therefore seek to maximize these two factors. Note that the unlikely occurrence of homeowner compliance approaching 100% may have implications in the optimal choice of strategy by which to screen homes, as we have shown.

The question of whether or not to implement a national radon control program is more difficult to answer, and will require consideration of what one is willing to pay for a unit of health (e.g., a QALY), and the cost-effectiveness of alternative approaches to managing health risk. Research is underway within both of these areas, and may provide valuable direction to decision-makers within the coming years.

At this point, however, evidence indicates that directing public dollars towards smoking-cessation could achieve a much larger return on investment. Further, as radon-related lung cancer risks are currently modelled as a relative increase from baseline risk, the large reductions in population lung cancer risk that result from even small improvements in societal rates of tobacco use imply that addressing smoking within the population may, in fact, be the very best way to reduce the risks of residential radon exposure in Canada.

REFERENCES

1. Wanner, H.U. 1993. Sources of pollutants in indoor air. *IARC Scientific Publications*, 109:19-30.

2. National Research Council (NRC). 1999. *Committee on the Biological Effects of Ionizing Radiations. Health Effects of Exposure to Radon: BEIR VI.* Washington, D.C.: National Academy Press.

3. International Commission on Radiation Protection (ICRP). 1987. Lung Cancer Risk from Indoor Exposures to Radon Daughters. ICRP Publication 50. *Annals of the ICRP*, 17.

4. National Research Council (NRC). 1991. Panel on Dosimetric Assumptions Affecting the Application of Radon Risk Estimates. Comparative Dosimetry of Radon in Mines and Homes. Washington, D.C.: National Academy Press.

5. International Agency for Research on Cancer (IARC). 1988. *International Agency for Research Monography on the Evaluation of Carcinogenic Risks to Humans. Overall Evaluations of Carcinogenicity: An Updating of IARC Monographs Volumes 1 to 42, Supplement 7.* Lyon: IARC.

6. Canada Mortgage and Housing Corporation (CMHC) and Health Canada. 1997. *Radon: A Guide for Canadian Homeowners.* Ottawa: CMHC and Health Canada.

7. Health Canada. 1995. Exposure Guidelines for Residential Indoor Air Quality: A Report of the Federal-Provincial Advisory Committee on Environmental and Occupational Health. Ottawa, ON: Minister of Supply and Services Canada.

8. Åkerblom, G. 1999. Radon Legislation and National Guidelines. Stockholm: Swedish Radiation Protection Institute and European Commission.

9. Environmental Protection Agency (EPA). 1992. A Citizen's Guide to Radon. The Guide to Protecting Yourself and Your Family from Radon. 3rd Edition. Washington, D.C.: U.S. Government Printing Office.

10. International Commission on Radiological Protection (ICRP). 1993. Protection against radon-222 at home and at work. ICRP Publication No. 65. *Annals of the ICRP*, 23(2):1-45.

11. World Health Organization (WHO). 2000. Air Quality Guidelines - 2nd Edition. Copenhagen, Denmark: WHO Regional Office for Europe.

12. Nero, A.V., Jr. 1990. Radon and its Decay Products in Indoor Air: An overview. In: *Radon and Its Decay Products In Indoor Air.* W.W. Nazaroff and A.V. Nero, Jr., (eds.). New York: John Wiley & Sons. pp. 1-53.

13. Cartwright, B.G., Shirk, E.K., and Price, P.B. 1978. A nuclear-track-recording polymer of unique sensitivity and resolution. *Nucl.Instrum.Methods*, 153:457.

14. Cassou, R.M. and Benton, E.V. 1978. Properties and applications of CR-39 polymeric nuclear track detector. *Nucl.Track Detection*, 2:173.

15. Nazaroff, W.W. 1990. Appendix: Measurement Techniques. In: *Radon and Its Decay Products.* W.W. Nazaroff and A.V. Nero, Jr., (eds.). New York: John Wiley & Sons. pp. 491-504.

16. Budnitz, R.J. 1974. Radon-222 and its daughters: A review of instrumentation for occupational and environmental monitoring. *Health Phys.*, 26:145.

17. Budnitz, R.J., Nero, A.V., Murphy, D.J., and Graven, R. 1983. *Instrumentation for Environmental Monitoring, 2nd Ed. Vol. 1., Radiation.* New York: Wiley.

18. United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). 2000. Sources and Effects of Ionizing Radiation. UNSCEAR 2000 Report to the General Assembly, with Scientific Annexes. Vol I: Sources. New York: United Nations.

19. Krewski, D., Lubin, J., Zielinski, J.M., Alavanja, M.C., Catalan, V., Field, R.W., Klotz, J.B., Létourneau, E., Lynch, C.F., Lyon, J., Sandler, D.P., Schoenberg, J.B., Steck, D.J., Stolwijk, J.A., Weinberg, C., and Wilcox, H.B. 2002. A combined analysis of North American case-control studies of residential radon and lung cancer. *N.Engl.J.Med.*, (Submitted).

20. Leech, J.A., Nelson, W.C., Burnett, R.T., Aaron, S., and Raizenne, M.E. 2002. It's about time: a comparison of Canadian and American time-activity patterns. *J.Expo.Anal.Environ.Epidemiol.*, 12(6):427-432.

21. Clarke, R.H. and Southwood, T.R.E. 1989. Risks from ionizing radiation. *Nature*, 338:197-198.

22. National Council of Radiation Protection and Measurements (NCRP). 1984. Exposures from the Uranium Series with Emphasis on Radon and Its Daughters. NCRP Report 77. Washington, DC: National Council on Radiation protection and Measurements.

23. Nielson, K.K., Rogers, V.C., Holt, R.B., Pugh, T.D., Grondzik, W.A., and de Meijer, R.J. 1993. Radon penetration of concrete slab cracks, joints, pipe penetrations, and sealants. *Health Physics*, 73(4):668-678.

24. Nazaroff, W.W., Moed, B.A., and Sextro, R.G. 1990. Soil as a Source of Indoor Radon: Generation, Migration, and Entry. In: *Radon and Its Decay Products.* W.W. Nazaroff and A.V. Nero, Jr., (eds.). New York: John Wiley & Sons. pp. 57-112.

25. National Research Council (NRC). 1999. Risk Assessment of Radon in Drinking Water. Washington, DC: National Academy Press.

26. Nazaroff, W.W., Doyle, S.M., Nero, A.V., Jr., and Sextro, R.G. 1990. Radon Entry via Potable Water. In: *Radon and Its Decay Products.* W.W. Nazaroff and A.V. Nero, Jr., (eds.). New York: John Wiley & Sons. pp. 131-157.

27. Stranden, E. 1990. Building Materials as a source of Indoor Radon. In: *Radon and Its Decay Products.* W.W. Nazaroff and A.V. Nero, Jr., (eds.). New York: John Wiley & Sons. pp. 113-157.

28. Knutson, E.O. 1990. Modeling Indoor Concentrations of Radon's Decay Products. In: *Radon and Its Decay Products.* W.W. Nazaroff and A.V. Nero, Jr., (eds.). New York: John Wiley & Sons. pp. 161-202.

29. Jalbert, P.P.U.S.E. 2004. Radon Policy in the U.S. Presentation delivered at the Health Canada Radon Workshop, Ottawa, March 3-4, 2004; Radiation Protection Bureau.

30. Miles, J.C.H. and Algar, R.A. 1988. Variations in radon-222 concentrations. *J.Radiol.Prot.*, 8(2):103-105.

31. McGregor, R., Vasudev, P., Létourneau, E., McCullough, R., Prantl, F., and Taniguchi, H. 1980. Background Concentration of Radon and Radon Daughters in Canadian Homes. *Health Phys*, 39:285-289.

32. Nero, A.V., Schwehr, M.B., Nazaroff, W.W., and Revzan, K.L. 1986. Distribution of airborne radon-222 concentrations in U.S. homes. *Science*, 234(4779):992-997.

33. United States Environmental Protection Agency (EPA). 1992. National residential radon survey. EPA 402/R-92-011.

34. Marcinowski, F. 1992. Nationwide survey of residiential radon levels in the U.S. *Radiat.Prot.Dosimetry.*, 45(1):419-424.

35. Commission of the European Communities. 1987. Radiation Protection, Exposure to Natural Radiation in Dwellings of the European Communities. Luxembourg: CEC.

36. Swedjemark, G.A., Mellander, H., and Mjönes, L. 1993. Radon levels in the 1988 Swedish Housing Stock. in: Indoor Air '93. Proceeds of the 6th International Symposium on Indoor Air Quality and Climate, Helsinki, July 4-8, 1993.

37. Surbeck, H. and Völkle, H. 1991. Radon in Switzerland. Presented at the 1991 International Symposium on Radon and Radon Reduction Technology, Philidelphia, April, 1991.

38. Castren, O. 1994. Dealing with radon in dwellings: the Finnish experience. *Radiat.Prot.Dosimetry.*, 56:375-378.

39. Wrixon, A.D., Green, B.M.R., Lomas, P.R., Miles, J.C.H., Cliff, K.D., Francis, E.A., Driscoll, C.M., James, A.C.a., and O'Riordan, M. 1988. Natural radiation exposure in UK dwellings. NRPB-R190.

40. Letourneau, E.G., Zielinski, J.M., Krewski, D., and McGregor, R.G. 1992. Levels of Radon Gas in Winnipeg Homes. *Radiation Protection Dosimetry*, 45(1):531-534.

41. Bale, W.F. 1951. Memorandum to the Files, March 14, 1951: Hazards Associated with Radon and Thoron. *Health Phys*, 38:1061-1066.

42. Harris, S.J. 1954. Radon Levels in Mines in New York State. *Archives of Industrial Hygiene and Occupational Medicine*, 10:54-60.

43. Morken, D.A. 1955. Acute Toxicitiy of Radon. *Archives of Industrial Health*, 12:435.

44. Shapiro, J. 1954. An Evaluation of the Pulmonary Radiation Dosage from Radon and Its Daughter Products. Project report UR-298 to the US Atomic Energy Commission, University of Rochester, Rochester, NY.

45. Cross, F.T., Palmer, R.F., Filipy, R.E., Busch, R.H., and Stuart, B.O. 1978. Study of the Combined Effects of Smoking and Inhalation of Uranium Ore Dust, Radon Daughters and Diesel Oil Exhause Fumes in Hamsters and Dogs. PNL-2744. Springfield, VA: National Technical Information Service.

46. National Research Council (NRC). 1988. Committee on the Biological Effects of Ionizing Radiations. Health risks of radon and other internally deposited alpha-emitters: BEIR IV. Washington, DC: National Academy Press.

47. Cross, F.T. 1990. Evidence of Lung Cancer from Animal Studies. In: *Radon and Its Decay Products.* W.W. Nazaroff and A.V. Nero, Jr., (eds.). New York: John Wiley & Sons. pp. 373-404.

48. Little, M.P. and Wakeford, R. 2001. The bystander effect in C3H 10T cells and radon-induced lung cancer. *Radiat.Res.*, 156(6):695-699.

49. Evans, H. 1991. Cellular and molecular effects of radon and other alpha particle emitters. *Adv Mutat Res*, 3:28-52.

50. Metting, N., Palayoor, S., Macklis, R., Atcher, R., Liber, H., and Little, J. 1992. Induction of mutations by bismuth-212 a-particles at two genetic loci in human B-lymphoblasts. *Radiat.Res.*, 132:339-345.

51. Petersen, D., Gonzalez, F., Rapic, V., Kozak, C., Lee, J., Jones, J., and Nevert, D. 1989. Marked increases in hepatic NAD(P)H:oxidoreductase gene transcription and mRNA levels correlated with a mouse chromosome 7 deletion. *Proc Natl Acad Sci USA*, 86(17):6699-6703.

52. Dalla-Favera, R., Martinotti, S., Gallo, R., Erikson, J., and Croce, C. 1983. Translocation and rearrangement of the c-myc oncogene locus in human undifferentiated B-cell lymphomas. *Science*, 219:963-997.

53. Stanbridge, E. 1976. Suppression of malignancy in human cells. *Nature*, 260:17-20.

54. Kasten, M., Onyuuekwere, O., Sidransky, D., Vogelstein, B., and Craig, R. 1991. Participation of p53 protein in the cellular response to DNA damage. *Cancer Res*, 51:6304-6311.

55. Smith, M., Zhan, Q., Bae, I., and Fornace, A., Jr. 1994. Role of retinoblastoma gene porduct in p53-mediated DNA damage response. *Exp Cell Res*, 215:386-389.

56. Sabatier, L., Dutrillaux, B., and Martins, M.B. 1992. Chromosomal instability. *Nature*, 357:548.

57. Martins, M.B., Sabatier, L., Ricoul, M., Pinoton, A., and Dutrillaux, B. 1993. Specific chromosome instability induced by heavy ions: a step towards transformation of human fibroblasts. *Mutat Res*, 285:229-237.

58. Marder, B.A. and Morgan, W.F. 1993. Delayed chromosomal instability induced by DNA damage. *Mol Cell Biol*, 13:6667-6677.

59. Sankaranarayanan, K. and Chakraborty, R. 1995. Cancer predisposition, radiosensitivity and the risk of radiation-induced cancers. I. Background. *Radiat Res*, 143:121-143.

60. Swift, M., Morrell, D., Massey, R., and Chase, C. 1991. Incidence of cancer in 161 families affected by ataxia-telangiectasia. *N.Engl.J.Med.*, 325:1831-1836.

61. Cox, R. 1994. Human cancer predisposition and the implications for radiological protection. *Int J Radiat Biol*, 66:643-647.

62. Cox, R. 1994. Molecular mechanisms of radiation oncogenesis. *Int J Radiat Biol*, 65:57-64.

63. Lavin, M., Bennett, I., Ramsay, J., Gardiner, R., Seymour, G., Farrel, A., and Walsh, M. 1994. Identification of a potentially radiosensitive subgroup among patients with breast cancer. *J Natl Cancer Inst*, 86(1627):1634.

64. Lucke-Huhle, C., Comper, W., Hieber, L., and Pech, M. 1982. Comparative study of G2 delay and survival after 241Americium-alpha and 60cobalt-gamma irradiation. *Radiat Environ Biophys*, 20:171-185.

65. Murname, J. 1995. Cell cycle regulation in response to DNA damage in mammalian cells: A historical perspective. *Cancer and Metastasis Reviews*, 14:17-29.

66. Stewart, B. 1994. Mechanisms of apoptosis: integration of genetic, biocehmical, and cellular indicators. *J Natl Cancer Inst*, 86(17):1286-1296.

67. Thompson, C. 1995. Apoptosis in the pathogenesis and treatment of disease. *Science*, 267(5203):1456-1462.

68. Taya, A., Morgan, A., Baker, S., Humphreys, J., Bisson, M., and Collier, C. 1994. Changes in the rat lung after exposure to radon and its progeny: effects on incorporation of bromodeoxyuridine in epithelial cells and on the incidence of nuclear aberrations in alveolar macrophages. *Radiat Res*, 139:170-177.

69. Atencio, E. 1994. Epithelial cell kinetics of the upper respiratory tract of Wistar rats following radon exposure. Thesis, Masters of Science in Environmental Science, Washinton State University.

70. Cohen, S., Garland, E., and Ellwein, L. 1992. Cancer enhancement by cell proliferation. *Prog Clin Biol Res*, 374:213-229.

71. Krewski, D., Zielinski, J.M., Hazelton, W.D., Moolgavkar, S.H., and Garner, M. 2002. The Use of Biologically Based Cancer Risk Models in Radiation Epidemiology. *Radiation Protection Dosimetry*, 104(1) (In Press).

72. Armitage, P. and Doll, R. 1954. The age distribution of cancer and a multi-stage theory of carcinogenesis. *Br.J.Cancer*, 8:1-12.

73. Armitage, P. and Doll, R. 1957. A two-stage theory of carcinogeneisis in relation to the age distribution of human cancer. *Br.J.Cancer*, 11:161-169.

74. Fisher, J. 1958. Multiple mutation theory of carcinogenesis. *Nature*, 181:651-2.

75. Cook, P., Doll, R., and Fellingham, S. 1969. A mathematical model for the age distribution of cancer in man. *Int.J Cancer*, 4:93-112.

76. Moolgavkar, S. and Knudson, A. 1981. Mutation and cancer: a model for human carcinogenesis. *J Natl Cancer Inst*, 66:1037-1052.

77. Moolgavkar, S.H. 2002. The Neyman-Scott carcinogenesis model for low-dosage extrapolation. *Mathematical Biosciences*, 50:155-156.

78. Heidenreich, W.F., Luebeck, E.G., Hazelton, W.D., Paretzke, H.G., and Moolgavkar, S.H. 2002. Multistage models and the incidence of cancer in the cohort of atomic bomb survivors. *Radiat Res*, 158:607-614.

79. Hazelton, W.D., Moolgavkar, S.H., Curtus, S., Zielinski, J.M., Ashmore, J., and Krewski, D. 2002. Biologically based analysis of lung cancer incidence in a large Canadian occupational cohort with low-dose ionizing radiation exposure, and comparison with Japanese atomic bomb survivors.

80. Moolgavkar, S.H., Luebeck, E.G., Krewski, D., and Zielinski, J.M. 1993. Radon, cigarette smoke, and lung cancer: a re-analysis of the Colorado Plateau uranium minters; data. *Epidemiology*, 4:204-217.

81. Hazelton, W., Luebeck, E.G., Heidenreich, W.F., and Moolgavkar, S.H. 2001. Analysis of a historical cohort of Chinese tin miners with arsenic, radon, cigarette smoke, and pipe smoke expsoures using the biologically based two-stage clonal expansion model. *Radiat Res*, 156:78-94.

82. Luebeck, E.G., Heidenreich, W.F., Hazelton, W.D., Paretzke, H.G., and Moolgavkar, S.H. 1999. Biologically based analysis of the data for the Colorado uranium miners cohort: age, dose and dose-rate effects. *Radiat.Res.*, 152(4):339-351.

83. Zielinski, J.M., Kodell, R., and Krewski, D. 2001. Interaction between two carcinogens in the two-stage clonal expansion model of carcinogenesis. *J Epidemiol.Biostat.*, 6:219-228.

84. James, A.C. 1990. Lung Dosimetry. In: *Radon and Its Decay Products*. W.W. Nazaroff and A.V. Nero, Jr., (eds.). New York: John Wiley & Sons. pp. 259-309.

85. International Commission on Radiation Protection (ICRP). 1990. Recommendations of the International Commission on Radiation Protection, ICRP Publication 60. *Annals of the ICRP*, 21.

86. Hoover, H. and Hoover, L. 1950. *Georgius Agricola De Re Metalallica*. New York: Dover Publications Inc.

87. Ludwig, P. and Lorenser, S. 1924. Untersuchungen der grubenluft in den Schneeberger gruben auf den gehalt an radiumemanation. *Z Physik*, 22:178-185.

88. Pirchan, A. and Sikl, H. 1932. Cancer of the lung in the miners of Jachymov. *American Journal of Cancer*, 16:681-722.

