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# Examining the relationship between lung cancer and radon in small areas across Scotland

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## Abstract

Numerous studies have suggested that long-term exposure to radon gas may be an important cause of lung cancer, yet the precise effects are still not fully understood, especially in residential settings. This paper considers whether there is a relationship between the distribution of naturally occurring radon gas and lung cancer incidence in Scotland, for the period 1988–1991. We use regression analysis to test whether exposure to radon was a significant cause of lung cancer in Scotland, once smoking and other possible confounding factors were controlled for. The results demonstrate that for the population aged over 54, there was no significant relationship between radon exposure and lung cancer incidence. However, for those aged less than 55, lung cancer rates were significantly higher in places expected to have the highest levels of radon. These results suggest that more research is needed into the relationship between exposure to naturally occurring radon gas and lung cancer in Scotland, particularly among younger age groups.

*Keywords:* Lung cancer; Radon; Scotland

## Introduction

Approximately 85 % of the radiation to which the majority of the UK population is exposed is naturally occurring and, of this, radon gas is the largest source (Hughes and O'Riordan, 1993). Radon is generated in rock, soil, and building materials and diffuses readily in the open air. However, it can attain relatively high levels, particularly in poorly ventilated rooms (Green et al., 1996). A number of studies have suggested that long-term exposure to radon may be an important cause of lung cancer (Darby et al., 1998), yet the precise effects are still not fully understood, especially in residential settings.

This confusion has arisen because most studies, both ecological and individual-level, have been compromised by the paucity of accurate geographically detailed estimates of radon exposure. Radon levels are difficult to estimate because they fluctuate over small distances in response to differences in the geology, drift, transmission characteristics of the soil, and details of housing construction (Ball et al., 1991). This may be one reason why some ecological studies have found a significant positive relationship between radon exposure and lung cancer (Magnus et al., 1994) whereas others have found no correlation (Etherington et al., 1996).

Some argue that individual-level studies, which include personal exposures to radon, are more informative than ecological studies, which rely on broad

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predictions for large areas (Darby et al., 2001). Thus, Darby et al. (1998) collected radon measurements at the addresses at which subjects of their study in south-west England had lived over the previous 30 years, in order that time-weighted average indoor radon concentrations could be assessed. They found that the relative risk of lung cancer increased by 0.08 per 100 Bqm$^{-3}$ rise in the time-weighted residential radon concentration. However, other studies that have followed the long-term exposure of lung cancer subjects and their controls have found that there is either no relationship or only a rather weak one (Alavanja et al., 1994; Auvinen et al., 1996).

Individual studies are often prohibitively expensive because the residential history of each individual has to be tracked and radon measurements taken at each address in order that precise personal estimates of radon exposure can be estimated. Sample sizes in these types of study are often small. Even then, it is usually impossible to know if radon levels have fluctuated at a particular address during the period between exposure and diagnosis or whether the individual has been exposed to radon in a non-residential setting. Therefore, despite the perceived advantages of individual studies over ecological studies, the results from the former could be argued to be no more conclusive. Furthermore, although the radon exposure estimates used in ecological studies have been criticised, a parallel study using individual and ecological radon data in south-west England demonstrated that both types of studies are subject to many biases. Given the different biases associated with individual and ecological studies of radon, the authors suggest that it is 'at least logically possible' that the true effect of radon may be closer to the ecological associations than to the individual level associations (Darby et al., 2001), particularly because ecological estimates provide a general estimate of radon in the local area, rather than only in the residential setting. Unfortunately, most of the ecological studies that have been conducted to date have relied on radon estimates for relatively broad geographical units (Haynes, 1988). Few studies have considered radon estimates derived for small geographical units and in those cases where small area estimates of radon did exist other confounding variables, such as smoking, were not available for these geographical units (Etherington et al., 1996).

This study addresses this issue by examining the incidence of lung cancer in 38,254 small areas across Scotland for which radon estimates have been estimated. In addition, other explanatory variables, including smoking behaviour, were also available for these areas, allowing us to examine whether lung cancer incidence is related to radon levels, controlling for other factors thought to be associated with lung cancer.

