Residential Radon and Risk of Lung Cancer

A Combined Analysis of 7 North American Case-Control Studies

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Background: Underground miners exposed to high levels of radon have an excess risk of lung cancer. Residential exposure to radon is at much lower levels, and the risk of lung cancer with residential exposure is less clear. We conducted a systematic analysis of pooled data from all North American residential radon studies.

Methods: The pooling project included original data from 7 North American case–control studies, all of which used long-term α-track detectors to assess residential radon concentrations. A total of 3662 cases and 4966 controls were retained for the analysis. We used conditional likelihood regression to estimate the excess risk of lung cancer.

Results: Odds ratios (ORs) for lung cancer increased with residential radon concentration. The estimated OR after exposure to radon at a concentration of 100 Bq/m^3 in the exposure time window 5 to 30 years before the index date was 1.11 (95% confidence interval = 1.00–1.28). This estimate is compatible with the estimate of 1.12 (1.02–1.25) predicted by downward extrapolation of the miner data. There was no evidence of heterogeneity of radon effects across studies. There was no apparent heterogeneity in the association by sex, educational level, type of respondent (proxy or self), or cigarette smoking, although there was some evidence of a decreasing radon-associated lung cancer risk with age. Analyses restricted to subsets of the data with presumed more accurate radon dosimetry resulted in increased estimates of risk.

Conclusions: These results provide direct evidence of an association between residential radon and lung cancer risk, a finding predicted using miner data and consistent with results from animal and in vitro studies.

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Radon-222 is a decay product of radium-226 and ultimately of uranium-238 (2 elements that are ubiquitous in soils and rocks, thereby providing a continual source of radon). Radon can accumulate in enclosed areas such as underground mines and houses. When inhaled into the lung, alpha particles emitted by short-lived decay products of radon can damage cellular DNA. Cellular mutagenesis studies, experimental research in animals, and occupational epidemiologic studies have established radon as a human lung carcinogen.[1,2]

A combined analysis of lung cancer mortality among 11 cohorts of underground miners confirmed that high levels of exposure to radon are associated with increased lung cancer risk.[3] A subsequent evaluation of updated miner data conducted by the National Research Council[2] estimated that 10% to 15% of the 157,400 lung cancer deaths occurring annually in the United States may be attributed to residential radon, with an uncertainty range of 3300 to 32,000 deaths.[4]

Results of miner studies unambiguously demonstrate an excess risk of lung cancer resulting from occupational exposure to radon. However, differences in breathing characteristics of miners and residents at home are substantial, as are other differences between the mine and home environments.[2] Although low-exposed miners experienced exposures comparable to long-term residence in high radon houses, the mean cumulative exposure among miners is approximately 30-fold higher than that associated with long-term residency in a typical home.[2]

A combined analysis of data from residential radon case–control studies would help to resolve ambiguity in the evidence of increased lung cancer risk at residential radon exposure levels.[5] To date, 20 case–control studies of residential radon and lung cancer have been completed, including 7 studies in North America, 11 in Europe, and 2 in China (Table 1). Some of these studies reported a positive or weakly positive association between lung cancer risk and residential radon concentrations, whereas others have reported results consistent with no association. To date, no case–control study has reported a statistically significant negative association.

Based on results reported by the original investigators, the excess odds ratios for lung cancer at a radon concentration of 100 Bq/m^3 ranged from −0.05 in Shenyang, China, to 0.56 in New Jersey (Table 1). Although excess odds ratios for all but 2 studies (West Germany and Shenyang, China) were positive, confidence limits included the null value of zero in all but 4 studies (Iowa, the Swedish national study, the Czech