89. Lorenz, E. 1944. Radioactivity and lung cancer: A critical review of lung cancer in the miners of Schneeberg and Joachimsthal. *J Natl Cancer Inst*, 5:1-15.

90. Xuan, X.Z., Lubin, J.H., Li, J.Y., Yang, L.F., Luo, A.S., Lan, Y., Wang, J.Z., and Blot, W.J. 1993. A cohort study in southern China of tin miners exposed to radon and radon decay products. *Health Phys.*, 64(2):120-131.

91. Sevc, J., Tomasek, L., Kunz, E., Placek, V., Chmelevsky, D., Barclay, D., and Kellerer, A.M. 1993. A survey of the Czechoslovak follow-up of lung cancer mortality in uranium miners. *Health Phys.*, 64(4):355-369.

92. Sevc, J., Kunz, E., Tomasek, L., Placek, V., and Horacek, J. 1988. Cancer in man after exposure to Rn daughters. *Health Phys.*, 54(1):27-46.

93. Tomasek, L. and Placek, V. 1999. Radon exposure and lung cancer risk: Czech cohort study. *Radiat.Res.*, 152(6 Suppl):S59-S63.

94. Tomasek, L., Swerdlow, A.J., Darby, S.C., Placek, V., and Kunz, E. 1994. Mortality in uranium miners in west Bohemia: a long-term cohort study. *Occup.Environ.Med.*, 51(5):308-315.

95. Tomasek, L. and Darby, S.C. 1995. Recent results from the study of West Bohemian uranium miners exposed to radon and its progeny. *Environ.Health Perspect.*, 103 Suppl 2:55-57.

96. Hornung, R.W. and Meinhardt, T.J. 1987. Quantitative risk assessment of lung cancer in U.S. uranium miners. *Health Phys.*, 52(4):417-430.

97. Hornung, R.W., Deddens, J., and Roscoe, R. 1995. Modifiers of exposure-response estimates for lung cancer among miners exposed to radon progeny. *Environ.Health Perspect.*, 103 Suppl 2:49-53.

98. Hornung, R.W., Deddens, J.A., and Roscoe, R.J. 1998. Modifiers of lung cancer risk in uranium miners from the Colorado Plateau. *Health Phys.*, 74(1):12-21.

99. Muller, J., Wheeler, W., Gentleman, J., Suranji, J., and Kusick, R. 1983. Study of mortality of Ontario miners 1955-1957 Part 1. Toronto: Ontario Ministry of Labour.

100. Kusiak, R.A., Springer, J., Ritchie, A.C., and Muller, J. 1991. Carcinoma of the lung in Ontario gold miners: possible aetiological factors. *Br.J.Ind.Med.*, 48(12):808-817.

101. Kusiak, R.A., Ritchie, A.C., Muller, J., and Springer, J. 1993. Mortality from lung cancer in Ontario uranium miners. *Br.J.Ind.Med.*, 50(10):920-928.

102. Morrison, H.I., Semenciw, R.M., Mao, Y., and Wigle, D.T. 1988. Cancer mortality among a group of fluorspar miners exposed to radon progeny. *Am.J.Epidemiol.*, 128(6):1266-1275.

103. Radford, E. and St.Clair Renard, K. 1984. Lung cancer in Swedish iron miners exposed to low doses of radon daughters. *N Engl J Med*, 310(23):1485-1494.

104. Samet, J.M., Pathak, D.R., Morgan, M.V., Marbury, M.C., Key, C.R., and Valdivia, A.A. 1989. Radon progeny exposure and lung cancer risk in New Mexico U miners: a case-control study. *Health Phys.*, 56(4):415-421.

105. Samet, J.M. 1991. Diseases of uranium miners and other underground miners exposed to radon. *Occup.Med.*, 6(4):629-639.

106. Samet, J.M., Pathak, D.R., Morgan, M.V., Coultas, D.B., James, D.S., and Hunt, W.C. 1994. Silicosis and lung cancer risk in underground uranium miners. *Health Phys.*, 66(4):450-453.

107. Howe, G., Nair, R.C., Newcombe, H.B., Miller, A.B., and Abbatt, J. 1986. Lung cancer mortality (1950-80) in relation to radon daughter exposure in a cohort of workers at the Eldorado Beaverlodge Uranium Mine. *J Natl Cancer Inst*, 77:357-362.

108. L'Abbe, K.A., Howe, G.R., Burch, J.D., Miller, A.B., Abbatt, J., Band, P., Choi, W., Du, J., Feather, J., Gallagher, R., and . 1991. Radon exposure, cigarette smoking, and other mining experience in the Beaverlodge uranium miners cohort. *Health Phys.*, 60(4):489-495.

109. Howe, G.R. and Stager, R. 1996. Risk of lung cancer mortality after exposure to radon decay products in the Beaverlodge cohort based on revised exposure estimates. *Radiat Res*, 146:37-42.

110. Howe, G.R., Nair, R.C., Newcombe, H.B., Miller, A.B., Burch, J.D., and Abbatt, J.D. 1987. Lung cancer mortality (1950-1980) in relation to radon daughter exposure in a cohort of workers at the Eldorado Port Radium uranium mine: Possible modification by exposure rate. *J Natl Cancer Inst*, 79:1255-1260.

111. Woodward, A., Roder, D., McMichael, A.J., Crouch, P., and Mylvaganam, A. 1991. Radon daughter exposures at the Radium Hill uranium mine and lung cancer rates among former workers, 1952-87. *Cancer Causes Control*, 2(4):213-220.

112. Tirmarche, M., Raphalen, A., and Chameaud, J. 1992. Epidemiological study of French uranium miners. *Cancer Detect.Prev.*, 16(3):169-172.

113. Tirmarche, M., Raphalen, A., Allin, F., Chameaud, J., and Bredon, P. 1993. Mortality of a cohort of French uranium miners exposed to relatively low radon concentrations. *Br.J.Cancer*, 67(5):1090-1097.

114. Lubin, J.H., Boice, J.D.Jr., Edling, C., Hornung, R.W., Howe, G., Kunz, E., Kusiak, R.A., Morrison, H.I., Radford, E.P., Samet, J.M., Tirmarche, M., Woodward, A., Yao, S.X., and Pierce, D. 1994. Radon and Lung Cancer Risk: A joint Analysis of 11 Underground Miners Studies. National Institutes of Health, National Cancer Institute. NIH Publication No. 94-3644. Washington, DC: U.S. Department of Health and Human Services.

115. Lubin, J.H., Boice, J.D., Jr., Edling, C., Hornung, R.W., Howe, G.R., Kunz, E., Kusiak, R.A., Morrison, H.I., Radford, E.P., Samet, J.M., and . 1995. Lung cancer in radon-exposed miners and estimation of risk from indoor exposure. *J.Natl.Cancer Inst.*, 87(11):817-827.

116. Lubin, J.H., Tomasek, L., Edling, C., Hornung, R.W., Howe, G., Kunz, E., Kusiak, R.A., Morrison, H.I., Radford, E.P., Samet, J.M., Tirmarche, M., Woodward, A., and Yao, S.X. 1997. Estimating lung cancer mortality from residential radon using data for low exposures of miners. *Radiat.Res.*, 147(2):126-134.

117. Krewski, D., Rai, S.N., Zielinski, J.M., and Hopke, P.K. 1999. Characterization of uncertainty and variability in residential radon cancer risks. *Ann.N.Y.Acad.Sci.*, 895:245-272.

118. Brand, K., Zielinski, J., and Krewski, D. 2004. Residential Radon in Canada: An Uncertainty Analysis of Population and Individual Lung Cancer Risk. *Health Physics (In Press).*

119. Steinhäusler, F. 1990. Epidemiological evidence of Radon-Induced Health Risks. In: *Radon and its Decay Products in Indoor Air.* W.W. Nazaroff and A.V. Nero, Jr., (eds.). New York: John Wiliey & Sons. pp. 311-371.

120. Archer, V.E. 1987. Association of lung cancer mortality with precambrian granite. *Arch.Environ.Health*, 42(2):87-91.

121. Bean, J.A., Isacson, P., Hahne, R.M., and Kohler, J. 1982. Drinking water and cancer incidence in Iowa. II. Radioactivity in drinking water. *American Journal of Epidemiology*, 116(6):924-932.

122. Bean, J.A., Isacson, P., Hausler, W.J., Jr., and Kohler, J. 1982. Drinking water and cancer incidence in Iowa. I. Trends and incidence by source of drinking water and size of municipality. *American Journal of Epidemiology*, 116(6):912-923.

123. Dousset, M. and Jammet, H. 1985. Comparison of cancer mortality in Limousin and Poitou-Charentes. *Radioprotection*, 20:61-70.

124. Ennemoser, O., Ambach, W., Brunner, P., and Schneider, P. 1994. Unusual high radon exposure in homes and lung cancer. *Lancet*, 344(8915):127-128.

125. Fleischer, R.L. 1981. A possible association between lung cancer and phosphate mining and processing. *Health Physics*, 41(1):171-175.

126. Fleischer, R.L. 1986. A possible association between lung cancer and a geological outcrop. *Health Physics*, 50(6):823-827.

127. Forastiere, F., Valesini, S., Arca, M., Magliola, M.E., Michelozzi, P., and Tasco, C. 1985. Lung cancer and natural radiation in an Italian province. *Science of the Total Environment*, 45:519-526.

128. Hoffmann, W., Katz, R., and Zhang, C.X. 1985. Lung cancer incidence in a Chinese high background area--epidemiological results and theoretical interpretation. *Science of the Total Environment*, 45:527-534.

129. Neuberger, J.S., Lynch, C.F., Kross, B.C., Field, R.W., and Woolson, R.F. 1994. Residential radon exposure and lung cancer: evidence of an urban factor in Iowa. *Health Phys.*, 66(3):263-269.

130. Vonstille, W. and Sacarello, H. 1990. Radon and cancer: Florida study finds no evidence of increased risk. *J Environ Health*, 53:25-28.

131. Edling, C., Comba, P., Axelson, O., and Flodin, U. 1982. Effects of low-dose radiation - a correlation study. *Scand.J.Work Environ.Health*, 8 Suppl 1:59-64.

132. Hess, C.T., Weiffenbach, C.V., and Norton, S.A. 1983. Environmental radon and cancer correlations in Maine. *Health Phys.*, 45:339-348.

133. Létourneau, E., Mao, Y., McGregor, R., Semenciw, R., Smith, M., and Wigle, D. 1983. Lung cancer mortality and indoor radon concentrations in 18 Canadian cities: Epidemiology applied to health physics. Albuquerque, NM :. pp. 470-483.

134. Haynes, R. 1988. The distribution of domestic radon concentrations and lung cancer mortality in England and Wales. *Radiat Prot Dos*, 25:93-96.

135. Cohen, B.L. 1997. Lung cancer rate vs. mean radon level in U.S. counties of various characteristics. *Health Phys.*, 72(1):114-119.

136. Ruostennoja, E. 1991. Indoor radon and risk of lung cancer: An epidemiologic study in Finland. Thesis, Ph.D, Department of Public Health, University of Tampere.

137. Magnus, K., Engeland, A., Green, B.M., Haldorsen, T., Muirhead, C.R., and Strand, T. 1994. Residential radon exposure and lung cancer--an epidemiological study of Norwegian municipalities. *Int.J.Cancer*, 58(1):1-7.

138. Cohen, B.L. 1995. Test of the linear-no threshold theory of radiation carcinogenesis for inhaled radon decay products. *Health Physics*, 68(2):157-174.

139. Stranden, E. 1987. Radon-222 in Norwegian dwellings. In: *Radon and Its Decay Products: Occurence, Properties, and Health Effects* Washington, DC: American Chemical Society.

140. Ruosteenoja, E. 1991. Indoor radon and risk of lung cancer: An epidemiologic study in Finland. Thesis, Ph.D, Department of Public Health, University of Tampere.

141. Greenland, S. and Robins, J. 1994. Invited commentary: ecologic studies - biases, misconceptions, and counterexamples. *Am.J.Epidemiol.*, 139:747-760.

142. Greenland, S. and Robins, J. 1994. Accepting the limits of ecologic studies: Drs. Greenland and Robins reply to Drs. Piantadosi and Cohen. *Int J Epidemiol*, 139:769-771.

143. Gilbert, E. 1994. Smoking as an explanation for the negative relationship between exposure to radon and certain types of cancer. *Health Phys*, 67:192.

144. Cohen, B.L. 1994. Response to Gilbert. *Health Phys*, 167:197-198.

145. Lubin, J.H. 1998. On the discrepancy between epidemiologic studies in individuals of lung cancer and residential radon and Cohen's ecologic regression. *Health Phys.*, 75(1):4-10.

146. Cohen, B.L. 1998. Response to Lubin's proposed explanations for our discrepancy. *Health Phys*, 75(1):18-22.

147. Lubin, J.H. 1998. Rejoinder: Cohen's response to "On the discrepancy between epidemiologic studies in individuals of lung cancer and residential radon and Cohen's ecologic regression". *Health Phys*, 75(1):29-30.

148. Smith, B.J., Field, R.W., and Lynch, C.F. 1998. Residential ^{222}Rn exposure and lung cancer: testing the linear no-threshold theory with ecologic data. *Health Phys.*, 75(1):11-17.

149. Cohen, B.L. 1998. Response to criticisms of Smith et al. *Health Phys*, 75(1):23-28.

150. Field, R.W., Smith, B.J., and Lynch, C.F. 1998. Ecologic bias revisited, a rejoinder to Cohen's response to "Residential ^{222}Rn expsoure and lung cancer: Testing the linear no-threshold theory with ecologic data". *Health Phys*, 75(1):31-33.

151. Puskin, J.S. 2003. Smoking as a confounder in ecologic correlations of cancer mortality rates with average country radon levels. *Health Phys*, 84(4):526-532.

152. Greenland, S. and Morgenstern, H. 1989. Ecological bias, confounding, and effect modification. *Int J Epidemiol*, 18:269-274.

153. Breener, D., Savitz, D., Jockel, K., and Greenland, S. 1992. The effect of nondifferential exposure misclassification in ecological studies. *Am J Epidemiol*, 135(1):85-95.

154. Morgenstern, H. 1995. Ecologic studies in epidemiology: Concepts, principles and methods. *Ann Rev Public Health*, 16:61-81.

155. Stidley, C.A. and Samet, J.M. 1993. A review of ecologic studies of lung cancer and indoor radon. *Health Phys.*, 65(3):234-251.

156. Axelson, O., Edling, C., and Kling, H. 1979. Lung cancer and residency - a case-referent study on the possible impact of exposure to radon and its daughters in dwellings. *Scand.J.Work Environ.Health*, 5:10-15.

157. Edling, C., Kling, H., and Axelson, O. 1984. Radon in homes--a possible cause of lung cancer. *Scand.J.Work Environ.Health*, 10(1):25-34.

158. Pershagen, G., Damber, L., and Falk, R. 1984. Exposure to radon in dwellings and lung cancer: A pilot study. In: *Indoor Air. Radon, Passive Smoking, Particulates and Housing Epidemiology*, Vol. 2. B. Berglung, T. Lindvall, and J. Sundell, (eds.). Stockholm: Stockholm Council for Building Research. pp. 29-34.

159. Damber, L.A. and Larsson, L.G. 1987. Lung cancer in males and type of dwelling. An epidemiologic pilot study. *Acta Oncol.*, 26(3):211-215.

160. Svensson, C., Eklund, G., and Pershagen, G. 1987. Indoor exposure to radon from the ground and bronchial cancer in women. *Int.Arch.Occup.Environ.Health*, 59(2):123-131.

161. Axelson, O., Andersson, K., Desai, G., Fagerlund, I., Jansson, B., Karlsson, C., and Wingren, G. 1988. Indoor radon exposure and active and passive smoking in relation to the occurrence of lung cancer. *Scand.J.Work Environ.Health*, 14(5):286-292.

162. Svensson, C., Pershagen, G., and Klominek, J. 1989. Lung cancer in women and type of dwelling in relation to radon exposure. *Cancer Res*, 49:1861-1865.

163. Simpson, S.G. and Comstock, G.W. 1983. Lung cancer and housing characteristics. *Arch.Environ.Health*, 38(4):248-251.

164. New Jersey State Department of Health. 1989. A case-control study of radon and lung cancer among New Jersey women. Technical Report - Phase I.

165. Schoenberg, J.B., Klotz, J.B., Wilcox, H.B., Nicholls, G.P., Gil-del-Real, M.T., Stemhagen, A., and Mason, T.J. 1990. Case-control study of residential radon and lung cancer among New Jersey women. *Cancer Res.*, 50(20):6520-6524.

166. Schoenberg, J.B., Klotz, J.B., Wilcox, H.B., and Szmaciasz, S.F. 1992. A case-control study of radon and lung cancer among New Jersey women. *Twenty-Ninth Hanford Symposium on Health and the Environment, Indoor Radon and Lung Cancer: Reality or Myth?*, Sponsord by the United States Department of Energy and Battelle, Pacific Northwest Laboratories. Columbus, Richland, Washington :. pp. 905-918.

167. Letourneau, E.G., Krewski, D., Choi, N.W., Goddard, M.J., McGregor, R.G., Zielinski, J.M., and Du, J. 1994. Case-control study of residential radon and lung cancer in Winnipeg, Manitoba, Canada. *Am.J.Epidemiol.*, 140(4):310-322.

168. Alavanja, M.C., Brownson, R.C., Lubin, J.H., Berger, E., Chang, J., and Boice, J.D., Jr. 1994. Residential radon exposure and lung cancer among nonsmoking women. *J.Natl.Cancer Inst.*, 86(24):1829-1837.

169. Alavanja, M.C., Lubin, J.H., Mahaffey, J.A., and Brownson, R.C. 1999. Residential radon exposure and risk of lung cancer in Missouri. *Am.J.Public Health*, 89(7):1042-1048.

170. Field, R.W., Steck, D.J., Smith, B.J., Brus, C.P., Fisher, E.L., Neuberger, J.S., Platz, C.E., Robinson, R.A., Woolson, R.F., and Lynch, C.F. 2000. Residential radon gas exposure and lung cancer: the Iowa Radon Lung Cancer Study. *Am.J.Epidemiol.*, 151(11):1091-1102.

171. Sandler, D., Weinberg, C.R., Archer, V.E., Rothney-Kozlak, L., Bishop, M., Lyon, J., and Stolwijk, J. 1999. Proceedings of the American Statistical Association Conference on Radiation and Health, Indoor Radon and Lung Cancer Risk: A case-control study in Connecticut and Utah. *Radiat Res*, 151:103-105.

172. Pershagen, G., Liang, Z.H., Hrubec, Z., Svensson, C., and Boice, J.D., Jr. 1992. Residential radon exposure and lung cancer in Swedish women. *Health Phys.*, 63(2):179-186.

173. Pershagen, G., Akerblom, G., Axelson, O., Clavensjo, B., Damber, L., Desai, G., Enflo, A., Lagarde, F., Mellander, H., Svartengren, M., and . 1994. Residential radon exposure and lung cancer in Sweden. *N.Engl.J.Med.*, 330(3):159-164.

