Asthma prevalence and deprivation: a small area analysis

Clare Salmond, Peter Crampton, Simon Hales, Simon Lewis, Neil Pearce

Abstract

Study objective—To investigate the relation between the prevalence of asthma symptoms in adults and deprivation in the area of residence.

Design—Two complementary surveys carried out between 1991–1993 yielding adult asthma symptom prevalence throughout New Zealand. Deprivation is measured by the NZDep91 index of deprivation for small areas.

Setting—New Zealand.

Participants—A random sample of 25 042 adults aged 20–50 years.

Main results—After controlling for possible confounding by age, gender, and ethnicity, the 12 month period prevalence rates of asthma in this representative sample of New Zealand adults are significantly higher in the three most deprived area categories than in the least deprived (tenth) category. The prevalence ratio for the most deprived category compared with the least deprived category is 1.29 with 95% confidence intervals (CI) 1.14, 1.47. There is a linear increase in asthma prevalence with increasing area deprivation ($\chi^2 = 32.20, p < 0.001$). Independently, the rates are also 1.41 (95% CI 1.29, 1.54) times higher among Maori and 1.29 (95% CI 1.10, 1.52) times higher among the Pacific Island group than among the remaining, mostly European, respondents.

Conclusions—The relation between asthma in adults and area deprivation is unlikely to be attributable to study biases or confounding. Further work should examine the possible role of modifiable deprivation factors in this relation.

Asthma prevalence and deprivation: a small area analysis

(J Epidemiol Community Health 1999;53:476–480)

Asthma is an increasingly common problem in New Zealand and worldwide. Although many causes of asthma are known or suspected further work is needed to identify potentially modifiable environmental factors. Socioeconomic status may be one such broad factor. This paper focuses on the specific socioeconomic concept of deprivation.

There is abundant evidence in the international literature of a strong relation between health status and deprivation. This relation has been demonstrated for a large number of health states and health status measures. In particular there is some recent evidence of relations between asthma prevalence, treatment and severity, and deprivation. In considering this evidence, however, it is important to distinguish between studies in children that mainly reflect cumulative asthma incidence, and studies in adults that may be influenced by differences in the prolongation and exacerbation of symptoms that may affect the duration of the condition.

It is mainly in studies done in the 1960s and 1970s (for example, Mitchell and Dawson) that asthma has been found to be more common in children in the higher social classes, but there has been less evidence of social class differences as the diagnosis of asthma has become more widespread. A recent review of 24 studies in children published since the 1960s concluded that negative associations were about as numerous as positive associations, and most studies showed no association at all. The authors posited that the lack of clarity may be attributable to differing definitions of asthma, differing methods for assessing asthma, and different age groups studied. However, severe asthma seems to be more common in children in the lower social classes and hospital admissions because of asthma are higher in low socioeconomic status groups. For example, a study of schoolchildren in Aberdeen published in 1969 showed that the association between childhood asthma and socioeconomic status is strongly modified by degree of asthma severity.

There have been fewer asthma prevalence surveys in adults, but most studies show evidence of greater symptom prevalence or reduced lung function in lower socioeconomic status groups. Some of these findings could be attributable to social class differences in diagnostic labelling of wheezing in adults, but it is more likely that they reflect real differences in asthma prevalence that are because of prolongation and exacerbation of symptoms into adulthood.

A national survey of asthma prevalence among 25 666 New Zealanders aged 20–50 during 1991–3 provides an opportunity to gain substantive evidence concerning the relation between asthma prevalence and a new area-based measure of deprivation in adults.

Methods

The 1991–1993 asthma survey has been described in detail previously. A one page questionnaire was mailed to a national random sample of 35 888 adults aged 20–44 years (at enrolment) selected from electoral rolls. The overall response (excluding ineligibles) was 82%. We used the same operational definition of asthma as used in the European Community Respiratory Health Survey. A person was
considered to have had asthma in the previous
year if they had a positive response to one or
more of the following three questions: (1) Have
you been woken by an attack of shortness of
breath at any time in the last 12 months? (2)
Have you had an attack of asthma in the last 12
months? (3) Are you currently taking any
medicine (including inhalers, aerosols or tab-
lets) for asthma?