TABLE 1. Case-Control Studies of Residential Radon and Lung Cancer

Region	No. of Cases	No. of Controls	Average Radon Concentration (Bq/m^3)*	Excess Odds Ratio† (95% CI)
North America‡				
New Jersey (NJ)[9]	480	442	26	0.56 (−0.22–2.97)
Winnipeg (Winn)[10]	738	738	120	0.02 (−0.05–0.25)
Missouri-I (MO-I)[12]	538	1183	63	0.01 (<0.00–0.42)
Missouri-II (MO-II)[13]	512	553	56	0.27 (−0.12–1.53)
Iowa (IA)[14]	413	614	127	0.44 (0.05–1.59)
Connecticut (CT)[16]	963	949	33	0.02 (−0.21–0.51)
Utah–South Idaho (UT-ID)[16]	511	862	57	0.03 (−0.20–0.55)
Europe§				
Sweden (Stockholm)[40]	201	378	128	0.16 (−0.14–0.92)
Sweden (national)[41]	1281	2576	107	0.10 (0.01–0.22)
South Finland[42]	291	495	213	0.28 (−0.21–0.78)¶
Finland (national)[43]	517	517	96	0.11 (−0.06–0.31)
Southwest England[31]	982	3185	56	0.08 (−0.03–0.20)
Italy[44]	384	404	96	0.14 (−0.11–0.46)
East Germany[45]	1192	1640	74	0.08 (−0.03–0.20)
West Germany[46]	1449	2297	50	−0.02 (−0.18–0.17)
Sweden (nonsmokers)[47]	258	487	79	0.28 (−0.05–1.05)
France[48]	688	1428	128	0.05 (−0.01–0.12)
Czech Republic[49]	210	12,004	509	0.09 (0.02–0.21)
China				
Shenyang[50]	308	356	85	−0.05 (<0.00–0.08)
Gansu[51]	768	1659	223	0.19 (0.05–0.47)

*Values given are mean residential radon concentrations, except for the study from Italy (geometric mean) and the study from Shenyang, China (median).
†Excess odds ratio at 100 Bq/m^3.
‡Excess odds ratio values and radon levels have been recalculated from original data submitted for combined analysis based on α-track dosimetry only.
§Except for South Finland, Italy, France, and Czech Republic, results have been previously summarized by Kreuzer et al.(2003).[44]
¶Recalculated based on reported odds ratios.

Republic, and Gansu, China). These results reflect a range of lung cancer risks, including the possibility of no risk, suggesting the need for an overall assessment of the findings from different studies.

Metaanalyses of published odds ratios from North American and other residential radon case–control studies found a statistically significant increase in lung cancer risk.[6,7] However, the odds ratios in 13 studies included in the more recent metaanalysis exhibited heterogeneity among studies, possibly as a result of the inability to directly adjust for other confounding factors.

To better characterize results for these seemingly disparate studies, and to obtain direct estimates of potential lung cancer risks associated with radon in homes, we conducted a combined analysis of the original data from all North American case–control studies of residential radon and lung cancer, including studies in New Jersey,[8,9] Winnipeg,[10,11] Missouri nonsmoking women (denoted Missouri-I),[12] Missouri women (Missouri-II),[13] Iowa,[14,15] and Connecticut and Utah–South Idaho.[16] These studies included a total of 3662 cases and 4966 controls. The specific objectives of this combined analysis are to test the null hypothesis that residential radon does not increase risk of lung cancer, to evaluate the consistency of effects among the different studies, to evaluate variations in the exposure–response relationship with other lung cancer risk factors, and to compare risk estimates from the pooled residential data with extrapolations from miner-based risk models.

METHODS

Subject Selection

An overview of the studies included in the present combined analysis is provided in the appendix (available with the electronic version of this article). In all studies, cases were ascertained through state and provincial cancer registries and were histologically or cytologically confirmed. Controls were population-based, matched to cases on the basis of age ($\pm$ 5 years) and sex (Iowa, Missouri-I, Missouri-II, and New Jersey included only females). Smoking status was used as a matching variable in Connecticut, Utah–South Idaho, and Missouri-II. Frequency matching or randomized recruitment[17] was used for control selection, except in New Jersey and Winnipeg, where pair matching was used.

Radon Dosimetry

All studies used long-term α-track detectors to measure the concentration of radon progeny in indoor air for 12 months. Although the primary radon measurements in Missouri-II were made with a new technology that monitors alpha particles embedded in glass surfaces, we did not use these glass-based measurements in the present analysis to maintain comparable dosimetry among studies. In New Jersey, a small number of measurements (9%) made using short-term (3–7 days) charcoal canisters were also excluded from this analysis. Contemporaneous measurements were made in homes that subjects had occupied or were currently occupying; these measurements were used to estimate historical radon concentrations in those homes. Detectors were placed in the living area and bedroom areas of the home in which subjects had spent the majority of their time. The mean radon concentrations measured by α-track dosimeters in the living area were highest in Winnipeg (131 Bq/m^3) and Iowa (127 Bq/m^3) and lowest in New Jersey (25 Bq/m^3). Becquerels per cubic meter (Bq/m^3) is the SI measure of activity, with 1 Bq equaling 1 disintegration per second. Pico-curies per liter (pCi/L) is an historical unit still commonly used, with 1 pCi/L = 37 Bq/m^3.