174. Lagarde, F., Axelsson, G., Damber, L., Mellander, H., Nyberg, F., and Pershagen, G. 2001. Residential radon and lung cancer among never-smokers in Sweden. *Epidemiology*, 12(4):396-404.

175. Ruosteenoja, E., Makelainen, I., Rytomaa, T., Hakulinen, T., and Hakama, M. 1996. Radon and lung cancer in Finland. *Health Phys.*, 71(2):185-189.

176. Auvinen, A., Makelainen, I., Hakama, M., Castren, O., Pukkala, E., Reisbacka, H., and Rytomaa, T. 1996. Indoor radon exposure and risk of lung cancer: a nested case-control study in Finland. *J.Natl.Cancer Inst.*, 88(14):966-972.

177. Darby, S., Whitley, E., Silcocks, P., Thakrar, B., Green, M., Lomas, P., Miles, J., Reeves, G., Fearn, T., and Doll, R. 1998. Risk of lung cancer associated with residential radon exposure in south-west England: a case-control study. *Br.J.Cancer*, 78(3):394-408.

178. Bochicchio, F., Forastiere, F., Abeni, D., and Rapiti, E. 1998. Epidemiologic studies on lung cancer and residential exposure to radon in Italy and other countries. *Radiat Prot Dosimetry*, 78(1):33-38.

179. Wichmann, H.E., Gerken, M., Wellman, J., Kreuzer, M., Kreienbrock, L., Keller, G., Woelke, G., and Heinrich, J. 1999. Lungenkrebsrisiko durch Radon in der Bundesrepublik Deutschland (Ost). *Theringen und Sachsen.Ecomed, Lansberg/Lech.*

180. Kreienbrock, L., Kreuzer, M., Gerken, M., Dingerkus, G., Wellmann, J., Keller, G., and Wichmann, H.E. 2001. Case-control study on lung cancer and residential radon in western Germany. *Am.J.Epidemiol.*, 153(1):42-52.

181. Blot, W.J., Xu, Z.Y., Boice, J.D., Jr., Zhao, D.Z., Stone, B.J., Sun, J., Jing, L.B., and Fraumeni, J.F., Jr. 1990. Indoor radon and lung cancer in China. *J.Natl.Cancer Inst.*, 82(12):1025-1030.

182. Wang, Z. and Steinhausler, F. 1996. Elevated indoor exposure in Chinese carbon brick and cave dwellings. *Health Phys.*, 71(3):374-378.

183. Dosemeci, M., Wacholder, S., and Lubin, J.H. 1990. Does non-differential misclassification of exposure always bias a true effect toward the null value? *Am J Epidemiol*, 132:746-8.

184. Wacholder, S., Silverman, D.T., McLaughlin, J.K., and Mandel, J. 1995. Non-differential misclassification and bias towards the null: a clarification. *Occup Environ Med*, 52:157-8.

185. Lubin, J.H., Boice, J.D., Jr., and Samet, J.M. 1995. Errors in exposure assessment, statistical power and the interpretation of residential radon studies. *Radiat.Res.*, 144(3):329-341.

186. Field, R.W., Steck, D.J., Lynch, C.F., Brus, C.P., Neuberger, J.S., and Kross, B.C. 1996. Residential radon-222 exposure and lung cancer: exposure assessment methodology. *J.Expo.Anal.Environ.Epidemiol.*, 6(2):181-195.

187. Warner, K.E., Mendez, D., and Courant, P.N. 1996. Toward a more realistic appraisal of the lung cancer risk from radon: the effects of residential mobility. *Am.J.Public Health*, 86(9):1222-1227.

188. Upfal, M., Divine, G., and Siemiatycki, J. 1995. Design issues in studies of radon and lung cancer: implications of the joint effect of smoking and radon. *Environ.Health Perspect.*, 103(1):58-63.

189. Field, R.W., Steck, D.J., Smith, B.J., Brus, C.P., Fisher, E.F., Neuberger, J.S., and Lynch, C.F. 2001. The Iowa radon lung cancer study--phase I: Residential radon gas exposure and lung cancer. *Sci.Total Environ.*, 272(1-3):67-72.

190. Lubin, J.H. and Boice, J.D., Jr. 1997. Lung cancer risk from residential radon: meta-analysis of eight epidemiologic studies. *J.Natl.Cancer Inst.*, 89(1):49-57.

191. Auvinen, A. 1996. Lung cancer risk from indoor radon. *Lancet*, 348(9042):1662-1663.

192. Greenland, S. 1987. Quantitative methods in the review of epidemiologic literature. *Epidemiol.Reviews*, 9:1-30.

193. Lubin, J.H., Liang, Z., Hrubec, Z., Pershagen, G., Schoenberg, J.B., Blot, W.J., Klotz, J.B., Xu, Z.Y., and Boice, J.D., Jr. 1994. Radon exposure in residences and lung cancer among women: combined analysis of three studies. *Cancer Causes Control*, 5(2):114-128.

194. Friendenreich, C. 1993. Methods for pooled analyses of epidemiologic studies. *Epidemiology*, 4:295-302.

195. United States Department of Energy and The Commission of European Committees. 1989. Workshop on Residential Radon Epidemiology. CONF 8907178. Springfield, Virginia :.

196. United States Department of Energy and The Commission of European Committees. 1991. Report of the International Workshop on Residential Radon. CONF 9107220. Springfield, Virginia :.

197. United States Department of Energy and The Commission of European Committees. 1995. Third International DOC/CEC Residential Radon Workshop, February 1995 (Part I). DOE/ER - 0668. Springfield, Virginia :.

198. Kendall, G.M. and Smith, T.J. 2002. Doses to organs and tissues from radon and its decay products. *J.Radiol.Prot.*, 22(4):389-406.

199. Lucie, N.P. 1989. Radon exposure and leukemia. *Lancet*, 2(8654):99-100.

200. Henshaw, D.L., Eatough, J.P., and Richardson.R.B. 1990. Radon as a causative factor in induction of myeloid leukemia and other cancers. *Lancet*, 335:1008-1012.

201. Henshaw, D.L., Eatough, J.P., and Richardson.R.B. 1992. Is radon a causative factor in inducing myeloid leukemia and other cancers in adults and children? In: *Indoor radona nd Lung Cancer: Reality or Myth?* F.T. Cross, (ed.). Columbus, OH; Richland, WA: Battelle Press. pp. 935-958.

202. Lyman, G.H., Lyman, C.G., and Johnson, W. 1985. Association of leukaemia with radium groundwater contramination. *JAMA*, 254:621-626.

203. Alexander, F.E. 1990. Radon and leukemia (Letter). *Lancet*, 335(1336):1337.

204. Butland, B.K., Muirhead, C.R., and Draper, G.J. 1990. Radon and leukaemia (Letter). *Lancet*(335):1338-1339.

205. Lucie, N.P. 1990. Radon and acute lymphoblastic leukaemia. *Leukaemia and lymphoma*, 1:213-216.

206. Collman, G.W., Loomis, D.P., and Sandler, D.P. 1991. Childhood cancer mortality and radon concentration in drinking water in North Carolina. *Br.J.Cancer*, 63:626-629.

207. Forastiere, F., Quercia, A., Cavariana, F., Miceli, M., Biggeri, A., and Axelson, O. 1992. Cancer risk and radon exposure. *Lancet*, 339:1115.

208. Muirhead, C.R., Butland, B.K., Green, B.M., and Draper, G.J. 1992. Childhood leukaemia and natural radiation in Britain. *Lancet*, 337:503-504.

209. Mifune, M., Sobue, T., Arimoto, H.G., Komoto, Y., Kundo, S., and Tanooka, H. 1992. Cancer mortality survey in a spa area (Misasa, Japan) with a high radon background. *Japan J of Cancer Res*, 83:1-5.

210. Hoffmann, W., Kranefield, A., and Schmitz-Feuerhake, I. 1993. Radium-226 contaminated drinking water: hypothesis on exposure pathway in a population with elevated childhood leukaemia. *Environ.Health Perspect.*, Supplement 101:113-115.

211. Cohen, B.L. 1993. Relationship between exposure to radon and various types of cancer. *Health Phys.*, 65(5):529-531.

212. Viel, J. 1993. Radon exposure and leukaemia in childhood. *International Journal of Epidemiology*, 22:627-631.

213. Richardson, R.B., Monfort, C., Green, B.M., Draper, G.J., and Muirhead, C.R. 1995. Spatial variation of natural radiation and leukaemia incidence in Great Britain. *British Journal of Radiology*, 64:608-624.

214. Thorne, R., Foreman, N., and Mott, M. 1996. Radon in Devon and Cornwall and peadiatric malignancies. *European Journal of Cancer*, 32A(2):282-285.

215. Kohli, S., Brage, H., and Lofman, O. 2000. Childhood leukaemia in areas with different radon levels: a spatial and temporal analysis using GIS. *Journal of Epidemiology and Community Health*, 54:822-826.

216. Mole, R.H. 1990. Radon and leukemia (Letter). *Lancet*, 335:1336.

217. Bowie, S.H.U. 1990. Radon and leukaemia (Letter). *Lancet*, 335:1336.

218. Prentice, A.G. and Copplestone, J.A. 1990. Radon and leukaemia (Letter). *Lancet*, 335:1337.

219. Baverstock, K.F. 1990. Radon and leukaemia (Letter). *Lancet*, 335:1337-1338.

220. Peto, J. 1990. Radon and the risk of cancer. *Nature*, 345:389-390.

221. Wolff, S.P. 1991. Leukaemia risks and radon. *Nature*, 352:288.

222. Stjernfeldt, M., Samuelsson, L., and Ludvigsson, J. 1987. Radiation in dwellings and cancer in children. *Paediatric Haematology and Oncology*, 4:55-61.

223. Wakefield, M. and Kohler, J. 1991. Indoor radon and childhood cancer. *Lancet*, 338:1537-1538.

224. Pobel, D. and Viel, J. 1997. Case-control study of leukaemia among young people near La Hague nuclear reprocessing plant: the environmental hypothesis revisited. *British Medical Journal*, 314:101-106.

225. Forastiere, F., Sperati, A., Cherubini, G., Miceli, M., Biggeri, A., and Axelson, O. 1998. Adult myeloid leukaemia, geology and domestic exposoure to radon and gamma radiation. *Occupational and Environmental Medicine*, 55:106-110.

226. Lubin, J.H., Linet, M., Boice, J.D., Jr., Buckley, J., Conrath, S., Hatch, E., Kleinerman, R., Tarone, R., Wacholder, S., and Robison, L. 1998. Case-control study of childhood acute lymphoblastic leukaemia and residential radon exposure. *J Natl Cancer Inst*, 90(4):294-300.

227. Law, G., Kane, E., Roman, E., Smith, A., and Cartwright, R. 2000. Residential radon exposure and adult acute leukaemia. *Lancet - Research Letters*, 355:1888.

228. Tomasek, L., Darby, S.C., Swerdlow, A.J., Placek, V., and Kunz, E. 1993. Radon exposure and cancers other than lung cancer among uranium miners in West Bohemia. *Lancet*, 341(8850):919-923.

229. Darby, S.C., Whitley, E., Howe, G.R., Hutchings, S.J., Kusiak, R.A., Lubin, J.H., Morrison, H.I., Tirmarche, M., Tomasek, L., Radford, E.P., and . 1995. Radon and cancers other than lung cancer in underground miners: a collaborative analysis of 11 studies. *J.Natl.Cancer Inst.*, 87(5):378-384.

230. Drummond, M., O'Brien, B., Stoddart, G., and Torrance, G. 1997. *Methods for the Economic Evaluation of Health Care Programmes (Second Edition).* Oxford University Press.

231. Åkerman, J. 1988. Economic Valuation of Risk Reduction; The Case of In-door Radon. EFI Research Report, ISBN 91 7258 266-9.: Stockholm School of Economics.

232. Åkerman, J., Reed Johnson, F., and Bergman, L. 1991. Paying for Safety: Voluntary Reduction of Residential Radon Risks. *Land Economics*, 67(4):435-46.

233. Spiegel, J. 2000. A Comparison of Economic Valuation Methods for Environmental Health Risk Reduction: Assessing Residential Radon Mitigation in Manitoba. Thesis, Ph.D., University of Manitoba.

234. Spiegel, J. and Krewski, D. 2002. Using Willingness to Pay to Evaluate the Implementation of Canada's Residential Radon Exposure Guideline. *Canadian Journal of Public Health*, 93(3):223-228.

235. Kennedy, C.A. 2000. A Revealed Preference Approach to Valuations of Risk Reductions: A Contractor's Sample. *Proceedings of the 10th International Congress of the International Radiation Protection Association.*May 14-19, 2000, Hiroshima. pp. P-1b-45.

236. Drummond, M. 1980. *Principles of economic appraisal in health care.* Oxford: Oxford University Press.

237. Warner, K.E., Hutton, R.C., and Luce, B.R. 1981. *Cost-benefit and cost-effectiveness in health care: principles, practice and potential.* Ann Arbor, Michigan: Health Administration Press.

238. Luce, B. and Elixhauser, A. 1990. *Standards for socioeconomic evaluation of health care products and services.* Berlin Heidelberg: Springer-Verlag.

239. Gold, M.R., Siegel, J.E., Russell, L.B., and Weinstein, M.C. 1996. *Cost-effectiveness in health and medicine.*

240. Letourneau, E.G. 1985. Limitation of exposure to natural radioactivity in Canada. *Science of the Total Environment*, 45:647-656.

241. Létourneau, E. 1985. A Radon Standard for Canada. *Transactions of Indoor Ait Quality in Cold Climates: Hazards and Abatement Measures*:488-195.

242. Proskiw, G. 1992. The Performance and Costs of Residential Radon Mitigation Measures for Manitoba. Winnipeg: Manitoba Environment.

243. Létourneau, E., Krewski, D., Zielinski, J., and McGregor, R. 1992. Cost Effectiveness of Radon Mitigation in Canada. *Radiation Protection Dosimetry*, 45(1):593-598.

244. Moeller, D.W. and Fujimoto, K. 1984. Cost evaluation of control measures for indoor radon progeny. *Health Physics*, 46(6):1181-1193.

245. Bierma, T.J. 1990. A Cost Effectiveness Analysis of Policies for Reducing the Risks from Indoor Radon. *Dissertation Abstracts International: Section B: the Sciences & Engineering*, 51(4).

246. Mossman, K.L. and Sollitto, M.A. 1991. Regulatory control of indoor Rn. *Health Phys.*, 60(2):169-176.

247. United States Environmental Protection Agency (EPA). 1992. Technical Support Document for the 1992 Citizen's Guide to Radon. EPA publication 400-R-92-011. Washington, D.C.: USEPA.

248. Marcinowski, F. and Napolitano, S. 1993. Reducing the risks from radon. *J.Air Waste Manage.Assoc.*, 43(7):955-962.

249. Bogen, K.T. and Layton, D.W. 1998. Risk management for plausibly hormetic environmental carcinogens: the case of radon. *Human & Experimental Toxicology*, 17(8):463-467.

250. Dornsife, W.P. and Bhattacharyya, A. 1998. A risk based methodology for assessing the efficacy and priorities for the control of various radiation sources. [erratum appears in Health Phys 1998 Mar;74(3):399.]. *Health Physics*, 74(2):190-215.

251. Ford, E.S., Kelly, A.E., Teutsch, S.M., Thacker, S.B., and Garbe, P.L. 1999. Radon and lung cancer: a cost-effectiveness analysis. *Am.J.Public Health*, 89(3):351-357.

252. Lin, C.Y., Gelman, A., Price, P.N., and Krantz, D.H. 1999. Analysis of Local Decision Using Hierarchical Modeling, Applied to Home Radon Measurement and Remediation. *Statistical Science*, 14(3):305-337.

253. Denman, A., Barker, S., Parkinson, S., and Phillips, P. 1997. The health benefits and cost effectiveness of the radon mitigation programme in NHS properties in Northamptonshire. *Journal of Radiological Protection*, 17(4):253-259.

254. Denman, A.R. and Phillips, P.S. 1998. A review of the cost effectiveness of radon mitigation in domestic properties in Northamptonshire. *Journal of Radiological Protection*, 18(2):119-124.

255. Kennedy, C.A., Gray, A.M., Denman, A.R., and Phillips, P.S. 1999. A cost-effectiveness analysis of a residential radon remediation programme in the United Kingdom. *Br.J.Cancer*, 81(7):1243-1247.

256. Denman, A.R., Phillips, P.S., and Tornberg, R. 2000. A Comparative Review of the Effectiveness of Radon Remediation Programmes in Hospitals, Schools and Homes in Northamptonshire, UK. *Hiroshima*, Proceedings of the 10th IRPA Congress.

257. Denman, A.R., Phillips, P.S., and Tornberg, R. 2000. A comparison of the costs and benefits of radon remediation programmes in new and existing houses in Northamptonshire. *Journal of Environmental Management*, 59:21-30.

258. Coskeran, T., Denman, A., and Phillips, P. 2001. The costs of radon mitigation in domestic properties. *Health Policy*, 57(2):97-109.

259. Denman, A., Fraser, J., and Phillips, P.S. 2000. An Evaluation of the Effectiveness of the UK Programme to Protect new buildings in Radon Affected Areas. *Proceedings of the 10th International Congress of the International Radiation Protection Agency.*May 14-19, 2000, Hiroshima pp. P-1b-29.

260. Denman, A.R. and Phillips, P.S. 1998. The cost-effectiveness of radon mitigation in schools in Northamptonshire. *Journal of Radiological Protection*, 18(3):203-208.

261. Denman, A.R., Kennedy, C., and Phillips, P.S. 1999. Recent Developments in Evaluating Radon Remediation Strategies. *Proceedings of the 6th International Symposium of the Society for Radiological Protection.*June 14-18, 1999, Southport pp. 3-2.

262. Kennedy, C.A. and Gray, A.M. 2000. The Cost-Effectiveness of Radon-Induced Lung Cancer Prevention in Schools. *Int J of Environ Health Res*, 10(3):181-190.

263. Denman, A.R., Phillips, P.S., and Tornberg, R. 2001. The cost effectiveness of radon remediation programmes in hospitals, schools and homes in radon affected areas in the UK. *Science of the Total Environment*, 272(1-3):23-24.

264. Denman, A.R., Phillips, P.S., and Tornberg, R. 2002. The costs and benefits of radon remediation programmes in existing homes: case study of action level selection. *Journal of Environmental Radioactivity*, 62(1):17-27.

265. Kennedy, C.A., Gray, A.M., Denman, A.R., and Phillips, P.S. 2002. The cost-effectiveness of residential radon remediation programmes: assumptions about benefits stream profiles over time. *J.Environ.Radioact.*, 59(1):19-28.

266. Coskeran, T., Denman, A.R., Phillips, P.S., and Gillmore, G.K. 2002. A critical comparison of the cost-effectiveness of domestic radon remediation programmes in three counties of England. *Journal of Environmental Radioactivity*, 62(2):129-144.