## Data and methods

### Lung cancer in Scotland

An individual level dataset on lung cancer incidence was provided by the Information and Statistics Division (ISD) of NHS Scotland for the period 1988–1991. The records included the age of the patient at diagnosis, their sex and the census unit in which they lived. The date of diagnosis is the date of the first consultation or admission to hospital for the cancer (ISD, 2002). The census output-area (OA) identifier was also attached which enabled 1991 census data to be associated with each record. The OA is the smallest level of dissemination in the Scottish census and represents approximately 50 households (Dale, 1993). There were 18,632 new lung cancer patients identified in this period, of which 12,073 cases were male and 6559 female.

### Other explanatory variables

In an analysis of the relationship between lung cancer and radon, it is important to control for other important explanatory variables, particularly the age–structure of the population and smoking behaviour. Comprehensive and spatially disaggregated data on smoking are not generally available in Scotland. Therefore, age- and sex-specific estimates of smoking behaviour were calculated for all OAs across the country using a combination of individual-level data from the Scottish Household Survey and areal data from the 1991 Scottish Census. The Scottish Household Survey questioned 13,784 individuals aged over 15, distributed across 7127 of the 38,254 Scottish OAs, on a range of social issues including their smoking behaviour. These data were juxtaposed with a combination of OA and pseudo-postcode sector (PPS) data from the census to model smoking behaviour using a multilevel logit model. The parameters from this model were then used to predict smoking behaviour for all 38,254 OAs across Scotland. Further details on the methods used to estimate smoking behaviour in Scotland are provided elsewhere (Pearce et al., 2003).

A number of studies have suggested that deprivation has an independent effect on cancer incidence (Benach et al., 2001) and morbidity in general (Carstairs and Morris, 1991; Macintyre et al., 1993; Boyle et al., 1999). Therefore, the Carstairs Index of Deprivation was included as an explanatory variable at the PPS level. Furthermore, previous studies have identified a slight urban excess in lung cancer rates in Scotland, even when other risk factors have been controlled for (Pearce and Boyle, 2005). Therefore, the OA-level population density was included as an urban–rural proxy (Martin et al., 2000).

### *Radon potential*

The relationship between radon and lung cancer in Scotland has received little attention, despite some parts of Scotland having levels of radon that exceed national limits (Green et al., 1996). A total of 5700 radon measurements have been collected by the National Radiological Protection Board (NRPB) on behalf of the Scottish Executive. Fig. 1 shows the average radon concentration in postcode districts across Scotland. Low mean concentrations were found in southern and western Scotland and relatively high values in Aberdeenshire and the Highlands. There were no radon measurements taken in the main population centres of Glasgow and Edinburgh as these cities represent areas of low radon potential. Fig. 2 provides postcode sector averages for two areas of the Grampian and Highland regions where more measurements were collected. The highest values were in south-west Aberdeenshire and on the north-east coast of the Highlands. Unfortunately, the NRPB was unwilling to release the data at the household or small area level for our analysis.

