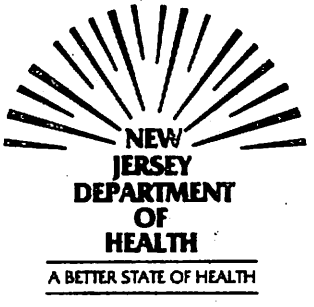
A CASE-CONTROL STUDY OF RADON AND LUNG CANCER AMONG NEW JERSEY WOMEN

TECHNICAL REPORT - PHASE I

AUGUST, 1989



NEW JERSEY STATE DEPARTMENT OF HEALTH

DIVISION OF EPIDEMIOLOGY AND DISEASE CONTROL
DIVISION OF OCCUPATIONAL AND ENVIRONMENTAL HEALTH

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EXECUTIVE SUMMARY

The New Jersey State Department of Health (NJDOH) has been conducting an epidemiologic study of radon and lung cancer in New Jersey women. This study focuses on the questions of whether and to what extent radon in homes is associated with increased lung cancer risk. The importance of this question arose from the 1985 finding of very high levels of radon, a known cause of lung cancer among underground miners, in some Eastern Pennsylvania residences.

The New Jersey research reported here is the first large-scale epidemiological study of radon and lung cancer based on actual measurements in homes and detailed smoking histories for individual subjects. It is an extension of a case-control study of lung cancer which previously had been conducted among New Jersey women. The cases in that study were women newly diagnosed with lung cancer from August 1982 through September 1983, while the controls were women without lung cancer but similar in age and race to the cases. Information on smoking, residential, occupational and dietary histories was collected for 994 cases and 995 controls.

The radon substudy initially focused on those New Jersey dwellings which met a residence criterion, i.e., where subjects had lived the longest and for at least 10 years during the period from 10-30 years prior to lung cancer diagnosis or control selection. Both long-term and short-term radon measurements were made in these houses. Radon exposures for subjects were estimated by year-long alpha track detector measurements in the living areas. Four-day measurements of radon were made using charcoal canisters in basements to provide quick screening measurements for current residents, in case radon levels were so high that immediate remediation was needed, and to provide back-up data in case year-long measurements of radon were not completed.

This report is based on radon exposure data from 433 cases and 402 controls. Some of the original cases and controls were not included in the radon substudy because address-specific information could not be collected, because no house met the

residence criterion, or because radon tests could not be conducted at a house which did meet this criterion.

The overall distribution of radon exposure was generally low: only 24 cases (5.6%) and 12 controls (3.0%) had year-round living area radon concentrations of 2 pCi/L or greater. After smoking, age and occupation were taken into account, the estimated lung cancer risk for those exposed to the highest radon category (2-11 pCi/L) was 80% greater than the risk for those at the lowest exposure level (less than 1.0 pCi/L). Because the number of subjects in the higher exposure category was small, however, the relative risk estimate was not statistically significant. In contrast, the trend for increasing risk with increasing radon exposure was statistically significant; the probability that this trend was due to chance alone was only 4%.

When duration of exposure was also taken into account, similar patterns of increasing risk with increasing cumulative radon exposure were seen. The estimated lung cancer risk for those exposed to the highest cumulative radon category (50-155 pCi/L-years) was 40% greater than the risk for those at the lowest exposure level (less than 25 pCi/L-years). Furthermore, the increase in lung cancer risk over background risk per unit of cumulative exposure was consistent with that generally found in the studies of underground miners.

Study analyses also showed that lung cancer risk for women who smoked about one pack a day was 1,000% greater than risk for lifetime nonsmokers. This again confirmed that **smoking is the major cause of lung cancer.**

Some of the results of this study must be interpreted cautiously because of the small number of subjects in the highest radon exposure categories. Extensive data analyses and discussion throughout the technical report and its appendices are designed to consider the extent of any possible biases introduced by reduction of the potential study population to those with actual radon exposure estimates.

Nevertheless, the study suggests that the findings of radon-related lung cancer in miners can be applied to the residential setting. Excess radon exposures typical of homes may increase risk of lung cancer; extremely high residential exposures would be associated with very serious lung cancer risks. These results support the comprehensive interdepartmental radon-related effort initiated in 1985 by the NJDOH and the New Jersey Department of Environmental Protection, including provision of technical information and services, citizen education, and research activities. The study also confirms that smoking avoidance education should be strongly emphasized along with radon reduction activities.

The exposure data yielded by this study also suggest that the relationship between screening measurements and year-round living area measurements need better characterization for public policy purposes and clearer understanding by the public before remediation decisions are made. In addition, building code modification to prevent radon entry may be an effective means for reducing overall population risks from radon exposure.

Further data analyses may refine the results of this study. A second, still ongoing phase of data collection will add more subjects to the substudy, and will result in more complete exposure histories from additional houses for those subjects already included.

The findings of this study also need to be corroborated by other residential radon studies currently underway worldwide. In the meantime, existing actions to reduce radon exposure to the lowest feasible levels should be maintained. Remedial action should be taken in residences when follow-up testing indicates that typical exposures of occupants are above 4 pCi/L. This recommendation is not based upon the absence of any risk below 4 pCi/L; rather, it is based upon the limited feasibility of remediating residences below that level.

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This report on the New Jersey Radon-Female Lung Cancer Case-Control Study was written by Janet Schoenberg (NJDOH, Division of Epidemiology and Disease Control) and by Judith Klotz (NJDOH, Division of Occupational and Environmental Health). Other persons who made important contributions to this study include Homer Wilcox, Maria Gil-del-Real, and Annette Stemhagen (NJDOH, Division of Epidemiology and Disease Control), Gerald Nicholls and Mary Cahill (NJDEP), Zdenek Hrubec (National Cancer Institute, Radiation Epidemiology Branch), and Thomas Mason (National Cancer Institute, Environmental Epidemiology Branch).

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INTRODUCTION

In early 1985, the New Jersey State Department of Health (NJDOH) and the New Jersey State Department of Environmental Protection (NJDEP) considered public policy implications of the extremely high concentrations of radon[1] in homes on the Reading Prong in Pennsylvania. The Reading Prong geologic region was known to extend into New Jersey. Moreoever, gamma radiation surveys by aerial overflights suggested that the radon problem in New Jersey might extend beyond the Reading Prong. Subsequently, elevated radon levels were measured in homes in other regions of the state. Some New Jersey homes were identified for which the lifetime cumulative exposures of residents would exceed those for most uranium miners.

Among the initial decisions, and prior to any systematic testing of homes in New Jersey, was the commitment to extend a recently-conducted statewide female lung cancer study by including data on radon exposures. The collaborators in this study extension included staff and managers of the Division of Epidemiology and Disease Control and the Division of Occupational and Environmental Health of the NJDOH, the Division of Environmental Quality of the NJDEP, and both the Radiation Epidemiology Branch and the Environmental Epidemiology Branch of the National Cancer Institute.

The evidence that lung cancer is caused primarily by cigarette smoking is voluminous and incontrovertible (USDHEW, 1979, 1980). Evidence is also strong regarding other risk factors, including various occupations (Fraumeni, 1975) and diet (Ziegler et al., 1986). The roles of environmental pollution, urban-rural differences independent of smoking, and other potential risk factors are not as clear (Fraumeni and Blot, 1982).

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[1] For the rest of this report, "radon" refers to both the gas itself and its short-lived particulate decay products. Two principal types of units appear in this report, those that denote radon or radon progeny concentrations (pCi/L, WL, Bq/m^3, Bq/m^3 EER), and those which denote cumulative exposure to residents or mine workers (pCi/L-years, WLM, Bq/m^3 a, Bq/m^3 EER a, J hr/m^3). See Appendix A for equivalences.

Epidemiologic studies of miners have shown a strong and consistent dose-response relationship between lung cancer mortality and radon exposure (NCRP, 1984; NRC, 1988). The miner studies upon which the NCRP and NRC reviews and analyses were based spanned many years and many countries, including the United States, Canada, Sweden, and Czechoslovakia. They involved diverse types of underground mines including iron, tin, and fluorspar as well as uranium. Most of the study designs were "historical cohort," meaning that the population studied was classified according to past exposure history and followed forward in time for observation of health outcomes. Rates of specific causes of death were the health outcomes observed. The lung cancer rates of the mining groups were compared to those of the general male populations in their respective countries. Analogous case-control studies of miners were also conducted (Samet et al., 1984, 1989).

While these studies probably had reasonable accuracy regarding vital status and cause of death of subjects, exposure estimates were often less precise. Since radon concentrations in mines usually had been measured not for epidemiological purposes but for industrial hygiene and regulation purposes, overestimates or underestimates of typical exposures may have resulted. Smoking data on individuals were collected in some but not all of these studies. However, other possible causes of lung cancer, e.g., arsenic and other toxic exposures, have not been shown to be explanatory of the excess associated with radon. Mining cumulative radon exposures ranged from those representative of lifetime residential exposures to two orders of magnitude higher in some members of the US and Czech mining groups. Despite the wide range of cumulative exposures, a remarkable degree of consistency in specific dose-response has been observed in this body of research.

Furthermore, predictive models for the radiation dose to the lung under various scenarios suggest similar doses per unit of radon exposure in homes as in mines (NRC, 1988). Extrapolation of the miner data to the levels of exposure found in houses

suggests a substantial risk for residents of some homes (NRC, 1988; Harley, 1984; Radford, 1985; Klotz, 1986; Jacobi et al, 1987). However, there are some uncertainties in this extrapolation. This includes questions on the applicability of findings in working age men to women and to children. There also are questions regarding the extent of excess risk at the lower radon concentrations typically encountered in homes. Clarification of the degree of lung cancer risk from indoor radon has far-reaching economic and public health policy implications because of the vast public and private resources needed in order to identify and remediate residences with elevated radon concentrations. Therefore, estimates of risk from elevated radon exposures are required specifically for the residential setting.

Direct information on residential risk from radon has been very limited to date. Most reports involve only correlation or "ecological" studies, i.e., those comparing lung cancer rates and mean radon exposure or exposure potential in different geographical areas but without including any specific information on individuals (Hess et al, 1983; Letourneau et al, 1983; Stranden et al, 1987; Archer et al., 1987; Cohen et al., 1988). Because the results of such studies are sensitive to confounders such as smoking and to biases due to migration, strong conclusions cannot be drawn from those reports alone, particularly since their results have been conflicting.

Individual-based epidemiologic studies, especially case-control studies (because of their feasibility), are the design of choice for addressing public health policy questions.

Case-control studies of residential radon and lung cancer.

A number of small case-control studies have examined the association between lung cancer and housing construction, or between lung cancer and residential radon exposure. Six of these studies are summarized in Table 1, and are reviewed in detail below (Axelson et al., 1979; Edling et al., 1984; Svensson et al., 1987; Axelson et al., 1988; Svensson et al., 1989; Lees et al, 1987). Other studies, including some not yet

published, have been reviewed by Borak and Johnson (1989). In the review below, the exposure units used by the respective papers are quoted, although conversion to alternate units are also shown. (See Appendix A for brief descriptions, comparisons, and conversions of the various types of units used for radon exposure.)

(a) Axelson and his colleagues first conducted a case-control study which examined the association between housing type and lung cancer risk among rural residents age 40 and over in the Swedish counties of Ostergotland and Orebro (Axelson et al., 1979). Rural residents were presumed to have few significant industrial exposures and to have lived in the same houses for long periods of time. The study included 37 lung cancer deaths recorded from 1965 to 1977, and 178 controls selected from the death registers, excluding any cancer diagnoses.

The subjects' registered addresses were classified into three housing categories: wooden house without basements; brick, concrete, or granite houses with basements; and a mixed category. The age-and-sex-adjusted rate ratios were 1.7 for the mixed houses, and 4.8 for the stone houses with basements, relative to 1.0 for wooden houses without basements, showing a statistically significant trend.

Neither the actual lengths of residence in the houses nor the smoking habits of the subjects were known. Based on earlier Swedish studies which compared measurements of radon according to housing type, it was assumed that stone houses with basements had higher radon levels, but no measurements of radon actually were conducted in this study.

(b) Another Swedish study by Edling et al. (1984) was based on the primarily-rural population of Oeland, an island with a narrow strip of alum shale-containing ground which was associated with locally high levels of gamma radiation. The 23 cases included all registered lung cancer deaths age 40 and over from 1960 - 1979, who had lived in the same house for at least 30 years just prior to death. Apartment dwellers were excluded. The 202 controls were a random sample of all non-cancer deaths in the

same years, applying the same age and residence criteria. Smoking data were collected for all cases and for most of the controls.

The houses were classified according to three criteria: building material, (stone, brick, or plaster vs. wood); presence or absence of a basement; and whether or not the house was built on alum shale ground. The eight combinations based on these criteria were then grouped into three categories, the lowest being wooden houses without a basement on normal ground (see footnote b, Table 1). The age, sex, and smoking adjusted odds ratios were 1.2 for the middle category and 3.5 for the highest, relative to 1.0 for the lowest, again showing a significant trend in risk with housing type.

Radon measurements were conducted in 86% of the houses, which were then classified as <50 Becquerels per meter cubed (Bq/m^3) equilibrium equivalent radon (EER) [<0.0135 WL], 50-150 Bq/m^3 [0.0135-0.04 WL], and >150 Bq/m^3 [>0.04 WL]. The age, sex and smoking adjusted odds ratio for the middle exposure group was 2.3, and for the highest, 5.1, relative to the lowest exposure group (1.0), again showing a significant trend in risk.

(c) A third Swedish study (Svensson et al., 1987) included 292 female long-term residents of Stockholm who had been diagnosed from 1972-1980 with lung cancer, specifically the histologic subgroup "unspecified epithelial" (primarily small cell undifferentiated and large cell cancers). The 584 controls were long-term residents who were selected from the population registry at random, and matched to the cases by year of birth. No data on smoking habits of these subjects were obtained.

All of the addresses where subjects had lived up to 5 years prior to diagnosis of the case were classified as positive if they were located on the radon-emitting ground types[2] and if they were single family detached houses or multi-family houses in which

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[2] In Stockholm, ground types with increased likelihood of high radon emanation have been extensively mapped.

the subjects lived on the ground floor. Twenty-two of the case houses (7.5%) were positive, compared to 21 of the controls houses (3.6%), giving an odds ratio of 2.2.

To validate the exposure classification, all positive houses and a sample (n=110) of negative houses were selected for single grab sample measurements of radon daughters. Case addresses which had been classified as positive had the highest measurements, but these were not significantly higher than those for negative case addresses or for control addresses.

(d) An incident lung cancer case-control study of women in Stockholm county was conducted by Svensson et al (1989). For the 210 cases, two series of matched controls were used, one population-based (n=209), the other hospital-based (n=191). The latter were drawn from the same clinical department as the cases, but were found not to have lung cancer. Details on individual smoking histories (active and passive), diet (foods rich in vitamins A and C), and occupation were collected by interviewing the subjects.

As with some of the other studies, the exposure index was constructed by characterizing former residences by soil and house type and by testing a sample of those residences. The measurements were of a two-week duration and were conducted during the heating season.

Relative risks were calculated on the basis of cumulative exposure estimates, using years of occupancy and estimated radon concentration for each former residence occupied for two or more years. For subjects with estimated cumulative exposures over 4,500 Bq/m^3-years [24.3 WLM, or 121.5 pCi/L-years], the relative risk for all lung cancer was 1.8. The trend was strongest for women over 70 years old and for small cell carcinoma. The authors found a greater than additive lung cancer risk associated with radon exposure in combination with smoking.

(e) Axelson et al (1988) also studied 177 population-based cases who had died of lung cancer between 1960 and 1981 and 673 non-cancer deaths. Each subject had lived at only one address for the last thirty years prior to death. Exposure classification was

based on house-type (construction material, presence or absence of a basement, and underlying ground-types) and on measurements of a stratified sample of the residences. Individual smoking data were collected from next of kin.

A positive trend for lung cancer and radon was found for the rural but not for the urban subjects. Male and female subjects showed similar trends, with males having a slightly stronger association between lung cancer and radon. Among the rural subjects, "occasional" and passive smokers showed a stronger association than "regular" smokers.

(f) Another case-control study was conducted in Port Hope, Ontario, where many of the houses constructed since 1933 have been built on waste materials from a radium and uranium processing plant (Lees et al, 1987). The 27 cases with lung cancer, all diagnosed or deceased from 1969-1979, were Port Hope residents for at least seven years prior to diagnosis, and did not work in the uranium plant. The 49 birth-date and sex-matched controls were selected with similar residence and occupation restrictions from non-respiratory cancer registry files and from physicians' records. Smoking, occupational, and residential histories were collected.

Radon measurements of all residences in the town had been made in 1975. Comparing subjects with "non-zero" cumulative background-corrected exposures to those with background or "zero" values gave a non-significant smoking adjusted odds ratio of 2.4. A statistically significant relationship resulted from analyses of exposures on a continuous log scale, but the authors stated that the measurement precision may have only justified the classification by high vs. low exposure.

Cohort studies of indoor radon and lung cancer.

A retrospective cohort mortality study of radon exposure among residents of radium-contaminated neighborhoods in New Jersey was conducted recently by the NJDOH (NJDOH, 1988; Klotz et al, 1989). Although the numbers of residents (752) in the index houses limited the statistical power of the study design, a relative risk of 1.7 (95%

confidence interval, 0.83, 3.2), based on 10 lung cancer deaths, was observed among white males when compared to expected rates derived from the New Jersey general population. Females showed no excess, but only one case was found, while 1.5 was expected. Possible confounding by smoking and occupation could not be assessed. A cumulative exposure index in WLM was utilized; however, no dose-response gradient was observed.

In summary, all six of the case-control studies as well as the cohort study are suggestive of an association between residential radon exposures and lung cancer risk. However, most of the studies did not include actual measurements of radon in the houses of all subjects. Four of the case-control studies took smoking into account, but two used only a crude adjustment. It was clear to the NJDOH and to the other collaborating agencies that a large case-control study could address many limitations of the former reports. By extending a prior New Jersey-based female lung cancer study, it would be possible to include extensive data which had already been collected on smoking, diet, and occupation, all of which were potentially important influences on lung cancer risk. Thus, it was intended that the New Jersey radon study could help to resolve the vital questions for public health policy as to whether the findings for underground miners could be extrapolated to residential exposure settings. Consequently, the study could help guide public agencies and citizens on radon testing and remediation decisions.

NEW JERSEY RADON STUDY - METHODS

This radon study is an extension of a statewide population-based incident case-control interview study of lung cancer previously conducted among New Jersey women (Schoenberg et al., 1989; see Appendix B).

Original subjects: selection and data collection.

The original study cases included all female New Jersey residents who were newly diagnosed with histologically confirmed primary cancer of the lung from August 1982 through September 1983. For cases who were interviewed themselves, controls (frequency matched to cases by 5-year age groups and race) were selected during the same time period from New Jersey drivers' license files (age <65) and from Health Care Financing Administration files of persons enrolled for Medicare (age 65+). For cases with next of kin interviews, individually matched controls were selected from state death certificate files.

During the original study, personal interviews were completed for 994 (76.1%) of the 1,306 cases identified and for 995 (68.7%) of the 1,449 controls identified. Details on the reasons for nonresponse are summarized in Appendix B. Fifty-three percent of the interviews were conducted with the subjects themselves. The remaining interviews were conducted with the spouse (17%) or with other next of kin (30%), mainly daughters, sons and sisters.

The questionnaire included a lifetime brand-specific smoking history, information on smoking habits of other household members, lifetime residential and occupational histories, and a history of consumption of foods containing vitamin A. These data have been analysed in some detail, specifically with respect to lung cancer risk associated with active and passive smoking, occupational exposure, and diet (vegetable consumption), after adjusting for age, race, respondent type, education, and county of

residence. The distribution of all cases and controls with respect to these variables, as well as odds ratios estimated from these data, are also summarized in Appendix B.

Subjects and residences for radon substudy.

In order to collect data on radon exposures, the original female lung cancer study was extended, beginning in 1985. Based on the literature available at that time, a minimum 10 year period was initially assumed between relevant exposure to radon and diagnosis of lung cancer (see below, p. 14, for updates regarding this assumption). To allow for sufficient duration of exposure, and to remain within available budgetary resources for radon measurements, the extension study identified one New Jersey address at which each subject had lived the longest and for at least 10 years during the twenty-year period 10-30 years prior to case diagnosis or control selection (approximately 1953-1972).

The residential information which had been collected previously specified only the towns in which each subject had lived. Therefore, the subjects or their next of kin were recontacted in the extended study to collect information on exact street addresses during the period 1953-1982 and to identify an "index residence" which met the above residence criterion (10+ years, approximately 1953-1972). Interviewers were not aware whether the subjects were cases or controls.

For each index residence, the current occupant was identified and requested to participate in the measurement portion of the radon study. Sometimes the current occupant was the original study subject or a relative; frequently, however, the current occupant was a person who was not related to the study subject.

A comparison of those subjects from the original study who were included in the radon substudy and those not included is given in Appendix C.

Radon exposure data collection.

Radon study data collection at the index residences started in October 1986. At each residence, information was collected on the house construction and ventilation, including questions regarding any changes in construction which had occurred since the current occupant or his/her family had lived in the house (see Appendix D). These changes in construction have not yet been taken into account in the analyses presented in this preliminary report.

Four-day screening measurements of radon were made using charcoal canisters provided by and analyzed by the NJDEP (Parsa, 1986; EPA, 1987). More details on the charcoal canister methodology are presented in Appendix E. The charcoal canister measurements served two purposes: first, to provide a relatively quick screening measurement of radon for current residents, in case some had dangerously high levels which necessitated immediate remediation; second, to provide some back-up data, in case long-term measurements of radon (see below) were not completed.

Two charcoal canisters were installed in each index house by trained field staff between October 1986 and April 1987. One canister was usually placed in the basement or lowest living level of the house, the other usually in the master bedroom. The residents were asked to maintain "closed house conditions" (windows closed, outside doors closed when not in use). After four days of sampling, the residents closed and sealed the exposed canisters and mailed them to the laboratory for analysis. The laboratory staff were not aware of the case-control status of the houses. Standard quality control procedures for analysis of the canisters were used (see Appendix E).

The principal exposure measurements for this study were year-long measurements of radon using alpha track detectors (Type SF, Terradex Radon Detection Products, Glenwood, IL) (Alter & Fleischer, 1981). More detail on the alpha track detectors is also presented in Appendix E. Two alpha track detectors were installed in each house, generally in the same locations as the charcoal canisters. For about 15% of the

addresses, a third alpha track detector was paired with one of the first two as a quality control check on the measurement precision (See Appendix E). Alpha track detectors were retrieved from the houses after about one year, except at a small number of houses (29 out of 719, or 4.0%; see Appendix E for details) where a change in ownership necessitated retrieval of the detectors before 11 months of exposure.

The detectors were shipped to Terradex for processing in 12 batches beginning in September 1987. Most batches also included some quality control detectors which had been exposed to known concentrations of radon gas at the Environmental Measurements Laboratory (US Department of Energy, New York City), as well as some blank detectors which had not been exposed at all (see Appendix E, Quality Control). Terradex was not informed of the presence of the quality control detectors, which were prepared in such a way as to resemble the other detectors submitted with each batch. All alpha track detector results returned from the laboratory were reviewed carefully to verify that the correct exposure dates had been used in the calculation of radon concentrations. Two basement alpha track detector results were suspected as being artifacts because the high reported concentrations were completely inconsistent with the charcoal canister results and the living area alpha track results. All measurements at these two houses were repeated (the alpha track measurements only for three months); the earlier basement results were not replicated, and were deleted from the data set.

It was assumed that living area (non-basement) alpha track measurements would provide the best estimate of the year-round radon concentrations to which the subjects had been exposed when they were living in these houses. Therefore, the charcoal canister measurements were not used for analyses except to estimate the year-round living area radon concentration when no alpha track measurements were available.

Residents were informed of their charcoal canister results approximately 6-8 weeks after the measurements were conducted. To our knowledge, remediation efforts were undertaken at only one house as a result of these screening measurements (this was

taken into account by placement of two sets of alpha-track detectors for intervals of three months before and nine months after the change). All residents were contacted six to nine months after canister placement to determine if any significant changes in construction or remediation had occurred. Residents with screening results between 4 and 20 pCi/L had been advised to wait for the results of the alpha-track detectors before undertaking any remediation, in accordance with NJDEP guidelines at that time.

Duration of exposure to the radon concentration measured in the index house was estimated from the dates provided by the respondent (original subject or next of kin) in the residential history. Tax office records were used, whenever possible, to validate the residential histories provided by the respondents (See Appendix F).

Statistical methods.

Because of concern regarding the precision of the radon measurements, particularly at the low concentrations found in this study (see Appendix E), the distribution of radon measurements was considered as a categorical variable. Results were expressed as <1, 1-1.9, 2-3.9, and 4.0+ pCi/L. These cutpoints provide reasonably grouped frequencies for a log-normal distribution (Nero, 1985), and also conform to the exposure groups generally reported by the NJDEP. There were too few living area alpha track results above 8.0 pCi/L to separate this group. Some analyses were also conducted considering radon exposure as a continuous variable.

Standard non-parametric statistical procedures were utilized for comparison of the distributions of cases and controls (Snedecor and Cochran, 1967; Fleiss, 1981). All analyses ignore the age, race and respondent type matching in the original data; therefore, adjustments were made for these variables in the analyses. Odds ratios (OR: estimates of the lung cancer risk associated with radon exposure, after adjusting for other factors) and 90% confidence intervals (CI) (equivalent to one-sided 95% CI) were calculated using multiple logistic regression analysis (Breslow & Day, 1980), with the

microcomputer-based LOGRESS program (McGee, 1985). Log-linear trends in risk with increasing exposure were also calculated using logistic regression techniques, for a weighted categorical exposure variable or for a continuous exposure variable. The significance of trends in risk was evaluated using the model Z statistic for the trend term, with a one-sided p value of $\leq$ 0.05. The difference in the likelihood ratio statistics between successive models, evaluated as a Chi-square statistic, allowed for determination of the goodness of fit of the models (Breslow and Day, 1980).

Parallel categorical analyses were conducted to estimate Mantel-Haenszel OR and 90% CI (Mantel & Haenszel, 1959) and the Mantel Chi-trend statistic (Mantel, 1963). Because these results were essentially similar to the logistic regression results, only the latter will be presented.

Phase II of data collection.

A second phase of data collection is currently underway. Two factors led to this second phase. One was the publication, since 1985, of data from miner studies indicating a shorter period (five years) between relevant radon exposure and lung cancer than had been assumed previously (Howe, 1986; NRC, 1988). The other factor was an additional appropriation in 1988 by the NJ State Legislature providing funds for testing of additional residences of the original subjects.

To date, as part of Phase II, approximately 200 additional houses have had canister measurements and installation of alpha track detectors. These measurements will result in more complete exposure histories for some subjects already included in Phase I of the radon study, as well as the inclusion of additional subjects not in Phase I. The results of these Phase II measurements are not included in the analyses in this report.

The criteria for houses being measured in Phase II, and examples of they will affect inclusion in the study, are shown in Table 2.

RESULTS

Inclusion in the radon study.

The status of the original 994 female lung cancer cases and 995 controls in the current radon substudy was examined (Table 3) in order to determine the extent of any possible bias in the composition of the radon substudy population relative to the original study population. Cases and controls did not differ significantly with respect to their status in the substudy. However, slightly more controls did not have measurements at the index residences, particularly due to refusal by the current occupant to participate. More often than for cases, original control subjects were still the occupants of the index residences. Some of these controls, who had already spent considerable time being interviewed, did not want any further involvement with the study.

Analyses in this radon study include the 411 cases and 385 controls whose index residence was successfully tested for radon with alpha track detectors and/or charcoal canisters. In addition, most analyses also include the 22 cases and 17 controls whose index residence was an apartment above the second floor or a trailer, for whom radon exposures were estimated (see Appendix J). Therefore, a total of 835 subjects (433 cases, 402 controls) are included in the radon study.

Table 4 shows the distribution of these subjects by age, respondent type and race, and by active smoking (lifetime average number of cigarettes per day, total years smoked, years since smoking cessation, tar content of cigarettes smoked from 1973-1982), vegetable consumption, occupation, education, county of residence at diagnosis, and passive smoking (for non-smokers only: exposure to spouse tobacco smoke). These variables were considered as possible confounders in the analysis of any association between radon exposure and lung cancer. Odds ratios which were estimated for these variables in the original female lung cancer study are summarized in Appendix B. These

analyses confirm the importance of smoking as the most significant risk factor for female lung cancer (particularly, number of cigarettes per day and years since smoking cessation). In addition, age, occupation, vegetable consumption, and respondent type (i.e., differences in smoking-related odds ratios by respondent type) also contribute signficantly to the observed lung cancer risk.

More detailed analyses on the characteristics of those women included in the radon substudy, compared to those women not included, are presented in Appendix C. Overall, controlling for all the potential confounders, there was significantly greater participation for cases from the original study than for controls. Moreoever, radon study subjects were more often older, whites, either nonsmokers, light smokers, or ex-smokers, residents of counties with higher radon levels, and more highly educated. However, there were relatively few significant case-control differences between those included and not included, except among heavy smokers, who showed some unusual risk factor distributions (Appendix C).

Type of measurement results.

One or more alpha track measurements were completed for 719 (90%) of the 796 index residences tested in the radon study. One or more charcoal canister measurements were obtained for 788 (99%) addresses. The canister measurements were not used for analyses, except to estimate the year-round living area radon concentration when no alpha track measurements were completed (see below, p. 17).

The charcoal canister results were also used to determine how the sample of houses included in this study compared to other New Jersey houses. In Appendix G, the distribution of basement or lower floor charcoal canister measurements from the 788 houses tested in this study has been compared, by county, to the distribution of basement or lower floor charcoal canister measurements obtained in a statewide survey of 5,727 homes conducted for the NJDEP. There was relatively good agreement

between the NJDEP survey and the case-control study, once the results were population-weighted. However, the case-control study had significantly fewer houses with lower floor charcoal canister measurements at 20+ pCi/L. This difference is at least partially attributable to differences in sampling between the two studies (the population based case-control study included more urban residents) and differences in the ages of the houses tested (the case-control study houses were all at least 22 years old). See Appendix G for further discussion of these issues.

A more detailed description of the type of radon measurements completed is shown in Table 5. There were no differences in the distribution of measurement types between cases and controls. Living area (non-basement) alpha track measurements were completed for 664 of the index houses. This included 347 addresses with first floor measurements and 317 addresses with second floor measurements. There were no significant differences in the distribution of radon results between those houses with first floor measurements and those with second floor measurements (see Apppendix H).

Living area alpha track measurements were not obtained for 171 of the index residences. For 55 residences, only basement alpha track measurements were completed. For 77 residences, only charcoal canister measurements (usually, both basement and living area) were completed. No radon measurements were conducted at the 39 addresses which were apartments above the second floor.

Living area radon concentrations for these 171 residences were estimated. The procedures used for this estimation are described in Appendix J. The resulting estimates were less than 1 pCi/L for 159 residences (including all 39 in the apartment 3+ category) and 1-1.9 pCi/L for 12 residences. The living area radon concentrations werre not estimated as 2 pCi/L or higher at any of the 171 residences. Most analyses have been carried out using the complete data set, including the estimates. However, some analyses have been repeated on a data set which excludes the houses without actual alpha track measurements of living area radon concentration (see Appendix K).