267. Colgan, P. and Guitiérrez, J. 1995. Justification and optimisation in the choice of Reference Levels for radon in existing Spanish dwellings. *Journal of Radiological Protection*, 15(4):289-301.

268. Colgan, P. and Guitiérrez, J. 1996. Cost effectiness of reducing radon exposure in Spanish dwellings. *Journal of Radiological Protection*, 16(3):181-190.

269. Monchecourt, H.D. 2000. Efficiency of Radon Reduction Techniques and Strategies. *Proceedings of the 10th International Congress of the International Radiation Protection Agency.*May 14-19, 2000, Hiroshima pp. P-1b-34.

270. Maringer, F.J., Akis, M.G., Kaineder, H., Kindl, P., Kralik, C., Lettner, H., Lueginger, S., Nadschlager, E., Ringer, W., Rolle, R., Schonhofer, F., Sperker, S., Stadtmann, H., Steger, F., Steinhausler, F., Tschurlovits, M., and Winkler, R. 2001. Results and conclusions of the Austrian radon mitigation project 'SARAH'. *Science of the Total Environment*, 272(1-3):159-167.

271. Snihs, J. 1992. Swedish radon programme. *Radiat.Prot.Dosim.*, 42:177-184.

272. Reid, G.W., Lassovszky, P., and Hathaway, S. 1985. Treatment, waste management and cost for removal of radioactivity from drinking water. *Health Physics*, 48(5):671-694.

273. Kukacka, L. and Isler, R.J. 1978. Cost Estimate for Remedial Action Program for Residence in the Grand Junction, Colorado Area. Upton, NY: BNL Report No. 17433, Brookhave National Laboratory.

274. Bergman, L., Söderqvist, T., and Åkerman, J. 2003. Subjective Valuation of Radon Radiation Reduction. *British Journal of Cancer.*

275. Torrance, G. and Krewski, D. 1985. Economic Evaluation of Toxic Chemical Control Programs. *Toxic Substances Journal*, 7:53-71.

276. Canada Mortgage and Housing Corporation. Canadian Housing Observer 2003. CMHC, Ottawa (2003).

277. Statistics Canada. Census of Population, 2001.

278. Létourneau, E., Krewski, D., Zielinski, J., and McGregor, R. 1992. Cost Effectiveness of Radon Mitigation in Canada. *Radiation Protection Dosimetry*, 45(1):593-598.

279. Microsoft Corporation. 2002.

280. Decisionerring, Inc. 2002.

281. Statistics Canada. Census of Population, 2001.

282. Statistics Canada. Census of Population, 2001.

283. Canada Mortgage and Housing Corporation. Canadian Housing Observer 2003. CMHC, Ottawa (2003).

284. Brand, K. 2004. Approximations and Heuristics for the Cause-Modified Life Table. *Risk Analysis (accepted).*

285. Statistics Canada, CAN-SIM II Table 051-0011.

286. Statistics Canada. CAN-SIM II Table 051-0001

287. Villeneuve, P. and Mao, Y. 1994. Lifetime probablity of developing lung cancer by smoking status, Canada. *Can.J.of Public Health*, 85(6):385-388.

288. Mattson, M., Pollack, E., and Cullen, J. 1987. What are the odds that smoking will kill you? *Am.J.Public Health*, 77(3):425-431.

289. Statistics Canada. National Population Health Survey (NPHS) 1996-97.

290. American Cancer Society (ACS) Cohort Study, 1997 estimates.

291. Reed, J.L. and Merrell, M. 1939. A short method for constructing an abridged life table. *Am.J.Hyg.*, 30:33-62.

292. Greville, T.N.E. 1943. Short methods of constructing abridged life tables. *Rec.Am.Inst.Actuar.*, 32:29-43.

293. Keyfitz, N. and Frauenthal, J. 1975. An improved life table method. *Biometrics*, 31:889-899.

294. Chiang, C.L. 1984. *The Lifetable and Its Applications.* Malabar, FL: Krieger Publishing.

295. Cause-specific mortality rates obtained from: Statistics Canada. CAN-SIM II Table 102-0522.

296. Chiang, C.L. 1991. Competing Risks in Mortality Analysis. *Annu.Rev.of Publ.Health*, 12:281-307.

297. Statistics Canada. Catalogue No. 82F0075XCB.

298. Statistics Canada. Catalogue No. 82F0075XCB.

299. Statistics Canada. Census of Population, 2001.

300. Statistics Canada. CAN-SIM II Table 051-0011.

301. Personal communication. Statistics Canada, Demography Division;. March 2004

302. Statistics Canada, 2004.

303. Personal Communication, D. Krewski, Director, MgLaughlin Centre for Population Health Risk Assessment, March 2004.

304. Estimates provided by C. Halliwell, Director General, Applied Research, Human Resources Development Canada

305. Evans, W.K., Will, B.P., Berthelot, J.M., and Wolfson, M.C. 1995. Estimating the cost of lung cancer diagnosis and treatment in Canada: the POHEM model. *Can.J.Oncol.*, 5(4):408-419.

306. Torrance, G. 1992. Economic Evaluation Methods for the Determination of the Cost-Effectiveness and Cost-Utility of Radon Mitigation in Canada. Ottawa: Health Canada.

307. Coyle, D., Barbeau, M., and Baladi, J.-F. 2004. Using Probabilistic Analysis to Assess the Cost Effectiveness of Health Interventions: An Application to the Treatment of Parkinson's Disease. *International Journal of Technology Assessment in Health Care*, (submitted).

308. Arrow, K. and Lind, R. 1970. Uncertainty and the Evaluation of Public Investment Decisions. *American Economic Review*, 60:364-78.

309. Doubilet, P., Weinstein, M.C., and McNeil, B.J. 1986. Use and misuse of the term "cost effective" in medicine. *N.Engl.J.Med.*, 314(4):253-256.

310. Tracy, B., Krewski, D., Chen, J., Zielinski, J.M., Brand, K., and Meyerhof, D. 2004. Assessment and Management of Residential Radon Health Risks: a Report from the Health Canada Radon Workshop. *Journal of Toxicology and Environmental Health (In press).*

311. Bradley, E. and Thomas, J. 1996. An Analysis of Responses to Radon Remediation Advice. *NRPB-M707,*

312. Laupacis, A., Feeny, D., Detsky, A.S., and Tugwell, P.X. 1992. How attractive does a new technology have to be to warrant adoption and utilization? Tentative guidelines for using clinical and economic evaluations. *CMAJ.*, 146(4):473-481.

313. Tengs, T.O., Adams, M.E., Pliskin, J.S., Safran, D.G., Siegel, J.E., Weinstein, M.C., and Graham, J.D. 1995. Five-hundred life-saving interventions and their cost-effectiveness. *Risk Anal.*, 15(3):369-390.

314. Graham, J.D., Corso, P.S., Morris, J.M., Segui-Gomez, M., and Weinstein, M.C. 1998. Evaluating the cost-effectiveness of clinical and public health measures. *Annu.Rev.Public Health*, 19:125-152.

315. Graham, J.D., Thompson, K.M., Goldie, S.J., Segui-Gomez, M., and Weinstein, M.C. 1997. The cost-effectiveness of air bags by seating position. *JAMA*, 278(17):1418-1425.

316. Tengs, T.O., Adams, M.E., Pliskin, J.S., Safran, D.G., Siegel, J.E., Weinstein, M.C., and Graham, J.D. 1995. Five-hundred life-saving interventions and their cost-effectiveness. *Risk Anal.*, 15(3):369-390.

317. Tengs, T.O., Adams, M.E., Pliskin, J.S., Safran, D.G., Siegel, J.E., Weinstein, M.C., and Graham, J.D. 1995. Five-hundred life-saving interventions and their cost-effectiveness. *Risk Anal.*, 15(3):369-390.

318. Krewski, D., Gaylor, D., and Szyszkowicz, M. 1991. A model-free approach to low dose extrapolation. *Environmental Health Perspectives*, 90:279-285.

319. Health Canada. 2004. Canadian Tobacco Use Monitoring Survey (CTUMS), 1999-2003.

320. Katz, D.A. and Welch, H.G. 1993. Discounting in cost-effectiveness analysis of healthcare programmes. *Pharmacoeconomics.*, 3(4):276-285.

321. West, R.R. 1996. Discounting the future: influence of the economic model. *J.Epidemiol.Community Health*, 50(3):239-244.

322. Parsonage, M. and Neuburger, H. 1992. Discounting and health benefits. *Health Econ.*, 1(1):71-76.

323. Gravelle, H. and Smith, D. 2001. Discounting for health effects in cost-benefit and cost-effectiveness analysis. *Health Econ.*, 10(7):587-599.

324. Keeler, E.B. and Cretin, S. 1983. Discounting of Life-Saving and Other Nonmonetary Effects. *Management Science*, 29(3):300-306.

325. Coyle, D. and Tolley, K. 1992. Discounting of health benefits in the pharmacoeconomic analysis of drug therapies: an issue for debate? *Pharmacoeconomics.*, 2(2):153-162.

APPENDIX A
SUPPLEMENTARY RESULTS TABLES

This section provides tables containing all outputs of the decision-model, upon which the results in Chapter 3 were based. The data tables are divided here by 'simulation', as follows.

No.	Compliance Rate	Reduction in Radon Levels	Radon Distribution	Page
1	50%	90%	Canada	159
2	10%	90%	Canada	164
3	100%	90%	Canada	169
4	50%	70%	Canada	174
5	10%	70%	Canada	179
6	100%	70%	Canada	184
7	50%	90%	'Sudbury'	189
8	50%	90%	'Winnipeg'	194

Each section contains the results for simulation of all five national mitigation strategies under the conditions listed in the above table, replicated for the 3 action levels (800, 400 and 200 Bqm^{-3}) and 3 annual discount rates (0, 5, and 10% per year) considered in this analysis.

The results are presented in the following order.

- Net Total Cost (Millions)
- Initial Screen Costs per 'At-Risk' Home Identified
- % of Total Cost (Gross), Screening
- % of Total Cost (Gross), Mitigation
- % of Total Cost (Gross), Upkeep
- % of Total Cost (Gross), Program Administration
- Lung Cancer Deaths Prevented, by Smoking Status
- Life-Years (LY) Gained, by Smoking Status
- Quality-Adjusted Life-Years (QALY) Gained, by Smoking Status
- Cost per Lung Cancer Death Prevented
- Cost per Life-Year (LY) Gained
- Cost per Quality-Adjusted Life-Year (QALY) Gained

Simulation 1: 50% Homeowner Compliance with Radon Guideline
90% Reduction in Radon Levels Within Mitigated Homes
Indoor Radon Distribution: Canada ~LN(11.2 Bqm^{-3}, 3.9)

TABLE A-1. Net Total Cost ($Millions) – Simulation 1

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$462	$370	$334
	400	$633	$442	$380
	200	$1,240	$661	$556
2	800	$574	$112	$68
	400	$667	$115	$68
	200	$1,065	$155	$88
3	800	$972	$467	$394
	400	$1,236	$543	$439
	200	$2,241	$801	$636
4	800	$670	$191	$123
	400	$831	$220	$136
	200	$1,430	$316	$195
5	800	$861	$257	$169
	400	$1,078	$288	$181
	200	$1,840	$411	$255

TABLE A-2. Initial Screen Costs per 'At-Risk' Home Identified – Simulation 1

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$39,492	$35,904	$32,933
	400	$7,898	$7,181	$6,587
	200	$2,338	$2,126	$1,950
2	800	$34,829	$6,938	$4,249
	400	$8,878	$1,768	$1,083
	200	$2,434	$485	$297
3	800	$36,729	$18,741	$15,937
	400	$8,420	$4,296	$3,654
	200	$2,391	$1,220	$1,038
4	800	$42,128	$12,390	$7,996
	400	$8,986	$2,643	$1,705
	200	$2,436	$716	$462
5	800	$39,676	$12,314	$8,109
	400	$8,715	$2,705	$1,781
	200	$2,439	$757	$498

Note: The results presented in Table A-2 are identical for simulations 1-6, and will therefore only be presented again for simulations 7 and 8.

TABLE A-3. % of Total Cost (Gross), Screening – Simulation 1

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	74.39%	90.60%	92.98%
	400	49.86%	74.41%	78.91%
	200	25.55%	49.32%	55.35%
2	800	81.57%	84.83%	85.64%
	400	68.53%	81.77%	84.06%
	200	41.62%	59.48%	65.92%
3	800	83.15%	91.99%	93.74%
	400	61.59%	77.87%	81.18%
	200	33.45%	52.13%	57.55%
4	800	84.37%	88.42%	88.71%
	400	65.30%	75.62%	77.68%
	200	37.08%	51.92%	55.82%
5	800	84.94%	90.38%	91.05%
	400	65.12%	79.44%	82.02%
	200	37.22%	54.82%	59.86%

TABLE A-4. % of Total Cost (Gross), Mitigation – Simulation 1

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	2.22%	2.66%	2.68%
	400	7.15%	10.71%	11.39%
	200	12.06%	23.22%	25.96%
2	800	1.75%	1.04%	0.91%
	400	5.46%	2.92%	1.94%
	200	11.74%	13.95%	12.69%
3	800	2.09%	2.35%	2.43%
	400	6.63%	9.36%	10.16%
	200	12.22%	21.83%	24.48%
4	800	1.51%	1.96%	2.61%
	400	6.44%	9.16%	9.90%
	200	13.44%	22.10%	24.10%
5	800	1.66%	1.91%	2.27%
	400	6.57%	7.88%	8.08%
	200	12.73%	20.38%	21.68%

TABLE A-5. % of Total Cost (Gross), Upkeep – Simulation 1

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	10.64%	2.89%	1.77%
	400	34.45%	11.72%	7.53%
	200	58.01%	25.37%	17.15%
2	800	5.78%	1.32%	0.84%
	400	16.86%	2.96%	1.63%
	200	41.08%	17.59%	11.69%
3	800	8.52%	2.61%	1.65%
	400	27.16%	10.19%	6.77%
	200	51.82%	24.31%	16.63%
4	800	4.78%	2.12%	1.72%
	400	21.02%	8.81%	6.33%
	200	45.38%	21.57%	15.69%
5	800	6.20%	2.15%	1.60%
	400	22.78%	7.79%	5.33%
	200	46.89%	21.42%	15.12%

TABLE A-6. % of Total Cost (Gross), Program Administration – Simulation 1

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	12.74%	3.84%	2.57%
	400	8.54%	3.16%	2.18%
	200	4.38%	2.09%	1.53%
2	800	10.90%	12.81%	12.61%
	400	9.15%	12.35%	12.37%
	200	5.56%	8.98%	9.70%
3	800	6.24%	3.05%	2.18%
	400	4.62%	2.58%	1.89%
	200	2.51%	1.73%	1.34%
4	800	9.34%	7.50%	6.96%
	400	7.23%	6.41%	6.09%
	200	4.11%	4.40%	4.38%
5	800	7.21%	5.56%	5.08%
	400	5.53%	4.89%	4.58%
	200	3.16%	3.37%	3.34%

TABLE A-7. Lung Cancer Deaths Prevented – Simulation 1

Strategy	AL	0% Discount Rate			5% Discount Rate			10% Discount Rate		
		Total	Ever	Never	Total	Ever	Never	Total	Ever	Never
1	800	1,775	1,498	278	242	204	38	62	53	10
	400	5,111	4,311	799	673	568	105	169	143	26
	200	9,797	8,265	1,532	1,298	1,095	203	324	274	50
2	800	641	540	101	41	35	6	5	4	1
	400	1,456	1,226	230	84	71	13	9	8	1
	200	3,858	3,249	608	260	219	41	34	28	5
3	800	2,417	2,038	379	282	238	44	68	57	11
	400	6,566	5,537	1,029	757	638	118	178	150	28
	200	13,656	11,516	2,141	1,557	1,313	244	358	302	56
4	800	692	584	109	76	64	12	18	15	3
	400	2,426	2,045	381	237	200	37	46	39	7
	200	5,715	4,817	898	544	459	85	102	86	16
5	800	1,253	1,056	197	115	97	18	23	19	4
	400	3,587	3,023	564	309	260	49	55	46	9
	200	8,258	6,960	1,298	730	615	115	129	109	20

TABLE A-8. Life-Years (LY) Gained – Simulation 1

Strategy	AL	0% Discount Rate			5% Discount Rate			10% Discount Rate		
		Total	Ever	Never	Total	Ever	Never	Total	Ever	Never
1	800	26,212	16,787	9,425	2,298	1,510	788	383	260	123
	400	75,121	48,135	26,986	6,381	4,193	2,188	1,034	702	332
	200	144,848	92,778	52,070	12,316	8,093	4,223	1,986	1,347	638
2	800	6,547	4,322	2,226	329	220	110	31	21	10
	400	14,258	9,451	4,808	649	434	214	52	35	16
	200	41,779	27,440	14,339	2,137	1,423	714	197	135	63
3	800	32,760	21,109	11,651	2,627	1,729	898	413	281	133
	400	89,382	57,588	31,794	7,030	4,628	2,402	1,086	737	349
	200	186,642	120,228	66,413	14,453	9,516	4,937	2,183	1,482	701
4	800	9,052	5,848	3,205	703	463	240	107	73	34
	400	31,522	20,390	11,132	2,146	1,416	730	280	190	90
	200	73,887	47,818	26,069	4,903	3,237	1,666	618	420	198
5	800	15,258	9,917	5,340	1,024	677	347	137	93	44
	400	43,550	28,324	15,227	2,720	1,799	920	328	223	105
	200	102,911	66,796	36,115	6,478	4,283	2,195	773	525	247

TABLE A-9. Quality-Adjusted Life-Years (QALY) Gained – Simulation 1

Strategy	AL	0% Discount Rate			5% Discount Rate			10% Discount Rate		
		Total	Ever	Never	Total	Ever	Never	Total	Ever	Never
1	800	22,286	14,323	7,963	1,968	1,297	671	330	224	105
	400	63,884	41,079	22,805	5,464	3,603	1,862	891	607	285
	200	123,171	79,171	44,000	10,546	6,952	3,594	1,712	1,165	547
2	800	5,602	3,710	1,892	283	189	93	26	18	8
	400	12,210	8,119	4,091	557	374	183	45	31	14
	200	35,718	23,536	12,182	1,832	1,224	608	170	116	54
3	800	27,888	18,033	9,855	2,250	1,486	764	356	243	114
	400	76,096	49,199	26,897	6,021	3,977	2,044	936	637	299
	200	158,902	102,715	56,186	12,379	8,176	4,202	1,882	1,281	600
4	800	7,709	4,997	2,712	602	398	204	92	63	29
	400	26,865	17,437	9,428	1,838	1,217	621	241	164	77
	200	62,982	40,899	22,083	4,200	2,782	1,418	532	363	170
5	800	13,014	8,488	4,526	877	582	296	118	81	38
	400	37,159	24,248	12,911	2,330	1,547	784	283	193	90
	200	87,777	57,165	30,612	5,549	3,681	1,869	666	454	212