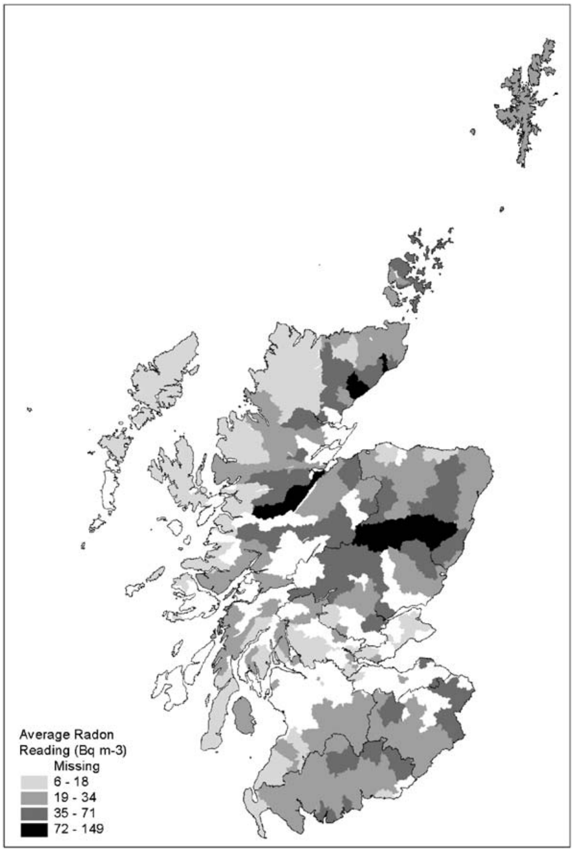

Fig. 1. Average radon reading in postcode districts in Scotland (NRPB data).

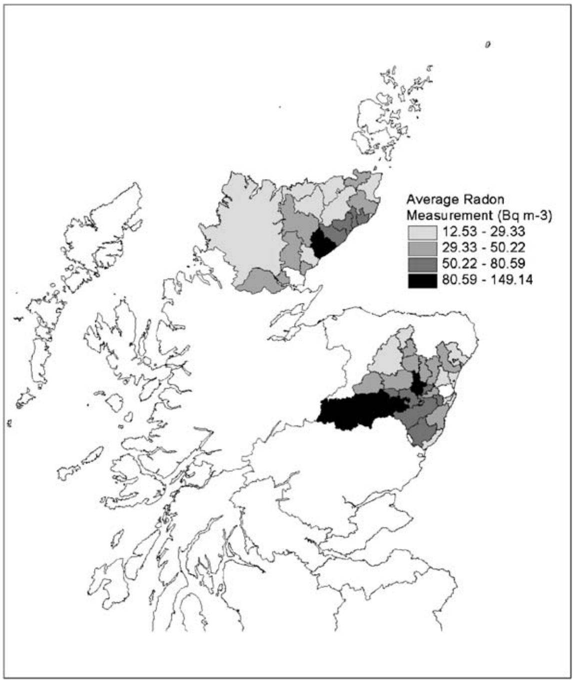

Fig. 2. Average radon levels in postcode sectors in parts of the Grampian and Highland regions (NRPB data).

In the absence of comprehensive and geographically specific radon data in Scotland, radon potential estimates that had been produced by the British Geological Survey (BGS) were used as proxies of radon exposure. Log-normal modelling was used by the BGS to estimate the percentage of dwellings that exceeded the UK action level for each geological unit of the 1:625,000 geological map of Scotland, using the radon measurements collected by the NRPB. This involved subtracting the average outdoor radon concentration from the measured indoor values, setting negative values to a small positive value, taking natural logarithms and calculating the mean and standard deviation for each geological unit. The emission of radon will be modified by the thickness, composition, source, permeability and wetness of any unconsolidated deposits that overlay the solid geology. The radon potential estimates were modified according to the nature of the overlying drift deposits (Table 1). It should be noted that the interaction between geology and drift can be more complex as, for example, glacial or river gravels may contain uranium or radium deposits that can contribute to indoor radon levels. Each combination of geology and drift was classified into one of five categories: low; low-moderate; moderate; high and very high unless there was a paucity of radon measurements to determine the radon potential, in which case the group was labelled unclassified (Appleton and Ball, 1995). These values

Table 1
Effect of unconsolidated deposits on radon potential

| Type and characteristics of unconsolidated deposits | Impact of unconsolidated deposits on radon potential class indicated by underlying rocks |
|---|---|
| Generally impermeable: peat and lacustrine clays | Reduces radon potential to low |
| Variable permeability: alluvium, boulder clay and morainic drift | Reduces, or has no significant effect, on radon potential, depending upon thickness, composition and derivation of unconsolidated deposit (no effect if radon potential is low) |
| Generally permeable: blown sand, river terrace, raised beach and marine, glacial sand and gravel | Increases or has no significant effect, on radon potential (normally no effect if radon potential is high or very high) |

were used to calculate the proportion of homes above the action level (200 Bq $m^{-3}$) for each section of the geological map (Miles, 1998). It has been estimated that approximately 2000 Scottish dwellings exceed the UK action level, although these are concentrated in particular parts of the country (Kendall et al., 1994).