In most studies, an attempt was made to monitor homes occupied for at least 1 year within the exposure time window considered to be most directly related to lung cancer risk. In New Jersey, only the last residence occupied for at least 10 years during the exposure time window 10 to 30 years before recruitment was monitored. The Iowa study also measured only one home, but enrollment required occupancy for 20 years or more in the current home. Based on the extensive analysis of cohort studies conducted by the National Research Council,[2] the present analysis is focused on the exposure time window 5 to 30 years before the index date. The proportion of time within this exposure time window covered by radon measurements ranged from 75.2% in Winnipeg to 92.4% in Iowa.

Statistical Analysis

Data were aggregated using a common format. Information included age at index date (date of diagnosis for cases and date of interview or recruitment for controls), year of ascertainment, source of information (subject or proxy interview), sex, smoking-related variables, education, family income, ethnicity, and historical profiles of radon concentrations in houses based on detector measurements or on the original investigators' best estimates. All analyses were conducted using conditional likelihood regression[18] with a linear model for the odds ratio (OR) of the form $OR(x) = 1 + \beta x$, where x is the mean residential radon concentration in the exposure time window[2] in Bq/m^3 and β is the excess odds ratio for each unit increase in x. This model was fit with the PECAN module in the Epicure software package using the conditional analytic method of parameter estimation.[19] Results are based on the best estimates of radon concentrations, including both measured and imputed radon values supplied by the collaborating investigators. (There was virtually no difference in the estimated excess odds ratios when the imputed values provided by the collaborating investigators were replaced with imputed values corresponding to the study-specific control means, as recommended by Weinberg et al.[20]) We stratified baseline risk by sex, age at index date

(<60, 60–64, 65–69, 70–74, 75+ years), number of cigarettes smoked per day (never-smoker, 1–9, 10–19, 20–29, 30+), duration of cigarette smoking (never, 1–24, 25–34, 35–44, 45+ years), number of residences occupied (<3, 3+), years with α-track monitoring within the exposure time window (<20, 20+ years), and an indicator variable for each study to control for confounding. We also included an offset parameter to control for the randomized recruitment design in the Connecticut, Utah–South Idaho, and Missouri-II studies.

Although our main analyses are based on the full dataset comprising 3662 cases and 4966 controls, we also analyzed restricted datasets. Restrictions focused on subjects for which measured, rather than imputed, radon concentrations were used for dosimetry[21] and on subjects who occupied only 1 or 2 residences. The latter restriction potentially offsets both the reduction in the range of exposures conferred by population mobility[5,22,23] and exposure measurement error associated with the monitoring of former residences. Data restrictions were imposed under the assumption that the restrictions resulted in more accurate radon dosimetry. The restricted datasets involved fewer subjects; for example, there were 1910 cases and 2651 controls in the subgroup of subjects for which measured rather than imputed radon concentrations were available for at least 20 years within the exposure time window of interest and who had occupied, at most, 2 residences.

RESULTS

Characteristics of Study Subjects

The majority of the study subjects (86%) had some secondary school or higher education. Among cases, 38% were diagnosed with adenocarcinoma, 22% with squamous cell carcinoma, and 16% with small/oat cell carcinoma. Because 4 studies (Iowa, Missouri-I, Missouri-II and New Jersey) enrolled only women (who typically spent more time in the home than men), the database includes more women (2556 cases and 3596 controls) than men (1106 cases and 1370 controls). Smoking status varied among the study participants; although some studies were restricted to nonsmoking cases, the majority of cases were smokers.

Risk Estimates

Table 2 shows estimated odds ratios for lung cancer by categories of mean radon concentration and the excess odds ratio at 100 Bq/m^3, along with 95% confidence intervals (CIs). The excess odds ratio for individual studies ranged from 0.01 (<0.00–0.42) in Missouri-I to 0.56 (−0.22–2.97) per 100 Bq/m^3 in New Jersey but did not demonstrate heterogeneity ($P = 0.59$). The overall results are also presented in Figure 1, where the abscissa of each category-specific odds ratio is the mean radon concentration within its category. Odds ratios exhibit no apparent evidence of nonlinearity throughout the range of radon concentrations observed in these studies.

