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Original Paper

An Ecological Study of Cancer Incidence and Radon Levels in South West England

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To investigate the relationship of domestic radon levels and cancer, the incidence of 14 major cancers in Devon and Cornwall were examined in relation to the local radon levels. Cancer registrations for 1989–1992 were provided by the South-Western Regional Cancer Registry. The average radon levels for postcode sectors were sorted into ten categories from low ($<40 \text{ Bq/m}^3$) to extremely high ($\geq 230 \text{ Bq/m}^3$) and age-standardised incidence rates were calculated for each radon category. The incidence rates for lung cancer, where radon has been claimed to be a risk factor, were very similar across all domestic radon categories. Only non-melanoma skin cancers, showed a significant increase in incidence in the high-radon postcode sectors ($\geq 100 \text{ Bq/m}^3$) compared with the low-radon sectors ($<60 \text{ Bq/m}^3$) and this effect was observed for both sexes. The remaining 12 cancer sites showed no significant trend in incidence rates with increasing radon concentration. There was no significant difference in corrected survival rates for any cancer site between the low- and high-radon areas. The possible contribution of confounding factors to the results of this study is discussed. Copyright © 1996 Elsevier Science Ltd

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INTRODUCTION

RADON is a naturally occurring radioactive gas emitted from the earth surface. The daughter breakdown products of radon are α particles with short half-lives [1] and have been implicated as causative agents for a number of cancers, especially those of the lung. Numerous studies have been undertaken to assess the extent of radon-induced lung cancer in miners and others in the population [2–4]. Elevated domestic radon levels have also been linked to the development of acute myeloid leukaemia [5–7], skin [8, 9] and prostatic [10] cancers. Alpha particles from radon can penetrate only a short distance into cells and may cause changes at topical sites. Radon can also be absorbed since it is water soluble, but as it is known to be much more soluble in the lipid components of tissues, where its decay products may promote damage to DNA at deeper cell locations in the body such as the haemopoietic tissue in bone marrow [1, 11–13]. Chromosomal damage has also been observed in blood lymphocytes of people with long-term exposure to high domestic levels of radon [14].

The data for radon-induced cancers are not conclusive.

Lung cancer in miners has been linked to exposure to high radon levels underground. However, a number of studies have now failed to reveal any significant link between elevated domestic radon levels and the incidence of various cancers, including lung [3, 15–18]. Difficulties in the interpretation of ecological studies for lung cancer have been reviewed [19]. In part, the problem of assessing the risk from domestic radon is in identifying the actual levels of exposure. The airborne levels are normally low, but radon emissions can accumulate in houses, and exposure to residents within the home depends on a number of factors, such as structure, siting of bedrooms and ventilation, and these have been examined in detail. Due to individual domestic lifestyles, radon levels can also vary with the season and there may be large differences between neighbouring houses in the same street [1].

Many analyses of the risk from domestic radon have examined large geographical areas such as counties or even countries, relying on published data for the average domestic levels in these areas, which dilutes the input from any local clusters. In South West England, high emission levels of radon are associated with the igneous granitic rocks of Devon and Cornwall where the gas can accumulate in poorly ventilated dwellings and 12% of homes in these counties have been

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found to have levels above 200 Bq m^{-3} . Due to the perceived risk of cancer, this is the action level set by the National Radiological Protection Board for sealing the basement area and improving the ventilation of homes [1]. This study was undertaken using cancer registry data to identify any variation in incidence and survival rates across these two counties associated with radon, based on the average emission values published for the smaller areas defined by postcode sectors.

MATERIALS AND METHODS

The tumour records in the South-Western Cancer Registry have been postcoded routinely since 1985 and these are validated from the regional address file. The postal areas of Exeter, Plymouth, Torquay and Truro cover all of Cornwall and almost all of Devon. All cancer records from 1989 to 1992 were extracted by postcode sector for these areas and sorted by domestic radon emission level into ten categories of approximately equal numbers of residents, using the published table for average levels in these postcode sectors [20].

The domestic radon concentration limits (Bq m^{-3}) in the ten categories were allocated as follows: (1) 0–39, (2) 40–44, (3) 45–49, (4) 50–59, (5) 60–74, (6) 75–99, (7) 100–129, (8) 130–159, (9) 160–229, (10) ≥ 230 . The small urban areas of Penzance and Falmouth were in high-radon categories, whereas the larger urban areas of Exeter and Plymouth were both in lower-radon categories. The 1991 population estimates for postcode sectors from OPCS [21] show that the total population covered was 1.47 million with the population in the ten radon categories ranging from 110–200000 and an average of 148000.