The highest radon potential classes were located in the Grampian and Highland regions (Fig. 3), which corresponds with the NRPB postcode district maps in Figs. 1 and 2. Low radon potential levels were found in the Central Belt and most of the Borders region. The map reflects the limitations of the data, as localised or anomalous features may not be represented and the boundaries shown are approximate (Appleton and Ball, 1995).

Using Geographical Information Systems (GIS) the population-weighted centroid of each of the 38,254 OAs in Scotland was assigned to a radon potential class to provide an estimate of the radon potential to which the majority of the population within the OA are exposed.

### Modelling lung cancer incidence in Scotland

We used a Poisson regression model to examine whether radon influenced lung cancer incidence (1988–91), controlling for age, sex, smoking behaviour, the Carstairs Index of Deprivation and population density. For each OA, the population and lung cancer counts were calculated for 12 age-sex groups. The log of the age- and sex-specific population count was treated as an offset, effectively making this a model of rates (Knudsen, 1992). The age and sex groups were chosen to coincide with the age- and sex-specific smoking estimates (males and females aged 16–24, 25–34, 35–44, 45–54, 55–64 and 65 and over). Two categorical variables were included to define which age and sex group each record represented. Smoking probabilities could not be calculated for 347% or 0.9% of the OAs (Pearce et al., 2003) and these were excluded from the analysis, leaving a total of 37,907 OAs in Scotland. Because each OA was represented 12 times there were 454,884 records in the modelled data set.

Previous studies have noted that the age at which an individual is exposed to radon has an important influence upon their chances of developing lung cancer (Bijwaard et al., 2001). For example, it has been suggested that radon-induced lung cancer is approximately ten times higher for people exposed at the age of about 15 than at about 50 (Leenhouts, 1999). Therefore, in this study separate models of the younger (16–54; 1473 cases) and older (over 54; 16,929 cases) age groups were examined.

Each of the potential explanatory variables were tested individually, both in their raw and logged form, where applicable, and the more significant of the two was used in subsequent models. Interaction terms were also examined for all pairs of explanatory variables. The goodness of fit was tested by comparing the deviance (a measure of unexplained variation) with the critical value of chi-square (Lovett and Flowerdew, 1989).

## Results

Models 1–3 in Table 2 show the results for the younger age groups. The addition of the radon variable to the null model demonstrated that not all of the radon categories were significantly different to the base category (low radon potential). When the differences were significant, the parameter estimates were negative indicating that lung cancer was actually lower in areas with higher levels of radon. The parameter estimate of the very high radon potential category was positive but not significant in this model. Once age, sex, deprivation and smoking were controlled for, all of the radon categories had negative parameter estimates and were insignificant except for the very high radon category where the parameter estimate was positive and significant. The incidence of lung cancer was higher among the older members of this young sample, for males and in areas with higher deprivation scores and smoking rates. The interaction between sex and smoking was negative and significant which suggests that that the effect of age- and sex-specific smoking probabilities on lung cancer