Age at the time of the survey was calculated
from the date of birth given on the asthma
questionnaire. Ethnicity was obtained as
Maori, Pacific Islander, European, and/or
Other, and then classified hierarchically into
Maori or part (10.2%), Pacific Islander or part
(2.5%), and a third group with European
and/or Other ancestry (79.6%+7.6%).

Deprivation in an area was measured by the
NZDep91 index of deprivation for small
areas. The index was created from 1991
census data and is available for all meshblocks
(the smallest administrative area used in the
New Zealand Census of Population and
Dwellings). The full index is a weighted
combination of 10 proportions in a small area,
typically consisting of one or two geographi-
cally contiguous meshblocks having in total at
least 100 persons usually resident in them. The
proportions are standardised for age (in four
groups) and gender, and two are adjusted for
household size and composition. The overall
distribution of this index was then split into
deciles to produce a 10 point scale of depriva-
tion where 1 is least deprived, and 10 is most
deprived. The deprivation value for a small
area was assigned to each of its constituent
meshblocks. These deprivation scores may be
considered to have had asthma in the previous

Table 1  New Zealand asthma survey response by area deprivation score

<table>
<thead>
<tr>
<th>Deprivation score*</th>
<th>Respondents‡ (%)</th>
<th>Asthma study sample‡ (%)</th>
<th>New Zealand population (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (least deprived areas)</td>
<td>11.32</td>
<td>10.39</td>
<td>10.65</td>
</tr>
<tr>
<td>2</td>
<td>10.85</td>
<td>10.26</td>
<td>10.26</td>
</tr>
<tr>
<td>3</td>
<td>10.30</td>
<td>9.76</td>
<td>9.97</td>
</tr>
<tr>
<td>4</td>
<td>9.96</td>
<td>9.44</td>
<td>9.97</td>
</tr>
<tr>
<td>5</td>
<td>9.94</td>
<td>9.56</td>
<td>9.68</td>
</tr>
<tr>
<td>6</td>
<td>9.48</td>
<td>9.36</td>
<td>9.71</td>
</tr>
<tr>
<td>7</td>
<td>9.59</td>
<td>9.90</td>
<td>9.77</td>
</tr>
<tr>
<td>8</td>
<td>9.45</td>
<td>9.88</td>
<td>9.76</td>
</tr>
<tr>
<td>9</td>
<td>9.74</td>
<td>10.47</td>
<td>10.06</td>
</tr>
<tr>
<td>Number 10 (most deprived areas)</td>
<td>9.36</td>
<td>10.97</td>
<td>10.17</td>
</tr>
<tr>
<td>Total</td>
<td>25 042</td>
<td>34 869</td>
<td></td>
</tr>
</tbody>
</table>

*NZDep91 value. ‡Excludes 624 respondents who could not be geocoded.

Results

NZDep91 values were assigned to the 25 042 respondents (97.6%) with a meshblock geoco-
dable address. The deprivation profile of the
asthma study sample and the respondents is
shown in table 1 along with the full distribution
in New Zealand. There are slightly more
persons living in deprived areas in the asthma
sample than in the New Zealand population.
Among those who responded, there are slightly
closer persons living in the most deprived areas,
and conversely, slightly more in the least
deprived areas, than in the whole of the country.

Overall 3912 (15.2%) respondents are con-
sidered to have had asthma in the previous 12
months. The proportion with asthma varies
significantly across the 10 deciles of area depriv-
ation, from 12.8% to 19.5% (table 2). A gen-
eralised linear model showed that there was a
significant linear trend in these proportions ($\chi^2 = 77.7, p < 0.001$).