TABLE A-10. Cost per Lung Cancer Death Prevented – Simulation 1

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$260,384	$1,531,913	$5,355,189
	400	$123,952	$656,866	$2,253,117
	200	$126,585	$509,082	$1,716,303
2	800	$894,738	$2,727,767	$13,000,834
	400	$458,161	$1,372,773	$7,488,865
	200	$275,945	$596,632	$2,613,458
3	800	$402,131	$1,654,229	$5,820,952
	400	$188,268	$717,039	$2,471,027
	200	$164,066	$514,375	$1,776,633
4	800	$967,943	$2,498,012	$7,036,952
	400	$342,437	$927,020	$2,942,801
	200	$250,290	$580,232	$1,899,569
5	800	$686,820	$2,233,097	$7,444,848
	400	$300,491	$933,091	$3,320,354
	200	$222,872	$563,371	$1,983,326

TABLE A-11. Cost per Life-Year (LY) Gained – Simulation 1

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$17,637	$161,022	$873,360
	400	$8,433	$69,267	$367,864
	200	$8,562	$53,634	$280,276
2	800	$87,598	$339,251	$2,230,841
	400	$46,771	$177,342	$1,309,024
	200	$25,480	$72,467	$446,310
3	800	$29,663	$177,873	$952,985
	400	$13,831	$77,182	$404,800
	200	$12,004	$55,418	$291,329
4	800	$74,040	$271,483	$1,154,896
	400	$26,360	$102,423	$486,802
	200	$19,360	$64,380	$314,832
5	800	$56,412	$250,863	$1,232,025
	400	$24,751	$105,975	$552,916
	200	$17,884	$63,501	$330,156

TABLE A-12. Cost per Quality-Adjusted Life-Year (QALY) Gained – Simulation 1

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$20,744	$188,041	$1,012,982
	400	$9,916	$80,889	$426,707
	200	$10,068	$62,636	$325,123
2	800	$102,385	$395,554	$2,591,036
	400	$54,617	$206,658	$1,520,408
	200	$29,803	$84,516	$518,364
3	800	$34,845	$207,679	$1,105,449
	400	$16,246	$90,115	$469,582
	200	$14,100	$64,706	$337,982
4	800	$86,943	$316,964	$1,339,830
	400	$30,929	$119,576	$565,040
	200	$22,711	$75,160	$365,469
5	800	$66,136	$292,793	$1,429,691
	400	$29,009	$123,685	$641,851
	200	$20,968	$74,122	$383,299

Simulation 2: 10% Homeowner Compliance with Radon Guideline
90% Reduction in Radon Levels Within Mitigated Homes
Indoor Radon Distribution: Canada ~LN(11.2 Bqm^{-3}, 3.9)

TABLE A-13. Net Total Cost ($Millions) – Simulation 2

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$450	$358	$323
	400	$486	$374	$334
	200	$603	$418	$371
2	800	$621	$116	$70
	400	$869	$139	$81
	200	$1,777	$228	$122
3	800	$938	$454	$382
	400	$1,002	$470	$392
	200	$1,193	$521	$432
4	800	$659	$189	$121
	400	$696	$194	$123
	200	$811	$213	$133
5	800	$840	$253	$166
	400	$893	$259	$168
	200	$1,051	$283	$180

TABLE A-14. % of Total Cost (Gross), Screening – Simulation 2

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	82.97%	94.91%	96.55%
	400	74.35%	90.50%	92.84%
	200	57.82%	80.17%	83.81%
2	800	76.60%	82.35%	83.64%
	400	54.47%	68.70%	72.04%
	200	26.66%	41.74%	47.89%
3	800	90.41%	95.90%	97.06%
	400	82.83%	92.20%	93.85%
	200	67.77%	82.51%	85.44%
4	800	87.61%	90.11%	90.39%
	400	81.82%	87.15%	88.30%
	200	68.97%	79.01%	82.00%
5	800	89.26%	92.48%	92.92%
	400	82.70%	90.03%	91.29%
	200	68.81%	81.78%	85.29%

TABLE A-115. % of Total Cost (Gross), Mitigation – Simulation 2

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	0.50%	0.51%	0.47%
	400	2.22%	2.70%	2.77%
	200	5.53%	7.83%	8.35%
2	800	3.05%	2.35%	2.10%
	400	8.52%	9.11%	8.94%
	200	15.20%	22.52%	23.24%
3	800	0.55%	0.46%	0.41%
	400	2.08%	2.32%	2.40%
	200	5.29%	7.05%	7.53%
4	800	0.56%	1.07%	1.52%
	400	2.10%	2.80%	2.92%
	200	5.22%	7.38%	7.08%
5	800	0.61%	0.91%	1.14%
	400	2.11%	2.32%	2.22%
	200	5.46%	6.70%	5.99%

TABLE A-16. % of Total Cost (Gross), Upkeep – Simulation 2

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	2.32%	0.55%	0.31%
	400	10.69%	2.95%	1.83%
	200	26.75%	8.59%	5.52%
2	800	10.11%	2.87%	1.94%
	400	29.73%	11.81%	8.41%
	200	54.57%	29.44%	21.82%
3	800	2.26%	0.46%	0.27%
	400	8.87%	2.42%	1.57%
	200	21.85%	7.71%	5.04%
4	800	2.13%	1.17%	1.00%
	400	7.03%	2.66%	1.86%
	200	18.17%	6.92%	4.48%
5	800	2.55%	0.92%	0.75%
	400	8.18%	2.11%	1.40%
	200	19.89%	6.48%	3.96%

TABLE A-17. % of Total Cost (Gross), Program Administration – Simulation 2

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	14.21%	4.03%	2.67%
	400	12.74%	3.84%	2.56%
	200	9.90%	3.40%	2.31%
2	800	10.23%	12.44%	12.31%
	400	7.28%	10.38%	10.61%
	200	3.56%	6.30%	7.05%
3	800	6.79%	3.18%	2.26%
	400	6.22%	3.05%	2.18%
	200	5.09%	2.73%	1.99%
4	800	9.70%	7.64%	7.09%
	400	9.06%	7.39%	6.93%
	200	7.64%	6.70%	6.43%
5	800	7.57%	5.69%	5.19%
	400	7.02%	5.54%	5.10%
	200	5.84%	5.03%	4.76%

TABLE A-18. Lung Cancer Deaths Prevented – Simulation 2

Strategy	AL	0% Discount Rate			5% Discount Rate			10% Discount Rate		
		Total	Ever	Never	Total	Ever	Never	Total	Ever	Never
1	800	25	21	4	5	5	1	2	2	0
	400	747	630	117	103	87	16	27	22	4
	200	1,922	1,621	301	259	218	40	65	55	10
2	800	242	204	38	11	9	2	1	1	0
	400	505	425	80	22	19	4	1	1	0
	200	1,010	850	160	58	49	9	6	5	1
3	800	267	225	42	16	14	3	2	2	0
	400	1,253	1,056	197	125	106	20	28	24	4
	200	2,932	2,471	460	317	267	50	71	60	11
4	800	89	75	14	13	11	2	5	4	1
	400	489	412	77	50	42	8	11	9	2
	200	1,247	1,051	196	118	100	19	22	18	3
5	800	292	246	46	23	19	4	5	4	1
	400	892	752	141	68	58	11	12	10	2
	200	2,026	1,707	319	166	140	26	27	23	4

TABLE A-19. Life-Years (LY) Gained – Simulation 2

Strategy	AL	0% Discount Rate			5% Discount Rate			10% Discount Rate		
		Total	Ever	Never	Total	Ever	Never	Total	Ever	Never
1	800	434	274	159	54	35	19	11	8	4
	400	11,218	7,177	4,041	981	644	337	163	110	52
	200	28,732	18,390	10,341	2,461	1,617	844	401	272	129
2	800	2,169	1,452	717	75	51	24	4	2	1
	400	4,444	2,980	1,464	152	104	49	7	5	2
	200	10,223	6,758	3,465	452	303	149	33	23	11
3	800	2,603	1,726	876	129	86	43	15	10	5
	400	15,662	10,158	5,504	1,133	748	385	170	115	54
	200	38,955	25,149	13,806	2,913	1,919	994	434	295	139
4	800	1,068	692	376	126	83	43	29	19	9
	400	6,264	4,054	2,210	452	298	154	65	44	21
	200	16,172	10,463	5,709	1,064	703	362	131	89	42
5	800	3,052	2,010	1,042	197	130	66	32	22	10
	400	10,018	6,558	3,459	583	387	196	71	48	23
	200	24,505	15,946	8,559	1,446	958	488	160	109	51

TABLE A-20. No. Quality-Adjusted Life-Years (QALY) Gained – Simulation 2

Strategy	AL	0% Discount Rate			5% Discount Rate			10% Discount Rate		
		Total	Ever	Never	Total	Ever	Never	Total	Ever	Never
1	800	367	233	134	46	30	16	10	7	3
	400	9,537	6,123	3,413	840	553	286	140	95	45
	200	24,429	15,691	8,738	2,107	1,389	718	346	235	110
2	800	1,862	1,250	612	65	44	21	3	2	1
	400	3,816	2,566	1,249	131	89	42	6	4	2
	200	8,753	5,804	2,948	388	261	127	29	20	9
3	800	2,230	1,484	746	111	74	37	13	9	4
	400	13,353	8,690	4,663	971	643	328	146	100	47
	200	33,183	21,496	11,686	2,495	1,649	846	374	255	119
4	800	908	591	317	108	71	37	25	17	8
	400	5,337	3,466	1,871	387	256	131	56	38	18
	200	13,784	8,948	4,836	911	604	308	113	77	36
5	800	2,610	1,724	885	169	112	56	28	19	9
	400	8,559	5,622	2,937	500	333	167	61	42	19
	200	20,914	13,655	7,259	1,239	823	416	138	94	44

TABLE A-21. Cost per Lung Cancer Death Prevented – Simulation 2

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$17,962,444	$65,951,210	$177,686,955
	400	$650,524	$3,632,053	$12,585,517
	200	$313,813	$1,618,284	$5,666,451
2	800	$2,566,061	$10,628,523	$103,258,192
	400	$1,720,389	$6,200,020	$59,312,856
	200	$1,759,788	$3,924,575	$20,890,065
3	800	$3,514,588	$27,800,584	$152,923,067
	400	$800,207	$3,751,969	$14,060,365
	200	$406,928	$1,645,635	$6,061,312
4	800	$7,404,303	$14,191,742	$26,170,635
	400	$1,425,225	$3,905,580	$11,611,795
	200	$650,296	$1,801,458	$6,157,411
5	800	$2,874,464	$10,993,886	$31,476,239
	400	$1,000,728	$3,789,231	$14,212,787
	200	$519,032	$1,706,761	$6,734,974

TABLE A-22. Cost per Life-Year (LY) Gained – Simulation 2

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$1,038,596	$6,648,165	$28,714,301
	400	$43,331	$380,786	$2,052,162
	200	$20,991	$170,042	$924,561
2	800	$286,187	$1,537,379	$19,801,943
	400	$195,609	$909,333	$11,468,364
	200	$173,786	$503,665	$3,668,515
3	800	$360,528	$3,512,993	$25,815,067
	400	$63,994	$414,465	$2,310,327
	200	$30,623	$178,856	$994,770
4	800	$616,742	$1,497,852	$4,223,382
	400	$111,171	$429,660	$1,911,725
	200	$50,131	$200,006	$1,021,966
5	800	$275,100	$1,287,190	$5,165,474
	400	$89,147	$443,942	$2,371,490
	200	$42,908	$195,645	$1,127,502

TABLE A-23. Cost per Quality-Adjusted Life-Year (QALY) Gained – Simulation 2

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$1,225,652	$7,763,634	$33,285,959
	400	$50,970	$444,691	$2,380,255
	200	$24,689	$198,578	$1,072,417
2	800	$333,376	$1,787,680	$22,977,851
	400	$227,799	$1,057,179	$13,306,270
	200	$202,974	$586,916	$4,261,315
3	800	$420,879	$4,092,215	$29,932,356
	400	$75,060	$483,730	$2,679,730
	200	$35,950	$208,802	$1,153,981
4	800	$725,375	$1,747,654	$4,891,659
	400	$130,468	$501,591	$2,218,004
	200	$58,816	$233,511	$1,186,482
5	800	$321,705	$1,500,282	$5,984,139
	400	$104,340	$517,820	$2,751,482
	200	$50,275	$228,325	$1,309,153

Simulation 3: 100% Homeowner Compliance with Radon Guideline 90% Reduction in Radon Levels Within Mitigated Homes Indoor Radon Distribution: Canada ~LN(11.2 Bqm^{-3}, 3.9)

TABLE A-24. Net Total Cost ($Millions) – Simulation 3

Strategy	AL	Discount Rate 0%	5%	10%
1	800	$477	$382	$345
	400	$812	$519	$433
	200	$1,990	$944	$774
2	800	$590	$113	$70
	400	$788	$132	$74
	200	$1,597	$214	$120
3	800	$1,003	$481	$406
	400	$1,536	$637	$498
	200	$3,522	$1,143	$885
4	800	$693	$197	$127
	400	$1,009	$253	$149
	200	$2,177	$427	$256
5	800	$888	$264	$174
	400	$1,303	$333	$200
	200	$2,830	$570	$344

TABLE A-25. % of Total Cost (Gross), Screening – Simulation 3

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	67.46%	86.77%	89.81%
	400	36.57%	62.30%	67.84%
	200	15.51%	34.07%	39.64%
2	800	76.60%	82.35%	83.64%
	400	54.47%	68.70%	72.04%
	200	26.66%	41.74%	47.89%
3	800	76.46%	88.27%	90.65%
	400	46.42%	64.97%	69.58%
	200	20.59%	35.93%	41.13%
4	800	77.64%	84.56%	85.74%
	400	50.43%	64.24%	68.09%
	200	23.57%	37.74%	42.27%
5	800	78.55%	86.85%	88.25%
	400	50.43%	67.07%	71.31%
	200	23.36%	38.74%	44.15%

TABLE A-26. % of Total Cost (Gross), Mitigation – Simulation 3

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	3.63%	4.57%	4.65%
	400	9.83%	16.75%	18.23%
	200	14.09%	30.81%	35.69%
2	800	3.05%	2.35%	2.10%
	400	8.52%	9.11%	8.94%
	200	15.20%	22.52%	23.24%
3	800	3.51%	4.18%	4.31%
	400	9.55%	15.53%	17.11%
	200	14.79%	29.62%	34.34%
4	800	3.08%	4.10%	4.55%
	400	9.88%	15.31%	16.05%
	200	16.81%	30.07%	33.00%
5	800	3.15%	3.76%	4.00%
	400	9.64%	14.03%	14.46%
	200	15.86%	28.48%	31.10%

TABLE A-27. % of Total Cost (Gross), Upkeep – Simulation 3

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	17.36%	4.98%	3.07%
	400	47.33%	18.31%	12.05%
	200	67.74%	33.66%	23.58%
2	800	10.11%	2.87%	1.94%
	400	29.73%	11.81%	8.41%
	200	54.57%	29.44%	21.82%
3	800	14.29%	4.63%	2.94%
	400	40.55%	17.36%	11.69%
	200	63.07%	33.27%	23.56%
4	800	10.68%	4.17%	2.99%
	400	34.10%	15.01%	10.52%
	200	57.01%	28.99%	21.42%
5	800	11.63%	4.04%	2.83%
	400	35.65%	14.77%	10.25%
	200	58.80%	30.40%	22.29%

TABLE A-28. % of Total Cost (Gross), Program Administration – Simulation 3

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	11.56%	3.68%	2.48%
	400	6.27%	2.64%	1.87%
	200	2.66%	1.45%	1.09%
2	800	10.23%	12.44%	12.31%
	400	7.28%	10.38%	10.61%
	200	3.56%	6.30%	7.05%
3	800	5.74%	2.92%	2.11%
	400	3.48%	2.15%	1.62%
	200	1.55%	1.19%	0.96%
4	800	8.60%	7.17%	6.72%
	400	5.58%	5.45%	5.34%
	200	2.61%	3.20%	3.32%
5	800	6.66%	5.35%	4.93%
	400	4.28%	4.13%	3.98%
	200	1.98%	2.38%	2.46%

TABLE A-29. Lung Cancer Deaths Prevented – Simulation 3

Strategy	AL	0% Discount Rate			5% Discount Rate			10% Discount Rate		
		Total	Ever	Never	Total	Ever	Never	Total	Ever	Never
1	800	3,376	2,848	528	444	374	69	112	94	17
	400	9,215	7,774	1,441	1,219	1,028	191	305	258	48
	200	18,436	15,553	2,883	2,435	2,055	381	607	513	95
2	800	1,595	1,344	251	119	100	19	18	15	3
	400	4,076	3,434	642	304	256	48	47	39	7
	200	8,869	7,471	1,397	661	557	104	99	84	16
3	800	4,971	4,192	779	563	475	88	130	110	20
	400	13,293	11,209	2,084	1,523	1,284	238	352	297	55
	200	27,312	23,031	4,281	3,097	2,612	485	707	596	110
4	800	2,317	1,953	364	206	174	32	37	31	6
	400	6,050	5,099	951	542	457	85	94	79	15
	200	12,197	10,279	1,917	1,092	920	172	191	161	30
5	800	3,215	2,709	506	282	238	44	50	43	8
	400	8,518	7,178	1,340	744	627	117	129	109	20
	200	17,676	14,897	2,779	1,554	1,310	244	269	227	42

TABLE A-30. Life-Years (LY) Gained – Simulation 3

Strategy	AL	0% Discount Rate			5% Discount Rate			10% Discount Rate		
		Total	Ever	Never	Total	Ever	Never	Total	Ever	Never
1	800	49,274	31,586	17,688	4,206	2,764	1,442	684	464	220
	400	135,803	87,002	48,801	11,565	7,599	3,965	1,870	1,269	601
	200	272,146	174,333	97,813	23,105	15,183	7,922	3,718	2,523	1,195
2	800	17,732	11,615	6,116	1,004	666	337	108	74	34
	400	45,584	29,848	15,736	2,574	1,709	865	278	189	89
	200	100,634	65,822	34,812	5,616	3,728	1,887	589	401	188
3	800	67,007	43,202	23,805	5,209	3,430	1,779	792	537	254
	400	181,402	116,860	64,542	14,139	9,309	4,830	2,148	1,458	690
	200	372,846	240,201	132,645	28,724	18,914	9,810	4,307	2,924	1,383
4	800	28,928	18,772	10,155	1,831	1,211	621	221	150	71
	400	76,784	49,771	27,013	4,829	3,192	1,638	565	384	181
	200	154,220	99,993	54,227	9,725	6,428	3,297	1,149	781	368
5	800	39,534	25,685	13,849	2,497	1,651	846	303	206	97
	400	105,639	68,595	37,044	6,579	4,351	2,228	772	525	247
	200	220,744	143,262	77,482	13,776	9,109	4,667	1,615	1,098	517