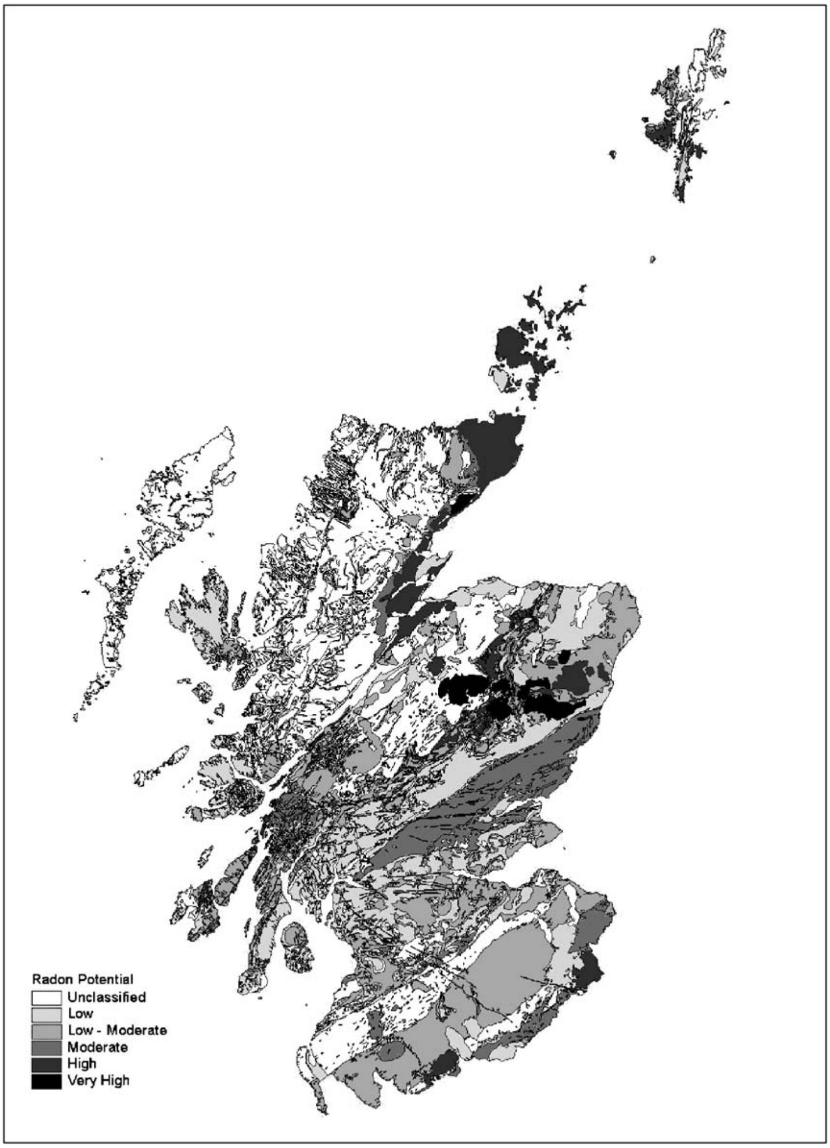

Fig. 3. Map of radon potential in Scotland.

incidence was less for women than it was for men. The population density variable was not significant.

The results for the older age groups (Table 3) show that lung cancer was less common in the moderate, high and very high categories compared to the base category (low), and the differences for these groups were significant (Model 2). The pattern was similar once age, sex, deprivation, smoking and population density were controlled for. The incidence of lung cancer was lower in each of the radon categories compared to the base category and all were significant except for the low-moderate and very high categories. The parameter estimates of the other variables followed the pattern of the model of young cases (Table 2), except that the population density variable was significant.

## Discussion

The relationship between lung cancer incidence and exposure to radon gas is not fully understood (Darby, 1999), particularly in Scotland where the health effects of radon have received little attention (Green et al., 1996). This analysis has examined the relationship between exposure to radon and lung cancer incidence in small areas across Scotland, which has allowed

