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Health risk attributable to environmental exposures: Radon☆

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Abstract

Alternate attributable risk methodologies are examined and applied to the problem of estimating the number of annual lung cancer deaths in the United States that can be attributed to residential exposure to radon and radon progeny. The US Environmental Protection Agency's lifetable analysis, using modeled lung cancer death rates among various populations, is compared to Levin's measure of attributable risk, using lung cancer death rates in exposed and non-exposed populations as reported in epidemiological studies. Average annual residential radon concentrations are examined on the national, state, and county level. Results obtained by Levin's methodology are comparable to those obtained by EPA's most recent methodology.

1. Introduction

Attributable risk is a statistical concept that underlies statements such as this US newspaper headline, "*Radon in homes could kill 30,000 yearly*" [1]. Estimating attributable risk is part of the health risk assessment process, which uses information about toxicity and exposure to a potential hazard in order to qualitatively and quantitatively characterize risk. The qualitative description includes a discussion of methodologies, assumptions, uncertainty, and professional judgements that contribute to the conclusions about risk. Quantitative description of risk may be statistical expressions called risk descriptors, such as lifetime excess risk or attributable cases in a study population.

Risk analysts understand that risk is incompletely characterized by an unqualified numeric statement such as this headline, because any assessment integrates information

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from many different sources, with varying assumptions and degrees of scientific certainty. Attributable risk is a quantitative risk descriptor that is often used outside the context of a formal risk assessment in order to convey information about potential hazards to non-specialists. While it can be argued that successful communication of risk sometimes requires translation of the complexities of the risk assessment process into simple concepts, easily grasped by the general public, the fact remains that an unqualified statement of attributable risk is an incomplete representation of risk, open to misinterpretation by non-specialists. In this paper we will examine the concept of attributable risk within the context of the carcinogenic risk assessment process, and use it to describe the extent of lung cancer risk associated with residential exposure to radon in the United States.

2. The risk assessment process

Almost all risk assessments involve making inferences and assumptions about some factors because of lack of direct evidence. In the absence of clear scientific evidence, a public health agency must make many conservative assumptions that presumably would be least likely to underestimate risk to human health. Depending on the scope of the risk assessment, these uncertainties and assumptions may be addressed in a discussion of professional judgement involved, or they may be quantified by estimating risk as a range of possible values under varying conditions, or by subjecting the estimates to analytic techniques such as sensitivity analysis.

The hazard identification component of a risk assessment is the qualitative, descriptive evaluation of a hazardous agent's potential to cause cancer. Data on the toxicity and pharmacokinetics of a potentially harmful substance are gathered and evaluated in order to determine to what degree toxic effects observed in one setting will occur in other settings. The dose–response assessment is a quantitative description of the relationship between the magnitude of administered, applied, or internal dose and a specific biological response. Responses from human epidemiologic studies are preferred, but in the absence of appropriate human data, the dose–response assessment usually involves extrapolating the human response at low doses from the observed responses to artificially high doses administered to experimental animals of a species whose physiological responses are most like humans. Less frequently, responses observed in connection with higher occupational exposures are extrapolated to lower, more widely prevalent environmental levels of exposures.

The exposure assessment describes the magnitude, duration, and route of exposure to a hazardous agent, and the various populations that have been, are currently, or will be exposed, and estimates dose as the result of actual or possible exposures in the human environment. Environmental concentrations of a hazardous agent may be obtained by direct measurement or by modeling. For carcinogens, standard regulatory agency practice is to average exposure over the lifetime. Exposed populations may be categorized according to the magnitude of the dose received, age, sex, occupation, or the size of the population or subgroups. This information is then combined with dose–response data to estimate risk, or probability of harmful effect in the populations at risk.

The risk characterization integrates the hazard and dose–response data with the exposure assessment results, to quantitatively and qualitatively describe the probability that the populations potentially at risk will experience any of the various forms of toxicity associated with the hazardous agent in question, under known or anticipated conditions of exposure. The various estimates of risk that are calculated are collectively referred to as risk descriptors.

