Lung cancer incidence attributable to residential radon exposure in Alberta in 2012

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Abstract

Background: Radon is carcinogenic, and exposure to radon has been shown to increase the risk of lung cancer. The objective of this study was to quantify the proportion and number of lung cancer cases in Alberta in 2012 that could be attributed to residential radon exposure.

Methods: We estimated the population attributable risk of lung cancer for residential radon using radon exposure data from the Cross-Canada Survey of Radon Concentrations in Homes from 2009–2011 and data on all-cause and lung cancer mortality from Statistics Canada from 2008–2012. We used cancer incidence data from the Alberta Cancer Registry for 2012 to estimate the total number of lung cancers attributable to residential radon exposure. Estimates were also stratified by sex and smoking status.

Results: The mean geometric residential radon level in Alberta in 2011 was 71.0 Bq/m³ (geometric standard deviation 2.14). Overall, an estimated 16.6% (95% confidence interval 9.4%–29.8%) of lung cancers were attributable to radon exposure, corresponding to 324 excess attributable cancer cases. The estimated population attributable risk of lung cancer due to radon exposure was higher among those who had never smoked (24.8%) than among ever smokers (15.6%). However, since only about 10% of cases of lung cancer occur in nonsmokers, the estimated total number of excess cases was higher for ever smokers (274) than for never smokers (48).

Interpretation: With about 17% of lung cancer cases in Alberta in 2012 attributable to residential radon exposure, exposure reduction has the potential to substantially reduce Alberta’s lung cancer burden. As such, home radon testing and remediation techniques represent important cancer prevention strategies.
Ontario. The objectives of this study were to estimate the population attributable risk of lung cancer due to residential radon exposure in Alberta and to estimate the number of cases of lung cancer that could be attributed to this exposure in 2012.

Methods

This manuscript is part of a series of exposure-specific manuscripts concerning the proportion of cancer cases attributable to modifiable lifestyle and environmental risk factors in the general population of Alberta. The methodologic framework for this series has previously been described.

Data sources

We estimated the excess risk ratio using data on radon exposure in Alberta and an estimate of the proportion of Albertans living in apartments in 2011. People living in apartments above the second floor typically have negligible exposure to residential radon, and thus the proportion of the Alberta population residing in apartment buildings was of interest and was estimated from Statistics Canada 2011 census data at 20%. These people were assumed to be unexposed (zero radon exposure). We estimated radon exposure levels using data from the Cross-Canada Survey of Radon Concentrations in Homes (CCSRCH), conducted by Health Canada over the fall/winter of 2009–2010 and 2010–2011. Eligible participants were heads of households over 18 years of age who were homeowners, did not live on First Nations reserves or on military bases, and did not plan to move or be away during the proposed time of the study (October to March) and whose homes were not high-rise condominiums or built on stilts. Participating households were asked to place an α-track radon detector on the lowest lived-in level in their home for at least 3 months. The overall study response rate was roughly 21%. Among the 1131 tests completed in Alberta, data were missing for 55 (4.9%), and these 55 tests were excluded, leaving 1076 tests from which mean radon exposure levels for Alberta were estimated. We estimated mean radon exposure levels for the province as a whole and for the 5 Alberta Health Services zones individually (www.albertahealthservices.ca/abs-map-abs-zones.pdf).

We estimated the prevalence of ever smoking in Alberta using data from 2000–2001, 2003, 2005 and 2007–2008 Canadian Community Health Survey. The mean prevalence value across these survey years was used in each age–sex group (15–19 yr, 20–24 yr, 25–34 yr, 35–54 yr and ≥ 55 yr) in the analysis to enhance the stability of the estimates, similar to the method employed by Peterson and colleagues. Ever smokers were people who reported being current or former daily or occasional smokers, and never smokers were those who reported that they had never smoked. Data on all-cause mortality for 2008–2012 were obtained from Statistics Canada, and data on lung cancer mortality for the same period were obtained from the Alberta Cancer Registry.

