RSP-0205

UPDATED ANALYSIS OF THE ELDORADO URANIUM MINERS' COHORT: PART I OF THE SASKATCHEWAN URANIUM MINERS' COHORT STUDY

March 16, 2006

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ABSTRACT

A cohort study has been completed in which records for uranium miners and processors who worked for Eldorado Nuclear Limited have been linked to national mortality records (1950–1999) and national cancer incidence records (1969–1999). This study, thus, updates an earlier study in which mortality in the cohort between 1950 and 1980 was ascertained.

This report presents the results of the statistical analysis of a cohort of 17,660 individuals known to have worked for Eldorado somewhere between 1930 and 1999. Based on a total of 5,332 deaths between 1950 and 1999, and 2,355 individuals who developed at least one cancer between 1969 and 1999, several types of analyses have been conducted.

The first analysis was a comparison of the mortality of the cohort with the mortality of the general Canadian population between 1950 and 1999. Lung cancer was elevated not only in the whole cohort, but in various sub-cohorts defined by gender, site of working, underground and mill workers and sub-cohorts defined by first working date for Eldorado. There can be no doubt that much of this excess is attributable to exposure to radioactive radon delay products (RDP) as discussed in more detail subsequently.

For most other the causes of death, the cohort as a whole and the various sub-cohorts had reduced risks relative to the population. This probably represents the healthy worker effect, a supposition which is supported, for example, by the major decrease in ischaemic heart disease to reflect the fact that the risk of heart disease is lower in the cohort being studied than in the population at large. The latter condition would probably prevent people working in a strenuous physical occupation such as mining.

The most notable exceptions for causes of death where rates in the Eldorado cohort were elevated relative to the population include hypertensive causes, and external causes such as homicides, suicides and both traffic and non-traffic-related accidents.

Comparisons of the cohort with the general Canadian population with respect to cancer incidence rates between 1969 and 1999 are also reported. The only cancer that is consistently elevated is lung cancer, thus, mirroring the mortality results discussed above.

The second analysis presented in the present report is that of mortality from lung cancer with respect to RDP exposure. This is primarily based on 618 lung cancer deaths amongst men in the cohort. This compares with previous analyses of the Eldorado cohort when the total number of such deaths was 122. Thus, the present analysis represents a substantial increase in the power of the study and, thus, should produce more precise estimates.

There is a strong positive monotonic increase in risk of lung cancer death with increasing RDP exposure which is highly statistically significant. This increase generally manifests itself for the three main sites (Port Hope, Port Radium and Beaverlodge sites), although it is likely that the exposure of the "Port Hope" sub-cohort comes primarily from exposure received at other sites. However, fitting a simple linear excess relative risk model to the data although, again, demonstrating a strong relationship with RDP exposure does provide excess relative risk estimates that are inconsistent across the sites.

Application of the BEIR VI-type risk model which allows for effect modification by time since exposure, exposure rate and age at risk reduces this statistical heterogeneity in terms of the RDP effect and it is clear that these modifying factors contribute to the apparent heterogeneity seen in the simple linear excess relative risk model. Using the same parameterization as chosen by the BEIR VI Committee leads to coefficient estimates that are very similar in the present study as reported by the BEIR VI Committee.

The lung cancer mortality analysis detected no effect of gamma ray exposure on risk of lung cancer mortality and all estimates did not change by including or excluding those with non-Eldorado work histories or those with zero exposure to RDP.

An analysis of lung cancer incidence amongst males was also conducted. The results mirror those of the mortality analysis and as relative measures are used, this is hardly surprising. It

should also be noted that the two analyses are not independent in that a substantial proportion of cancer cases contributed to the corresponding death analysis.

The final type of analysis conducted was that examining mortality and cancer incidence for diseases other than lung cancer with RDP exposure and gamma ray exposure. In summary, there is no meaningful evidence of any causal relationship between RDP exposure and increased risk of any of these other diseases, nor was there any meaningful evidence of a relationship with gamma ray dose.

Several limitations should be borne in mind when considering the above results. No data were available on smoking or other possible carcinogens among the cohort. Further, measurement error in exposure estimates could not be taken into account. The implication of these limitations is also discussed in the report.

Exposure to RDP is one of the best-studied carcinogenic phenomena in epidemiology. The results obtained from these studies, primarily of underground miners, are very consistent in showing increases in lung cancer risk from such exposure, but no increase in any other disease.

The present study (which is essentially independent of the data set used by the BEIR VI Committee) further supports these conclusions based on 50 years' mortality experience and 31 years of cancer incidence experience. They certainly support the use of BEIR VI-type models to predict any group's future risk of lung cancer from RDP exposure either from past or current such exposure.

Finally, it is worth noting that as yet only about 25% of the cohort has died. Future follow-up and analysis of this cohort with respect to both mortality and cancer incidence should shed further light on our knowledge on the effects of uranium mining and processing in both Saskatchewan and other provinces upon the resulting health of those employed in such occupations.

1. INTRODUCTION AND BACKGROUND

1.1. The Original Eldorado Study:

It has been known for many years that certain underground miners appear to have an excess of lung cancer. More recently, this increase has been ascribed to exposure to decay products of radon from occupationally-related exposure. In this report, the decay products are referred to as RDP (radon decay products). The potential carcinogenic properties of RDP have been demonstrated conclusively in both humans and animals.

Various cohort studies have been conducted to examine the relationship between RDP exposure and the risk of lung cancer in underground miners. At least 11 such studies have been reported (Lubin, Boice et al. 1994). Such studies have generally shown an approximately linear relationship of exposure with risk as would be expected for alpha particle emitters from radiobiologic theory (BEIR VI 1999), (Brenner and Sachs 2003).

Two of these studies were of underground miners employed by Eldorado Nuclear Ltd., namely, their facilities at Port Radium in the Northwest Territories and Beaverlodge in Saskatchewan. These original studies were funded by Eldorado Nuclear Ltd. and the National Cancer Institute of Canada, the scientific work being conducted by Dr. J.D. Abbatt and Mr. S.E. Frost of Eldorado and members of the National Cancer Institute of Canada, Epidemiology Unit, based at the University of Toronto.

The original study was carried out by assembling a nominal roll of all current and ex-employees on the Eldorado employment records at the time of the study, and then linking these records to national mortality records maintained by Statistics Canada for the period 1940–1980. Exposure reconstruction techniques were used to construct individual exposure estimates for each member of the cohort.

The results of the study were published in 1986 (Beaverlodge) and 1987 (Port Radium) (Howe, Nair et al. 1986)and (Howe, Nair et al. 1987).

Based on 65 observed lung cancer deaths in the Beaverlodge cohort (N=8487, expected = 34.24), a linear dose-response relationship was observed with a relative risk coefficient of 3.28% per WLM (occupational exposure to RDP is traditionally measured in units of working level months (WLM) with risk coefficients being expressed per 100 working level months or as a percentage per working level month).

Based on 57 lung cancer deaths observed in the Port Radium cohort (N=2103, expected = 24.73), the corresponding relative risk coefficient was estimated to be 0.27%. Both these relationships were highly statistically significant though, obviously, the magnitude of the coefficients for the two cohorts were substantially different. It was conjectured (Howe, Nair et al. 1987) that an inverse exposure rate effect might be responsible for this difference with higher exposure rates typically seen at Port Radium leading to lower risk coefficients.

A later study used a nested case-control within a cohort approach to study the 65 lung cancer deaths in the Beaverlodge cohort, using revised exposure estimates based on the arithmetic mean rather than the geometric mean of radon/RDP measurements and detailed examination of the work history of individual subjects (Howe and Stager 1996). Although exposure estimates are generally increased resulting from the use of the arithmetic mean rather than geometric mean of area measurements, the coefficient was not greatly affected having a value of 3.25%. It may be that the increased precision of the new estimates led to an increase in the estimated risk which compensated for the reduction due to higher exposure estimates.

1.2. Results of Other Studies:

Combined analyses of the data from 11 underground miners' studies have been conducted (Lubin, Boice et al. 1994), (Lubin, Tomasek et al. 1997). The Port Radium and Beaverlodge cohorts were two of the studies that contributed to these analyses. In summary, the relationship between RDP exposure and lung cancer was found to be linear with a slope that depended on time since exposure, exposure rate and age at risk. Risks per unit of exposure decreased with increasing time since exposure, with increasing exposure rate and increasing age at risk. Details of this model are given later in this report.

1.3. Current Study (Updated Eldorado Study):

In August 1991, a Joint Federal-Provincial Panel on Uranium Mining Developments in Northern Saskatchewan was appointed to review various proposed uranium mining developments in Northern Saskatchewan (Panel, 1993). In 1993, the Panel "*recommended that arrangements be made to conduct an on-going epidemiological study of all Saskatchewan uranium miners (past, present and future). It is recommended that the study begin as soon as possible and that the results be promptly communicated to the public*". The federal and Saskatchewan governments agreed to support such a study in their responses to the Panel recommendations.

As discussed in Section 1.1 above, Eldorado Nuclear Limited first sponsored an epidemiological study of past employees in the early 1980s. Eldorado operated uranium mines at Port Radium in the Northwest Territories and at Beaverlodge Lake near Uranium City in Northern Saskatchewan, as well as a uranium refinery and conversion plant in Port Hope, Ontario. In 1988, Eldorado merged with Saskatchewan Mining Development Corporation to form Cameco Corporation.

Public concerns mounted in Port Hope in the 1990s over possible public health risks from the radium and uranium refining in the town. The Port Hope Community Health Concerns Committee (PHCHCC) wanted health studies to investigate the risks. In response to these concerns, Cameco was interested in further follow-up of the Port Hope portion of the Eldorado cohort, about which little was published.

These two events were the impetus to update of the original Eldorado Nuclear Epidemiological Study.

The Atomic Energy Control Board (AECB) (now the Canadian Nuclear Safety Commission (CNSC)), Saskatchewan government, industry and workers groups agreed to the Panel's recommended epidemiology study. In 1995, the Saskatchewan Uranium Miners' Cohort (SUMC) Study Working Group formed to set the overall purpose, direction and focus of the study and keep all stakeholders well informed. The group represented the CNSC, Saskatchewan Health, Saskatchewan Labour, Saskatchewan Northern Health Districts Co-management

Partnership, Saskatchewan Cancer Agency, and both miner and manager representatives of Cameco Corporation and COGEMA Resources Incorporated. Cameco and COGEMA are the two current uranium mine companies in Northern Saskatchewan.

The Study Working Group divided the Panel's recommended epidemiology study into two parts. Part I would update the historical Saskatchewan Beaverlodge miners' cohort study. Part II would create and follow-up a new cohort of modern uranium miners, employed in Saskatchewan since 1975, though currently such a study does not appear to be feasible (SENES Consultants Limited 2003) (the first of the modern Saskatchewan uranium mines, Rabbit Lake, opened in 1974).

In 2002, five Saskatchewan government departments (Labour, Health, Industry and Resources, Northern Affairs and Environment), industry (Cameco and COGEMA), and the CNSC agreed to co-fund the Saskatchewan Uranium Mines' Cohort (SUMC) Study. Financial agreements are in place. A Steering Committee oversees the overall plan and budget related to the SUMC Study.

For Part I, the CNSC and Cameco initially agreed to share the costs of updating Beaverlodge and Port Hope. However, it was more cost-effective to link the entire Eldorado cohort (rather than separate links for the Beaverlodge and Port Hope cohorts) to the Canadian Mortality Data Base (CMDB) and the Canadian Cancer Data Base (CCDB). It was also sensible to include Port Radium in the update since over 600 Beaverlodge miners had radiation exposures from the Port Radium mine. These exposures were important to include and Port Radium could be updated with little added cost. Cameco funded the Port Hope cohort of the study.

The study design for the updated Eldorado study (Beaverlodge, Port Radium, Port Hope, and other sites) was very similar to that used in the original study. The nominal roll was that used in the original study with the addition of workers who joined the Beaverlodge Operation between the cut-off of the original study (31 December 1980) and the final shut-down of the mine in 1982. In fact, the last new entry to the nominal roll was hired on 2 December 1981. A great deal of effort was put into improving the quality and quantity of identifying data in the nominal roll and work histories and this resulted in some deletions and additions for the pre-1980 period This

work was carried out under the supervision of S.E. Frost and is been reported in detail elsewhere (Frost 2005).

At Statistics Canada, the Eldorado cohort file was first linked to itself to identify and remove any duplicate entries. The cohort file was then linked to the tax summary files (1984 to latest year available) using primarily SIN. Matches were validated using full names and dates of birth. Indications of tax filing or death were appended to the cohort file.

This nominal roll was then linked to the Canadian Mortality Database between 1940 and 1999. This database contains records of all deaths amongst Canadians resident in Canada and those Canadians who died in the United States and is thought to be essentially complete. Some studies use linkage to income tax rolls to supplement mortality linkage (Morrison 1997), but, such data were not available to the present study. Identifiers such as given name, surname and day, month and year of birth were used to identify which of the cohort records had a corresponding match in the mortality database. From 1940–1949, the database does not contain causes of death, only date and fact of death. Therefore, this was used for "death clearance" between 1940 and 1949. Those who died between 1940-1949 were eliminated from further analysis. The cause of death is only available from 1950 on, so meaningful statistical analyses can only be conducted for the period 1950–1999.

The Canadian Cancer Registry has existed in computerized form since 1969. It was formed by contributions from individual provincial and territorial cancer registries. Again, it is thought that the Registry is essentially complete for all cases of cancer occurring in Canada. Also, it has the advantage that cancers occurring in provinces different to the original place of residence will be identified. The same linkage techniques were used to identify cancer incidence occurring in the cohort between 1969 and 1999. These linkages have been described in detail previously (Westland 2004).

Exposure estimates and estimates of gamma ray doses were accumulated for the cohort. Radiation dose is expressed in terms of energy absorbed per unit mass of tissue multiplied by modifying factors that take into account the different types and effectiveness of the radiation and

the radiation sensitivity of the various tissues. However, for radon decay products (RDP), because of the complexity and uncertainties of the dose calculations, it is customary to consider only exposure, the product of time in the workplace and the concentration of RDP in the workplace air, usually expressed in Working Level Months (WLM), where 1 WLM is equivalent to one working month (170 hours) in a concentration of RDP that would result in the ultimate release of 1.3×10^5 MeV of potential alpha energy per litre of air.

At Port Hope, film badges were used on some personnel from the late 1940s, coverage was increased in the 1960s, and full external dosimetry was in place by about 1970. At Port Radium, film badges were used for a few short campaigns of personnel and area measurements in the 1950s. At Beaverlodge, similar short campaigns of personnel and area monitoring with film badges took place in the 1950s and starting in 1963 a sampling of workers wore film badges full time. This coverage was gradually increased through the 1970s. For the original Eldorado study, these film results were used to establish annual average dose rates for various jobs, and personal gamma doses were calculated from the average dose rates and time on the job. Other than correcting the odd error that was noted in going through the files, no further work was done on gamma radiation doses for the current study.

At Port Radium workplace measurements, initially of radon and later RDP, started in 1945 and carried on sporadically through the 1940s and 1950s. No personal exposures were calculated. At Beaverlodge both radon and RDP measurements started in 1954 and continued at increasing frequency throughout the life of the mine. Personal exposures were assigned, starting with underground miners in November 1966 and expanding to cover all personnel in the 1970s. No radon monitoring data were available for Port Hope. For the original study, individual annual exposures in WLM were estimated for Port Radium using the annual geometric mean RDP concentration underground and in the mill and time spent in the workplace. For Beaverlodge, the same calculations were done for the period before assignment of individual exposures. For Port Hope, assumptions were made on the radon source term (based on the ore and radium inventory) and building ventilation rates to permit estimates of RDP concentrations for the calculations.

In the current study, the annual individual RDP exposures were recalculated for all Port Radium personnel and for all Beaverlodge personnel for whom no individual exposure assignments had been made during the operation. No new calculations were done for Port Hope. This time annual or seasonal average RDP in WL were used based on the work of SENES Consultants Ltd. For Beaverlodge (SENES Consultants Limited 1996a), the annual mean was calculated by summing over the WL measurements available for each type of workplace, the proportion of employees in each occupation and the proportion of time spent in each type of workplace by employees in each occupation. For Port Radium (SENES Consultants Limited 1996b), a similar approach was taken, but seasonal averages were determined to account for different winter and summer mine ventilation rates, and the rather scant measurement data was augmented by ventilation modeling.