Analyses of radon concentrations by case-control status.

Table 6 shows the distribution of year-round living area radon concentrations (measurements and estimates) for the 433 cases and 402 controls. Radon levels were less than 1 pCi/L for 666 of the subjects (79.0% of cases, 80.6% of controls), 1-1.9 pCi/L for 133 subjects (15.5% of cases, 16.4% of controls), 2-3.9 pCi/L for 28 subjects (4.2% of cases, 2.5% of controls), and 4+ pCi/L for only 8 subjects (1.4% of cases, 0.5% of controls). The total unadjusted odds ratios showed an increase in risk with increasing radon exposure.

Table 6 also shows the data within each of four smoking categories according to lifetime average daily cigarette consumption. The reference group for these odds ratios is lifetime nonsmokers exposed to radon at less than 1 pCi/L. The unadjusted odds ratios increased with radon exposure more for the light smokers (less than 15 cigarettes/day) and, to a lesser extent, for the moderate smokers (15-24 cigarettes/day). Paradoxically, the heavy smokers showed a pattern of decreasing odds ratios with increasing radon exposure. (Some possible selection biases and other factors related to this observation are discussed below on p. 42). For the lifetime nonsmokers, the pattern was inconsistent.

The right-hand column in Table 6 summarizes the unadjusted odds ratios for smoking in this subset of the original study. The odds ratios show a strong increase with increasing amount smoked. Smokers of about one pack a day had a greater than nine-fold increase in risk relative to lifetime nonsmokers.

Odds ratios adjusted by smoking. Table 7 again shows the unadjusted odds ratios for the association of lung cancer with radon in all subjects, as well as the odds ratio after adjustment for smoking (lifetime average number of cigarettes per day). There was some confounding by smoking, i.e., the adjusted odds ratios were different from the unadjusted odds ratios. The odds ratio estimated for 2-3.9 pCi/L decreased with adjustment, while the odds ratio estimated for 4+ pCi/L increased. Because of the

small numbers of subjects with radon levels in each of the upper two categories, the odds ratio was also estimated for the combined 2+ pCi/L category; this also decreased after adjustment. The smoking adjusted odds ratios for all subjects, for all radon exposure categories, were not statistically significant (i.e., the 90% confidence intervals included 1.0). However, the trend statistics still showed a pattern of increasing risk with increasing radon concentration at a borderline significance level (for weighted categorical analysis, p = 0.068; for continuous analyses, p = 0.089).

The heavy smokers showed a pattern of decreasing odds ratios with increasing radon exposure, which was opposite to the pattern shown by the light and moderate smokers. When the difference in the slopes was evaluated (Rothman and Boice, 1982), including all four smoking groups in the analysis showed significant heterogeneity (p = 0.035). Exclusion of the heavy smokers reduced the heterogeneity statistic to nonsignificance (p=0.36). Therefore, analyses were also conducted for all subjects except heavy smokers. The unadjusted and smoking adjusted odds ratios for these subjects are also shown in Table 7. The adjusted odds ratio for 4+ pCi/L and for 2+ pCi/L in all but heavy smokers were statistically significant. Moreover, there was a highly significant increasing trend with increasing radon concentration (for weighted categorical analysis, p = 0.008; for continuous analysis, p = 0.017).

Adjustment for other variables in addition to smoking. The odds ratios in Table 7 are adjusted only for the lifetime average number of cigarettes smoked per day. There are many other factors which contribute to lung cancer risk among New Jersey *women* (Table 4, see also Appendix B), as well as other subject characteristics which should be also be taken into account in the analyses. Table 8 shows the results of analyses in all subjects adjusting not only for the lifetime average number of cigarettes smoked per day, but also for that factor together with one or more of eleven additional factors [age, respondent type, race, vegetable consumption, high risk occupation, education, county of residence, number of years of cigarette smoking, numbers of years since

smoking cessation, and average tar content of the cigarettes smoked from 1973-1982, plus the interaction between respondent type and numbers of cigarettes per day (see Appendix B)]. In Table 8, the models are ranked by decreasing improvement in the fit of the model, as evaluated by the difference in the likelihood ratio statistics; the significance level for the improvement in fit is also shown in Table 8. Adjusting for occupation, years since smoking cessation, or average cigarette tar content had the greatest effect on the odds ratios and trend statistic; in these three models, the categorical trend statistic passed below the p=0.05 significance level.

Multiple risk factor adjustment. Given the small number of subjects at the higher concentrations of radon, it was considered inappropriate to adjust for all factors simultaneously. The logistic model which took into account the number of years since smoking cessation gave a much better overall fit to the data (improvement in the likelihood ratio statistic) than the model which considered duration of smoking, and gave a slightly better fit than the model which considered cigarette tar content. Therefore, in further stepwise logistic regression modeling, only the smoking cessation variable was used (along with lifetime average number of cigarettes per day). Adding the age and occupation variables to those two smoking variables gave further, significant improvement to the fit of the model. The odds ratios (90% CI), trend statistics, and likelihood ratio statistics for this model are shown in Table 9.

A further model which added respondent type and the interaction between respondent type and cigarettes smoked per day also gave significant improvement in fit, and is also shown in Table 9. A final model which added vegetable consumption, respondent type and race, but excluded the respondent type interaction terms is also shown. There is no further improvement in fit for this model. For all three of these models, the overall results were similar: There was a pattern of increasing odds ratios with increasing radon level which was statistically significant (considering the categorical trend variable) or marginally significant (considering the continuous trend

variable). The odds ratios at the 4+ pCi/L level was statistically significant for two of the three models (the lower 90% confidence limit was 1.0 or greater).

Some multivariate analyses were also conducted examining the contribution of additional interaction terms (respondent type*radon, radon*smoking, respondent type*radon*smoking) to the fit of the model. Inclusion of the radon*smoking terms gave a significant improvement in fit; however, this has already been considered by conducting some analyses excluding heavy smokers. Additional interaction terms gave no further improvement in the fit of the models.

Analyses excluding heavy smokers. Similar analyses were conducted excluding heavy smokers and are summarized in Table 10. The logistic model which took into account cigarettes per day, number of years since smoking cessation, age, and occupation gave a significant improvement to the fit of the model (compare to Table 7). Addition of the respondent type and respondent type*smoking interaction terms gave a marginal improvement in the fit of the model. The model which added vegetable consumption and race but excluded the interaction terms gave a slightly greater improvement in fit. For all three of these models, the overall results were essentially the same: In the subgroup excluding heavy smokers, there was a pattern of increasing odds ratios with increasing radon level which was highly significant. In addition, the odds ratios at both the 4+ pCi/L level and the 2+ pCi/L level were statistically significant (the lower 90% confidence limit was 1.0 or greater).

Analyses for heavy smokers only. A few analyses were conducted for heavy smokers alone, in order to determine the magnitude of the negative trend in risk observed. The 2-sided p value for the negative categorical trend term in the unadjusted analysis was 0.120. Adjusting for age, occupation, respondent type, education, time since smoking cessation, and vegetable consumption reduced the magnitude of the negative trend so that the 2-sided p value was 0.360.

Analyses using a logarithmic continuous exposure variable. The categorical analyses group the exposure variable as on a logarithmic scale, whereas the continuous variable analyses have utilized an untransformed variable. A few analyses were also conducted using a logarithmic transformation for the continuous exposure variable. Analyses were conducted for all subjects, adjusting for cigarettes smoked per day, time since cessation, age, occupation, respondent type, and the respondent type*smoking interaction (comparable to the second model in Table 9). The Z statistic for the logarithmic trend term was 0.50, with a one-sided p value of 0.309. For all subjects excluding heavy smokers, a similar analysis (comparable to the second model in Table 10) yielded a Z statistic of 1.04, with a one-sided p value of 0.149.

Histologic type. Analyses were also conducted according to the histologic type of the case. Table 11 shows the distribution of all cases by histologic type (and controls) by year-round living area radon concentrations. Of the six cases with radon levels at 4+ pCi/L, three were small cell carcinomas; the remaining three included one squamous cell carcinoma, one adenocarcinoma, and one other histologic type. Of the 24 cases with radon levels at 2+ pCi/L, seven were small cell carcinomas and eight were adenocarcinomas; the remaining nine included three squamous cell carcinomas, three large cell carcinomas, and three other histologic types. All histologic types except squamous cell carcinoma had a greater percentage of cases with elevated radon exposures (2+ pCi/L) than did the controls.

Table 12 shows the adjusted odds ratios for the association of each histologic type of lung cancer with radon. The pattern of increasing odds ratios with increasing radon level was found for all histologic types, with the possible exception of squamous cell carcinoma. Only large cell carcinoma showed a statistically significant trend in odds ratios for the weighted categorical analysis ($p = 0.027$) but there were no cases exposed at the 4+ pCi/L level. The odds ratios was significant for small cell carcinoma at 4+ pCi/L (OR=13.2; 90% CI = 1.5, 118.2). The odds ratios was also significantly high for

"other histologic types" at 1-1.9 pCi/L (OR=2.1, 90% CI = 1.2, 3.8), but insignificant at higher radon levels.

Passive smoking. Analyses were also conducted taking into account passive smoking exposure by lifetime nonsmokers (see Appendix B for background from the orginal case control study). Table 13 shows the distribution of the 274 nonsmokers (61 cases, 213 controls) by year-round living area radon concentrations, as well as by exposure to spouse tobacco smoke (no exposure, exposure to spouse cigarette smoke, exposure to spouse tobacco smoke only from pipes and cigars). Adjusting for exposure to spouse tobacco smoke had very little effect on the odds ratios estimated for radon exposure among nonsmokers, or on the trend statistic in this subgroup.

Similar analyses (not shown) were also conducted considering exposure to tobacco smoke from any household member, not just the spouse. Adjusting for any household tobacco exposure had no effect on the odds ratios estimated for radon among nonsmokers. Therefore, neither of the nonsmoker-passive smoking exposure variables were considered in the overall model for all subjects.

Analyses of cumulative radon exposures.

All of the analyses described above have considered only the radon concentration measured in the living area of the index residence. The number of years of residence at the index address had not yet been taken into account. **A cumulative exposure index multiplies the radon concentration by years of residence.** In the development of the cumulative exposure index used in these anslyses below, several assumptions have been made:

(1) A minimum period of five years since relevant radon exposure has been assumed, rather than ten years, making the exposure period of interest the years from 5-30 years prior to case diagnosis or control selection. This assumption is based on the

publication, since 1985, of data from miner studies indicating a shorter time period between radon exposure and lung cancer incidence (Howe, 1986; NRC, 1988).

(2) Based on the median radon concentration for control subjects in this study, a minimum exposure of 0.6 pCi/L has been assumed for each year during the index period when a subject lived in a house other than the index residence, because these other houses were not tested for radon in Phase I.

The resulting cumulative exposure distribution has been divided into subgroups of <25, 25-49, 50-99, and 100+ pCi/L- years, corresponding to <5, 5-9, 10-19, and 20+ WLM (assuming 50% equilibrium and 80% occupancy). Each level represents the equivalent of 25 years of exposure at <1, 1-1.9, 2-3.9, or 4+ pCi/L, respectively.

Table 14 shows the distribution of cumulative radon exposures for the 433 cases and 402 controls. Cumulative exposures were less than 25 pCi/L-years for 701 of the subjects (83.4% of cases, 84.6% of controls), 25-49 pCi/L years for 108 subjects (12.9% of both cases and controls), 50-99 pCi/L years for 21 subjects (2.8% of cases, 2.2% of controls), and 100+ pCi/L years for only 5 subjects (0.9% of cases, 0.2% of controls). The unadjusted odds ratios showed an increase with increasing cumulative radon exposure, with the greatest increase for the small numbers of subjects with 100+ pCi/L-year exposure.

Table 14 also shows the distribution of the cases and controls within each of the four smoking categories according to lifetime average daily cigarette consumption, and the odds ratios relative to nonsmokers with <25 pCi/L-years exposure. The pattern of increasing odds ratios with increasing cumulative radon exposure was strongest for light smokers and, to a lesser extent, for moderate smokers. The pattern was inconsistent for both lifetime nonsmokers and heavy smokers.

Table 15 shows the odds ratios among all subjects after adjustment only for smoking (lifetime average daily cigarette consumption) or after adjustment for lifetime average daily cigarette consumption, years since smoking cessation, age, occupation,

respondent type, and the respondent type*smoking interaction. Odds ratios were statistically significant for exposures of 100+ pCi/L-years. The categorical trend statistic showed a marginal pattern of increasing odds ratios with increasing cumulative exposure ($p = 0.090$).

Table 15 also shows the adjusted odds ratios among all subjects excluding heavy smokers. None of the cumulative radon exposure categories had odds ratios which were statistically significant. However, the trend statistics showed patterns of increasing odds ratios with increasing cumulative exposure which were statistically significant ($p = 0.029, 0.030$).

Histologic type. Analyses were also conducted according to the histologic type of the case. Table 16 shows the distribution of all cases by histologic type (and controls) by cumulative radon exposure. Of the four cases with cumulative radon exposures at 100+ pCi/L, two were small cell carcinoma, one squamous cell carcinoma, and one adenocarcinoma. Of the 16 cases with cumulative radon exposures at 50+ pCi/L, six were small cell carcinoma, two squamous cell, four adenocarcinoma, two large cell, and two other histologic types.

Table 17 shows the adjusted odds ratios for the association of each histologic type of lung cancer with cumulative radon. A pattern of increasing odds ratios with increasing cumulative radon exposure was found to varying extents for all histologic types except for squamous cell carcinoma. The pattern was strongest and most consistent for the undifferentiated histologic types, including small cell, large cell, and other types. Only the odds ratio for small cell carcinoma at 100+ pCi/L-years and for other histologic types at 25-49 pCi/L-years were statistically significant.

Relative risk coefficients.

In order to compare the results of this study with those of others, particularly among miners, the continuous cumulative exposure analyses were used to calculate the

increase in risk per pCi/L-year, and the corresponding increase in risk per WLM. This result is usually called the "relative risk coefficient" and is expressed as a percentage increase in risk per unit of exposure. The results of these calculations are shown in Table 18. For comparability with other studies, results are shown not only for all subjects but also for all smokers and all nonsmokers. The relative risk coefficient for all subjects was 3.4% (90% CI: 0%, 8.0%). Only the relative risk coefficient for all subjects excluding heavy smokers was statistically significant (5.9%; 90% CI, 0.7%, 11.2%). Table 18 also shows the calculated relative risk coefficients for all subjects by histologic type, which range from a low of 0% for squamous cell carcinoma to a high of 6.7% (0, 17.4%) for large cell carcinoma.

DISCUSSION

In this section, the results of the first phase of this case-control study of radon and lung cancer among New Jersey women are discussed with regard to inferences about causality which can be drawn, contributions to the body of knowledge about indoor radon and lung cancer, and public health policy implications.

As described previously, the study was undertaken to test the hypothesis that higher indoor radon and radon decay product exposures are associated with excess lung cancer risk. Validation or rejection of this hypothesis is important to citizens and public policy makers because of the considerable effort and resources which are being devoted to testing and remediating elevated indoor radon concentrations. Although the cause-effect link between radon and lung cancer is incontrovertible for the high concentrations which have been seen in the occupational setting, many have questioned the extrapolation of that link to lower radon concentrations usually seen in residences.

This study supports a radon-lung cancer link in residences, but some of its results must be interpreted cautiously for reasons which are described below.

Evaluation of causality: Guides to drawing inferences from epidemiologic data.

First, the results are considered in the context of how epidemiologists interpret statistical associations between an exposure and a health outcome and draw conclusions about a cause-effect relationship (Rothman, 1986).

Consistency with previous findings. In a specific study, an association between exposures and health effects supports the inference that the association may be causal if it is consistent with other research and if similar findings have been previously reported in other populations. As described in the Introduction, there is a vast body of evidence indicating that decay products of radon cause lung cancer. Our results are consistent with those of uranium and other hard-rock miners and with those of residential case-control studies already reported from other countries (See Table 1).

Biological plausibility and coherence. When there is an internally consistent body of knowledge which provides a biologically plausible basis for relating an exposure and hypothetical effect, there is more support for such an association to be interpreted as representing causation. There is ample information from human and animal observations and from radiation biology to predict **a priori** (that is, independently) that the alpha and other radiation emitted by radon and its decay products would be carcinogenic to the respiratory system when inhaled. These predictions are based on: (a) the highly damaging quality of alpha radiation, (b) the behavior of radon decay product particles in the respiratory system, and (c) the experimental and predicted radiation dose to the lungs resulting from typical and high radon concentrations in indoor air. All previous modeling, based on animal and human data, predicts that the radiation dose to the living cells of the lung lining from radon inhalation far exceeds the radiation dose to all other organs combined. The estimated dose to the human lung, even from typical indoor radon concentrations, is more than twice the dose typically absorbed by people outside of medical, unusual occupational or accidental circumstances (NCRP, 1984). Dosimetry models (NRC, 1988) suggest that radiation dose to dwelling occupants are

similar to those of miners for similar cumulative radon exposures, even accounting for differences of breathing patterns and particle characteristics between mines and homes.

Dose-response issues. Causality is supported when an exposure-effect relationship increases in strength with increasing exposure. Our study found that overall relative risks increased directly with exposure (Tables 7,9,10,15), whether intensity or cumulative exposure was considered. Statistical tests for trend were used to quantify strength of this dose-response relationship. The trend in our study was found to be strongest among light and moderate smoking subgroups (Tables 6,7,10) and, depending upon the exposure index used, for the undifferentiated histologic types, predominantly "small cell" and "large cell" (Tables 12,17). However, there were variations in dose-response among smoking groups, as discussed below.

Strength of association between health outcomes and exposure indices. In interpreting epidemiological data on the effects of exposure to an agent, a greater rate of disease or degree of biological response is interpreted as indicating a higher likelihood that an observed association is causal. The method we used to assess strength of association, or degree of risk, is the "relative risk" (RR) as estimated by the "odds ratio" (OR). The relative risk (the risk of lung cancer in the radon-exposed subjects divided by the lung cancer risk in the "unexposed") must be adjusted for numerous other variables, especially smoking and age, which are in themselves important predictors of lung cancer probability. A relative risk (or odds ratio) greater than 2.0 is often interpreted as indicating a strong association, and above 5.0, one that is extremely strong.

In the current study, relative risks for women in the groups with higher exposure intensity (2+ pCi/L) were 1.6 to 1.8, depending upon the number of variables included in the adjustment used (Tables 7-9). That is, when controlling for other variables, the risks of lung cancer were 60% to 80% greater for women exposed to the range of 2.0 pCi/L to 11.3 pCi/L (on an annual basis in the living area) compared to the risks for background indoor concentrations of less than 1.0 pCi/L. Similarly, after making

conservative assumptions about the years not measured, the relative risks for women who accumulated over 50 pCi/L-years during the twenty-five-year exposure period under study were 1.3 - 1.4. That is, when controlling for other variables, the lung cancer risks were 30-40% greater for women exposed to the range of 50-155 pCi/L-years than women who accumulated the typical 25 pCi/L years (see Table 15). For the occasional household in New Jersey with extreme radon levels, such as 200 pCi/L, lung cancer risks are probably much higher, although not necessarily proportionally higher.

Specificity. A classic cause-effect relationship in communicable disease depends upon a unique microbe's association with a particular clinical syndrome. In environmental health, it is rare to find a disease caused by only one agent and lung cancer is no exception. Lung cancer in modern society is, of course, primarily due to cigarette smoking. However, occupation and diet are also important factors. For these reasons, the effects of smoking and other factors were carefully controlled in the analyses (See Tables 7-9,15).

Sequence (order of occurrence) of exposure and health effect. The long latency (that is, time between exposure and initial diagnosable effect) of lung cancer after its initiation is one of the factors which makes epidemiologic research on its causality so difficult. In contrast with correlation studies (see Introduction), the design of this study specifically addressed the estimated exposures of each individual subject from five to thirty years prior to diagnosis of the cases.

Internal consistency of results among subgroups. Causality is supported when all subgroups or strata of important variables which could confound the study show similar results. Therefore, the difference among smoking subgroups in our findings weakens our causal inferences. However, it should be noted that the subgroup of heavy smokers is particularly prone to other competing causes of death and may also be subject to certain selection biases (see below, pp. 30-32 and Appendix C).

Other considerations. The observed association between lung cancer and radon became stronger with more rigorous adjustment (Tables 7-9) and this fact strengthens the causal inference. However, when duration of exposure as well as intensity (i.e. cumulative exposure) was considered, the strength of the association decreased, thereby weakening the causal interpretation (Table 15).

Strengths and weaknesses of the study.

The issues in this section affect the validity of application of this study's findings to the general population of women in New Jersey, and, by implication, to people in other locations.

Health outcome data. Among the strong features of this study are the objectivity, validation, and systematic nature of the lung cancer data. As discussed earlier and in Appendix B, the original cases and controls were drawn systematically from the entire New Jersey population, and the cases were all validated through review of pathology reports and other medical records. Thus, health outcome misclassification should be infrequent or absent. As discussed below, however, the designation of specific histologic type was not independently validated, so that analyses by cell type may be somewhat affected by misclassification.

Possible selection biases. Although the cases and controls in the prior statewide female lung cancer study were population based, the residence criteria and the need to have the cooperation of both subjects and current occupants of former residences resulted in a reduction of the original study subjects by about 60% and could have introduced some biases into the current study. Such biases could have resulted in observing either a greater or lesser radon-lung cancer association. As described more fully in Appendix C, the women included in the radon study extension were not completely representative,with respect to certain factors, of those in the original lung cancer study.

(a) In general, **more cases than controls were included in the study** extension, particularly because control subjects who were still the current residents of the index houses tended to have higher refusal rates. This may have biased the results if the missing controls tended to have higher radon exposures. Future analyses using New Jersey geographic data on radon potentials as a surrogate for missing years may help to indicate the magnitude of any possible bias (see pp. 51-52).

(b) Both the cases and controls who were included in the study extension were **older** than those from the original study who were not included because of the residency criterion of at least 10 years in one house. Since many people tend to move more frequently at younger ages, this shift in age was expected. If the risks due to radon exposure are proportionally greater in older age groups, as has been suggested by some occupational studies (NRC, 1988; NCRP, 1984), our findings may be slightly exaggerated. Conversely, if radon-induced lung cancer is proportionally more prominent in younger women, the findings might slightly underrepresent the true hazard to the general population.

(c) For both cases and controls, the subjects in the study extension were **less likely to have had respondents who were next-of-kin other than spouses** (see Appendix C). For most variables, it is not known whether any bias in the findings could have resulted from differences in accuracy or completeness of data supplied by a relative other than a spouse. For smoking, a detailed discussion of potential biases is given on p. 42).

(d) The statistically significant **underrepresentation of non-whites** from the original study in the radon extension was also a function of the residence criterion. However, since a similar pattern resulted for both cases and controls, and since race was not an important predictive variable in either the multivariate analyses on the original data set (see Appendix B, Table B3) or the radon extension (see Appendix C2, C3), we do not believe that major bias was thus introduced.

(e) With regard to **educational level**, the representation of cases and controls included from the original study did differ. The cases included fewer women with less than eight years of school. Again from Tables B3, C2 and C3, that variable was not seen to have an important effect on lung cancer outcome when considered together with smoking and radon exposure, and was not included in the final models (Tables 9,10,15). However, the striking underrepresentation of cases with less years of schooling among the heavy smokers suggests that this subgroup may be particularly biased. This possibility is reinforced by an underrepresentation of controls with high vegetable consumption among the heavy smokers.

(f) With respect to **residence at diagnosis** the nonsmokers in the original study showed a significant risk associated with residence in the "low radon" counties, while heavy smokers showed a marginally significant risk associated with residence in the moderately low radon counties (see Appendix C). These observations, together with selective underrepresentation of "low radon" county controls among the heavy smokers, suggest that other as yet undetermined geographically associated risk factors might be operating to mask any slight radon effect in nonsmokers or heavy smokers.

(g) The design of the study did not allow the evaluation of effects of **residence mobility** per se on the likelihood of high radon exposure or lung cancer. Since relatively low mobility during the thirty years prior to diagnosis was a criterion for inclusion, any independent relation of lung cancer or radon concentration to mobility could have increased or decreased the observed association. Future analyses (see later section in this Discussion) will treat the issue of mobility with regard to other variables.

Exposure data. In contrast to most other studies of this type reported *previously*, **actual radon measurements were made in each and every index house except for apartments above the second floor.** The technicians who placed the detectors and the laboratory technicians who assayed them did not know the case or control status of any

detector. Residential duplicates, "spiked" samples, and blanks were used for quality assurance (See Appendix E).

Another strength of the study is the use of **year-long measurements** in living areas as the exposure index and the use of short-term measurements for screening, consistency checks, and as contingency measurements. Short term measurements under "worst case conditions" are generally used by citizens to screen their homes and by agencies to rapidly assess radon exposure potential and needs for service. "Worst case" conditions are: heating season, "closed-house", and floor closest to the underlying soil. Such screening conditions are appropriate for initiating decisions on whether further testing isneeded, but they are, by definition and intent, exaggerations of the radon concentrations inhaled by occupants of a dwelling on a year-round basis (see Appendix E). When "worst case" exposures are used to make quantitative risk assessments, the resulting risk assessment tends to underestimate the hazard per unit of exposure, and subsequent application of these unit risks to true annual average measurements result in risk assessments which are unrealistically low.

Further, year-long measurements have the advantage of smoothing over the daily and seasonal radon fluctuations due to meteorological variations, varying proportions of time spent at home, and varying amounts of time spent on each floor of the house.

As discussed above (also see Appendix J), certain index houses did not have long-term living area measurements completed. The relationships generated between the set of contingency samples (short-term canister measurements) and long-term alpha tracks, on both lowest and upper floors, appeared stable enough (see Appendix E,Tables E4a, E4b) to enable us to confidently assign an exposure interval for missing measurements. When analyses were conducted without the estimated values, the results agreed with those which included the estimates although there were inevitable losses in statistical power (Appendix K, Tables K1-4).

Because residency data were collected from respondents and validated through tax records, a **cumulative** radon exposure index could be constructed. The cumulative index was utilized in addition to the radon **intensity** concentration as an exposure variable. A cumulative exposure index has generaly been used in mining studies (NRC, 1988; Howe et al., 1986) and has been seen to vary directly with risk in a dose-response gradient.

A necessary weakness of any retrospective study of this type is the **collection of exposure data in the present time when the exposure of interest actually occurred in the past.** The factors which cause daily and seasonal radon fluctuations are not likely to have significantly affected the measurements we made, but there is a possibility that changes in house construction, heating, ventilation, occupants' activity, and hours per week of occupancy could cause major inaccuracies in the exposure estimates.

Equally important are the **"missing years" of observation.** The distribution of years not accounted for by the measurements leaves the possibility that high or low radon exposure in the unaccounted years could have caused significant exposure misclassification for some subjects. This is especially true for those subjects (10.0% of cases and 10.9% of controls) for which less than half of the 5-30 year index residence period was accounted. "Phase II" measurements currently in progress are expected to address this issue.

Remaining lifetime years (beyond the 25-year index residence period) may also contribute radon exposure which are important 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.

House occupancy pattern (rooms in which subjects spent most time) is a factor which could not be addressed in this study; the proportion of time spent at home per year may be addressed by later analyses of time and place of occupation (see pp. 52-53).

Radon measurements in the lower ranges, such as were found in this study, have a greater probability of **random misclassification.** Random exposure misclassification tends to bias results so as to reduce the observed difference between groups. Since a significant dose relationship was, in fact, found for the main study hypothesis, an erroneous conclusion probably did not result from any random exposure misclassification caused by the above factors. We have no reason to believe that there was any systematic exposure misclassification, especially between cases and controls. Furthermore, the distribution of measurement results are overall in excellent agreement with those of NJDEP after adjusting for population distributions (see Appendix G).

The quality control results described in Appendix E suggest that the precision of the measurements may not be sufficient to analyze all of the data on a continuous scale, particularly given the low levels of exposure prevalent in this study. Therefore, categorical analyses (<1, 1-1.9, 2-3.9, 4+ pCi/L) were used predominantly and the continuous variable analyses of exposure should be interpreted very cautiously. On the other hand, cumulative exposure, which includes the additional component of residence duration, may be less sensitive to the imprecision of the measurement, and the continuous cumulative exposure analyses may be more reliable. **This is the justification for presenting results of both continuous and categorical analyses in this report.**

Potential confounders. Confounders are factors which can selectively influence both exposure observations and health effect observations. They may thereby distort an observed association between the exposure effect of interest.

The effect of smoking, by far the most important determinant of lung cancer, is distinguished from the effect of radon in this study by stratifying in some analyses by smoking and by controlling for smoking in multivariate analyses using logistic regression. Smoking is important when evaluating radon as a lung carcinogen in women as shown in this study by the differences between the relative risks for smoking-adjusted versus non-adjusted analyses. These observations also underscore the importance of

individual-based study designs such as case-control, in contrast to "ecological" or correlation studies (see the Introduction). Ecological studies are unable to examine and control for smoking differences among individuals and geographical areas. Differences in the radon-related risks by smoking, combined with the overwhelming risk of smoking itself, may mask any overall radon effect in the general population. (A more detailed discussion of the smoking-radon interactions we found, and their possible interpretation appear later in this Discussion).

Among the strengths of this study were the detailed dietary and occupational histories and complete data on various demographic characteristics such as educational level. As can be seen from Table 7, demographic and socio-economic factors such as race and educational level were not important in modifying the effect of radon on lung cancer after smoking was taken into account. Occupation, dietary factors and respondent type were included in the final multivariate models along with smoking parameters and age.

Numbers of subjects and measurements; Statistical power. The small number of residences which had high annual exposure measurements (especially the very few above 4 pCi/L) limit the inferences which can be drawn because of the statistical instability in those categories. **Therefore, our results must be interpreted cautiously.** However, the total number of subjects for whom residential measurements were made exceeds most previous individual-based residential studies and is a major strength of the study.

It should be noted that this study was not designed on the basis of prior statistical power calculations. This usual practice was not followed because of the particular history of the study as an extension of a prior one (see Methods and Appendices B and C). The statistical power to find an association of lung cancer and radon was extremely limited. Considering the annual upstairs radon distribution found in the controls of 3% above 2 pCi/L (Table 6), we would have predicted that this study could only detect a risk of 2.3 or greater with 80% statistical power using a "one-tailed"

test for statistical significance at the 0.1 probability level. This latter probability denotes the frequency of accepting a radon effect when, in fact, the observed difference arose by chance and sampling error only (Schlesselman, 1974). Nevertheless, a relationship has been supported by the data, although the possibility cannot be ruled out that confounders which could not be controlled for are producing the observed association. Extra caution should be used in interpreting all findings on subgroups of the study population such as smoking categories and histological. types, since multiple statistical tests on subgroups increase the probability of artifacts of "statistical significance" due to chance alone.

Risk per unit of exposure.

In comparing miner studies and especially in deriving from them predictions about the degree of hazard from indoor radon exposure under various scenarios, the concept of excess risk per unit exposure of radon decay product has been a convenient method for assessing the potency of radon as a carcinogen.

Risk models: Absolute (attributable) and relative risk. Incremental risks from radon exposure can be understood and expressed in two different ways:

(1) as the **additional** (absolute) increment, or **number of cases beyond those that would have occurred** in a given population without the extra exposure. This is "attributable risk".