TABLE A-31. No. Quality-Adjusted Life-Years (QALY) Gained – Simulation 3

Strategy	AL	0% Discount Rate			5% Discount Rate			10% Discount Rate		
		Total	Ever	Never	Total	Ever	Never	Total	Ever	Never
1	800	41,904	26,956	14,948	3,601	2,374	1,227	589	401	188
	400	115,482	74,244	41,239	9,903	6,528	3,374	1,612	1,097	515
	200	231,421	148,767	82,654	19,785	13,043	6,741	3,205	2,181	1,024
2	800	15,149	9,956	5,193	860	573	287	93	64	30
	400	38,943	25,584	13,359	2,206	1,469	737	239	163	76
	200	85,962	56,412	29,551	4,813	3,206	1,608	507	346	161
3	800	57,055	36,914	20,141	4,462	2,948	1,514	682	465	218
	400	154,438	99,836	54,602	12,109	7,998	4,111	1,852	1,261	591
	200	317,439	205,217	112,222	24,601	16,251	8,350	3,713	2,528	1,185
4	800	24,672	16,065	8,607	1,569	1,040	528	190	130	61
	400	65,477	42,585	22,892	4,137	2,743	1,394	487	332	155
	200	131,517	85,560	45,957	8,331	5,524	2,807	990	675	315
5	800	33,725	21,985	11,740	2,140	1,419	720	261	178	83
	400	90,112	58,709	31,403	5,636	3,739	1,897	665	454	212
	200	188,282	122,605	65,676	11,802	7,829	3,973	1,391	949	442

TABLE A-32. Cost per Lung Cancer Death Prevented – Simulation 3

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$141,405	$860,688	$3,090,112
	400	$88,138	$425,541	$1,416,354
	200	$107,935	$387,488	$1,274,268
2	800	$369,860	$955,648	$3,822,259
	400	$193,206	$435,435	$1,590,999
	200	$180,054	$323,764	$1,208,721
3	800	$201,797	$855,040	$3,126,362
	400	$115,523	$418,008	$1,415,040
	200	$128,972	$369,160	$1,252,771
4	800	$298,869	$954,363	$3,465,029
	400	$166,858	$466,561	$1,584,844
	200	$178,476	$390,690	$1,336,559
5	800	$276,264	$933,983	$3,449,741
	400	$152,980	$447,717	$1,552,797
	200	$160,084	$367,073	$1,277,590

TABLE A-33. Cost per Life-Year (LY) Gained – Simulation 3

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$9,689	$90,842	$504,580
	400	$5,981	$44,850	$231,276
	200	$7,312	$40,841	$208,119
2	800	$33,266	$113,008	$644,263
	400	$17,278	$51,410	$267,911
	200	$15,868	$38,135	$203,757
3	800	$14,971	$92,340	$512,746
	400	$8,465	$45,021	$231,997
	200	$9,448	$39,805	$205,510
4	800	$23,941	$107,422	$576,203
	400	$13,147	$52,362	$263,938
	200	$14,115	$43,870	$222,495
5	800	$22,465	$105,563	$574,006
	400	$12,335	$50,602	$258,801
	200	$12,819	$41,403	$212,885

TABLE A-34. Cost per Quality-Adjusted Life-Year (QALY) Gained – Simulation 3

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$11,392	$106,085	$585,300
	400	$7,033	$52,377	$268,280
	200	$8,599	$47,696	$241,424
2	800	$38,937	$131,841	$748,129
	400	$20,224	$59,977	$311,066
	200	$18,576	$44,491	$236,596
3	800	$17,582	$107,814	$594,860
	400	$9,944	$52,567	$269,148
	200	$11,097	$46,476	$238,429
4	800	$28,071	$125,388	$668,881
	400	$15,417	$61,125	$306,457
	200	$16,552	$51,211	$258,326
5	800	$26,334	$123,212	$666,350
	400	$14,460	$59,064	$300,478
	200	$15,029	$48,329	$247,171

Simulation 4: 50% Homeowner Compliance with Radon Guideline
70% Reduction in Radon Levels Within Mitigated Homes
Indoor Radon Distribution: Canada ~LN(11.2 Bqm^{-3}, 3.9)

TABLE A-35. Net Total Cost ($Millions) – Simulation 4

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$471	$371	$334
	400	$659	$445	$384
	200	$1,290	$667	$558
2	800	$615	$115	$70
	400	$855	$138	$80
	200	$1,731	$224	$122
3	800	$984	$469	$394
	400	$1,270	$546	$443
	200	$2,310	$809	$638
4	800	$674	$191	$124
	400	$843	$221	$137
	200	$1,459	$318	$195
5	800	$867	$257	$169
	400	$1,096	$290	$183
	200	$1,882	$415	$256

TABLE A-36. % of Total Cost (Gross), Screening – Simulation 4

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	74.39%	90.60%	92.98%
	400	49.86%	74.41%	78.91%
	200	25.55%	49.32%	55.35%
2	800	76.60%	82.35%	83.64%
	400	54.47%	68.70%	72.04%
	200	26.66%	41.74%	47.89%
3	800	83.15%	91.99%	93.74%
	400	61.59%	77.87%	81.18%
	200	33.45%	52.13%	57.55%
4	800	84.37%	88.42%	88.71%
	400	65.30%	75.62%	77.68%
	200	37.08%	51.92%	55.82%
5	800	84.94%	90.38%	91.05%
	400	65.12%	79.44%	82.02%
	200	37.22%	54.82%	59.86%

TABLE A-37. % of Total Cost (Gross), Mitigation – Simulation 4

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	2.22%	2.66%	2.68%
	400	7.15%	10.71%	11.39%
	200	12.06%	23.22%	25.96%
2	800	3.05%	2.35%	2.10%
	400	8.52%	9.11%	8.94%
	200	15.20%	22.52%	23.24%
3	800	2.09%	2.35%	2.43%
	400	6.63%	9.36%	10.16%
	200	12.22%	21.83%	24.48%
4	800	1.51%	1.96%	2.61%
	400	6.44%	9.16%	9.90%
	200	13.44%	22.10%	24.10%
5	800	1.66%	1.91%	2.27%
	400	6.57%	7.88%	8.08%
	200	12.73%	20.38%	21.68%

TABLE A-38. % of Total Cost (Gross), Upkeep – Simulation 4

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	10.64%	2.89%	1.77%
	400	34.45%	11.72%	7.53%
	200	58.01%	25.37%	17.15%
2	800	10.11%	2.87%	1.94%
	400	29.73%	11.81%	8.41%
	200	54.57%	29.44%	21.82%
3	800	8.52%	2.61%	1.65%
	400	27.16%	10.19%	6.77%
	200	51.82%	24.31%	16.63%
4	800	4.78%	2.12%	1.72%
	400	21.02%	8.81%	6.33%
	200	45.38%	21.57%	15.69%
5	800	6.20%	2.15%	1.60%
	400	22.78%	7.79%	5.33%
	200	46.89%	21.42%	15.12%

TABLE A-39. % of Total Cost (Gross), Program Administration – Simulation 4

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	12.74%	3.84%	2.57%
	400	8.54%	3.16%	2.18%
	200	4.38%	2.09%	1.53%
2	800	10.23%	12.44%	12.31%
	400	7.28%	10.38%	10.61%
	200	3.56%	6.30%	7.05%
3	800	6.24%	3.05%	2.18%
	400	4.62%	2.58%	1.89%
	200	2.51%	1.73%	1.34%
4	800	9.34%	7.50%	6.96%
	400	7.23%	6.41%	6.09%
	200	4.11%	4.40%	4.38%
5	800	7.21%	5.56%	5.08%
	400	5.53%	4.89%	4.58%
	200	3.16%	3.37%	3.34%

TABLE A-40. Lung Cancer Deaths Prevented – Simulation 4

Strategy	AL	0% Discount Rate			5% Discount Rate			10% Discount Rate		
		Total	Ever	Never	Total	Ever	Never	Total	Ever	Never
1	800	1,381	1,165	216	188	158	29	49	41	8
	400	3,975	3,353	622	523	442	82	131	111	20
	200	7,619	6,428	1,192	1,009	851	158	252	213	39
2	800	499	420	79	32	27	5	4	3	1
	400	1,132	953	179	65	55	10	7	6	1
	200	3,000	2,527	473	202	170	32	26	22	4
3	800	1,879	1,585	295	220	185	34	53	44	8
	400	5,107	4,307	801	589	496	92	138	117	22
	200	10,621	8,956	1,665	1,211	1,022	190	278	235	43
4	800	539	454	84	59	50	9	14	12	2
	400	1,887	1,591	296	184	155	29	36	30	6
	200	4,445	3,747	698	423	357	66	80	67	12
5	800	975	821	153	89	75	14	18	15	3
	400	2,790	2,351	439	240	202	38	42	36	7
	200	6,423	5,413	1,010	568	479	89	100	84	16

TABLE A-41. Life-Years (LY) Gained – Simulation 4

Strategy	AL	0% Discount Rate			5% Discount Rate			10% Discount Rate		
		Total	Ever	Never	Total	Ever	Never	Total	Ever	Never
1	800	20,387	13,057	7,330	1,787	1,174	613	298	202	96
	400	58,427	37,438	20,989	4,963	3,262	1,702	804	546	258
	200	112,656	72,158	40,498	9,579	6,294	3,284	1,544	1,048	496
2	800	5,092	3,361	1,731	256	171	85	24	16	8
	400	11,090	7,351	3,739	504	338	166	40	28	13
	200	32,494	21,342	11,152	1,662	1,107	555	153	105	49
3	800	25,480	16,418	9,062	2,043	1,345	698	321	218	103
	400	69,518	44,790	24,728	5,468	3,599	1,868	844	573	271
	200	145,159	93,506	51,653	11,241	7,401	3,840	1,698	1,153	545
4	800	7,041	4,548	2,493	547	360	186	83	56	27
	400	24,517	15,859	8,658	1,669	1,102	568	217	148	70
	200	57,467	37,191	20,276	3,814	2,518	1,296	481	327	154
5	800	11,867	7,713	4,154	796	526	270	107	73	34
	400	33,872	22,029	11,843	2,115	1,399	716	255	173	82
	200	80,039	51,951	28,088	5,038	3,331	1,707	601	409	192

TABLE A-42. No. Quality-Adjusted Life-Years (QALY) Gained – Simulation 4

Strategy	AL	0% Discount Rate			5% Discount Rate			10% Discount Rate		
		Total	Ever	Never	Total	Ever	Never	Total	Ever	Never
1	800	17,333	11,140	6,193	1,530	1,009	522	257	175	82
	400	49,687	31,950	17,737	4,250	2,802	1,448	693	472	221
	200	95,796	61,575	34,221	8,202	5,407	2,795	1,331	906	425
2	800	4,357	2,885	1,472	220	147	73	20	14	6
	400	9,497	6,315	3,182	433	291	142	35	24	11
	200	27,781	18,306	9,475	1,425	952	473	132	90	42
3	800	21,691	14,026	7,665	1,750	1,156	594	277	189	88
	400	59,185	38,265	20,920	4,683	3,093	1,590	728	496	232
	200	123,584	79,886	43,699	9,628	6,359	3,268	1,463	996	467
4	800	5,996	3,887	2,109	468	309	159	72	49	23
	400	20,895	13,562	7,333	1,430	947	483	187	128	60
	200	48,985	31,810	17,176	3,267	2,164	1,103	414	282	132
5	800	10,122	6,602	3,520	682	452	230	92	63	29
	400	28,901	18,859	10,042	1,812	1,203	609	220	150	70
	200	68,269	44,460	23,808	4,316	2,863	1,453	518	353	165

TABLE A-43. Cost per Lung Cancer Death Prevented – Simulation 4

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$341,317	$1,976,015	$6,891,559
	400	$165,906	$850,957	$2,922,414
	200	$169,296	$660,958	$2,213,013
2	800	$1,233,386	$3,620,162	$17,122,200
	400	$755,163	$2,112,513	$11,338,718
	200	$576,943	$1,111,751	$4,642,268
3	800	$523,566	$2,133,283	$7,490,406
	400	$248,601	$928,328	$3,204,309
	200	$217,493	$667,770	$2,290,591
4	800	$1,251,037	$3,218,137	$9,053,811
	400	$446,816	$1,198,293	$3,816,429
	200	$328,346	$752,427	$2,448,619
5	800	$889,594	$2,877,536	$9,578,257
	400	$392,888	$1,206,104	$4,305,255
	200	$293,098	$730,756	$2,556,320

TABLE A-44. Cost per Life-Year (LY) Gained – Simulation 4

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$23,119	$207,702	$1,123,921
	400	$11,287	$89,733	$477,139
	200	$11,450	$69,635	$361,388
2	800	$120,753	$450,238	$2,938,034
	400	$77,091	$272,904	$1,981,963
	200	$53,273	$135,033	$792,777
3	800	$38,620	$229,384	$1,226,301
	400	$18,263	$99,925	$524,925
	200	$15,913	$71,943	$375,605
4	800	$95,695	$349,745	$1,485,900
	400	$34,395	$132,394	$631,318
	200	$25,397	$83,486	$405,830
5	800	$73,066	$323,258	$1,585,075
	400	$32,362	$136,982	$716,924
	200	$23,519	$82,367	$425,539

TABLE A-45. Cost per Quality-Adjusted Life-Year (QALY) Gained – Simulation 4

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$27,191	$242,553	$1,303,600
	400	$13,272	$104,790	$553,461
	200	$13,465	$81,322	$419,215
2	800	$141,136	$524,961	$3,412,414
	400	$90,021	$318,017	$2,302,013
	200	$62,312	$157,484	$920,765
3	800	$45,367	$267,821	$1,422,492
	400	$21,452	$116,669	$608,930
	200	$18,691	$84,001	$435,755
4	800	$112,371	$408,338	$1,723,838
	400	$40,356	$154,567	$732,783
	200	$29,794	$97,465	$471,103
5	800	$85,662	$377,288	$1,839,385
	400	$37,928	$159,874	$832,240
	200	$27,574	$96,144	$494,035

Simulation 5: 10% Homeowner Compliance with Radon Guideline 70% Reduction in Radon Levels Within Mitigated Homes Indoor Radon Distribution: Canada ~LN(11.2 Bqm^{-3}, 3.9)

TABLE A-46. Net Total Cost ($Millions) – Simulation 5

Strategy	AL	Discount Rate 0%	5%	10%
1	800	$450	$358	$323
	400	$490	$374	$334
	200	$613	$420	$371
2	800	$553	$110	$67
	400	$583	$111	$67
	200	$659	$117	$70
3	800	$940	$454	$382
	400	$1,009	$470	$393
	200	$1,208	$523	$432
4	800	$659	$189	$121
	400	$699	$194	$124
	200	$817	$213	$134
5	800	$841	$253	$166
	400	$898	$259	$168
	200	$1,062	$284	$181

TABLE A-47. % of Total Cost (Gross), Screening – Simulation 5

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	82.97%	94.91%	96.55%
	400	74.35%	90.50%	92.84%
	200	57.82%	80.17%	83.81%
2	800	86.04%	86.58%	87.07%
	400	81.07%	85.99%	86.88%
	200	70.87%	80.80%	83.60%
3	800	90.41%	95.90%	97.06%
	400	82.83%	92.20%	93.85%
	200	67.77%	82.51%	85.44%
4	800	87.61%	90.11%	90.39%
	400	81.82%	87.15%	88.30%
	200	68.97%	79.01%	82.00%
5	800	89.26%	92.48%	92.92%
	400	82.70%	90.03%	91.29%
	200	68.81%	81.78%	85.29%

TABLE A-48. % of Total Cost (Gross), Mitigation – Simulation 5

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	0.50%	0.51%	0.47%
	400	2.22%	2.70%	2.77%
	200	5.53%	7.83%	8.35%
2	800	0.53%	0.24%	0.08%
	400	1.74%	0.72%	0.23%
	200	4.56%	3.40%	2.22%
3	800	0.55%	0.46%	0.41%
	400	2.08%	2.32%	2.40%
	200	5.29%	7.05%	7.53%
4	800	0.56%	1.07%	1.52%
	400	2.10%	2.80%	2.92%
	200	5.22%	7.38%	7.08%
5	800	0.61%	0.91%	1.14%
	400	2.11%	2.32%	2.22%
	200	5.46%	6.70%	5.99%

TABLE A-49. % of Total Cost (Gross), Upkeep – Simulation 5

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	2.32%	0.55%	0.31%
	400	10.69%	2.95%	1.83%
	200	26.75%	8.59%	5.52%
2	800	1.94%	0.10%	0.03%
	400	6.36%	0.31%	0.09%
	200	15.10%	3.59%	1.87%
3	800	2.26%	0.46%	0.27%
	400	8.87%	2.42%	1.57%
	200	21.85%	7.71%	5.04%
4	800	2.13%	1.17%	1.00%
	400	7.03%	2.66%	1.86%
	200	18.17%	6.92%	4.48%
5	800	2.55%	0.92%	0.75%
	400	8.18%	2.11%	1.40%
	200	19.89%	6.48%	3.96%

TABLE A-50. % of Total Cost (Gross), Program Administration – Simulation 5

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	14.21%	4.03%	2.67%
	400	12.74%	3.84%	2.56%
	200	9.90%	3.40%	2.31%
2	800	11.49%	13.08%	12.82%
	400	10.83%	12.99%	12.79%
	200	9.47%	12.20%	12.31%
3	800	6.79%	3.18%	2.26%
	400	6.22%	3.05%	2.18%
	200	5.09%	2.73%	1.99%
4	800	9.70%	7.64%	7.09%
	400	9.06%	7.39%	6.93%
	200	7.64%	6.70%	6.43%
5	800	7.57%	5.69%	5.19%
	400	7.02%	5.54%	5.10%
	200	5.84%	5.03%	4.76%

TABLE A-51. Lung Cancer Deaths Prevented – Simulation 5

Strategy	AL	0% Discount Rate			5% Discount Rate			10% Discount Rate		
		Total	Ever	Never	Total	Ever	Never	Total	Ever	Never
1	800	20	16	3	4	4	1	1	1	0
	400	581	490	91	80	67	12	21	17	3
	200	1,495	1,261	234	201	170	31	51	43	8
2	800	188	158	30	8	7	1	1	0	0
	400	393	331	62	17	15	3	1	1	0
	200	785	661	124	45	38	7	5	4	1
3	800	208	175	33	13	11	2	2	2	0
	400	974	821	153	97	82	15	22	18	3
	200	2,280	1,922	358	246	208	39	55	47	9
4	800	69	58	11	10	9	2	4	3	1
	400	380	320	60	39	33	6	8	7	1
	200	970	817	152	92	77	14	17	14	3
5	800	227	191	36	18	15	3	4	3	1
	400	694	585	109	53	45	8	9	8	1
	200	1,577	1,329	248	129	109	20	21	18	3