Effect Modification

We also examined potential modifying effects of demographic and smoking-related factors (Table 3). There was no apparent heterogeneity in the excess odds ratio estimates by sex ($P = 0.21$) or educational level ($P = 0.23$), although there was some evidence of decreasing radon-associated lung cancer risk with age ($P = 0.09$). Overall, 57% of case information was derived from the subjects themselves rather than proxies. The excess odds ratio was higher when information was obtained from the subject rather than from a

TABLE 2. Odds Ratios* for Lung Cancer by Categories of Residential Radon Concentration and Excess Odds Ratio Per 100 Bq/M^3 Radon in the 5- To 30-Year Exposure Time Window

Radon Concentration (Bq/m^3)	No. of Cases (n = 3662)	No. of Controls (n = 4966)	Odds Ratio (95% CI)
<25[†]	832	934	1.00
25–49	1021	1432	1.13 (0.95–1.35)
50–74	669	1052	1.09 (0.89–1.34)
75–99	349	501	1.16 (0.91–1.48)
100–149	450	569	1.24 (0.96–1.60)
150–199	163	228	1.22 (0.87–1.71)
≥200	178	250	1.37 (0.98–1.92)
Excess odds ratio[‡] ($\beta \times 100$)			0.11 (0.00–0.28)

*Odds ratios stratified by sex and categories of age, duration of smoking, number of cigarettes smoked per day, number of residences, and years with α-track measurements in the exposure time window.

[†]Reference category.

[‡]Excess odds ratio ($\beta \times 100$) based on the linear model: $OR(x) = 1 + \beta x$, where x is the mean radon concentration in the 5- to 30-year exposure time window.