The effect of non-response on this trend
depends on the relation between deprivation
and asthma and the likelihood of response. If all people with asthma responded, regardless
of their area deprivation, the linear trend in propor-
tions is still significant ($\chi^2 = 16.6, p <
0.001$). If response among people with asthma
was inversely proportional to deprivation, a
more pronounced trend would result. A
diminished trend would result only from
proportionately fewer asthmatics in the most
deprived areas not responding, which seems
unlikely.

The proportion with asthma is significantly
higher among Maori (22.0%) and Pacific
Islanders (20.5%) than among the remaining,
mostly European, group (14.3%). The respec-
tive crude prevalence ratios and 95% confi-
dence intervals are 1.54 (1.42, 1.67) and 1.43
(1.23, 1.67).

Controlling for age and gender, asthma
prevalence varied with area deprivation score,
being significantly greater in the three most

Table 2  One year period prevalence of asthma among New Zealand adults aged 20–50
years, by area deprivation score

<table>
<thead>
<tr>
<th>Deprivation score*</th>
<th>Respondents*</th>
<th>Prevalence of asthma (%)</th>
<th>Prevalence ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (least deprived areas)</td>
<td>2 836</td>
<td>13.1</td>
<td>1.0</td>
</tr>
<tr>
<td>2</td>
<td>2 717</td>
<td>12.8</td>
<td>0.98</td>
</tr>
<tr>
<td>3</td>
<td>2 580</td>
<td>13.1</td>
<td>1.00</td>
</tr>
<tr>
<td>4</td>
<td>2 493</td>
<td>15.0</td>
<td>1.14</td>
</tr>
<tr>
<td>5</td>
<td>2 490</td>
<td>14.9</td>
<td>1.14</td>
</tr>
<tr>
<td>6</td>
<td>2 374</td>
<td>14.7</td>
<td>1.13</td>
</tr>
<tr>
<td>7</td>
<td>2 492</td>
<td>14.9</td>
<td>1.14</td>
</tr>
<tr>
<td>8</td>
<td>2 366</td>
<td>16.4</td>
<td>1.25</td>
</tr>
<tr>
<td>9</td>
<td>2 440</td>
<td>18.6</td>
<td>1.43</td>
</tr>
<tr>
<td>10 (most deprived areas)</td>
<td>2 344</td>
<td>19.5</td>
<td>1.49</td>
</tr>
<tr>
<td>address not geocodable</td>
<td>624</td>
<td>16.8</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>25 666</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*NZDep91 value.
Table 3  Generalised linear models* for asthma prevalence among New Zealand adults

<table>
<thead>
<tr>
<th>Explanatory variables</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
<th>Model 4</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PR‡ 95% CI</td>
<td>PR 95% CI</td>
<td>PR 95% CI</td>
<td>PR 95% CI</td>
</tr>
<tr>
<td>Area deprivation†</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(least deprived) 1</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>2</td>
<td>0.98</td>
<td>0.85, 1.12</td>
<td>0.97</td>
<td>0.85, 1.12</td>
</tr>
<tr>
<td>3</td>
<td>1.00</td>
<td>0.87, 1.15</td>
<td>1.00</td>
<td>0.87, 1.15</td>
</tr>
<tr>
<td>4</td>
<td>1.15</td>
<td>1.00, 1.31</td>
<td>1.14</td>
<td>1.00, 1.30</td>
</tr>
<tr>
<td>5</td>
<td>1.13</td>
<td>0.99, 1.29</td>
<td>1.12</td>
<td>0.98, 1.28</td>
</tr>
<tr>
<td>6</td>
<td>1.11</td>
<td>0.97, 1.27</td>
<td>1.09</td>
<td>0.95, 1.25</td>
</tr>
<tr>
<td>7</td>
<td>1.13</td>
<td>0.99, 1.29</td>
<td>1.08</td>
<td>0.95, 1.24</td>
</tr>
<tr>
<td>8</td>
<td>1.23</td>
<td>1.08, 1.41</td>
<td>1.19</td>
<td>1.04, 1.35</td>
</tr>
<tr>
<td>9</td>
<td>1.41</td>
<td>1.24, 1.59</td>
<td>1.30</td>
<td>1.14, 1.48</td>
</tr>
<tr>
<td>10 (most deprived)</td>
<td>1.47</td>
<td>1.29, 1.66</td>
<td>1.29</td>
<td>1.14, 1.47</td>
</tr>
</tbody>
</table>

