Case-Control Study of Residential Radon and Lung Cancer among New Jersey Women

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ABSTRACT

To evaluate the association of indoor radon exposure with lung cancer risk, a 1-year track detector measurements of radon were conducted in dwellings which had been occupied for at least 10 years by 433 New Jersey female lung cancer cases and 402 controls who were subjects in a larger population-based study. Adjusted odds ratios were 1.1 (90% confidence interval, 0.79–1.7), 1.3 (90% confidence interval, 0.62–2.9), and 4.2 (90% confidence interval, 0.99–17.5) for exposures of 1.0–1.9, 2.0–3.9, and 4.0–11.3 pCi/liter, respectively, relative to exposures of less than 1.0 pCi/liter, showing a significant trend (1-sided P = 0.04) with increasing radon concentration. The trend was strongest among light smokers (less than 15 cigarettes/day, 1-sided P = 0.01). The trend for lung cancer risk with estimated cumulative radon exposure was slightly weaker (1-sided P = 0.09). The increase in relative risk for each unit of cumulative exposure, 0.8% (90% confidence interval, 0.0–8.0%) per working level month, was consistent with the range of 0.5–4.0% per working level month generally reported for underground miner studies, supporting the extrapolation of the occupational data to the residential setting. However, the possibility of selection biases, the small number of high exposures, and other uncertainties necessitate caution in interpretation of these data.

INTRODUCTION

Prolonged exposures to high levels of radon have been identified as a cause of lung cancer in underground miners, with numerous studies showing a strong and consistent dose-response relationship (1, 2). Based on extrapolation from the miner data, it has been postulated that concentrations of radon found in some houses may also result in a substantial lung cancer risk (3–5). However, direct information on risk from residential radon exposures has been very limited thus far (6). Some correlation studies have reported a positive association between lung cancer mortality and average radon concentrations, while others have reported no relationship or a negative association (6); none of these studies has taken into account the possibility of selection biases, the small number of high exposures, and other uncertainties necessitate caution in interpretation of these data.

A case-control study of lung cancer was conducted among New Jersey women from 1982 to 1984; extensive data on smoking, occupation, and diet were collected (13). This study was extended to include data on radon exposures, in order to evaluate the association of indoor radon with lung cancer risk.

MATERIALS AND METHODS

The cases in the original study included all female New Jersey residents who were newly diagnosed with histologically confirmed primary cancer of the lung from August 1982 through September 1983. Population-based controls were chosen from three sources. For cases who were themselves interviewed, controls were selected from a random sample of New Jersey driver's license files (age <65) or Health Care Financing Administration files (age 65+), and were frequency matched to cases by race and age. For cases with next of kin respondents, death certificates with no mention of any respiratory disease were used for random selection of controls who were individually matched to cases by race, age, and closest date of death. Of 1306 cases and 1449 controls originally identified, 994 (76%) cases and 995 (69%) controls or their next of kin were interviewed and were potential subjects for the radon substudy. Further details on case ascertainment and control selection have been reported previously (13).

The questionnaire for this study included a lifetime brand-specific smoking history, information on smoking by other household members, lifetime residential and occupational histories, and a dietary history of foods containing vitamin A. The residential history specified only the towns in which each subject had lived. For the radon substudy, subjects or their next of kin were recontacted to determine exact street addresses and dates of residence. Because the budget allowed for measurements in only one house per subject, a residence criterion was established; subjects were included in the radon substudy if they had lived in a single “index” residence for at least 10 years in the 10–30-year period prior to diagnosis or selection. This residence criterion also allowed sufficient duration of exposure and assumed a minimum 10-year period between relevant radon exposure and lung cancer diagnosis (14). The dates of residence at the index residences were validated whenever possible using tax office records (15).

A yearlong α track detector measurement of radon was conducted in the living area of the index residence (usually the master bedroom) and was assumed to provide the best estimate of the average radon concentration to which a subject had been exposed when she was a resident of this house. A second α track detector was also placed in each house, usually in the basement. For about 15% of the houses, a third α track detector was placed with one of the first two as a quality control check on the measurement precision (17), as determined by the coefficient of variation (18). Spiked detectors (n = 37; 4–5 each at eight concentrations from 0.5 to 2.1 pCi/liter) and blank detectors (n = 17) were also submitted to the laboratory for processing with study detectors.