Accurate population data by ageband are available only for enumeration districts (EDs), which are smaller (about 300–500 persons), but not coterminous with postcode sectors (about 3000–10000 persons). The ED population data from the 1991 census were supplied by the Information Unit of the Regional Health Authority. Pseudo-EDs, which identify those EDs having the best fit for individual postcodes, were collated for each postcode sector. The full population profile for each radon category was then calculated from the aggregated postcode sector populations.

The cancer records for 14 key sites were extracted and sorted by the 3-digit ICD9 code (4-digit codes for *in situ* breast and cervical cancers) and radon category. The key sites selected were: 150 (oesophagus), 151 (stomach), 153 (colon), 154 (rectum), 157 (pancreas), 162 (lung), 172 (melanoma), 173 (other skin), 174/2330 (breast), 180/2331 (cervix uteri), 182 (corpus uteri), 183 (ovary), 185 (prostate) and 188 (bladder). Counts for *in situ* breast (ICD 2330) and cervical (ICD 2331) cancers were added to the respective invasive tumour counts as the screening programmes have facilitated earlier diagnosis at these two sites. Initial checks were made to determine the percentage of cases occurring in the population for each of the ten radon categories, the average age at diagnosis for each cancer and the incidence of multiple cancers. Three cancer sites had been claimed in past studies to be linked to radon emissions (lung, prostate, skin) and the others were selected as they are major cancers, suitable for a broadly based ecological study.

Standard procedures were employed for data analysis [22]. Age-standardised incidence rates (ASRs) with 95% confidence intervals were calculated for all key cancer sites in each radon category, using the combined Devon and Cornwall population as the standard. Corrected survival rates were also

calculated for the 14 sites. The statistical facilities of Microsoft Excel were used for the calculation of confidence intervals, and SAS [23] was used for the determination of correlation coefficients.

Social indicators were examined to evaluate any possible systematic differences between the ten area categories for radon emissions. The Carstairs index of social deprivation [24] for EDs in the South-Western region was supplied by Dr Paul Elliott of the Small Areas Health Statistics Unit, London. The EDs in each radon category were sorted by the Carstairs quintile value and the distributions plotted. The average Carstairs score for each category was calculated, after weighting the individual ED scores according to number of households present.

RESULTS

A total of 28989 cancers were registered for the 14 key sites and diagnosed in the years 1989–1992 for residents living in the postcode areas EX, PL, TQ and TR, which cover almost all of Devon and Cornwall. Counts of the tumour records by ICD code and radon category are shown in Table 1, together with the resident population figures in each category. Initial examination of the percentage counts in each category showed that only other skin cancer (ICD 173) was increased in the high-radon categories. The average age at registration and number of multiple tumours identified failed to indicate any differences across the range of domestic radon concentrations (data not shown).

The ASRs for the 14 key cancer sites are shown in Figure 1. There was some variation across the ten radon categories, but these were not consistent nor significant except for other skin cancer. This cancer (ICD 173) showed a significant increase for both sexes in the high-radon categories. However, there was a slight trough in the incidence rate where the domestic radon level was slightly raised at 50–74 Bq m^{-3} and, against the lowest radon category, the ASR was significantly reduced in females. The ASR of lung cancer (ICD 162) was essentially unchanged across the range of domestic radon levels indicating no overall effect on the resident population from elevated domestic radon emissions. These data are summarised in Table 2.

Due to the non-normality and ranking of the radon variable, the determination of correlation coefficients for these ASRs was undertaken using Spearman's rank correlation test. The correlation coefficients and probability values are shown in Table 3. Although a number of correlations were individually significant, the *P* values had to be adjusted because the chance of at least one correlation being significant at $P=0.05$ increased with the number of tests done. Hence, predominantly non-significant correlations were found in these plots between radon and incidence. Only the correlation between radon and malignant melanoma for females approached significance, so there was slight evidence to suggest that high ranks in radon levels corresponded to high ranks in the ASRs for women with malignant melanoma for the data observed.