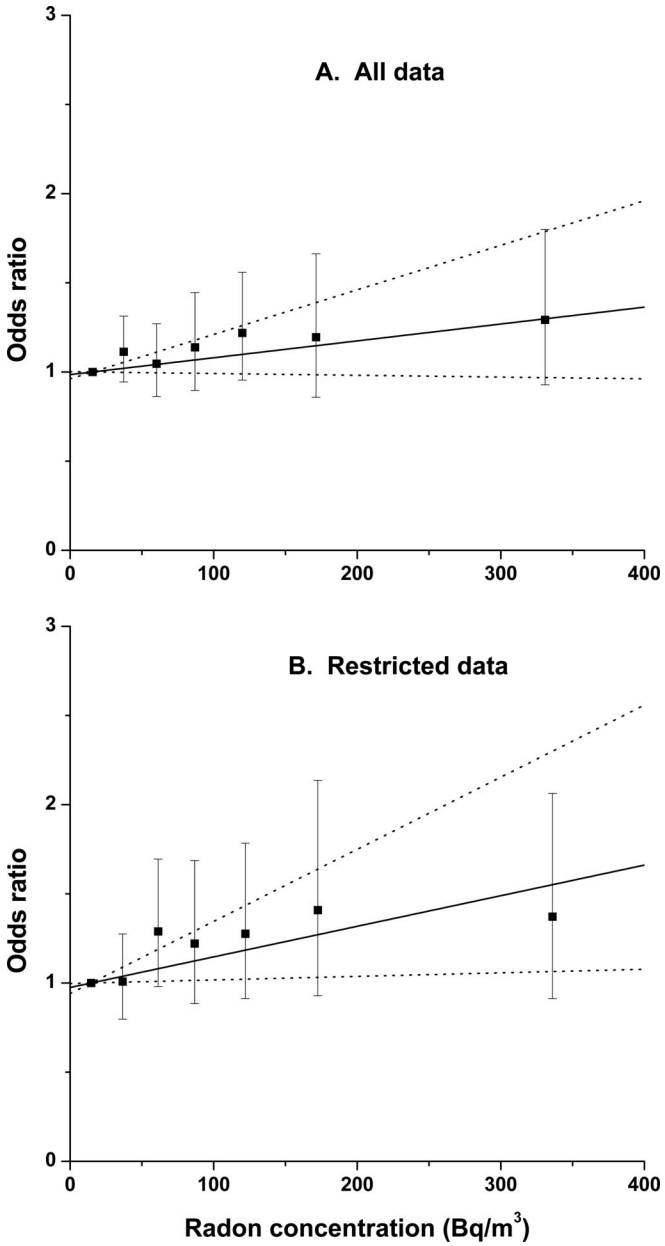

FIGURE 1. ORs and 95% CIs for categories of mean radon concentration within the 5- to 30-year exposure time window from the fitted model for the linear excess OR (solid line) and its 95% CIs (dotted lines). A, All data (n = 3662 cases, 4966 controls) and (B) restricted data, limited to subjects residing in 1 or 2 residences during the 5- to 30-year exposure time window and at least 20 years' coverage with α-track monitors (n = 1910 cases, 2651 controls).

proxy. There were no substantial differences in the excess odds ratios by categories of cigarette smoking, number of cigarettes smoked per day ($P = 0.94$), duration of cigarette smoking ($P = 0.55$), or time since quitting smoking ($P = 0.89$).

Histology

The histologic type of lung cancer was available for all but 166 lung cancer cases. There was a preponderance of adenocarcinomas (1380 of 3662 cases) as a result of the emphasis on women and (current) nonsmokers within several of the case series. The largest excess odds ratio (0.23 per 100 Bq/m^3) was observed for small cell carcinoma, although the confidence limits overlapped with other histologic types of lung cancer (Table 4). Only lesions of unknown histology failed to demonstrate a positive excess odds ratio (−0.16 per 100 Bq/m^3). Because of the reduced number of subjects, all of the confidence limits for the excess odds ratios for specific histologic types of lung cancer included zero. Similar results were obtained when cases were restricted by sex.

Data Restriction

Table 5 illustrates the consequences of increasingly stringent restrictions on radon dosimetry based on increasing the number of years in the 5- to 30-year exposure time window for which radon measurements were available using α-track monitors and limiting the number of residences occupied by the study subjects. With either no restrictions on mobility or limiting the analysis to subjects who lived in 1 or 2 homes, the excess odds ratios increased with increasing number of years monitored. Excess odds ratios were uniformly larger when data were restricted to subjects living in 1 or 2 houses as compared with no restriction on mobility.

DISCUSSION

Although radon is one of the most extensively investigated human lung carcinogens, the weight of evidence for radon carcinogenicity derives largely from occupational studies of underground miners[24] exposed to much higher radon levels than those typically encountered in homes. There are also marked differences between the conditions of exposure in mines and in houses. These differences include the relative proportion of radon itself to its decay products (which affects the amount of energy deposited in the lung), respiratory rate (which affects the rate radon and its decay products are inhaled and retained in the lung), and particle size distributions (which affect the fraction of radon progeny attached to particles and the depth of penetration and site of deposition within the lung). All of these factors complicate the direct extrapolation of occupational data on radon lung cancer risks to residential settings.[2,25]

Laboratory studies have shown direct damage of cellular DNA after the traversal of cultured mammalian cells by single alpha particles and provide direct evidence of the potential for radon carcinogenicity at low levels of exposure.[2,26] Indirect genotoxic effects of radon (including mutation and micronucleated cells) and nongenotoxic effects of radon (including sister chromatid exchange and cellular proliferation) may play a role in carcinogenesis and have been

TABLE 3. Excess Odds Ratio for Lung Cancer and 95% Confidence Interval (CI) for Time-Weighted Radon Concentration in the 5- to 30-Year Interval Before the Index Date by Categories of Demographic Factors and Categories of Cigarette Smoking-Related Factors*

