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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.[1] Deprivation was measured using the Index of Multiple Deprivation (IMD) 2000[2] 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.[3–9] 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 authors of this study use a weighted average to determine IMD 2000 scores for PCTs. However, with an average of almost 22 wards per PCT in England and an average PCT population of over 128 000 in the study, it is not clear that ''the use of a weighted average IMD score is an appropriate way of including deprivation in the analysis''.[1] Secondly, with only 17 data points, it is probable that Battersby *et al*'s study lacks sufficient power to detect any but the strongest of associations.

Although the focus of Battersby *et al*'s article is primarily on a methodological technique, rather than on the particulars of the example data used, the failure to detect an association between deprivation and incidence of lung cancer suggests some problem with this technique. Furthermore, presentation of these results to non-specialised audiences, as is intended, may give the impression that deprivation is not an important determinant of incidence of lung cancer—which the majority of other evidence suggests it is.

Although public health practice requires methods that are quick, easy, and cheap to perform, this should not be at the expense of accuracy to such a degree that important, strong, and known associations are overlooked.

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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.[1]

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 $r = 0.76$ (95% CI 0.44 to 0.91), and women $r = 0.59$ (95% CI 0.15 to 0.83). 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.[2]

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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Bidirectional associations between depression/anxiety and bowel disease in a population based cohort

Kurina and colleagues[1] reported that neurotic disorders preceded inflammatory bowel disease (IBD), adding credence to the suggestion that depression/anxiety may either play an aetiological part in the onset of IBD or that both conditions share a common abnormality.[2] As their findings were based on record linkage of people with neurotic disorder who received treatment in mental health services, representing only a small minority of such cases, replication independent of mental health service treatment setting is necessary to exclude the well known bias attributable to selection of the most severely ill with the highest comorbidity rates.

This analysis is a part of the Netherlands mental health survey and incidence study

Table 1 Prospective associations between depression and severe bowel disease

	SBD at baseline in those without depression (n = 63)	No SBD at baseline in those without depression (n = 2840)	OR (95% CI)	Adjusted* OR (95% CI)
Rate depression at follow up (n, %)	7 (11.1)	108 (3.8)	3.2 (1.4 to 7.1)	2.5 (1.1 to 5.8)
	Depression at baseline	**No depression at baseline**	**OR (95% CI)**	**Adjusted† OR (95% CI)**
Rate SBD at follow up (n, %)	14 (2.8)	35 (1.2)	2.3 (1.2 to 4.3)	2.0 (1.01 to 3.8)

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

(NEMESIS), a longitudinal study of the prevalence, incidence, course, consequences, and somatic comorbidities of psychiatric disorders in the Dutch general population, approved by a recognised ethics committee. The sample consists of 7076 people who were examined at three measurement points (baseline, T1, and T2) over a period of three years.[3][4] Subjects were seen at home by trained interviewers using the composite international diagnostic interview (CIDI),[5] 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 3 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 comorbidity 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% (of which 85% in receipt of medical treatment) 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[6] 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[7] that may predispose to no only IBD, but also to depression.[8]

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BOOK REVIEW

Cannabis use and dependence: public health and public policy

Written by two scholars with outstanding reputations in the drugs field, this book assessed current knowledge on very controversial topics: the health, psychological, and social effects of cannabis use and misuse, the effectiveness and costs of cannabis prohibition, and policy alternatives. Well written and well organised, with chapter summaries and a final topic by topic summing up, this book is probably the most comprehensive and honest attempt to improve the quality of public policy debate on cannabis.

The most comprehensive, because the authors address the whole range of relevant issues, including some that remain "taboo" in several developed countries: they compare the health effects of cannabis with alcohol and tobacco; they investigate the possible benefits of cannabis use; they scrutinise the necessary conditions to implement the legalisation of cannabis… Then "drug warriors" should be interested in reading about topics they usually refuse to discuss. For example, the authors showed very convincingly that in a legal market the government should regulate the quality, potency, price, and bulk of cannabis that is sold to consumers, so the removal of criminal sanctions for sale and possession of cannabis would not eliminate the costs of enforcing cannabis laws.

The most honest, because unlike propagandists from both sides and other self proclaimed "experts" who are prompt to express great certainty about studies flawed by methodological shortcomings, the authors admit that many results remain uncertain and controversial, and they identify promising avenues for future research. With regard to honesty, they also mention the frequent dishonest and selective citation of evidence on the health effects of cannabis that blurs the public debate and discredits health education among the youth. Last but not least, they remind us that beyond scientific evidence deciding which policy to adopt remains a moral issue depending on which social values we defend.

Patrick Peretti-Watel