Many Eldorado workers had indicated prior experience in other uranium mines and it was also known that many obtained employment in other uranium mines or other industries with radiation exposure potential after leaving Eldorado. The National Dose Registry (NDR) collects and records radiation exposure and dose data for all exposed workers in Canada with some records going back to 1944. However, the NDR had no early Eldorado records and no records for any of the other Western Canadian uranium mines. Estimates of non-Eldorado exposure were made based on the Beaverlodge WL data, to cover work in the other Western mines. The nominal roll was matched against the NDR records to collect all data on non-Eldorado radiation exposure. This exposure/dose estimation procedure was conducted by S. Frost and J.P. Ashmore and has been described in detail elsewhere (Ashmore 2005), (Frost 2004).

These procedures thus produced data files for potential analysis for three types of data, i.e., factors such as age and gender from the formation of the nominal roll process, deaths and cancer incidence from the record linkage and exposures and doses from the combination of Eldorado records and the National Dose Registry.

This report adds a further 19 years of mortality data for the original Eldorado cohort and also includes cancer incidence results for 31 years, i.e., 1969–1999. It also summarizes the various data processing steps in amalgamating and checking these records, and detailed statistical

analysis which focuses particularly on the relationship between RDP exposure and lung cancer both for mortality and incidence. Other related topics are also considered.

2. MATERIAL AND METHODS

2.1. Material:

The first step was the checking, recoding and data reduction applied to the nominal roll file received from S. E. Frost. This has been described in detail in a previous progress report. Table 2.1 summarizes the number of individuals at various steps in the preprocessing and the corresponding numbers eliminated, e.g., those who had no birth year could not be used in the analysis. The data from the exposure/dose estimation procedure were added to this file by J.P. Ashmore and were then sent to Statistics Canada, though, as can be seen from Table 2.1, not all nominal roll records had corresponding exposure estimates. To be conservative, it was assumed that these exposure/doses were unknown and were dropped from the analysis.

The file that was produced from the above processing was then sent to Statistics Canada to add the deaths and cancer incidence information. Because of confidentiality restrictions under the Canadian Statistics Act hands-on analysis had to be conducted within Statistics Canada in Ottawa. This analysis is described in more detail below.

In total, there were 17,660 records in the final analysis file. Of these, 4,450 have been identified as dying between 1950 and 1999, and occurrence of at least one cancer has been identified for 2,036 individuals. Multiple cancers occurring in the same person were also recorded in these records, though as the respective incidence dates were known the order of occurrence could be identified. These cancers are primary rather than metastatic. The resulting file was the basis for the statistical analysis.

2.2. Characteristics of the Cohort:

The basic characteristics of the cohort for analysis are shown in Table 2.2. As will be seen from Table 2.2, the great majority of the cohort was male which accounted for more than 90% of the cohort. This represents the nature of the occupations involved, i.e., uranium mining and processing Women who were employed generally held office or other jobs not involving much exposure to RDP.

The cohort was subdivided into four groups based upon the employment site where they spent the longest period of time working for Eldorado. By far the largest numbers worked at the Beaverlodge mine in Northern Saskatchewan, with smaller numbers working at the Port Radium mine in the Northwest Territories and Port Hope in Ontario where uranium processing was carried out. As will be seen, a small number of individuals worked at other sites including Head Office, Research and Development, etc. Analyses were carried out using all four subcohorts combined since the primary interest of the study was the relative risk of RDP exposure and all four subcohorts had some RDP exposure. However, some analyses are presented for the subcohorts individually.

The mode of distribution for birth year is in the decade 1920–1929. These individuals would be nearly all in the decade of 70–79 years of age when the mortality and cancer surveillance ended.

Finally, in Table 2.2, the mean values of RDP exposure are shown for the cohort as a whole and for the four sub-cohorts. As would be expected, the maximum mean exposure was at Port Radium which operated primarily in the 1930s and ‘40s when exposure standards would have been less stringent. There is a noticeable drop in the mean value for Beaverlodge which was primarily mined in the 1950s, ‘60s and ‘70s and exposures at Port Hope and for the other sites were minimal.

2.3. Methods of Statistical Analysis:

Two general types of comparison were used for the cohort data. Firstly, observed and expected values were used to estimate standardized mortality ratios (SMR) and standardized incidence ratios (SIR). Expected values were derived from Canadian national population rates for mortality between 1950 and 1999 and for cancer incidence between 1969 and 1999 (R. Semenciw, personal communication). National rather than regional rates were used as death and cancers were spread across Canada and did not appear to be concentrated in specific locations. Expected values were adjusted for gender, age (five-year intervals) and calendar year at risk (five-year intervals). Interval estimates for the SMR and SIR, and p-values testing departure of these values from 1.0 were based on treating the observed numbers of deaths as Poisson variates in the usual way.

The second series of comparisons was based upon internal comparisons, i.e., with no reference to an external population. These were conducted by using Poisson regression analyses (Preston, Lubin et al. 1993). The relative rate estimated from the latter techniques may be expressed as:

$$\text{Relative Rate} = 1.0 + (\beta X) \exp(\Sigma_i \gamma_i z_i)$$

where X represents factors such as RDP exposure or gamma ray dose, z_i are potential modifying factors such as time since exposure and β and γ_i are coefficients estimated using likelihood techniques. Beta is referred to as the excess relative risk (ERR); by adding 1.0 to the ERR one obtains the relative risk at 100 WLM for RDP exposure and per one sievert for gamma ray doses. Regression parameters and p-values were estimated using the method of maximum likelihood using EPICURE software (Preston, Lubin et al. 1993).

Risks may conveniently be modeled as relative risks which multiply the background rate or additive or absolute risks which add to the background rate.

It should be noted that the use of relative rather than absolute risk measures is based on the following considerations:

A. Studies where record linkage has been used to determine outcome are less susceptible to bias in relative rather than absolute measures (Howe, Lindsay et al. 1979).
B. The preferred risk model for RDP exposure and lung cancer chosen by the BEIR VI Committee (BEIR VI 1999) is a relative risk model and thus may be compared directly with the estimates given in this report.
C. Relative risk is probably a more meaningful measure to an individual employed in underground mining who is interested in knowing whether his risk for a particular disease is increased by 50% or 100% or whatever, though of course, the excess absolute risk is also helpful for miners to interpret their own particular risk.

The rest of this report gives results with respect to SMR and SIR analyses (Section 3), RDP exposure and lung cancer mortality (Section 4), RDP exposure and lung cancer incidence

(Section 5) and mortality from other causes and incidence of other cancers and RDP exposure and gamma ray doses (Section 6). In Section 7, the results are summarized and discussed in more detail.

3. COMPARISON OF COHORT WITH GENERAL POPULATION RATES (SMRs AND SIRs)

3.1. Introduction:

A traditional method of analyzing data from cohort studies, particularly occupational cohort studies, is to compare the observed number of outcomes (deaths or cancer cases) from a particular disease with the number which would be expected if gender-, age- and calendar year-specific rates for a comparison population applied to the particular person-years at risk in that cohort. The observed and expected numbers are rates in the cohort and in the population, standardized to the person-year structure of the cohort. Given that the expected rates are based on a large comparison population, it may be shown that the SMR and SIR are maximum likelihood estimates of a relative risk assumed constant across the various strata of gender, age and calendar year, though this latter assumption was not tested.

Such comparisons are of some interest in comparing the mortality or cancer incidence experience of the cohort to a general population. However, they have severe limitations because any factors that differ between the cohort and the population could lead to SMRs or SIRs that are different from 1.0. Such factors could be occupationally related, but also are almost certainly related to environmental and lifestyle factors such as smoking, diet, exercise, etc. A further complication is the so-called "healthy worker effect" (Howe, Chiarelli et al. 1988). It is generally observed that workers are "healthier" than the general population. There are a number of potential reasons for this phenomenon and one reason may be more important than another in a particular cohort. The magnitude of the healthy worker effect also depends upon the specific disease and the specific occupation and also on factors such as time of employment (Howe, Chiarelli et al. 1988). Therefore, interpreting SMRs and SIRs for an occupational cohort must be done with a good deal of caution and departures from 1.0 must be viewed extremely cautiously as, in general, they are more likely to represent differences in lifestyle factors than occupational exposure. If an observed association is very strong, this may be more indicative of an occupational factor, but, unless this is the case, little weight can be placed on such findings.

Nevertheless, the results of such analyses are now presented because of their potential interest to workers in the cohort and because of requests for such analyses.

3.2. Mortality:

Tables 3.1–3.12 give the standardized mortality ratios for up to 39 causes of death. These causes were selected because they had adequate numbers for statistical stability (in general N>10).

Tables 3.1 and 3.2 give the results for males and females, respectively. These are presented separately since occupational exposures are very different for males and females, and risk of death in general also can be affected substantially by gender.

Tables 3.3–3.6 show results for males from the four sub-cohorts contained in the study, i.e., workers at Port Hope, Port Radium, Beaverlodge and other sites. Designation of a worker to a particular sub-cohort was based on the site at which he spent his longest period of employment for Eldorado. There is of course some overlap of subcohorts, but this overlap was small.

SMRs for underground workers at Port Radium and Beaverlodge are presented in Tables 3.7 and 3.8 with results for mill workers at Beaverlodge being presented in Table 3.9 (these definitions were based on the job held for longest period). Finally, Tables 3.10–3.12 represent results for sub-cohorts determined by the start of employment at Port Radium and Beaverlodge, thus, these three sub-cohorts represent workers who started work at later dates than other members of the cohort and, in some cases, it is thought that exposures are more precisely estimated (S. Frost, personal communication) and the interest in others is to see whether reduced RDP exposure in later years reduced the corresponding lung cancer risk.

It should be noted that apart from Table 3.2, all of the other tables are restricted to males because the number of females is too small to permit sensible sub-division.

For males (Table 3.1) generally, the cohort shows reduced risk of cancer mortality compared to the general population. The only SMR which is significantly elevated, as might be expected, is lung cancer, where the cohort displays a 30% higher rate than the general population. The excess of lung cancer certainly has a contribution from RDP exposure, but, also it may well have a contribution from smoking; it is impossible to distinguish the two factors in the present analysis. The reduction in risk for the other cancers probably represents a combination of the

healthy worker effect and different lifestyle factors. For example, miners have a physically strenuous job and thus might be expected to be more physically active than the general population. Amongst non-cancer causes of death, there is a deficit of and ischaemic heart disease, presumably due both to the healthy worker effect and healthy lifestyle. In general, the cohort has similar lower mortality for other non-cancer causes presumably for similar reasons.

However, there are some diseases in which the cohort shows substantially elevated risk compared with the population, namely, hypertensive disease, alcohol-related diseases, motor vehicle accidents, suicide, homicide and other external causes. The excess of hypertensive-related disease has no obvious explanation, though increased medical surveillance for a working population and/or misclassification with other related causes of death could contribute to the excess. For the other elevated causes, this clearly is a matter of lifestyle. One must also recognize that the availability of medical treatment may have been less in the north of Canada where the mines were located as compared to the whole of Canada.

For females (Table 3.2), it is of interest to note that lung cancer mortality is elevated with an SMR of 1.5. It is not statistically significantly different from 1.0, but, given the extremely limited exposure of females to RDP, it might provide an indication of heavier smoking amongst female employees than the general population. For the other causes with insufficient numbers to be shown in the table, general mortality is lower amongst the female members of the cohort than amongst the general population, though none of the differences are statistically significant presumably reflecting smaller numbers.

When the analysis is restricted to sub-cohorts, it will be seen from Table 3.3. that mortality for the Port Hope site (uranium processing rather than uranium mining) generally is less than that of the Canadian population. Lung cancer is only slightly elevated, though deaths from hypertensive disease are significantly elevated ($p=0.003$). However, none of the causes that were elevated in the general male cohort (Table 3.1) such as suicides or homicides are elevated. Presumably, this represents a different set of socioeconomic and lifestyle factors operating for the processors as opposed to the miners.

The results for Port Radium (Table 3.4) in large part reflect those shown for the entire male cohort (Table 3.1). Lung cancers are elevated by 60% likely reflecting the increased exposure to RDP in Port Radium which was in operation in earlier years than, for example, Beaverlodge, though one cannot rule out a contribution from smoking.

The results for Beaverlodge (Table 3.5) again show elevated lung cancer mortality rates with a somewhat lower SMR than for Port Radium (1.3 for Beaverlodge vs. 1.6 for Port Radium). Again external causes are generally elevated for the Beaverlodge cohort, a reflection presumably of lifestyle. The results for other sites (Table 3.6) have few causes of death with at least 10 observed deaths and no statistically significant results. Since this is a very mixed sub-cohort and numbers are small, Table 3.6 is largely uninformative.

For those who worked underground at Port Radium (Table 3.7), there is a notable excess of lung cancer with an SMR of 2.1, presumably reflecting their greater exposure to RDP than surface workers. For other causes of death, external causes are also notably elevated, again likely due to lifestyle considerations.

The pattern for underground miners at Beaverlodge is very similar to that for underground workers at Port Radium (Table 3.8). In contrast, for mill workers at Beaverlodge (Table 3.9), there is no elevation in lung cancer risk. Although several other causes are elevated (suicides and other external causes), of these, only the elevation in suicide rates is statistically significant.

Finally, Tables 3.10–3.12 show results for the various sub-cohorts defined by the date on which they first worked for Eldorado. For those who worked at Port Radium on or after the 1st of April 1956 (Table 3.10) numbers of deaths are small, amounting to about one-quarter of the total Port Radium cohort. Though their lung cancer risk appears to be substantially reduced from the whole Port Radium cohort, this is based on small numbers of deaths so one cannot attribute too much to this observation. For those who first worked at Beaverlodge from 1st of January 1956, this constitutes most of the Beaverlodge cohort and the results are very similar to those for the Beaverlodge cohort as a whole (Table 3.5). Table 3.12 shows SMRs for those first employed at Beaverlodge on or after 1st of January 1970. This is based on approximately one third of the

total Beaverlodge cohort. As will be seen, lung cancer risk is still elevated, with an SMR of 1.8 which in fact is higher than for the Beaverlodge cohort as a whole. However, because of smaller numbers the confidence interval is, of course, wider so it is difficult to interpret this result as reflecting changes in RDP exposure.

3.3. Cancer Incidence:

The SIR results for the same subgroups of the cohort as reported previously for mortality are shown in Tables 3.13–3.24. It should be noted that the population incidence rates are for incidence of newly diagnosed cancer cases and, thus, a single individual can contribute more than one case of cancer to the rates. Thus, the numbers of cases shown in these tables may differ from those where internal comparisons are being made, for example, for lung cancer where a person would contribute only one lung cancer to that analysis.

For all males in the cohort (Table 3.13), results are quite analogous to those for mortality (Table 3.1). For cancer as a whole, there is a deficit amongst Eldorado employees with an SIR of 0.8 which is highly statistically significant. This is reflected in the data for individual cancers, all of which show deficits among Eldorado employees though not all of these are statistically significant. The only exception is lung cancer where overall there is an elevated SIR of 1.2, presumably primarily due to RDP exposure.

For females (Table 3.14), very few cancers have sufficient numbers to give statistically stable estimates. It is of interest to note that lung cancer is elevated in the incidence data as in the mortality data, but cancer as a whole shows a deficit (SIR=0.8).

The site-specific data for the individual four sub-cohorts corresponding to site are shown in Tables 3.15–3.18. For Port Hope (Table 3.15), there are no statistically significant departures of the SIRs from the value of 1.0 and, in general, the Port Hope cohort seems to have cancer incidence that is very similar to the general population. Although lung cancer is elevated, there is only a small and not statistically significant elevation and meaningful interpretation is thus very limited.

Workers at Port Radium (Table 3.16) generally show decreased cancer rates relative to the population with a number of these decreases being statistically significant. However, for lung cancer, as might be expected, there is a significantly elevated SIR; thus, this sub-cohort continued to show an excess of lung cancer many years after the mine was closed.

Table 3.17 gives the results for the Beaverlodge sub-cohort. This shows a similar pattern to the Port Radium sub-cohort with most cancers being decreased relative to the population rate and the rate for lung cancer being increased with an SIR of 1.3. With regard to other sites (Table 3.18), no sensible evaluation can be made because of small numbers other than to observe that overall there is a decreased cancer rate relative to the population (SIR=0.7).