Unit cancer risk is the excess lifetime risk due to a continuous constant lifetime exposure of one unit of carcinogen concentration. This estimate is based on an assumption of linearity of response at low doses.

Dose per level of risk is a way of expressing risk when using non-linear response extrapolation models. It is the dose or exposure at which a specific level of risk is estimated to occur.

Individual risk descriptors are an expression of the distribution of risk within a population, including measures of central tendency among a population and the high-end portions of the risk distribution. Measures of central tendency may be either the arithmetic mean risk or the median risk (geometric mean). High-end descriptors estimate the risks that are expected to occur in small but definable segments of the subject population. These measures do not necessarily represent a particular individual within the population.

Population risk descriptors refer to the population as a whole. Population-attributable risk estimates the number of cases of a particular effect or that is probabilistically estimated to occur in a study population over a period of time. This is a hypothetical prediction, based on a combination of science fact, science policy, assumptions, and uncertainty. It is not intended to be an accurate prediction of real, individual cases of disease in the population. The attributable risk estimate translates hypothetical risk to an understandable statement, and should not be interpreted literally as a statement of real individual cases of a disease.

3. Radon and attributable risk

Radon-222 is an inert gas produced by the decay of uranium and is found throughout the geosphere. Radon formed in rocks and soil can enter buildings through any penetration in walls and floors, such as foundation joints, cracks in floors and walls, piping and drains. In a confined space, such as mines or inside buildings, low rates of air change can result in a buildup of radon and its decay products to levels tens of thousands of times higher than those typically observed outdoors. Radon can also enter the indoor environment from building construction materials, from natural gas, and from well waters. In the US, these sources usually contribute much less than soil to the total radon level inside a building [2].

Recognition of radon as a possible health hazard is relatively recent, beginning with the observation of excess lung cancer in underground miners in various parts of the US and other countries during the middle of this century. As radon decays, it generates intermediate products or radon progeny, which emit α radiation. Although α-emitting particles travel less than 100 μm into the tissue, their high energy causes an

intense local ionization, causing damage at the cellular and molecular level, with subsequent risk for cancer development. Radon decay products are easily inhaled, allowing the α-emitting particles to kill or damage cells, particularly the stem cells of the bronchial epithelium. Stem cells, precursors of many different kinds of cells, divide frequently, and the α-particle bombardment increases the possibility of a cancer-causing mutation of the stem cell's DNA.

3.1. Exposure to radon

Measurement of exposure. The activity concentration of radon in air is expressed in becquerels per cubic meter of air (Bq/m^3) or in picocuries per liter of air (pCi/l). A concentration of 150 Bq/m^3 is about equal to 4 pCi/l, the level at which the US Environmental Protection Agency (EPA) recommends taking remedial action. The concentration of radon decay products in air is expressed as the total potential α energy concentration of the radon decay product mixture present. The unit of measurement of the potential α energy concentration of radon decay products in air developed in assessing the exposure of miners is known as the working level (WL). A WL is defined as any combination of short-lived radon decay products in one liter of air that will result in the emission of 1.3×10^5 million electron volts (MeV) of potential α energy.

The equilibrium relationship between air concentration of radon (pCi/l) and radon decay products (WL) depends upon the ratio of the decay products to radon. Radioactive equilibrium occurs when every short-lived radon decay product is present at the same activity as radon. In confined air spaces the activity concentration of radon never reaches radioactive equilibrium with radon, due mainly to ventilation and the deposition of radon decay products on surfaces. In a mine, one WL of potential α energy is about equivalent to 300 pCi/l of radon; in a home it is about 200 pCi/l, and outdoors it is about 150 pCi/l [3].