TABLE A-52. Life-Years (LY) Gained – Simulation 5

Strategy	AL	0% Discount Rate			5% Discount Rate			10% Discount Rate		
		Total	Ever	Never	Total	Ever	Never	Total	Ever	Never
1	800	337	213	124	42	27	14	9	6	3
	400	8,725	5,582	3,143	763	501	262	126	86	41
	200	22,347	14,303	8,043	1,914	1,257	657	312	212	100
2	800	1,687	1,129	558	59	40	19	3	2	1
	400	3,456	2,318	1,138	119	81	38	5	4	2
	200	7,951	5,256	2,695	352	235	116	26	18	8
3	800	2,024	1,343	682	101	67	33	11	8	4
	400	12,181	7,900	4,281	881	582	300	132	90	42
	200	30,298	19,560	10,738	2,266	1,493	773	338	229	108
4	800	831	538	292	98	64	34	22	15	7
	400	4,872	3,153	1,719	352	232	120	50	34	16
	200	12,578	8,138	4,440	828	546	281	102	69	33
5	800	2,374	1,563	810	153	101	51	25	17	8
	400	7,791	5,101	2,690	454	301	153	55	37	18
	200	19,085	12,419	6,666	1,129	748	381	126	85	40

TABLE A-53. No. Quality-Adjusted Life-Years (QALY) Gained – Simulation 5

Strategy	AL	0% Discount Rate			5% Discount Rate			10% Discount Rate		
		Total	Ever	Never	Total	Ever	Never	Total	Ever	Never
1	800	286	182	104	36	24	12	8	5	2
	400	7,417	4,763	2,655	653	430	223	109	74	35
	200	19,000	12,204	6,796	1,639	1,080	559	269	183	86
2	800	1,448	972	476	50	34	16	2	2	1
	400	2,968	1,996	972	102	70	32	5	3	1
	200	6,808	4,514	2,293	302	203	99	22	15	7
3	800	1,734	1,154	580	86	58	28	10	7	3
	400	10,385	6,759	3,627	755	500	255	114	77	36
	200	25,808	16,719	9,089	1,941	1,283	658	291	198	93
4	800	706	459	247	84	55	29	19	13	6
	400	4,151	2,696	1,455	301	199	102	43	29	14
	200	10,721	6,960	3,761	709	470	239	88	60	28
5	800	2,030	1,341	689	131	87	44	22	15	7
	400	6,657	4,373	2,284	389	259	130	47	32	15
	200	16,288	10,634	5,654	967	643	325	108	74	34

TABLE A-54. Cost per Lung Cancer Death Prevented – Simulation 5

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$23,101,108	$84,800,824	$228,460,968
	400	$842,925	$4,676,195	$16,206,227
	200	$410,013	$2,087,065	$7,291,757
2	800	$2,940,811	$13,001,910	$127,535,166
	400	$1,483,100	$6,369,798	$63,254,242
	200	$839,315	$2,599,025	$15,379,494
3	800	$4,525,298	$35,750,046	$196,621,738
	400	$1,035,378	$4,830,380	$18,106,390
	200	$529,736	$2,122,236	$7,799,446
4	800	$9,526,356	$18,252,931	$33,654,256
	400	$1,838,971	$5,027,868	$14,959,437
	200	$842,635	$2,322,571	$7,922,981
5	800	$3,702,280	$14,141,410	$40,475,753
	400	$1,293,191	$4,878,284	$18,310,692
	200	$673,235	$2,193,887	$8,594,346

TABLE A-55. Cost per Life-Year (LY) Gained – Simulation 5

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$1,335,715	$8,548,287	$36,919,407
	400	$56,147	$490,254	$2,642,546
	200	$27,426	$219,299	$1,189,752
2	800	$327,982	$1,880,681	$24,457,562
	400	$168,629	$934,233	$12,230,441
	200	$82,886	$333,548	$2,700,800
3	800	$464,207	$4,517,518	$33,191,872
	400	$82,800	$533,592	$2,975,148
	200	$39,865	$230,655	$1,280,028
4	800	$793,498	$1,926,485	$5,431,079
	400	$143,444	$553,125	$2,462,868
	200	$64,958	$257,862	$1,315,004
5	800	$354,325	$1,655,708	$6,642,357
	400	$115,201	$571,534	$3,055,250
	200	$55,629	$251,350	$1,438,170

TABLE A-56. Cost per Quality-Adjusted Life-Year (QALY) Gained – Simulation 5

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$1,576,285	$9,982,569	$42,797,416
	400	$66,045	$572,531	$3,065,027
	200	$32,257	$256,101	$1,380,017
2	800	$382,062	$2,186,875	$28,380,155
	400	$196,379	$1,086,127	$14,190,476
	200	$96,807	$388,681	$3,137,225
3	800	$541,913	$5,262,365	$38,485,700
	400	$97,120	$622,765	$3,450,851
	200	$46,800	$269,273	$1,484,894
4	800	$933,266	$2,247,773	$6,290,453
	400	$168,343	$645,725	$2,857,447
	200	$76,211	$301,059	$1,526,692
5	800	$414,353	$1,929,809	$7,695,091
	400	$134,834	$666,645	$3,544,803
	200	$65,181	$293,332	$1,669,804

Simulation 6: 100% Homeowner Compliance with Radon Guideline 70% Reduction in Radon Levels Within Mitigated Homes Indoor Radon Distribution: Canada ~LN(11.2 Bqm^{-3}, 3.9)

TABLE A-57. Net Total Cost ($Millions) – Simulation 6

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$495	$384	$345
	400	$859	$525	$439
	200	$2,084	$956	$777
2	800	$598	$114	$70
	400	$808	$134	$76
	200	$1,642	$217	$121
3	800	$1,028	$484	$407
	400	$1,603	$644	$506
	200	$3,661	$1,159	$889
4	800	$704	$198	$128
	400	$1,040	$256	$152
	200	$2,239	$432	$257
5	800	$904	$265	$174
	400	$1,346	$337	$204
	200	$2,920	$578	$345

TABLE A-58. % of Total Cost (Gross), Screening – Simulation 6

Strategy	AL	Discount Rate 0%	5%	10%
1	800	67.46%	86.77%	89.81%
	400	36.57%	62.30%	67.84%
	200	15.51%	34.07%	39.64%
2	800	76.60%	82.35%	83.64%
	400	54.47%	68.70%	72.04%
	200	26.66%	41.74%	47.89%
3	800	76.46%	88.27%	90.65%
	400	46.42%	64.97%	69.58%
	200	20.59%	35.93%	41.13%
4	800	77.64%	84.56%	85.74%
	400	50.43%	64.24%	68.09%
	200	23.57%	37.74%	42.27%
5	800	78.55%	86.85%	88.25%
	400	50.43%	67.07%	71.31%
	200	23.36%	38.74%	44.15%

TABLE A-59. % of Total Cost (Gross), Mitigation – Simulation 6

Strategy	AL	Discount Rate 0%	5%	10%
1	800	3.63%	4.57%	4.65%
	400	9.83%	16.75%	18.23%
	200	14.09%	30.81%	35.69%
2	800	3.05%	2.35%	2.10%
	400	8.52%	9.11%	8.94%
	200	15.20%	22.52%	23.24%
3	800	3.51%	4.18%	4.31%
	400	9.55%	15.53%	17.11%
	200	14.79%	29.62%	34.34%
4	800	3.08%	4.10%	4.55%
	400	9.88%	15.31%	16.05%
	200	16.81%	30.07%	33.00%
5	800	3.15%	3.76%	4.00%
	400	9.64%	14.03%	14.46%
	200	15.86%	28.48%	31.10%

TABLE A-60. % of Total Cost (Gross), Upkeep – Simulation 6

Strategy	AL	Discount Rate 0%	5%	10%
1	800	17.36%	4.98%	3.07%
	400	47.33%	18.31%	12.05%
	200	67.74%	33.66%	23.58%
2	800	10.11%	2.87%	1.94%
	400	29.73%	11.81%	8.41%
	200	54.57%	29.44%	21.82%
3	800	14.29%	4.63%	2.94%
	400	40.55%	17.36%	11.69%
	200	63.07%	33.27%	23.56%
4	800	10.68%	4.17%	2.99%
	400	34.10%	15.01%	10.52%
	200	57.01%	28.99%	21.42%
5	800	11.63%	4.04%	2.83%
	400	35.65%	14.77%	10.25%
	200	58.80%	30.40%	22.29%

TABLE A-61. % of Total Cost (Gross), Program Administration – Simulation 6

Strategy	AL	Discount Rate 0%	5%	10%
1	800	11.56%	3.68%	2.48%
	400	6.27%	2.64%	1.87%
	200	2.66%	1.45%	1.09%
2	800	10.23%	12.44%	12.31%
	400	7.28%	10.38%	10.61%
	200	3.56%	6.30%	7.05%
3	800	5.74%	2.92%	2.11%
	400	3.48%	2.15%	1.62%
	200	1.55%	1.19%	0.96%
4	800	8.60%	7.17%	6.72%
	400	5.58%	5.45%	5.34%
	200	2.61%	3.20%	3.32%
5	800	6.66%	5.35%	4.93%
	400	4.28%	4.13%	3.98%
	200	1.98%	2.38%	2.46%

TABLE A-62. Lung Cancer Deaths Prevented – Simulation 6

Strategy	AL	0% Discount Rate			5% Discount Rate			10% Discount Rate		
		Total	Ever	Never	Total	Ever	Never	Total	Ever	Never
1	800	2,626	2,215	411	345	291	54	87	73	14
	400	7,167	6,046	1,121	948	800	148	238	201	37
	200	14,338	12,096	2,242	1,894	1,598	296	472	399	74
2	800	1,240	1,045	195	92	78	15	14	12	2
	400	3,170	2,671	500	236	199	37	36	31	6
	200	6,898	5,811	1,087	514	434	81	77	65	12
3	800	3,866	3,260	606	438	369	68	101	85	16
	400	10,338	8,718	1,620	1,184	999	185	274	231	43
	200	21,240	17,910	3,329	2,409	2,032	377	550	464	86
4	800	1,802	1,519	283	160	135	25	29	24	4
	400	4,705	3,966	740	422	355	66	73	62	11
	200	9,486	7,995	1,491	849	716	133	149	126	23
5	800	2,500	2,107	393	220	185	34	39	33	6
	400	6,625	5,583	1,042	578	487	91	100	84	16
	200	13,747	11,585	2,162	1,209	1,019	190	209	177	33

TABLE A-63. Life-Years (LY) Gained – Simulation 6

Strategy	AL	0% Discount Rate			5% Discount Rate			10% Discount Rate		
		Total	Ever	Never	Total	Ever	Never	Total	Ever	Never
1	800	38,323	24,566	13,757	3,271	2,150	1,121	532	361	171
	400	105,621	67,665	37,956	8,995	5,911	3,084	1,455	987	467
	200	211,654	135,583	76,072	17,970	11,809	6,161	2,892	1,962	929
2	800	13,791	9,034	4,757	781	518	262	84	57	27
	400	35,454	23,215	12,239	2,002	1,329	673	216	147	69
	200	78,268	51,193	27,075	4,368	2,900	1,468	458	312	146
3	800	52,116	33,601	18,515	4,052	2,668	1,383	616	418	198
	400	141,084	90,886	50,198	10,997	7,240	3,757	1,671	1,134	536
	200	289,963	186,804	103,159	22,339	14,710	7,630	3,350	2,274	1,075
4	800	22,499	14,601	7,899	1,424	942	483	172	117	55
	400	59,719	38,710	21,010	3,756	2,482	1,274	440	299	141
	200	119,943	77,768	42,175	7,564	4,999	2,564	894	608	286
5	800	30,748	19,977	10,771	1,942	1,284	658	236	160	75
	400	82,161	53,350	28,811	5,117	3,384	1,733	601	408	192
	200	171,678	111,418	60,260	10,715	7,085	3,630	1,256	854	402

TABLE A-64. No. Quality-Adjusted Life-Years (QALY) Gained – Simulation 6

Strategy	AL	0% Discount Rate			5% Discount Rate			10% Discount Rate		
		Total	Ever	Never	Total	Ever	Never	Total	Ever	Never
1	800	32,592	20,966	11,626	2,801	1,847	954	458	312	146
	400	89,817	57,743	32,074	7,702	5,078	2,624	1,254	853	401
	200	179,982	115,699	64,283	15,387	10,144	5,243	2,493	1,697	796
2	800	11,782	7,744	4,039	669	446	223	72	49	23
	400	30,288	19,898	10,390	1,716	1,143	573	186	127	59
	200	66,857	43,874	22,983	3,744	2,493	1,250	395	269	125
3	800	44,375	28,710	15,665	3,470	2,292	1,178	531	361	169
	400	120,113	77,646	42,466	9,418	6,221	3,197	1,440	981	460
	200	246,873	159,597	87,276	19,133	12,639	6,494	2,887	1,966	921
4	800	19,189	12,495	6,694	1,220	809	411	148	101	47
	400	50,926	33,121	17,805	3,218	2,133	1,084	379	258	120
	200	102,287	66,544	35,743	6,479	4,296	2,183	770	525	245
5	800	26,230	17,099	9,131	1,664	1,104	560	203	138	65
	400	70,085	45,661	24,424	4,384	2,908	1,475	517	353	165
	200	146,432	95,353	51,079	9,179	6,089	3,090	1,082	738	344

TABLE A-65. Cost per Lung Cancer Death Prevented – Simulation 6

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$188,344	$1,113,012	$3,979,320
	400	$119,861	$553,543	$1,846,611
	200	$145,323	$504,632	$1,644,701
2	800	$482,072	$1,235,097	$4,920,637
	400	$254,948	$566,254	$2,087,146
	200	$238,045	$422,682	$1,560,386
3	800	$265,995	$1,105,755	$4,025,936
	400	$155,075	$543,863	$1,847,061
	200	$172,382	$481,079	$1,617,080
4	800	$390,799	$1,233,448	$4,461,346
	400	$221,074	$606,278	$2,074,528
	200	$236,020	$508,739	$1,724,763
5	800	$361,737	$1,207,249	$4,441,697
	400	$203,233	$582,054	$2,033,464
	200	$212,379	$478,381	$1,648,961

TABLE A-66. Cost per Life-Year (LY) Gained – Simulation 6

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$12,905	$117,474	$649,776
	400	$8,133	$58,341	$301,532
	200	$9,844	$53,187	$268,619
2	800	$43,358	$146,053	$829,401
	400	$22,799	$66,855	$351,457
	200	$20,978	$49,785	$263,037
3	800	$19,733	$119,416	$660,282
	400	$11,364	$58,576	$302,826
	200	$12,627	$51,871	$265,271
4	800	$31,305	$138,835	$741,881
	400	$17,418	$68,042	$345,488
	200	$18,666	$57,125	$287,118
5	800	$29,415	$136,448	$739,059
	400	$16,386	$65,785	$338,912
	200	$17,006	$53,958	$274,766

TABLE A-67. Cost per Quality-Adjusted Life-Year (QALY) Gained – Simulation 6

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$15,174	$137,185	$753,725
	400	$9,564	$68,131	$349,777
	200	$11,577	$62,114	$311,605
2	800	$50,750	$170,394	$963,113
	400	$26,687	$77,996	$408,071
	200	$24,559	$58,083	$305,430
3	800	$23,176	$139,426	$766,023
	400	$13,348	$68,393	$351,320
	200	$14,831	$60,565	$307,763
4	800	$36,705	$162,055	$861,208
	400	$20,426	$79,429	$401,145
	200	$21,888	$66,684	$333,356
5	800	$34,482	$159,261	$857,955
	400	$19,210	$76,786	$393,490
	200	$19,938	$62,983	$319,018

Simulation 7: 50% Homeowner Compliance with Radon Guideline
90% Reduction in Radon Levels Within Mitigated Homes
Indoor Radon Distribution: Canada ~LN(21.5 Bqm^{-3}, 4.0)

TABLE A-68. Net Total Cost ($Millions) – Simulation 7

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$538	$451	$409
	400	$1,001	$650	$524
	200	$2,340	$1,138	$939
2	800	$503	$113	$75
	400	$750	$135	$64
	200	$1,725	$238	$151
3	800	$1,099	$561	$478
	400	$1,913	$792	$600
	200	$4,336	$1,393	$1,088
4	800	$791	$221	$143
	400	$1,320	$295	$162
	200	$2,793	$531	$320
5	800	$987	$296	$196
	400	$1,658	$393	$222
	200	$3,639	$705	$432

TABLE A-69. Initial Screen Costs per 'At-Risk' Home Identified – Simulation 7

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$10,568	$9,609	$8,815
	400	$2,797	$2,544	$2,333
	200	$928	$844	$774
2	800	$11,711	$2,381	$1,453
	400	$2,928	$595	$363
	200	$913	$186	$113
3	800	$11,182	$5,726	$4,860
	400	$2,869	$1,469	$1,247
	200	$919	$471	$400
4	800	$10,562	$3,108	$1,987
	400	$2,791	$821	$525
	200	$913	$269	$172
5	800	$10,910	$3,395	$2,215
	400	$2,869	$893	$583
	200	$911	$284	$185

TABLE A-70. % of Total Cost (Gross), Screening – Simulation 7

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	52.58%	76.75%	81.19%
	400	27.62%	52.04%	58.08%
	200	13.30%	30.04%	35.19%
2	800	74.14%	81.82%	83.00%
	400	45.47%	62.88%	66.48%
	200	21.91%	36.06%	40.14%
3	800	65.73%	79.71%	82.92%
	400	36.34%	55.03%	60.05%
	200	17.28%	31.50%	36.18%
4	800	67.97%	79.32%	81.39%
	400	39.33%	57.91%	63.48%
	200	19.39%	32.42%	36.15%
5	800	69.28%	81.71%	84.06%
	400	40.06%	60.10%	65.54%
	200	19.16%	33.61%	37.81%

TABLE A-71. % of Total Cost (Gross), Mitigation – Simulation 7

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	6.75%	9.69%	10.09%
	400	11.72%	21.95%	24.35%
	200	14.55%	32.86%	38.49%
2	800	3.77%	2.90%	3.02%
	400	11.17%	12.13%	12.63%
	200	16.70%	25.14%	27.91%
3	800	5.57%	8.44%	9.13%
	400	11.76%	20.40%	22.96%
	200	15.68%	31.67%	37.25%
4	800	6.15%	7.40%	7.75%
	400	13.78%	19.33%	19.41%
	200	18.35%	32.63%	36.79%
5	800	5.66%	6.70%	6.85%
	400	12.73%	17.91%	18.12%
	200	17.32%	30.99%	34.81%

TABLE A-72. % of Total Cost (Gross), Upkeep – Simulation 7

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	32.35%	10.56%	6.66%
	400	56.30%	23.97%	16.09%
	200	70.04%	35.92%	25.43%
2	800	12.98%	4.14%	2.92%
	400	37.77%	16.42%	12.03%
	200	58.69%	33.90%	26.61%
3	800	24.16%	9.42%	6.18%
	400	49.39%	22.90%	15.70%
	200	65.84%	35.87%	25.80%
4	800	18.94%	7.09%	4.93%
	400	42.87%	18.24%	12.48%
	200	60.29%	32.42%	24.42%
5	800	19.66%	6.98%	4.75%
	400	44.09%	18.60%	12.96%
	200	62.03%	33.50%	25.42%