Covariate	Cases (n = 3662) %	Control (n = 4966) %	Excess Odds Ratio (95% CI)†
Sex			
Women	70	72	0.19 (0.02–0.46)
Men	30	28	0.03 (−0.04–0.24)
Age at disease occurrence (years)			
<60	25	27	0.02 (—–0.35)
60–64	17	15	0.80 (0.13–2.57)
65–69	21	20	0.02 (−0.05–0.28)
70–74	18	17	0.33 (0.01–1.02)
75+	19	20	−0.02 (–0.10–0.30)
Highest grade level of education			
0–7	14	13	−0.06 (−0.28–0.54)
8–13	60	53	0.26 (0.04–0.64)
14+	25	34	0.02 (—–0.28)
Type of respondent			
Subject	57	94	0.18 (0.01–0.45)
Surrogate	43	6	−0.05 (—–0.93)
Cigarette smoking status			
Never smoked	18	44	0.10 (−0.09–0.42)
Ever smoked	80	54	0.10 (−0.02–0.33)
Number of cigarettes smoked per day			
1–9	8	11	0.31 (−0.13–2.56)
10–19	28	22	0.14 (−0.08–0.62)
20–29	27	14	0.04 (−0.04–0.36)
30+	18	7	0.12 (−0.13–1.00)
Duration of cigarette smoking (years)			
1–24	7	14	0.05 (−0.05–0.68)
25–34	14	11	−0.03 (−0.08–0.32)
35–44	27	14	0.39 (0.01–1.40)
45+	33	14	0.11 (−0.07–0.50)
Years since stopping cigarette smoking			
0	44	21	0.11 (—–0.54)
1–9	18	13	0.01 (−0.13–0.55)
10–19	9	8	0.53 (−0.21–4.45)
20+	9	13	0.29 (−0.31–2.87)

*Data limited to never smokers and cigarette-only smokers for excess odds ratios of smoking-related factors.

†Based on the linear OR model: $OR(x) = 1 + \beta x$, where x is mean radon concentration within 5- to 30-year exposure time window. Models stratified by study, sex and categories of age, duration of smoking and number cigarettes smoked per day, and number of residences and years with α-track measurements in the exposure time window. Combined estimates based on the fixed effects modeling. Numbers of cases and controls vary as a result of missing data.

demonstrated in unexposed cells in the neighborhood of cells irradiated with alpha particles.[27] Occupational studies have also demonstrated an inverse dose-rate effect of radon,[28,29] which results in higher lung cancer risks when the same cumulative exposure is experienced over a longer period of time.

The National Research Council[2] has estimated that residential radon may account for 10% to 15% of the lung cancer burden in the United States. However, there has been no unambiguous direct evidence of an increased lung cancer risk associated with residential exposures. The present pooled analysis has several strengths. It provides the largest aggre-

TABLE 4. Excess Odds Ratios* for Lung Cancer Per 100 Bq/M^3 Radon in the 5- to 30-Year Exposure Time Window by Histologic Type

Histologic Type	No. of Cases (n = 3662)	Excess Odds Ratio (95% CI)
Adenocarcinoma	1380	0.09 (−0.05–0.35)
Squamous cell	799	0.09 (−0.04–0.42)
Small/oat cell	577	0.23 (−0.08–0.88)
Other	740	0.19 (−0.02–0.62)
Unknown	166	−0.16 (—–0.06)
All	3662	0.11 (0.00–0.28)

*Excess odds ratio ($\beta \times 100$) based on fitting the linear OR model: $OR(x) = 1 + \beta x$, where x is mean radon concentration within 5- to 30-year exposure time window. Models stratified by study, sex, categories of age, duration of smoking and number cigarettes smoked per day, and number of residences and years with α-track measurements in the exposure time window.

gation of data on residential radon lung cancer risks to date. Radon dosimetry was based on long-term α-track monitors placed in current and former homes of the study subjects. Case and control selection was population-based, and we had histologic or pathologic confirmation of case diagnosis. Finally, the studies included a wide range in residential radon exposures and data on modifying factors, including age, sex, and smoking.

The analysis is inherently limited by the quality and reporting of the original residential radon studies, as well as the need for a common data format. For example, in the interest of comparability across studies, we used air radon measurements in living areas to characterize exposure in the Iowa study, even though those investigators had detailed radon measurements both inside and outside the house with links to historical patterns of mobility.[14] Results for specific histologic types of lung cancer also require cautious interpretation because only the Missouri and Iowa studies obtained consensus diagnoses by a panel of blinded expert pathologists. Brownson et al.[30] observed overall concordance between original histologic diagnoses of lung cancer and a consensus histopathologic review of only 66%.