Table 2
Poisson regression models of lung cancer incidence among those aged $<55$

| | Model 1 | | | Model 2 | | | Model 3 | | |
|---|---|---|---|---|---|---|---|---|---|
| Scaled deviance | 16,238 | | | 16,216 | | | 12,999 | | |
| Reduction in scaled deviance | | | | | | | | | |
| Degrees of freedom | 303,255 | | | 303,250 | | | 303,243 | | |
| | Parameter estimate | Std. error | *T*-value | Parameter estimate | Std. error | *T*-value | Parameter estimate | Std. error | *T*-Value |
| Intercept | −7.502 | 0.0260 | −288.54 | −7.447 | 0.0406 | −183.38 | −10.400 | 0.3630 | −28.65 |
| Age (25–34) | | | | | | | 1.503 | 0.3896 | 3.86 |
| Age (35–44) | | | | | | | 3.843 | 0.3494 | 11.00 |
| Age (45–54) | | | | | | | 5.562 | 0.3448 | 16.13 |
| Sex (female) | | | | | | | −0.844 | 0.1369 | −6.16 |
| Carstairs score | | | | | | | 0.042 | 0.0099 | 4.24 |
| Log smoking | | | | | | | 1.149 | 0.1060 | 10.84 |
| Radon (low-mod) | | | | −0.008 | 0.0564 | −0.15 | −0.037 | 0.0564 | −0.66 |
| Radon (mod) | | | | −0.279 | 0.1040 | −2.68 | −0.186 | 0.1049 | −1.77 |
| Radon (high) | | | | −0.335 | 0.1301 | −2.58 | −0.186 | 0.1317 | −1.41 |
| Radon (very high) | | | | 1.096 | 0.5788 | 1.89 | 1.457 | 0.5787 | 2.52 |
| Radon (unclassified) | | | | −0.375 | 0.1624 | −2.31 | −0.181 | 0.1641 | −1.10 |
| Log smoking.sex | | | | | | | −0.419 | 0.1340 | −3.13 |

Table 3
Poisson regression models of lung cancer incidence among those aged $>54$

| | Model 4 | | | Model 5 | | | Model 6 | | |
|---|---|---|---|---|---|---|---|---|---|
| Scaled deviance | 72,733 | | | 72,553 | | | 69,416 | | |
| Reduction in scaled deviance | | | | 180 | | | 3137 | | |
| Degrees of freedom | 151,627 | | | 151,622 | | | 151,616 | | |
| | Parameter estimate | Std. error | *T*-value | Parameter estimate | Std. error | *T*-value | Parameter estimate | Std. error | *T*-value |
| Intercept | −4.538 | 0.0077 | −589.35 | −4.494 | 0.0121 | −370.49 | −3.976 | 0.0649 | −61.27 |
| Age (65+) | | | | | | | 0.673 | 0.0229 | 29.37 |
| Sex (female) | | | | | | | −0.737 | 0.0516 | −14.29 |
| Carstairs score | | | | | | | 0.028 | 0.0031 | 8.86 |
| Log smoking | | | | | | | 0.702 | 0.0320 | 21.98 |
| Log population density | | | | | | | 0.016 | 0.0049 | 3.23 |
| Radon (low–mod) | | | | 0.003 | 0.0168 | 0.17 | −0.021 | 0.0168 | −1.27 |
| Radon (mod) | | | | −0.164 | 0.0281 | −5.83 | −0.085 | 0.0282 | −3.03 |
| Radon (high) | | | | −0.337 | 0.0392 | −8.59 | −0.223 | 0.0395 | −5.65 |
| Radon (very high) | | | | −0.723 | 0.3511 | −2.06 | −0.402 | 0.3505 | −1.15 |
| Radon (unclassified) | | | | −0.356 | 0.0455 | −7.81 | −0.131 | 0.0468 | −2.80 |
| Log smoking.sex | | | | | | | −0.233 | 0.0351 | −6.63 |

localised variations in the disease and associated risk factors to be considered. The geographically detailed radon potential estimates used in this study reflect the combination of solid geology and the overlying drift deposits and this improves upon previous ecological studies, which have tended to rely upon radon estimates for quite large and geologically heterogeneous administrative areas.

The results of the modelling procedure for both age groups demonstrate that age, sex, deprivation and smoking were all significantly related to the incidence of lung cancer between 1988 and 1991. In addition, population density was significant for older people. The parameter estimates were intuitively reasonable as lung cancer incidence was higher in older age groups, among males, in more deprived areas, in areas with higher smoking rates and higher population densities (for the elderly). These results confirm those of Pearce and Boyle (2004), although it appears that population density is only significant for those in older age groups. This is probably because exposures to all of the potential risk factors associated with the urban excess are likely to be greater among the older population.