Underground miners at Port Radium (Table 3.19) and Beaverlodge (Table 3.20) show very similar patterns to each other with, again, generally decreased cancer rates relative to the population except for lung cancer where, as expected, there is a highly statistically significant elevation in rate. For mill workers at Beaverlodge (Table 3.21), there is no elevation in lung cancer rates, although, for all causes of cancer there is, again, a decreased SIR of 0.8.

Finally, for the three sub-cohorts defined by date of first working for Eldorado (Tables 3.22–3.24), the Port Radium sub-cohort does not show an elevated lung cancer rate, though this is based on fairly small numbers. For the Beaverlodge sub-cohort, lung cancer is still elevated both in the early cohort (Table 3.23) and the later cohort (Table 3.24).

In summary, the cancer incidence comparisons show consistently elevated rates for lung cancer both in the cohort as a whole and in the various sub-cohorts. Of course, most of this can probably be attributed to RDP exposure, though there could be a component from smoking differences between the cohort and the general population. Other types of cancer are generally decreased relative to the general population and in many cases these decreases are statistically significant. This can have a number of causes including the healthy worker effect (Howe, Chiarelli et al. 1988) and differences in lifestyle in factors such as diet and exercise. It is of interest to note that smoking-related cancers other than lung cancer are generally not elevated in the cohort which would suggest that smoking in the cohort is not substantially elevated relative

to the general Canadian population. This is somewhat surprising since previous studies have shown that miners in general are heavy smokers. One final caveat should be noted in interpreting both SMR and SIR analyses. Mortality and incidence in the cohort is determined by record linkage whereas this is not true of the general population (Krewski 2005). Thus, choice of a threshold of value for accepting links as true matches is more critical than when results are based solely on record linkage. If, for example, too high a threshold is chosen in the former case, it will bias relative risk measures (i.e, SMRs and SIRs) downwards whereas in the latter case (i.e., internal analyses) any possible bias will be much less. Therefore, in interpreting SMRs and SIRs as well as the caveats stressed at the start of this section, this possible bias should be borne in mind.

4. INTERNAL COMPARISON OF RDP EXPOSURE AND LUNG CANCER MORTALITY (1950–1999)

4.1. Introduction:

Lung cancer is by far the most important long-term health consequence of exposure to RDP experienced by underground miners. In fact, there is little, if any, evidence of any other serious long-term health effect arising from such exposure (Darby, Whitley et al. 1995).

This section describes the statistical analysis with respect to the 639 deaths from lung cancer identified in the cohort between 1950 and 1999. There have been three publications addressing this topic based on the previous linkage of Eldorado cohort to mortality between 1950 and 1980 (Howe, Nair et al. 1986), (Howe, Nair et al. 1987), (Howe and Stager 1996). These papers focused on the previous experience of the Beaverlodge cohort (Howe, Nair et al. 1986), (Howe and Stager 1996) and the Port Radium cohort (Howe, Nair et al. 1987). The corresponding results have been described in Section 1.

Data from the previous Beaverlodge and Port Radium studies contributed to a combined analysis of 11 previous studies of underground miners conducted by authors from these studies which were reported initially in 1994 (Lubin, Boice et al. 1994) and updated in 1997 (Lubin, Tomasek et al. 1997). The results from these analyses were the basis for the preferred risk model chosen by the BEIR VI Committee. The latter model assumes a linear dose-response relationship between RDP exposure and excess relative risk of lung cancer, the slope of that relationship being modified by time since exposure with risks decreasing with increasing time, exposure rate with the risks, again, decreasing with increasing exposure rate and age at risk with older individuals being at lower risk than younger individuals. (For further details see Section 4.4.) Allowance for effect modification in the risk model removes much of the apparent heterogeneity among previous results published for these individual studies. This Section describes the application of both the simple linear excess relative risk model and the more complex BEIR VI type model to the new mortality data accumulated for the updated Eldorado study.

4.2. Numbers of Lung Cancer Deaths and Person-Years at Risk:

Table 4.1 shows the distribution of the number of lung cancer deaths and person-years at risk for various categories of RDP exposure expressed as working level months (WLM). Data are shown

separately for men and women and for men by sub-cohort (Port Hope, Port Radium, Beaverlodge and other sites). Membership in a sub-cohort is defined as the site where the individual had spent most time of employment at Eldorado. It was not feasible to split females in this way due to small numbers.

In total, there were 639 deaths from lung cancer and 553,608 person-years at risk. The mean RDP exposure (weighted by person-years) for the cohort was 117 WLM (excluding those with no exposure).

It will be noted that in Table 4.1, the distribution of deaths and person-years for women and for those in the "other sites" sub-cohort are not given. This is because the cells for the number of deaths for these two groups contain less than five deaths and, as stated before, because of the confidentiality requirements of Statistics Canada, these cannot be given. The small number of lung cancer deaths (eight) observed in the "other sites" sub-cohort precludes any meaningful analysis, and the small number of lung cancer deaths among women (21) and their very low exposures to RDP also means that no sensible analyses can be conducted with respect to women. Therefore, subsequent analyses are limited to either all men, or to men employed at Port Hope, Port Radium or Beaverlodge.

The total number of lung cancer deaths observed for men in the Port Radium sub-cohort has increased from 57 (in the 1980 analysis) to 230 in the present analysis, and for Beaverlodge, the corresponding numbers are 65 and 279. Thus, there should be substantial improvement in the power of the study in the new analyses compared to the original analyses.

4.3. Simple Linear Excess Relative Risk Models:

The analyses of the Eldorado cohort reported to date based on mortality until 1980 were presented in terms of the simple linear excess relative risk model (see Section 2.3). These models are useful in understanding the general pattern seen in the cohort and particularly in the sub-cohorts with respect to lung cancer mortality.

To account for potential confounding factors, the background term was stratified by sub-cohort, age at risk and calendar year at risk (see Section 2.3). These terms are all associated both with RDP exposure and independently with risk of lung cancer. Two other variables were available which could be investigated for any potential confounding effect. The total length of employment at Eldorado was statistically significantly associated with risk of lung cancer, with those who worked more than six months having a relative risk of 0.76 as compared to those who worked for less than six months ($X^2_1 = 6.93$, $p = 0.009$). However, the risks did not decrease any further for those who worked increasingly longer periods of time. This phenomenon, i.e., those who worked short term having increased risks of mortality from various causes has been observed previously in a number of studies of workers in various industries (Howe, Chiarelli et al. 1988). When time of employment was included in the background term as a dichotomous variable, i.e., less or more than six months, the estimate for WLM was substantially increased since, of course, time of employment is correlated with exposure and, as stated, inversely associated with risk of lung cancer. For subsequent analyses time of employment (less than six months vs. six months or more), was included as a stratum variable in addition to sub-cohort, age and year at risk.

The other variable available for investigation as a potential confounder was gamma ray dose. Because gamma ray doses were low, this term was included in the background term as a continuous variable, i.e., assuming a log linear relationship which approximates to a linear relationship at low doses. However, the addition of this risk term did not statistically significantly add to the fit of the model ($X^2_1 = 0.02$, $p = 0.88$).

Table 4.2 shows the results of fitting simple linear excess relative risk models to all males, and with males subdivided into the three sub-cohorts.

With the exception of Port Hope, the relationship between RDP exposure and lung cancer mortality risk is highly statistically significant ($p < 0.0001$) based on a test for linear trends. The excess relative risk for Port Hope is positive, though, as stated, not statistically significant. This may reflect the smaller number of cases, and the lower exposures recorded for those in the Port

Hope sub-cohort. It is probably true that most of their exposure was accumulated in other sites, but their longest time of employment was at Port Hope rather than at these other sites.

The estimate of the excess relative risk for Port Radium is 0.37% which may be compared with the value of 0.27% found in the 1980 mortality analysis. For Beaverlodge, the current estimate is 0.96% which, again, may be compared with the previous estimate of 3.25%. Thus, it appears that the ERR for Port Radium is essentially unchanged whereas the ERR for Beaverlodge has decreased substantially. This could be accounted for in part by the early exposures of Port Radium workers for whom the time dependent effect modifiers (time since exposure and age at risk) may be of less importance than for the Beaverlodge sub-cohort.

The models shown in Table 4.2 show no evidence of any curvature with the addition of exposure^2 term in the model ($X^2_1 = 0.19$, $p = 0.66$ for the entire male sub-cohort). Similarly, restricting the analysis to those who reported no non-Eldorado work experience and limiting the analysis to those who had complete time of employment data made little difference to the results shown in Table 4.2 (data not shown). Finally, excluding individuals with no exposure (who may well represent a different group of individuals to those who have some exposure), again, made little difference to the results.

A test of the heterogeneity of the excess relative risk with sub-cohort was conducted (including the subtotal of "other sites"). This yielded a X^2_3 value of 12.45, $p = 0.006$. Thus, the hypothesis of equal ERRs by sub-cohort would be rejected. Again, this emphasizes the importance of considering effect modifiers when expressing risk models for RDP exposure and lung cancer risk (see next section). One must also recognize the possibility of differential confounding among the subcohorts due to possible differential exposure to such confounders.

Table 4.3 shows estimated relative risks by category of RDP exposure, again, for males, and for males subdivided by sub-cohort. The exposure cutpoints were chosen to equally distribute all cases among males between the seven categories. All these comparisons yield highly statistically significant associations, including the risks for Port Hope. The relative risk in the highest exposure category for all males is in excess of 10.0.

In figure 4.1, these relative risks are plotted against the person-year-weighted mean exposure. There is little evidence from this figure to suggest any departure from linearity, other than that which might be due to statistical fluctuations.

4.4. Effect Modification:

The model preferred by the BEIR VI Committee (BEIR VI 1999) has effect modification by time since exposure, exposure rate and age at risk. When potential effect modifiers were investigated in the present data, these were the only variables that suggested some evidence of effect modification. It was decided to fit the preferred BEIR VI risk model to the present data (i.e., all subjects) using the same parameterization as was used for that model. Table 4.4 shows the deviances for the various models and appropriate X^2 values for testing the effect of adding effect modifying terms to the basic simple linear excess relative risk model.

As can be seen from Table 4.4, the addition of time since exposure terms (subdivided into time windows of 5–14, 15–24 and 25+ years since exposure) yields a significant X^2_2 of 14.37, p = 0.0008. The excess relative risks decrease monotonically with increasing years since exposure (see Table 4.5).

Similarly, adding six categories of exposure rate (<0.5, 0.5–1.0, 1.0–3.0, 3.0–5.0, 5.0–15.0 and 15.0+ WLs) provides a further significant improvement in the fit (X^2_5 = 20.17, p = 0.001) again with the excess relative risk showing monotonically decreasing values with increasing exposure rate (see Table 4.5). Finally, the addition of age at risk terms (<55, 55–64, 65–74 and 75+ years) does not lead to a statistically significant improvement in the fit with a X^2_3 value of 7.01, p = 0.07. Thus, there is some suggestion of an improvement in fit, though, not formally statistically significant. Nevertheless, it seems wise to include an age-at-risk term in view of its importance in the BEIR VI model. This is primarily driven in the present data by the large decrease in risk for those aged 75+ years (see Table 4.5). It may be that the number of deaths in the <55 age group is too small to provide stable estimates compared to the other age groups.

Table 4.5 summarizes the parameter estimates for the final interaction model shown in Table 4.4, model 4. It also provides the corresponding parameter estimates from the preferred BEIR VI

model (BEIR VI 1999) In the pooled data, there were 115 lung cancer cases among workers with no occupational WLM exposure and 2,674 among exposed miners, with 353 and 562 lung cancer cases in miners with <50 WLM and <100 WLM, respectively (Lubin, Tomasek et al. 1997). Given the fact that only 122 of the deaths contained in the present analyses contributed to the BEIR VI model, the present results may be regarded as essentially independent of the BEIR VI data set. Generally, there is consistency amongst the present parameter estimates and those provided by the BEIR VI model, apart from potentially random variations. In fact, most of the parameter estimates of the BEIR VI model all lie within the confidence intervals of the present parameter estimates.

Thus, the present data set provides further evidence of the importance of effect modifiers in the relationship between RDP exposure and lung cancer mortality risk and suggests that the BEIR VI model performs well in an essentially independent data set.

Finally, with the model shown in Table 4.5, tests of the heterogeneity of the exposure effect were conducted by adding appropriate interaction terms for the sub-cohort. This yielded an X^2_3 of 4.45, $p = 0.22$. It should be noted that this model assumes the same modifying effects and parameter values for all sub-cohorts and only the main effect of exposure is being tested. It further assumes that the size of the modification by sub-cohort applies equally to all time windows of exposure. However, the study lacks power to further test heterogeneity of all the parameters shown in the model.

5. INTERNAL COMPARISON OF RDP EXPOSURE AND LUNG CANCER INCIDENCE (1969–1999)

5.1. Introduction:

Most of the previously conducted studies of underground miners exposed to RDP have been of mortality. This generally has occurred because incidence records with which to identify new cancer cases in a cohort have been less readily available, particularly in the past, than mortality records.

The existence of the Canadian Cancer Database maintained by Statistics Canada and covering all new cases of cancer found in Canada between 1969 and 1999 has provided a useful opportunity to examine cancer incidence in general and lung cancer incidence in particular in the Eldorado cohort, both as a partly independent check of the results of the mortality analysis and for direct comparison with mortality results themselves. Since relative risk measures are the primary statistic of interest in the present context, given that survival of lung cancer cases is generally short and independent of the source of lung cancer, relative risk measures from mortality and incidence analyses should approximate closely to each other.

Table 5.1 compares the number of lung cancer deaths and lung cancer cases (i.e., incidence) found in the Eldorado cohort for various years. As will be seen from the table, in total, 789 individuals in the cohort developed lung cancer and died from lung cancer during the study period, or died of lung cancer without a previous incidence record or developed lung cancer, but, did not die from the disease.

The total number of deaths (1950–1999) and the total number of cancer cases (1969–1999) were numerically similar though, of course, many of these refer to the same individual. There was a relatively small number of deaths before 1969 when the cancer surveillance started (N=83) with the majority of deaths occurring during the period 1969–1999. A total of 48 individuals died of lung cancer between 1969 and 1999 without there being a corresponding incidence record. Some of these could have been diagnosed with lung cancer earlier than 1969, but, it appears there is a small percentage of deaths which were simply not picked up by the cancer registration system or where lung cancer was established by autopsy and not reported to the Cancer Registry.

Finally, there are 150 cases of lung cancer for which as yet there is no death record; these then represent a contribution to the incidence analysis which is independent of the mortality analysis.

5.2. Cases and Person-years at Risk:

Table 5.2 (parallel to Table 4.1) shows the observed cases and person-years at risk for males and females separately, again with the males being subdivided by sub-cohort (Port Hope, Port Radium, Beaverlodge and "other sites"). Each subject could contribute at most one cancer since multiple cancers at one site for an individual are hard to interpret. Again, females could not be meaningfully so divided because of small numbers. As for the mortality analysis, numbers are not shown for some cells in Table 5.2 to preserve confidentiality under Statistic Canada's rules, i.e., for cells with less than five observed. Further, no meaningful analysis could be performed for females or for workers in the "other sites" category because of small numbers and for women because of very low exposures.

5.3. Simple Linear Excess Relative Risk Models:

As with the mortality model (Section 4), potential confounders of an association between RDP and lung cancer incidence risk were first identified. Again, age at risk, calendar year at risk, sub-cohort and days of employment all turned out to be covariates affecting lung cancer risk, and subsequent analyses described in this section are all controlled for the effects of these variables using stratification. In particular, lower risk of lung cancer was incurred by those who worked for six months or more for Eldorado with a relative risk of 0.72 compared to those who worked less than six months ($p=0.0007$).

There was no significant effect of gamma ray doses (expressed as a log linear relationship as with mortality) with a slope of 0.064 per sievert and a corresponding p-value of 0.84.

The inclusion of a quadratic term in WLM showed no evidence of an improvement in fit with a p-value of 0.93 and excluding various subgroups of the cohort, e.g., those with some recorded non-Eldorado experience, those with incomplete Eldorado dates of employment and those with zero exposure essentially made little difference to the results (results not shown).

Table 5.3 shows the estimated excess relative risks for men as a whole and for three of the sub-cohorts, i.e., Port Hope, Port Radium and Beaverlodge. The estimate for males combined is 0.55 (95% confidence interval: 0.36, 0.81), a result which is very similar to that reported in Section 4 for the mortality analysis.

The estimate for Beaverlodge based on incidence is somewhat reduced from the estimate obtained for mortality whereas the Port Radium estimate is quite similar between the two analyses. Again, the relationships for Port Radium and Beaverlodge are highly statistically significant ($p<0.0001$).