Corrected survival percentages were determined for each key site and sex in the low- and high-radon areas, using combined data from the two radon categories at each end of the range. Only registrations for the three earlier years, 1989–1991, were analysed and the three-year corrected survival percentages are shown in Table 4. None of these data showed any significant differences between the low and high domestic emission levels.

Table 1. Tumour registrations 1989–1992 for Devon and Cornwall. Key sites segregated for counting in ten categories determined by the domestic radon level (average for postcode sector). The resident population in each radon category is shown

Domestic radon levels (average values for postcode sectors)											
Radon category (Bq/m³)	1 (0–39)	2 (40–44)	3 (45–49)	4 (50–59)	5 (60–74)	6 (75–99)	7 (100–129)	8 (130–159)	9 (160–229)	10 (≥ 230)	
Resident population (thousands)											
Total males	67.2	61.0	67.1	84.0	95.9	87.0	57.5	79.4	55.8	52.6	
Total females	74.1	65.4	73.4	92.7	104.1	93.9	62.9	86.0	59.1	57.0	
ICD9/Site	Sex	Counts									
150	M	39	25	41	52	58	34	39	48	28	29
Oesophagus	F	28	28	31	44	31	32	33	35	20	21
151	M	61	64	63	62	108	79	59	67	45	42
Stomach	F	36	30	34	54	43	37	33	34	28	30
153	M	99	100	105	152	148	144	112	145	71	78
Colon	F	134	122	134	190	187	173	117	162	108	126
154	M	68	79	77	98	109	84	69	83	54	45
Rectum	F	86	64	64	105	88	81	54	92	57	61
157	M	37	27	38	51	48	43	33	33	19	24
Pancreas	F	36	27	27	54	36	48	29	39	31	19
162	M	246	246	266	321	327	278	224	298	206	208
Lung	F	112	110	133	186	157	157	112	116	89	98
172	M	20	31	26	27	48	51	38	37	35	19
Melanoma	F	39	35	40	51	63	71	45	62	44	44
173	M	283	262	263	319	358	427	399	512	337	394
Other skin	F	283	215	223	262	240	342	332	451	283	346
174/2330	M	–	–	–	–	–	–	–	–	–	–
Breast	F	406	316	407	539	557	494	375	460	330	314
180/2331	M	–	–	–	–	–	–	–	–	–	–
Cervix uteri	F	288	247	273	308	351	282	184	249	194	224
182	M	–	–	–	–	–	–	–	–	–	–
Corpus uteri	F	48	43	58	76	73	65	51	59	38	46
183	M	–	–	–	–	–	–	–	–	–	–
Ovary	F	55	46	51	76	84	67	66	79	47	39
185	M	168	161	164	227	270	215	180	248	158	156
Prostate	F	–	–	–	–	–	–	–	–	–	–
188	M	93	83	78	117	134	100	79	111	91	56
Bladder	F	41	29	27	46	46	40	25	47	31	27