Exposure is defined in terms of the working level month (WLM). One WLM is defined as exposure to one WL of potential α energy for a working month of 170 h. A residential indoor air concentration of radon measured at 4 pCi/l (150 Bq/m^3) would be equal to 0.02 WL of decay products. Continuous full-time exposure to that level of radon for one year would result in an exposure of 1 WLM/year.

Prevalence of indoor exposure. The prevalence of exposure of the US population to indoor radon and radon progeny is difficult to estimate. Due to varying construction, ventilation, and building use characteristics, indoor radon concentrations can vary widely within the same geographic area having the same bedrock type and soil permeability. To estimate the average annual exposure across the US, it is necessary to take accurate measurements in a statistical sample of the entire US residential building stock, taking into account these differences. Because of seasonal variations in ventilation, measurements have to be taken several times during a year, and averaged to estimate an accurate annual concentration.

Nero and colleagues [4] systematically analyzed the full range of US radon home monitoring data available through 1984, and aggregated them in a consistent fashion to estimate a frequency distribution of estimated annual average concentrations.

Based on 800 homes in 17 states, Nero estimated that in 7% of the homes in the US, average annual radon concentrations exceeded EPA's remedial action level of 4 pCi/l [5].

In 1986, Cohen [6] examined year-long monitoring of radon concentrations in 453 homes from 42 states and found 5% of the homes had concentrations above 4 pCi/l. His final compilation of mean concentrations for US counties and states was recently published, in which about 272,000 measurements from the University of Pittsburgh Radon Project, 40,000 EPA state-level measurements, and measurements from several state-sponsored studies were normalized to a national average in agreement with the EPA's recently completed National Residential Radon Survey [7]. The combined county data set may be interpreted as actual average radon levels in each of 1705 counties; the study also lists average radon levels in each state, derived from population-weighted averages for the counties in each state. These county and state estimates are illustrated in Figs. 1 and 2.

The National Residential Radon Survey (NRRS) estimates a national frequency distribution of average annual radon concentrations in occupied housing units across the 50 United States. Alpha track detectors were placed for 12 months in approximately 7100 of 11,000 eligible homes, randomly selected in a nationwide multi-stage probability sample of US housing stock in regular use. Single family detached homes, multi-unit structures, and mobile homes were covered in the survey.

The results of the NRRS indicated the arithmetical average annual residential concentration of radon in the US as a whole, was 1.25 pCi/l (46 Bq/m^3), 95% CI = $\pm$ 9%. Median value was 0.67 pCi/l (25 Bq/m^3). The average annual radon concentration exceeded the US EPA action level of 4 pCi/l (150 Bq/m^3) in 6.01% (SE $\pm$ 0.68) of the US housing units, found in all parts of the US. Conclusions from this survey are applicable only to indoor radon concentrations for the entire country, and for the ten EPA regions [8, 9].

3.2. Attributable risk methodologies

Ideally, to estimate population-attributable risk, one would conduct an experiment measuring disease rates before and after the complete elimination of the risk factor from the population under study. Since this would be impractical for the most part among human populations, the most widely used approach is to study disease incidence or mortality rates in a representative sample of the population, then carefully extrapolate the results to the larger population of interest.

EPA's estimates. Increased incidence of lung cancer has been associated with occupational exposure of uranium miners to high levels of radon [10–12], but the magnitude of the risk from exposure to radon at lower indoor levels is still under debate. Since a sizeable portion of the mining cohorts is still alive, the mortality data needed to estimate the risk of exposure to occupational levels of radon relative to background exposure levels is not yet complete. Epidemiologists must use numeric models to predict rather than observe the future lung cancer mortality rate of this population. Relative risk prediction models have been developed by the International Commission on Radiation Protection (ICRP), the National Academy of Science's

