Failure to identify association between deprivation and incidence of lung cancer

Surprising

Battersby et al present a method of performing equity audit where data on incidence, deprivation, and surgical resection rates of non-small cell lung cancer are compared. Deprivation was measured using the Index of Multiple Deprivation (IMD) 2000 and all analyses were performed at the primary care trust (PCT) level. Battersby et al report no statistically significant associations between their measure of deprivation and age and sex standardised incidence of non-small cell lung cancer. This is highly unusual and in contrast with findings from a large number of different populations. Without clear evidence that there is something exceptional about the population studied by Battersby et al, the lack of association between deprivation and incidence of lung cancer is likely to be an artefact.

Two possible explanations of Battersby et al’s failure to find an association between deprivation and incidence of lung cancer are possible. Firstly, calculating deprivation at the PCT level may be highly inaccurate. The IMD 2000 is a ward level variable and the PCT level may be highly inaccurate. The construction of an IMD score for PCTs is an appropriate way of including aggregation populations requires population weighting. This is an accepted methodology fore, the construction of an IMD score for PCTs is highly inaccurate. However, we would dispute the suggestion that calculating deprivation at primary care trust (PCT) level is inaccurate. It seems more probable that this lack of statistically significant association can be explained by the fact that the range of deprivation scores across PCTs in the east of England is comparatively narrow. IMD 2000 population weighted average scores vary from 7.1 to 38.0 across PCTs in Norfolk, Suffolk, and Cambridgeshire. This compares with a much wider range of 4.4 to 61.3 across local authorities in England. A further weakening of the association may also be attributable to the exclusion of small cell lung cancers (about 13% of all lung cancers), which are almost exclusively attributable to smoking. However, this effect is likely to be small.

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References


Authors’ reply

Dr Adams is correct in her assertion that the lack of statistically significant association between the Index of Multiple Deprivation 2000 (IMD 2000) score and the incidence of non-small cell lung cancer is unusual. However, we would dispute the suggestion that calculating deprivation at primary care trust (PCT) level is inaccurate.

We suggest that the use of population weighted average scores is an appropriate method of calculating PCT deprivation scores. The IMD is a proportion and, therefore, the construction of an IMD score for aggregated populations requires population weighting. This is an accepted methodology and is widely applied.

We did report that crude incidence of non-small lung cancer correlated with the IMD 2000 score. Moreover, when looking at mortality from all lung cancers during a similar period, there was good correlation between IMD 2000 score and age specific death rates for lung cancer, both for men and women. It was only the relation between IMD 2000 score and the age-sex standardised incidence of non-small cell lung cancer that, although present, did not reach statistical significance.

It seems more probable that this lack of statistically significant association can be explained by the fact that the range of deprivation scores across PCTs in the east of England is comparatively narrow. IMD 2000 population weighted average scores vary from 7.1 to 38.0 across PCTs in Norfolk, Suffolk, and Cambridgeshire. This compares with a much wider range of 4.4 to 61.3 across local authorities in England. A further weakening of the association may also be attributable to the exclusion of small cell lung cancers (about 13% of all lung cancers), which are almost exclusively attributable to smoking. However, this effect is likely to be small.
The question on bowel symptoms included a range of somatic conditions in the study, and trained interviewers asked subjects about a variety of conditions at baseline and at T2. Subjects were then diagnosed using the composite international diagnostic interview (CIDI), yielding DSM-III-R diagnoses. At baseline and at T2, trained interviewers asked subjects about a range of somatic conditions in the past 12 months. The question on bowel disease was: “Did you suffer, in the past 12 months, from severe bowel disease for a period of longer than three months?”

There were 3428 people with complete data on depression and somatic conditions, including cancer, at both baseline and T2. At baseline, 88 people (2.6%) reported “severe bowel disease” (SBD), for which 76% received medical treatment, and 525 (15.3%) reported a history of depression according to CIDI interview. There was significant reciprocal influence of SBD and depression at baseline (OR = 2.3, 95% CI: 1.4 to 3.6) (table 1).

After three years, when assessments were repeated, the incidences for SBD and depression were 1.5%, and 4.0% respectively. A baseline composite variable was made indicating presence of any of 29 somatic illnesses other than SBD. Cases with cancer were excluded from the SBD case definition. People with depression who did not have a history of SBD at baseline had a greater risk than those without depression of reporting SBD at follow up, independent of other baseline somatic illnesses, medication use, age, class, sex, and depression at follow up (logistic regression adjusted OR = 2.0, 95% CI: 1.01 to 3.8). Similarly, people with SBD at baseline who were free of depression had a greater risk than those without SBD of having depression at follow up, independent of any other baseline somatic illness at baseline, age, class, and sex (adjusted OR: 2.5, 95% CI: 1.1 to 5.8).

The measures of SBD were not precise, and probably included not only people with IBD but also people with severe irritable bowel syndrome (IBS). However, IBD and IBS themselves are associated with each other over time and therefore the bidirectional gut-brain-relations shown in this study are compatible with the hypothesis that severe non-cancerous bowel disease and depressive illness have reciprocal aetiological influences or, more parsimoniously, are varying expressions of a partly shared aetiological process. In support of the latter hypothesis is recent work showing that IBD is associated with familial transmission of an endophenotype characterised by subclinical inflammation that may predispose to no only IBD, but also to depression.

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References

Table 1 Prospective associations between depression and severe bowel disease

<table>
<thead>
<tr>
<th>SBD at baseline in those without depression (n = 63)</th>
<th>No SBD at baseline in those without depression (n = 2840)</th>
<th>OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rate depression at follow up (n, %)</td>
<td>Depression at baseline</td>
<td>No depression at baseline</td>
</tr>
<tr>
<td>Rate SBD at follow up (n, %)</td>
<td>7 (11.1)</td>
<td>108 (3.8)</td>
</tr>
<tr>
<td>14 (2.8)</td>
<td>35 (1.2)</td>
<td>OR (95% CI) Adjusted* OR (95% CI)</td>
</tr>
<tr>
<td>2.3 (1.2 to 4.3)</td>
<td>2.0 (1.01 to 3.8)</td>
<td></td>
</tr>
</tbody>
</table>

*Adjusted for baseline somatic illness, age, class, and sex.
†Adjusted for baseline somatic illnesses, medication use, age, class, sex, and depression at follow up.
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J Adams

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