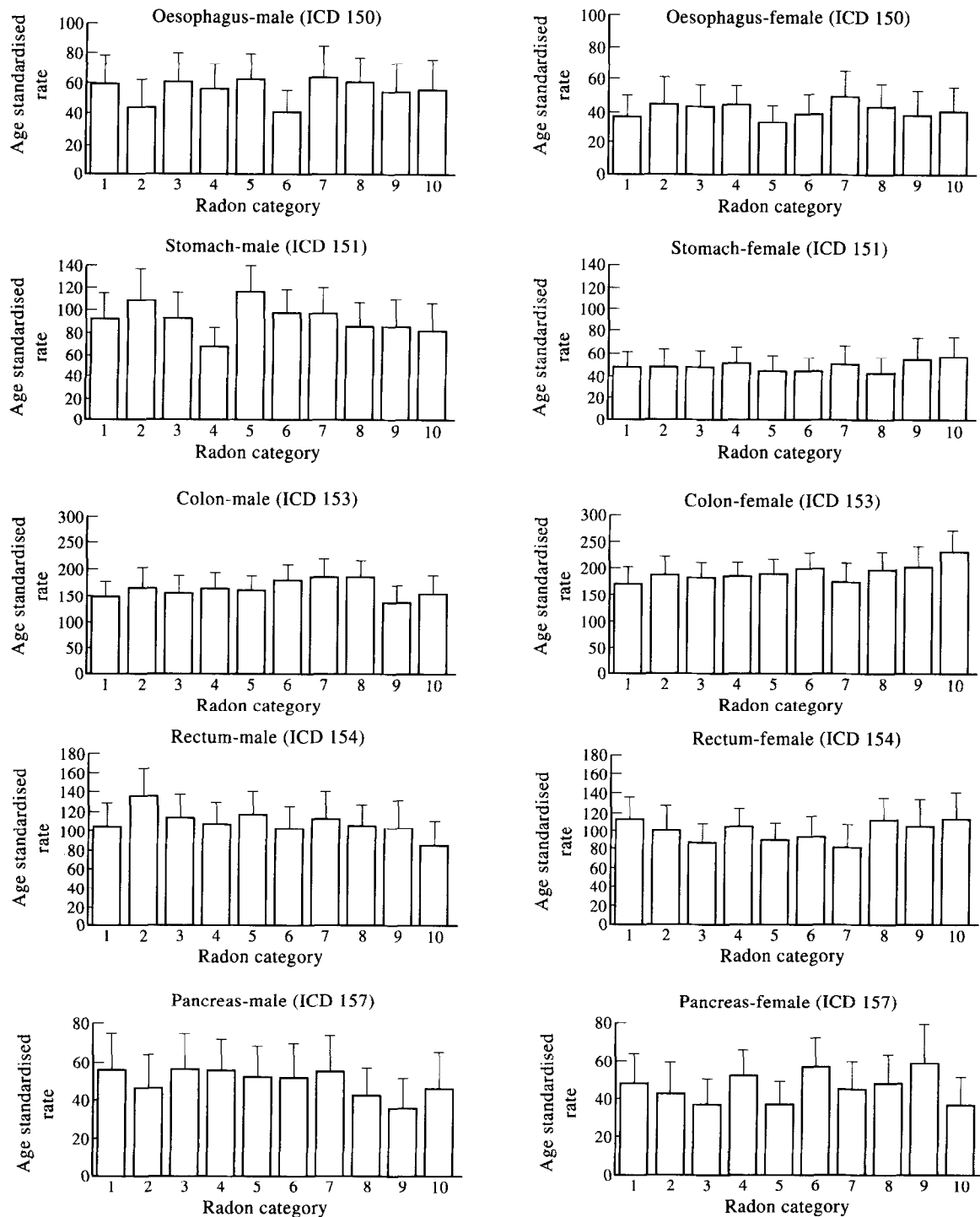
Of the confounding factors in this study, any widespread differences in social status are likely to contribute to possible changes in incidence rates across the ten categories for the domestic radon emissions. The national quintile value for the Carstairs index was applied to each ED and the distribution of Carstairs quintile values by EDs is shown for the whole area and the ten sub-areas in Figure 2. Each domestic radon category was represented by EDs from all quintiles and there was little visual evidence that these histograms showed any difference in socio-economic status between the categories with the exception of the 5th quintile (i.e. the most deprived) which was reduced in the high-radon categories. Superficially, socio-economic status appears to be related to radon level, but this simply reflects the fact that the deprived inner-city EDs are low-radon areas because the larger urban areas are situated on the alluvial plains of river estuaries.

The average Carstairs score was calculated for each radon category after weighting the individual ED scores by the number of households that are present. These average scores revealed only small differences, with those living in the middle

of the radon emission range being slightly more affluent. For the sixth category, 75–99 Bq/m³, the score moved into the second quintile whereas all other category average scores fell within the range of the Carstairs third quintile.

DISCUSSION

There is now substantial evidence to link radon to the development of lung cancer in miners who are exposed to high concentrations of this gas, particularly in uranium mines. In contrast to this, the evidence for domestic radon being a risk factor for cancer is controversial [19]. The counties of Devon and Cornwall in the South-Western peninsula of England are rich in granitic rocks, emitting large amounts of radon which can accumulate in poorly ventilated houses. Tables published by the National Radiological Protection Board [20] provide estimates of the domestic radon concentration for each postcode sector from a number of random measurements at selected indoor sites in each sector. This study has used these data to examine the incidence of 14 key site-specific cancers in relation to domestic radon emissions and to test if elevated

Figure 1. *Continued opposite*

emissions can be shown to be a risk factor. For ease and convenience of analysis, postcode sectors were sorted into ten categories according to the average domestic radon estimate, such that the population numbers in each category did not differ widely.