These changes, i.e., between incidence and mortality, may have a contribution from chance, or are possibly due to the effects of time-dependent effect modifiers representing a different time pattern occurring in the mortality and incidence analyses.

Finally, testing the heterogeneity of the ERR across sub-cohort yields a χ^2_3 value of 10.25, $p=0.02$. Thus, there still remains an apparent heterogeneity of effect by sub-cohort when using a simple excess relative risk model.

Relative risks estimated for various categories of WLM exposure are shown in Table 5.4 and Figure 5.1, again, for all males combined and for the three individual sub-cohorts (Port Hope, Port Radium and Beaverlodge). The pattern of relative risks are generally monotonically increasing with the highest risks being seen in the highest category, and the risk for the highest exposure having a value of more than eleven-fold for the combined male cohort.

5.4. Effect Modification:

The same interaction models used for the mortality analysis described in Section 4 were fitted to the incidence data. Again, the effect modifiers considered were those selected by the BEIR VI Committee, i.e., time since exposure, exposure rate and age at risk, which again were the only variables displaying some evidence of effect modification. The deviances for the various models and corresponding χ^2 tests are given in Table 5.5 which shows the effect of adding one modifying effect variable at a time.

Splitting total WLM exposure into three time windows (5–14, 15–24 and 25+ years) significantly improves the fit of the model (p=0.0002). Inclusion of exposure rate in the model using the same six exposure rates as previously defined, does not lead to a statistically significant improvement (p=0.13), though the corresponding estimates for the exposure rate effect (Table 5.6) again show in general decreasing effects per exposure unit with increasing exposure rate.

Finally, adding terms in age at risk leads to a p-value of 0.28 with a somewhat irregular pattern of corresponding estimates (Table 5.6). This again may reflect the comparatively small number of lung cancer cases below age 55 which, in turn, leads to a correspondingly somewhat unstable relative risk estimate for the other age categories. As with the mortality, however, those past age 75 appear to have a decreased risk per unit of exposure as compared to those developing lung cancer at younger ages.

Table 5.6 presents the estimate derived from Model 4 in Table 5.5, again, with a corresponding estimate from the preferred BEIR VI model. These estimates can also be compared with those given for the corresponding mortality analysis in Table 4.5.

Generally, the estimates for WLM time windows, and for exposure rate are similar between the present study and the BEIR VI estimates, and are very similar to the estimates for the mortality analysis. With respect to age at risk, the estimates do differ somewhat from the BEIR VI estimates and also from the mortality analysis, though the risk for all three series is substantially decreased for those age 75 years or more. These latter differences could be due to limitations of statistical power for the Eldorado cohort incidence and mortality analyses.

Overall, therefore, the incidence analysis for lung cancer shows very similar patterns to the mortality analysis. Although there is a substantial overlap in the "cases" used in the two analyses with 508 individuals contributing to both analyses, there do remain in both analyses more than 100 individuals contributing independently to the two analyses.

A test of the heterogeneity of effect across sub-cohorts of male Eldorado employees yields a χ^2_3 test of 5.40, p=0.17. As with the mortality analysis, this only reflects differences in the effects of

the various time windows of RDP exposure and assumes the other modifying effects have the same value for each of the sub-cohorts. There was insufficient power in the data to test separately for any differences in the other modifying effects. Thus, again, inclusion of modifying effects, as shown in Tables 5.5 and 5.6, reduces any statistical heterogeneity in the RDP effect among the sub-cohorts.

5.5. Effect of Cell Type:

Unlike the mortality data, the incidence data contains codes describing the cell type of the various lung cancers observed during the cancer linkage. Squamous cell cancers represented the largest single histologic type (32.9%), and small cell and adenocarcinoma types were present in smaller, but similar proportions, 16.2% and 17.8% respectively. There were only 22 cases of large cell lung cancer which were included with other cancers (33.1% of the total number of cancers).

BEIR VI (BEIR VI 1999) cites several reviews of histologic types of lung cancer in underground miners and in the general population, and concludes that none of them has proven to be a definitive indicator of exposure to radon decay products. Several studies, however, have observed higher prevalence of small cell histologic types among radiation-associated lung cancers in uranium miners (Yao 1994); (Land 1993); (BEIR IV 1988). Continued follow-up of these cohorts showed significant changes over time in the histopathology of lung cancer, with small cell predominance usually decreasing over time and falling to the level observed in the general population (Saccomanno 1988).

Table 5.7 shows that the distribution of the three main histologic types of lung cancer was very similar for Port Radium and Beaverlodge facilities. This distribution of cases was also similar to the distribution of cell types of lung cancer in the general population of Alberta where in 1973-1997 (the latest data available), 36 percent of cases were squamous cell type, 19% adenocarcinoma and 16% small cell type (Cancer Incidence on Five Continents, Vol. VIII) (Parkin 2003). While the distribution of cases differed somewhat in the Port Hope subcohort, it was not very different from the distribution of cell type in the general population of the province of Ontario. From 1978-1997 (the latest data available), more than 14,000 cases of lung cancer

have been diagnosed in Ontario (Cancer Incidence on Five Continents, Vol. VIII). The relative contribution of the squamous, adeno- and small cell types of lung cancer during that time was 29%, 21% and 13% respectively, which is very comparable to the distribution of cases observed in the Port Hope facility (25%, 21% and 8% respectively). The distribution of the main histologic types remained relatively stable over time.

Table 5.8 presents the ERR estimates for the three main histologic types of lung cancer. There was some heterogeneity of risks depending on the histologic type, with the highest risk observed for squamous cell lung cancers and the lowest for adenocarcinomas, ERR=0.80 and ERR=0.33 respectively. A formal tests showed that this heterogeneity approached statistical significance (p=0.07). Numbers were too small to fit a BEIR VI-type model to the cell specific data which might have accounted for some of this heterogeneity.

6. INTERNAL COMPARISON OF RDP EXPOSURE AND GAMMA RAY DOSE WITH MORTALITY (1950–1999) AND CANCER INCIDENCE (1969–1999) FOR DISEASES OTHER THAN LUNG CANCER

6.1. Introduction:

Generally, there is little evidence that exposure to RDP will increase the risk of diseases other than lung cancer in those so exposed. An analysis of cancer mortality based on the 11 underground miners studies used in the BEIR VI report showed essentially no evidence of any increased risk of cancers other than lung cancer (Darby, Whitley et al. 1995). Nor have individual studies reported any meaningful evidence of increases for diseases other than lung cancer.

On the basis of an ecological study, it was suggested that domestic radon exposure was correlated with rates of leukemia internationally (Laurier, Valenty et al. 2001) though, essentially, since that suggestion there has been no confirmatory evidence.

The other exposure of interest in the present study is the dose accumulated from gamma ray exposure. It is known, of course, that comparatively high doses of gamma rays can increase the risk of a number of cancers (UNSCEAR 2000), with leukemia being particularly sensitive to such exposure. However, in the present context, the mean gamma ray dose of subjects in the Eldorado cohort was fairly low, so it is certainly possible that the study lacks statistical power to detect an effect of exposure to gamma rays.

This section, therefore empirically examines the relationship between RDP exposure and gamma ray doses and causes of death and cancer incidence other than lung cancer. Unlike the material presented in Section 3 which simply presents the observed and expected values for such deaths and cancers, in this section, the presence of any dose-response relationship with the two exposures is examined by using a simple linear excess relative risk model. This may be regarded as a "screening technique," though radiobiologic theory would suggest that a radiation dose-response relationship might be expected if any detectable risk occurs.

The criterion used for choosing the various causes of death and the various cancer incidence data examined was that it was restricted to those situations in the cohort where there are 50 or more deaths, and/or 50 or more cases of the disease under consideration. The only exception to this latter rule was to include leukemia where there were fewer than 50 deaths and/or cases. The "50" rule was applied because generally numbers less than this tend to give quite unstable risk estimates and, indeed, even with numbers of 50 or more, some of the models proved unstable and could not provide a maximum likelihood estimate. Leukemia was included because of the special interest in leukemia referred to above because of its known radiation sensitivity.

6.2. RDP Exposure and Mortality:

The results of fitting the linear excess relative risk model to RDP exposure are shown in Table 6.1 for 22 causes of death with 50 or more deaths observed in the Eldorado cohort between 1950 and 1999 (for leukemia n=34). It should be noted that mortality analysis utilizes all leukemia together since further subdivision was not available until 1969 and analyses restricted to the period 1969–1999 would further reduce the already small number of deaths for analysis.

For two causes of death, namely, stomach cancer and alcohol-related causes, a stable estimate of the ERR could not be obtained, presumably because the estimate was approaching its lower limit which would lead to a negative relative risk for some cells.

None of the results shown in Table 6.1 are statistically significant except for stroke which shows a negative association and accidents other than motor vehicle accidents where there is a positive association with a p-value<0.01. Given the fact that 22 comparisons have been made, it is hardly surprising that one or two could be statistically significant by chance. However, the positive association with accidents could obviously be related to employment particularly in underground mining though there appears to be no obvious reason why the ***rate*** of deaths from accidents should increase with increasing RDP exposure. Although the results shown in Table 6.1 are adjusted for time of employment (six or more months vs. <6 months) a second analysis was carried out in which the adjustment for time of employment was carried out using a continuous variable, i.e., days of employment. With this adjustment, the accident risk estimate shown in Table 6.1 was reduced from 0.18 to 0.06% and the corresponding p-value increased to 0.10.

This would suggest that time of employment explains the relationship shown in Table 6.1 rather than actual exposure to RDP, as might be expected. Although for some chronic diseases the healthy worker effect ((Howe, Chiarelli et al. 1988) decreases with increasing time of employment, it is hard to see how accidents would be affected by this phenomena.

Overall, generally, there is no evidence from Table 6.1 that mortality from causes other than lung cancer is increased by exposure to RDP.

6.3. Gamma Ray Dose and Mortality:

Table 6.2 shows the results of fitting a simple linear excess relative risk model in gamma ray dose for the same 22 causes of death shown in Table 6.1. In this case, three of the models failed to give a maximum likelihood estimate of the risk and two of these (infectious diseases and pancreatic cancer) are at or approaching their lower bound, and one of them (suicide) gives a positive value for the excess relative risk.

The only statistically significant association seen in Table 6.2 is again for accidents other than motor vehicle accidents where there is a positive relationship ($p=0.03$). Again, adjusting for time of employment as a continuous variable substantially reduces this association and it is no longer statistically significant ($p=0.25$).

Overall, therefore, the data shown in Table 6.2 show no evidence of any detectable association between gamma ray doses and risk of mortality for the diseases shown. In particular, there is no evidence of a positive association with total leukemia. As stated above, this could be a reflection of the fact that gamma ray doses were too low in the cohort to have sufficient statistical power to detect any such effect, but, it is reassuring that at the doses experienced by the Eldorado cohort, there appears to be no demonstrable effect on risk of mortality.

6.4. RDP Exposure and Cancer Incidence:

The results of fitting the simple linear excess relative risk model for RDP exposure and for the 11 cancers that yielded 50 or more cases for the period 1969–1999 are shown in Table 6.3.

None of the results shown in Table 6.3 are statistically significant. In particular, neither chronic lymphatic leukemia nor leukemia excluding chronic lymphatic leukemia has a positive association.

Overall, therefore, these results display no meaningful evidence of any detectable association between RDP exposure and the cancers listed in this table.

6.5. Gamma Ray Dose and Cancer Incidence:

Finally, Table 6.4 shows the results of fitting the simple linear excess relative risk model to the data for gamma ray doses and risk of the same 11 cancers shown in the previous table. For two of these cancers, the maximum likelihood estimate of the ERR could not be obtained, again, presumably due to approaching the lower limit of the estimation procedure.

None of the results in Table 6.4 are statistically significant. Although there is a positive estimate for bladder cancer of 2.83% per sievert, this is not statistically significant (p=0.15). For the leukemia analyses, CLL shows a positive association with an ERR of 7.28 per sievert whereas leukemia other than CLL gives a negative estimate. However, neither of these observations is statistically significant.

Thus, it appears that again there is no detectable increase in risk associated with gamma ray dose for any of the cancers shown in Table 6.4.

7. SUMMARY AND DISCUSSION

This report presents the results of the statistical analysis of a cohort of 17,660 individuals known to have worked for Eldorado somewhere between 1930 and 1999. Based on a total of 5,332 deaths between 1950 and 1999, and 2,355 individuals who developed at least one cancer between 1969 and 1999, several types of analyses have been conducted.

Several considerations should be taken into account in interpreting the present results. First of all is the potential effect of smoking. Smoking is, of course, the primary cause of lung cancer with relative risks for current smokers being of the order of 20-fold. Thus, it is important to consider the potential impact of smoking on RDP risk estimates.

The present analysis has assumed that risk factors are acting multiplicatively in causing lung cancer for reasons previously specified. If this is the case, and smoking is not correlated with RDP exposure for a given cohort, age and year then smoking should not affect the risk estimates for RDP exposure. If smoking under the same circumstances is positively correlated with RDP exposure, RDP relative risk estimates would be inflated and vice versa. Although no data on smoking are available for the present cohort, a previous case-control study of the Beaverlodge cohort (L'Abbe, Howe et al. 1991) suggested that such correlation was not present and, in general, occupational studies frequently show a lack of any strong correlation between occupational exposures and smoking. Thus, the background level of smoking in the subcohorts under these conditions, i.e., a multiplicative effect and smoking and RDP exposure uncorrelated would not affect relative risk estimates for the various subcohorts.

If the relative risks of smoking and RDP exposure are not multiplicative, e.g., if they are additive, then the background level of smoking will affect the relative risk estimates with a population that smokes more having increased biases in the relative risk estimate towards the null. In practice, according to the BEIR VI Committee and based on limited data, the interaction between smoking and RDP exposure on risk of lung cancer appears to be intermediate between multiplicative and additive. In the absence of smoking data for the cohort, this possibility must be recognized, but cannot be adjusted quantitatively.

Similarly, if the effect of smoking and RDP exposure is not strictly multiplicative, smokers will have different risks from RDP exposure for lung cancer than non-smokers when considering the relative risk scale. This would correspondent to effect modification of the RDP exposure by smoking. Again, in the absence of smoking data, one cannot address this possible phenomenon, but recognize the possibility of its existence.

The second issue that should be taken into account in interpreting the results is the possible presence of other risk factors for lung cancer in the Eldorado environment. Again, there are no data available for the cohort on such potential risk factors and, again, the interpretation with respect to such factors will have the same considerations as those given for smoking.

A third consideration in interpreting results is measurement error in exposure estimation. Such error almost certainly has decreased with calendar time and, thus, the Port Radium cohort will have a greater measurement error than the Beaverlodge cohort and recent employees will have lower mean errors than those working further back in time. A further consideration in this respect is that of domestic exposure to radon which likely would have a relatively greater contribution to total exposure in recent times when occupational exposures have been lower.

The impact of such measurement error depends on a number of factors, in particular, the quantitative nature of the error and the risk function that has been considered. For example, if there is no correlation between domestic exposure and total occupational exposure, the relative risk relationship would be unaffected and no bias would result. In general, however, random misclassification is known to bias relative risk estimates towards the null and this too should be borne in mind in interpreting the present results.

The first analysis was a comparison of the mortality of the cohort with the mortality of the general Canadian population between 1950 and 1999. Lung cancer was elevated not only in the whole cohort, but in various sub-cohorts defined by gender, site of working, underground and mill workers and sub-cohorts defined by first working date for Eldorado. There can be no doubt that much of this excess is attributable to RDP exposure as discussed in more detail subsequently. However, females and processors who worked at Port Hope still appear to have

elevated though non-statistically significant lung cancer risks which could suggest that the smoking habits of Eldorado employees differed from the general population and this too might have contributed to increased lung cancer risk in the cohort as a whole.

For most of the causes of death, the cohort as a whole and the various sub-cohorts had reduced risks relative to the population. This probably represents the healthy worker effect, a supposition which is supported, for example, by the major decrease in ischaemic heart disease to reflect the fact that the risk of heart disease is lower in the cohort being studied than in the population at large. The latter condition would probably prevent people working in a strenuous physical occupation such as mining.

The most notable exceptions for causes of death where rates in the Eldorado cohort were elevated relative to the population include hypertensive causes, and external causes such as homicides, suicides and both traffic and non-traffic-related accidents. The excess of hypertensive disease has no obvious explanation, but is present in a number of the sub-cohorts, but with statistical significance in only one, and is in contrast to the deficit of cardiovascular disease such as stroke and ischaemic heart disease. If this proves to be a real phenomenon and is still operative in the cohort, this could suggest a program of regular blood pressure checks and appropriate medication, but such a recommendation is clearly beyond the scope of the present report.