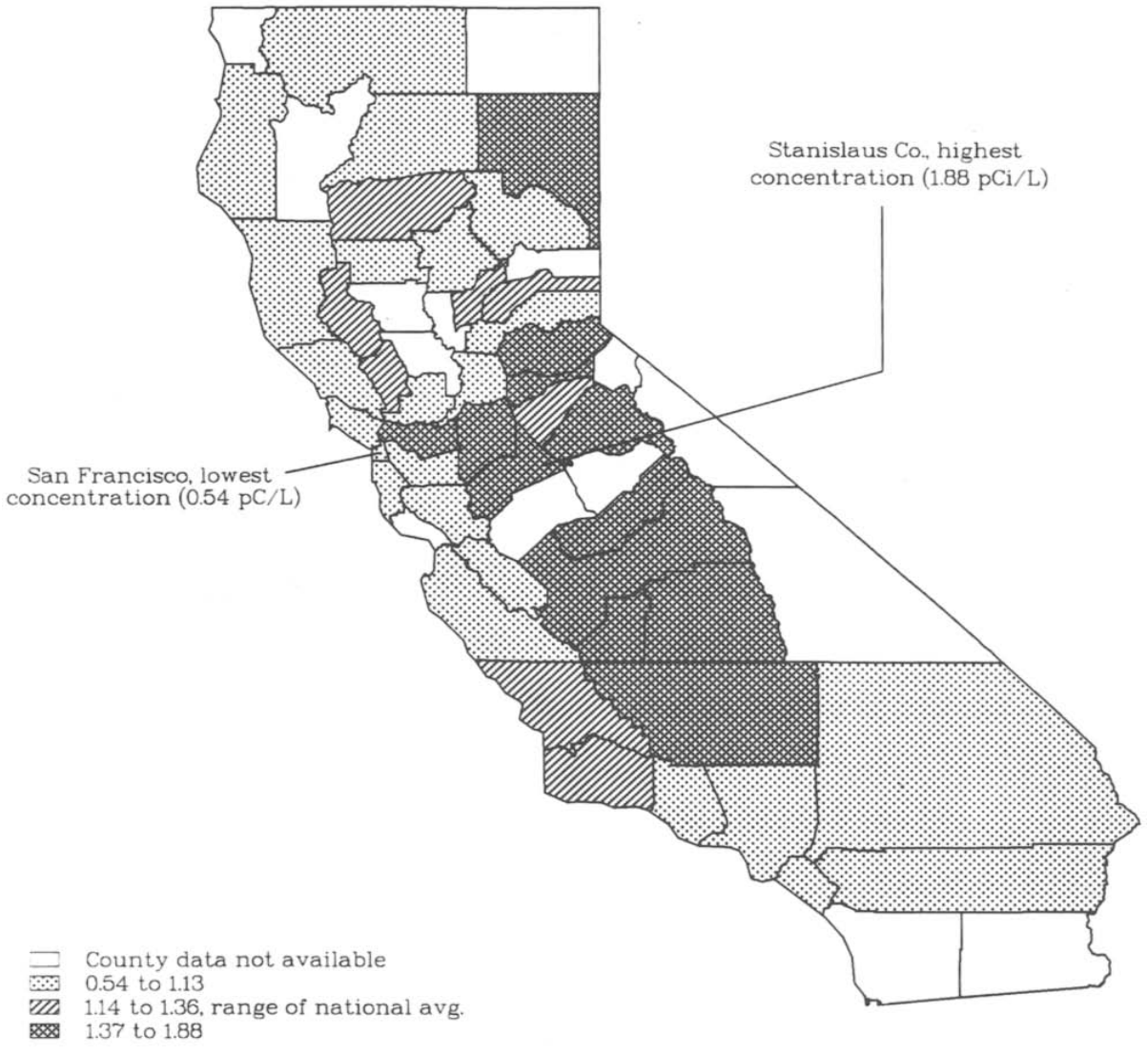

Fig. 1. Estimated annual average radon concentrations in California homes, by county, based on data compiled by Cohen [7].