The years covered were 1989–1992 and a total of over 28900 tumours were counted for these key sites in a resident population of nearly 1.5 million. Initial checks showed that overall the crude incidence rates for cancer sites in each radon category were similar except for non-melanoma skin cancers

where the rate was elevated in the high-radon sectors. There appeared to be no apparent change in the average age at diagnosis, nor in the incidence of second tumours and no further studies were made of these parameters.

The ASRs for each cancer site and sex in each of the radon categories confirmed the increased rate only for non-melanoma skin cancer (ICD 173) in the four higher radon categories (≥ 100 Bq/m³). That this was statistically significant for both sexes is shown by the 95% confidence limits in Figure 1. However, there was a distinct decline in those post-

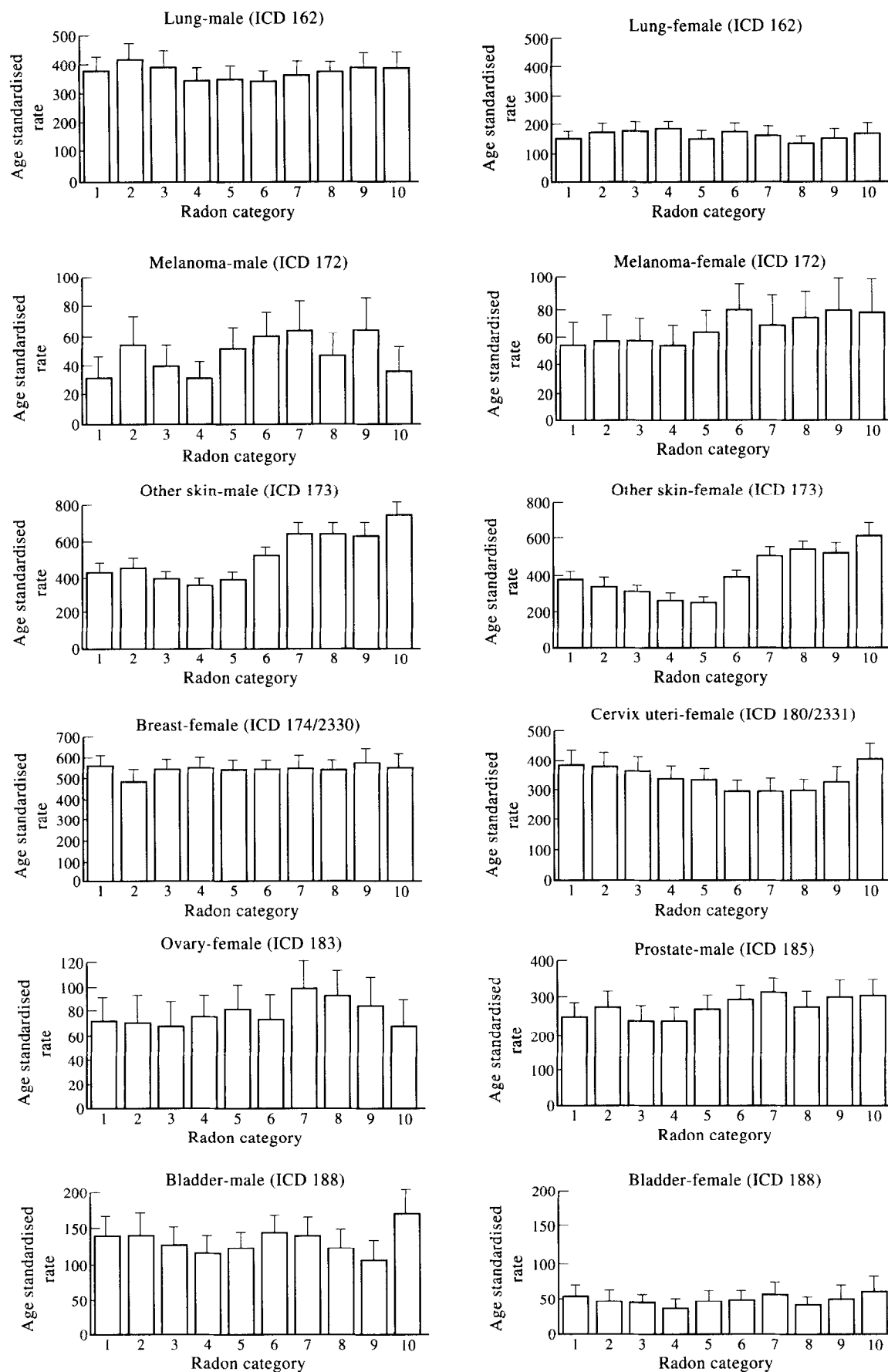


Figure 1. Age-standardised incidence rates, with confidence limits, for 14 key cancer sites registered 1988–1992 in Devon and Cornwall and sorted according to the published domestic radon levels. The radon categories (Bq/m^3) were: (1) 0–39, (2) 40–44, (3) 45–49, (4) 50–59, (5) 60–74, (6) 75–99, (7) 100–129, (8) 130–159, (9) 160–229, (10) ≥ 230 .

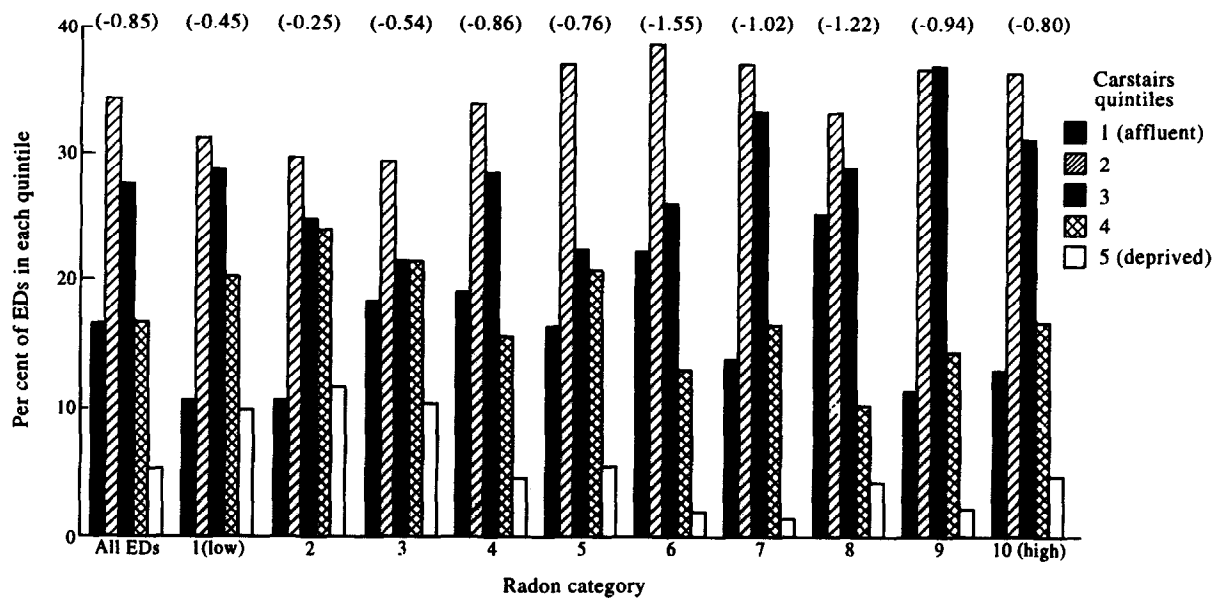


Figure 2. Distribution of Carstairs deprivation score quintiles for EDs in the ten radon category areas with the average score (weighted) for each category shown in parentheses.

Table 2. Tumour registrations 1989–1992 for Devon and Cornwall. Summary of age-standardised rates per 100000 population for the key sites are shown by radon category