With respect to the excess of external causes, this probably represents a combination of job-related accidents and lifestyle factors amongst miners as a whole. The observation of a substantially elevated suicide rate is of particular interest, but there are no data for the cohort to examine this phenomenon further other than note its existence.

Comparisons of the cohort with the general Canadian population with respect to cancer incidence rates between 1969 and 1999 are also reported. The only cancer that is consistently elevated is lung cancer, thus, mirroring the mortality results discussed above. For cancer as a whole and for specific cancers, generally rates are lower than for the population which again is likely to be a manifestation of the healthy worker effect.

The comparison of the cohort with the general Canadian population rates needs very cautious interpretation. Differences could of course be due to various occupational exposures, but, also could and are perhaps more likely to have a more substantial contribution from lifestyle and environmental factors such as exercise and diet. The healthy worker effect which has a contribution from differences in lifestyle and environmental factors as well as other considerations also needs to be borne in mind. Finally, mortality and cancer incidence of the cohort has been determined by using record linkage whereas the population data have been determined by direct ascertainment. This produces a risk of bias in the estimation of both SMRs and SIRs. Both mortality and incidence may be underestimated by choosing a threshold for record linkage that is too high which tends to happen when one requires a high degree of certainty that the chosen links are valid. This bias does not affect to such an extent measures based on internal comparisons (see below), but is more of a problem when comparing with an external population (Krewski 2005).

Nevertheless, the main findings of increased lung cancer rates, increased rates of hypertensive diseases and external causes of death and decreased rates for other diseases is probably not accounted for in total by such a potential bias and this pattern is probably reflective of the true mortality and cancer incidence experience of the cohort.

The second analysis presented in the present report is that of mortality from lung cancer with respect to RDP exposure (Section 4). This is primarily based on 618 lung cancer deaths amongst men in the cohort. This compares with previous analyses of the Eldorado cohort when the total number of such deaths was 122. Thus, the present analysis represents a substantial increase in the power of the study and, thus, should produce more precise estimates.

There is a strong positive monotonic increase in risk of lung cancer death with increasing RDP exposure which is highly statistically significant. This increase generally manifests itself for the three main sites (Port Hope, Port Radium and Beaverlodge sites), although it is likely that the exposure of the “Port Hope” sub-cohort comes primarily from exposure received at other sites. However, fitting a simple linear excess relative risk model to the data although, again,

demonstrating a strong relationship with RDP exposure does provide excess relative risk estimates that are inconsistent across the sites.

Application of the BEIR VI-type risk model which allows for effect modification by time since exposure, exposure rate and age at risk reduces this statistical heterogeneity in terms of the RDP effect and it is clear that these modifying factors contribute to the apparent heterogeneity seen in this simple linear excess relative risk model. Using the same parameterization as chosen by the BEIR VI Committee leads to coefficient estimates that are very similar in the present study as reported by the BEIR VI Committee. The BEIR VI estimates were based on 11 underground miners' studies including the previous analysis of the Port Radium and Beaverlodge cohorts, but, as stated above, the present study is based on a substantially greater number of lung cancers. Therefore, the present results may be regarded as essentially independent of the data used by the BEIR VI Committee and thus, provide a useful confirmation of the results obtained by BEIR VI. There is some irregularity in the present analysis of the effect of age at risk as compared to the BEIR VI model, but the present data do indicate a substantial drop in risk for those age 75 years or more.

The lung cancer mortality analysis detected no effect of gamma ray exposure on risk of lung cancer mortality and all estimates did not change by including or excluding those with non-Eldorado work histories or those with zero exposure to RDP.

An analysis of lung cancer incidence amongst males was also conducted (Section 5). The results mirror those of the mortality analysis and as relative measures are used, this is hardly surprising. It should also be noted that the two analyses are not independent in that a substantial proportion of cancer cases contributed to the corresponding death analysis.

The final type of analysis conducted was that examining mortality and cancer incidence for diseases other than lung cancer with RDP exposure and gamma ray exposure (Section 6). In summary, there is no meaningful evidence of any causal relationship between RDP exposure and increased risk of any of these other diseases, nor was there any meaningful evidence of a relationship with gamma ray dose. Where there was a suggestion of such a relationship, this

appeared to be accounted for in large part by higher doses representing more years of work, a phenomenon seen in many occupational studies which is part of the healthy worker effect. Similarly, there is no meaningful evidence of any consistent association with either RDP exposure or gamma ray dose for the cancer incidence results.

Exposure to RDP is one of the best-studied carcinogenic phenomena in epidemiology. The results obtained from these studies, primarily of underground miners, are very consistent in showing increases in lung cancer risk from such exposure, but no increase in any other disease. These results are consistent with physiological considerations (i.e., where radon decay products are deposited in the body) and with many animal studies. The quantitative results of these studies are also very consistent with the model proposed by the BEIR VI Committee providing a good fit to most data sets.

The present study (which is essentially independent of the data set used by the BEIR VI Committee) further supports these conclusions based on 50 years' mortality experience and 31 years of cancer incidence experience. They certainly support the use of BEIR VI-type models to predict any group's future risk of lung cancer from RDP exposure either from past or current such exposure. No measurable risks of leukemia, lung cancer or any other disease were demonstrated to be associated with gamma ray exposure in the present cohort. For predicting risk of the effects of gamma ray exposure, use of currently accepted risk models as published by the BEIR Committees and UNSCEAR Committees would appear to be appropriate and not contradicted by the results of the present study.

Finally, it is worth noting that as yet only about 25% of the cohort has died. Future follow-up and analysis of this cohort with respect to both mortality and cancer incidence should shed further light on our knowledge on the effects of uranium mining and processing in both Saskatchewan and other provinces upon the resulting health of those employed in such occupations.

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TABLES 2.1 – 6.4

TABLE 2.1

NUMBER OF REJECTIONS DURING DATA PROCESSING

Original nominal roll file as received	19855
Rejections:	
Missing gender	3
Missing birth year	1866
No occupational record*	42
Age of employment (15-75) out of range	35
Missing dose data	116
Last contact before 1940	24
Recorded exposure after postulated death link	9
Age >100 and no death link found	100
Total rejections	2195
Total left for analysis	17660

* Due to error in occupational record

TABLE 2.2

BASIC CHARACTERISTICS OF THE COHORT

Characteristics	Value	N	%
Sex	Males	16236	91.9
	Females	1424	8.1
Site	Port Hope	3003	17.0
	Port Radium	3300	18.7
	Beaverlodge	10050	56.9
	Other sites	1307	7.4
Birth Year	-1900	414	2.3
	-1910	1028	5.8
	-1920	1803	10.2
	-1930	4030	22.8
	-1940	3913	22.2
	-1950	2790	15.8
	-1960	3118	17.7
	1960+	564	3.2

Cohort/Sub-cohort	Mean RDP Exposure (WLM)	Standard Deviation
Entire cohort	48.0	182.6
Port Hope	12.5	43.4
Port Radium	174.2	369.1
Beaverlodge	23.2	81.7
Other sites	1.9	32.9

TABLE 3.1

STANDARDISED MORTALITY RATIOS (SMR) AND 95% CONFIDENCE INTERVAL COMPARED TO CANADIAN MORTALITY RATES (1950-1999) FOR SELECTED CAUSES

MALES

Cause	Observed	Expected	SMR	Lower Limit	Upper Limit	p-value
All infectious diseases	53	58.6	0.9	0.7	1.2	0.513
Buccal cavity cancer	25	37.7	0.7	0.4	1	0.038
Esophageal cancer	25	37.1	0.7	0.4	1	0.047
Liver cancer	16	16.7	1	0.5	1.6	0.991
Stomach cancer	75	81.7	0.9	0.7	1.2	0.501
Colon cancer	82	134.3	0.6	0.5	0.8	<0.001
Rectal cancer	46	44.7	1	0.8	1.4	0.888
Pancreatic cancer	67	72.5	0.9	0.7	1.2	0.568
Laryngeal cancer	19	21.3	0.9	0.5	1.4	0.713
Lung cancer	618	470.3	1.3	1.2	1.4	<0.001
Skin cancer	17	19.1	0.9	0.5	1.4	0.739
Prostate cancer	98	123.8	0.8	0.6	1	0.019
Kidney cancer	25	35.6	0.7	0.5	1	0.08
Bladder and other urinary cancer	29	39.4	0.7	0.5	1.1	0.106
Brain and other cns cancer	33	41.6	0.8	0.5	1.1	0.204
Non-Hodgkins lymphoma	42	46.3	0.9	0.7	1.2	0.59
Multiple myeloma	18	22.1	0.8	0.5	1.3	0.45
All leukemia	34	49	0.7	0.5	1	0.031
Diabetes mellitus	64	97.8	0.7	0.5	0.8	<0.001
All mental disorders	29	66	0.4	0.3	0.6	<0.001
All nervous system diseases	61	92.2	0.7	0.5	0.8	0.001
Hypertensive disease	42	24.5	1.7	1.2	2.3	0.002
Ischemic heart disease	1235	1508.1	0.8	0.8	0.9	<0.001
Stroke	244	309.4	0.8	0.7	0.9	<0.001
All other cardiovascular disease	317	363.7	0.9	0.8	1	0.014
Chronic obstructive lung disease	79	210.8	0.4	0.3	0.5	<0.001
Pneumonia	134	130.9	1	0.9	1.2	0.809
All digestive diseases	179	222.5	0.8	0.7	0.9	0.003
Alcoholism and related	54	34.8	1.6	1.2	2	0.003
Genitourinary diseases	51	73.1	0.7	0.5	0.9	0.008
Musculoskeletal and connective	11	11.5	1	0.5	1.7	0.999
Motor vehicle accidents	180	139.1	1.3	1.1	1.5	0.001
Suicide	208	122.7	1.7	1.5	1.9	<0.001
Homicide	28	14.7	1.9	1.3	2.7	0.003
Other external causes	365	215.6	1.7	1.5	1.9	<0.001
All cancers	1406	1425.7	1	0.9	1	0.613
All causes	5148	5284.2	1	0.9	1	0.061

Adjusted for age and calendar year at risk

TABLE 3.2

STANDARDISED MORTALITY RATIOS (SMR) AND 95% CONFIDENCE INTERVAL COMPARED TO CANADIAN MORTALITY RATES (1950-1999) FOR SELECTED CAUSES

FEMALES

Cause	Observed	Expected	SMR	Lower Limit	Upper Limit	p-value
Lung cancer	21	14.4	1.5	0.9	2.2	0.121
Breast cancer	16	17.8	0.9	0.5	1.5	0.777
Ischemic heart disease	34	44.9	0.8	0.5	1.1	0.112
Stroke	14	18.3	0.8	0.4	1.3	0.381
All other cardiovascular disease	10	17	0.6	0.3	1.1	0.098
All cancers	74	81.4	0.9	0.7	1.1	0.447
All causes	184	234.2	0.8	0.7	0.9	0.001

Adjusted for age and calendar year at risk

TABLE 3.3

STANDARDISED MORTALITY RATIOS (SMR) AND 95% CONFIDENCE INTERVAL COMPARED TO CANADIAN MORTALITY RATES (1950-1999) FOR SELECTED CAUSES

MALES: PORT HOPE

Cause	Observed	Expected	SMR	Lower Limit	Upper Limit	p-value
Stomach cancer	14	18.4	0.8	0.4	1.3	0.371
Colon cancer	22	26.9	0.8	0.5	1.2	0.404
Rectal cancer	16	9.4	1.7	1	2.8	0.064
Pancreatic cancer	11	14.7	0.7	0.4	1.3	0.411
Lung cancer	101	92	1.1	0.9	1.3	0.376
Prostate cancer	21	25.9	0.8	0.5	1.2	0.388
Bladder and other urinary cancer	11	8.3	1.3	0.7	2.4	0.433
Diabetes mellitus	14	19.5	0.7	0.4	1.2	0.253
All nervous system diseases	12	17.6	0.7	0.4	1.2	0.213
Hypertensive disease	13	4.9	2.7	1.4	4.5	0.003
Ischemic heart disease	345	324.2	1.1	1	1.2	0.261
Stroke	72	69.3	1	0.8	1.3	0.777
All other cardiovascular disease	85	79.4	1.1	0.9	1.3	0.555
Chronic obstructive lung disease	25	43.4	0.6	0.4	0.9	0.004
Pneumonia	29	26.8	1.1	0.7	1.6	0.715
All digestive diseases	41	43.9	0.9	0.7	1.3	0.735
Genitourinary diseases	19	16	1.2	0.7	1.9	0.518
Motor vehicle accidents	24	23.2	1	0.7	1.5	0.931
Suicide	18	20.2	0.9	0.5	1.4	0.736
Other external causes	28	38.4	0.7	0.5	1.1	0.101
All cancers	272	283.8	1	0.8	1.1	0.505
All causes	1104	1077.5	1	1	1.1	0.427

Adjusted for age and calendar year at risk

TABLE 3.4

STANDARDISED MORTALITY RATIOS (SMR) AND 95% CONFIDENCE INTERVAL COMPARED TO CANADIAN MORTALITY RATES (1950-1999) FOR SELECTED CAUSES

MALES: PORT RADIUM

Cause	Observed	Expected	SMR	Lower Limit	Upper Limit	p-value
All infectious diseases		13	0.8	0.4	1.4	0.563
Stomach cancer	21	27.1	0.8	0.5	1.2	0.277
Colon cancer	27	42.3	0.6	0.4	0.9	0.016
Rectal cancer	14	14.4	1	0.5	1.6	0.999
Pancreatic cancer	24	22.6	1.1	0.7	1.6	0.829
Lung cancer	230	142.7	1.6	1.4	1.8	<0.001
Prostate cancer	35	44.2	0.8	0.6	1.1	0.184
Non-Hodgkins lymphoma	14	13.3	1	0.6	1.8	0.928
Diabetes mellitus	16	31.5	0.5	0.3	0.8	0.004
All nervous system diseases	17	29.8	0.6	0.3	0.9	0.016
Hypertensive disease	14	8.5	1.7	0.9	2.8	0.101
Ischemic heart disease	395	508.2	0.8	0.7	0.9	<0.001
Stroke	81	111	0.7	0.6	0.9	0.003
All other cardiovascular disease	102	126.5	0.8	0.7	1	0.029
Chronic obstructive lung disease	33	75.1	0.4	0.3	0.6	<0.001
Pneumonia	48	49.3	1	0.7	1.3	0.924
All digestive diseases	45	69.3	0.6	0.5	0.9	0.002
Alcoholism and related	23	9.1	2.5	1.6	3.8	<0.001
Genitourinary diseases	20	26.3	0.8	0.5	1.2	0.25
Motor vehicle accidents	46	32.4	1.4	1	1.9	0.028
Suicide	48	25.7	1.9	1.4	2.5	<0.001
Other external causes	110	58.1	1.9	1.6	2.3	<0.001
All cancers	459	442.4	1	0.9	1.1	0.443
All causes	1616	1700.5	1	0.9	1	0.04

Adjusted for age and calendar year at risk

TABLE 3.5

STANDARDISED MORTALITY RATIOS (SMR) AND 95% CONFIDENCE INTERVAL COMPARED TO CANADIAN MORTALITY RATES (1950-1999) FOR SELECTED CAUSES