Committee on the Biological Effects of Ionizing Radiation (BEIR), and the EPA [13–15]. All risk prediction models incorporate risk coefficients based on incomplete studies of miners, and extrapolation of occupational exposure to lower indoor exposures. They all differ in their underlying assumptions and consequently in the resulting risk projections.

In conjunction with modified versions of both the BEIR and ICRP models, and 1980 US age-specific mortality rates and vital statistics, EPA used a standard lifetable analysis to calculate a lifetime, age-averaged death rate for radon-induced lung cancer of 360 deaths per 10^6 person-WLM (90% CI = 140–720 deaths) in the general US population [16]. The ICRP assumed the amount of radiation actually absorbed per WLM for the general population was only 80% of the amount absorbed by miners;

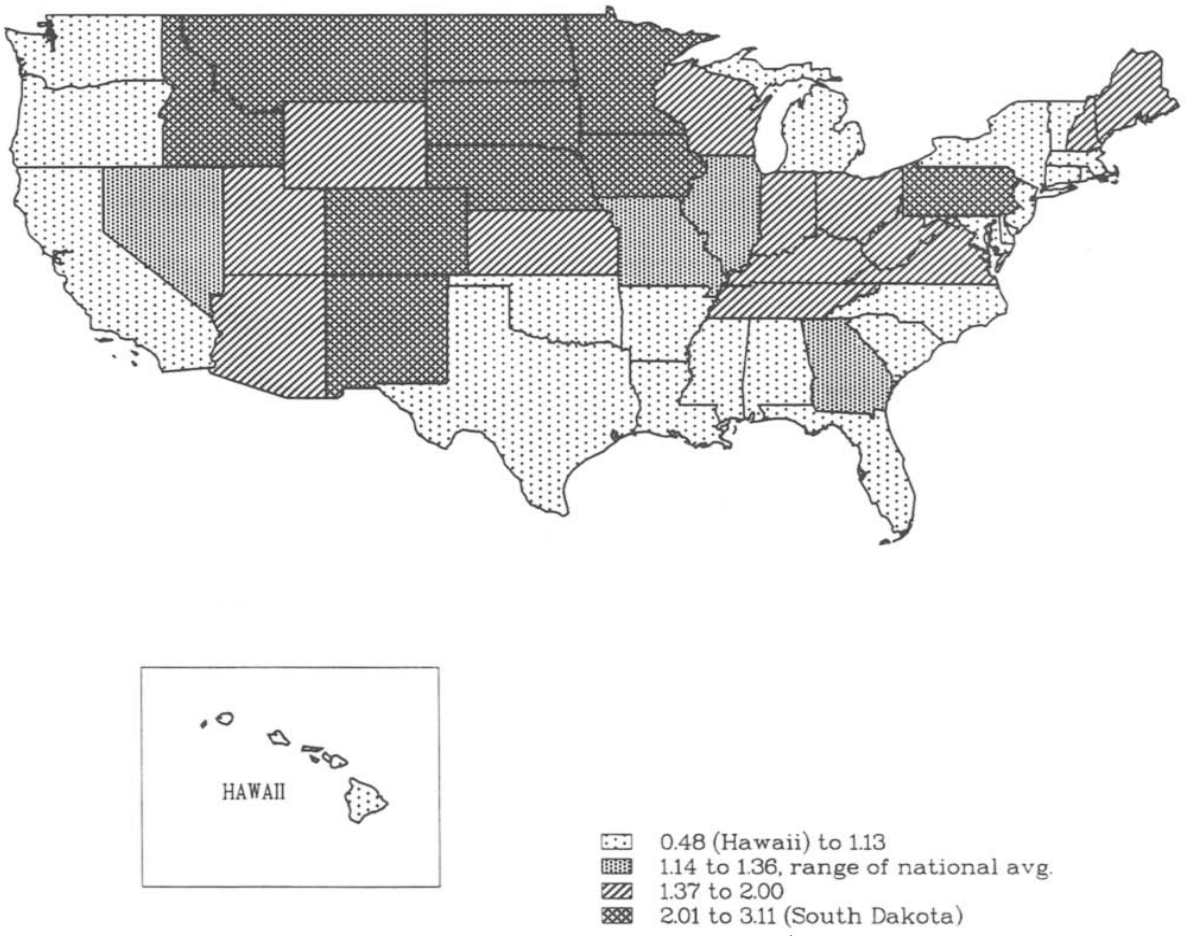

Fig. 2. Estimated annual average radon concentrations in US homes, by state, based on data compiled by Cohen [7].