At each residence, 4-day measurements of radon were conducted under closed house conditions during the heating season using charcoal canisters (17) from the NJDEP. The charcoal canister measurements served two purposes: to provide back-up data if α track measurements were not completed; and to provide current residents with a screening measurement to identify any high radon levels which might need immediate remediation. Remediation efforts were undertaken at only one house as a result of these screening measurements; α track detector measurements completed before the remedial work were used in these analyses. Details on the comparison between canister and α track detector measurements in this study will be presented elsewhere.

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2. To whom requests for reprints should be addressed, at Chronic Disease Epidemiology Program, New Jersey State Department of Health, CN369, Trenton, NJ 08625.
3. The abbreviations used are: NJDEP, New Jersey State Department of Environmental Protection; OR, odds ratio; CI, confidence interval; WLM, working level month.
The paired α track detectors had coefficients of variation of 0.36, 0.22, and 0.12 for measurements of <1.0, 1.0–1.9, and 2.0–3.9 pCi/liter, respectively; one pair was above 4.0 pCi/liter and had a difference of 0.2 pCi/liter (15). Spiked and blank α track detectors also showed poorer precision and accuracy in readings below 1.0 pCi/liter (15). Because of this measurement uncertainty and the large number of readings in this low range, data analyses were conducted using the broad categories of <1.0, 1.0–1.9, 2.0–3.9, and 4.0+ pCi/liter, consistent with a lognormal distribution (19). Some analyses were also conducted considering radon concentration as a continuous variable.

Cumulative radon exposure in pCi/liter-years was estimated from the living area radon concentrations and the number of years the subject lived in the index residence during the period 5–30 years prior to diagnosis or selection. This time period was used because data had been published (2, 20) which indicated a shorter period (5 years) between relevant radon exposure and diagnosis of lung cancer than had been assumed at the time of study design. For each year during this period when the subject did not live at the index residence, an exposure of 0.6 pCi/liter, the median concentration among controls, was assumed.

ORs and 90% CIs for the association of lung cancer with radon concentration or with cumulative radon exposure were estimated by multiple logistic regression analysis (21) using the microcomputer-based LOGRESS program (22). The significance of the trend in risk with increasing radon exposure was evaluated using the model Z statistic for a weighted categorical exposure variable, with a one-sided P value. All analyses were adjusted for lifetime average number of cigarettes smoked per day (lifetime nonsmokers; smokers of <15, 15–24, 25+ cigarettes/day). Additional categorical variables considered in the logistic models were based on age, respondent type, race, education, county of residence, years since smoking cessation, cigarette tar content, total years smoked, high-risk occupation, and vegetable consumption. No attempt was made to adjust for possible differences in house occupancy, based on the occupational history. The difference in the likelihood ratio statistics between successive models, evaluated as a χ² statistic, was used to determine the goodness of fit of these models. The best control for confounding was obtained with the model with the greatest improvement in fit, which adjusted for number of cigarettes per day plus time since smoking cessation, age, occupation, respondent type, and the interaction between respondent type and number of cigarettes per day.

RESULTS

Of 994 cases and 995 controls in the original lung cancer study, 433 cases and 402 controls were included in the radon substudy (Table 1). Actual radon measurements were conducted for 411 cases and 385 controls; living area α track measurements were completed for 346 cases and 318 controls and were estimated from baseline α track results for 27 cases and 28 controls or from charcoal canister results for 38 cases and 39 controls. Another 22 cases and 17 controls who lived in apartments above the second floor were assumed to have radon exposures less than 1.0 pCi/liter.

The remaining 561 cases and 593 controls from the original study were not included in the radon substudy because no address-specific information could be collected, no address met the residence criterion, or radon tests could not be conducted at the index residence (Table 1).

The distributions of subjects in the radon substudy according to demographic characteristics and various risk factors are shown in Table 2. The substudy subjects were fairly representative of the women in the original study except with respect to age, race, and education. Only 35% of the original subjects under age 58 were included in the substudy, compared to 44% of women ages 58–71 years and 43% of women over age 71 years (χ² = 12.8; P = 0.002). Similarly, inclusion of original study subjects was 17% for nonwhites compared to 44% for whites (χ² = 48.7; P < 0.001), and 32% for women with less than 8 years of education compared to 41 and 51% for women with 8–12 or more than 12 years, respectively (χ² = 29.5; P < 0.001). In further comparisons of subjects who were included in the substudy with those who were not included, there were few significant differences except among heavy smokers, who showed some unusual risk factor distributions. For example, for the heavy smokers included in the substudy, lung cancer risk increased with increasing vegetable consumption, which