Population attributable risk estimation

We estimated the population attributable risk of lung cancer in Alberta due to radon exposure using the method of Brand and colleagues, which was previously used for a similar analysis in Ontario. This method is based on the exposure–age–concentration risk model described in the 1999 BEIR VI report on the health effects of radon exposure. Briefly, excess rate ratios were estimated with the use of data on radon exposure and other modifying factors, and were used to estimate the population attributable risk percent of lung cancer mortality attributable to radon exposure. These estimates of excess rate ratios were then combined with data on all-cause and lung cancer mortality by means of life-table methods to estimate the lifetime risk of lung cancer mortality among radon-exposed and radon-unexposed people. The lifetime risk estimates were then used to estimate the excess lifetime risk ratio for lung cancer associated with radon exposure, which was used to estimate the population attributable risk.

Similar to the approach used by Parkin and Darby, we considered the population attributable risk associated with lung cancer mortality to approximate the population attributable risk associated with lung cancer incidence. This assumption is based on the idea that the risk of death from lung cancer is the same for cases that were and were not caused by radon exposure, an assumption supported by a recent study among uranium miners, such that the population attributable risk based on data on lung cancer mortality approximates the measure that would be obtained with data on lung cancer incidence.

Specifically, we estimated excess rate ratios of lung cancer mortality using the equations described by Brand and colleagues. We used data on radon exposure and the proportion of Albertans living in apartments as inputs for these estimates. We employed estimates of lifetime risk of lung cancer mortality among those exposed and unexposed to estimate the proportion of cancer cases attributable to this exposure in 2012. We then performed abridged life-table calculations to estimate the lifetime risk of lung cancer mortality, performed for ever smokers, never smokers, and ever and never smokers combined. We conducted these calculations using age- and sex-specific rates of all-cause and lung cancer mortality, along with age- and sex-specific data on the prevalence of ever smoking. We derived estimates for men and women combined using age-specific data only. We then combined lifetime calculations with the previously estimated values of excess rate ratios to produce estimates of the lifetime risk of lung cancer mortality among those exposed.

We employed estimates of lifetime risk of lung cancer mortality among those exposed and unexposed to estimate the excess lifetime risk ratio using equation 1:

$$ELRR = (LRE - LR)/LR$$
where ELRR = excess lifetime risk ratio, LRE = lifetime risk of lung cancer mortality among exposed people and LR = lifetime risk of lung cancer among those exposed and unexposed.

We then used the expected value of excess lifetime risk ratio (taken across the distribution of radon exposure) to estimate the population attributable risk of lung cancer due to radon exposure employing equation 2:

\[ \text{Equation 2: population attributable risk} = \frac{(LRE - LR)}{LRE} = 1 - \frac{1}{[ELRR + 1]} \]

**Statistical analysis**

To estimate the number of cases of lung cancer in Alberta in 2012 that could be attributed to radon exposure, we applied the population attributable risk estimates to data on lung cancer incidence. Our uncertainty analysis provided a histogram of population attributable risk estimates, with the spread representing uncertainty and hyperparameter uncertainty in the estimates of excess relative risk. We singled out the arithmetic mean for our estimates of attributable cases, but percentiles for population attributable risk can be applied to obtain estimates of attributable cases that reflect the upper/lower percentiles of output uncertainty. We derived estimates of the number of lung cancer cases attributable to radon exposure in ever smokers and never smokers separately by assuming that 10% of lung cancers are diagnosed in never smokers. All analyses were conducted in RStudio (version 0.98.1080, RStudio, Inc.).

**Ethics approval**

Ethics approval was obtained from the Conjoint Health Research Ethics Board, University of Calgary.

**Results**

Measured radon levels for Alberta for 2011 from the CCSRCH followed a log-normal distribution, with a geometric mean of 71.0 Bq/m³ and a geometric standard deviation of 2.14. The geometric mean of radon varied across Alberta Health Services zones, with the lowest concentrations seen in the Calgary Zone (64.9 Bq/m³) and the highest concentrations in the Central Zone (78.5 Bq/m³) (Table 1).