(2) as the **proportional** (percent) increase in the underlying lung cancer risk. This is called "relative risk". Its calculation requires knowledge of the baseline risk.

The former expression is generally easier to describe and understand; however, it cannot be estimated directly from case-control studies. The current scientific consensus is that in radon carcinogenesis a relative risk model is appropriate, especially with regards to age. It has also been suggested that both relative and attributable risk models may be appropriate for different populations, depending upon age and smoking

status (Archer, 1988). The roles of gender and smoking in risk from radon have yet to be resolved. The effect of a pure relative risk process would be to produce the largest number of excess cases in smokers, in people between 55 and 65 years old, and in males. So far, all of these patterns have been generally observed where such comparisons were possible (NRC, 1988).

Estimates of risk per unit of exposure from miner studies. Excess risk per unit dose has historically been expressed in one of two ways using "working level month" (WLM) as a unit for cumulative exposure. Most unit risk estimates from the miner studies assume a linear dose-effect relationship at lower radon intervals and fall between these intervals:

Attributable (absolute) risks:

a) $\frac{\text{5-50 excess lung cancer cases}}{\text{million people per year per WLM exposure}}$

b) $\frac{\text{100-800 excess lung cancer cases}}{\text{million lifetimes per WLM exposure}}$

Relative (proportional) risks:

a) $\frac{\text{1\% - 4\% increase in lung cancer}}{\text{WLM exposure}}$

b) "Doubling dose" between 25 WLM and 100 WLM (i.e., cumulative exposure of 25-100 WLM would result in doubling of baseline risk.

The National Research Council's "BEIR IV" report (NRC, 1988) analyzed a compilation of underground miner data and calculated a relative risk of about two percent per WLM, when considering certain additional factors such as time interval since exposure. It should be noted that the above estimates were all derived from males only.

Limits of extrapolation using risk per unit of exposure. A simple proportionality of excess risk per unit of radon dose cannot be realistically applied without some bound. Without some modification, over 100% risk could be calculated for some of the extremely high occupational or residential exposures which have been documented,

clearly an absurdity. In fact, groups of miners who had accumulated in excess of about 1,000 WLM did **not** show as steep an increase in lung cancer risk per unit of exposure as did their counterparts with lower cumulative exposures, indicating some plateauing of effects (NCRP, 1984). In the current study, however, and in the overwhelming majority of residences, such huge exposures are not seen. There is also evidence from the miner studies that low dose rates are associated with **greater** risks per unit of dose (Sevc et al., 1988).

Risk per unit of exposure from previous residential studies. Previous residential study estimates have been quite consistent with the occupational estimates (Edling et al., 1986; Svensson et al., 1989; Axelson et al., 1988). However, as reviewed in the Introduction, most former residential studies did not include enough measurements to generate unit risk estimates.

Risk per unit of exposure yielded by the current study. The coefficients which were derived from this study are relative risks per unit of radon exposure. They are calculated directly from the odds ratios found in Table 18 for the trends of lung cancer risk in relation to cumulative radon exposure using the continuous exposure variable. The slope of the excess relative risk (RR - 1.0) per pCi/L-yr. was used to calculate the equivalent percentage increase in risk per WLM (using the equivalents in Appendix A).

In Table 18, the estimates of the percentage increase of relative risk of lung cancer per WLM are given for various smoking categories, in order to permit comparison with former studies. The relative risk per unit dose for the entire study population combined was 3.4% (0.034) per WLM (90% C.I. 0%,8.0%), somewhat in excess of the BEIR IV coefficient, but definitely within the range of underground miner studies generally, especially considering the sampling variability of our estimates. The coefficient for all smokers combined had about the same magnitude. Non-smokers had the smallest risk coefficient, 2.0% (90% C.I. = 0%, 10.2%). The coefficient for all

subjects excluding heavy smokers was 5.9% (0.7%.,11.2%) and was statistically significant.

Attributable or "absolute risk" coefficients were then derived from the unit relative risks when applied to typical lung cancer incidence rates. For women, current annual lung cancer incidence in New Jersey is approximately 300 per million, and for men, 900 per million. Therefore, an attributable risk coefficient per WLM - person year derived from this study is about 3.4% x 300, or about ten excess cases per million person years per WLM for women. If the same coefficient were applied to males, about 30 excess cases per million person-years per WLM would be estimated. These attributable risk coefficients are also within the ranges previously found in mining studies.

Issues of smoking interaction, histology, age, and gender interaction.

Previous epidemiologic studies of radon exposure in miners, and residents and experimental studies in animals have produced differing findings with regard to the interaction of smoking and radon (Cross et al., 1982; Axelson and Sundell, 1978; Whittemore and McMillan, 1983; Damber and Larsson, 1982; Samet et al., 1989). The interactions suggested by those reports have ranged from less than additive to multiplicative. It is possible that the roles of age and radon dose (intensity and rate) may be responsible for the different observations (Archer, 1988).

Given the small number of nonsmokers among the cases, the generally low radon exposures, and the prior estimates of the risk per unit of radon exposure, we would not have expected to observe a large radon effect on lung cancer in this subgroup. Therefore, we do not interpret the absence of an observable trend in the nonsmokers as inconsistent with former studies. Consequently, it is prudent and consistent with all other available evidence to continue to advise nonsmokers to avoid unnecessary radon exposure.

Since cigarette smoke contains a mixture of potent carcinogens and is believed to act as both an initiator and a promoter of cancer (Van Duuren, 1976), it is plausible that a combination of both radon and cigarette exposure might produce a carcinogenic effect in excess of either exposure's single effect. However, the net result of the complex changes which cigarette smoke exerts on the respiratory tract could plausibly increase and/or decrease the alpha radiation dose to the lung lining cells (Axelson and Sundell, 1978; NRC, 1988).

In former radon studies, characterization of smoking history has rarely been conducted with the detail we used in the current investigation (See Appendix B). Most prior distinctions have been between smokers, vs nonsmokers and exsmokers, regular vs "occasional" smokers, or between those who smoked more or less than ten cigarettes per day (Svensson et al., 1989).

Some evidence from animal and occupational investigations are not inconsistent with a less than additive effect of radon in combination with heavy smoking. Experiments with beagles which suggested protective effects of tobacco smoke for radon-induced lung cancer used very high tobacco doses (Cross et al, 1982). There is also circumstantial evidence that many miners who smoked would have been classified as light or moderate smokers. According to Archer et al. (1973), most lung cancer cases among their cohort smoked a pack per day or less, and the authors state that only a 49% increase in lung cancer was attributable to smoking among this cohort (compared to approximately 80-90% in the general population). In addition, it is plausible that there would be a selection against heavy smokers continuing to work as miners and/or to survive until the ages at which lung cancers usually appear (sixth to seventh decades); other lung diseases and heart diseases tend to decrease fitness for mining and to increase early death.

In our study, the small number of nonsmokers hampered the ability to analyze the effect of active smoking or passive smoking on radon risk. In addition, as described in

Appendix C, there could have been misclassification of smoking by non-spouse next-of-kin or a selection bias with regard to geography for heavy smokers or for nonsmokers. Because of the loss of subjects due to the residence criterion and to and other reasons, the radon study was no longer strictly population-based as was the prior lung cancer study.

It is important to consider that our own sample of heavy smokers might be biased in some ways, particularly with respect to education and diet (see Appendix C). Heavy smokers were the most under-represented subgroup of the prior lung cancer study in the radon study extension. We surmise that smokers tend to have smoking spouses. Women who were heavy smokers would therefore be more likely to be widowed than women who were nonsmokers, and might tend to relocate after becoming widowed. Also, smokers themselves are more likely to die of smoking-related disease other than lung cancer. All these phenomena would tend to reduce the proportion of heavy smokers in our prior study who met the residence criteria for radon measurements or might make it more difficult for us to find the appropriate former residence and gain access to it. In addition, we have already observed differences in smoking-related risk by respondent type which are possibly related to misclassification of smoking by next-of-kin respondents (Schoenberg et al, 1989; see Appendix B). The possibility of a case response bias, and resultant misclassification even by subject respondents is purely speculative but cannot be ruled out. Possible misclassification of smoking must be taken into account before accepting the smoking-related differences in radon risk suggested by these results.

It is therefore unclear what phenomena are responsible for specific patterns seen in this study, i.e., an apparent multiplicative interaction of radon with light smoking, an apparently negative interaction with heavy smoking, and an inconsistent effect in nonsmokers (no trend). It is important for public health practice that we resolve this issue, and it is hoped that our own ongoing work, as well as forthcoming reports from

other states and from other countries, will eventually clarify the interpretation of the results presented here.

It is also essential to emphasize that **smoking is by far the most serious risk factor for lung cancer**, and, in particular, that **smoking about one pack per day increases the risks fourteen-fold in women who currently smoke** (Appendix C; Schoenberg et al., 1989) compared to the overall less than two-fold risk of radon found in this study.

Histology. Particular cell or histological types of lung cancer which are differentially associated with radon exposure could have medical significance for early diagnosis and treatment. In early analyses, underground miner studies suggested that small cell lung cancer was the major histologic type induced by radon (U.S.D.O.E., 1988, NCRP, 1984) Later analyses, however, indicated that squamous cancer (also called epidermoid) was also found in excess in uranium and other miners, and that even other histologies, such as adenocarcinoma and large cell cancer, were elevated, although less dramatically (NCRP, 1984; NRC, 1988; U.S.D.O.E., 1988).

One mining study (Sevc et al., 1988) found that small cell excesses predominated at lower cumulative radon exposures while the increase in squamous cancers continued beyond 500 WLM. It is possible that the much lower concentrations in dwellings would produce a histology pattern at variance with that seen in male workers exposed to much higher levels. Since females tend to have a somewhat different distribution of lung cancer cell types, it is of particular interest to observe the histological findings of the present study. Smoking is a stronger risk factor for squamous and small cell carcinoma, but it also causes adenocarcinoma and large cell lung cancer.

The cumulative exposure results (Table 17) suggest that the undifferentiated histologic types including small cell and large cell lung cancer are most closely associated with radon exposure while squamous cell cancer is least affected. However, analyses of these subgroups are unstable due to the small numbers at the higher radon

exposures. Furthermore, histologic type was not validated by independent pathology review in this study, so that misclassification of histology is possible (Schoenberg et al., 1989). Our findings need to be corroborated by forthcoming residential studies before they are judged to be definitive.

Age and gender comparisons. Since underground miner research was conducted on working age males, only residential studies can address radon-related risk comparisons of adults vs children, as well as males vs females. The design of this study did not permit the consideration of effects of exposure during childhood, but our focus on females, for reasons given earlier, permits tentative gender comparisons on degree of risk, smoking interaction, and histological types, among other issues.

Males have greater baseline lung cancer risks than females. In extrapolating from male-based mining studies to females, our initial hypothesis was that we would find similar **proportional** lung cancer risks but lower **absolute** (attributable) risks in females compared to males. This prior expectation is based on the studies on A-bomb survivors (NRC, 1988) former residential radon case control studies (Edling et al., 1984; Axelson et al., 1988;) and our own former (not statistically significant) observations (NJDOH, 1988). Application of the present type of design to both sexes in future studies will be needed to evaluate the degree to which the current findings can be extrapolated to males.

Implications of exposure findings.

The annual exposures found in the study group were lower than had been expected on the basis of a statewide survey (NJDEP, 1989) which included primarily screening measurements. Several factors contribute to this difference:

1) The ratio of annual average exposures (using alpha track detectors in living areas) to "worst case" screening concentrations (using charcoal canisters in basements in winter under "closed house" conditions) decreased as the screening concentrations increased (see Appendix E).

2) Previous estimates of the "annual average" radon concentrations (see data summarized by Nero et al, 1986) have often been calculated as the average of two screening measurements, one in the winter and one in the summer. This may overestimate the true annual average concentration.

3) The houses in this study also tended to have lower screening measurements; only one house had a basement screening measurement of 20+ pCi/L. The houses may not have been typical of all New Jersey houses, e.g., they were at least 25 years old due to the residence criterion (dwellings occupied by our cases and controls at least 20 years prior to 1982-1983 when the subjects were identified). It is possible that new houses tend to be "tighter" and tend to have higher radon concentrations because of less ventilation and to be built in areas of New Jersey with higher radon potential (see Appendix G). Additional study subjects and index houses which are being added in Phase II of the study may be newer and "tighter" and may have higher radon levels.

4) The geographically-based screening study conducted for NJDEP (1989) was not population-weighted (see Appendix G). That is, dwellings in rural areas were more likely to be tested than dwellings in more urban areas with higher population densities. In contrast, the original cases and controls in our study were derived from a population-based sample with higher proportions of subjects from low radon counties. The exposures in urban areas in New Jersey are likely to be lower, partially due to a greater probability of residence in high-rise apartments. In addition, even detached houses in urban areas have lower radon levels than detached houses in suburban or rural ares (see Appendix G). This may be related to differences in the underlying geology of the areas which happen to be urban in New Jersey. Consequently, the exposure distribution of this study is likely to be much lower than that based on a geographically-stratified sample.

There are several important implications of these exposure findings:

(a) The results support the **use of follow-up tests rather than screening tests when making remediation decisions.** Such procedures are already advised by the USEPA, NJDEP, and NJDOH, but are not necessarily understood by the public.

(b) If our findings concerning the ratio of average annual to worst case radon concentrations are confirmed by other studies, remediation may not be necessary for as many dwellings in the state (and the nation) as previously had been believed based on the distribution of measurements.

Population attributable risk. Estimates of average exposure as well as risk per unit dose are used to calculate total population attributable risks from radon. It should be noted that the relative risk coefficients (excess risk per unit of exposure) yielded by this study are slightly higher than those extrapolated from most occupational studies. However, if the differences between screening measurements and the actual annual exposures in the living areas are corroborated, estimates of the number of excess lung cancer cases due to radon in the state and in the nation may decrease. Testing and remediation recommendations to individuals and agencies located in areas with high radon potential would **not** be modified by such results.

Policy implications.

The results of this study have important implications with respect to the policies which have been followed concerning radon-related issues.

Degree of health concern about radon exposure. The results of this study, in combination with previous data, suggest that radon is a carcinogen in the residential setting. From our data, the excess lung cancer risk per unit of radon exposure appears to be consistent with underground mining studies. These findings also suggest that even the relatively low exposures typical of dwellings may increase risk of lung cancer and

that high exposures would be associated with very serious lung cancer risks. If our results are corroborated, there does not appear to be an exposure limit for radon which can be used to totally avoid risks; instead, reduction of excess lung cancer risk must be based on avoidance of unnecessary exposure.

Governmental radon programs. The observation of probable lung cancer risk at even moderately elevated radon exposures supports governmental actions to educate citizens, provide technical information and services, and conduct research on health effects, testing, and remediation. Furthermore, smoking avoidance education should be included and strongly emphasized in all governmental radon risk education activities. The distinction between radon screening and annual average tests should be emphasized.

Remedial action level recommendations. **Given that radon appears to be a lung carcinogen even at low, unavoidable exposures, the recommended action levels must be based on feasibility of remediation. The current guidance remains:**

1) Follow-up testing should be conducted when a screening test under worst case conditions (heating season, ground level closed house) exceeds 4 pCi/L, in order to characterize the annual exposures to occupants of the dwelling. The length of the follow-up testing should depend upon the screening result: over about 20 pCi/L, follow-up tests should be short term; below 20 pCi/L, long-term measurements are better, but short-term testing may also be useful under certain circumstances.

2) Remedial action should be taken when follow-up testing indicates that typical exposures of occupants are elevated and when remediation is feasible, i.e., when typical exposures are greater than 4 pCi/L.

The above action levels are not based only on acceptability of risks, because a true health-based guideline for a carcinogen such as radiation would be associated with zero exposure or with extremely small calculated risks such as less than one per million. However, even outdoor radon levels (about 0.1 - 0.2 pCi/L) and baseline indoor levels (0.2 - 1.0 pCi/L) are predicted to result in considerably more lung cancer than one in a

million on the assumption of no "threshold" exposure and linearity of risk for lung cancer risk for radon. The forseeable future does not appear to hold any promise for changing this unavoidable exposure or the associated risks.

There are two types of practical limitations to reduction of radon exposure:

a) Remediation efficacy: As the indoor concentrations approach the baseline, there are diminishing returns in radon concentration reduction as a result of remedial action. For any house, at some point of radon concentration further actions are increasingly expensive and decreasingly effective. (This phenomenon is true of pollution abatement generally).

b) Validation of remediation: As the indoor radon concentrations are reduced closer to background, normal daily/weekly/seasonal fluctuations due to weather ventilation, etc., can easily mask any improvements in radon gas levels which result from further remediation. In order to be sure whether any action has succeeded, testing must be increasingly long in duration and sophisticated in sensitivity. It therefore becomes even more difficult to reduce radon concentrations below a certain point because the results of such actions cannot be easily verified.

There has been consensus from national radon technology experts that 4 pCi/L is currently an achievable goal for most dwellings. There is also intensive research underway throughout the world to increase the effectiveness of both new construction and remediation techniques for citizens. It is hoped that these efforts will contribute to the long-range goals of limiting indoor radon to outdoor (background) concentrations.

Meanwhile, it has been the policy of the NJDOH and NJDEP to urge attainment of the lowest radon exposure which is currently feasible for citizens, to support a decrease in the officially-recommended exposure limit as soon as such technology is considered practical, and to support the implementation of building construction codes so that radon entry resistant dwellings will be built in areas with high exposure potential. The findings of this case-control study on lung cancer and radon in New Jersey women

support these policies, since they are consistent with the belief that even radon concentrations at or below the current guidance levels probably cause small increases in the chances of lung cancer. However, it is important that public health agencies periodically review new data on health risk, monitoring and remediation, and that policy recommendations to citizens are updated whenever necessary.

Recommendations on specific geographic areas. This study did not address potential radon exposures in various counties or municipalities. The ongoing data collection and updated guidance in this regard by the NJDEP continues to be the best guide to citizen testing.

Policies addressing maximum individual risks vs population risks. Indoor radon is an example of a public health hazard in which some individuals are subject to much higher exposures than most others. **It is appropriate for public health policy to address reduction of risk both to the most highly exposed individuals (maximum individual risk) and to the public as a whole (population attributable risk).**

Extensive media attention and resources by NJDEP have been devoted to the discovery of houses with extremely high radon levels (e.g. over 200 pCi/L). Some occupants of such houses are exposed to higher concentrations than those typical of some uranium miners and may have lung cancer risks approaching or even exceeding those of cigarette smokers. While identification and remediation of such houses do not make a large impact on population-attributable risks, they may have a dramatic effect on reducing the lung cancer risks for the specific occupants.

The excess risks to individual occupants of houses with low radon exposures are quite modest compared to other causes of lung cancer to which members of the public are subject, specifically, smoking and certain occupational exposures. However, in the population as a whole, most of the lung cancer risk due to radon is a result of relatively low exposures. Locating and assuring reduction of moderately elevated indoor radon concentrations is not likely to make a large impact on rates of lung

cancer unless conducted on an extremely wide scale. Despite massive publicity and educational activities of public agencies and the media, it is believed (G. Nicholls, personal communication) that considerably less than half of New Jersey dwellings in the areas with the highest radon potential have been tested to date. There is little information regarding the proportion of houses screened which have had follow-up testing and even less data on the population of houses with annual averages over the current guideline 4 pCi/L, which have been remediated.

Moreover, because remediation is not yet feasible at levels less than 4 pCi/L, revised building codes designed to render new dwellings more resistant to radon entry may have a far-reaching effect on overall population risks. To be effective such codes need to be widely implemented, especially in areas with high radon potential.

Considering these issues of population and individual risks also help to underscore the importance of public policies and resources devoted to avoidance and cessation of smoking in addressing lung cancer hazards. There has been a fallacious historical distinction between the involuntary assumption of risks due to exposure such as radon, and the voluntary assumption of risks due to smoking. However, the overwhelming majority of smokers began as children, and by the time they reached adulthood were addicted. The allocation of public health resources should reflect the magnitude of the relative and population attributable risks, regardless of any presumed voluntary nature of smoking exposures.

Future analyses.

This report is neither a final point in the data collection nor of data analyses for this study. Nonetheless, we consider it important to share with the New Jersey public and the scientific community the findings to date because of their important implications in validating the activities conducted and recommended so far by the

NJDOH and the NJDEP. Several important additions to the study are planned for the near future.

"Phase II" houses. As described in the text above, the original study design and resources limited radon assessment to only one residence per subject. Phase II residence measurements are currently under way and are intended to add more years of radon measurements to those included in this report as well as more subjects from the previous lung cancer study to the radon extension. The accuracy of cumulative radon exposure estimates and the statistical power of the study will be increased, and exposure misclassification may be decreased in this manner. All additional data analyses described below will include the Phase II residence data.

Time analysis of radon exposure. The 1988 "BEIR IV" report of the National Research Council proposed a model linking radon exposure and lung cancer in underground miners which included a factor called "time since exposure" (NRC, 1988). In particular, the NRC concluded that exposures during the time interval 5 - 15 years before diagnosis or death from lung cancer produced twice the risk increment as exposures before that time. Further analyses of the data in this report combined with Phase II residences will be used to test the hypothesis that the BEIR IV model applies to residences in that respect.

Quantitative modeling of smoking interaction. Further statistical analysis of the interaction of smoking and radon will be conducted. The BEIR IV and other models proposed by other research reports will be explored. As described above, ages and rates at which exposure to radon and smoking occur may be important in determining degree of risk.

New Jersey geographic data on radon potential as a surrogate for missing years. As described earlier and in Appendix G, the NJDEP has conducted a statewide stratified sampling of about 6,000 residences and other buildings. As a result, they have generated municipality-based and county-based estimates of average radon exposure.

As illustrated in Appendix G, there is generally good agreement between the results of the NJDEP study and the distribution of the radon concentrations from our own study set taking into account differences in sampling between the two studies and the house age restrictions in the case-control study.

In a subsequent analysis, we will use municipality-specific or county-specific radon data from the DEP study to estimate the remaining missing years (among the 25-year exposure window) for New Jersey houses in our study (after Phase II houses have been added). A comparison of the dimensions and significance of risk estimates and trends of the data will then be made with and without use of these surrogates. We hypothesize that the use of the geographically-based surrogates will improve the predictive value of radon exposure for lung cancer risk in our data (i.e., the significance of dose-response trends will increase).

Urban-rural gradient. There have been several indications that the urban or rural character of a dwelling locality may predict radon exposure or the observable association of radon with lung cancer. For example, the results of Axelson et al. (1988) found a significant radon-lung cancer association in rural but not urban areas of Stockholm county. Therefore, in a subsequent analysis, population density, our subjects' own characterization of the urban-rural nature of their residence, and other factors will be used to explore the effect of an urban-rural factor on the multivariate model for lung cancer risk. The distribution pattern of radon in New Jersey, i.e. the highest radon is generally found in rural areas, particularly suggest this analysis.

House construction changes. We will explore whether data from the house construction characteristics and changes described in Appendix D can be useful. That is, we will consider whether using modifications of radon concentration indices on the basis of such data can increase the predictive value of the radon exposure for lung cancer risk.

Use of occupational data to improve exposure estimates. In a future analysis, details on time and place of occupation for these women subjects will be used to improve

estimates of number of hours per week spent at home. Modest improvement in both cumulative radon exposure estimates and the trends for cumulative exposure and lung cancer are expected.

Relocation frequency. In a future analysis, the relative frequency with which subject subgroups moved their residence will be observed in order to further consider factors which could have influenced our results because of the residency requirement in our design.

CONCLUSIONS

Radon exposure is universal; everyone is exposed to radon to some degree. This interim report is intended to contribute to decisions by public agencies and individuals regarding the importance of limiting radon exposure, wherever it is feasible to do so.

The findings of the first phase of the New Jersey epidemiologic study of radon and lung cancer in women are consistent with recommendations to reduce exposure which have been made by the New Jersey Department of Health, New Jersey Department of Environmental Protection, U.S. Environmental Protection Agency, and other federal agencies. These recommendations have been in effect since the widespread problem of elevated radon exposure from naturally-occurring sources became known in the mid-1980's.

This study found statistically significant or marginally significant trends in lung cancer risk with increasing radon exposure. However, the number of subjects in this study with annual exposures above 4 pCi/L was very small; therefore, the results should be interpreted very cautiously. The degree of excess risk per unit of radon exposure which were found are in good agreement with the few previous individual-based residential studies and with the many occupational studies of underground miners.

Forthcoming analyses of additional measurement data may improve the confidence of the risk estimates from this study.

The exposure data yielded by the study suggest that a relatively small percentage of houses in New Jersey which are more than 25 years old have annual averages above 4 pCi/L, although in certain geographic areas, the proportion is larger. Moreover, the relationship of screening to annual average exposures may need better characterization for public policy purposes and clearer understanding by the public before remediation decisions are made.

One potentially important finding was that the strongest effects of radon exposure were seen in light and moderate smokers. However, the possible contribution of misclassification of smoking and selective underrepresentation of heavy smokers cannot be ruled out. It is clear that cigarette smoking, even at the level of one pack per day, remains by far the most important risk factor for lung cancer in most women and men.

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Table 1

Reported case-control studies of housing, residential radon, and lung cancer

First author	Number of Cases/controls	Exposure group	Results
Axelson (1979)	37/178	Wooden houses without basements	1.0[a]
		Mixed houses	1.7
		Stone houses with basements	4.8
Edling (1984)	23/178	Low exposure houses[b]	1.0[c]
		Middle exposure houses	1.2 (0.5, 3.0)
		High exposure houses	3.5 (1.3, 9.2)
	19/159	<50 Bq/m^3 EER[d]	1.0[c]
		50-149 Bq/m^3 EER	2.3 (0.9, 5.8)
		150+ Bq/m^3 EER	5.1 (1.4, 18.5)
Svensson (1987)	292/584	Negative exposure history[e]	1.0[f]
		Positive exposure history	2.2 (1.2, 4.0)
Svensson (1989)	187/377	<4,500 Bq/m^3-years	1.0[g]
		4,500-6,000 Bq/m^3-years	1.8 (1.2, 2.9)
		>6,000 Bq/m^3-years	1.7 (0.9, 3.3)
Axelson (1988)	85/296	Rural:Low exposure houses[b]	1.0[h]
		Middle exposure houses	1.4 (0.82, 2.4)
		High exposure houses	1.8 (0.94, 3.4)
	67/161	Urban:Low exposure houses[b]	1.0[h]
		Middle exposure houses	0.65 (0.22, 1.9)
		High exposure houses	0.54 (0.17, 1.7)
Lees (1987)	27/49	Cumulative background-corrected exposure:	
		0 WLM	1.0[i]
		>0 WLM	2.4 (0.79,7.1)

Table 1 (contd)

[a] Age and sex adjusted rate ratio (no confidence intervals reported); Mantel-Haenszel rate ratio (90% confidence interval) for mixed + stone houses vs wooden houses is 1.8 (0.99, 0.32).

[b] Exposure defined by stone (+) vs. wooden (-) houses; basement (+) vs. no basement(-); alum shale ground (+) vs. no alum shale ground (-). Low exposure houses = -/-/-. Middle exposure houses =+/-/-, -/+/-, -/-/+. High exposure houses = +/+/-, +/-/+, -/+/+, +/+/+.

[c] Age, sex and smoking adjusted odds ratios (90% confidence interval).

[d] EER = Equilibrium Equivalaent Radon.

[e] Positive exposure history defined as residence in a single family detached house (or on the ground floor of a multi-family house) located on radon-emitting ground types.

[f] Odds ratio (95% confidence interval) from age-matched triplet analysis

[g] Age, smoking and municipality adjusted odds ratios (95% confidence interval).

[h] Smoking-adjusted odds ratios (90% confidence interval)

[i] Smoking adjusted odds ratio (95% confidence interval)

TABLE 2
Criteria for inclusion of houses in Phase II of data collection,
New Jersey radon-female lung cancer case-control study, 1982-1988

CRITERIA FOR HOUSE INCLUDED IN PHASE II OF RADON SUBSTUDY	RESIDENCE TIMES FOR SUBJECTS ALREADY INCLUDED IN RADON STUDY UNDER PHASE I WITH FURTHER RESULTS UNDER PHASE II	RESIDENCE TIMES FOR NEW SUBJECTS TO BE INCLUDED IN RADON STUDY UNDER PHASE II
1. residence of original subject for at least 10 years in the 25 year period from 5-30 years prior to diagnosis	10-14 years (Phase I) + 10-14 YEARS (PHASE II)	10+ YEARS (PHASE II)
2. residence of original subject which would add at least 7 years in the 25 year period to the already collected exposure history	10-17 years (Phase I) + 7-9 YEARS (PHASE II)	--------
3. residences of original subject, each for at least 7 years in the 25 year period, which would result in a total known exposure history of at least 14 years	----------	7-9 YEARS (PHASE II) + 7-9 YEARS (PHASE II)
4. residence of original subject in six counties with high radon potential[a] which would add at least 4 years in the 25 year period to the already collected exposure history	10-21 years (Phase I) + 4-6 YEARS (PHASE II)	-------
5. residences of original subject in six counties with high radon potential[3], each for at least 4 years in the 25 year period, which would result in a total known exposure history of at least 14 years.	----------	4-6 YEARS (PHASE II) + 4-6 YEARS (PHASE II) + 4-6 YEARS (PHASE II)

[a] Warren, Hunterdon, Sussex, Morris, Somerset, and Mercer counties

TABLE 3
Distribution of the original New Jersey female lung cancer cases and controls by their status in the radon substudy
New Jersey radon-female lung cancer case-control study, 1982-1988

	No. of cases (%)		No. of controls (%)	
INCLUDED IN RADON STUDY	**433**	**(43.6%)**	**402**	**(40.4%)**
Radon testing at index address [a]	411	(41.4%)	385	(38.7%)
Index address is apartment 3+ [b]	22	(2.2%)	17	(1.7%)
NOT INCLUDED IN RADON STUDY	**561**	**(56.4%)**	**593**	**(59.6%)**
No address specific information [c]	140	(14.1%)	126	(12.7%)
No address met residence criterion [d]	253	(25.5%)	256	(25.7%)
No radon testing at index address [e]	168	(16.9%)	211	(21.2%)
TOTAL [f]	994		995	

Chi-square for case vs. control distributions: 7.1, 4 d.f., p = 0.13

[a] Subjects whose index address was successfully tested for radon with alpha track detectors and/or charcoal canisters. Includes subjects whose index address was an apartment on the first or second floor.

[b] Subjects whose index address was an apartment above the second floor or a trailer.

	No. of cases (%)		No. of controls (%)	
[c] No address specific information				
Refused further contact after interview	29	(2.9%)	27	(2.7%)
Lost to follow-up	58	(5.8%)	43	(4.3%)
Refused address-specific interview	31	(3.1%)	35	(3.5%)
Inadequate address-specific information	22	(2.2%)	21	(2.1%)
[d] No address met residence criterion				
No New Jersey town, 10+ yrs, 1953-1972	164	(16.5%)	170	(17.1%)
No New Jersey address, 10+ yrs, 1953-1972	89	(9.0%)	86	(8.6%)

(contd)

Table 3 (contd.)

[e] No radon testing at index address				
Index address demolished	23	(2.3%)	20	(2.0%)
Refusal by current resident	112	(11.3%)	169	(17.0%)
No contact with current resident	33	(3.3%)	22	(2.2%)

[f] 994 cases represent 76.1% of 1,306 cases identified in original study; 995 controls represent 68.7% of 1,449 controls identified in original study.