TABLE A-73. % of Total Cost (Gross), Program Administration – Simulation 7

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	8.32%	3.01%	2.07%
	400	4.37%	2.04%	1.48%
	200	2.10%	1.18%	0.90%
2	800	9.12%	11.14%	11.06%
	400	5.59%	8.57%	8.86%
	200	2.69%	4.91%	5.35%
3	800	4.55%	2.43%	1.77%
	400	2.51%	1.67%	1.29%
	200	1.20%	0.96%	0.77%
4	800	6.93%	6.19%	5.93%
	400	4.01%	4.52%	4.63%
	200	1.98%	2.53%	2.63%
5	800	5.40%	4.61%	4.34%
	400	3.12%	3.39%	3.38%
	200	1.49%	1.90%	1.95%

TABLE A-74. Lung Cancer Deaths Prevented – Simulation 7

Strategy	AL	0% Discount Rate			5% Discount Rate			10% Discount Rate		
		Total	Ever	Never	Total	Ever	Never	Total	Ever	Never
1	800	10,185	8,589	1,596	1,299	1,095	203	319	269	50
	400	20,404	17,207	3,198	2,600	2,193	407	636	537	99
	200	30,872	26,035	4,837	3,972	3,350	622	977	825	152
2	800	8,744	7,366	1,377	755	636	119	132	112	21
	400	17,309	14,582	2,726	1,482	1,249	233	253	213	40
	200	28,575	24,075	4,500	2,503	2,109	394	441	372	69
3	800	13,561	11,432	2,129	1,534	1,293	240	354	299	55
	400	27,816	23,449	4,367	3,141	2,649	492	719	607	112
	200	44,806	37,772	7,035	5,025	4,238	788	1,144	966	179
4	800	5,853	4,932	921	538	453	85	98	83	15
	400	12,132	10,222	1,910	1,066	898	168	182	153	29
	200	19,590	16,506	3,084	1,754	1,478	276	309	261	48
5	800	8,744	7,366	1,377	755	636	119	132	112	21
	400	17,309	14,582	2,726	1,482	1,249	233	253	213	40
	200	28,575	24,075	4,500	2,503	2,109	394	441	372	69

TABLE A-75. Life-Years (LY) Gained – Simulation 7

Strategy	AL	0% Discount Rate			5% Discount Rate			10% Discount Rate		
		Total	Ever	Never	Total	Ever	Never	Total	Ever	Never
1	800	147,209	94,420	52,788	12,267	8,063	4,204	1,953	1,325	628
	400	296,125	189,895	106,230	24,573	16,151	8,422	3,895	2,643	1,252
	200	450,376	288,708	161,669	37,583	24,699	12,884	5,982	4,059	1,923
2	800	106,923	69,488	37,435	6,659	4,404	2,255	794	540	254
	400	212,498	138,069	74,429	13,062	8,640	4,422	1,516	1,031	485
	200	353,846	229,745	124,101	22,171	14,658	7,513	2,649	1,800	848
3	800	183,229	118,099	65,130	14,217	9,360	4,857	2,160	1,466	694
	400	377,704	243,376	134,328	29,128	19,177	9,952	4,389	2,979	1,410
	200	607,453	391,478	215,975	46,555	30,653	15,903	6,980	4,738	2,242
4	800	74,297	48,142	26,155	4,812	3,178	1,634	592	402	190
	400	152,554	98,940	53,614	9,465	6,256	3,208	1,092	742	350
	200	247,310	160,336	86,974	15,632	10,330	5,303	1,859	1,264	596
5	800	106,923	69,488	37,435	6,659	4,404	2,255	794	540	254
	400	212,498	138,069	74,429	13,062	8,640	4,422	1,516	1,031	485
	200	353,846	229,745	124,101	22,171	14,658	7,513	2,649	1,800	848

TABLE A-76. Quality-Adjusted Life-Years (QALY) Gained – Simulation 7

Strategy	AL	0% Discount Rate			5% Discount Rate			10% Discount Rate		
		Total	Ever	Never	Total	Ever	Never	Total	Ever	Never
1	800	125,232	80,607	44,625	10,506	6,927	3,578	1,684	1,146	538
	400	251,913	162,110	89,803	21,044	13,876	7,168	3,358	2,285	1,073
	200	383,103	246,445	136,658	32,186	21,220	10,966	5,157	3,510	1,648
2	800	91,232	59,490	31,741	5,706	3,786	1,920	684	467	218
	400	181,313	118,203	63,111	11,192	7,427	3,766	1,306	891	416
	200	301,868	196,657	105,211	18,996	12,599	6,397	2,282	1,556	726
3	800	156,031	100,920	55,112	12,178	8,043	4,134	1,862	1,267	594
	400	321,630	207,966	113,664	24,950	16,479	8,471	3,783	2,575	1,208
	200	517,291	334,531	182,760	39,877	26,340	13,537	6,017	4,096	1,921
4	800	63,356	41,192	22,164	4,122	2,731	1,391	510	347	162
	400	130,125	84,677	45,447	8,109	5,377	2,732	941	641	299
	200	210,926	137,209	73,717	13,393	8,878	4,515	1,602	1,092	510
5	800	91,232	59,490	31,741	5,706	3,786	1,920	684	467	218
	400	181,313	118,203	63,111	11,192	7,427	3,766	1,306	891	416
	200	301,868	196,657	105,211	18,996	12,599	6,397	2,282	1,556	726

TABLE A-77. Cost per Lung Cancer Death Prevented – Simulation 7

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$52,792	$347,280	$1,282,925
	400	$49,043	$249,854	$822,869
	200	$75,803	$286,415	$961,050
2	800	$57,534	$149,199	$567,171
	400	$43,356	$91,310	$253,462
	200	$60,367	$95,023	$343,587
3	800	$81,072	$365,634	$1,349,931
	400	$68,784	$252,008	$834,188
	200	$96,770	$277,265	$950,526
4	800	$135,088	$411,242	$1,457,722
	400	$108,833	$277,130	$892,404
	200	$142,578	$302,730	$1,035,542
5	800	$112,916	$392,717	$1,480,579
	400	$95,769	$265,002	$876,808
	200	$127,333	$281,896	$980,313

TABLE A-78. Cost per Life-Year (LY) Gained – Simulation 7

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$3,652	$36,762	$209,586
	400	$3,379	$26,439	$134,436
	200	$5,196	$30,267	$156,970
2	800	$4,705	$16,910	$94,420
	400	$3,531	$10,359	$42,262
	200	$4,875	$10,726	$57,165
3	800	$6,000	$39,445	$221,255
	400	$5,066	$27,178	$136,754
	200	$7,138	$29,929	$155,861
4	800	$10,643	$45,941	$241,923
	400	$8,655	$31,212	$148,709
	200	$11,294	$33,972	$172,217
5	800	$9,234	$44,510	$246,481
	400	$7,801	$30,064	$146,197
	200	$10,283	$31,820	$163,101

TABLE A-79. Cost per Quality-Adjusted Life-Year (QALY) Gained – Simulation 7

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$4,293	$42,926	$243,112
	400	$3,972	$30,872	$155,942
	200	$6,109	$35,342	$182,079
2	800	$5,514	$19,735	$109,603
	400	$4,139	$12,089	$49,063
	200	$5,714	$12,519	$66,358
3	800	$7,046	$46,050	$256,665
	400	$5,949	$31,729	$158,644
	200	$8,382	$34,941	$180,811
4	800	$12,480	$53,625	$280,818
	400	$10,147	$36,430	$172,655
	200	$13,242	$39,653	$199,926
5	800	$10,822	$51,945	$286,114
	400	$9,142	$35,086	$169,725
	200	$12,053	$37,138	$189,330

Simulation 8: 50% Homeowner Compliance with Radon Guideline
90% Reduction in Radon Levels Within Mitigated Homes
Indoor Radon Distribution: Winnipeg ~LN(57.0 Bqm^{-3}, 4.6)

TABLE A-80. Net Total Cost ($Millions) – Simulation 8

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$1,430	$1,005	$884
	400	$3,885	$2,004	$1,481
	200	$7,994	$3,495	$2,797
2	800	$1,929	$274	$159
	400	$3,509	$450	$229
	200	$6,614	$780	$429
3	800	$2,527	$1,203	$1,024
	400	$6,412	$2,358	$1,619
	200	$13,550	$4,166	$3,199
4	800	$2,348	$502	$309
	400	$4,921	$952	$504
	200	$9,187	$1,620	$950
5	800	$2,679	$652	$421
	400	$5,823	$1,200	$626
	200	$11,502	$2,089	$1,256

TABLE A-81. Initial Screen Costs per 'At-Risk' Home Identified – Simulation 8

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$1,562	$1,420	$1,303
	400	$610	$554	$508
	200	$311	$283	$259
2	800	$1,443	$293	$179
	400	$634	$129	$79
	200	$312	$63	$39
3	800	$1,493	$762	$647
	400	$623	$318	$270
	200	$312	$159	$135
4	800	$1,466	$431	$276
	400	$616	$181	$116
	200	$312	$92	$59
5	800	$1,476	$458	$299
	400	$620	$192	$126
	200	$310	$96	$63

TABLE A-82. % of Total Cost (Gross), Screening – Simulation 8

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	20.20%	41.47%	47.23%
	400	8.63%	20.85%	24.96%
	200	4.76%	12.22%	14.94%
2	800	30.86%	47.74%	52.14%
	400	16.35%	28.39%	32.50%
	200	8.83%	16.35%	19.14%
3	800	25.44%	43.18%	48.35%
	400	11.84%	22.29%	26.05%
	200	6.46%	12.99%	15.53%
4	800	28.14%	43.91%	48.01%
	400	13.27%	22.83%	25.94%
	200	7.39%	13.52%	15.53%
5	800	27.80%	45.05%	49.53%
	400	13.43%	24.29%	27.87%
	200	7.35%	14.21%	16.53%

TABLE A-83. % of Total Cost (Gross), Mitigation – Simulation 8

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	13.29%	27.34%	31.20%
	400	15.54%	37.51%	44.88%
	200	16.28%	41.76%	51.04%
2	800	14.78%	20.14%	21.73%
	400	17.98%	29.13%	32.82%
	200	19.67%	34.75%	40.35%
3	800	14.16%	26.16%	30.03%
	400	16.63%	36.23%	43.51%
	200	17.74%	40.64%	49.77%
4	800	16.41%	26.79%	29.68%
	400	19.51%	37.99%	43.67%
	200	20.95%	43.02%	50.27%
5	800	15.40%	25.40%	28.02%
	400	18.42%	35.92%	41.21%
	200	19.76%	40.88%	47.90%

TABLE A-84. % of Total Cost (Gross), Upkeep – Simulation 8

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	63.98%	29.90%	20.62%
	400	74.76%	41.00%	29.66%
	200	78.36%	45.65%	33.72%
2	800	51.39%	27.01%	20.69%
	400	64.10%	39.44%	31.28%
	200	70.64%	47.15%	38.51%
3	800	59.01%	29.63%	20.80%
	400	70.88%	40.95%	30.00%
	200	75.45%	46.06%	34.44%
4	800	53.19%	26.59%	19.54%
	400	66.15%	37.77%	28.90%
	200	71.06%	42.62%	33.31%
5	800	55.09%	27.54%	20.43%
	400	67.33%	38.70%	29.78%
	200	72.44%	44.28%	34.89%

TABLE A-85. % of Total Cost (Gross), Program Administration – Simulation 8

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	2.53%	1.28%	0.95%
	400	1.08%	0.65%	0.50%
	200	0.60%	0.38%	0.30%
2	800	2.98%	5.11%	5.44%
	400	1.58%	3.04%	3.39%
	200	0.85%	1.75%	2.00%
3	800	1.39%	1.04%	0.82%
	400	0.65%	0.54%	0.44%
	200	0.35%	0.31%	0.26%
4	800	2.27%	2.71%	2.77%
	400	1.07%	1.41%	1.50%
	200	0.60%	0.84%	0.90%
5	800	1.71%	2.01%	2.02%
	400	0.82%	1.08%	1.14%
	200	0.45%	0.63%	0.67%

TABLE A-86. Lung Cancer Deaths Prevented – Simulation 8

Strategy	AL	0% Discount Rate			5% Discount Rate			10% Discount Rate		
		Total	Ever	Never	Total	Ever	Never	Total	Ever	Never
1	800	48,410	43,410	5,000	5,299	4,872	427	936	902	34
	400	89,839	78,734	11,105	10,322	9,180	1,142	2,011	1,838	173
	200	121,161	105,271	15,890	14,239	12,510	1,729	2,895	2,597	299
2	800	9,665	9,532	133	374	427	-53	-14	6	-20
	400	24,047	22,112	1,935	1,130	1,111	18	43	64	-20
	200	39,384	35,287	4,097	2,009	1,889	120	122	138	-16
3	800	91,654	80,242	11,412	8,413	7,529	885	1,380	1,285	95
	400	154,043	132,979	21,064	15,091	13,214	1,878	2,726	2,446	280
	200	204,018	175,129	28,889	20,466	17,765	2,701	3,845	3,401	444
4	800	20,783	19,440	1,343	1,331	1,314	17	96	117	-21
	400	46,554	41,636	4,918	3,211	2,963	247	317	319	-2
	200	67,997	59,864	8,134	4,819	4,352	467	522	502	20
5	800	46,953	41,939	5,014	2,990	2,770	220	271	279	-8
	400	85,108	74,348	10,760	5,889	5,271	618	646	612	34
	200	117,626	101,854	15,772	8,434	7,449	985	997	920	77

TABLE A-87. Life-Years (LY) Gained – Simulation 8

Strategy	AL	0% Discount Rate			5% Discount Rate			10% Discount Rate		
		Total	Ever	Never	Total	Ever	Never	Total	Ever	Never
1	800	511,779	345,788	165,991	35,240	25,016	10,224	3,884	2,992	891
	400	1,110,041	731,367	378,674	80,906	55,346	25,560	10,124	7,311	2,813
	200	1,567,493	1,025,249	542,244	117,259	79,359	37,900	15,387	10,920	4,468
2	800	-2,558	8,243	-10,801	-2,121	-829	-1,291	-515	-285	-230
	400	112,719	87,853	24,867	2,062	2,205	-143	-347	-140	-207
	200	254,511	183,402	71,109	7,816	6,225	1,591	-32	102	-134
3	800	990,714	660,980	329,733	60,989	42,241	18,748	6,434	4,751	1,682
	400	1,833,837	1,205,676	628,161	121,169	82,136	39,033	14,330	10,188	4,142
	200	2,508,807	1,640,974	867,833	170,168	114,499	55,670	20,995	14,747	6,248
4	800	114,691	87,611	27,080	3,746	3,411	335	-159	14	-174
	400	402,478	277,996	124,482	17,975	13,137	4,838	911	793	118
	200	650,968	441,077	209,892	30,801	21,797	9,004	1,974	1,548	426
5	800	378,951	263,975	114,975	15,604	11,575	4,029	645	608	37
	400	823,804	555,733	268,072	38,847	27,255	11,593	2,609	2,000	609
	200	1,210,576	808,443	402,133	59,926	41,377	18,549	4,534	3,346	1,188

TABLE A-88. Quality-Adjusted Life-Years (QALY) Gained – Simulation 8

Strategy	AL	0% Discount Rate			5% Discount Rate			10% Discount Rate		
		Total	Ever	Never	Total	Ever	Never	Total	Ever	Never
1	800	419,554	285,943	133,611	28,679	20,605	8,075	3,075	2,421	653
	400	927,265	614,480	312,786	67,594	46,557	21,037	8,402	6,125	2,277
	200	1,315,982	865,158	450,824	98,647	67,146	31,501	12,915	9,231	3,684
2	800	-10,278	2,235	-12,513	-2,317	-1,015	-1,302	-503	-283	-220
	400	86,560	69,549	17,011	1,117	1,505	-388	-381	-171	-209
	200	206,794	150,956	55,838	5,938	4,897	1,040	-129	26	-155
3	800	828,117	555,838	272,279	50,692	35,386	15,306	5,258	3,934	1,325
	400	1,545,361	1,020,965	524,396	102,077	69,581	32,496	12,017	8,606	3,411
	200	2,120,093	1,392,935	727,158	143,986	97,357	46,629	17,738	12,533	5,205
4	800	86,593	68,342	18,252	2,419	2,458	-39	-241	-52	-190
	400	330,581	230,473	100,108	14,420	10,708	3,712	645	599	45
	200	542,037	369,793	172,244	25,309	18,094	7,215	1,538	1,238	300
5	800	311,048	218,801	92,248	12,418	9,382	3,036	419	442	-22
	400	689,586	468,040	221,546	32,155	22,758	9,397	2,072	1,621	451
	200	1,019,154	684,172	334,982	50,117	34,841	15,276	3,704	2,768	936

TABLE A-89. Cost per Lung Cancer Death Prevented – Simulation 8

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$29,530	$189,590	$944,633
	400	$43,249	$194,125	$736,493
	200	$65,977	$245,454	$966,000
2	800	$199,628	$732,572	-$11,288,019
	400	$145,925	$398,166	$5,265,175
	200	$167,944	$388,262	$3,521,330
3	800	$27,571	$142,979	$742,298
	400	$41,625	$156,221	$593,935
	200	$66,415	$203,565	$831,963
4	800	$112,955	$377,423	$3,215,336
	400	$105,697	$296,496	$1,587,375
	200	$135,112	$336,227	$1,820,102
5	800	$57,057	$217,880	$1,551,644
	400	$68,424	$203,813	$969,644
	200	$97,782	$247,751	$1,260,197

TABLE A-90. Cost per Life-Year (LY) Gained – Simulation 8

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$2,793	$28,508	$227,657
	400	$3,500	$24,767	$146,264
	200	$5,100	$29,806	$181,772
2	800	-$754,142	-$129,253	-$307,705
	400	$31,131	$218,105	-$658,602
	200	$25,988	$99,806	-$13,238,239
3	800	$2,551	$19,723	$159,161
	400	$3,497	$19,457	$112,993
	200	$5,401	$24,482	$152,359
4	800	$20,469	$134,128	-$1,941,118
	400	$12,226	$52,957	$552,828
	200	$14,113	$52,608	$481,232
5	800	$7,070	$41,752	$651,807
	400	$7,069	$30,897	$239,925
	200	$9,501	$34,867	$277,040

TABLE A-91. Cost per Quality-Adjusted Life-Year (QALY) Gained – Simulation 8

Strategy	AL	Discount Rate		
		0%	5%	10%
1	800	$3,407	$35,030	$287,529
	400	$4,190	$29,644	$176,248
	200	$6,074	$35,429	$216,575
2	800	-$187,722	-$118,329	-$315,034
	400	$40,539	$402,683	-$600,364
	200	$31,985	$131,378	-$3,317,355
3	800	$3,051	$23,730	$194,737
	400	$4,149	$23,096	$134,749
	200	$6,391	$28,934	$180,333
4	800	$27,110	$207,731	-$1,279,426
	400	$14,885	$66,013	$781,235
	200	$16,949	$64,024	$617,806
5	800	$8,613	$52,466	$1,002,790
	400	$8,445	$37,327	$302,149
	200	$11,285	$41,691	$339,055