Overall, the odds ratios for lung cancer increased with increasing radon exposure categories, with an odds ratio of 1.37 (0.98–1.92) for concentrations exceeding 200 Bq/m^3 relative to concentrations under 25 Bq/m^3. The overall estimate of the excess odds ratio for lung cancer was 0.11 (0.00–0.28) per 100 Bq/m^3. Restrictions that increased coverage of the exposure time window resulted in increasing excess odds ratios. For example, those subjects who had resided in only 1 or 2 houses in the period 5 to 30 years before recruitment with at least 20 years covered by α-track monitors had an excess odds ratio of 0.18 (0.02–0.43) per 100 Bq/m^3.

It is possible that the findings for the restricted data were the consequence of differentially excluding participants in the negative studies. To explore this, we examined the proportions of cases and controls from each study who contributed to the combined risk estimates. There were slightly larger proportions of subjects from Iowa (cases and controls) and Connecticut (cases only) with increasing stringency of restrictions, smaller proportions from Missouri-II and Winnipeg and similar proportions from Missouri-I, New Jersey, and Utah–South Idaho. Overall, however, there was little indication that the increasing excess odds ratios in Table 5 are the result of differential contributions from particular studies.

Our overall excess odds ratio estimate of 0.11 is consistent with the predicted excess odds ratio of 0.12 (0.02–0.25) per 100 Bq/m^3 based on a linear model developed by the National Research Council[2] using data on low-exposed miners whose exposures were similar to long-term residents of high radon homes; similar risk projections were also obtained from risk models derived from the full range of miner data by the National Research Council. The consistency of the residential estimates of risk with the results from data on all miners and low-exposed miners increases the confidence that current radon estimates are not the result of unknown latent factors such as confounding or study-specific biases.

The residential radon measurements in these case–control studies are subject to measurement error. No formal adjustment for this source of error was attempted. Such adjustments require repeated radon measurement in the same home, which were generally not available. Adjustment for exposure measurement error in studies conducted in South West England,[31] Sweden,[32] and Gansu, China[33] resulted in an increase in the estimated excess odds ratio of 50% or more. Our restricted analyses of the 7 North American case–control studies resulted in an increase in the estimated excess odds ratio of approximately 50%, most likely by reducing exposure misclassification. Although the increased risks observed in the subset analyses may be attributable to some unidentified systematic or differential bias, we are unaware of specific sources of bias that could affect our analyses. In most case–control studies, nondifferential misclassification of exposure results in a bias toward the null.[34–36] Field et al.[37] have recently demonstrated that empiric models with improved retrospective radon exposure estimates were more likely to detect an association between prolonged residential radon exposure and lung cancer.

Collectively, our results provide direct evidence of an association between residential radon exposure and lung cancer, a finding predicted by downward extrapolation of epidemiologic data on underground miners exposed to higher

TABLE 5. Excess Odds Ratios* for Lung Cancer Per 100 Bq/m^3 Radon in the 5- to 30-Year Exposure Time Window by Years of Radon Monitoring, With and Without Restrictions on Mobility

Years Monitored With α-Track Detectors	No. Cases	No. Controls	Excess Odds Ratio (95% CI)
No restrictions on mobility			
>0	3662	4966	0.11 (0.00–0.28)
≥10	3148	4321	0.13 (0.01–0.32)
≥15	2764	3857	0.13 (0.00–0.31)
≥20	2263	3172	0.14 (0.01–0.35)
25	1621	2323	0.21 (0.03–0.50)
Subjects residing in 1 or 2 houses			
>0	2467	3430	0.15 (0.01–0.37)
≥10	2308	3194	0.15 (0.00–0.37)
≥15	2171	3009	0.17 (0.01–0.41)
≥20	1910	2651	0.18 (0.02–0.43)
25	1552	2170	0.21 (0.03–0.52)

*Excess odds ratio (ß × 100) based on the linear odds ratio model: OR(x) = 1 + βx, where x is the mean radon concentration in the 5- to 30-year exposure time window. Models stratified by study, sex and categories of age, duration of smoking, number of cigarettes smoked per day, number of residences, and years with α-track measurements in the exposure time window.

levels of radon and consistent with toxicologic results from animal and in vitro studies. Additional support for this conclusion has been provided by a combined analysis of the 2 Chinese case–control studies,[38] involving a total of 1050 cases and 1996 controls. Further information on residential radon lung cancer risks will be provided by an ongoing analysis of European case–control studies,[39] to be followed by a global combined analysis of all residential radon case–control studies.

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