TABLE 4
Number of lung cancer cases and controls in radon study, by various risk factors and subject characteristics
New Jersey radon-female lung cancer case-control study, 1982-1988

	No. of cases (%)		No. of controls (%)	
TOTAL	433		402	
AGE AT DIAGNOSIS[a]				
<58 years	98	(22.6%)	78	(19.4%)
58-71 years	215	(49.7%)	216	(53.7%)
72+ years	120	(27.7%)	108	(26.9%)
RESPONDENT TYPE				
self	246	(56.8%)	212	(52.7%)
spouse	74	(17.1%)	89	(22.1%)
other next of kin	113	(26.1%)	101	(25.1%)
RACE				
white, including hispanic	418	(96.5%)	386	(96.0%)
nonwhite	15	(3.5%)	16	(4.0%)
CIGARETTES/DAY [b]				
Lifetime nonsmoker	61	(14.1%)	213	(53.0%)
< 15 cigarettes/day	83	(19.2%)	90	(22.4%)
15-24 cigarettes/day	178	(41.1%)	67	(16.7%)
25+ cigarettes/day	111	(25.6%)	32	(8.0%)
TOTAL NUMBER OF YEARS SMOKED				
Lifetime nonsmoker	61	(14.1%)	213	(53.0%)
< 35 years	90	(20.8%)	72	(17.9%)
35+ years	282	(65.1%)	117	(29.1%)
NUMBER OF YEARS QUIT SMOKING				
Lifetime nonsmoker	61	(14.1%)	213	(53.0%)
Current smoker, quit 0-1 years	289	(66.7%)	112	(27.9%)
Ex-smoker, quit 2-9 years	49	(11.3%)	27	(6.7%)
Ex-smoker, quit 10+ years	34	(7.9%)	50	(12.4%)
AVERAGE CIGARETTE TAR CONTENT, 1973-1982				
Lifetime nonsmoker	61	(14.1%)	213	(53.0%)
Nonsmoker, 1973-1982	31	(7.2%)	45	(11.2%)
Smoker, tar <21 mg/cigarette	284	(65.6%)	126	(31.3%)
Smoker, tar 21+ mg/cigarette	57	(13.2%)	18	(4.5%)
VEGETABLE CONSUMPTION[a]				
<35 servings/month	118	(27.3%)	83	(20.7%)
35-74 servings/month	241	(55.7%)	209	(52.0%)
75+ servings/month	74	(17.1%)	110	(27.4%)

(contd)

TABLE 4 (contd.)
Number of lung cancer cases and controls in radon study,
by various risk factors and subject characteristics
New Jersey radon-female lung cancer case-control study, 1982-1988

	No. of cases (%)		No. of controls (%)	
TOTAL	433		402	
HIGH-RISK OCCUPATION [c]				
no high-risk occupation	350	(80.8%)	363	(90.3%)
high-risk occupation	83	(19.2%)	39	(9.7%)
EDUCATION				
<8 years completed	35	(8.1%)	51	(12.7%)
8-12 years completed	278	(64.2%)	232	(57.7%)
13+ years completed	120	(27.7%)	119	(29.6%)
COUNTY AT DIAGNOSIS[d]				
low radon	112	(25.9%)	89	(22.1%)
moderately low radon	163	(37.6%)	178	(44.3%)
moderate radon	84	(19.4%)	76	(18.9%)
high radon	74	(17.1%)	59	(14.7%)
LIFETIME NONSMOKERS ONLY, BY PASSIVE SMOKING				
No exposure to spouse tobacco	18	(29.5%)	70	(32.9%)
Exposure to spouse cigarette smoke	38	(62.3%)	116	(54.5%)
Exposure to spouse pipe/cigar only	5	(8.2%)	27	(12.7%)

[a] Cutpoints based on distribution of controls in original female lung cancer study (1st quartile; 2nd+3rd quartile; 4th quartile)
[b] Lifetime average number of cigarettes smoked per day. Cutpoints based on bimodal distribution of controls in original female lung cancer study, with peaks at 10 and 20 cigarettes/day, and a long tail starting at 25 cigarettes/day.
[c] Ever employed in any occupational group shown to have a smoking adjusted risk of 1.5 or greater in the original female lung cancer study. This is an a posteriori definition, used only for the purpose of adjusting in the radon analyses for the possible effect of occupational exposure. See Appendix b for further clarification of this variable.
[d] County at diagnosis for cases, or county at ascertainment for controls. Low radon counties include include Atlantic, Cape May, Essex, Hudson, and Ocean counties. Moderately low radon counties include Bergen, Burlington, Cumberland, Gloucester, Middlesex, and Union counties. Moderate radon counties include Camden, Monmouth, Passaic, and Salem counties. High radon counties include Hunterdon, Mercer, Morris, Somerset, Sussex, and Warren counties. Grouping of counties was determined by the percentage of houses with basement or lower level screening values above 4 pCi/L in the statewide survey conducted for the New Jersey State Department of Environmental Protection (see Appendix G).

TABLE 5
Distribution of lung cancer cases and controls included in radon study by type of radon measurement results at index residence, New Jersey radon-female lung cancer case-control study, 1982-1988

	No. of cases (%)	No. of controls (%)	Total No. (%)
Living area alpha track measurements			
First floor[a]	184 (42.5%)	163 (40.5%)	347 (41.6%)
Second floor[a]	162 (37.4%)	155 (38.6%)	317 (38.0%)
Estimates of living area alpha track results from basement alpha track measurements [b]	27 (6.2%)	28 (7.0%)	55 (6.6%)
Estimates of living area alpha track results from canister measurements [b]	38 (8.8%)	39 (9.7%)	77 (9.2%)
Living area alpha track results estimated as < 1 pCi/L; no actual radon measurements; index residence is apartment 3+[b,c]	22 (5.1%)	17 (4.2%)	39 (4.7%)
TOTAL	433	402	835

Chi-square for case vs. control distributions: 0.95, 4 d.f., p = 0.92.

[a] Includes measurements made in apartments which were below the third floor.

[b] See Appendix J .

[c] Subjects whose index address was an apartment above the second floor or a trailer.

TABLE 6
Distribution of lung cancer cases and controls by radon level (year-long living area alpha track measurements, n=664; estimates, n=171) and by lifetime average daily cigarette consumption, New Jersey radon-female lung cancer case-control study, 1982-1988

Smoking status	Radon (pCi/L) <1.0 [a]	1-1.9	2-3.9	4-11.3	Total
Nonsmokers					
Cases	48 (78.7%)	11 (18.0%)	1 (1.6%)	1 (1.6%)	61
Controls	168 (78.9%)	39 (18.3%)	5 (2.3%)	1 (0.5%)	213
Unadjusted OR	**1.0[b]**	**0.99**	**0.70**	**3.5**	**1.0[c]**
<15 cigs/day					
Cases	61 (73.5%)	16 (19.3%)	3 (3.6%)	3 (3.6%)	83
Controls	77 (85.6%)	13 (14.4%)	0 (-)	0 (-)	90
Unadjusted OR	**2.8**	**4.3**	**oo**	**oo**	**3.2**
15-24 cigs/day					
Cases	139 (78.1%)	28 (15.7%)	10 (5.6%)	1 (0.6%)	178
Controls	55 (82.1%)	10 (14.9%)	2 (3.0%)	0 (-)	67
Unadjusted OR	**8.8**	**9.8**	**17.5**	**oo**	**9.3**
25+ cigs/day					
Cases	94 (84.7%)	12 (10.8%)	4 (3.6%)	1 (0.9%)	111
Controls	24 (75.0%)	4 (12.5%)	3 (9.4%)	1 (3.1%)	32
Unadjusted OR	**13.7**	**10.5**	**4.7**	**3.5**	**12.1**
Total					
Cases	342 (79.0%)	67 (15.5%)	18 (4.2%)	6 (1.4%)	433
Controls	324 (80.6%)	66 (16.4%)	10 (2.5%)	2 (0.5%)	402
Unadjusted OR	**1.0[d]**	**0.96**	**1.7**	**2.8**	

[a] Includes subjects whose index address was an apartment above the second floor or a trailer.
[b] Unadjusted odds ratio (an estimate of the lung cancer risk associated with radon exposure and smoking, but not adjusted for any other factors), relative to nonsmokers with < 1.0 pCi/L radon exposure.
[c] Unadjusted odds ratio (an estimate of the lung cancer risk associated with smoking, but not adjusted for radon exposure or any other factors), relative to lifetime nonsmokers.
[d] Unadjusted odds ratio (an estimate of the lung cancer risk associated with radon exposure, but not adjusted for smoking or any other factors), relative to subjects with < 1.0 pCi/L radon exposure.

TABLE 7

Odds ratios[a] (90% confidence intervals) for association of lung cancer with radon (year-long living area alpha track measurements, n=664; estimates, n=171) in ALL SUBJECTS, and EXCLUDING HEAVY SMOKERS.

New Jersey radon-female lung cancer case-control study, 1982-1988

Smoking status	Radon (pCi/L) <1.0[b]	1-1.9	2-3.9	4-11.3	Trend Zcat[c] (p)	Trend Zcnt[d] (p)
ALL SUBJECTS						
Unadjusted OR	1.0	0.96 (0.70,1.3)	1.7 (0.88,3.3)	2.8 (0.74,10.9)	1.51 (0.066)	1.36 (0.087)
[LR=2.4, 1 df]+			1.9[e] (1.0,3.4)			
Adjusted by cigarettes/day	1.0	1.1 (0.78,1.6)	1.3 (0.61,2.7)	3.5 (0.82,15.2)	1.49 (0.068)	1.35 (0.089)
[LR=189.1, 4 df]+			1.6 (0.82,3.1)			
ALL EXCEPT HEAVY SMOKERS						
Unadjusted OR	1.0	1.1 (0.77,1.5)	2.4 (1.1,5.2)	6.0 (0.99,36.9)	2.33 (0.010)	2.12 (0.017)
[LR=5.8, 1 df]+			2.9 (1.4,5.8)			
Adjusted by cigarettes/day	1.0	1.2 (0.81,1.7)	2.0 (0.84,4.8)	8.7 (1,3,56.8)	2.40 (0.008)	2.11 (0.017)
[LR=144.6, 3 df]+			2.8 (1.3,6.1)			

+ Likelihood ratio statistic for model including categorical "radon trend" term (with degrees of freedom)

[a] Odds ratios (OR) and 90% confidence intervals from logistic regression analyses.

[b] Includes subjects whose index address was an apartment above the second floor or a trailer.

[c] Z statistic (1-sided p value) for categorical "radon trend" term in logistic regression model. This term equals 0.4 if radon is <1 pCi/L, 1.2 (1-1.9 pCi/L), 2.3 (2-3.9 pCi/L), or 4.55 (4+ pCi/L). These values are the medians of the respective intervals for controls. This model gives results equivalent to the Mantel Chi-extension procedure for stratified analyses.

[d] Z statistic (1-sided p Value) for continuous radon variable in logistic regression model.

[e] OR (90% confidence interval) for radon = 2+ pCi/L.

TABLE 8

Odds ratios[a] for association of lung cancer with radon (year-long living area alpha track measurements, n=664; estimates, n=171) in all subjects, adjusting for other risk factors and subject characteristics, New Jersey radon-female lung cancer case-control study, 1982-1988

Adjusted by:	Radon (pCi/L) <1.0[b]	1-1.9	2-3.9	4-11.3	Trend Zcat[c] (p)
Cigarettes/day[d]	1.0	1.1	1.3	3.5	1.49 (0.068)
[LR=189.1, 4df]+			1.6[e]		
Cigarettes/day + occupation[f]	1.0	1.1	1.4	3.6	1.65 (0.049)
[LR=200.4, 5df]+			1.7		
(p < 0.001)++					
Cigarettes/day + age[g]	1.0	1.1	1.2	3.8	1.44 (0.075)
[LR=202.3, 6df]+			1.6		
(p = 0.001)++					
Cigarettes/day + yrs quit smoking[h]	1.0	1.2	1.2	4.2	1.66 (0.048)
[LR=207.1, 10df]+			1.6		
(p = 0.006)++					
Cigarettes/day + cigarette tar[i]	1.0	1.2	1.2	4.3	1.69 (0.046)
[LR=205.0, 10df]+			1.6		
(p = 0.014)++					
Cigarettes/day + respondent type[j] + resptype+cigs/day[k]	1.0	1.1	1.3	3.1	1.41 (0.079)
[LR=200.6, 8df]+			1.6		
(p = 0.021)++					
Cigarettes/day + vegetables[l]	1.0	1.1	1.3	3.6	1.55 (0.061)
[LR=195.0, 6df]+			1.7		
(p = 0.052)++					
Cigarettes/day + respondent type[j]	1.0	1.1	1.3	3.6	1.51 (0.066)
[LR=190.5, 5df]+			1.6		
(p = 0.237)++					

TABLE 8 (contd)
Odds ratios[a] for association of lung cancer with radon
(year-long living area alpha track measurements, n=664; estimates, n=171)
in all subjects, adjusting for other risk factors and subject characteristics,
New Jersey radon-female lung cancer case-control study, 1982-1988

Adjusted by:	Radon (pCi/L) <1.0[b]	1-1.9	2-3.9	4-11.3	Trend Zcat[c] (p)
Cigarettes/day + years smoked[m] [LR=193.2, 7df]+ (p = 0.251)++	1.0	1.1	1.2	3.6	1.47 (0.071)
			1.6		
Cigarettes/day + county[n] [LR=191.5, 7df]+ (p = 0.494)++	1.0	1.1	1.2	3.2	1.36 (0.087)
			1.5		
Cigarettes/day + education[o] [LR=189.4, 6df]+ (p = 0.861)++	1.0	1.1	1.3	3.5	1.48 (0.069)
			1.6		
Cigarettes/day + race[p] [LR=189.1, 5df]+ (p = 0.99)++	1.0	1.1	1.3	3.5	1.48 (0.069)
			1.6		

+ Likelihood ratio statistic for model including categorical "radon trend" term (with degrees of freedom)
++ Significance of Chi-square statistic evaluating the improvement in fit, i.e., the difference in likelihood ratio statistics between this model and the base model (including only adjustment for cigarettes/day)

[a] Odds ratios from logistic regression analyses. Models are ranked by the improvement in the fit of the model, as determined by the difference in the likelihood ratio statistics of the new model and the likelihood ratio statistic of the base model including only terms for cigarettes per day and radon. This difference was evaluated as a Chi-square statistic with degrees of freedom equivalent to the difference in degrees of freedom between the new model and the base model.

[b] Includes subjects whose index address was an apartment above the second floor or a trailer.

[c] Z statistic (1-sided p value) for categorical "radon trend" term in logistic regression model. This term equals 0.4 if radon is <1 pCi/L, 1.2 (1-1.9 pCi/L), 2.3 (2-3.9 pCi/L), or 4.55 (4+ pCi/L). These values are the medians of the respective intervals for controls. This model gives results equivalent to the Mantel Chi-extension procedure for stratified analyses. (contd)

TABLE 8 (contd.)

[d] adjusted by lifetime average daily cigarette consumption (nonsmokers, <15 cigarettes/day, 15-24 cigarettes/day, 25+ cigarettes/day).

[e] OR for radon = 2+ pCi/L.

[f] adjusted by lifetime average daily cigarette consumption and high risk occupation (ever employed in any occupational group shown to have a smoking adjusted risk of 1.5 or greater in the original female lung cancer study [see Appendix B for further clarification of this variable]; never employed in any of these occupational groups)

[g] adjusted by lifetime average daily cigarette consumption and age (<58, 58-71, 72+)

[h] adjusted by lifetime average daily cigarette consumption and number of years since smoking cessation (lifetime nonsmoker, quit 0-1 year, quit 2-9 years, quit 10+ years).

[i] adjusted by lifetime average daily cigarette consumption and time-weighted average tar content of cigarettes smoked during 1973-1982 (lifetime nonsmoker, tar <21 mg/cigarette, tar 21+ mg/cigarette, smoker but did not smoke during 1973-1982). See Appendix B for further clarification of this variable.

[j] adjusted by lifetime average daily cigarette consumption and respondent type (self, next of kin). There was little difference if next of kin were separated into spouse and other next of kin groups.

[k] adjusted by lifetime average daiily cigarette consumption, respondent type (see above, note j), and the interaction between respondent type and number of cigarettes smoked per day. See Appendix B for further clarification of this variable.

[l] adjusted by lifetime average daily cigarette consumption and vegetable consumption as a measure of dietary Vitamin A (<35 servings/month, 35-74 servings/month, 75+ servings/month).

[m] adjusted by lifetime average daily cigarette consumption and total duration of smoking (lifetime nonsmoker, <35 years, 35+ years).

[n] adjusted by lifetime average daily cigarette consumption and county of residence at diagnosis (low radon, moderately low radon, moderate radon, high radon). See Appendix B or footnote d, Table 3, for further clarification of this variable.

[o] adjusted by lifetime average daily cigarette consumption and education (<8 years, 8-12 years, 13+ years)

[p] adjusted by lifetime average daily cigarette consumption and race (white including hispanic, nonwhite)

TABLE 9

Odds ratios[a] (90% confidence intervals) for association of lung cancer with radon (year-long living area alpha track measurements, n=664; estimates, n=171) in ALL SUBJECTS, adjusting for multiple risk factors and subject characteristics
New Jersey radon-female lung cancer case-control study, 1982-1988

Adjusted by:[b]	Radon (pCi/L) <1.0[c]	1-1.9	2-3.9	4-11.3	Trend Zcat[d] (p)	Trend Zcnt[e] (p)
Cigarettes/day, age, occupation, yrs quit smoking [LR=236.3, 13df]+	1.0	1.2 (0.81,1.7)	1.2 (0.58,2.7)	4.7 (1.1,20.3)	**1.80 (0.036)**	**1.48 (0.069)**
			1.7[f] (0.87,3.4)			
Cigarettes/day, age, occupation, yrs quit smoking, respondent type, resptype*cigs/day [LR=246.6, 17df]+	1.0	1.1 (0.79,1.7)	1.3 (0.62,2.9)	4.2 (0.99,17.5)	**1.75 (0.040)**	**1.45 (0.074)**
			1.8 (0.89,3.5)			
Cigarettes/day, age, occupation, yrs quit smoking, vegetables, respondent type, race [LR=243.2, 17df]+	1.0	1.1 (0.79,1.6)	1.4 (0.63,2.9)	4.8 (1.1,21.5)	**1.82 (0.034)**	**1.50 (0.067)**
			1.8 (0.92,3.6)			

+ Likelihood ratio statistic for model including categorical "radon trend" term (with degrees of freedom).

[a] Odds ratios and 90% confidence intervals from logistic regression analyses.
[b] See footnotes to Table 8 for definitions of variables
[c] Includes subjects whose index address was an apartment above the second floor or a trailer.
[d] Z statistic (1-sided p value) for categorical "radon trend" term in logistic regression model. This term equals 0.4 if radon is <1 pCi/L, 1.2 (1-1.9 pCi/L), 2.3 (2-3.9 pCi/L), or 4.55 (4+ pCi/L). These values are the medians of the respective intervals for controls. This model gives results equivalent to the Mantel Chi-extension procedure for stratified analyses.
[e] Z statistic (1-sided p Value) for continuous radon variable in logistic regression model.
[f] OR (90% confidence interval) for radon = 2+ pCi/L.

TABLE 10

Odds ratios[a] (90% confidence intervals) for association of lung cancer with radon (year-long living area alpha track measurements, n=559; estimates, n=133), in ALL SUBJECTS EXCLUDING HEAVY SMOKERS, adjusting for multiple risk factors and subject characteristics, New Jersey radon-female lung cancer case-control study, 1982-1988

	Radon (pCi/L)				Trend	
Adjusted by:[b]	<1.0[c]	1-1.9	2-3.9	4-11.3	Zcat[d] (p)	Zcnt[e] (p)
Cigarettes/day, age, occupation, yrs quit smoking [LR=187.7, 10df]+	1.0	1.2 (0.81,1.8)	2.0 (0.82,5.1)	9.8 (1.5,65.6)	2.47 (0.007)	2.08 (0.019)
			2.9[f] (1.3,6.6)			
Cigarettes/day, age, occupation, yrs quit smoking, respondent type, resptype*cigs/day [LR=192.4, 13df]+	1.0	1.2 (0.81,1.8)	2.0 (0.82,5.0)	8.6 (1.3,57.4)	2.38 (0.009)	2.01 (0.022)
			2.8 (1.3,6.3)			
Cigarettes/day, age, occupation, yrs quit smoking, vegetables, respondent type, race [LR=195.3, 14df]+	1.0	1.2 (0.80,1.8)	2.2 (0.88,5.3)	11.6 (1.6,84.6)	2.54 (0.006)	2.10 (0.018)
			3.1 (1.4,7.0)			

+ Likelihood ratio statistic for model including categorical "radon trend" term (with degrees of freedom).

[a] Odds ratios and 90% confidence intervals from logistic regression analyses.
[b] See footnotes to Table 8 for definitions of variables
[c] Includes subjects whose index address was an apartment above the second floor or a trailer.
[d] Z statistic (1-sided p value) for categorical "radon trend" term in logistic regression model. This term equals 0.4 if radon is <1 pCi/L, 1.2 (1-1.9 pCi/L), 2.3 (2-3.9 pCi/L), or 4.55 (4+ pCi/L). These values are the medians of the respective intervals for controls. This model gives results equivalent to the Mantel Chi-extension procedure for stratified analyses.
[e] Z statistic (1-sided p Value) for continuous radon variable in logistic regression model.
[f] OR (90% confidence interval) for radon = 2+ pCi/L.

TABLE 11
Distribution of lung cancer cases (by histologic type), and controls, by radon level
(year-long living area alpha track measurements, n=664; estimates, n=171)
New Jersey radon-female lung cancer case-control study, 1982-1988

	Radon (pCi/L)				
	<1.0 [a]	1-1.9	2-3.9	4-11.3	Total
Cases					
Squamous cell	92 (83.6%)	15 (13.6%)	2 (1.8%)	1 (0.9%)	110
Small cell	78 (82.1%)	10 (10.5%)	4 (4.2%)	3 (3.2%)	95
Adenocarcinoma	102 (79.1%)	19 (14.7%)	7 (5.4%)	1 (0.8%)	129
Large cell	24 (68.6%)	8 (22.9%)	3 (8.6%)	0 (0.0%)	35
Other types [b]	46 (71.9%)	15 (23.4%)	2 (3.1%)	1 (1.6%)	64
Controls	324 (80.6%)	66 (16.4%)	10 (2.5%)	2 (0.5%)	402

[a] Includes subjects whose index address was an apartment above the second floor or a trailer

[b] Other histologic types include: mixed adeno-squamous, undifferentiated, anaplastic, poorly differentiated, and malignant (not otherwise specified).

TABLE 12
Adjusted odds ratios[a] (90% confidence intervals) for association of lung cancer, by histologic type, with radon level
(year-long living area alpha track measurements, n=664; estimates, n=171)
New Jersey radon-female lung cancer case-control study, 1982-1988

Histologic type	Radon (pCi/L) <1.0[b]	1-1.9	2-3.9	4-11.3	Trend Zcat[c] (p)	Trend Zcnt[d] (p)
Squamous cell	1.0	0.98 (0.53,1.8)	0.43 (0.09,1.9)	5.3 (0.53,53.7)	0.07 (0.472)	0.60 (0.274)
[LR=148.9, 17df]+			0.73[e] (0.20,2.6)			
Small cell	1.0	0.83 (0.39,1.7)	0.87 (0.26,2.9)	13.2 (1.5,118.2)	1.41 (0.079)	0.88 (0.189)
[LR=178.2, 17df]+			1.8 (0.66,5.0)			
Adenocarcinoma	1.0	1.1 (0.66,1.8)	2.0 (0.79,5.1)	2.4 (0.26,22.1)	1.29 (0.099)	0.81 (0.209)
[LR=71.7, 17df]+			2.1 (0.87,4.9)			
Large cell[f]	1.0	2.2 (0.96,5.2)	3.2 (0.83,12.2)	0.0 (--,--)	1.93 (0.027)	1.00 (0.159)
[LR=69.1, 14df]+			3.2 (0.83,12.0)			
Other types[g]	1.0	2.1 (1.2,3.8)	0.96 (0.24,3.9)	3.5 (0.35,34.7)	1.46 (0.072)	1.39 (0.082)
[LR=51.1, 17df]+			1.3 (0.40,4.2)			

+ Likelihood ratio statistic for model including categorical "radon trend" term (with degrees of freedom).

[a] Odds ratios (OR) and 90% confidence intervals from logistic regression analyses, adjusted by lifetime average daily cigarette consumption, age, occupation, years since smoking cessation, respondent type, and interaction between respondent type and cigarettes/day. See footnotes, Table 8, for further definitions of these variables.
(contd)

TABLE 12 (contd)

[b] Includes subjects whose index address was an apartment above the second floor or a trailer.

[c] Z statistic (1-sided p value) for categorical "radon trend" term in logistic regression model. This term equals 0.4 if radon is <1 pCi/L, 1.2 (1-1.9 pCi/L), 2.3 (2-3.9 pCi/L), or 4.55 (4+ pCi/L). These values are the medians of the respective intervals for controls. This model gives results equivalent to the Mantel Chi-extension procedure for stratified analyses.

[d] Z statistic (1-sided p Value) for continuous radon variable in logistic regression model.

[e] OR (90% confidence interval) for radon = 2+ pCi/L.

[f] Model including respondent type*cigarettes/day interaction was indeterminate. Results are shown for model without the interaction terms.

[g] Other histologic types include: mixed adeno-squamous, undifferentiated, anaplastic, poorly differentiated, and malignant (not otherwise specified).

TABLE 13
Distribution of lung cancer cases and controls (LIFETIME NONSMOKERS ONLY), by radon level
(year-long living area alpha track measurements, n=221; estimates, n=53) and by exposure to spouse tobacco smoke
New Jersey radon-female lung cancer case-control study, 1982-1988

Passive smoking status	Radon (pCi/L) <1.0 [a]	1-1.9	2-3.9	4-11.3	Total
No exposure					
Cases	17	0	1	0	18
Controls	54	15	1	0	70
Exposure to spouse cigarette smoke					
Cases	29	8	0	1	38
Controls	96	17	2	1	116
Exposure to spouse tobacco smoke (pipes/ cigars only)					
Cases	2	3	0	0	5
Controls	18	7	2	0	27
Total nonsmokers					
Cases	48	11	1	1	61
Controls	168	39	5	1	213
Unadjusted OR	**1.0**	**0.99 (0.53,1.8)**	**0.70 (0.11,4.3)**	**3.5 [c] (0.34,36.4)**	
Adjusted OR b	**1.0**	**1.0 (0.55,1.9)**	**0.79 (0.13,5.0)**	**3.1 [d] (0.30,32.7)**	

[a] Includes subjects whose index address was an apartment above the second floor or a trailer.

[b] Adjusted by exposure to spouse tobacco smoke (no exposure, exposed to spouse cigarettes, exposed to spouse pipes/cigars only)

[c] OR for 2+ pCi/L: 1.2 (0.30,4.6); trend (Zcat) = 0.43, p = 0.33.
[d] OR for 2+ pCi/L: 1.2 (0.31,5.0); trend (Zcat) = 0.51, p = 0.32.

TABLE 14

Distribution of lung cancer cases and controls by cumulative radon exposure[a] and by lifetime average daily cigarette consumption, New Jersey radon-female lung cancer case-control study, 1982-1988

Smoking status	Cumulative radon (pCi/L-years) <25	25-49	50-99	100-155	Total
Nonsmokers					
Cases	52 (85.2%)	8 (13.1%)	0 (-)	1 (1.6%)	61
Controls	175 (82.2%)	33 (15.5%)	4 (1.9%)	1 (0.5%)	213
Unadjusted OR	**1.0[b]**	**0.82**	**0.0**	**3.4**	**1.0[c]**
<15 cigs/day					
Cases	64 (77.1%)	14 (16.9%)	3 (3.6%)	2 (2.4%)	83
Controls	82 (91.1%)	8 (8.9%)	0 (-)	0 (-)	90
Unadjusted OR	**2.6**	**5.9**	**oo**	**oo**	**3.2**
15-24 cigs/day					
Cases	146 (82.0%)	24 (13.5%)	8 (4.5%)	0 (-)	178
Controls	58 (86.6%)	7 (10.4%)	2 (3.0%)	0 (-)	67
Unadjusted OR	**8.5**	**11.5**	**13.5**	**—**	**9.3**
25+ cigs/day					
Cases	99 (89.2%)	10 (9.0%)	1 (0.9%)	1 (0.9%)	111
Controls	25 (78.1%)	4 (12.5%)	3 (9.4%)	0 (-)	32
Unadjusted OR	**13.3**	**8.4**	**1.1**	**oo**	**12.1**
Total					
Cases	361 (83.4%)	56 (12.9%)	12 (2.8%)	4 (0.9%)	433
Controls	340 (84.6%)	52 (12.9%)	9 (2.2%)	1 (0.2%)	402
Unadjusted OR	**1.0[d]**	**1.0**	**1.3**	**3.8**	

[a] Cumulative radon exposure during 25 years from 5-30 years prior to case diagnosis or control selection; assumes exposure of 0.6 pCi/L (median for controls) for any of the 25 years during which the subject did not live in the index address where the measurements were made.

b Unadjusted odds ratio (an estimate of the lung cancer risk associated with radon exposure and smoking, but not adjusted for any other factors), relative to nonsmokers with < 25.0 pCi/L-years cumulative radon exposure.

[c] Unadjusted odds ratio (an estimate of the lung cancer risk associated with smoking, but not adjusted for radon exposure or any other factors), relative to lifetime nonsmokers.

[d] Unadjusted odds ratio (an estimate of the lung cancer risk associated with cumulative radon exposure, but not adjusted for smoking or any other factors), relative to subjects with < 25.0 pCi/L-years cumulative radon exposure.

TABLE 15

Odds ratios[a] (90% confidence intervals) for association of lung cancer with cumulative radon exposure[b] in ALL SUBJECTS, and EXCLUDING HEAVY SMOKERS. New Jersey radon-female lung cancer case-control study, 1982-1988

Smoking status	Cumulative radon (pCi/L-years) <25	25-49	50-99	100-155	Trend Zcat[c] (p)	Trend Zcnt[d] (p)
ALL SUBJECTS						
Adjusted by cigarettes/day [LR=188.1, 4df]+	1.0	1.2 (0.87,1.7)	0.87 (0.38,2.0)	7.0 (1.0,48.8)	**1.15 (0.125)**	**1.05 (0.147)**
			1.3[e] (0.61,2.7)			
Adjusted by cigarettes/day, age, occupation, yrs quit smoking, respondent type, resptype*cigs/day [LR=245.3, 17df]+	1.0	1.2 (0.83,1.9)	0.94 (0.41,2.2)	7.2 (1.0,50.3)	**1.34 (0.090)**	**1.22 (0.115)**
			1.4 (0.65,3.0)			
ALL EXCEPT HEAVY SMOKERS						
Adjusted by cigarettes/day [LR=142.5, 3df]+	1.0	1.3 (0.86,2.0)	1.6 (0.63,4.2)	6.8 (0.95,48.5)	**1.98 (0.024)**	**1.90 (0.029)**
			2.2 (0.94,5.2)			
Adjusted by cigarettes/day, age, occupation, yrs quit smoking, respondent type, resptype*cigs/day [LR=190.1, 13df]+	1.0	1.3 (0.85,2.0)	1.6 (0.60,4.1)	6.6 (0.90,48.9)	**1.90 (0.029)**	**1.85 (0.032)**
			2.2 (0.90,5.2)			

+ Likelihood ratio statistic for model including categorical "cumulative radon rend" term (with degrees of freedom)

[a] Odds ratios and 90% confidence intervals from logistic regression analyses.