The results suggest that, for the young, exposure to very high levels of radon may contribute to the premature development of lung cancer. This supports previous experimental and population-based studies which suggest that radon exposure at a young age has a greater influence on lung cancer incidence than exposure at an older age (Leenhouts, 1999; Langholz, et al., 1999). Radon is a risk factor that people can be exposed to at an early stage in life, whereas exposure to some of the other risk factors, such as smoking, tends not to commence until later in life.

However, it should be noted that in this study the average age of the lung cancer cases in the 'young' age group was 48.46. Also, we would expect a linear relationship between the radon potential measure and lung cancer incidence, but this did not occur. The rates in the 'high' radon potential areas were lower than those in the 'low' potential areas, although the difference was not significant.

The results presented here suggest that radon is not a cause of lung cancer in older age groups as incidence of the disease was highest in the lowest radon potential group. Again, this may be because the carcinogenic effects of radon exposure are age-dependent. Furthermore, the relative importance of radon exposure for older individuals may be minor compared to a long-term smoking habit (Leenhouts, 1999). Alternatively, the estimates of radon exposure may be compromised by the higher total lifetime migration of people in this age group when compared to the younger age group (Boyle et al., 1998).

There are a number of reasons why the results only provide some marginal support for the effect of radon on lung cancer. First, the simplest explanation is that radon levels are not an important factor in controlling lung cancer incidence in Scotland and this is supported by many ecological and individual studies that have found no link between radon and lung cancer in other areas (Etherington et al., 1996; Blot et al., 1990). Most studies, including this one, certainly suggest that other factors are more important than radon.

Second, the radon variable may be insignificant because of the paucity of radon measurements that have been taken in Scotland, resulting in a classification scheme that is too generalised and cannot account for local variations that exist due to small-scale geological differences such as contrasting lithology and permeability or unmapped shear and fracture zones. Clearly, the very localised variations in radon levels were impossible to measure in this study due to the complex interaction of factors that affect the radon reading. Note that the radon potential maps only represented the *potential* for having high levels of radon rather than being based on actual radon levels. Furthermore, the radon potential estimates are based upon the 1:625,000 geological map of Scotland and hence provide a generalised assessment of radon. Therefore, it is possible that there were some high radon readings in the low radon potential classes and vice versa, although such occurrences should have been relatively rare.

Third, the radon potential measures take no account of non-geological factors that may affect radon levels; the most obvious example would be differences in the degree of housing ventilation. In order to make a more confident assessment as to which of these factors is the more likely explanation, it would be necessary to undertake a comprehensive programme of radon measurement collection. This would allow for a more accurate set of models to be developed that better reflect more localised variations.

Fourth, data on the residential history of the lung cancer patients was not available and therefore it was not possible to associate historical information on radon exposure. Therefore, variations in exposure to radon at different places could not be accounted for. This could be important because the relationship between radon and lung cancer may be confounded by the movement of people between areas that have substantially different radon levels. This is a feature that is common to all ecological studies of radon exposure and lung cancer (Stidley and Samet, 1993). The assumption made here is that the radon levels experienced at the time of diagnosis will have influenced the probability of acquiring lung cancer, but some people will have moved prior to diagnosis. However, there is no evidence to suggest that migration during the study period should be selective in relation to radon and, therefore, we would not expect younger people with lung cancer to be more likely to migrate to areas with high levels of radon.

Despite these reservations, this research is the first to examine the relationship between lung cancer and radon in Scotland for small geographical areas. It is also the first ecological study to control for the effect of smoking in small areas. The study is consistent with a number of previous studies that have found that radon is a significant cause of lung cancer incidence but that age and smoking behaviour are by far the most important causes of the disease. At the very least, our results suggest there is a need to examine further the health effects of radon in Scotland among the young.

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