The prevalence of asthma in the developed world has increased over recent decades. At least part of this increase is real and not due to changes in diagnostic practices. This increase in genetically stable populations must be due to environmental or lifestyle factors. Since asthma is more common in westernised/wealthy nations, the increases in prevalence within these countries suggest that asthma may be a disease of affluence.

Within countries there have been many attempts to relate the prevalence of asthma and atopy to socioeconomic status (SES). Unlike atopy, which is more common in higher SES groups, the evidence for socioeconomic patterning in asthma is conflicting. Studies have found both increased and decreased prevalence of asthma in higher SES groups, while other studies have found no relationship.

There may be several explanations for these findings, including the methodology of the studies. The use of symptoms such as wheeze and cough to indicate asthma may be misleading. This may be because of the occurrence of non-asthmatic wheeze and cough (due to bronchitis, for example) or because of differences in reporting of these symptoms between socioeconomic groups. Alternatively, physician diagnosis and treatment of asthma may differ between economic groups leading to either an apparent increase in prevalence in those with better access to care or an apparent increase in asthma severity in those with inadequate treatment. Finally, it is probable that SES interacts with specific environmental factors to have different effects on asthma prevalence in different populations. For example, less affluent people in the USA have greater exposure to cockroaches and therefore may have more asthma symptoms due to cockroach allergy. House dust mites are more important in other populations and exposure may be greater in higher SES homes.

Clouding the issue further, many studies of SES have been cross sectional and most have been restricted to children. We report a longitudinal cohort study from birth to age 26 that recorded both subjective and objective measures of asthma and atopy to examine the relationship between SES and asthma.

**METHODS**

**Participants**

The Dunedin Multidisciplinary Health and Development Study is a longitudinal investigation of health and behaviour in a birth cohort. Study members were born in Dunedin, New Zealand between April 1972 and March 1973. Of these, 1037 children (91% of eligible births; 52% male) participated in the first follow up assessment at age 3, constituting the base sample for the remainder of the study. Cohort families represent the full range of SES in the general population of New Zealand and are primarily white of European descent. Follow up assessments occurred at ages 5, 7, 9, 11, 13, 15, 18, 21, and at age 26 years when we assessed 980 (96%) of 1019 study members still alive. The study was approved by the Otago ethics committee and written informed consent was obtained at each assessment.

**Measurements of socioeconomic status (SES)**

The primary measures of SES used in this study were based on the scale of Elley and Irving. The scale places each occupation into one of six categories (6 = unskilled labourer, 1 = professional) based on the educational levels and income associated with that occupation in data from the latest New Zealand census.

**Childhood SES**

Socioeconomic conditions may change during childhood, and rather than take the measurement from a single point at
birth or early in life, we calculated childhood SES as the average of the higher SES level of either parent, assessed repeatedly from the study member’s birth through to age 15 (n = 1031). Although homemakers, unemployed, and students are not classified, only six study members could not be assigned an SES category between birth and age 15.

The distribution of SES classifications was 1–1.9 = 14.3%; 2–2.9 = 19.9%; 3–3.9 = 34.8%; 4–4.9 = 23.2%; 5–6 = 7.2%.

Adult SES

To address the issues of whether adult SES has an impact on current asthma, and also whether the experience of asthma during childhood impacts on adult SES, the adult SES score was measured according to the study member’s own Elley-Irving category at age 26 (n = 934, the remaining cohort seen at 26 were homemakers, students, or unemployed and were omitted). 45

Alternative measures of SES

Parental income taken as the total income of both parents recorded on two occasions (ages 13 and 15) and expressed as the mean of the totals at these ages (n = 955) was used as an alternative measure of childhood SES. The highest level of educational achievement by age 26 (1 = no qualifications; 2 = school certificate only (the most basic New Zealand qualification); 3 = higher level school qualification (e.g. sixth form certificate), or post-school qualification (e.g. trade certificate diploma); 4 = bachelors degree or higher) was used as an alternative measure of adult SES (n = 980).

Outcome measures

Asthma, wheeze and cough

At age 9 the accompanying adult (usually mother) answered questions on current wheezing and coughing symptoms and also provided a retrospective history of respiratory symptoms and illnesses since birth. The frequency, severity, trigger factors, and treatment of symptoms were recorded, including whether a diagnosis of asthma had been made. 46 At age 11, 13, and 15 questions were asked regarding symptoms since the last assessment. At age 18, 21, and 26 a self-administered questionnaire, 47 to which we added questions from the American Thoracic Society questionnaire, 48 was completed by the study member before the interviewer administered questionnaire.

Current asthma was defined as diagnosed asthma with symptoms in the previous year. Current wheeze was defined as all reported wheezing, excluding those with only one or two episodes of wheezing each lasting for less than 1 hour in the previous year. Asthma treatment included any bronchodilator, corticosteroid, or cromoglycate medication.