(contd)

TABLE 15 (contd)

[b] Cumulative radon exposure during 25 years from 5-30 years prior to case diagnosis or control selection; assumes exposure of 0.6 pCi/L (median for controls) for any of the 25 years during which the subject did not live in the index address where the measurements were made.

[c] Z statistic (1-sided p value) for categorical "cumulative radon trend" term in logistic regression model. This term equals 11.8 if cumulative radon is <25 pCi/L-years, 29.4 (25-49 pCi/L-years), 69.4 (50-99 pCi/l-years, or 109.5 (100+ pCi/l-years). These values are the medians of the respective intervals for controls. This model gives results equivalent to the Mantel Chi-extension procedure for stratified analyses.

[d] Z statistic (1-sided p Value) for continuous cumulative radon variable in logistic regression model.

[e] Odds ratio (90% confidence interval) for cumulative radon=50+ pCi/L-years.

TABLE 16
Distribution of lung cancer cases (by histologic type) and controls, by cumulative radon exposure[a]
New Jersey radon-female lung cancer case-control study, 1982-1988

	Cumulative radon (pCi/L-years)				
	<25	25-49	50-99	100-155	Total
Cases					
Squamous cell	97 (88.2%)	11 (10.0%)	1 (0.9%)	1 (0.9%)	110
Small cell	80 (84.2%)	9 (9.5%)	4 (4.2%)	2 (2.1%)	95
Adenocarcinoma	107 (83.0%)	18 (14.0%)	3 (2.3%)	1 (0.8%)	129
Large cell	27 (77.2%)	6 (17.1%)	2 (5.7%)	0 (0.0%)	35
Other types [b]	50 (78.1%)	12 (18.9%)	2 (3.1%)	0 (0.0%)	64
Controls	340 (84.6%)	52 (12.9%)	9 (2.2%)	1 (0.2%)	402

[a] Cumulative radon exposure during 25 years from 5-30 years prior to case diagnosis or control selection; assumes exposure of 0.6 pCi/L (median for controls) for any of the 25 years during which the subject did not live in the index address where the measurements were made.

[b] other histologic types include: mixed adeno-squamous, undifferentiated, anaplastic, poorly differentiated, and malignant (not otherwise specified).

TABLE 17

Adjusted odds ratios[a] (90% confidence intervals) for association of lung cancer, by histologic type, with cumulative radon exposure[b] New Jersey radon-female lung cancer case-control study, 1982-1988

Histologic type	Cumulative radon (pCi/L-years) <25	25-49	50-99	100-155	Trend Zcat[c] (p)	Trend Zcnt[d] (p)
Squamous cell	1.0	0.87 (0.43,1.8)	0.21 (0.03,1.5)	15.4 (1.3,188.5)	-0.21 (—)	-0.22 (0.371)
[LR=149.0, 17df]+			0.58[e] (0.13,2.6)			
Small cell	1.0	1.0 (0.48,3.4)	1.2 (0.35,4.0)	∞ (--,--)	1.41 (0.079)	0.76 (0.224)
[LR=178.2, 17df]+			2.1 (0.68,6.2)			
Adenocarcinoma	1.0	1.5 (0.88,2.5)	0.86 (0.25,2.9)	5.9 (0.52,68.4)	1.00 (0.159)	0.86 (0.195)
[LR=71.0, 17df]+			1.2 (0.40,3.6)			
Large cell[f]	1.0	1.8 (0.71,4.4)	2.2 (0.46,10.9)	0.0 (--,--)	1.14 (0.127)	1.04 (0.149)
[LR=67.0, 14df]+			2.2 (0.45,10.7)			
Other types[g]	1.0	2.1 (1.1,4.1)	0.97 (0.24,4.0)	0.0 (--,--)	0.75 (0.227)	1.11 (0.133)
[LR=49.6, 17df]+			0.94 (0.23,3.8)			

+ Likelihood ratio statistic for model including categorical "cumulative radon trend" term (with degrees of freedom).

[a] Odds ratios (OR) and 90% confidence intervals from logistic regression analyses, adjusted by lifetime average daily cigarette consumption, age, occupation, years since smoking cessation, respondent type, and interaction between respondent type and cigarettes/day. See footnotes, Table 8, for further definitions of these variables. (contd)

TABLE 17 (contd)

[b] Cumulative radon exposure during 25 years from 5-30 years prior to case diagnosis or control selection; assumes exposure of 0.6 pCi/L (median for controls) for any of the 25 years during which the subject did not live in the index address where the measurements were made.

[c] Z statistic (1-sided p value) for categorical "cumulative radon trend" term in logistic regression model. This term equals 11.8 if cumulative radon is <25 pCi/L-years, 29.4 (25-49 pCi/L-years), 69.4 (50-99 pCi/l-years, or 109.5 (100+ pCi/l-years). These values are the medians of the respective intervals for controls. This model gives results equivalent to the Mantel Chi-extension procedure for stratified analyses.

[d] Z statistic (1-sided p value) for continuous cumulative radon variable in logistic regression model.

[e] Odds ratio (90% confidence interval) for cumulative radon=50+ pCi/L-years.

[f] Model including respondent type*cigarettes/day interaction was indeterminate. Results are shown for model without the interaction terms.

[g] Other histologic types include: mixed adeno-squamous, undifferentiated, anaplastic, poorly differentiated, and malignant (not otherwise specified).

TABLE 18
Adjusted odds ratios[a] (90% confidence intervals) for log-linear trend in the association of lung cancer with cumulative radon exposure[b], and derived relative risk coefficients per WLM[c], New Jersey radon-female lung cancer case-control study, 1982-1988

Subject category [p value]	O.R. (90% CI)	O.R. - 1.0[d] (per pCi/L-yr)	Excess Relative Risk (%) per WLM (90% CI)
All subjects [0.115]	1.0067 (0.9977,1.0159)	0.0067	3.4% (0%,8.0%)
All except heavy smokers [0.032]	1.0117 (1.0013,1.0223)	0.0117	5.9% (0.7%,11.2%)
Nonsmokers [0.348]	1.0039 (0.9877,1.0204)	0.0039	2.0% (0%,10.2%)
All Smokers [0.152]	1.0071 (0.9958,1.0185)	0.0071	3.6% (0%,9.3%)
Histologic type:			
Squamous cell [---]	0.9976 (0.9803,1.0153)	------	---- (-------)
Small cell [0.224]	1.0067 (0.9922,1.0214)	0.0067	3.4% (0%,10.7%)
Adenocarcinoma [0.195]	1.0066 (0.9940,1.0193)	0.0066	3.3% (0%,9.7%)
Large cell [0.149]	1.0133 (0.9923,1.0348)	0.0133	6.7% (0%,17.4%)
Other types[e] [0.133]	1.0110 (0.9947,1.0276)	0.0110	5.5% (0%,13.8%)

[a] Odds ratios (OR) and 90% confidence intervals from logistic regression analyses, adjusted by lifetime average daily cigarette consumption, age, occupation, years since smoking cessation, respondent type, and interaction between respondent type and cigarettes/day. See footnotes, Table 8, for further definitions of these variables.

[b] Cumulative radon exposure during 25 years from 5-30 years prior to case diagnosis or control selection; assumes exposure of 0.6 pCi/L (median for controls) for any of the 25 years during which the subject did not live in the index address where the measurements were made. (contd)

TABLE 18 (contd)

c Relative risk coefficient/WLM equals excess relative risk per pCi/L-year (OR - 1.0) divided by 0.20 WLM per pCI/L-year. See Appendix A for equivalences.

d OR - 1.0 equals the log-linear increase in risk per pCi/L-year.

e Other histologic types include: mixed adeno-squamous, undifferentiated, anaplastic, poorly differentiated, and malignant (not otherwise specified).

APPENDICES

A CASE-CONTROL STUDY OF RADON AND LUNG CANCER AMONG NEW JERSEY WOMEN

A. Comparison of units of measurement for radon and cumulative radon exposure.

B. Summary of original New Jersey female lung cancer case-control study

C. Detailed comparison of characteristics of women included vs. not included in the radon substudy

D. Considerations with respect to changes in house construction.

E. Detailed information on radon measurements methods, including quality control aspects and relationships between different radon measurements.

F. Validation of residential histories

G. Comparison with results of statewide radon survey conducted for New Jersey State Department of Environmental Protection

H. Considerations with respect to the house floor on which radon measurements were made.

J. Estimates of year-round living area radon concentrations.

K. Analyses excluding estimates of year-round living area radon concentrations.

APPENDIX A

Units of Exposure for Radon

Exposure intensity concentration

Traditional units used in previous underground mining studies and still in general use in the United States:

Radon gas: picocuries per liter (pCi/L)

Radon decay products: Working Levels (WL)
(radon daughters, radon progeny)

At 100% equilibrium of radon and its decay products, 100 pCi/L corresponds to 1 WL.

At 50% equilibrium (usual assumption), 200 pCi/L corresponds to 1 WL.

S.I.(International) units now in general use in Europe and in most scientific journals:

Radon gas: Becquerels per cubic meters (Bq/m^3)

Radon decay products: Becquerels per cubic meters equilibrium equivalent radon (Bq/m^3 EER)

Cumulative exposure

Traditional units: Working Level Months (WLM)

1 WLM = 1 WL for one "working month" for 170 hr/month

Occupational: 12 WLM/WL per year

Residential (assuming 80% occupancy): 40 WLM/WL per year

S.I. units: Becquerels per cubic meters equilibrium equivalent radon annum (Bq/m^3 EER-a)

Joules hours per cubic meters (J hr/m^3)

Equivalences

Radon gas concentration

1 pCi/L = 37 Bq/m^3

1 Bq/m^3 = 0.027 pCi/L

150 Bq/m^3 = 4 pCi/L (approximately)

Radon decay product concentration

1 WL = 3,700 Bq/m^3 EER

100 Bq/m^3 EER = 0.027 WL

Cumulative exposure

1 WLM = 0.0035 J hr/m^3

(assuming 80% occupancy in residences)

1 WLM = 92.5 Bq/m^3 EER a

1,000 Bq/m^3 EER a = 10.8 WLM

(assuming 80% occupancy and 50% equilibrium between radon and its decay products):

1 WLM = 5 pCi/L-year

1 pCi/L-year = 0.20 WLM

1 pCi/L-year = 18.5 Bq/m^3 EER a

1,000 Bq/m^3 EER a = 27 pCi/L-year

1 pCi/L-year = 0.0007 J hr/m^3

APPENDIX B

Original NJ Female Lung Cancer Case-Control Study: Methods

Cases included all female state residents who were newly diagnosed with histologically confirmed primary cancer of the lung, trachea or bronchus (code 162, International Classification of Diseases, 9th Revision) from August 1982 through September 1983. They were ascertained through a rapid reporting system that the NJ State Department of Health (NJDOH) established with local hospital pathology departments, and by periodic review of hospital pathology records and of State Cancer Registry and death certificate files. Pathology reports, from which the histologic type was determined, and other medical records were reviewed by NJDOH physicians to verify the diagnosis. No slide review was conducted. Some adenocarcinoma cases were designated as "probably in-scope," rather than "*in-scope*," if there was insufficient documentation to rule out completely the possibility of another primary site, particularly breast cancer.

Population-based controls were selected using one of three files. For cases who were themselves interviewed, controls were selected using a random sample of either New Jersey drivers' license files (for ages less than 65) or Health Care Financing Administration Files (for ages 65 or older), and were frequency matched to the cases within race and 5-year age groups. For deceased or incapacitated cases, with next-of-kin respondents, New Jersey State mortality files were used to *select* controls who were individually matched to the cases by race, age, and closest date of death (or date of death closest to date of diagnosis, for incapacitated cases). Controls selected from mortality files were excluded if lung cancer or any other respiratory disease was mentioned on the death certificate.

Subjects or their next-of-kin were personally interviewed in their homes by trained interviewers. Questionnaire items included demographic data, a detailed

brand-specific smoking history, a history of passive exposure to smoking by other household members, a dietary history to determine consumption of foods containing vitamin A, and lifetime residential and occupational histories.

In the cigarette use section of the questionnaire, a smoker first was asked to recall the years in which she smoked cigarettes for any period 6 months or longer. Second, she was asked to recall the brands of cigarettes she smoked during each period, the specific years in which she smoked each brand, the number of cigarettes of each brand smoked per day, and the depth of inhalation for each. The interviewer probed for any changes in number per day of a particular brand; a change greater than 10 per day generated a new data entry. The sequence of temporal episodes yielded the summary measures of years actually smoked and years since cessation (if any). The collection of brand name and intensity records for each episode yielded a lifetime intensity measure, or average number of cigarettes smoked per day. The tar content per cigarette for any brand in any year was determined from historical estimates (Tobacco Merchants Association, 1978; USDHHS, 1981) and test data (Federal Trade Commission, 1976; Federal Trade Commission,1983).

Time-weighted average tar levels were calculated for the interval 1973-1982. This period was selected because (a) precise figures for tar content of all domestically produced cigarettes were available; (b) except for the latter part of the interval, when ultra-low-tar cigarettes became available, this period did not show as sharp a decline in tar content as the two previous decades; and (c) this proximal portion of the smoking history was assumed to be recalled more accurately by both self- and next of kin respondents.

Diet was assessed by asking about the usual frequency of consumption, approximately 4 years earlier, of 59 food items, including major sources of preformed retinol and carotenoids. For fruits and vegetables that the respondent

said were eaten primarily in certain seasons, frequency of consumption both in season and out of season and the length of season were obtained. In these analyses, the average frequency of consumption was calculated for the food group "vegetables", which in an earlier study of lung cancer among New Jersey white males (Ziegler, 1986) had shown the strongest, most consistent inverse association with lung cancer risk. The variable used in these analyses was calculated using exactly the same food items as had been used in the earlier study. However, it should be noted that the questionnaire used in this study also asked about consumption of several additional vegetables; this additional information is not yet included in the analyses. Therefore, the dietary associations presented here are preliminary. However, it is doubtful that this will strongly affect the degree to which diet confounds any association with radon.

In the occupational history section of the questionnaire, information was obtained on each full-time or part-time job held for 3 months or more since age 12. This included the name and address of employer; type of business; job title; duties performed; materials handled; exposure to solvents, fumes, or dust; and time period of employment. All industry and job title information was coded using the 1970 census index system (US Bureau of Census, 1971). Job title categories and industry-job title categories (selected job titles from specified industries; only those potentially exposed, excluding most clerical, administrative, and sales personnel) were chosen for analysis after an extensive literature review, with particular attention to Dubrow and Wegman's summary of occupational surveillance studies (Dubrow and Wegman, 1983). For this study, categories were also chosen if they represented occupations in which women were frequently employed.

In the passive smoking exposure section of the questionnaire, a subject was asked whether any member of her household ever smoked. This included parents and other members of her family while she was growing up, and her spouse if she

was ever married. This was followed by questions relating to who these individuals were, how long the subject lived with them while they smoked, what they smoked (cigarettes, cigars, pipes, or a combination of these), and how much they smoked.

Original NJ Female Lung Cancer Study: Results

Interviews were successfully completed for 994 (76%) of the 1306 female lung cancer cases identified, and for 995 (69%) of the 1449 female controls identified. Reasons for non-response are detailed in Table B1. Response rates were similar for all major histologic types. Of the cases interviewed, 269 (27%) were squamous cell carcinoma, 220 (22%) were small cell carcinoma, 290 (29%) were adenocarcinoma, and 215 (22%) were other histologic types. Of the cases with adenocarcinoma, 191 were judged "in-scope" and 99, "probably in-scope."

Table B2 shows the distribution of the 994 cases and 995 controls by various risk factors and subject characteristics. The overall distributions of cases and controls by age, race, and respondent type were by design very similar. The median ages for cases and controls were both 65 years. [However, the age distributions varied significantly among cases by histologic type, with a greater proportion of adenocarcinoma cases in the younger age stratum. The median ages for squamous cell, small cell, adenocarcinoma, and other histologic types were 65, 67, 63, and 63, respectively.] Ten per cent of the cases and 9 per cent of the controls were non-white. Interviews for 54 per cent of the cases and 53 per cent of the controls were conducted with the subjects themselves. The remaining interviews were conducted with next of kin, either the spouse (16% for cases, 19% for controls), or other next of kin (31% for cases, 28% for controls). The majority of the other next of kin respondents were daughters, sons, or sisters (e.g., for controls, 13%, 8%, and 3%, respectively). The distribution of respondent type among controls and among

cases varied significantly with age. For the younger subjects, a larger percentage were interviewed themselves; for the older subjects, a larger percentage had next of kin interviews, with an increasing proportion of other (nonspouse) respondents.

The percentages of cases and controls varied significantly with several different measures of cigarette smoking, including lifetime average number of cigarettes smoked per day, total duration of smoking, number of years since smoking cessation, and average tar content of cigarettes smoked during 1973-1982 (Table B2). More cases than controls were heavy smokers (25+ cigarettes/day) or moderate smokers (15-24 cigarettes/day) than light smokers (<15 cigarettes/day) or lifetime nonsmokers. More cases than controls had smoked for 35+ years rather than <35 years. More cases than controls were current smokers (quit 0-1 years) or recent ex-smokers (quit 2-9 years) than long-term ex-smokers (quit 10+ years). More cases than controls were smokers of high tar (21+ mg) cigarettes than lower tar (<21 mg) cigarettes.

More cases than controls were low consumers of vegetables (<35 servings/month) than high consumers (75+ servings/month).

In analyses of occupation, many of the job title categories considered to be high risk based on our literature review and/or found to be high risk for New Jersey males (Schoenberg et al, 1987) were not represented in the female data set. For example, there were no women ever employed as blacksmiths, boilermakers, brickmasons, automobile mechanics, plasterers, plumbers and pipefitters, roofers, or stationary engineers and firemen. There were only one or two metal molders, sheetmetal workers, asbestos insulation workers, furnacemen, and construction laborers. The smoking adjusted odds ratios (OR) were significantly high for professional and photographic equipment manufacturing workers [22 cases, 9 controls; OR=2.3; 95% confidence interval (CI) = 1.0, 5.1] and for laundry and dry cleaning workers [73 cases, 41 controls; OR=1.5; 95% CI = 1.0, 2.3]. The smoking

adjusted OR was also high for plastics manufacturing workers [29 cases, 15 controls; OR=1.9; 95% CI = 0.98, 3.9] and for food counter workers [36 cases, 21 controls, OR=1.8; 95% CI = 0.98, 3.3]. In addition, women who had worked as restaurant or food service workers for more than 20 years had a significantly high smoking adjusted risk [28 cases, 9 controls; OR=2.8; 95% CI = 1.2, 6.8].

Some of the industry and job title categories showed an excess of cases over controls, but the numbers of subjects were very small. These categories included petroleum industry [2 cases, 0 controls], construction industry [2 cases, 0 controls], lumber and wood products manufacturing [3 cases, 2 controls], asbestos products manufacturing [4 cases, 2 controls], primary iron and steel manufacturing [4 cases, 1 control], transportation industry workers excluding drivers [4 cases, 3 controls], drivers (irrespective of industry) [10 cases, 3 controls], gas stations and garage workers [4 cases, 1 control], painters [7 cases, 5 controls] and bartenders [3 cases, 0 controls]. It is not possible, given the limited number of subjects in these latter categories, to attribute any statistical significance to the findings of excess cases. However, in carrying out the analyses for the radon study, it was important to control adequately for any potential confounding by occupation in these data. Therefore, it was decided to include these small categories, along with the five larger categories mentioned above (professional and photographic equipment, laundry and dry cleaning, etc.) in an overall a posteriori high-risk occupation category, which would represent the potential influence of occupation. As shown in Table B2, 203 cases and 103 controls were represented in this high-risk occupation category. However, because this category was constructed after looking at the data, it is not possible to attribute any statistical significance to a derived risk estimate.

There was little difference between cases and controls in the percentage of subjects by educational level (Table B2). Slightly more cases than controls lived in the "low radon" counties (Atlantic, Cape May, Essex, Hudson, Ocean), while slightly

more controls than cases lived in the "moderately low radon" counties (Bergen, Burlington, Cumberland, Gloucester, Middlesex, and Union). [See footnote e, Table B2, for further details on the grouping of counties according to radon level.]

Among lifetime nonsmokers only [116 cases, 499 controls], there were slightly more cases than controls who were exposed to spouse cigarette smoke, and fewer cases than controls who were exposed to spouse smoke from only pipes or cigars.

Table B3 shows the odds ratios and 95% confidence intervals estimated for these risk factors and subject characteristics using multiple logistic regression analysis (Breslow and Day, 1980) as carried out using the microcomputer-based LOGRESS program (McGee, 1986). Of the several variables for smoking, only lifetime average number of cigarettes smoked per day was considered in this initial analysis. Smoking was the major risk for lung cancer among these women, with risks ranging from 4.4 for light smokers to 14.4 for heavy smokers. After controlling for smoking, the age variable (72+ years compared to 58-71 years)[1], the vegetable consumption variable (both low and moderate consumption) and the occupation variable showed moderate increases in risk. Respondent type, race, education, and county group had little influence on lung cancer risk, after adjusting for smoking, age, diet, and occupation.

Even though respondent type was not an appreciable confounder for the associations between lung cancer and smoking, there were significant differences in the smoking-related risk by respondent type, i.e., there was significant interaction (Schoenberg et al, 1989). Table B4 shows results of an analysis similar to that shown in Table B3, but including interaction terms between respondent type and each of the three variables for the smoking groups according to cigarettes per day.

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[1] The risk associated with increased age emerges despite the fact the the original case and control series were age matched. This reflects reverse confounding by smoking. It also reflects a cohort effect, in that subjects age 72+ were more often nonsmokers than were subjects younger than age 72.

The OR for the three cigarettes per day groups (i.e., the main effects in the model) now reflect the smoking-associated risk in subjects who were self-respondents, with risks ranging from 6.2 in light smokers to 27.7 in heavy smokers. The OR for the three respondent type* cigarettes per day groups (i.e., the interaction terms), when multiplied by the OR for the main effects, yield the OR for subjects with next of kin respondents, with risks ranging from 3.2 for light smokers to 9.1 for heavy smokers. The hypothesized reasons for this significant interaction have been discussed extensively elsewhere (Schoenberg et al, 1989). Actual differences in smoking between living and deceased controls may explain some of the risk differences by respondent type. However, misclassification by next of kin respondents seems as likely an explanation, given the significantly lower percentage of smokers reported by next of kin for cases. The possibility of misclassification is also consistent with differences in the degree of respondent type heterogeneity observed in histologic type specific smoking risks.

Because smoking is such an important risk factor, it was not considered sufficient to control only for lifetime average number of cigarettes smoked per day. Table B5 shows the results of analyses considering total duration of smoking, number of years since smoking cessation, or average cigarette tar content (1973-1982), in addition to lifetime average daily cigarette consumption. All of these analyses are adjusted for age, race, respondent type, vegetable consumption, occupation, and education (but not for the interaction between smoking and respondent type). Inclusion of any of the three detailed smoking variables shows a highly signficant improvement in the overall fit of the model, as measured by the increase in the likelihood ratio statistic [duration of smoking, Chi-square=40.9, 4 df, $p < 0.0001$; years since smoking cessation, Chi-square=78.6, 6 df, $p < 0.0001$; cigarette tar content, Chi-square=59.0, 6 df, $p < 0.0001$]. Within light, moderate, or heavy smokers, risk increases systematically with increasing number of years

smoked, with decreasing number of years since smoking cessation (if any), or with increasing cigarette tar content. The maximum risk is 18.8 for heavy smokers who smoked 35+ years, 20.5 for heavy current smokers, or 33.2 for heavy smokers of high tar cigarettes.

Logistic regression analyses for passive smoking were limited to lifetime nonsmokers (Table B6). After adjusting for age, race, respondent type, vegetable consumption, occupation, and education, there was a very slight, non-significant increase in risk associated with exposure to spouse cigarette smoke, and a nonsignificant decrease in risk associated with exposure to spouse smoke only from pipes or cigars. Previous analyses (not shown) gave the same results when exposure to smoke from any household member, not just the spouse, was considered. Also, previous analyses according to the duration of exposure to spouse cigarette smoke, or the reported intensity of exposure (number of cigarettes smoked per day in the house) did not show any systematic relationship, after adjusting for age. Therefore, only the spouse smoking variable shown in these tables has been used in the radon study analyses.

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TABLE B1
Distribution of lung cancer cases and controls by survey outcome, New Jersey female lung cancer case-control study, 1982-1983

	No. of Cases	(%)	No. of Controls	(%)
Total eligible subjects	1,306	(100%)	1,449	(100%)
Cases with no contact attempted*	81	(6.2%)	0	(0%)
Subjects with no respondents available for interview+	71	(5.4%)	91	(6.3%)
Subjects contacted for interview	1,154	(88.4%)	1,358	(93.7%)
Refusal	160	(12.3%)	363	(25.1%)
Completed interviews	994	(76.1%)	995	(68.7%)

* Includes 54 (4.1%) cases for whom physician refused permission to contact, and 27 (2.1%) deceased cases for whom the mandatory waiting period before contacting next of kin had not elapsed by the end of the survey.

+ Includes 14 (1.1%) live cases who moved from the area or were untraceable, 45 (3.1%) live controls who were untraceable, 4 (0.3%) live controls with language problems, and 57 (4.4%) deceased cases and 42 (2.9%) deceased controls with no next of kin, with next of kin who were unfamiliar with subject's history, or with next of kin who had moved or were untraceable.

TABLE B2
Number of lung cancer cases and controls in original study, by various risk factors and subject characteristics, New Jersey female lung cancer case-control study, 1982-1983

	No. of cases	(%)	No. of controls	(%)
TOTAL	994		995	
AGE AT DIAGNOSIS[a]				
<58 years	255	(25.7%)	249	(25.0%)
58-71 years	489	(49.2%)	485	(48.7%)
72+ years	250	(25.2%)	261	(26.2%)
RESPONDENT TYPE				
self	532	(53.5%)	528	(53.1%)
spouse	155	(15.6%)	188	(18.9%)
other next of kin	307	(30.9%)	279	(28.0%)
RACE				
white, including hispanic	899	(90.4%)	910	(91.5%)
nonwhite	95	(9.6%)	85	(8.5%)
CIGARETTES/DAY [b]				
Lifetime nonsmoker	116	(11.7%)	499	(50.2%)
< 15 cigarettes/day	198	(19.9%)	210	(21.1%)
15-24 cigarettes/day	414	(41.7%)	195	(19.6%)
25+ cigarettes/day	266	(26.8%)	91	(9.2%)
TOTAL NUMBER OF YEARS SMOKED				
Lifetime nonsmoker	116	(11.7%)	499	(50.2%)
< 35 years	219	(22.0%)	235	(23.6%)
35+ years	659	(66.3%)	261	(26.2%)
NUMBER OF YEARS QUIT SMOKING				
Lifetime nonsmoker	116	(11.7%)	499	(50.2%)
Current smoker, quit 0-1 years	712	(71.6%)	289	(29.1%)
Ex-smoker, quit 2-9 years	103	(10.4%)	78	(7.8%)
Ex-smoker, quit 10+ years	63	(6.3%)	129	(13.0%)
AVERAGE CIGARETTE TAR CONTENT, 1973-1982				
Lifetime nonsmoker	116	(11.7%)	499	(50.2%)
Nonsmoker, 1973-1982	60	(6.0%)	116	(11.7%)
Smoker, tar <21 mg/cigarette	664	(66.8%)	336	(33.8%)
Smoker, tar 21+ mg/cigarette	154	(15.5%)	44	(4.4%)
VEGETABLE CONSUMPTION[a]				
<35 servings/month	284	(28.6%)	238	(23.9%)
35-74 servings/month	532	(53.5%)	510	(51.3%)
75+ servings/month	178	(17.9%)	247	(24.8%)

(contd)

TABLE B2 (contd)
Number of lung cancer cases and controls in original study, by various risk factors and subject characteristics, New Jersey female lung cancer case-control study, 1982-1983

	No. of cases (%)	No. of controls (%)
TOTAL	994	995
HIGH-RISK OCCUPATION [c]		
no high-risk occupation	791 (79.6%)	892 (89.6%)
high-risk occupation	203 (20.4%)	103 (10.4%)
EDUCATION		
<8 years completed	127 (12.8%)	144 (14.5%)
8-12 years completed	648 (65.2%)	605 (60.8%)
13+ years completed	219 (22.0%)	246 (24.7%)
COUNTY AT DIAGNOSIS[d]		
Low radon	300 (30.2%)	265 (26.6%)
Moderately low radon	354 (35.6%)	389 (39.1%)
Moderate radon	190 (19.1%)	195 (19.6%)
High radon	150 (15.1%)	146 (14.7%)
LIFETIME NONSMOKERS ONLY, BY PASSIVE SMOKING		
No exposure to spouse tobacco	43 (37.1%)	196 (39.3%)
Exposure to spouse cigarette smoke	66 (56.9%)	250 (50.1%)
Exposure to spouse pipe/cigar only	7 (6.0%)	53 (10.6%)

[a] Cutpoints based on distribution of controls in original female lung cancer study (1st quartile; 2nd+3rd quartiles; 4th quartile).
[b] Lifetime average number of cigarettes smoked per day. Cutpoints based on bimodal distribution of controls in original female lung cancer study, with peaks at 10 and 20 cigarettes per day, and a long tail starting at 25 cigarettes per day.
[c] Ever employed in any occupational group shown to have a smoking adjusted risk of 1.5 or greater in the original female lung cancer study. This is an a posteriori definition, used only for the purpose of adjusting in the radon analyses for the possible effect of occupational exposure. See text for further clarification of this variable.
[d] County at diagnosis for cases, or county at ascertainment for controls. Low radon counties include include Atlantic, Cape May, Essex, Hudson, and Ocean counties. Moderately low radon counties include Bergen, Burlington, Cumberland, Gloucester, Middlesex, and Union counties. Moderate radon counties include Camden, Monmouth, Passaic, and Salem counties. High radon counties include Hunterdon, Mercer, Morris, Somerset, Sussex, and Warren counties. Grouping of counties was determined by the percentage of houses with basement or lower level screening values above 4 pCi/L in the statewide survey conducted for the New Jersey State Department of Environmental Protection (see Appendix G).