Radon category		1 (low)	2	3	4	5	6	7	8	9	10 (high)
ICD9/Site	Sex	Age-standardised rates									
150	M	59	44	61	57	63	41	63	60	53	55
Oesophagus	F	35	43	41	42	31	37	49	42	37	39
151	M	92	109	93	67	116	97	96	85	84	79
Stomach	F	46	46	45	51	43	42	49	41	53	55
153	M	148	169	155	166	160	177	182	182	134	148
Colon	F	171	187	179	185	188	196	173	196	199	227
154	M	104	135	113	107	118	102	112	103	101	84
Rectum	F	112	100	85	103	88	92	80	109	103	109
157	M	56	46	56	55	52	52	53	41	35	45
Pancreas	F	47	42	36	51	36	55	43	47	57	34
162	M	375	418	390	349	354	341	365	372	389	389
Lung	F	148	169	178	185	158	178	167	137	159	174
172	M	31	54	38	31	51	59	63	46	64	35
Melanoma	F	52	55	54	52	62	77	68	72	77	76
173	M	430	446	387	349	386	520	645	639	629	742
Other skin	F	372	333	300	259	240	382	495	538	514	616
174/2330	M	—	—	—	—	—	—	—	—	—	—
Breast	F	571	493	544	555	545	544	554	534	578	550
180/2331	M	—	—	—	—	—	—	—	—	—	—
Cervix uteri	F	384	378	367	339	333	296	297	294	329	404
182	M	—	—	—	—	—	—	—	—	—	—
Corpus uteri	F	64	67	79	75	72	72	76	69	66	79
183	M	—	—	—	—	—	—	—	—	—	—
Ovary	F	72	71	69	77	83	75	98	93	84	68
185	M	247	274	239	239	293	268	272	312	301	299
Prostate	F	—	—	—	—	—	—	—	—	—	—
188	M	140	141	115	128	144	122	121	140	170	105
Bladder	F	53	45	36	44	46	45	37	56	58	49

Table 3. Correlation coefficients and probability values for age-standardised incidence rates across the ten domestic radon categories for 14 key cancer sites. Data from 1989–1992 registrations for Devon and Cornwall

ICD9/Site	Sex	Correlation coefficient	Probability (*<0.01)
150	M	-0.0545	0.881
Oesophagus	F	0.0303	0.9338
151	M	-0.3818	0.2763
Stomach	F	0.2848	0.425
153	M	-0.0545	0.881
Colon	F	0.7576	0.0111
154	M	-0.6970	0.0251
Rectum	F	0.0424	0.9074
157	M	-0.7091	0.0217
Pancreas	F	0.0303	0.9338
162	M	-0.1151	0.7514
Lung	F	-0.1151	0.7514
172	M	0.3697	0.2931
Melanoma	F	0.7818	0.0075*
173	M	0.7333	0.0158
Other skin	F	0.7454	0.0133
174/2330	M	—	—
Breast	F	0.1634	0.6515
180/2331	M	—	—
Cervix uteri	F	-0.9000	0.0374
182	M	—	—
Corpus uteri	F	0.3091	0.3848
183	M	—	—
Ovary	F	0.3212	0.3655
185	M	0.7091	0.0217
Prostate	F	—	—
188	M	-0.1879	0.6032
Bladder	F	0.4182	0.2291

code sectors where the radon levels were slightly raised and the reasons for this are unclear. There was perhaps some evidence to imply an association between radon levels and melanoma in females for this data (Table 3). This, together with non-melanoma skin cancer, were the only cancers identified in this study where there appeared to be an association with domestic radon emissions. As a reference point in making comparisons, the national average domestic radon level is 20 Bq/m³, which is centrally located within the limits of the first radon category of this study.

Other workers [8, 9, 25] have also found an association between the strength of radon emissions and non-melanoma skin cancers. Eatough and coworkers [9] have calculated the theoretical dose of radiation received at the basal layer of skin as being 2.5 mSv/y, for the population average radon concentration (20 Bq/m³), which provides a basis for the calculation of radiation doses in those areas where radon emissions are high, assuming a linear relationship.

Regression analysis of the incidence rates for all key cancers by individual postcode sectors was impracticable as the number of cases of a specific cancer in any postcode sector was too small. In addition, since the radon value was an approximation from random sampling, specific levels within individual EDs, from which the population data were aggregated, cannot be assumed. Instead, Spearman's rank correlation test was applied, although, with only ten pairs of observations, the results should be interpreted with caution. The calculated correlation coefficients generally showed only weak associations.

For cancer sites other than skin, several investigations in

South-West England have failed to demonstrate elevated domestic radon levels as a risk factor. Bowie and coworkers [16] have previously examined the evidence for lung cancer in Devon and Cornwall and were unable to identify a clear link with the domestic levels of radon. We have also shown that the ASRs for lung cancer remain unchanged across the ten bands for domestic radon activity. In another study, Foreman and coworkers [26] failed to find any elevation for childhood cancers in Devon and Cornwall compared with the remaining, low-radon counties in South-West England and also in the West Midlands where the levels are generally very low.