MALES: BEAVERLODGE

Cause	Observed	Expected	SMR	Lower Limit	Upper Limit	p-value
All infectious diseases	30	27.8	1.1	0.7	1.5	0.726
Buccal cavity cancer	12	17.9	0.7	0.3	1.2	0.194
Esophageal cancer	14	17.4	0.8	0.4	1.4	0.501
Stomach cancer	39	33.5	1.2	0.8	1.6	0.382
Colon cancer	31	60.2	0.5	0.3	0.7	<0.001
Rectal cancer	14	19.3	0.7	0.4	1.2	0.27
Pancreatic cancer	29	32.5	0.9	0.6	1.3	0.615
Lung cancer	279	217.8	1.3	1.1	1.4	<0.001
Prostate cancer	38	49.4	0.8	0.5	1.1	0.112
Kidney cancer	16	16.6	1	0.6	1.6	0.999
Bladder and other urinary cancer	11	16.1	0.7	0.3	1.2	0.243
Brain and other cns cancer	19	20.9	0.9	0.5	1.4	0.78
Non-Hodgkins lymphoma	19	22.3	0.9	0.5	1.3	0.562
Multiple myeloma	12	10	1.2	0.6	2.1	0.611
All leukemia	22	22.6	1	0.6	1.5	0.999
Diabetes mellitus	32	43.3	0.7	0.5	1	0.092
All mental disorders	16	30.1	0.5	0.3	0.9	0.007
All nervous system diseases	26	41.1	0.6	0.4	0.9	0.016
Hypertensive disease	14	10.2	1.4	0.7	2.3	0.308
Ischemic heart disease	460	625.1	0.7	0.7	0.8	<0.001
Stroke	86	119.3	0.7	0.6	0.9	0.002
All other cardiovascular disease	121	145.8	0.8	0.7	1	0.04
Chronic obstructive lung disease	20	85	0.2	0.1	0.4	<0.001
Pneumonia	57	50.6	1.1	0.9	1.5	0.402
All digestive diseases	88	101.2	0.9	0.7	1.1	0.204
Alcoholism and related	27	18.1	1.5	1	2.2	0.06
Genitourinary diseases	11	28.4	0.4	0.2	0.7	<0.001
Motor vehicle accidents	105	76.2	1.4	1.1	1.7	0.002
Suicide	139	69.8	2	1.7	2.4	<0.001
Homicide	20	8.7	2.3	1.4	3.5	0.001
Other external causes	219	109.2	2	1.7	2.3	<0.001
All cancers	635	646.1	1	0.9	1.1	0.681
All causes	2289	2311.3	1	1	1	0.653

Adjusted for age and calendar year at risk

TABLE 3.6

STANDARDISED MORTALITY RATIOS (SMR) AND 95% CONFIDENCE INTERVAL COMPARED TO CANADIAN MORTALITY RATES (1950-1999) FOR SELECTED CAUSES

MALES: OTHER SITES

Cause	Observed	Expected	SMR	Lower Limit	Upper Limit	p-value
Ischemic heart disease	35	50.5	0.7	0.5	1	0.027
All cancers	40	53.4	0.7	0.5	1	0.069
All causes	139	194.9	0.7	0.6	0.8	<0.001

Adjusted for age and calendar year at risk

TABLE 3.7

STANDARDISED MORTALITY RATIOS (SMR) AND 95% CONFIDENCE INTERVAL COMPARED TO CANADIAN MORTALITY RATES (1950-1999) FOR SELECTED CAUSES

MALES: PORT RADIUM UNDERGROUND WORKERS

Cause	Observed	Expected	SMR	Lower Limit	Upper Limit	p-value
Stomach cancer	14	13.8	1	0.6	1.7	0.999
Colon cancer	15	22.1	0.7	0.4	1.1	0.146
Pancreatic cancer	13	11.8	1.1	0.6	1.9	0.81
Lung cancer	159	75.5	2.1	1.8	2.5	<0.001
Prostate cancer	17	23.3	0.7	0.4	1.2	0.223
Diabetes mellitus	10	16.5	0.6	0.3	1.1	0.123
All nervous system diseases	10	16.1	0.6	0.3	1.1	0.148
Ischemic heart disease	183	263.5	0.7	0.6	0.8	<0.001
Stroke	36	56.2	0.6	0.4	0.9	0.005
All other cardiovascular disease	57	65.2	0.9	0.7	1.1	0.343
Chronic obstructive lung disease	20	40.1	0.5	0.3	0.8	0.001
Pneumonia	24	25.8	0.9	0.6	1.4	0.818
All digestive diseases	19	36.4	0.5	0.3	0.8	0.002
Alcoholism and related	13	4.8	2.7	1.4	4.6	0.003
Genitourinary diseases	12	13.6	0.9	0.5	1.5	0.795
Motor vehicle accidents	24	16.7	1.4	0.9	2.1	0.11
Suicide	27	13.4	2	1.3	2.9	0.001
Other external causes	74	30.6	2.4	1.9	3	<0.001
All cancers	286	232.2	1.2	1.1	1.4	0.001
All causes	896	887	1	0.9	1.1	0.773

Adjusted for age and calendar year at risk

TABLE 3.8

STANDARDISED MORTALITY RATIOS (SMR) AND 95% CONFIDENCE INTERVAL COMPARED TO CANADIAN MORTALITY RATES (1950-1999) FOR SELECTED CAUSES

MALES: BEAVERLODGE UNDERGROUND WORKERS

Cause	Observed	Expected	SMR	Lower Limit	Upper Limit	p-value
All infectious diseases	11	15.2	0.7	0.4	1.3	0.344
Stomach cancer	21	17.9	1.2	0.7	1.8	0.516
Colon cancer	17	32.8	0.5	0.3	0.8	0.004
Pancreatic cancer	20	17.8	1.1	0.7	1.7	0.66
Lung cancer	198	120.7	1.6	1.4	1.9	<0.001
Prostate cancer	16	25.3	0.6	0.4	1	0.065
Kidney cancer	14	9.2	1.5	0.8	2.6	0.169
Brain and other cns cancer	10	11.9	0.8	0.4	1.5	0.718
Non-Hodgkins lymphoma	11	12.4	0.9	0.4	1.6	0.825
All leukemia	14	12.4	1.1	0.6	1.9	0.712
Diabetes mellitus	22	23.3	0.9	0.6	1.4	0.89
All mental disorders	11	16.2	0.7	0.3	1.2	0.231
All nervous system diseases	11	22	0.5	0.2	0.9	0.015
Ischemic heart disease	271	332.6	0.8	0.7	0.9	0.001
Stroke	40	61	0.7	0.5	0.9	0.006
All other cardiovascular disease	61	76.2	0.8	0.6	1	0.086
Chronic obstructive lung disease	11	43.8	0.3	0.1	0.4	<0.001
Pneumonia	26	25.5	1	0.7	1.5	0.976
All digestive diseases	49	55.8	0.9	0.6	1.2	0.399
Alcoholism and related	15	10.4	1.4	0.8	2.4	0.213
Motor vehicle accidents	63	42.4	1.5	1.1	1.9	0.004
Suicide	84	39.6	2.1	1.7	2.6	<0.001
Homicide	14	5	2.8	1.5	4.7	0.001
Other external causes	148	61.1	2.4	2	2.8	<0.001
All cancers	395	354.1	1.1	1	1.2	0.034
All causes	1348	1244	1.1	1	1.1	0.004

Adjusted for age and calendar year at risk

TABLE 3.9

STANDARDISED MORTALITY RATIOS (SMR) AND 95% CONFIDENCE INTERVAL COMPARED TO CANADIAN MORTALITY RATES (1950-1999) FOR SELECTED CAUSES

MALES: BEAVERLODGE MILL WORKERS

Cause	Observed	Expected	SMR	Lower Limit	Upper Limit	p-value
Lung cancer	24	28.1	0.9	0.5	1.3	0.507
Ischemic heart disease	47	75.4	0.6	0.5	0.8	0.001
Stroke	12	14.1	0.9	0.4	1.5	0.704
Motor vehicle accidents	13	12.8	1	0.5	1.7	0.999
Suicide	22	11.3	1.9	1.2	3	0.006
Other external causes	25	16.4	1.5	1	2.2	0.059
All cancers	68	83.5	0.8	0.6	1	0.094
All causes	247	297.6	0.8	0.7	0.9	0.003

Adjusted for age and calendar year at risk

TABLE 3.10

STANDARDISED MORTALITY RATIOS (SMR) AND 95% CONFIDENCE INTERVAL COMPARED TO CANADIAN MORTALITY RATES (1950-1999) FOR SELECTED CAUSES

MALES: PORT RADIUM – FIRST EMPLOYED AFTER 1 APRIL 1956

Cause	Observed	Expected	SMR	Lower Limit	Upper Limit	p-value
Lung cancer	22	20.2	1.1	0.7	1.7	0.742
Ischemic heart disease	49	58.7	0.8	0.6	1.1	0.227
Suicide	15	5	3	1.7	4.9	<0.001
Other external causes	11	9.1	1.2	0.6	2.2	0.623
All cancers	39	59.5	0.7	0.5	0.9	0.006
All causes	181	209.5	0.9	0.7	1	0.049

Adjusted for age and calendar year at risk

TABLE 3.11

STANDARDISED MORTALITY RATIOS (SMR) AND 95% CONFIDENCE INTERVAL COMPARED TO CANADIAN MORTALITY RATES (1950-1999) FOR SELECTED CAUSES

MALES: BEAVERLODGE – FIRST EMPLOYED AFTER 1 JANUARY 1956

Cause	Observed	Expected	SMR	Lower Limit	Upper Limit	p-value
All infectious diseases	19	18.4	1	0.6	1.6	0.958
Stomach cancer	26	17.5	1.5	1	2.2	0.07
Colon cancer	15	33.5	0.4	0.3	0.7	0.001
Rectal cancer	10	10.5	1	0.5	1.8	0.999
Pancreatic cancer	18	18.2	1	0.6	1.6	0.999
Lung cancer	158	124	1.3	1.1	1.5	0.004
Prostate cancer	18	22.8	0.8	0.5	1.2	0.369
Brain and other cns cancer	10	13.5	0.7	0.4	1.4	0.423
Non-Hodgkins lymphoma	14	13.7	1	0.6	1.7	0.999
All leukemia	14	13.2	1.1	0.6	1.8	0.9
Diabetes mellitus	19	23.7	0.8	0.5	1.3	0.397
All mental disorders	13	16.6	0.8	0.4	1.3	0.459
All nervous system diseases	14	22.5	0.6	0.3	1	0.078
Ischemic heart disease	249	321.4	0.8	0.7	0.9	<0.001
Stroke	50	56.7	0.9	0.7	1.2	0.411
All other cardiovascular disease	65	72.2	0.9	0.7	1.1	0.435
Chronic obstructive lung disease	10	39.5	0.3	0.1	0.5	<0.001
Pneumonia	25	22.8	1.1	0.7	1.6	0.693
All digestive diseases	50	57.7	0.9	0.6	1.1	0.343
Alcoholism and related	16	12	1.3	0.8	2.2	0.306
Motor vehicle accidents	72	55.4	1.3	1	1.6	0.036
Suicide	106	53	2	1.6	2.4	<0.001
Homicide	17	6.9	2.5	1.4	4	0.002
Other external causes	156	72.4	2.2	1.8	2.5	<0.001
All cancers	368	364.5	1	0.9	1.1	0.87
All causes	1375	1275.5	1.1	1	1.1	0.006

Adjusted for age and calendar year at risk

TABLE 3.12

STANDARDISED MORTALITY RATIOS (SMR) AND 95% CONFIDENCE INTERVAL COMPARED TO CANADIAN MORTALITY RATES (1950-1999) FOR SELECTED CAUSES

MALES: BEAVERLODGE – FIRST EMPLOYED AFTER 1 JANUARY 1970

Cause	Observed	Expected	SMR	Lower Limit	Upper Limit	p-value
Lung cancer	29	15.9	1.8	1.2	2.6	0.004
Ischemic heart disease	38	36.2	1	0.7	1.4	0.809
All other cardiovascular disease	15	8.7	1.7	1	2.9	0.064
All digestive diseases	12	8.5	1.4	0.7	2.5	0.3
Motor vehicle accidents	34	17	2	1.4	2.8	<0.001
Suicide	48	19.8	2.4	1.8	3.2	<0.001
Homicide	10	2.6	3.9	1.9	7.2	0.001
Other external causes	50	19.4	2.6	1.9	3.4	<0.001
All cancers	64	51.1	1.3	1	1.6	0.09
All causes	325	204.9	1.6	1.4	1.8	<0.001

Adjusted for age and calendar year at risk

TABLE 3.13

STANDARDISED INCIDENCE RATIOS (SIR) FOR VARIOUS CANCERS AND 95% CONFIDENCE INTERVAL COMPARED TO CANADIAN INCIDENCE RATES (1969-1999)

MALES

Cause	Observed	Expected	SIR	Lower Limit	Upper Limit	p-value
Buccal cavity cancer	52	112.8	0.5	0.3	0.6	<0.001
Esophageal cancer	28	34.8	0.8	0.5	1.2	0.285
Liver cancer	15	22.5	0.7	0.4	1.1	0.128
Stomach cancer	71	94.9	0.7	0.6	0.9	0.013
Colon cancer	131	230.3	0.6	0.5	0.7	<0.001
Rectal cancer	103	137.8	0.7	0.6	0.9	0.002
Pancreatic cancer	61	70	0.9	0.7	1.1	0.307
Laryngeal cancer	36	54.1	0.7	0.5	0.9	0.012
Lung cancer	688	555.8	1.2	1.1	1.3	<0.001
Malignant melanoma	46	56.2	0.8	0.6	1.1	0.19
Breast cancer	46	58.1	0.8	0.6	1.1	0.121
Prostate cancer	354	501.9	0.7	0.6	0.8	<0.001
Kidney cancer	36	78.5	0.5	0.3	0.6	<0.001
Bladder and other urinary cancer	100	161.2	0.6	0.5	0.8	<0.001
Brain and other CNS cancer	36	46.9	0.8	0.5	1.1	0.12
Non-Hodgkins Lymphoma	85	96	0.9	0.7	1.1	0.283
Multiple myeloma	24	32.5	0.7	0.5	1.1	0.152
Leukemia	55	70.5	0.8	0.6	1	0.066
All cancers	2210	2651.2	0.8	0.8	0.9	<0.001

Adjusted for age (five-year intervals) and calendar year at risk (five-year intervals)

TABLE 3.14

STANDARDISED INCIDENCE RATIOS (SIR) FOR VARIOUS CANCERS AND 95% CONFIDENCE INTERVAL COMPARED TO CANADIAN INCIDENCE RATES (1969-1999)

FEMALES

Cause	Observed	Expected	SIR	Lower Limit	Upper Limit	p-value
Lung cancer	28	18.7	1.5	1	2.2	0.054
Breast cancer	44	53	0.8	0.6	1.1	0.239
All cancers	145	171.3	0.8	0.7	1	0.044

Adjusted for age (five-year intervals) and calendar year at risk (five-year intervals)

TABLE 3.15

STANDARDISED INCIDENCE RATIOS (SIR) FOR VARIOUS CANCERS AND 95% CONFIDENCE INTERVAL COMPARED TO CANADIAN INCIDENCE RATES (1969-1999)

MALES: PORT HOPE

Cause	Observed	Expected	SIR	Lower Limit	Upper Limit	p-value
Stomach cancer	13	17.5	0.7	0.4	1.3	0.344
Colon cancer	34	39.9	0.9	0.6	1.2	0.392
Rectal cancer	23	24	1	0.6	1.4	0.938
Pancreatic cancer	10	12.4	0.8	0.4	1.5	0.61
Laryngeal cancer	11	9.6	1.1	0.6	2.1	0.731
Lung cancer	110	100.3	1.1	0.9	1.3	0.359
Malignant melanoma	11	8.3	1.3	0.7	2.4	0.421
Prostate cancer	91	94.7	1	0.8	1.2	0.753
Bladder and other urinary cancer	27	29.7	0.9	0.6	1.3	0.703
Non-Hodgkins Lymphoma	15	15.4	1	0.5	1.6	0.999
Leukemia	10	12.2	0.8	0.4	1.5	0.66
All cancers	426	455.3	0.9	0.8	1	0.174

Adjusted for age (five-year intervals) and calendar year at risk (five-year intervals)

TABLE 3.16

STANDARDISED INCIDENCE RATIOS (SIR) FOR VARIOUS CANCERS AND 95% CONFIDENCE INTERVAL COMPARED TO CANADIAN INCIDENCE RATES (1969-1999)

MALES: PORT RADIUM

Cause	Observed	Expected	SIR	Lower Limit	Upper Limit	p-value
Buccal cavity cancer	13	30	0.4	0.2	0.7	0.001
Stomach cancer	19	27.8	0.7	0.4	1.1	0.105
Colon cancer	37	63.5	0.6	0.4	0.8	<0.001
Rectal cancer	23	37.4	0.6	0.4	0.9	0.016
Pancreatic cancer	21	19.7	1.1	0.7	1.6	0.832
Laryngeal cancer	13	14.7	0.9	0.5	1.5	0.794
Lung cancer	204	155.8	1.3	1.1	1.5	<0.001
Prostate cancer	112	154.2	0.7	0.6	0.9	<0.001
Bladder and other urinary cancer	28	47.2	0.6	0.4	0.9	0.004
Non-Hodgkins Lymphoma	21	22.5	0.9	0.6	1.4	0.864
All cancers	596	710.1	0.8	0.8	0.9	<0.001