TABLE B3
Odds ratios[a] (95% confidence intervals) for association of lung cancer with various risk factors and subject characteristics
New Jersey female lung cancer case-control study, 1982-1983

	Odds ratio	(95% CI)
AGE		
<58 years	0.83	(0.64, 1.1)
58-71 years	1.0[b]	---
72+ years	1.7	(1.3, 2.2)
RESPONDENT TYPE[c]		
self	1.0[b]	---
spouse, other next of kin	0.88	(0.71, 1.1)
RACE		
white, including hispanic	1.0[b]	---
nonwhite	1.2	(0.81, 1.7)
CIGARETTES/DAY [d]		
Lifetime nonsmoker	1.0[b]	---
< 15 cigarettes/day	4.4	(3.3, 5.9)
15-24 cigarettes/day	10.4	(7.9, 13.8)
25+ cigarettes/day	14.5	(10.4, 20.2)
VEGETABLE CONSUMPTION		
<35 servings/month	1.5	(1.1, 2.0)
35-74 servings/month	1.4	(1.1, 1.8)
75+ servings/month	1.0[b]	---
HIGH-RISK OCCUPATION		
no high-risk occupation	1.0[b]	---
high-risk occupation	2.0	---[e]
EDUCATION		
<8 years completed	0.95	(0.70, 1.3)
8-12 years completed	1.0[b]	---
13+ years completed	0.96	(0.75, 1.2)
COUNTY AT DIAGNOSIS[f]		
Low radon	1.0[b]	
Moderately low radon	0.91	(0.70, 1.2)
Moderate radon	0.80	(0.60, 1.1)
High radon	0.91	(0.66, 1.3)

[a] Odds ratios (95% confidence interval) from logistic regression analysis for 994 cases and 995 controls in original female lung cancer study.
[b] Reference group: odds ratio=1.0; no confidence interval calculated.
[c] Analyses showed no appreciable confounding by respondent type, and no differences in other estimated odds ratios if next of kin were separated into spouse next of kin and other next of kin; therefore, results are shown only for the self vs. next of kin comparison. (contd)

Table B3 (contd)

[d] lifetime average number of cigarettes per day
[e] no significance testing or confidence limits shown for a posteriori occupation category.
[f] County at diagnosis for cases, or county at ascertainment for controls. Low radon counties include include Atlantic, Cape May, Essex, Hudson, and Ocean counties. Moderately low radon counties include Bergen, Burlington, Cumberland, Gloucester, Middlesex, and Union counties. Moderate radon counties include Camden, Monmouth, Passaic, and Salem counties. High radon counties include Hunterdon, Mercer, Morris, Somerset, Sussex, and Warren counties. Grouping of counties was determined by the percentage of houses with basement or lower level screening values above 4 pCi/L in the statewide survey conducted for the New Jersey State Department of Environmental Protection (see Appendix G).

TABLE B4

Odds ratios[a] (95% confidence intervals) for association of lung cancer with various risk factors and subject characteristics, including interaction between smoking and respondent type, New Jersey female lung cancer case-control study, 1982-1983

	Odds ratio (95% CI)	
AGE		
<58 years	0.81	(0.63, 1.1)
58-71 years	1.0[b]	---
72+ years	1.6	(1.3, 2.1)
RESPONDENT TYPE[c]		
self	1.0[b]	---
spouse, other next of kin	1.6	(1.0, 2.4)
RACE		
white, including hispanic	1.0[b]	---
nonwhite	1.2	(0.81, 1.7)
CIGARETTES/DAY [d]		
Lifetime nonsmoker	1.0[b]	---
< 15 cigarettes/day	6.2	(4.1, 9.3)
15-24 cigarettes/day	14.7	(9.9, 22.0)
25+ cigarettes/day	27.7	(16.3, 47.2)
RESPONDENT TYPE * CIGARETTES/DAY		
Next of kin*Nonsmoker	1.0[b]	---
Next of kin*<15 cigarettes/day	0.51	(0.28, 0.91)
Next of kin*15-24 cigarettes/day	0.51	(0.30, 0.89)
Next of kin*25+ cigarettes/day	0.33	(0.17, 0.63)
VEGETABLE CONSUMPTION		
<35 servings/month	1.5	(1.1, 2.0)
35-74 servings/month	1.4	(1.1, 1.8)
75+ servings/month	1.0[b]	---
HIGH-RISK OCCUPATION		
no high-risk occupation	1.0[b]	---
high-risk occupation	2.0	---[e]
EDUCATION		
<8 years completed	0.96	(0.70, 1.3)
8-12 years completed	1.0[b]	---
13+ years completed	0.96	(0.75, 1.2)
COUNTY AT DIAGNOSIS[f]		
Low radon	1.0[b]	
Moderately low radon	0.92	(0.72, 1.2)
Moderate radon	0.79	(0.59, 1.1)
High radon	0.91	(0.66, 1.3)

(contd)

Table B4 (contd)

[a] Odds ratios (95% confidence interval) from logistic regression analysis for 994 cases and 995 controls in original female lung cancer study.
[b] Reference group: odds ratio=1.0; no confidence interval calculated.
[c] Analyses showed no appreciable confounding by respondent type, and no significant differences in other estimated odds ratios if next of kin were separated into spouse next of kin and other next of kin; therefore, results are shown only for the self vs. next of kin comparison.
[d] lifetime average number of cigarettes per day
[e] no significance testing or confidence limits shown for a posteriori occupation category.
[f] County at diagnosis for cases, or county at ascertainment for controls. Low radon counties include include Atlantic, Cape May, Essex, Hudson, and Ocean counties. Moderately low radon counties include Bergen, Burlington, Cumberland, Gloucester, Middlesex, and Union counties. Moderate radon counties include Camden, Monmouth, Passaic, and Salem counties. High radon counties include Hunterdon, Mercer, Morris, Somerset, Sussex, and Warren counties. Grouping of counties was determined by the percentage of houses with basement or lower level screening values above 4 pCi/L in the statewide survey conducted for the New Jersey State Department of Environmental Protection (see Appendix G).

TABLE B5

Odds ratios[a] (95% confidence intervals) for association of lung cancer with detailed smoking characteristics
New Jersey female lung cancer case-control study, 1982-1983

	Odds ratio	(95% CI)
CIGARETTES/DAY+TOTAL YEARS SMOKED		
Lifetime nonsmoker	1.0[b]	---
< 15 cigarettes/day		
<35 years	2.9	(2.0, 4.1)
35+ years	6.1	(4.3, 8.6)
15-24 cigarettes/day		
<35 years	5.8	(4.0, 8.6)
35+ years	12.7	(9.3, 17.2)
25+ cigarettes/day		
<35 years	6.1	(3.7, 10.1)
35+ years	18.8	(13.0, 27.3)
CIGARETTES/DAY+YEARS QUIT SMOKING		
Lifetime nonsmoker	1.0[b]	---
< 15 cigarettes/day		
Ex-smoker, quit 10+ years	1.7	(1.1, 2.8)
Ex-smoker, quit 2-9 years	4.8	(2.7, 8.4)
Current smoker, quit 0-1 years	6.8	(4.8, 9.6)
15-24 cigarettes/day		
Ex-smoker, quit 10+ years	4.0	(2.3, 7.1)
Ex-smoker, quit 2-9 years	7.0	(4.2, 11.6)
Current smoker, quit 0-1 years	13.3	(9.8, 18.0)
25+ cigarettes/day		
Ex-smoker, quit 10+ years	2.0	(0.80, 5.4)
Ex-smoker, quit 2-9 years	7.1	(3.6, 14.0)
Current smoker, quit 0-1 years	20.5	(14.1, 29.6)
CIGARETTES/DAY+CIGARETTE TAR, 1973-1982		
Lifetime nonsmoker	1.0[b]	---
< 15 cigarettes/day		
Nonsmoker, 1973-1982	1.9	(1.2, 3.0)
Smoker, tar <21 mg/cigarette	5.4	(3.9, 7.6)
Smoker, tar 21+ mg/cigarette	10.2	(4.8, 21.7)
15-24 cigarettes/day		
Nonsmoker, 1973-1982	4.2	(2.3, 7.6)
Smoker, tar <21 mg/cigarette	11.1	(8.2, 15.0)
Smoker, tar 21+ mg/cigarette	14.0	(8.4, 23.2)
25+ cigarettes/day		
Nonsmoker, 1973-1982	2.1	(0.81, 5.6)
Smoker, tar <21 mg/cigarette	15.0	(10.5, 21.4)
Smoker, tar 21+ mg/cigarette	33.2	(15.2, 72.5)

[a] Odds ratios (95% confidence interval) from logistic regression analysis for 994 cases and 995 controls in original female lung cancer study, adjusting for age, race, respondent type, vegetable consumption, occupation, and education.
[b] Reference group: odds ratio=1.0; no confidence interval calculated.

TABLE B6
Odds ratios[a] (95% confidence intervals) for association of lung cancer with passive smoke exposure (lifetime nonsmokers only)
New Jersey female lung cancer case-control study, 1982-1983

	Odds ratio	(95% CI)
PASSIVE SMOKE EXPOSURE		
No exposure to spouse tobacco	1.0[b]	---
Exposure to spouse cigarette smoke	1.2	(0.75,1.8)
Exposure to spouse pipe/cigar smoke only	0.52	(0.22,1.3)

[a] Odds ratios (95% confidence interval) from logistic regression analysis for 116 cases and 499 controls in original female lung cancer study, adjusting for age, race, respondent type, vegetable consumption, occupation, and education.

[b] Reference group: odds ratio=1.0; no confidence interval calculated.

APPENDIX C

Comparison of Women Included vs Not Included in Radon Study

The proportion of women from the original study who were included in the radon study was examined in subgroups defined by the variables shown in Table B2, i.e., by age, respondent type, etc. The results of these analyses are shown in Table C1. Only one subgroup, subjects who were smokers for less than 35 years, showed a statistically significant ($p < 0.05$) difference between cases and controls in the proportion of women included in the radon study. However, among both lifetime nonsmokers and moderate smokers (15-24 cigarettes/day), among long-term ex-smokers, among self respondents, and among whites, there also were proportionally fewer controls than cases who were included in the radon study ($0.05 \leq p < 0.10$). Most of these case-control differences were attributable to more refusals by current residents of control index residences. For short-duration smokers and for long-term ex-smokers, more controls did not meet the residence criterion.

Within cases and/or within controls, there were several statistically significant differences in the proportion of subjects who were included in the radon study. For both cases and controls, there were smaller proportions of young subjects (age < 58) who were included in the radon study, because of the higher percentage of these young subjects who did not meet the residence criterion. There were also smaller proportions of subjects with other next of kin (nonspouse) respondents who were included. Among cases, there were proportionally more subjects with other next of kin respondents for whom we could not obtain specific address information; among controls, there was a higher percentage of subjects who did not meet the residence criterion. There were also significantly fewer nonwhite subjects than white subjects from the original study who were included in the radon study. A higher percentage of nonwhite subjects did not meet the residence

criterion; there were also more nonwhites for whom specific address information could not be obtained.

Within cases and controls, there were no statistically significant differences in the proportion of subjects included in the radon study according to cigarettes smoked per day. However, radon study subjects had higher proportions of nonsmokers and light smokers, and lower proportions of moderate and heavy smokers. Controls who had smoked less than 35 years, cases who were current smokers, and cases who smoked high tar cigarettes had significantly lower proportions of subjects included in the radon study, primarily because fewer of these subgroups met the residence criterion.

There were significant increases in the proportion of original subjects included in the radon study with increasing educational level. This was related to differences in the percentage of subjects for whom address specific information could not be obtained, as well as differences in the percentages of subjects whose index residence could not be tested for radon (because of refusal or because the house had been demolished). These differences persisted when the analyses were restricted only to white subjects.

Those counties with lower proportions of cases or controls included in the radon study also had higher proportions who did not meet the residence criterion. There was little difference by county group in the proportion with no radon testing at the index residence, or in the proportion with refusal by the current resident.

Control nonsmokers with no reported spouse tobacco exposure also had a lower proportion included in the radon study, because there were more subjects for whom specific address information could not be obtained.

Although the case-control differences in radon study inclusion appeared to be minimal within most subgroups, the differences in risk factor distributions were frequent. Therefore, we repeated the logistic regression analyses shown in Appendix

B, with a variable representing inclusion in the radon substudy (Table C2). Among all subjects (TOTAL), adjusting for all other risk factors and subject characteristics shown, it was significantly more likely for a case than a control to be included in the radon substudy [OR = 1.3; 95% CI= 1.1, 1.6]. Given the smoking-related differences in the radon-associated risk observed in this study, it was also important to assess this bias potential within subgroups according to smoking. The radon study inclusion term was marginally significant for nonsmokers [OR = 1.5; 95% CI= 0.98, 2.3] and significant for moderate smokers [OR = 1.5; 95% CI= 1.0, 2.1]. Light smokers, who showed the strongest radon-associated risk, showed the least bias potential. Heavy smokers, who showed no radon-associated risk, also showed little bias potential.

Table C2 also shows the odds ratios for other risk factors within the smoking subgroups, after controlling for inclusion in the radon study. These odds ratios suggest that proportionally more cases with next of kin respondents were reported to be nonsmokers, while proportionally fewer cases with next of kin respondents were reported to be moderate or heavy smokers. The differences in smoking-associated risk according to respondent type discussed in Appendix B are consistent with the possibility of misclassification of smoking by next of kin respondents.

Among nonsmokers, the odds ratios for residence in the three higher radon county groups are significantly low, relative to residence in the low radon county group [moderately low: OR=0.57, 95% CI=0.34, 0.94; moderate: OR= 0.43, 95% CI=0.22, 0.85; high: OR = 0.59, 95% CI= 0.30, 1.2]. Among heavy smokers, the odds ratio for residence in the moderately low radon county group, relative to residence in the low radon counties, is marginally significantly high [OR = 2.0 (0.98, 3.9)]. These observations suggest the possibility that other, as yet undetermined, geographically-associated risk factors might be operating to mask any slight radon effect in nonsmokers or heavy smokers.

Another means of examining the possibility of bias in the radon substudy population is to compare odds ratios for other risk factors and subject characteristics in those subjects included vs. those subjects not included. Table C3 shows the results of such analyses, for the total study population, and for subgroups according to smoking. The 95% confidence intervals for the odds ratios are not shown; however, any statistically significant ($p < 0.05$) or marginally signficant ($p < 0.10$) differences in odds ratios between those included and not included are noted. In the total study population, the only marginally significant difference was in the odds ratio for moderate, long-term ex-smokers relative to lifetime nonsmokers. This difference also appeared in the comparisons for the moderate smokers. The only other statistically significant differences in odds ratios were for the heavy smokers. Those heavy smokers included in the radon study showed a pattern of increasing risk with increasing vegetable consumption, which was opposite to that observed for the heavy smokers not included in the radon study, and opposite to that observed for most other subgroups. The heavy smokers included in the radon study also showed a significantly low risk associated with fewer years of school, compared to no association in the heavy smokers who were not included in the radon study.

The absence of a radon-association among heavy smokers (in fact, the suggestion of a negative trend, although non-significant) suggested that the heavy smokers included in the radon study might be unusual in some respects. The differences observed in Table C3 are consistent with this suspicion, and suggest that the heavy smokers should be examined in greater detail. Table C4 replicates Table C1, but exclusively for heavy smokers. Although the numbers are small, several observations are noteworthy. There is a pronounced deficit of subjects with less than eight years of school among the heavy smoker cases included in the radon study. There is also a deficit of subjects with high vegetable consumption among the heavy smoker controls included in the radon study.

Conclusions The original female lung cancer study was population-based in design. However, in establishing the residence criterion for the radon substudy, and in eliciting cooperation from the current residents of the index addresses, the radon study was no longer population-based. Overall, there was significantly greater participation for cases in the original study than for controls. Moreoever, radon study subjects were more often older, whites, either nonsmokers, light smokers, or exsmokers, residents of counties with higher radon levels, and more highly educated. Nonetheless, there were relatively few significant differences in other risk factors and subject characteristics between those included and not included, except among heavy smokers, who showed some highly unusual risk factor distributions. The significant differences in smoking-related risk by respondent type discussed in Appendix B are consistent with the possibility of misclassification of smoking by next of kin respondents. The possibility of a case response bias and further misclassification of smoking, even by subject respondents, is purely speculative, but cannot be ruled out. All of these factors suggest that the results of this study with respect to differences in radon-associated risk according to smoking status need to be evaluated very carefully in other study populations before they are accepted as proven.

TABLE C1
Total number of women in original study,
and number and percentage (%) included in radon study[a],
by various risk factors and subject characteristics,
New Jersey radon-female lung cancer case-control study, 1982-1988

	CASES			CONTROLS			
	TOTAL	RADON		TOTAL	RADON		
	N	N	(%)	N	N	(%)	[p[b]]
TOTAL	994	433	(43.6%)	995	402	(40.4%)	[0.17]
AGE AT DIAGNOSIS							
<58 years	255	98	(38.4%)	249	78	(31.3%)	[0.11]
58-71 years	489	215	(44.0%)	485	216	(44.5%)	[0.91]
72+ years	250	120	(48.0%)	261	108	(41.4%)	[0.16]
[p[c] (d.f.)]	[0.09 (2)]			[0.002 (2)]			
RESPONDENT TYPE							
Self	532	246	(46.2%)	528	212	(40.2%)	[0.05]
Spouse	155	74	(47.8%)	188	89	(47.3%)	[0.99]
Other next of kin	307	113	(36.8%)	279	101	(36.2%)	[0.95]
[p[c] (d.f.)]	[0.02 (2)]			[0.06 (2)]			
RACE							
White	899	418	(46.5%)	910	386	(42.4%)	[0.09]
Nonwhite	95	15	(15.8%)	85	16	(18.8%)	[0.73]
[p[c] (d.f.)]	[<0.001 (1)]			[<0.001 (1)]			
CIGARETTES/DAY[b]							
Lifetime nonsmoker	116	61	(52.6%)	499	213	(42.7%)	[0.07]
<15 cigarettes/day	198	83	(41.9%)	210	90	(42.9%)	[0.93]
15-24 cigarettes/day	414	178	(43.0%)	195	67	(34.4%)	[0.05]
25+ cigarettes/day	266	111	(41.7%)	91	32	(35.2%)	[0.33]
[p[c] (d.f.)]	[0.21 (3)]			[0.13 (3)]			
TOTAL NO. YEARS SMOKED							
Lifetime nonsmoker	116	61	(52.6%)	499	213	(42.7%)	[0.07]
<35 years	219	90	(41.1%)	235	72	(30.6%)	[0.03]
35+ years	659	282	(42.8%)	261	117	(44.8%)	[0.63]
[p[c] (d.f.)]	[0.10 (2)]			[0.002 (2)]			
NO. YEARS QUIT SMOKING							
Lifetime nonsmoker	116	61	(52.6%)	499	213	(42.7%)	[0.07]
Quit 0-1 years	712	289	(40.6%)	289	112	(38.8%)	[0.64]
Quit 2-9 years	103	49	(47.6%)	78	27	(34.6%)	[0.11]
Quit 10+ years	63	34	(54.0%)	129	50	(38.8%)	[0.07]
[p[c] (d.f.)]	[0.02 (3)]			[0.45 (3)]			

(contd)

TABLE C1 (contd)
Total number of women in original study,
and number and percentage (%) included in radon study[a],
by various risk factors and subject characteristics,
New Jersey radon-female lung cancer case-control study, 1982-1988

	CASES			CONTROLS			
	TOTAL	RADON		TOTAL	RADON		
	N	N	(%)	N	N	(%)	[p[b]]
TOTAL	994	433	(43.6%)	995	402	(40.4%)	[0.17]
AVG. CIGARETTE TAR CONTENT, 1973-1982							
Lifetime nonsmoker	116	61	(52.6%)	499	213	(42.7%)	[0.07]
Nonsmoker, 1973-1982	60	31	(51.7%)	116	45	(38.8%)	[0.14]
Tar <21 mg/cigarette	664	284	(42.8%)	336	126	(37.5%)	[0.13]
Tar 21+ mg/cigarette	154	57	(37.0%)	44	18	(40.9%)	[0.77]
[p[c] (d.f.)]	[0.04 (3)]			[0.50 (3)]			
VEGETABLE CONSUMPTION							
<35 servings/month	284	118	(41.6%)	238	83	(34.9%)	[0.14]
35-74 servings/month	532	241	(45.3%)	510	209	(41.0%)	[0.18]
75+ servings/month	178	74	(41.6%)	247	110	(44.5%)	[0.61]
[p[c] (d.f.)]	[0.49 (2)]			[0.09 (2)]			
HIGH-RISK OCCUPATION[c]							
no high-risk occupation	791	350	(44.3%)	892	363	(40.7%)	[0.16]
high-risk occupation	203	83	(40.9%)	103	39	(37.9%)	[0.70]
[p[c] (d.f.)]	[0.43 (1)]			[0.65 (1)]			
EDUCATION							
<8 years	127	35	(27.6%)	144	51	(35.4%)	[0.21]
8-12 years	648	278	(42.9%)	605	232	(38.4%)	[0.11]
>12 years	219	120	(54.8%)	246	119	(48.4%)	[0.20]
[p[c] (d.f.)]	[<0.001 (2)]			[0.01 (2)]			
COUNTY AT DIAGNOSIS[d]							
Low radon	300	112	(37.3%)	265	89	(33.6%)	[0.71]
Moderately low radon	354	163	(46.1%)	389	178	(45.8%)	[0.99]
Moderate radon	190	84	(44.2%)	195	76	(39.0%)	[0.35]
High radon	150	74	(49.3%)	146	59	(40.4%)	[0.15]
[p[c] (d.f.)]	[0.05 (3)]			[0.02 (3)]			
LIFETIME NONSMOKERS ONLY, BY PASSIVE SMOKING							
No spouse tobacco exp.	43	18	(41.9%)	196	70	(35.7%)	[0.56]
Spouse cigarette exp.	66	38	(57.6%)	250	116	(46.4%)	[0.14]
Spouse pipe/cigar only	7	5	(71.4%)	53	27	(50.9%)	[0.54]
[p[c] (d.f.)]	[0.16 (2)]			[0.03 (2)]			

(contd)

TABLE C1 (contd)

[a] Number of women included in radon study includes those whose index address was tested for radon and those whose index address was an apartment higher than the second floor (see Table 2). Percentage represents number of women included in radon study divided by total number of women in original study.

[b] p value for Chi-square test (1 degree of freedom, with continuity correction) comparing the percentage of all cases vs. all controls included in the radon study.

[c] p value for Chi-square test (with degrees of freedom noted in parentheses) comparing the percentage of cases (or controls) included in the radon study, by subgroups of the various risk factors and subject characteristics.

[d] Lifetime average number of cigarettes smoked per day.

[e] Ever employed in any occupational group shown to have a smoking adjusted risk of 1.5 or greater in the original female lung cancer study. This is an a posteriori definition, used only for the purpose of adjusting in the radon analyses for the possible effect of occupational exposure. See text, Appendix B, for further clarification of this variable.

[f] County at diagnosis for cases, or county at ascertainment for controls. Low radon counties include include Atlantic, Cape May, Essex, Hudson, and Ocean counties. Moderately low radon counties include Bergen, Burlington, Cumberland, Gloucester, Middlesex, and Union counties. Moderate radon counties include Camden, Monmouth, Passaic, and Salem counties. High radon counties include Hunterdon, Mercer, Morris, Somerset, Sussex, and Warren counties. Grouping of counties was determined by the percentage of houses with basement or lower level screening values above 4 pCi/L in the statewide survey conducted for the New Jersey State Department of Environmental Protection (see Appendix G).

TABLE C2

Odds ratios[a] for association of lung cancer with various risk factors and subject characteristics and with inclusion in the radon substudy, for all subjects and by subgroups according to smoking
New Jersey radon-female lung cancer case-control study, 1982-1988

SUBGROUP:	TOTAL	NS[a]	LS[a]	MS[a]	HS[a]
NO. CASES:	994	116	198	414	266
NO. CONTROLS:	995	499	210	195	91
INCLUDED IN RADON STUDY:no	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]
yes	1.3*	1.5	1.1	1.5*	1.2
AGE:<58 years	0.78	0.60	0.65	0.96	0.63
58-71 years	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]
72+ years	1.8**	1.6	1.8*	2.2**	1.1
RESP TYPE: self	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]
spouse, other next of kin	0.87	1.6*	0.92	0.75	0.40**
RACE: white	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]
nonwhite	1.1	0.81	2.0	0.89	1.4
CIGARETTES/DAY+YEARS QUIT					
Lifetime nonsmoker (NS)	1.0[b]	---	---	---	---
< 15 cigarettes/day (LS)					
Ex-smoker, quit 10+ years	1.7*	---	1.0[b]	---	---
Ex-smoker, quit 2-9 years	4.8**	---	2.7**	---	---
Current smoker, quit 0-1 years	6.9**	---	4.0**	---	---
15-24 cigarettes/day (MS)					
Ex-smoker, quit 10+ years	4.1**	---	---	1.0[b]	---
Ex-smoker, quit 2-9 years	7.2**	---	---	1.7	---
Current smoker, quit 0-1 years	13.5**	---	---	3.3**	---
25+ cigarettes/day (HS)					
Ex-smoker, quit 10+ years	2.1	---	---	---	1.0[b]
Ex-smoker, quit 2-9 years	7.4**	---	---	---	4.8*
Current smoker, quit 0-1 years	20.9**	---	---	---	16.4**
VEGETABLE:<35 servings/mo	1.4*	1.4	0.90	1.7*	1.8
35-74 servings/mo	1.3*	2.0*	0.66	1.9**	0.95
75+ servings/mo	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]
HIGH-RISK OCCUPATION: no	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]
yes	2.0	2.6	1.1	2.2	2.9
EDUCATION: <8 years	0.95	0.91	1.3	1.2	0.58
8-12 years	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]
13+ years	0.99	1.2	0.97	0.86	1.2

(contd)

TABLE C2 (contd)
Odds ratios[a] for association of lung cancer with various risk factors and subject characteristics and with inclusion in the radon substudy, for all subjects and by subgroups according to smoking
New Jersey radon-female lung cancer case-control study, 1982-1988

SUBGROUP:	TOTAL	NS[a]	LS[a]	MS[a]	HS[a]
NO. CASES:	994	116	198	414	266
NO. CONTROLS:	995	499	210	195	91
COUNTY: low radon	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]
moderate low radon	0.88	0.57*	0.97	0.92	2.0
moderate radon	0.77	0.43*	0.86	0.82	1.1
high radon	0.97	0.59	1.0	1.1	1.3

* $p < 0.05$ ** $p < 0.01$
[a] Odds ratios from logistic regression analysis for 994 cases and 995 controls in original female lung cancer study. NS=lifetime nonsmoker; LS=light smoker (<15 cigarettes/day); MS=moderate smoker (15-24 cigarettes/day); HS=heavy smoker (25+ cigarettes/day).
[b] Reference group: odds ratio=1.0

TABLE C3

Odds ratios[a] for association of lung cancer with various risk factors and subject characteristics, for all subjects and for subgroups according to smoking, and by inclusion in the radon substudy, New Jersey radon-female lung cancer case-control study, 1982-1988

SUBGROUP:	TOTAL		NONSMOKER		LIGHT SMOKER <15 CIGS/DAY		MOD. SMOKER 15-24 CIGS/DAY		HEAVY SMOKER 25+ CIGS/DAY	
INCLUDED IN RADON STUDY:	YES	NO	YES	NO	YES	NO	YES	NO	YES	NO
NO. CASES:	433	561	61	55	83	115	178	236	111	155
NO. CONTROLS:	402	593	213	286	90	120	67	128	32	59
AGE:<58 years	1.0	0.67	0.61	0.53	0.86	0.51	1.3	0.82	0.81	0.51
58-71 years	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]
72+ years	2.3	1.5	2.1	1.1	1.5	2.2	3.8	1.8	0.99	1.1
RESP TYPE: self	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]
spouse, other next of kin	0.78	0.94	1.4	1.7	0.76	1.1	0.62	0.88	0.39	0.38
RACE: white	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]
nonwhite	0.78	1.3	0.47	1.0	1.0	2.8	0.70	0.94	0.90	1.3
CIGARETTES/DAY+YEARS QUIT										
Lifetime nonsmoker (NS)	1.0[b]	1.0[b]	---	---	---	---	---	---	---	---
<15 cigarettes/day (LS)										
Ex-smoker, quit 10+ years	1.5	2.0	---	---	1.0[b]	1.0[b]	---	---	---	---
Ex-smoker, quit 2-9 years	4.0	5.5	---	---	2.9	2.1	---	---	---	---
Current smoker, quit 0-1 years	5.5	8.0	---	---	3.9	4.0	---	---	---	---
15-24 cigarettes/day (MS)										
Ex-smoker, quit 10+ years	7.7+	2.5	---	---	---	---	1.0[b]	1.0[b]	---	---
Ex-smoker, quit 2-9 years	7.8	6.8	---	---	---	---	1.0	3.0	---	---
Current smoker, quit 0-1 years	12.0	14.8	---	---	---	---	1.5++	6.7	---	---
25+ cigarettes/day (HS)										
Ex-smoker, quit 10+ years	2.9	1.6	---	---	---	---	---	---	1.0[b]	1.0[b]
Ex-smoker, quit 2-9 years	17.7	4.8	---	---	---	---	---	---	6.1	3.8
Current smoker, quit 0-1 years	16.6	25.2	---	---	---	---	---	---	12.3	21.4

+ $p < 0.10$ ++ $p < 0.05$ for difference in OR between those included and not included in radon study (contd)

TABLE C3 (contd)

Odds ratios[a] for association of lung cancer with various risk factors and subject characteristics, for all subjects and for subgroups according to smoking, and by inclusion in the radon substudy, New Jersey radon-female lung cancer case-control study, 1982-1988

SUBGROUP:	TOTAL		NONSMOKER		LIGHT SMOKER <15 CIGS/DAY		MOD. SMOKER 15-24 CIGS/DAY		HEAVY SMOKER 25+ CIGS/DAY	
INCLUDED IN RADON STUDY:	YES	NO	YES	NO	YES	NO	YES	NO	YES	NO
NO. CASES:	433	561	61	55	83	115	178	236	111	155
NO. CONTROLS:	402	593	213	286	90	120	67	128	32	59
VEGETABLE:<35 servings/mo	1.7	1.2	2.4	0.77	0.85	1.0	2.8	1.3	0.45+	3.0
35-74 servings/mo	1.4	1.3	2.1	1.8	0.75	0.67	2.6	1.6	0.21++	1.7
75+ servings/mo	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]
HIGH-RISK OCCUPATION: no	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]
yes	2.4	1.8	3.4	2.2	1.0	1.1	3.7	1.8	1.9	2.6
EDUCATION: <8 years	0.75	1.1	0.76	0.95	1.1	1.4	1.6	1.1	0.10++	1.0
8-12 years	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]
13+ years	1.0	0.91	1.0	1.2	1.3	0.70	0.67	0.95	1.7	0.80
COUNTY: low radon	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]	1.0[b]
moderate low radon	0.77	0.95	0.45	0.74	1.0	0.75	0.74	1.1	1.1	2.3
moderate radon	0.86	0.68	0.38	0.41	1.2	0.84	1.2	0.59	0.78	1.2
high radon	0.95	0.89	0.53	0.59	1.7	0.68	0.82	1.2	0.87	1.4

+ $p < 0.10$ ++ $p < 0.05$ for difference in OR between those included and not included in radon study

[a] Odds ratios from logistic regression analysis for all subjects from original female lung cancer study.
[b] Reference group: odds ratio=1.0

TABLE C4
Total number of HEAVY SMOKERS in original study,
and number and percentage (%) included in radon study[a],
by various risk factors and subject characteristics,
New Jersey radon-female lung cancer case-control study, 1982-1988

	CASES			CONTROLS			
	TOTAL N	RADON N	(%)	TOTAL N	RADON N	(%)	[p[b]]
TOTAL	266	111	(41.7%)	91	32	(35.2%)	[0.33]
AGE AT DIAGNOSIS							
<58 years	81	27	(33.3%)	28	6	(21.4%)	[0.35]
58-71 years	142	66	(46.5%)	49	21	(42.9%)	[0.79]
72+ years	43	18	(41.9%)	14	5	(35.7%)	[0.93]
[p[c] (d.f.)]	[0.16 (2)]			[0.17 (2)]			
RESPONDENT TYPE							
Self	119	53	(44.5%)	28	9	(32.1%)	[0.33]
Spouse	43	17	(39.5%)	21	5	(23.8%)	[0.34]
Other next of kin	104	41	(39.4%)	42	18	(42.9%)	[0.84]
[p[c] (d.f.)]	[0.71 (2)]			[0.30 (2)]			
RACE							
White	250	109	(43.6%)	85	31	(36.5%)	[0.31]
Nonwhite	16	2	(12.5%)	6	1	(16.7%)	[0.99]
[p[c] (d.f.)]	[0.03 (1)]			[0.59 (1)]			
TOTAL NO. YEARS SMOKED							
<35 years	47	16	(34.0%)	36	6	(16.7%)	[0.13]
35+ years	219	95	(43.4%)	55	26	(47.3%)	[0.71]
[p[c] (d.f.)]	[0.31 (1)]			[0.006 (1)]			
NO. YEARS QUIT SMOKING							
Quit 0-1 years	235	95	(40.4%)	60	25	(41.7%)	[0.98]
Quit 2-9 years	24	12	(50.0%)	17	3	(17.7%)	[0.07]
Quit 10+ years	7	4	(57.1%)	14	4	(28.6%)	[0.43]
[p[c] (d.f.)]	[0.47 (2)]			[0.16 (2)]			
AVG. CIGARETTE TAR CONTENT, 1973-1982							
Nonsmoker, 1973-1982	7	4	(57.1%)	13	4	(30.8%)	[0.50]
Tar <21 mg/cigarette	205	89	(43.4%)	70	23	(32.9%)	[0.16]
Tar 21+ mg/cigarette	54	18	(33.3%)	8	5	(62.5%)	[0.23]
[p[c] (d.f.)]	[0.29 (2)]			[0.24 (2)]			

(contd)

TABLE C4 (contd)
Total number of HEAVY SMOKERS in original study,
and number and percentage (%) included in radon study[a],
by various risk factors and subject characteristics,
New Jersey radon-female lung cancer case-control study, 1982-1988

	CASES			CONTROLS			
	TOTAL	RADON		TOTAL	RADON		
	N	N	(%)	N	N	(%)	[p[b]]
TOTAL	994	433	(43.6%)	995	402	(40.4%)	[0.17]
VEGETABLE CONSUMPTION							
<35 servings/month	86	30	(34.9%)	23	8	(34.8%)	[0.99]
35-74 servings/month	135	62	(45.9%)	52	22	(42.3%)	[0.78]
75+ servings/month	45	19	(42.2%)	16	2	(12.5%)	[0.07]
[p[c] (d.f.)]	[0.27 (2)]			[0.09 (2)]			
HIGH-RISK OCCUPATION							
no high-risk occupation	204	89	(43.6%)	79	28	(35.4%)	[0.26]
high-risk occupation	62	22	(35.5%)	12	4	(33.3%)	[0.99]
[p[c] (d.f.)]	[0.32 (1)]			[0.99 (1)]			
EDUCATION							
<8 years	30	3	(10.0%)	14	6	(42.9%)	[0.03]
8-12 years	189	82	(43.4%)	60	22	(36.7%)	[0.44]
>12 years	47	26	(55.3%)	17	4	(23.5%)	[0.05]
[p[c] (d.f.)]	[<0.001 (2)]			[0.49 (2)]			
COUNTY AT DIAGNOSIS							
Low radon	76	32	(42.1%)	31	8	(25.8%)	[0.17]
Moderately low radon	93	39	(41.9%)	22	9	(40.9%)	[0.99]
Moderate radon	59	22	(37.3%)	23	9	(39.1%)	[0.99]
High radon	38	18	(47.4%)	15	6	(40.0%)	[0.86]
[p[c] (d.f.)]	[0.81 (3)]			[0.61 (3)]			

[a] Number of heavy smokers included in radon study includes those whose index address was tested for radon and those whose index address was an apartment higher than the second floor (see Table 2). Percentage represents number of heavy smokers included in radon study divided by total number of heavy smokers in original study.