however the BEIR committee in 1988 recommended considering the exposures identical, and EPA's above estimate of radon-induced death rate is based on the assumption that indoor and mining exposures result in the same dose per WLM. Based on a steady-state population, EPA estimated the annual lung cancer deaths due to a constant lifetime radon exposure of 0.25 WLM/yr to be 21,600. The estimate was based on an indoor equilibrium fraction of 50%, an average annual indoor radon level of approximately 1.3 pCi/l at 75% occupancy, and a population of 240,000,000, the 1985 total US population [15].

A later National Academy of Sciences report was found that the actual radiation dose to the bronchial epithelium for residential radon exposure had been overestimated by 30%. Consequently the EPA annual radon-induced lung cancer death estimate was revised downward to about 13,600 lung cancer deaths per year, with an uncertainty range of 7000 to 30,000 deaths per year. Also included in the revision was a new average annual exposure of 0.242 WLM/yr and an average residential radon level of about 1.25 pCi/l, from EPA's National Residential Radon Survey [17].

Levin's measure of attributable risk is a method widely used to estimate attributable risk from environmental risk factors where more complete relative mortality data is available from epidemiological studies [18, 19]. It is derived as follows.

Relative risk r is estimated by observing the ratio of different disease incidence or mortality rates among exposed and unexposed portions of a sample population, d_1 and d_0.

$$r = d_1/d_0. \tag{1}$$

Relative risk is combined with data on prevalence of exposure to obtain an estimate of the attributable risk among the larger population under study. Levin's measure of attributable risk summarizes the relationship between relative risk r in a sample population and prevalence of exposure p among a larger population.

$$a = \frac{p(r-1)}{p(r-1)+1}. \tag{2}$$

This attributable risk fraction a is then multiplied by the number of new cases or deaths D that occurred among the larger population during the same time period in which the extent of exposure was estimated. The result is attributable cases or deaths A, a probabilistic estimate of the number of premature occurrences of the disease or mortality that could have been avoided by elimination of exposure to the risk factor.

$$A = aD. \tag{3}$$

Eq. 2 shows how the attributable risk a depends on both the relative risk r and the prevalence p or proportion of total subject population exposed to given risk factor. A health hazard associated with a low relative risk and widespread population exposure can yield as high an attributable risk as a hazard associated with a very high relative risk and a very low prevalence of exposure. Assuming that the relative risk remains unchanged, an estimate of attributable cases or deaths will change as the time frame changes, because the number of new cases or deaths (D) in the subject population changes over time, as does the prevalence of exposure.

Attributable risk, then, is the proportion of all cases (deaths) in the total subject population that is statistically attributable to a given risk factor during a specified period of time. Epidemiological studies upon which the relative risk estimate is based present an observation of a statistical association between exposure to a risk factor and disease, allowing as much as possible for confounding factors. The finding of an increased relative risk in an epidemiological study is in itself insufficient to establish causation. The determination of causation requires consideration of several additional issues, which will not be addressed here. 'Attributable to' does not mean 'caused solely by'. What it does mean is that there is a high probability (within certain error limits) that a given risk factor plays a part in an estimated fraction of cases or deaths, limited by the degree of uncertainty that was incorporated into the epidemiological studies and surveys upon which the risk estimate was based. A statement of attributable risk does not necessarily include the notion of interaction between components of causal mechanisms – proportions of the same disease that are attributed to various risk factors are not mutually exclusive.