<table>
<thead>
<tr>
<th>Cases</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at diagnosis (yr)</td>
<td></td>
</tr>
<tr>
<td>&lt;58</td>
<td>98</td>
</tr>
<tr>
<td>58–71</td>
<td>215</td>
</tr>
<tr>
<td>72+</td>
<td>120</td>
</tr>
<tr>
<td>Respondent type</td>
<td></td>
</tr>
<tr>
<td>Self</td>
<td>246</td>
</tr>
<tr>
<td>Next of kin</td>
<td>187</td>
</tr>
<tr>
<td>Race</td>
<td></td>
</tr>
<tr>
<td>White, including Hispanic</td>
<td>418</td>
</tr>
<tr>
<td>Nonwhite</td>
<td>15</td>
</tr>
<tr>
<td>Education (yr completed)</td>
<td></td>
</tr>
<tr>
<td>&lt;8</td>
<td>35</td>
</tr>
<tr>
<td>8–12</td>
<td>278</td>
</tr>
<tr>
<td>13+</td>
<td>120</td>
</tr>
<tr>
<td>Cigarette smoking</td>
<td></td>
</tr>
<tr>
<td>Lifetime nonsmokers</td>
<td>61</td>
</tr>
<tr>
<td>Smokers (av. cigarettes/day)</td>
<td></td>
</tr>
<tr>
<td>&lt;15</td>
<td>83</td>
</tr>
<tr>
<td>15–24</td>
<td>178</td>
</tr>
<tr>
<td>25+</td>
<td>111</td>
</tr>
<tr>
<td>Smokers (yr since quit smoking)</td>
<td></td>
</tr>
<tr>
<td>0–1 (current)</td>
<td>289</td>
</tr>
<tr>
<td>2–9</td>
<td>49</td>
</tr>
<tr>
<td>10+</td>
<td>34</td>
</tr>
<tr>
<td>Vegetable consumption, servings/month</td>
<td></td>
</tr>
<tr>
<td>&lt;35</td>
<td>118</td>
</tr>
<tr>
<td>35–74</td>
<td>241</td>
</tr>
<tr>
<td>75+</td>
<td>74</td>
</tr>
<tr>
<td>Occupation</td>
<td></td>
</tr>
<tr>
<td>No high-risk occupation</td>
<td>350</td>
</tr>
<tr>
<td>High-risk occupation</td>
<td>83</td>
</tr>
</tbody>
</table>

* Cutpoints based on distribution of controls in original study (1st, 2nd + 3rd, 4th quartiles).

Table 2 Characteristics of 433 cases and 402 controls in substudy of radon among New Jersey women

Table 1 Original 994 New Jersey female lung cancer cases and 995 controls, by their status in the radon substudy

<table>
<thead>
<tr>
<th>Cases</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Status</td>
<td>No.</td>
</tr>
<tr>
<td>Included in radon study*</td>
<td>433</td>
</tr>
<tr>
<td>No address-specific information</td>
<td>140</td>
</tr>
<tr>
<td>No address met residence criterion</td>
<td>253</td>
</tr>
<tr>
<td>No radon testing at index address</td>
<td>168</td>
</tr>
</tbody>
</table>

* Index residence tested for radon with α track detectors and/or charcoal canisters (411 cases, 385 controls); index residence was an apartment above the second floor, radon exposures assumed to be <1 pCi/liter (22 cases, 17 controls).

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was opposite to the pattern observed for heavy smokers not included in the substudy as well as for most other subgroups.

The median length of residence at the index address was 26 years for cases and 27 years for controls. Considering the period from 5 to 30 years prior to diagnosis or selection, the median residence time was 20 years for cases and 21 years for controls; only 18% of cases and 21% of controls lived at the index residence for less than 15 years of this period. Validation of residential histories was conducted for the index residence of all but 17% of the cases and 15% of the controls. Unresolved discrepancies between the tax office date of purchase and the reported year of first residence were >5 years for only 2% of cases and 2% of controls.