Ecological studies of cancer as undertaken here do not reveal the nature or extent of any confounding factors which may influence the incidence rates. In particular, in South-West England, levels of radon emission are inversely correlated with levels of urbanisation, which themselves are associated with variations in socio-economic conditions and exposure to risk factors. However, analysis of the socio-economic status of families at the ED level, according to the Carstairs index of social deprivation, failed to indicate any association with domestic radon levels except with regard to the distribution of the fifth quintile, which only accounted for approximately 5% of all the EDs and 6% of all the households in the survey. Preliminary studies by us have found a small increase in the ASRs for several tumour sites with Carstairs quintile (unpublished data). Whether socio-economic condition *per se* is a confounder in this study cannot be excluded. To obtain conclusive evidence would require analysis of Carstairs scores for the individual family backgrounds in respect of the cases identified from the registry database.

Table 4. Corrected survival values for cases of invasive carcinoma living in areas with low and high domestic radon levels (average for postcode sectors). The counts for 1989–1991 registrations and 3 year survival data are shown for all key sites

ICD9/site	Sex	Low radon (<45 Bq/m ³)		High radon (>159 Bq/m ³)	
		Cases at start	% survival	Cases at start	% survival
150	M	56	16.5	42	16.8
Oesophagus	F	46	10.7	34	8.3
151	M	97	26.4	64	23.3
Stomach	F	51	19.1	44	8.7
153	M	134	43.6	93	54.8
Colon	F	170	50.2	154	59.2
154	M	100	53.4	67	50.0
Rectum	F	98	47.5	76	49.0
157	M	48	7.8	28	8.2
Pancreas	F	39	11.0	39	8.8
162	M	330	8.7	270	11.3
Lung	F	145	11.1	108	16.4
172	M	39	73.8	33	84.1
Melanoma	F	51	93.9	56	92.8
173	M	427	98.5	549	98.7
Other skin	F	389	97.9	468	99.1
174 (excl. 2330)	M	—	—	—	—
Breast	F	460	77.6	423	79.6
180 (excl. 2331)	M	—	—	—	—
Cervix uteri	F	73	74.9	53	62.6
182	M	—	—	—	—
Corpus uteri	F	66	76.6	59	82.7
184	M	—	—	—	—
Ovary	F	85	28.9	67	28.4
185	M	261	57.0	246	67.4
Prostate	F	—	—	—	—
188	M	152	73.8	116	77.2
Bladder	F	53	55.7	47	71.4

Other likely confounding factors are variations in the average daily exposure of the individual cases, and the duration of exposure. Exposure to radon is clearly dependent on lifestyle, place of work and other factors. Also, there is considerable migration within the population both across these counties and with other parts of the U.K., so that identifying the area of residence at the time of diagnosis may be quite misleading in terms of exposure to domestic radon. Consequently, the likely effect of such confounding factors would be to dilute any associations rather than create false positive ones. Therefore, the main purpose of a study of this nature is to identify areas of possible association for further study, rather than to obtain definite proof of causal associations.

The association between cancer and domestic radon levels is controversial [19] and several papers have reported data to show radon as a risk factor especially for lung cancer. The additional risk of developing lung cancer has been estimated [27, 28] and indicates that radon is more frequently a cause of cancer than other forms of radiation. The increased risks of developing acute myeloid or lymphoblastic leukaemias have also been calculated [29]. However, the data obtained in this population study have not revealed any significant positive correlation between the incidence of cancer and radon emissions except for the non-melanoma skin cancers, and suggest that the theoretical increased risks are of little significance at a population level where overall risk levels are attributable to complex interactions of a number of factors. Further studies are required to reconcile our data with that published elsewhere for this region of England. For many years, there have

been strong environmental pressures to provide protection from the accumulation of radon in homes, but from our data it appears that in recent years these risks may have been exaggerated. The recent case control study of domestic radon and lung cancer in Winnipeg [3], which failed to show any increased risk, and other reports of negative findings [30] have reinforced the view that if radon is a significant risk to the development of certain cancers then confounding factors can seriously distort the effects from domestic or industrial accumulations of the gas, both by masking positive associations and by producing apparently spurious negative ones.

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