[b] p value for Chi-square test (1 degree of freedom, with continuity correction) comparing the percentage of all cases vs. all controls included in the radon study.

[c] p value for Chi-square test (with degrees of freedom noted in parentheses) comparing the percentage of cases (or controls) included in the radon study, by subgroups of the various risk factors and subject characteristics.

APPENDIX D

Considerations about Changes in House Construction

One of the basic, underlying assumptions of this study is that the radon concentrations measured in 1986-1988 in the index addresses are adequate estimates of the exposures that were incurred by the subjects when they were living in these houses, particularly for residence in these houses from 1953 to 1978, the presumed latency period. However, it is possible that changes in construction of the house have occurred which have fundamentally altered the radon concentrations in the house. Let us consider two different scenarios:

(1) The house construction has been changed in such a way that the radon concentrations have been significantly reduced. There were not any houses in this study which were known to have undergone changes specifically for the purpose of radon remediation. However, this does not mean that there were no houses with other structural changes which might have reduced the radon levels in the house. Nevertheless, at this point in time, there is no way to estimate what the radon concentration might have been before, given certain structural changes.

(2) The house construction has been changed in such a way that the radon concentrations have been significantly increased. For example, a basement has been dug out where none existed previously, or significant cracks have developed in the foundation. Again, at this point in time, there is no way to estimate what the radon concentration might have been before, given this structural change.

The final problem in considering changes in construction is that the information which has been collected for the study houses is not complete. This is particularly true for houses in which the current resident was not the study subject or a relative. The new resident was often not aware of any structural changes which might have occurred.

Given all of the above, it is still important to determine the magnitude of the potential problem, particularly for the 36 houses with living area measurements at 2+ pCi/L which represent the driving force behind these results. Therefore, Table H1 presents key characteristics with respect to construction changes for these 36 houses, in addition to the residence period reported for the subject and the residence period of the current occupant. These changes have not yet been taken into account in the analyses. One focus of the additional analyses planned for the final study report will be whether the cumulative exposure estimate can be modified, based on this limited information on construction changes.

TABLE D1

Changes in house construction reported for 36 houses in radon study with living area alpha track measurements of 2+ pCi/L, New Jersey radon-female lung cancer case-control study, 1982-1988

NO.	SUBJ RES YRS[a]	CURR RES YRS[b]	REPORTED CHANGES IN CONSTRUCTION
1	1961-83	1962-present	refinished second floor (2 small bedrooms) in 1963; added attic insulation in 1963
2	1938-70	1973-present	added 2 story addition in 1976; heating system was expanded in 1976; took out old door, installed sliding glass door in dining room in 1982
3	1947-72	1975-present	remodeled house in 1981; no changes in basement; upgraded heating system in 1984; central air installed 1984; extra wall insulation 1976; attic insulation 1985; attic fan 1978
4	1952-64	1982-present	attic insulation 1983
5	1953-82	1945-present	porch addition 1953; new burner in oil furnace 1975
6	1960-83	1985-present	extra wall insulation, attic insulation, storm doors, storm windows 1986
7	1944-83	1948-present	porch added 1985; basement treated with sealant and paint for water leakage 1985; converted to gas heat 1954; added central air 1981
8	1942-70	1970-present	old well in basement filled in with dirt prior to present owner
9	1960-83	1985-present	central air added 1986; storm door 1985; attic fan 1985
10	1958-83	1986-present	no changes reported
11	1956-84	1986-present	total remodeling of upstairs 1986; added garage 1986; no changes in basement; extra wall insulation, attic insulation, 1986
12	1963-84	1984-present	attic insulation 1985; no other changes
13	1961-83	1960-present	added enclosed back porch 1964; added siding, storm doors, storm windows 1984 attic fan 1967 (contd)

TABLE D1 (contd)
Changes in house construction reported for 36 houses
in radon study with living area alpha track measurements of 2+ pCi/L,
New Jersey radon-female lung cancer case-control study, 1982-1988

NO.	SUBJ RES YRS[a]	CURR RES YRS[b]	REPORTED CHANGES IN CONSTRUCTION
14	1953-83	1953-present	replaced oil furnace 1980; extra wall insulation, attic insulation, storm doors, attic fan 1953; storm windows 1982
15	1963-75	commercial	building addition 1985; no changes in basement; no other reported changes
16	1959-83	1985-present	no reported changes
17	1919-79	1979-present	storm door 1984
18	1952-79	1948-present	replaced furnace 1985; attic insulation 1985; replaced storm door and windows 1962
19	1958-75	1977-present	converted from oil to gas furnace, 1982
20	1955-71	1986-present	no reported changes
21	1959-72	1974-present	installed central air 1980; installed new thermopane windows and doors 1984 attic insulation 1982; attic fan 1980
22	1962-83	1985-present	siding, storm doors, storm windows 1963
23	1926-83	1983-present	added French drain 1984; extra wall insulation 1985; storm doors, storm windows 1983; siding 1986
24	1940-82	1941-present	coal furnace converted to gas 1961; storm doors, storm windows 1952; siding 1942
25	1948-83	1985-present	addition 1985; remodeled old section of house 1985; no changes in basement; crawl space added with addition, 1985; changed heating system from oil (forced air) to gas (hot water), 1985 extra wall insulation, attic insulation 1985
26	1950-83	1951-present	no reported changes
27 (contd)	1950-84	1952-present	siding 1984; no other reported changes

TABLE D1 (contd)
Changes in house construction reported for 36 houses
in radon study with living area alpha track measurements of 2+ pCi/L,
New Jersey radon-female lung cancer case-control study, 1982-1988

NO.	SUBJ RES YRS[a]	CURR RES YRS[b]	REPORTED CHANGES IN CONSTRUCTION
28	1963-73	1985-present	storm doors, storm windows 1985; siding 1986
29	1937-82	1985-present	storm doors, storm windows 1947
30	1961-83	1961-present	basement dug out and added 1961; gas furnace added with basement 1961; window air conditioners 1964; siding, storm windows 1970; front storm door, 1986
31	1956-76	1980-present	addition in back (on slab) and new interior walls added, 1984; no changes to original crawl space; added fireplace, 1983; extra wall insulation, attic insulation, wood siding, storm doors, storm windows, 1983
32	1957-78	1978-present	extra wall insulation, siding, new storm doors, storm windows 1985
33	1953-82	1953-present	basement finished into an apartment, windows added, 1966; attic insulation, 1987; attic fan, 1986
34	1953-82	1986-present	tore porch down, built one room addition, 1986; crawl space built for addition, 1986; no changes in basement (new crawl space "open" to basement but window closed all the time); new furnace and new central air, 1986; new siding, storm doors, storm windows, 1986
35	1937-82	1985-present	attic being converted into living space, 1987; installed wood stove in attic, 1987; attic fan, 1982
36	1952-62	1972-present	storm doors, storm windows, 1980.

[a] Subject residence years as reported in the residential history

[b] Residence years for current occupant

APPENDIX E

Radon Measurement Methods

Charcoal canister measurements

A cylindrical metal canister about 4 inches in diameter and 3/4 inches in height is filled with 100 grams of activated carbon. Before sampling, the detector is purged of radon gas and water by baking for several hours, and then sealed with a metal cover and tape. The canister is weighed before deployment. At the sampling site, the tape and metal cover are removed, and the open side of the canister is exposed to the air for four days. During this period, radon in air passively diffuses into the canister and is adsorbed onto the carbon. During the decay of the radon gas, its decay product particles remain adsorbed. After the sampling period, the detector is closed, taped again, and then mailed to the NJDEP laboratory for counting.

Assay for radon gas concentration in the sampled air is accomplished in the laboratory by counting the gamma-ray activity of the specific radon decay products, lead-214 and bismuth-214, with a sodium iodide detector. Absorption of water by the charcoal (and loss of adsorption capability for radon) is measured by weighing the canister again and correcting for the water content.

The charcoal canister is a passive, integrating detector which depends only upon air diffusion. The method is not sensitive to the precise length of time that the detector was exposed. Instead, it yields an average radon concentration for approximately the last two days of exposure. The minimum detectable concentration (MDC) and precision of the detector are also very sensitive to the elapse of time after the canister is closed and to the amount of radon adsorbed. After more than two half-lives of radon-222 (two times 3.8 days), the MDC increases markedly. It often took up to a week from the midpoint of sampling for the mailed canisters to

reach the lab. Typical MDCs for this study were 0.6-0.9 pCi/L. Every effort was made to repeat canister measurements if the MDCs were greater than 1.0 pCi/L.

The charcoal canister measurement component of this study included quality control checks routinely utilized by the NJDEP laboratory. This includes processing of blanks, known calibration standards, and unknowns provided by the US Environmental Protection Agency. Of the canisters reported for this study, ten percent were selected at random and recounted on another detector. Any result discrepant by more than $\pm$ 1.96 standard deviations caused the laboratory personnel to recaliabrate the counting instrumentation until the specified level of agreement was achieved.

Alpha track detector measurements

The alpha track detector consists of a small cylindrical plastic cup, about one inch in diameter. The method is based on passive diffusion. The open top of the cup is covered with a membrane which is permeable to radon gas but impermeable to radon decay particles. Consequently, only the radon gas entering the open side of the cup (and the particles resulting from the decay of the gas inside the cup) are assayed with this method. The radon decay products in the room air are not assayed. Inside the cup is a plastic (acrylic) film onto which alpha particles resulting from the decay of the radon gas produce submicroscopic "tracks" when they impact. Etching this film with a caustic solution accentuates the tracks, enabling them to be counted visually. The number of tracks within a specific area of the film are proportional to the radon concentration in air multiplied by the precise length of exposure. Sensitivity depends both upon the area of the alpha-sensitive film which is counted and the time that the detector has been exposed. After the exposure period is completed, the detector is enclosed in an air-tight aluminum pouch (or, as in this study, in several layers of aluminum foil) and mailed

for analysis. The number of tracks are counted, and the reported exposure is calculated by dividing the calculated pCi/L-days by the reported exposure period. Therefore, errors in the designation of dates can result in erroneous reported exposures. The precision of the detectors varies, with a maximum of 25%, and a usual value of 10%. The MDC is 30 pCi/L-years; therefore, over a year's time, the MDC approaches the background outdoor radon concentration of 0.1-0.2 pCi/L. The detectors can be stored for long periods after exposure and, if desirable, recounted at a later date without loss of precision.

The alpha track detector monitoring in this study had two specific quality control components. For most houses in this study, two alpha track detectors had been installed, generally on different floors. Until the end of March 1987, there was a continuous problem with the supply of alpha track detectors which could be ordered. Therefore, no additional quality control detectors could be installed. After the end of March 1987, the supply problem was solved. At that time, in most houses, a third alpha track detector was paired with one of the first two as a quality control check.

Duplicate measurements were collected in this manner for 89 houses (12.4% of the 719 houses for which one or more alpha track measurements were obtained). The difference (mean $\pm$ standard deviation) between the detector results for the 89 pairs was 0.27 ± 0.26 pCi/L, with differences ranging from 0.0 to 1.3 (Table E1). The difference was 0.2 pCi/L or smaller for 57 (64%) of the 89 pairs. Only 3 (3%) of the pairs had differences of 1.0 pCi/L or greater. The overall precision of the measurements, as determined by the coefficient of variation, was 25%.

Table E1 also shows the difference within pairs by the average radon concentration which was measured and by which floor of the house was measured. The 52 detector pairs with an average less than 1 pCi/L had a smaller mean difference within pairs, but a greater coefficient of variation (36%), i.e., lower

precision. The 10 detector pairs with an average 2-3.9 pCi/L had a coefficient of variation of 12%. The 49 detector pairs in the basement had a larger mean difference within pairs, and a smaller coefficient of variation (21%). The 17 first floor pairs, with a mean exposure of 0.70 pCi/L, had a coefficient of variation of 24%. The 23 second floor pairs, with a mean exposure of 0.56 pCi/L, had a coefficient of variation of 38%.

As an additional quality control check, the NJDEP made arrangements with Andreas George of the Environmental Measurements Laboratory (EML), US Department of Energy, to expose 40 alpha track detectors to known concentrations of radon. These "spiked" detectors and a sample of "blank" (unexposed) detectors were then labeled, packaged, and shipped to Terradex in such a way as to resemble the other detectors submitted with each batch.

Table E2 shows the results for these detectors, according to the level of exposure (in pCi/L-days; and in pCi/L, if the detector had been exposed for 365 days, as were the majority of detectors in this study). The maximum level reported for any of the blank detectors was 129 pCi/L-days, equivalent to 0.35 pCi/L for a one year exposure period.

The absolute value of the difference between the reported pCi/L-days and the "true" pCi/L-days was calculated; the accuracy of the alpha track detector method (the average difference divided by the "true" pCi/L) ranged from 84% for the lowest known exposure level (0.5 pCi/L) to 9-28% for the highest known exposure levels (1.2 to 2.1 pCi/L). One of the detectors which had been exposed to 168 pCi/L-days (0.5 pCi/L) was reported as 636 pCi/L (1.7 pCi/L), even after a repeat reading. This was by far the largest discrepancy observed. The calculated accuracy of this group of detectors without this one apparent outlier was 36%. These results still suggest that there is much more uncertainty in readings below 1 pCi/L. However, the extent of error is not likely to result in a reading greater than 2 pCi/L.

The precision of the alpha track detector reading was also assessed with these spiked detectors. The standard deviation for each exposure level group, divided by the mean reported exposure, yielded the coefficient of variation. This statistic was 131% for the blank detectors, 65% for the lowest exposed level (0.5 pCi/L) [29% excluding the outlier], and then ranged from 6% to 32% for all other exposure levels.

These quality control results suggest that, given the low levels of exposure prevalent in this study, the precision of the measurements may not be sufficient to analyze all of the data on a continuous scale. Rather, a categorical analysis, based on low, medium, and high exposure (e.g., <1, 1-1.9, 2-3.9, 4+) may be preferable. Therefore, the continuous variable analyses for exposure should be interpreted cautiously. On the other hand, cumulative exposure, which includes the additional component of residence duration, may be less sensitive to the imprecision of the measurement, but also less sensitive to variation in the true values. This is the justification for presenting both the results of continuous and categorical analyses in this report.

Length of alpha track detector installation.

Table E3 shows the distribution of alpha track detector installation times, by case-control status, for the 664 living area alpha track detectors and for the 55 basement alpha track detectors which were used to estimate living area radon concentrations. Only 27 of the 664 living area alpha track detectors (4.1%) and 2 of the 55 basement alpha track detectors (3.6%) were installed for less than 11 months (48 weeks). This included one case alpha track detector with a measurement of 4.9 pCi/L which was installed for 15 weeks (March 10 - June 26), and one case alpha track detector with a measurement of 2.4 pCi/L which was installed for 22

weeks (August 31 - January 29). None of the control detectors with measurements above 2.0 pCi/L were installed for less than 11 months.

Relationship between canister and alpha track measurements

One of the strengths of this study is the fact that both canister and alpha track measurements of radon were obtained. If only alpha track measurements had been conducted, the low levels of reported exposures might have seemed unlikely and the entire study results might have been compromised. However, because the alpha track detector results were consistent with the canister measurements, both on an individual and on a group basis, the study conclusions are strengthened.

Table E4 shows the geometric means for basement canisters, living area canisters, basement alpha track detectors, and living area alpha track detectors, and the correlations between pairs of measurements. The top part, Table E4a, shows these statistics for the 516 houses with all four types of measurements, and all paired combinations. The bottom part, Table E4b, shows these statistics for all 796 houses with any measurements, with the number for each type of paired combination shown along with the statistics. The correlation between measurements was good, given the different time periods for the canister and alpha track measurements, and given the loss of precision attributable to the greater number of low measurements. Table E4b also shows that the basement canister results were slightly higher in those houses for which living area alpha track measurements were obtained; i.e., detector retrieval was much poorer for those houses with very low canister results.

Ratio of lower level (basement) canister to first floor alpha track measurements.

Initially it seemed inconsistent that 13.3 percent of the lower floor (usually basement) canister measurements in this study had results over 4 pCi/L, compared

to only 1.0 percent of the living area alpha track measurements. The relationship of the lower floor canister measurements to the living area alpha track measurements, at increasing radon concentrations (as measured by the lower floor canister), is shown in Table E5. The results are shown for all lower floor canisters, and for all basement canisters, for which there were living area alpha track measurements. The results of a similar analysis also taking into account the type of heat circulation (forced air vs. hot water/electric) are also shown in Table E5.

The relationship of the living area alpha track measurement to the lower floor or basement canister measurement was markedly non-linear. Those houses with a higher concentration of radon measured for four days during the heating season on the lower floor had a proportionally lower concentration of radon as measured by a year-round alpha track detector in the living area. This difference was less pronounced for forced air houses than for hot water/electric houses; however, the non-linearity was present in both groups. This non-linearity may be due, in part, to the relatively greater imprecision at low radon concentrations for both the canister and the alpha track detectors.

The observation that the ratio plateaus at levels greater than previously speculated may be related to the manner in which previous estimates of the ratio of screening measurements to annual averages were calculated. Many of these former ratios were actually based on the average of two screening measurements, one in the heating season, and one in the summer (see, for example, Nero et al., 1986). This may result in an artifactually high estimate of the "annual average." Also, reported ratios were often the ratio of the average basement canister to the average "annual average", rather than the average of the ratio calculated for each individual house.

Data from the NJDEP study (see Appendix G), which included a sample of about 200 heating season alpha track detectors installed for 3-5 months, also

suggest that the ratio of heating season basement canister results to living area alpha track results increase with increasing basement radon levels (NJDEP, 1989). These results, together with the results in Table E5, reiterate the necessity for screening measurements to be followed up by some type of confirmatory measurement before remediation decisions are made. If the screening measurement is between 4 and 20 pCi/L, it probably should be followed by a measurement of the annual average radon concentration in areas of the house where residents spend considerable amounts of time, although short-term measurements in the living area may also be useful. These recommendations are actually consistent with existing USEPA and NJDEP guidelines, but are not necessarily understood by the public.

If the results of the analyses comparing screening measurements to year-round average living area radon concentrations are confirmed by other studies, they also suggest that national surveys based only on screening results may overestimate the radon exposures of the general population.

TABLE E1
Analysis of results of paired alpha track detectors,
by radon concentration, and by house level,
New Jersey radon-female lung cancer case-control study, 1982-1988

	N	Mean[a]	Mean Diff[b]	Std Dev of Diff	CV[c]	Range of Difference
TOTAL PAIRED SAMPLES	89	1.07	0.27	0.26	0.25	0.0 - 1.3
RADON CONCENTRATION						
<1.0 pCi/L	52	0.56	0.19	0.20	0.36	0.0 - 0.9
1-1.9 pCi/L	26	1.44	0.36	0.32	0.22	0.0 - 1.3
2-3.9 pCi/L	10	2.47	0.41	0.30	0.12	0.0 - 1.0
4+ pCi/L	1	4.10	0.20	----	----	0.2 - 0.2
HOUSE LEVEL						
Basement	49	1.44	0.33	0.30	0.21	0.0 - 1.3
First floor	17	0.70	0.19	0.17	0.24	0.0 - 0.6
Second floor	23	0.56	0.18	0.21	0.38	0.0 - 0.8

[a] Average radon concentration for pairs of alpha track detectors

[b] Average of difference between members of pairs

[c] Coefficient of variation, standard deviation of the difference divided by the mean of the measured radon concentrations

TABLE E2
Difference between radon concentration determinations (pCi/L-days) and known concentration, for laboratory-exposed and blank alpha track detectors, by known radon concentration, New Jersey radon-female lung cancer case-control study, 1982-1988

Known radon conc pCi/L-days (pCi/L)[a]	N	Mean	Std Dev	CV[b]	Range of Difference	% Diff[c]
0 (0.0)[d]	17	28	37	1.31	0 - 129	---
168 (0.5)	5	300	196	0.65	-23 - 468[e]	84%
250 (0.7)	5	324	92	0.28	-9 - 228	31%
356 (1.0)	5	415	91	0.22	-51 - 186	23%
434 (1.2)	5	472	53	0.11	-25 - 119	11%
511 (1.4)	4	537	89	0.17	-76 - 140	12%
667 (1.8)	5	611	39	0.06	-110 - -16	9%
730 (2.0)	4	832	263	0.32	-38 - 337	28%
781 (2.1)	4	821	139	0.17	-90 - 195	15%

[a] pCi/L-days=days of exposure in chamber X radon exposure concentration in chamber. Number in parentheses (pCi/L) is hypothetical exposure concentration if monitor with the same reported number of tracks had been exposed for 365 days.

[b] coefficient of variation, standard deviation divided by the mean.

[c] % difference (accuracy) = mean of absolute differences for each group, divided by known radon concentration (pCi/L-days)

[d] Three of the 17 blanks in this group were prepared at the Environmental Measurements Laboratory. The average reported measurement was 66.1 pCi/L-years, compared to an expected value of 0.0. The coefficient of variation for these three was 0.26. The remaining 14 blanks detectors were opened, labeled, and then repackaged for shipment at the New Jersey State Department of Health. The average reported measurement was 20.2 pCi/L-years, compared to an expected value of 0.0. The coefficient of variation for these 14 was 1.76.

[e] This group contained one extreme outlier (reported value of 640.5 pCi/L-years. The accuracy and precision, respectively, of the other 4 measurements in this group was 29% and 0.36.

TABLE E3
Duration of exposure of alpha track detectors in index residences for living area alpha tracks (A), and basement installations (B), by case-control status,
New Jersey radon-female lung cancer case-control study, 1982-1988

A. SUCCESSFUL RETRIEVAL OF LIVING AREA DETECTORS

Duration	Cases N	Cases (%)	Controls N	Controls (%)	Total N	Total (%)
up to 6 months	3	(0.9)	4	(1.3)	7	(1.0)
7, 8, or 9 months	7	(2.0)	9	(2.8)	16	(2.4)
10 or 11 months	4	(1.2)	3	(0.9)	7	(1.0)
49 - 51 weeks	84	(24.3)	57	(27.4)	171	(25.8)
ONE YEAR	204	(59.0)	165	(51.9)	369	(55.6)
53 - 55 weeks	30	(8.7)	34	(10.7)	64	(9.6)
56-71 weeks	14	(4.0)	16	(5.0)	30	(4.5)
TOTAL	346		318		664	

B. RETRIEVAL OF BASEMENT DETECTORS ONLY[a]

Duration	Cases N	Cases (%)	Controls N	Controls (%)	Total N	Total (%)
up to 11 months	2	(7.4)	1	(3.6)	3	(5.5)
50 - 51 weeks	5	(18.5)	9	(32.1)	14	(25.5)
ONE YEAR	9	(33.3)	8	(28.6)	17	(30.9)
53 - 55 weeks	5	(18.5)	7	(25.0)	12	(21.8)
56-62 weeks	6	(22.2)	3	(10.7)	9	(16.4)
TOTAL	27		28		55	

[a] Living area measurement estimated from basement measurement for these 55 houses, see Appendix J.

TABLE E4
Geometric means and correlations[a] of radon concentration measurements by availability of measurements, New Jersey radon-female lung cancer case-control study, 1982-1988

A. All paired combinations of measurements available (n=516)

	Basement canister	Basement alpha track	Living area canister	Living area alpha track	GM[b] (pCi/L)
Basement canister	----	0.71	0.57	0.55	1.65
Basement alpha track	----	----	0.48	0.54	1.46
Living area canister	----	----	----	0.59	0.85
Living area alpha track	----	----	----	----	0.59

B. Including any available pair of measurements (total N=796)

		Basement canister	Basement alpha track	Living area canister	Living area alpha track
Basement canister	r	----	0.70	0.57	0.55
	N	678	569	670	560
	GM[b]	1.47	1.57	1.48	1.61
Basement alpha track	r	0.70	----	0.46	0.57
	N	569	581	567	526
	GM	1.41	1.40	1.41	1.45
Living area canister	r	0.57	0.46	----	0.55
	N	670	567	780	655
	GM	0.81	0.84	0.82	0.85
Living area alpha track	r	0.55	0.57	0.55	----
	N	560	526	655	664
	GM	0.58	0.59	0.58	0.58

[a] Correlation (r) of natural logarithms of radon concentrations
[b] Geometric mean

TABLE E5
Ratio of lower floor canister (CANL) or basement canister (CAN0) to living area alpha track (TRKU), by canister radon concentration and by type of heat distribution
New Jersey radon-female lung cancer case-control study, 1982-1988

RADON CONCENTRATION	N	Ratio (CANL/TRKU)[a]	N	Ratio (CAN0/TRKU)[a]
TOTAL	**650**		**556**	
MDC[b]	274	1.5 ± 1.8	204	1.6 ± 1.8
<1.0 pCi/L	16	1.3 ± 1.7	15	1.3 ± 1.8
1-1.9 pCi/L	87	2.7 ± 1.7	82	2.8 ± 1.7
2-3.9 pCi/L	181	4.0 ± 2.0	169	4.2 ± 1.9
4-7.9 pCi/L	75	5.1 ± 2.0	69	5.5 ± 1.8
8+ pCi/L	17	5.1 ± 2.4	17	5.1 ± 2.4
FORCED AIR HEAT	**242**		**195**	
MDC[b]	107	1.3 ± 1.7	72	1.3 ± 1.7
<1.0 pCi/L	5	0.86 ± 1.5	4	0.76± 1.8
1-1.9 pCi/L	32	2.4 ± 1.6	28	2.4 ± 1.6
2-3.9 pCi/L	66	3.1 ± 1.7	60	3.3 ± 1.5
4-7.9 pCi/L	27	4.0 ± 1.5	26	4.1 ± 1.5
8+ pCi/L	5	4.3 ± 1.9	5	4.3 ± 1.9
HOT WATER/ELEC HEAT	**408**		**361**	
MDC[b]	167	1.7 ± 1.9	132	1.7 ± 1.8
<1.0 pCi/L	11	1.5 ± 1.7	11	1.5 ± 1.7
1-1.9 pCi/L	55	2.9 ± 1.8	54	3.0 ± 1.8
2-3.9 pCi/L	115	4.7 ± 2.0	109	4.8 ± 2.0
4-7.9 pCi/L	48	5.9 ± 2.1	43	6.6 ± 1.9
8+ pCi/L	12	5.4 ± 2.6	12	5.4 ± 2.6

(continued)

[a] This table includes all houses with actual living area alpha track measurements and excludes any houses for which living area alpha track results were estimated. This also excludes houses for which only alpha track measurements and no canister measurements were conducted. Ratio statistic shown is: geometric mean of ratios $\pm$ geometric standard deviation.

[b] MDC=minimum detectable concentration. For these canisters, the value of CANL or CAN0 used to calculate the ratio was assumed to equal the MDC.

APPENDIX F

Validation of Residential Histories

Tax office records were searched in order to validate the information provided by the respondent with respect to occupancy of the index residence. There are several limitations to this approach. Tax office records list the owner(s) of record, and there is no guarantee that the owner of a building and the occupants were identical. There is also no reason to expect perfect agreement between the dates of ownership and the dates of residence. Nevertheless, it is important that there be some degree of consistency between the tax office records and the reported residential histories. Also, any major inconsistencies between the two sources of information were probed and many were resolved.