Table 3 shows the case-control distributions of index residence radon concentrations, as well as adjusted ORs at each exposure level. The number of subjects with high exposures was very small, and none of the ORs was statistically significant. However, lung cancer risk increased with radon and showed a statistically significant trend. When the small numbers of subjects in the 2.0–3.9 and 4.0–11.3 pCi/liter groups were combined, the OR was 1.8 (90% CI, 0.89–3.5). Adjusting only for the number of cigarettes smoked per day showed fairly similar results, with a slightly weaker trend (P = 0.068) based on an OR of 3.5 (90% CI, 0.82–15.2) at exposures of 4.0–11.3 pCi/liter.

Analyses by separate smoking groups showed some differences in the patterns of radon-related risk (Table 4). For lifetime nonsmokers, the pattern was inconsistent; taking spouse smoking into account did not result in any changes in the nonsmokers’ pattern of radon-related risk. The ORs increased significantly with radon exposure for smokers of less than 15 cigarettes/day and to a lesser extent for smokers of 15–24 cigarettes/day. Smokers of 25+ cigarettes/day showed a pattern of decreasing ORs with increasing radon. For all smokers combined, the ORs were 1.0, 1.3, and 2.0 for exposures of less than 1.0, 1.0–1.9, and 2.0–11.3 pCi/liter, respectively.

Lung cancer risks also increased with increasing estimated cumulative radon exposure (Table 5), but the trend was slightly weaker than for radon concentration. The OR for 100–155 pCi/liter-years, although statistically significant, was based on only 4 cases and 1 control. When the small numbers of subjects with 50–99 and 100–155 pCi/liter-years were combined, the OR was 1.4 (90% CI, 0.65–3.0). Patterns of risk by separate smoking groups (data not shown) were similar to those for radon concentration.

When analyses were limited to the 346 cases and 318 controls with actual living area a track measurements, the trends were slightly weaker (P = 0.085 for radon concentration and P = 0.192 for cumulative radon exposure). When a continuous exposure variable was used in the models in place of the weighted categorical term, the trends were also slightly weaker (P = 0.074 for radon concentration, and P = 0.111 for cumulative radon exposure).

In order to compare the results of this study with data from the miner studies, the continuous cumulative exposure analyses were used to calculate the increase in risk per pCi/liter-year, and the corresponding increase in risk per WLM (assuming 80% occupancy and 50% equilibrium between radon and its decay products, 1 pCi/liter-year equals 0.2 WLM). This relative risk coefficient was 3.4% (90% CI, 0.0–8.0%) per WLM for all subjects combined, 3.6% (90% CI, 0.0–9.3%) per WLM for smokers, and 2.0% CI, 0.0–10.2%) for nonsmokers.

Analyses conducted by lung cancer histological type also showed the pattern of increasing risk with increasing radon, except for squamous cell carcinoma. For radon concentration, the trend was strongest for large cell undifferentiated carcinomas (P = 0.027); while for cumulative radon exposure, the trend was strongest for small cell undifferentiated carcinomas (P = 0.079).

**DISCUSSION**

In this case-control study of lung cancer among New Jersey women, risk estimates showed a significant trend with increasing residential radon concentration, even after adjusting for detailed smoking and occupational histories as well as for other factors. Most of the earlier case-control studies which also suggested a radon-lung cancer association had used housing characteristics as surrogates for radon exposure and had conducted measurements only in a sample of houses (7, 9, 11–12).

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**Table 3** Lung cancer OR (90% CI) for New Jersey women by yearround living area radon concentrations

<table>
<thead>
<tr>
<th>Radon (pCi/liter)</th>
<th>Cases</th>
<th>Controls</th>
<th>Adjusted OR&lt;sup&gt;a&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1.0</td>
<td>342</td>
<td>79.0</td>
<td>324</td>
</tr>
<tr>
<td>1.0–1.9</td>
<td>67</td>
<td>15.5</td>
<td>16.4 (0.79–1.17)</td>
</tr>
<tr>
<td>2.0–3.9</td>
<td>18</td>
<td>4.2</td>
<td>2.5 (1.36–2.9)</td>
</tr>
<tr>
<td>4.0–11.3</td>
<td>6</td>
<td>1.4</td>
<td>0.5 (0.42–19.75)</td>
</tr>
</tbody>
</table>

Trend (1-sided P value): 0.040

<sup>a</sup> Yearround living area measurements for 346 cases, 318 controls; estimates of yearround living area concentrations for 87 cases, 84 controls.

<sup>b</sup> Adjusted for smoking (lifetime nonsmokers; smokers by lifetime average number of cigarettes/day and years since quit smoking); age; occupation; respondent type; and interaction terms between respondent type and number of cigarettes/day.