| Ethnicity             |         |         |         |         |
| European and other    | 1.0     | 1.0     | 1.0     | 1.0     |
| Maori                 | 1.52    | 1.40, 1.64 | 1.41 | 1.29, 1.54 | 1.66 | 1.49, 1.84 |
| Pacific Islander      | 1.41    | 1.24, 1.64 | 1.29 | 1.10, 1.52 | 1.41 | 1.17, 1.71 |

*Models 1–3 used the ECRHS definition of asthma, a positive answer to at least one of three questions—woken by shortness of breath, having “asthma” or having medication for asthma; model 4 used only a positive answer to the shortness of breath question; all models also included age and gender. †PR: prevalence ratio. Values are exponentiated parameter estimates.

Discussion

The 12 month age/gender adjusted period prevalence rates of asthma in this representative New Zealand sample of adults aged 20–50 years are 1.19–1.30 times higher in the most deprived areas than in the least deprived areas. Independently, the rates are also 1.41 times higher among Maori and 1.29 times higher among Pacific Islanders but we focus on the findings for area deprivation because the ethnic differences have been discussed elsewhere.12

As the sample of New Zealanders used in the asthma surveys is closely representative of the whole population it is unlikely that selection bias would have more than a minimal effect on the observed associations. Although a non-response bias is evident, the sensitivity analysis shows a stronger relation to area deprivation than the model for the ECRHS definition, which required a positive answer to any of the three questions, while the models for the other two questions (asthma attacks and asthma medication use) showed weaker and non-significant relation (not shown).

Models including interactions between age group and both deprivation and ethnicity showed, as found previously,1 that there was a significant interaction with ethnicity (p = 0.050), but not with deprivation as well (p = 0.19). However, when the interaction term with ethnicity was included in the model, the prevalence ratios for deprivation categories remained unchanged (to two decimal places).

Models were also developed for each of the three individual questions used in the European Community Respiratory Health Survey definition of asthma. The model for the first question (being woken by shortness of breath, table 3 model 4) showed a stronger relation to area deprivation than the model for the ECRHS definition, which required a positive
deprivation. In a previous report the authors considered that non-response bias had little effect on reported symptom prevalence. Thus the estimate of the effect of area deprivation on asthma prevalence, controlling for ethnicity, is also unlikely to be substantially affected by non-response bias.

There may be some random misclassification of asthma by the screening questions in the questionnaire. In particular, there may be some adults with respiratory symptoms such as cough or wheezing that are likely to be associated with smoking rather than asthma itself. However, the questions we used have validated well against physician diagnosed asthma, and have in fact been found to have greater validity than supposedly more “objective” measures such as bronchial hyperresponsiveness testing. Furthermore, the composite definition of asthma that we used is based on “asthma attacks”, use of asthma medications, or “waking with an attack of shortness of breath”, and does not focus on symptoms such as cough or wheeze; thus, it seems unlikely that our findings are explained by smoking related misclassification of asthma. Similarly, the use of deprivation of an area at the time of the 1991 census may have caused some minor, random misclassification of area deprivation at the time each questionnaire was answered because of changes of residence, although generally between neighbourhoods with similar levels of deprivation. The effect of these random misclassifications would be to slightly underestimate any real association of asthma with area deprivation.