Table F1 shows a tabulation of validation data for the 796 subjects (411 cases, 385 controls) whose index residence was tested for radon in this study. No validation was attempted for subjects whose residence was an apartment higher than the second floor. No validation was possible for 98 subjects (12.3%), because there was no record of ownership by the subject or the subject's family. Validation was not complete for 84 subjects (10.6%), but tax office records confirmed ownership by the subject or her family and were consistent with the residence dates reported for the subject. Tax office records (date of purchase) and reported residential histories (year of first residence) were within $\pm$ 1 year for 457 subjects (57.4%), within $\pm$ 3 years for 552 subjects (69.3%), and within $\pm$ 5 years for 579 subjects (72.7%).

Only 35 subjects (4.4%) had differences which were greater than $\pm$ 5 years. Attempts were made to recontact these subjects or their respondents to probe for further information regarding these discrepancies. Nineteen of the subjects or respondents indicated that the tax office dates were the correct period of residence for the subject; recorded residential history dates were changed accordingly. For eight of these subjects (5 cases, 3 controls), the corrected dates were such that the

subject no longer met the original residence criterion for the study (assuming a 10 year period between relevant radon exposure and diagnosis of lung cancer). Because the duration of residence, minus only a 5 year period, ranged from 8-14 years, the decision was made to leave these subjects in the study. However, they were excluded from the analyses by radon exposure shown in Appendix K, which also excluded subjects with estimated year-round living area radon concentrations.

TABLE F1
Distribution of New Jersey women included in radon study
by case-control status, and by difference between tax office dates and
reported residential history dates,
New Jersey radon-female lung cancer case-control study, 1982-1988

	No. of cases (%)	No. of controls (%)
No validation possible [a]	53 (12.9%)	45 (11.7%)
Validation not completely possible, but dates plausible [b]	47 (11.4%)	37 (9.6%)
Difference in dates [c]		
>-5 years	7 (1.7%)	5 (1.3%)
-4, -5 years	10 (2.4%)	9 (2.3%)
-3, -2 years	32 (7.8%)	25 (6.5%)
within ± 1 year	219 (53.3%)	238 (61.8%)
+2, +3 years	22 (5.4%)	16 (4.2%)
+4, +5 years	7 (1.7%)	1 (0.3%)
>+5 years	2 (0.5%)	2 (0.5%)
Discrepancy in dates >± 5 years; tax office dates confirmed by respondent; residential history dates changed [d]	12 (2.9%)	7 (1.8%)
TOTAL[e]	411	385

[a] No record of ownership by the subject or by the subject's family.
[b] Tax office dates indicated that subject's family owned the residence for a longer period of time than that reported as the residence period for the subject. Also includes a few houses for which available tax office records only covered a portion of the reported residential history years.
[c] Year of purchase by subject or family (minus) reported year of first residence by subject. Negative difference in dates indicates that tax office date is later than residential history date. Positive difference in dates indicates that tax office date is earlier than residential history date.
[d] Attempts were made to recontact respondents if there was a major inconsistency between the tax office dates and the residential history dates. For these 19 subjects, the respondents indicated that the tax office dates were the correct period of residence for the subject.
[e] No attempt made to validate residential histories for 39 subjects (22 cases, 17 controls) whose index residence was an apartment above the second floor.

APPENDIX G

Comparison with Statewide Radon Survey

The New Jersey Department of Environmental Protection (NJDEP) has conducted a geographically stratified sample of homes throughout New Jersey, weighted towards areas suspected of high radon potential (NJDEP, 1989). Measurements of radon concentrations were made using charcoal canisters in the basement or the lowest floor of 5,727 homes. Table G1 shows the distribution of these homes, by county, and the percentage with canister results of 4 pCi/L or higher, and 20 pCi/L or higher. The table also shows the results of comparable measurements from 788 homes in this case-control study (8 of the 796 homes only had alpha-track detector measurements).

Altogether, 1862 (32.5%) of the houses tested in the NJDEP study had lower floor canister measurements of 4 pCi/L or higher, compared to 105 (13.3%) of the houses tested in the case-control study. Furthermore, 263 (4.6%) of the houses tested in the NJDEP study had lower floor canister measurements of 20 pCi/L or higher, compared to 1 (0.1%) of the houses tested in the case-control study. These differences must be considered in the context of differences in the sampling procedures between the two studies.

The NJDEP study was a geographically stratified sample, while the case-control study was originally a population based sample. When the county-specific percentages for the two surveys were weighted by the county populations (1986 population estimates, New Jersey State Department of Labor), the percentages above 4 pCi/L were 14.0% for the NJDEP study and 13.7% for the case-control study. The population weighted average for the NJDEP study was further reduced, to 13.4%, when municipality specific percentages above 4 pCi/L and municipality specific populations were used in the calculation for Warren, Hunterdon, Sussex, Morris, and Somerset counties (the five counties with the greatest number of homes tested in

difference in the percentage of houses with canister results above 4 pCi/L in the two studies. For example, counties with the greatest percentage of high results in the NJDEP study (Warren, Sussex, Hunterdon, Morris, Somerset, Mercer) also tend to have high results in the case-control study. Counties with the lowest percentage of high results in the NJDEP study (Atlantic, Hudson, Essex, Ocean, Union, Cape May) also tend to have low results in the case-control study.

However, even adjusting by county population did not completely eliminate the differences between the two studies in the percentage of houses with canister results above 20 pCi/L. When the county-specific percentages for the two surveys were weighted by the county poopulations, the percentages at 20+ pCi/L were 1.6% for the NJDEP study, and 0.1% for the case-control study. After adjusting by county, there was a highly significant difference in the percentage with canister results of 4-19 pCi/L and 20+ pCi/L between the two studies.

There are several possible explanations for these observed differences. Again, the differences in the sampling procedures for the two studies must be taken into account. The geographic sampling for the NJDEP study resulted in the sampled houses being fairly uniformly distributed throughout rural areas, with relatively little sampling in the urban areas which represent much of the population. Preliminary analysis of the NJDEP data showed that there were distinct urban-rural gradients in radon concentrations (NJDEP, 1989). These differences persisted, even within subgroups defined by the six geologic provinces (ranked by decreasing radon concentrations: Highlands, Valley and Ridge, Southern Piedmont, Inner Coastal Plain, Northern Piedmont, Outer Coastal Plain). For example, within the Highlands (including parts of Somerset, Warren, Hunterdon, Sussex, Morris, Passaic and Bergen counties), the 1121 rural detached houses tested had an arithmetic mean radon level ($\pm$ standard deviation) of 9.2$\pm$17.8 pCi/L; the 283 suburban detached houses tested had a mean of 6.6$\pm$8.8 pCi/L; the 12 urban detached houses tested had a mean of

had a mean of 6.6± 8.8 pCi/L; the 12 urban detached houses tested had a mean of 5.3± 4.4. Within the Southern Piedmont (including parts of Hunterdon, Mercer, Morris, Somerset, Middlesex, and Union counties), the 1052 rural detached houses tested had a mean of 5.6± 10.3 pCi/L; the 459 suburban detached houses tested had a mean of 3.9± 7.0 pCi/L; the 30 urban detached houses tested had a mean of 2.3± 2.2 pCi/L.

Preliminary data from the NJDEP study also showed differences in the level of radon measured in houses according to the age of the house. The 1077 houses which were up to 10 years old had an arithmetic mean radon level of 6.8± 12.7 pCi/L. The 2851 houses which were 11-40 years old had a mean radon level of 5.1± 10.9 pCi/L. The 955 houses which were 41-90 years old had a mean radon level which was 3.8± 7.7 pCi/L. The 722 houses which were 91+ years old had a mean radon level of 5.7± 8.6 pCi/L. These differences according to house age persisted within geologic provinces for rural houses, and to a certain extent for suburban houses.

In the case-control study, the residence criterion necessitated that every house had to be at least 24 years old at the time of measurement in 1986-1987. Information on house age was obtained for 771 of the residences included in the case-control study. Of these 288 (37.4%) were from 22-40 years old, 421 (54.6%) were from 41-90 years old, and 62 (8.0%) were 91+ years old. Lower level canister results were available for most of these houses. The geometric mean (± geometric standard deviation) was 1.43 ± 2.48 for the 285 houses from 22-40 years old, 1.34 ± 2.44 for the 420 houses from 41-90 years old, and 1.30 ± 2.72 for the 60 houses which were 91+ years old.

Conclusions There was relatively good agreement between the NJDEP survey and the case-control study, once the NJDEP results were population-weighted. However, the case-control study had significantly fewer houses with lower floor

attributable to differences in sampling between the two studies (the population based case-control study included more urban residents) and differences in the ages of the houses tested (the case-control study houses were all at least 22 years old).

TABLE G1
Comparison of lowest floor charcoal canister results from statewide NJDEP survey (1986-1987) and from this case-control study (1986-1988), by county in New Jersey

	NJDEP Study			Case-Control Study		
County	Houses tested	% houses 4+ pCi/L	% houses 20+ pCi/L	Houses tested	% houses 4+ pCi/L	% houses 20+ pCi/L
Atlantic	25	0.0%	0.0%	18	5.6%	0.0%
Bergen	250	6.4%	0.0%	103	8.7%	0.0%
Burlington	268	9.7%	0.4%	29	17.2%	0.0%
Camden	73	17.8%	0.0%	52	3.9%	0.0%
Cape May	17	5.9%	0.0%	6	0.0%	0.0%
Cumberland	34	11.8%	0.0%	19	5.3%	0.0%
Essex	115	4.3%	0.0%	94	9.6%	0.0%
Gloucester	30	10.0%	3.3%	29	10.3%	3.4%
Hudson	28	3.6%	3.6%	49	4.1%	0.0%
Hunterdon	835	45.7%	6.7%	13	30.8%	0.0%
Mercer	358	26.3%	3.6%	37	24.3%	0.0%
Middlesex	365	11.8%	2.7%	60	21.7%	0.0%
Monmouth	355	16.9%	2.2%	53	15.1%	0.0%
Morris	718	36.4%	6.1%	40	30.0%	0.0%
Ocean	45	4.4%	0.0%	12	0.0%	0.0%
Passaic	207	21.7%	1.9%	44	15.9%	0.0%
Salem	86	15.1%	1.4%	7	14.3%	0.0%
Somerset	542	30.8%	4.6%	25	36.0%	0.0%
Sussex	622	49.2%	4.8%	3	33.3%	0.0%
Union	138	5.8%	0.0%	90	6.7%	0.0%
Warren	616	66.9%	12.7%	5	60.0%	0.0%
TOTAL	5,727	32.5%	4.6%	788	13.3%	0.1%
County population weighted average		**14.0%[a]**	**1.6%[b]**		**13.7%**	**0.1%**

[a] Mantel-Haenszel Chi-square, adjusted for county, for difference in percentage of houses at 4+ pCi/L, between NJDEP study and case-control study: 0.0, 1 d.f., p = 0.99.

[b] Mantel-Haenszel Chi-square, adjusted for county, for difference in percentage of houses at 4-19 pCi/L and 20+ pCi/L, between NJDEP study and case-control study: 10.5, 1 d.f., p = 0.005.

APPENDIX H

Considerations with Respect to Measurement Floor

The original protocol for this study called for making measurements of radon in the basement and in the master bedroom of the index house. There were several reasons for these decisions: The basement measurement was included as an index of the maximal potential for exposure in the house. It was also the part of the house in which the presence of a detector for a full-year's period was least likely to be an aesthetic problem for the resident; therefore, it was the part of the house where the detector was least likely to be disturbed and from which we were more likely to be able to retrieve the detector successfully after a full year. The master bedroom was selected as the room in the house where the subject probably spent the greatest period of time, i.e., 6-8 hours of sleeping per night. Ideally, it would have been optimal to make measurements on every floor of every house. However, the study budget did not allow this. Also, we had not collected information on the percentage of time spent by the subject in different areas of the house. Therefore, it would be difficult to assign appropriate weights to measurements from multiple living areas.

In many houses, the master bedroom was on the second floor; in many other houses, the master bedroom was on the first floor. In some houses, the current resident did not allow the placement of detectors in the master bedroom; therefore, they were placed in an alternate living area (another bedroom, the living room or dining room). Therefore, it was of some concern to us, whether there was any systematic difference between measurements on the second floor and measurements on the first floor, after controlling in some way for the "radon potential" of the house. If there were a systematic difference between floors, then we might have to standardize for the measurement floor in some way.

Therefore, we examined the ratio between the living area measurement and the basement measurement, in houses for which the measured living area was on the first floor versus houses for which the measured living area was on the second floor (Table H1). Because the results of the study conducted for the NJDEP (see Appendix G) had suggested that the inter-floor ratio could vary with the type of heat distribution (forced air vs. hot water/electric), we did this analysis controlling for the type of heat distribution. There was no significant difference ($p > 0.05$) in the ratio distribution for houses with first floor measurements vs. houses with second floor measurements, within either type of heat distribution. However, there were significant differences ($p < 0.001$) in the ratio distribution for forced air vs. hot water/electric houses within both first floor and second floor houses.

In this study, there were 39 houses in which both first floor and second floor measurements were made. For 28 of the 39 houses, the measurement result fell into the same interval (<1 pCi/L or 1-1.9 pCi/L) for both floors. For 9 of these houses, the measurement result was one interval higher for the first floor result than for the second floor result. For 2 of these houses, the measurement result was one interval lower for the first floor result than for the second floor result. The decision was made to use the first floor result.

For 92 of the 796 addresses with radon measurements, residential information indicated that the subject did not occupy the entire house. Rather, she lived in an apartment on the first or second floor or on the first or second floor of a multi-family house. Measurements were obtained on the correct index floor for 70 of these 92 addresses. Of the remaining 22 addresses, measurements were obtained on the second floor for 10 subjects who had lived on the first floor; measurements were obtained on the first floor for 9 subjects who had lived on the second floor. Only basement measurements were obtained for 3 addresses. All but one of these 22 addresses had a measured or estimated living area radon concentration of less than

1 pCi/L. Review of all of the results for these 22 addresses did not suggest that a higher measurement would have been obtained on the correct index floor.

TABLE H1

Ratio of living area alpha track detector results
to basement alpha track detector result,
by living area floor and by type of heat distribution,
New Jersey radon-female lung cancer case-control study, 1982-1988

	Forced Air Heating				Hot Water/Electric Heat			
	Floor=1		Floor=2		Floor=1		Floor=2	
Ratio	N	(%)	N	(%)	N	(%)	N	(%)
<0.2	0	(0%)	5	(6%)	25	(18%)	34	(17%)
0.2-0.39	33	(33%)	23	(26%)	53	(37%)	62	(32%)
0.4-0.59	32	(32%)	25	(28%)	25	(18%)	52	(27%)
0.6-0.79	18	(18%)	24	(27%)	18	(13%)	21	(11%)
0.8-0.99	9	(9%)	5	(6%)	8	(6%)	8	(4%)
1.0+	8	(8%)	6	(7%)	13	(9%)	19	(10%)

Comparison of distribution of ratio, by floor:
Within forced air heat houses: Chi-square = 9.2, df = 5, p = 0.10
Within hot water/electric heat houses: Chi-square = 4.4, df = 5, p = 0.50

Comparison of distribution of ratio, by type of heat distribution:
Within houses with 1st floor measurements: Chi-square=25.2, df = 5, $p < 0.001$
Within houses with 2nd floor measurements: Chi-square=18.1, df = 5, $p = 0.003$

APPENDIX J

Estimates of Year-round Living Area Radon Concentrations

Living area alpha track measurements were not obtained for 171 of the index addresses. Only basement alpha track measurements were completed for 55 addresses. Only charcoal canister measurements were completed for 77 addresses. No radon measurements were conducted at the 39 addresses which were apartments above the second floor. Living area radon concentrations were estimated for these 171 addresses. This estimation process was conducted without knowledge of the case-control status of the subject, and is described below. Analyses excluding the estimates are presented in Appendix K.

(1) Estimation from basement alpha track measurements. The analyses described in Appendix H suggest that there was a systematic difference in the ratio of living area alpha track measurements to basement alpha track measurements, depending on the type of heat distribution. The geometric mean of this ratio was calculated and found to be 0.48 for forced air houses and 0.36 for hot water/electric houses. For houses with only basement alpha track measurements, the living area radon level was estimated as 0.48 times the basement level for forced air houses and 0.36 times the basement level for hot water/electric houses. Of the 55 houses, only five had estimated living area radon concentrations in the 1-1.9 pCi/L range; these had basement concentrations of 3.8, 2.7, and 2.7 (forced air houses), and 4.1 and 3.5 (hot water/electric houses). The remainder had estimated living area radon concentrations which were less than 1 pCi/L.

(2) Estimation from charcoal canister measurements. In this study, living area alpha track results were measured as <1 pCi/L for 50 (86%) of 58 forced air houses with basement canister results below the minimum detectable concentration. Living area alpha track results were measured as <1 pCi/L for 120 (98%) of the 123 hot water/electric houses with basement canister results below the minimum detectable

basement canister results below the minimum detectable concentration. All but one also had living area charcoal canister results which were also below the minimum detectable concentration. For these 45 houses, the living area alpha track concentrations were estimated as less than 1 pCi/L.

Of the remaining houses with only charcoal canister results, 11 had no basement charcoal canister measurements, but only living area charcoal canister results which were all below the minimum detectable concentration. For these 11 houses, the living area alpha track concentrations were also estimated as less than 1 pCi/L.

The distributions of basement canister results, living area canister results, and living area alpha track results for those houses with complete measurements were used to estimate the living area alpha track results for the 21 remaining houses with only basement and living area charcoal canister results. This estimation process was done separately for forced air houses and hot water/electric houses. For 14 of these houses, the living area alpha track concentrations were also estimated as less than 1 pCi/L. The following is a list showing the characteristics of the 7 houses for which living area alpha track concentrations were estimated as 1-1.99:

Heat distribution	Basement canister	Living area canister
Forced air	4.4 pCi/L	MDC=0.5 pCi/L
Forced air	2.5 pCi/L	2.7 pCi/L
Forced air	6.6 pCi/L	2.2 pCi/L
Forced air	4.4 pCi/L	MDC=0.7 pCi/L
Hot water/electric	6.4 pCi/L	MDC=1.4 pCi/L
Hot water/electric	7.3 pCi/L	3.1 pCi/L
Hot water/electric	3.0 pCi/L	1.8 pCi/L

(3) Estimation for apartment 3+ addresses. As part of the initial study protocol, it was assumed that subjects whose index address was an apartment above the second floor would have negligible radon exposures[1]. Therefore, all of these addresses were estimated as having exposures less than 1 pCi/L.

[1]Cohen, BL, Gromicko, N. Radon-222 levels in low income households. Health Phys 56: 349-353 (1989).

APPENDIX K

Analyses Excluding Estimates of Radon Concentrations

In this study, living area radon concentrations corresponding to alpha track measurements were estimated for 171 addresses, as described in the text (p. 17) and in Appendix J. To determine the effect of this estimation process on the outcome of the results, key analyses were repeated excluding the subjects for whom radon concentrations were estimated. In addition, as reported in Appendix C, through validation of residential histories, eight subjects had corrected residence dates which no longer met the original eligibility criteria for the study. Two of these eight were already excluded because they had estimated living area radon concentrations. The other six were also excluded from the analyses presented in this Appendix. Table K1 presents results analagous to Table 6. Table K2 presents results analagous to key results in Tables 7, 9 and 10.

Analyses of cumulative radon concentrations were also repeated excluding the subjects for whom radon concentrations were estimated. Tables K3 and K4 present results analagous to Tables 14 and 15.

One important point about these sensitivity analyses needs to be made. Whereas the estimates themselves are approximations, analyses without the estimates are biased in a different respect. The index residences without living area alpha track measurements tended to be those with low canister results, because occupants who received reports of such results had a much poorer return rate for their alpha track detectors. Therefore, the analyses without the estimates are biased toward residences with higher results.

TABLE K1
Distribution of lung cancer cases and controls by radon level (year-long living area alpha track measurements, n=658)[a] and by lifetime average daily cigarette consumption, New Jersey radon-female lung cancer case-control study, 1982-1988

Smoking status	Radon (pCi/L) <1.0	1-1.9	2-3.9	4-11.3	Total
Nonsmokers					
Cases	36 (73.5%)	11 (22.5%)	1 (2.0%)	1 (2.0%)	49
Controls	126 (74.1%)	38 (22.4%)	5 (2.9%)	1 (0.6%)	170
Unadjusted OR	**1.0[b]**	**1.0**	**0.70**	**3.5**	**1.0[c]**
<15 cigs/day					
Cases	48 (72.7%)	12 (18.2%)	3 (4.6%)	3 (4.6%)	66
Controls	60 (84.5%)	11 (15.5%)	0 (-)	0 (-)	71
Unadjusted OR	**2.8**	**3.8**	**oo**	**oo**	**3.3**
15-24 cigs/day					
Cases	106 (74.1%)	26 (18.2%)	10 (7.0%)	1 (0.7%)	143
Controls	41 (78.9%)	9 (17.3%)	2 (3.9%)	0 (-)	52
Unadjusted OR	**9.0**	**10.1**	**17.5**	**oo**	**9.6**
25+ cigs/day					
Cases	69 (82.1%)	10 (11.9%)	4 (4.8%)	1 (1.2%)	84
Controls	15 (65.2%)	4 (17.4%)	3 (13.0%)	1 (4.4%)	23
Unadjusted OR	**16.1**	**8.8**	**4.7**	**3.5**	**12.7**
Total					
Cases	259 (75.7%)	59 (17.3%)	18 (5.3%)	6 (1.8%)	342
Controls	242 (76.6%)	62 (19.6%)	10 (3.2%)	2 (0.6%)	316
Unadjusted OR	**1.0[d]**	**0.89**	**1.7**	**2.8**	

[a] excludes subjects whose index address was an apartment above the second floor or a trailer, as well as other subjects whose living area alpha track results were estimated from basement alpha track results or from charcoal canister results. Also excludes eight subjects (six with measured living area alpha track results) for whom validation of residential history information showed that they no longer met the original criteria for eligibility for the study (see Appendix F).

[b] Unadjusted odds ratio (an estimate of the lung cancer risk associated with radon exposure and smoking, but not adjusted for any other factors), relative to nonsmokers with < 1.0 pCi/L radon exposure. (contd.)

Table K1 (contd)

[c] Unadjusted odds ratio (an estimate of the lung cancer risk associated with smoking, but not adjusted for radon exposure or any other factors), relative to lifetime nonsmokers.

[d] Unadjusted odds ratio (an estimate of the lung cancer risk associated with radon exposure, but not adjusted for smoking or any other factors), relative to subjects with < 1.0 pCi/L radon exposure.

TABLE K2

Odds ratios[a] (90% confidence intervals) for association of lung cancer with radon (year-long living area alpha track measurements, n=658)[b] in ALL SUBJECTS, and EXCLUDING HEAVY SMOKERS, adjusting for multiple risk factors and subject characteristics, New Jersey radon-female lung cancer case-control study, 1982-1988

	Radon (pCi/L)				Trend	
Smoking status:	<1.0[c]	1-1.9	2-3.9	4-11.3	Zcat[d] (p)	Zcnt[e] (p)
ALL SUBJECTS						
Adjusted by cigarettes/day	1.0	1.0 (0.71,1.5)	1.2 (0.58,2.7)	3.5 (0.80,14.9)	**1.30 (0.097)**	**1.13 (0.129)**
[LR=152.8, 4df]+			1.6[f] (0.79,3.0)			
Adjusted by cigarettes/day, age, occupation, yrs quit smoking, respondent type, resptype*cigs/day	1.0	1.1 (0.71,1.6)	1.3 (0.59,2.8)	3.5 (0.80,15.7)	**1.37 (0.085)**	**1.06 (0.145)**
[LR=219.4, 17df]+			1.6 (0.81,3.3)			
ALL EXCEPT HEAVY SMOKERS						
Adjusted by cigarettes/day	1.0	1.1 (0.77,1.7)	2.0 (0.83,4.8)	8.7 (1.3,56.9)	**2.30 (0.011)**	**1.99 (0.023)**
[LR=118.6, 3df]+			2.8 (1.3,6.0)			
Adjusted by cigarettes/day, age, occupation, yrs quit smoking, respondent type, resptype*cigs/day	1.0	1.1 (0.75,1.8)	2.0 (0.80,5.0)	8.1 (1.2,54.8)	**2.19 (0.014)**	**1.82 (0.034)**
[LR=164.9, 13df]+			2.8 (1.2,6.3)			

+ Likelihood ratio statistic for model including categorical "radon trend" term (with degrees of freedom).

[a] Odds ratios (OR) and 90% confidence intervals from logistic regression analyses. (contd).

Table K2 (contd)

[b] excludes subjects whose index address was an apartment above the second floor or a trailer, as well as other subjects whose living area alpha track results were estimated from basement alpha track results or from charcoal canister results. Also excludes eight subjects (six with measured living area alpha track results) for whom validation of residential history information showed that they no longer met the original criteria for eligibility for the study (see Appendix F).

[c] Includes subjects whose index address was an apartment above the second floor or a trailer.

[d] Z statistic (1-sided p value) for categorical "radon trend" term in logistic regression model. This term equals 0.4 if radon is <1 pCi/L, 1.2 (1-1.9 pCi/L), 2.3 (2-3.9 pCi/L), or 4.55 (4+ pCi/L). These values are the medians of the respective intervals for controls. This model gives results equivalent to the Mantel Chi-extension procedure for stratified analyses.

[e] Z statistic (1-sided p value) for continuous radon variable in logistic regression model.

[f] OR (90% confidence interval) for radon = 2+ pCi/L.

TABLE K3
Distribution of New Jersey female lung cancer cases and controls by cumulative radon exposure[a] (excluding all radon estimates)[b] and by lifetime average daily cigarette consumption, New Jersey radon-female lung cancer case-control study, 1982-1988

Smoking status	Cumulative radon (pCi/L-years) <25	25-49	50-99	100-155	Total
Nonsmokers					
Cases	40 (81.6%)	8 (16.3%)	0 (-)	1 (2.0%)	49
Controls	132 (77.7%)	33 (19.4%)	4 (2.4%)	1 (0.6%)	170
Unadjusted OR	**1.0[c]**	**0.80**	**0.00**	**3.3**	**1.0[d]**
<15 cigs/day					
Cases	51 (77.3%)	10 (15.2%)	3 (4.6%)	2 (3.0%)	66
Controls	65 (91.5%)	6 (8.5%)	0 (-)	0 (-)	71
Unadjusted OR	**2.6**	**5.5**	**oo**	**oo**	**3.2**
15-24 cigs/day					
Cases	113 (79.0%)	22 (15.4%)	8 (5.6%)	0 (-)	143
Controls	44 (84.6%)	6 (11.5%)	2 (3.8%)	0 (-)	52
Unadjusted OR	**8.5**	**12.1**	**13.2**	**—**	**9.5**
25+ cigs/day					
Cases	73 (86.9%)	9 (10.7%)	1 (1.2%)	1 (1.2%)	84
Controls	16 (69.6%)	4 (17.4%)	3 (13.0%)	0 (-)	23
Unadjusted OR	**15.1**	**7.4**	**1.1**	**oo**	**12.7**
Total					
Cases	277 (81.0%)	49 (14.3%)	12 (3.5%)	4 (1.2%)	342
Controls	257 (81.3%)	49 (15.5%)	9 (2.9%)	1 (0.3%)	316
Unadjusted OR	**1.0[e]**	**0.93**	**1.2**	**3.7**	

[a] cumulative radon exposure during 25 years from 5-30 years prior to case diagnosis or control selection; assumes exposure of 0.6 pCi/L (median for controls) for any of the 25 years during which the subject did not live in the index address where the measurements were made.
[b] excludes subjects for whom living area alpha track results were estimated.
[c] Unadjusted odds ratio (an estimate of the lung cancer risk associated with radon exposure and smoking, but not adjusted for any other factors), relative to nonsmokers with < 25.0 pCi/L-years cumulative radon exposure.
[d] Unadjusted odds ratio (an estimate of the lung cancer risk associated with smoking, but not adjusted for radon exposure or any other factors), relative to lifetime nonsmokers.
[e] Unadjusted odds ratio (an estimate of the lung cancer risk associated with cumulative radon exposure, but not adjusted for smoking or any other factors), relative to subjects with < 25.0 pCi/L-years cumulative radon exposure.

TABLE K4

Odds ratios[a] (90% confidence intervals) for association of lung cancer with cumulative radon exposure[b] (excluding radon estimates)[c] in ALL SUBJECTS, and EXCLUDING HEAVY SMOKERS.
New Jersey radon-female lung cancer case-control study, 1982-1988

Smoking status	Cumulative radon (pCi/L-years) <25	25-49	50-99	100-155	Trend Zcat[c] (p)	Trend Zcnt[d] (p)
ALL SUBJECTS						
Adjusted by cigarettes/day **[LR=152.0, 4df]+**	1.0	1.1 (0.73,1.7)	0.84 (0.37,1.9)	6.9 (0.99,48.3)	**0.96 (0.167)**	**0.86 (0.195)**
			1.2[e] (0.58,2.7)			
Adjusted by cigarettes/day, age, occupation, yrs quit smoking, respondent type, resptype*cigs/day **[LR=218.2, 17df]+**	1.0	1.2 (0.76,1.8)	0.82 (0.35,1.9)	6.0 (0.80,44.9)	**0.87 (0.192)**	**0.78 (0.218)**
			1.2 (0.53,2.6)			
ALL EXCEPT HEAVY SMOKERS						
Adjusted by cigarettes/day **[LR=116.4, 3df]+**	1.0	1.2 (0.80,1.9)	1.6 (0.62,4.1)	6.8 (0.95,48.6)	**1.85 (0.032)**	**1.78 (0.038)**
			2.2 (0.92,5.2)			
Adjusted by cigarettes/day, age, occupation, yrs quit smoking, respondent type, resptype*cigs/day **[LR=162.6, 13df]+**	1.0	1.3 (0.79,2.0)	1.5 (0.55,3.9)	6.2 (0.80,46.9)	**1.67 (0.047)**	**1.67 (0.047)**
			2.0 (0.83,5.0)			

+ Likelihood ratio statistic for model including categorical "cumulative radon rend" term (with degrees of freedom)

[a] Odds ratios and 90% confidence intervals from logistic regression analyses. (contd)

TABLE K4 (contd)

[b] cumulative radon exposure during 25 years from 5-30 years prior to case diagnosis or control selection; assumes exposure of 0.6 pCi/L (median for controls) for any of the 25 years during which the subject did not live in the index address where the measurements were made.

[c] excludes subjects for whom living area alpha track results were estimated.

[d] Z statistic (1-sided p value) for categorical "cumulative radon trend" term in logistic regression model. This term equals 11.8 if cumulative radon is <25 pCi/L-years, 29.4 (25-49 pCi/L-years), 69.4 (50-99 pCi/l-years, or 109.5 (100+ pCi/l-years). These values are the medians of the respective intervals for controls. This model gives results equivalent to the Mantel Chi-extension procedure for stratified analyses.

[e] Z statistic (1-sided p Value) for continuous cumulative radon variable in logistic regression model.

[f] Odds ratio (90% confidence interval) for cumulative radon=50+ pCi/L-years.