<sup>c</sup> Numbers in parentheses, 90% CI.

**Table 4** Lung cancer OR<sup>a</sup> by yearround living area radon concentrations for New Jersey female lifetime nonsmokers and smokers by average cigarette/day

<table>
<thead>
<tr>
<th>Radon (pCi/liter)</th>
<th>Lifetime nonsmokers</th>
<th>Smokers (cigarettes/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1.0</td>
<td>1.0 (48, 160)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1.0 (61, 77)</td>
</tr>
<tr>
<td>1.0–1.9</td>
<td>0.5 (89, 139, 55)</td>
<td>1.0 (94, 24)</td>
</tr>
<tr>
<td>2.0–11.3</td>
<td>1.2 (6, 0)</td>
<td>2.4 (11, 2)</td>
</tr>
</tbody>
</table>

Trend<sup>a</sup> P = 0.390

<sup>a</sup> OR adjusted by years since quit smoking (except nonsmokers), age, occupation, and respondent type.

<sup>b</sup> Numbers in parentheses, number of cases and controls.

**Table 5** Lung cancer OR (90% CI) for New Jersey women by estimated cumulative radon exposure

<table>
<thead>
<tr>
<th>Cumulative radon (pCi/liter-yr)</th>
<th>Cases</th>
<th>Controls</th>
<th>Adjusted OR&lt;sup&gt;a&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;25</td>
<td>361</td>
<td>83.4</td>
<td>340</td>
</tr>
<tr>
<td>25–49</td>
<td>56</td>
<td>12.9</td>
<td>52</td>
</tr>
<tr>
<td>50–99</td>
<td>12</td>
<td>2.8</td>
<td>9</td>
</tr>
<tr>
<td>100–155</td>
<td>4</td>
<td>0.9</td>
<td>12</td>
</tr>
</tbody>
</table>

Trend (1-sided P value): 0.090

<sup>a</sup> Estimated cumulative radon exposure from 5–30 years prior to case diagnosis or control selection; assumes exposure of 0.6 pCi/liter (median radon concentration for controls) for all years in this period when subject did not live at index residence.

<sup>b</sup> OR adjusted by years since quit smoking (except nonsmokers), age, occupation, and respondent type; and interaction terms between respondent type and number of cigarettes/day.

<sup>c</sup> Numbers in parentheses, 90% CI.
Two small studies included measurements for most or all subjects, 19 cases and 159 controls in one study (8) and 27 cases and 49 controls in the other (10).

The radon exposure data in this study were based on actual measurements for 411 cases and 385 controls, generally yearlong α track detectors in the living areas of long-term residences. However, a weakness of any retrospective study of this type is that exposure data are collected in the present time, although the exposure actually occurred over a longer period in the past. Major changes in house construction, heating, ventilation, and occupants’ activity could cause inaccuracies in the exposure estimates. For example, improved insulation and other energy conservation measures may have increased the radon concentrations to varying extents. Conversion from forced air to hot water heating may have decreased the radon concentrations in the living area. Differences in the past and present degree of ventilation of houses may also result in measured concentration which differ from actual exposures. Some subjects may have spent more time on floors with higher radon concentrations, although comparison of basement/first floor and basement/second floor radon ratios suggest that there was not a substantial systematic difference between concentrations on the first and second floors (15). Altogether, these exposure uncertainties provide another rationale, in addition to the lower precision of measurements under 1 pCi/liter, for the use of categorical rather than continuous analyses.

There are several other reasons for interpreting the data in this study cautiously, including the possibility of selection biases, the small number of subjects with high radon exposures, and the incomplete cumulative exposure assessments. The original population-based samples of 1306 eligible cases and 1449 eligible controls were reduced to 995 cases and 994 controls who were interviewed and further reduced to 433 cases and 402 controls whose data were analyzed in the radon substudy. The substudy subjects were not completely representative of all those interviewed. The younger, nonwhite, and less highly educated subjects more often were not included in the substudy because they did not meet the residence criterion. Heavy smokers included in the substudy showed unusual risk factor distributions, for example, with respect to vegetable consumption. We do not know if any of these differences resulted in a substudy sample which was biased with respect to radon distributions.