The preceding estimate of annual attributable lung cancer deaths, based on relative risk models and lifetable analysis, will be compared with the number of attributable deaths that can be estimated using Levin's model of attributable risk, and on relative risks calculated in recently completed epidemiological studies of human responses to residential levels of radon exposure. The EPA's lifetable-based estimate is valid only with respect to the 1980 background lung cancer rate; as the background rate changes, the risk estimates will also change. The estimates of radon-attributable lung cancer deaths, based on Levin's measure of attributable risk, are also dependent upon the lung cancer mortality rates in the general population during a specific time frame, in this case, the year 1985.

Residential radon studies. An early epidemiological study of lung cancer and residential radon exposure in Sweden used type of residence as a proxy for exposure, reporting a relative risk of 1.8 [20]. Later studies in Sweden characterized exposure by using residence type and soil parameter proxies, as well as measurements in at least a portion of the houses. Relative risks in these studies ranged from 2.0 to 2.7 [21–23].

A recent report of a case-control study of lung cancer incidence among women in Stockholm county, Sweden, and radon concentrations in a random sample of dwellings in which study subjects had lived, indicated dwellings with ground contact had an average concentration of approximately 160 Bq/m^3 (over 4 pCi/l), twice the average concentration of other dwellings. Based on a cumulated radon exposure index and the residential radon measurements, the relative risk of lung cancer for the total group, adjusted for smoking, age, and degree of urbanization, was 1.7 (95% CI = 0.9–3.3) for the higher environmental exposure level to radon [24]. Radon concentrations were eventually measured in all residences in which subjects had lived; based on these exposure measurements, relative risk for lung cancer was 1.7 (95% CI = 1.0–2.9) for women exposed to an average indoor radon concentration level greater than 150 Bq/m^3 (4 pCi/l) [25].

The New Jersey Department of Health's retrospective case-control study of lung cancer and exposure to radon in women also used measurements of radon levels in subjects' homes [26]. None of the individual odds ratios was found to be statistically significant, but a significant trend for lung cancer risk and increasing radon concentration was found ($p = 0.04$). Adjusted for other factors such as cigarette smoking and occupation, the odds ratios were 1.1 (90% CI = 0.79–1.7), 1.3 (90% CI = 0.62–2.9), and 4.2 (90% CI = 0.99–17.5) for year-round average living-area radon concentrations of 1.0–1.9 (37–73), 2.0–3.9 (74–147), and 4.0–11.3 (148–418) pCi/l (Bq/m^3) respectively.

Because of the small number of subjects in the two upper exposure groups (less than 5% of the study subjects), they were combined in a later analysis of the data, resulting in an odds ratio of 1.8 (90% CI = 0.89–3.5) for the upper levels of exposure. New information indicating a shorter period between relevant radon exposure and diagnosis of lung cancer allowed a reassessment of earlier residence criteria, resulting in more eligible subjects, as well as more residences for some earlier subjects. Analysis of this second set of subjects by time-weighted average radon exposures in one or more residences estimated a statistically significant odds ratio of 8.7 for the upper level of exposure. Since again so few subjects were found in the highest exposure group, the

Table 1
Estimates of attributable lung cancer deaths from residential radon exposure in the US, 1985, using Levin's measure of attributable risk; r is from cited study, $a = p(r-1)/[p(r-1)+1]$, and $A = aD$