Possible confounding of the relation between asthma and area deprivation by the usual demographic factors—age, gender, and ethnicity—has been controlled in the analyses. Smoking (in adults) and exposure to environmental tobacco smoke in children are known to be associated with the development of asthma but as smoking is part of the causal chain that leads from deprivation to asthma it is inappropriate to control for it when assessing the overall effect of deprivation. Nevertheless, it would be interesting to see to what extent smoking explained the observed association. However, smoking information was not collected in our survey. Family size (not obtained in this study) is negatively associated with asthma prevalence and is therefore unlikely to explain the association between deprivation and asthma prevalence. If such confounding is present our figures are likely to underestimate the size of the association. Other major confounders are unlikely.

Study biases, then, are unlikely explanations for the observed association. Thus, if the association is causal, deprivation must precede the onset of asthma symptoms. Although the present cross sectional survey cannot examine this issue, it is unlikely that the existence of a condition like asthma could lead to deprivation except in extreme cases (for example, if severe asthma affects educational or employment opportunities). The observed linear increase in asthma prevalence with increasing deprivation is support for a causal relation.

Various factors have been linked to asthma, including exposure to allergens, air pollution (especially tobacco smoke), diet, respiratory viral infections, fetal growth and family size—although findings have been inconsistent. It seems plausible that at least some of these factors could be on the causal pathway between material deprivation—acting at the level of communities and/or individuals—and asthma. For example, childhood patterns of respiratory infections may be influenced by extremes of overcrowding or social isolation; it may be more difficult to maintain a healthy diet in a deprived area because of cost or lack of shops or transport; smoking in adults and chronic long term exposure to environmental tobacco smoke in children are likely to be more common in deprived homes and communities.

As noted above, the ethnic differences have been discussed elsewhere but it is interesting to briefly consider possible explanations for the ethnic differences, as these may also be relevant to area deprivation. There is very little ethnic difference in asthma prevalence in children in New Zealand but prevalence differences emerge with increasing age. The reasons for this are unclear, but it seems likely to be attributable at least in part to a duration effect rather than an incidence effect—that is, the incidence of asthma is similar in Maori and non-Maori children but Maori children experience more exacerbations and prolongation of symptoms, and the prevalence does not therefore decline with age as it does in non-Maori. Although this is probably in part because of differences in the asthma risk factors mentioned above (particularly tobacco smoking), a major review concluded that these differences were also because of problems of access to adequate health care and asthma education for Maori with asthma. For example, Maori children who gain access to health care are less likely to be prescribed preventive medication. It is unclear whether the same process may be occurring for socioeconomic differences in asthma prevalence, and it is certainly possible that our findings are because of differences in disease duration rather than incidence. It would clearly be useful to examine area deprivation and asthma in children and adults in the same population, and to follow up populations over time to determine the causes of the asthma prevalence and severity differences in adults that we have observed.

Our findings lend further weight to the case for reducing material deprivation on public health grounds. In the primary prevention of asthma, social policies that tackle deprivation at the community level may prove effective, as well as attempting to modify personal risk factors.

Funding: this study was funded in part by the New Zealand Asthma Foundation, Lotteries Health and the Health Research Council of New Zealand. The Wellington Asthma Research Group is supported by a Programme Grant from the Health Research Council of New Zealand.

Conflicts of interest: none.


32. Martineau P. Role of viral infections in the inception of asthma. Arch Dis Child 1995;70:469–70.

33. Martineau P. Role of viral infections in the inception of asthma. Arch Dis Child 1995;70:469–70.

34. Martineau P. Role of viral infections in the inception of asthma. Arch Dis Child 1995;70:469–70.

35. Martineau P. Role of viral infections in the inception of asthma. Arch Dis Child 1995;70:469–70.


40. Martineau P. Role of viral infections in the inception of asthma. Arch Dis Child 1995;70:469–70.

41. Martineau P. Role of viral infections in the inception of asthma. Arch Dis Child 1995;70:469–70.