The small number of residences in this study with high yearround radon concentrations is attributable to several factors: (a) the original case-control study was population based and included more subjects from densely populated counties and from urban areas which in New Jersey tend to have low radon levels; (b) because of the residence criterion, index houses were at least 25 years old at the time of the radon measurements, and most were 41–90 years old; a statewide NJDEP survey of radon in New Jersey (23) found the highest concentrations in houses which were less than 10 years old, and the lowest concentrations in houses which were 41–90 years old; (c) charcoal canister (screening) results from this study and from the NJDEP survey suggest that about 14% of New Jersey residents live in houses with basement heating season concentrations of radon above 4 pCi/liter. However, in this study, the ratio of yearround living area α track to basement screening measurements decreased as the screening concentrations increased (15). Therefore, the percentage of subjects with living area annual exposures above 4 pCi/liter was only about 1%.

Because of the low exposure distribution, the risk estimates for concentrations of 4–11.3 pCi/liter or cumulative exposures of 100–155 pCi/liter-years have very wide confidence intervals. It may be more appropriate to consider the ORs of 1.8 for the concentration interval 2.0–11.3 pCi/liter and 1.4 for the exposure interval 50–155 pCi/liter-years, inasmuch as they are based on slightly larger numbers and their confidence intervals are narrower. Nevertheless, all of the risk estimates are relatively stable; adjustment for several other factors in addition to number of cigarettes per day resulted in only slight changes in the ORs or in the trend statistics. This finding is important because most of the earlier case-control studies were not able to take into account detailed aspects of smoking or other possible lung cancer risk factors, such as occupation and diet.

The small numbers of subjects with high measurements also limit the conclusions that can be drawn from analyses of subgroups. This applies particularly to the different patterns of radon-related risk among smoking subgroups. The possibility of selection biases among heavy smokers and the possibility of misclassification of smoking by next of kin respondents (13) also mean that apparent differences in risk according to smoking could be spurious. Nonetheless, some evidence from other studies is consistent with at least a weaker radon-related effect in heavy smokers. Experiments with beagles showed protective effects of high doses of tobacco smoke for radon-induced lung cancer, possibly because less α radiation could reach the bronchial epithelium through the thickened mucus layer (24). In the Colorado Plateau uranium miner studies, cohort members were usually not heavy smokers (25, 26). In addition, one residential radon case-control study also found a slightly stronger radon-lung cancer association for smokers of less than 10 cigarettes/day than for heavier smokers (12).

The OR for radon exposures of 2.0–11.3 pCi/liter was 1.2 for lifetime nonsmokers, compared to 2.0 for smokers, consistent with other analyses (27) which suggest that smoking prevention or cessation is a key factor in control of radon health effects. However, the small number of lifetime nonsmokers in this study is insufficient to rule out a significant radon hazard for this group.

The study findings do not suggest that the radon association is restricted to any single histological type, such as small cell carcinoma. However, the small numbers, the absence of a uniform pathology slide review, and some evidence suggesting the possibility of misclassification by histological type (13) also limit the conclusions that can be drawn.

In the miner studies, lung cancer risk varied directly with cumulative radon exposure (1–2, 6). In this study, the trends with cumulative radon exposure were slightly weaker than those for radon concentration. This may be due to the incomplete cumulative exposure assessments and the use of a conservative exposure estimate for those years when the subject did not live in the index residence. Exposure could have been misclassified, particularly for the 15–16% of the subjects who lived in the index residence for less than 15 years of the period from 5–30 years prior to diagnosis or selection. Additional houses which were also residences of the subjects during this 25-year period are now being tested for radon as part of a second phase of this study; more complete cumulative exposure assessments for these subjects might partially correct this misclassification.

Radon exposure during remaining lifetime years (beyond the 25-year period) may also have contributed to lung cancer risk. Analyses including all available data on the time window 5–40 years and 5+ years did not appreciably alter the relative risk coefficients. However, relatively few subjects had complete measurements for these longer time periods.
Edling et al. (28) used their residential radon data to calculate an attributable risk coefficient for nonsmokers of 5–7 cases/WLM/million persons at risk, which was within the range of 5–50 cases reported for the miner studies. In our study, the relative risk coefficient of 3.4%/WLM was also within the usual occupational range of 0.5–4.0% (5, 6). Therefore, the findings of increased lung cancer risk with increasing residential radon seem to be consistent with the miner studies and add support to the extrapolation of the occupational data to the residential setting. However, because of the limitations and uncertainties discussed above, the findings of this study need to be corroborated by other residential radon studies currently under way worldwide.

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REFERENCES

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