Estimates of the range of population attributable percent values for Alberta as a whole for 2012 are shown in Table 2. For men and women combined, a mean population attributable risk of 16.6% (95% confidence interval 9.4%–29.8%) was observed when not accounting for smoking status, which corresponded to 324 excess cases of lung cancer attributable to radon exposure (Table 2). When smoking status was
considered, the mean estimated population attributable risk was higher among never smokers (24.8% [95% CI 13.3%–43.4%]) than among ever smokers (15.6% [95% CI 9.2%–25.8%]). This pattern of a higher population attributable risk among never smokers was also observed among men and women: overall population attributable risk estimates were marginally higher among men than among women.

Table 3 presents the range of population attributable risk estimates for each Alberta Health Services zone. Despite some variation in the geometric mean radon concentration across zones, the estimated mean population attributable risks due to radon exposure were consistent. Owing to differences in population size between the zones, higher absolute numbers of excess attributable cases of lung cancer were estimated for the Calgary (91 excess cases) and Edmonton (112) zones, whereas lower numbers were estimated for the South (30), Central (46) and North (40) zones (Table 3). The population attributable risk estimates for men and women separately were similar to the overall estimates within each zone (data not shown).

### Interpretation

In this study, we estimated that 16.6% of lung cancer cases in Alberta in 2012 could be attributed to radon exposure. This corresponded to an estimated 324 excess cases of lung cancer, 85% of which were among ever smokers. Furthermore, consistent with previous analyses, no meaningful differences in population attributable risks between men and women were observed. Although we found small differences in mean radon level across the 5 Alberta Health Services zones, the estimated zone-specific population attributable risks were similar.

The estimated population attributable risk for Alberta is somewhat higher than that for Ontario (13.6%). Although the Ontario analysis was based on the same radon exposure survey as that used in the current analysis and employed the same analytic methods, arithmetic mean radon levels were lower in Ontario than in Alberta (43 Bq/m$^3$ v. 71 Bq/m$^3$). The observed difference in population attributable risk estimates
can be explained by the difference in exposure level between the 2 provinces.

The population attributable risk for lung cancer due to radon exposure for Canada as a whole was estimated in 3 previous studies.6–8 Brand and colleagues6 used national survey data from 1978 to characterize radon exposure. They employed the same exposure–age–concentration model recommended by BEIR VI that we used and estimated a population attributable risk of 7.8%. The mean radon level in their study was 11.2 Bq/m², substantially lower than the values observed for Alberta for 2011. Chen7 performed an analysis using the same 1970s survey data employed by Brand and colleagues6 but using the Environmental Protection Agency method stratified between ever (9.4% men, 8.8% women) and never (19% men, 18% women) smokers. She found that the estimated population attributable risks were a bit higher than those observed by Brand and colleagues6 (ever smokers 7.3%, never smokers 13.5%). More recently, Chen and colleagues8 applied the Environmental Protection Agency method to data from the CCSRCH and observed a geometric mean indoor radon level of 41.9 Bq/m³, which translated to an estimated population attributable risk of about 16%, virtually the same as our estimate. Outside Canada, Gray and colleagues19 estimated that with a mean indoor radon concentration of 21 Bq/m³, the proportion of lung cancer deaths attributable to radon exposure in the United Kingdom in 2006 was 3.3%. Parkin and Darby20 subsequently applied this proportion to 2010 data on incident lung cancer cases in the UK and estimated that 3.4% of incident lung cancer cases in 2010 were attributable to radon exposure. Finally, in a study from Germany, Steindorf and colleagues21 observed a geometric mean indoor radon level of 40 Bq/m³ and estimated a population attributable risk of 7%. Across all these previous studies, although the model used to estimate population attributable risk had some impact on the estimates observed, the primary reason for differences across studies was the difference in mean radon concentration.