Study	Exposure level (pCi/l)[a]	Relative risk (unitless) r	Prevalence (%) p [8]	Attributable risk (%) a	Total LC deaths D [28]	Attributable LC deaths A
Using point estimates of relative risk						
New Jersey, Phase I [26]	4.0–11.3	4.2	6.0	16.1	122,538	19,738
New Jersey, Phase I [26]	2.0–11.3 (upper levels combined)	1.8	17.0	12.0	122,538	14,670
New Jersey, Phase II [27]	4.0–11.3	8.7	6.0	31.6	122,538	38,723
New Jersey, Phase II [27]	2.0–11.3 (upper levels combined)	1.6	17.0	9.3	122,538	11,342
Sweden [25]	> 4.0	1.7	6.0	4.0	122,538	4939
Using upper limit of confidence interval estimates of relative risk						
New Jersey, Phase I [26]	2.0–11.3 (upper levels combined)	3.5	17.0	29.8	122,538	36,546
New Jersey, Phase II [27]	2.0–11.3 (upper levels combined)	3.1	17.0	26.3	122,538	32,237
Sweden [25]	> 4.0	2.9	6.0	10.2	122,538	12,540

[a] Annual average indoor concentration for US.

upper two exposure groups were again combined, resulting in an odds ratio of 1.6 (90% CI = 0.81–3.1) for the upper levels of exposure [27].

Using the New Jersey adjusted odds ratios and the Stockholm study's relative risk, and prevalence data from US EPA's National Residential Radon Survey, we have calculated a range of estimates of attributable lung cancer deaths in the US during 1985, using Levin's measure of attributable risk. The results are summarized in Table 1.

4. Discussion

The estimates of attributable deaths for 1985 in Table 1 show variation in the point estimates of about an order of magnitude, from the Swedish study's estimate of about 4900 deaths at exposure level > 4.0 pCi/l, to the New Jersey phase II upper exposure level estimate of about 39,000 deaths at a similar exposure level (from 4.0 to 11.3 pCi/l). The Swedish study involved 210 cases and 400 controls with radon concentrations measured in 1573 residences of the study subjects; the New Jersey

Table 2
Radon-attributable lung cancer deaths in the US from residential exposure estimated by different methodologies

Study		Estimated annual attributable lung cancer deaths
Risk projection model, lifetable method		
EPA, modified	point estimate, lower uncertainty limit	7000
dosimetry [17]	point estimate	13,600
	point estimate, upper uncertainty limit	30,000
Direct epidemiological studies, Levin's measure of attributable risk		
New Jersey,	point estimate[a]	14,600
Phase I [26]	upper limit, 90% CI[a]	36,500
New Jersey,	point estimate[a]	11,300
Phase II [27]	upper limit, 90% CI[a]	32,200
Sweden [25]	point estimate	4900
	upper limit, 95% CI	12,540

[a] Combined upper exposure levels.

phase II study included 480 cases and 442 controls, and radon measurements in one or more residences occupied by study subjects 5 to 30 years prior to selection or diagnosis. Both studies were controlled for smoking, and the New Jersey study was also controlled for occupation.

Because of the small numbers in the upper exposure level of both phases of the New Jersey study, it is necessary to interpret the results very cautiously. Considering only the combined upper exposure level results from the New Jersey study and the results from the Swedish study, estimates of attributable lung cancer deaths based on these two epidemiological studies are within the range of the deaths predicted by the latest EPA estimates. Leaving out all phase II results from the New Jersey study, on the basis that phase II is based on better exposure data, the range of deaths estimated is still similar to the range predicted by EPA. This comparison is summarized in Table 2.

Table 2 shows fairly good agreement between the two different methodologies for estimating annual attributable deaths, even though the epidemiological studies considered here are based on relatively small numbers of subjects. Studies involving larger cohorts will be able to more reliably estimate the true extent of the health risk from radon. Other uncertainties are involved in the design of future epidemiological studies of radon. As with the mining cohort studies, residential studies involve the task of estimating radon exposures that occurred in the past. Short-term measurements averaged over a year are used to estimate long term or lifetime exposures. The relatively high mobility of the middle to late twentieth century population in the US poses additional problems in assessing residential exposure to radon. Results from current and future epidemiological studies of residential radon exposure, as well as the

eventual completion of the studies on mining cohorts, will increase the accuracy of radon-attributable lung cancer estimates.

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