Adjusted for age (five-year intervals) and calendar year at risk (five-year intervals)

TABLE 3.17

STANDARDISED INCIDENCE RATIOS (SIR) FOR VARIOUS CANCERS AND 95% CONFIDENCE INTERVAL COMPARED TO CANADIAN INCIDENCE RATES (1969-1999)

MALES: BEAVERLODGE

Cause	Observed	Expected	SIR	Lower Limit	Upper Limit	p-value
Buccal cavity cancer	31	55.6	0.6	0.4	0.8	<0.001
Esophageal cancer	14	16.5	0.8	0.5	1.4	0.643
Stomach cancer	38	42.8	0.9	0.6	1.2	0.517
Colon cancer	50	103	0.5	0.4	0.6	<0.001
Rectal cancer	44	64.5	0.7	0.5	0.9	0.009
Pancreatic cancer	26	31.4	0.8	0.5	1.2	0.381
Lung cancer	337	259.7	1.3	1.2	1.4	<0.001
Malignant melanoma	24	29.5	0.8	0.5	1.2	0.357
Prostate cancer	135	233.7	0.6	0.5	0.7	<0.001
Kidney cancer	23	38.9	0.6	0.4	0.9	0.009
Bladder and other urinary cancer	37	74.4	0.5	0.4	0.7	<0.001
Brain and other CNS cancer	18	24	0.7	0.4	1.2	0.254
Non-Hodgkins Lymphoma	39	47.9	0.8	0.6	1.1	0.22
Multiple myeloma	11	14.8	0.7	0.4	1.3	0.403
Leukemia	34	33	1	0.7	1.4	0.909
All cancers	973	1212.1	0.8	0.8	0.9	<0.001

Adjusted for age (five-year intervals) and calendar year at risk (five-year intervals)

TABLE 3.18

STANDARDISED INCIDENCE RATIOS (SIR) FOR VARIOUS CANCERS AND 95% CONFIDENCE INTERVAL COMPARED TO CANADIAN INCIDENCE RATES (1969-1999)

MALES: OTHER SITES

Cause	Observed	Expected	SIR	Lower Limit	Upper Limit	p-value
Prostate cancer	16	19.4	0.8	0.5	1.3	0.53
All cancers	70	102.4	0.7	0.5	0.9	0.001

Adjusted for age (five-year intervals) and calendar year at risk (five-year intervals)

TABLE 3.19

STANDARDISED INCIDENCE RATIOS (SIR) FOR VARIOUS CANCERS AND 95% CONFIDENCE INTERVAL COMPARED TO CANADIAN INCIDENCE RATES (1969-1999)

MALES: PORT RADIUM: UNDERGROUND WORKERS

Cause	Observed	Expected	SIR	Lower Limit	Upper Limit	p-value
Buccal cavity cancer	11	15.8	0.7	0.3	1.2	0.274
Stomach cancer	13	14.7	0.9	0.5	1.5	0.792
Colon cancer	20	33.7	0.6	0.4	0.9	0.016
Rectal cancer	10	19.8	0.5	0.2	0.9	0.025
Pancreatic cancer	13	10.5	1.2	0.7	2.1	0.507
Lung cancer	138	83	1.7	1.4	2	<0.001
Prostate cancer	51	81.8	0.6	0.5	0.8	<0.001
Bladder and other urinary cancer	16	25.1	0.6	0.4	1	0.073
Non-Hodgkins Lymphoma	11	11.8	0.9	0.5	1.7	0.967
All cancers	344	376.3	0.9	0.8	1	0.098

Adjusted for age (five-year intervals) and calendar year at risk (five-year intervals)

TABLE 3.20

STANDARDISED INCIDENCE RATIOS (SIR) FOR VARIOUS CANCERS AND 95% CONFIDENCE INTERVAL COMPARED TO CANADIAN INCIDENCE RATES (1969-1999)

MALES: BEAVERLODGE: UNDERGROUND WORKERS

Cause	Observed	Expected	SIR	Lower Limit	Upper Limit	p-value
Buccal cavity cancer	19	31.6	0.6	0.4	0.9	0.022
Stomach cancer	19	23.6	0.8	0.5	1.3	0.401
Colon cancer	31	57.2	0.5	0.4	0.8	<0.001
Rectal cancer	27	36.2	0.7	0.5	1.1	0.137
Pancreatic cancer	19	17.5	1.1	0.7	1.7	0.778
Lung cancer	230	145.8	1.6	1.4	1.8	<0.001
Malignant melanoma	13	17	0.8	0.4	1.3	0.406
Prostate cancer	69	128.6	0.5	0.4	0.7	<0.001
Kidney cancer	17	22.1	0.8	0.4	1.2	0.326
Bladder and other urinary cancer	14	41.2	0.3	0.2	0.6	<0.001
Brain and other CNS cancer	11	13.7	0.8	0.4	1.4	0.563
Non-Hodgkins Lymphoma	22	27.2	0.8	0.5	1.2	0.371
Leukemia	20	18.4	1.1	0.7	1.7	0.763
All cancers	581	677.8	0.9	0.8	0.9	<0.001

Adjusted for age (five-year intervals) and calendar year at risk (five-year intervals)

TABLE 3.21

STANDARDISED INCIDENCE RATIOS (SIR) FOR VARIOUS CANCERS AND 95% CONFIDENCE INTERVAL COMPARED TO CANADIAN INCIDENCE RATES (1969-1999)

MALES: BEAVERLODGE: MILL WORKERS

Cause	Observed	Expected	SIR	Lower Limit	Upper Limit	p-value
Lung cancer	34	34.4	1	0.7	1.4	0.999
Prostate Cancer	16	30.5	0.5	0.3	0.9	0.006
All cancers	125	164.2	0.8	0.6	0.9	0.002

Adjusted for age (five-year intervals) and calendar year at risk (five-year intervals)

TABLE 3.22

STANDARDISED INCIDENCE RATIOS (SIR) FOR VARIOUS CANCERS AND 95% CONFIDENCE INTERVAL COMPARED TO CANADIAN INCIDENCE RATES (1969-1999)

MALES: PORT RADIUM: FIRST EMPLOYED AFTER 1 APRIL 1956

Cause	Observed	Expected	SIR	Lower limit	Upper limit	p-value
Lung cancer	24	23.9	1	0.6	1.5	0.999
Prostate cancer	12	21.9	0.5	0.3	1	0.031
All cancers	67	109.7	0.6	0.5	0.8	<0.001

Adjusted for age (five-year intervals) and calendar year at risk (five-year intervals)

TABLE 3.23

STANDARDISED INCIDENCE RATIOS (SIR) FOR VARIOUS CANCERS AND 95% CONFIDENCE INTERVAL COMPARED TO CANADIAN INCIDENCE RATES (1969-1999)

MALES: BEAVERLODGE: FIRST EMPLOYED AFTER 1 JANUARY 1956

Cause	Observed	Expected	SIR	Lower Limit	Upper Limit	p-value
Buccal cavity cancer	23	35.5	0.6	0.4	1	0.035
Stomach cancer	22	24.9	0.9	0.6	1.3	0.652
Colon cancer	33	60.9	0.5	0.4	0.8	<0.001
Rectal cancer	31	39.4	0.8	0.5	1.1	0.201
Pancreatic cancer	18	18.5	1	0.6	1.5	0.999
Lung cancer	204	153.8	1.3	1.2	1.5	<0.001
Malignant melanoma	15	21.7	0.7	0.4	1.1	0.172
Prostate cancer	80	130.8	0.6	0.5	0.8	<0.001
Kidney cancer	17	25.3	0.7	0.4	1.1	0.109
Bladder and other urinary cancer	23	43.3	0.5	0.3	0.8	0.001
Brain and other CNS cancer	12	16.8	0.7	0.4	1.2	0.291
Non-Hodgkins Lymphoma	30	32.7	0.9	0.6	1.3	0.723
Leukemia	21	20.5	1	0.6	1.6	0.966
All cancers	621	736.6	0.8	0.8	0.9	<0.001

Adjusted for age (five-year intervals) and calendar year at risk (five-year intervals)

TABLE 3.24

STANDARDISED INCIDENCE RATIOS (SIR) FOR VARIOUS CANCERS AND 95% CONFIDENCE INTERVAL COMPARED TO CANADIAN INCIDENCE RATES (1969-1999)

MALES: BEAVERLODGE: FIRST EMPLOYED AFTER 1 JANUARY 1970

Cause	Observed	Expected	SIR	Lower Limit	Upper Limit	p-value
Lung cancer	43	21.2	2	1.5	2.7	<0.001
Prostate cancer	12	14.9	0.8	0.4	1.4	0.548
All cancers	127	124.2	1	0.9	1.2	0.823

Adjusted for age (five-year intervals) and calendar year at risk (five-year intervals)

TABLE 4.1

LUNG CANCER DEATHS AND PERSON-YEARS BY CUMULATIVE EXPOSURE (WLM) FOR SUBGROUPS OF ELDORADO COHORT (1950-1999)

		Exposure (WLM)							
		0.0	**0.001-**	**3.58-**	**14.83-**	**53.12-**	**164.51-**	**614.38-**	**Total**
Port Hope, Males	Cases	17	33	31	11	7	-	-	101
	Person-years	30596	28829	12625	6575	3699	-	-	83397
Port Radium, Males	Cases	46	7	12	23	28	38	76	230
	Person-years	37187	9391	10782	12709	13986	18207	8960	111222
Beaverlodge, Males	Cases	41	43	43	53	47	44	8	279
	Person-years	82183	90740	46856	35771	20948	8673	793	285964
Other Sites, Males	Cases		-	-	-	-	-	-	8
	Person-years		-	-	-	-	-	-	28160
Females, all facilities	Cases		-	-	-	-	-	-	21
	Person-years		-	-	-	-	-	-	44865
Total	Cases	123	88	89	87	83	84	85	639
	Person-years	209470	136722	73807	56985	38855	27952	9817	553608

- indicates cells with potentially < 5 cases

TABLE 4.2

EXCESS RELATIVE RISK ESTIMATES FOR RADON DECAY PRODUCTS (PER 100 WLM) FOR VARIOUS SUBCOHORTS OF ELDORADO COHORT (1950-1999)*

	Cases N	Estimate	95% Lower Limit	95% Upper Limit	$X^2(1)$	p-value
Males**	618	0.546	0.373	0.782	130.6	<0.0001
Port Hope, Males	101	0.18	-0.102	1.492	0.29	0.59
Port Radium, Males	230	0.371	0.226	0.591	78.22	<0.0001
Beaverlodge, Males	279	0.963	0.56	1.563	55.42	<0.0001

* All models adjusted for sub-cohort, age at risk (five-year intervals), calendar year at risk (five-year intervals) and duration of employment by < than six months vs > than six months stratification.
** Including lung cancer cases occurring in the 'Other Sites' sub-cohort.

TABLE 4.3

RELATIVE RISKS BY EXPOSURE FOR VARIOUS SUB-COHORTS*

(MALES ONLY)

Entire Cohort

Exposure (WLM)	Cases	%	Person Years	%	Relative Risk	Lower Bound	Upper Bound	X^2	DOF	p- value
0.00-	110	17.8	175,143	34.4	1.00			98.05	1	<0.0001
0.0001-	83	13.4	130,688	25.7	0.85	0.62	1.17			
3.58-	86	13.9	70,919	13.9	1.30	0.96	1.75			
14.83-	87	14.1	55,349	10.9	1.51	1.12	2.03			
53.12-	83	13.4	38,776	7.6	1.79	1.31	2.43			
164.51-	84	13.6	27,976	5.5	2.50	1.82	3.42			
614.38-	85	13.8	9,822	1.9	7.34	5.13	10.53			
Total	618	100.0	508,673	100.0						

Port Hope

Exposure (WLM)	Cases	%	Person Years	%	Relative Risk	Lower Bound	Upper Bound	X^2	DOF	p-value
0.00-	17	16.8	30,565	36.7	1.00			2.75	1	0.0973
0.0001-	33	32.7	28,811	34.6	0.76	0.4	1.51			
3.58-	31	30.7	12,638	15.2	1.61	0.84	3.23			
14.83-	11	10.9	6,571	7.9	1.30	0.55	3.02			
53.12-	9	8.9	4,776	5.7	1.35	0.49	3.54			
Total	101	100.0	83,362	100.0						

Table 4.3 (cont.)

Port Radium

Exposure (WLM)	Cases	%	Person Years	%	Relative Risk	Lower Bound	Upper Bound	X^2	DOF	p-value
0.00-	46	20.0	37,158	33.4	1.00			42.38	1	<0.0001
0.0001-	7	3.0	9,414	8.5	0.72	0.29	1.56			
3.58-	12	5.2	10,802	9.7	0.95	0.47	1.76			
14.83-	23	10.0	12,758	11.5	1.43	0.84	2.37			
53.12-	28	12.2	13,992	12.6	1.38	0.85	2.22			
164.51-	38	16.5	18,221	16.4	1.43	0.91	2.24			
614.38-	76	33.0	8,951	8.0	5.35	3.54	8.2			
Total	230	100.0	111,297	100.0						

Beaverlodge

Exposure (WLM)	Cases	%	Person Years	%	Relative Risk	Lower Bound	Upper Bound	X^2	DOF	p-value
0.00-	41	14.7	82,135	28.7	1.00			52.44	1	<0.0001
0.0001-	43	15.4	90,674	31.7	1.13	0.73	1.77			
3.58-	43	15.4	46,853	16.4	1.44	0.93	2.24			
14.83-	53	19.0	35,739	12.5	1.82	1.19	2.79			
53.12-	47	16.8	20,957	7.3	2.36	1.5	3.72			
164.51-	44	15.8	8,681	3.0	4.62	2.91	7.37			
614.38-	8	2.9	807	0.3	9.19	3.91	19.17			
Total	279	100.0	285,846	100.0						

* Adjusted for cohort, age, years and days worked

TABLE 4.4

DEVIANCES AND X^2 TESTS FOR VARIOUS INTERACTION MODELS (1950-1999)

Model	Terms	Deviance	X^2	DOF	p-value
1	WLM	3248.18			
2	WLM 5, WLM 15, WLM 25	3233.8	14.37	2	0.0008
3	WLM 5, WLM 15, WLM 25, rate (2), rate (3), rate (4), rate (5), rate (6)	3213.63	20.17	5	0.0012
4	WLM5,WLM 15,WLM 25, rate (2), rate (3), rate (4), rate (5), rate (6), age (2), age (3), age (4)	3206.62	7.01	3	0.0715

WLM = Total WLM (per 100 WLM) lagged by 5 years
WLM 5 = WLM 5-14 Years previously (per 100 WLM)
WLM 15 = WLM 15-24 Years previously (per100 WLM)
WLM 25 = WLM 25 Years+ previously (per100 WLM)

Rate (2) = WL 0.5-1.0
Rate (3) = WL 1.0-3.0
Rate (4) = WL 3.0-5.0
Rate (5) = WL 5.0-15.0
Rate (6) = WL 15+

Age (2) = age at risk 55-64
Age (3) = age at risk 65-74
Age (4) = age at risk 75+

TABLE 4.5

PARAMETER ESTIMATES FOR FULL INTERACTION MODEL AND COMPARISON WITH BEIR VI MODEL ESTIMATES FOR MALES IN THE ELDORADO COHORT (1950-1999)

Parameter	Estimate	95% Lower Limit	95% Upper Limit	Estimate for BEIR VI
WLM 5	5.23	1.33	14.52	7.68
WLM 15	2.5	0.63	7.05	5.99
WLM 25	1.37	0.36	3.99	3.92
rate(1)	1			1
rate(2)	1.02	0.39	2.67	0.49
rate(3)	0.49	0.2	1.21	0.37
rate(4)	0.35	0.12	1.01	0.32
rate(5)	0.33	0.13	0.84	0.17
rate(6)	0.16	0.06	0.44	0.11
age(1)	1			1
age(2)	1.94	0.77	4.89	0.57
age(3)	1	0.37	2.72	0.29
age(4)	0.05	0	6266.67	0.09

Parameters as specified in footnotes to Table 4.4

TABLE 5.1

NUMBER OF LUNG CANCER DEATHS AND LUNG CANCER CASES IN ELDORADO STUDY COHORT

Total number of deaths and/or cases	1950–1999	789
Total number of deaths	1950–1999	639
Total number of cases	1969–1999	658
Number of deaths	1950–1968	83
Number of deaths	1969–1999	556
Number of deaths without incidence record	1969–1999	48
Number of cases without death record	1969–1999	150