The CCSRCH data were obtained with the use of long-term α track radon detectors, which are thought to provide more accurate measures of radon exposure than the grab sampling technique used in the earlier survey with data from the 1970s.8 However, although the mean radon level for Canada (41.9 Bq/m³) calculated with more recent data4 is closer to that observed for Alberta in the current study (71.0 Bq/m³), overall radon levels in Alberta were still higher than comparable values at the national level. Differences in the model used to estimate population attributable risk by Chen and colleagues8 and in our study likely explain why the estimates of population attributable risk are similar despite higher levels of radon exposure in Alberta.

Limitations

The CCSRCH collects data only from homeowners because there is no requirement for landlords to remediate high radon levels. This may have created a possible selection bias.22 If radon exposure is different in homes inhabited by tenants than in owner-inhabited homes (for example, if the absence of radon remediation requirements leads to higher radon levels in rented homes), the mean radon levels reported by the CCSRCH would be an underestimate. Given that no data on radon levels in homes inhabited by renters are available, we were unable to estimate whether this bias existed in our study. In a recent study in which radon levels were measured in over 2300 Calgary-area homes, Stanley and colleagues23 observed a mean radon level of 126 Bq/m³, which is higher than that observed by the CCSRCH for Alberta as a whole. This further suggests that our observed radon exposure levels may be underestimates. However, a portion of this difference may be attributable to the different geographic areas covered by the 2 surveys. The fact that the overall response rate in the CCSRCH was only 21% created a second potential source of selection bias if radon levels in the homes of nonparticipants were systematically higher or lower than those in the homes of participants. Furthermore, radon measurements in the CCSRCH were obtained during the winter, when levels are usually highest. The lack of adjustment for seasonality in our analysis likely led to overestimates in measured radon levels (and subsequently the estimated population attributable risks) that we cannot quantify. Our analysis assumed that radon exposure is constant over the lifetime and thus did not account for movement of people to different houses. As has been previously described,9 if residential mobility were accounted for, although estimates of the mean burden of lung cancer would not be affected, there would be less associated variability, with fewer estimates at the high and low ends of risk (an argument drawing from the central limit theorem). Finally, we estimated population attributable risk using data for lung cancer mortality and applied the risk values to incidence data to estimate the number of lung cancer cases attributable to radon exposure. If the assumption that lung cancer mortality is similar for both radon- and non–radon-induced cases is inaccurate, the number of radon-attributable cases of incident lung cancer would have been over- or underestimated, depending on whether people whose cancer was radon-induced are more or less likely to die than people with cancer not related to radon exposure.

We observed broad uncertainty intervals in our results, which is reflective of the uncertainty specified for all the inputs. This uncertainty implied an effective geometric standard deviation of about 1.3, a value that was upheld (roughly) for each of the smoking/sex permutations in Table 2. (By defining the applicable percentiles as Xα,1 and Xα,2, the effective geometric standard deviation can be calculated from a ratio of the 2 percentiles: (Xα,1/Xα,2)1/[2*1.96].) Such consistency across smoking strata was previously observed by Brand and colleagues6 and is likely to be upheld in analyses using the exposure–age–concentration model more generally.

Conclusion

We estimate that 16.6% of lung cancer cases in Alberta in 2012 were attributable to residential radon exposure. The geometric mean radon level, 71.0 Bq/m³, was higher than the value observed for Canada as a whole in a previous study (41.9 Bq/m³).4 As such, radon remediation strategies to reduce residential radon exposure in Alberta are needed and present a
non–smoking-related target that could affect the incidence of lung cancer in the province.

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Contributors: Christine Friedenreich and Darren Brenner were responsible for the study conception. Anne Grundy, Christine Friedenreich, Darren Brenner, Farah Khandwala and Abbey Poirier contributed substantially to the study design and interpretation of the data. Sierra Tamminen and Anne Grundy were responsible for data acquisition, and Farah Khandwala analyzed the data. Kevin Brand provided the R code and guidance on implementing the BEIR VI projections. All of the authors prepared the manuscript, approved the final version to be published and agreed to act as guarantors of the work.

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