TABLE 5.2

LUNG CANCER CASES AND PERSON-YEARS BY CUMULATIVE EXPOSURE (WLM) FOR SUBGROUPS OF ELDORADO COHORT (1969-1999)

		Exposure (WLM)							
		0.0	**0.001-**	**3.58-**	**14.83-**	**53.12-**	**164.51-**	**614.38-**	**Total**
Port Hope, Males	Cases	19	37	30	15	7	2	-	110
	Person-years	26308	17258	6843	3314	1507	411	-	55641
Port Radium, Males	Cases	39	9	13	18	30	40	47	196
	Person-years	16663	6713	7609	8587	9083	11190	4539	64383
Beaverlodge, Males	Cases	47	56	47	62	55	37	7	311
	Person-years	51974	83789	40370	28918	16429	6666	523	228669
Other Sites, Males	Cases	-	-	-	-	-	-	-	9
	Person-years	-	-	-	-	-	-	-	24451
Females, all facilities	Cases	-	-	-	-	-	-	-	28
	Person-years	-	-	-	-	-	-	-	35333
Total	Cases	130	108	93	95	93	79	56	654
	Person-years	144581	114259	57151	41961	27183	18267	5074	408477

- indicates cells with potentially < 5 cases

TABLE 5.3

EXCESS RELATIVE RISK ESTIMATES FOR RADON DECAY PRODUCTS (PER 100 WLM) FOR VARIOUS SUBCOHORTS OF ELDORADO COHORT (1969-1999)

	Cases N	ERR Estimate	95% Lower Limit	95% Upper Limit	$X^2(1)$	p-value
Males	626	0.55	0.37	0.81	102.7	<0.001
Port Hope, Males	110	0.68	-0.23	3.07	1.85	0.173
Port Radium, Males	196	0.40	0.23	0.68	58.15	<0.001
Beaverlodge, Males	311	0.70	0.38	1.17	37.89	<0.001

All models adjusted for sub-cohort, age at risk, calendar year at risk and duration of employment by stratification

TABLE 5.4

**RELATIVE RISKS BY EXPOSURE FOR VARIOUS SUB-COHORTS*
LUNG CANCER INCIDENCE (1969-1999)**

(MALES ONLY)

Entire cohort

Exposure (WLM)	Cases	%	Person Years	%	Relative Risk	Lower Bound	Upper Bound	X^2	DOF	p-value
0.00-	111	17.7	116,822	31.3	1.0			83.82	1	<0.0001
0.0001-	102	16.3	109,317	29.3	0.9	0.66	1.20			
3.58-	90	14.4	55,306	14.8	1.2	0.91	1.65			
14.83-	95	15.2	41,015	11.0	1.6	1.15	2.07			
53.12-	93	14.9	27,102	7.3	2.0	1.47	2.68			
164.51-	79	12.6	18,255	4.9	2.5	1.82	3.48			
614.38-	56	8.9	5,070	1.4	7.2	4.84	10.68			
Total	626	100.0	372,888	100.0						

Port Hope

Exposure (WLM)	Cases	%	Person Years	%	Relative Risk	Lower Bound	Upper Bound	X^2	DOF	p-value
0.00-	19	17.3	26,290	47.3	1.0			7.37	1	0.0066
0.0001-	37	33.6	17,246	31.0	0.9	0.45	1.64			
3.58-	30	27.3	6,838	12.3	1.7	0.91	3.44			
14.83-	15	13.6	3,311	6.0	2.2	0.99	4.70			
53.12-	9	8.2	1,917	3.4	1.9	0.71	4.96			
Total	110	100.0	55,603	100.0						

Table 5.4 (cont.)

Port Radium

Exposure (WLM)	Cases	%	Person Years	%	Relative Risk	Lower Bound	Upper Bound	X^2	DOF	p-value
0.00-	39	19.9	16,652	25.9	1.0			32	1	<0.0001
0.0001-	9	4.6	6,708	10.4	0.8	0.36	1.66			
3.58-	13	6.6	7,604	11.8	1.0	0.50	1.82			
14.83-	18	9.2	8,581	13.3	1.1	0.61	1.92			
53.12-	30	15.3	9,076	14.1	1.5	0.93	2.47			
164.51-	40	20.4	11,182	17.4	1.8	1.10	2.81			
614.38-	47	24.0	4,536	7.1	5.5	3.36	8.92			
Total	196	100.0	64,339	100.0						

Beaverlodge

Exposure (WLM)	Cases	%	Person Years	%	Relative Risk	Lower Bound	Upper Bound	X^2	DOF	p-value
0.00-	47	15.1	51,938	22.7	1.0			38.95	1	<0.0001
0.0001-	56	18.0	83,732	36.6	1.1	0.72	1.60			
3.58-	47	15.1	40,343	17.7	1.2	0.80	1.82			
14.83-	62	19.9	28,898	12.6	1.7	1.15	2.53			
53.12-	55	17.7	16,418	7.2	2.3	1.50	3.47			
164.51-	37	11.9	6,662	2.9	3.3	2.05	5.17			
614.38-	7	2.3	523	0.2	7.1	2.89	15.16			
Total	311	100.0	228,513	100.0						

* Adjusted for cohort, age, years and days worked

TABLE 5.5

DEVIANCES AND X^2 TESTS FOR VARIOUS INTERACTION MODELS (1969-1999)

Model	Terms	Deviance	X^2	DOF	p-value
1	WLM	3106.19			
2	WLM 5, WLM 15, WLM 25	3088.90	17.28	2	0.0002
3	WLM 5, WLM 15, WLM 25, rate (2), rate (3), rate (4), rate (5), rate (6)	3080.44	8.46	5	0.133
4	WLM5,WLM 15,WLM 25, rate (2), rate (3), rate (4), rate (5), rate (6), age (2), age (3), age (4)	3076.59	3.85	3	0.279

WLM = Total WLM (per 100 WLM) lagged by 5 years
WLM 5 = WLM 5-14 Years previously (per 100 WLM)
WLM 15 = WLM 15-24 Years previously (per100 WLM)
WLM 25 = WLM 25 Years+ previously (per100 WLM)

Rate (2) = WL 0.5-1.0
Rate (3) = WL 1.0-3.0
Rate (4) = WL 3.0-5.0
Rate (5) = WL 5.0-15.0
Rate (6) = WL 15+

Age (2) = age at risk 55-64
Age (3) = age at risk 65-74
Age (4) = age at risk 75+

TABLE 5.6

PARAMETER ESTIMATES FOR FULL INTERACTION MODEL AND COMPARISON WITH BEIR VI MODEL ESTIMATES FOR MALES IN THE ELDORADO COHORT (1969-1999)

Parameter	Estimate	95% Lower Limit	95% Upper Limit	Estimate for BEIR VI
WLM 5	6.53	1.76	18.84	7.68
WLM 15	2.45	0.72	6.71	5.99
WLM 25	0.93	0.23	3.07	3.92
rate(1)	1			1
rate(2)	0.68	0.28	1.69	0.49
rate(3)	0.41	0.18	0.90	0.37
rate(4)	0.28	0.09	0.85	0.32
rate(5)	0.36	0.14	0.91	0.17
rate(6)	0.23	0.09	0.59	0.11
age(1)	1			1
age(2)	2.51	0.92	6.86	0.57
age(3)	1.48	0.51	4.27	0.29
age(4)	0.77	0.03	18.13	0.09

Parameters as specified in footnotes to Table 4.4

TABLE 5.7

DISTRIBUTION OF HISTOLOGIC SUBTYPES OF LUNG CANCER FOR SUB-COHORTS OF ELDORADO COHORT

Histologic subtype	Port Hope	Port Radium	Beaverlodge
Squamous cell	28 (25.45%)	64 (32.65%)	111 (35.69%)
Small cell	9 (8.18%)	35 (17.86%)	56 (18.01%)
Adenocarcinoma	23 (20.91%)	38 (19.39%)	49 (15.76%)
Other types	50 (45.45%)	59 (30.10%)	95 (30.55%)

TABLE 5.8

EXCESS RELATIVE RISK ESTIMATES FOR RADON DECAY PRODUCTS (PER 100 WLM), BY HISTOLOGIC SUBTYPES OF LUNG CANCER FOR VARIOUS SUBCOHORTS OF THE ELDORADO COHORT (1950-1999)

Histologic subtype	Cases					
	N	Estimate	95% Lower Limit	95% Upper Limit	*$X^2(1)$	p-value
Squamous cell	206	0.80	0.43	1.42	57.15	<0.0001
Small cell	102	0.66	0.23	1.52	18.26	<0.0001
Adenocarcinoma	110	0.33	0.08	0.84	9.93	0.002
Other	208	0.51	0.22	1.03	27.82	<0.0001

All models adjusted for cohort, age at risk, calendar year at risk and duration of employment by stratification

*Tests statistical significance of adding a dose variable to the model

TABLE 6.1

RESULTS OF FITTING SIMPLE EXCESS RR MODEL TO WLM EXPOSURE FOR VARIOUS CAUSES OF DEATH (MORTALITY 1950-1999)

MALES ONLY

Cause	Number of Deaths	ERR	X^2_1	p-value
All infectious diseases (including TB)	53	-0.04	1.05	0.31
Stomach cancer*	75	-0.04	2	0.16
Colon cancer	82	0	0	0.99
Pancreatic cancer	67	-0.01	0.04	0.85
Prostate cancer	98	-0.03	0.42	0.52
Leukemia	34	0.02	0.06	0.81
Other cancer	113	0.06	0.44	0.51
Diabetes mellitus	64	0	0	0.98
Endocrine cancer	61	-0.04	1.12	0.29
Ischaemic heart disease	618	-0.02	2.24	0.13
Stroke	244	-0.04	6.23	0.01
Other (all) cardiovascular diseases	317	-0.02	0.49	0.49
Pneumonia	134	-0.01	0.26	0.61
Respiratory	158	0.02	0.3	0.58
Cirrhosis of liver	60	0.03	0.09	0.77
Digestive	179	-0.03	0.97	0.33
Alcoholism*	54	-0.04	1.68	0.19
Motor vehicle accidents	180	0.03	0.24	0.62
Suicide	208	0.05	0.9	0.34
Accidents	365	0.18	8.75	<0.0001
Other causes	113	0.07	1.18	0.28

Adjusted for sub-cohort, age and calendar year at risk, and days worked for Eldorado by stratification

*=Estimate may not be maximum likelihood estimate

TABLE 6.2

RESULTS OF FITTING SIMPLE EXCESS RR MODEL TO GAMMA RAY DOSE FOR VARIOUS CAUSES OF DEATH (MORTALITY 1950-1999)

MALES ONLY

Cause	Number of Deaths	ERR	X^2_1	p-value
All infectious diseases (including TB) *	53	-0.29	0.52	0.47
Stomach cancer	75	0.28	0.08	0.78
Colon cancer	82	0.82	0.31	0.57
Pancreatic cancer*	67	-0.29	0.6	0.44
Prostate cancer	98	0.19	0.06	0.8
Leukemia	34	-0.29	0.05	0.82
Other cancers	113	-0.07	0.01	0.94
Diabetes mellitus	64	0.29	0.03	0.86
Endocrine cancer	61	0.26	0.07	0.79
Ischaemic heart disease	618	0.22	1.1	0.29
Stroke	244	-0.29	1.58	0.21
Other (all) cardiovascular diseases	317	0.07	0.08	0.78
Pneumonia	134	0.68	1.33	0.25
Respiratory	158	0.12	0.03	0.86
Cirrhosis of liver	60	-0.1	0.02	0.89
Digestive	179	-0.29	0.57	0.45
Alcoholism	54	0.68	0.32	0.57
Motor vehicle accidents	180	0.81	1.18	0.28
Suicide*	208	1.48	1	0.32
Accidents	365	2.74	4.79	0.03
Other causes	113	0.43	0.36	0.55

Adjusted for sub-cohort, age and calendar year at risk, and days worked for Eldorado by stratification

*=Estimate may not be maximum likelihood estimate

TABLE 6.3

RESULTS OF FITTING SIMPLE EXCESS RR MODEL TO WLM EXPOSURE FOR VARIOUS CANCERS (INCIDENCE 1969-1999)

MALES ONLY

Cancer site	Number of Cases	ERR	X^2_1	p-value
Buccal cavity	50	-0.04	0.17	0.68
Stomach	69	-0.04	1.31	0.25
Colon	118	-0.04	0.71	0.4
Rectum	95	0.03	0.39	0.53
Pancreas	59	-0.03	0.11	0.74
Prostate	350	-0.01	0.09	0.77
Bladder	89	-0.04	0.36	0.55
Non-Hodgkins lymphoma	78	0.04	0.17	0.68
Other	139	0.09	1.23	0.27
Chronic lymphatic leukemia	22	-0.04	0.31	0.58
Leukemia, excluding CLL*	31	-0.04	0.48	0.49

Adjusted for sub-cohort, age and calendar year at risk, and days worked for Eldorado by stratification

*=Estimate may not be maximum likelihood estimate

TABLE 6.4

RESULTS OF FITTING SIMPLE EXCESS RR MODEL TO GAMMA RAY DOSE FOR VARIOUS CANCERS (INCIDENCE 1969-1999)

MALES ONLY

Cancer site	Number of Cases	ERR	X^2_1	p-value
Buccal cavity *	50	-0.34	0.25	0.62
Stomach	69	-0.34	0.58	0.45
Colon	118	0.31	0.3	0.59
Rectum	95	-0.34	0.13	0.72
Pancreas*	59	-0.34	0.43	0.51
Prostate	350	-0.34	1.57	0.21
Bladder	89	2.83	2.11	0.15
Non-Hodgkins lymphoma	78	-0.34	0.15	0.7
Other	139	0.13	0.02	0.88
Chronic lymphatic leukemia	22	7.28	0.78	0.38
Leukemia, excluding CLL	31	-0.34	0.12	0.73

Adjusted for sub-cohort, age and calendar year at risk, and days worked for Eldorado by stratification

*=Estimate may not be maximum likelihood estimate

FIGURES 4.1 AND 5.1

Figure 4.1. Plot of the Relative Risk Estimates For Lung Cancer Mortality and the Corresponding 95% Confidence Limits from the Categorical Analysis and a Fitted Least Squares Dose-Response Line

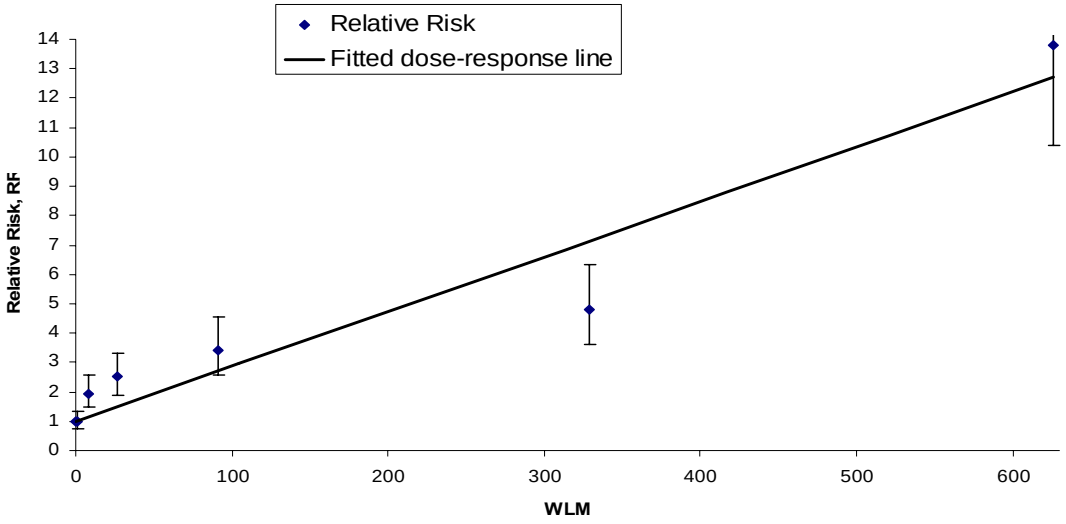

Figure 5.1. Plot of the Relative Risk Estimates For Lung Cancer Incidence and the Corresponding 95% Confidence Limits from the Categorical Analysis and a Fitted Least Squares Dose-Response Line