Lung function and airway responsiveness

Spirometric tests were performed at each assessment from age 9, recording the best of three acceptable forced expiratory manoeuvres without prior bronchodilator (within 6 hours). At 18 and 26 bronchodilator responsiveness was measured by repeating the spirometric tests 10 minutes after nebulised or metered dose salbutamol (200 μg) via a large volume spacer. At ages 9, 11, 13, 15, and 21 a methacholine challenge was performed using a modified Chai protocol 49 as previously described and validated. 50 Five deep inhalations of methacholine 0.025 mg/ml were administered through a Hudson Updraft nebuliser and spirometric tests were repeated after 30 seconds and 2 minutes. Provided the forced expiratory volume in 1 second (FEV1) fell less than 20%, further methacholine was administered at concentrations of 0.25, 2.5 and 25 mg/ml. The procedure was stopped when FEV1 fell by 20%, if there were symptoms of concern, or after the final concentration. The provoking concentration causing a 20% fall in FEV1 (PC20) was determined by linear interpolation. A PC20 of 8 mg/ml or less was regarded as increased airway responsiveness. Study members showing airflow obstruction at baseline (FEV1/FVC <75% at 9 and 11 years or <70% at older ages) were not challenged but instead were retested after salbutamol. An increase in the FEV1 of 10% of baseline or more was regarded as indicating bronchodilator airway responsiveness.

Atopic status

Skin prick testing was undertaken at age 13 in 714 study members using house dust mite (Dermatophagoides pteronyssinus, Bencard, UK), grass, cat, dog, horse, kapok, wool, Aspergillus fumigatus, Penicillium, and Cladosporium (Hollister-Stier, USA). 41 These were repeated at age 21 in 885 study members with the addition of cockroach allergen (Hollister-Stier, USA). A positive response was defined as a weal diameter 2 mm greater than the negative control. Serum IgE was measured at ages 11 and 21 in 571 and 786 study members, respectively. 52 IgE values were log-transformed before analysis to approximate a normal distribution.

Control variables

Parental asthma

The adult attending with the study member at age 7 was asked whether the natural mother and father had asthma, hay fever, or allergies. 43 This information was obtained again from the study member at age 18. Preference was given to the information obtained at age 7 if available.

Cigarette smoking

Current smoking was defined as smoking daily for at least 1 month of the previous year. Parental smoking during the study member’s life was ascertained at ages 9, 11 and 13 from the accompanying parent and used as an indicator of likely exposure to environmental tobacco smoke. Whether the study member’s mother had smoked during pregnancy was asked at age 9.

Breast feeding

Breast feeding and its duration. Study members were classified according to whether they had been breast fed for longer than 3 weeks. 43 The accuracy of recall of the duration of breast feeding was validated by comparison of data recorded prospectively by infant healthcare workers. 53

Birth order

Birth order was categorised according to whether the study member had no, one, two, or three or more older siblings.

Statistical analysis

Analyses of the effects of childhood SES were performed using the “xt” procedures of the Stata 8.0 software package (Stata Corporation, TX) for cross sectional, time series datasets (generalised estimating equations). Continuous variables were analysed using the xt regression model and binary outcomes by the xt logit model. Analyses were undertaken using asthma, atopy, FEV1/FVC ratio, and log IgE levels as dependent variables and childhood SES as the independent variable. All analyses included a term for the age of measurement and were adjusted for sex. Analyses were repeated with adjustment for potential covariates including family history of asthma, smoking, breast feeding, birth order, maternal smoking during pregnancy, and the presence of a smoker in the household. Analyses of the effect of childhood asthma (up to the age of 15) on adult (age 26) SES and of adult SES on adult asthma outcomes were performed...
analyses were not significant after adjustment for current
age for high, medium, and low Elley-Irving childhood SES
groups are shown in fig 1. These indicate no consistent
patterns of association between SES and the asthma outcomes
measured. Cross sectional time series analyses also indicate
no significant associations between mean childhood SES and
a diagnosis of asthma, wheeze, asthma treatment, time off
school or work for asthma, lung function, or airway
responsiveness to methacholine or salbutamol in either the
analyses adjusting for sex alone, or the analyses adjusting for
other covariates (table 1).

There was a trend towards increased atopy on skin prick
testing (any skin prick test positive) with increasing child-
hood SES (p = 0.044), but this was no longer significant after
controlling for birth order, family smoking, smoking during
pregnancy, and breast feeding (p = 0.084). There were no
significant associations between SES and log serum IgE.

Total parental income at ages 13 and 15 was not associated
with current asthma, significant wheeze, asthma treatment,
lung function, atopy, serum IgE, or airway responsiveness in
either the analyses adjusting for sex alone or the analyses
adjusting for other covariates.

Adult SES
The study member’s own SES and educational achievement
by age 26 were not significantly associated with current
asthma, doctor diagnosed asthma, current wheeze, time off
work due to asthma, or bronchodilator response at age 26.
Significant associations between being woken by a cough at
26 and both adult SES and educational status in unadjusted
analyses were not significant after adjustment for current
smoking (data not shown). The FEV1/FVC ratio tended to be
lower in those with lower educational achievement
(p = 0.026) and lower SES (p = 0.071), but these were not
significant in the fully adjusted analyses (p = 0.13 and 0.16,
respectively).

Impact of asthma on adult SES and educational
achievement
There were no significant associations between a diagnosis of
asthma in childhood and the educational achievement or
adult SES of the study member at age 26, even after adjusting
for childhood SES.

DISCUSSION
This study found no convincing evidence of an association
between childhood SES and asthma at any age from
childhood to early adulthood. The findings were consistent
whether we used parental occupation or parental income as
the measure of childhood SES. Furthermore, adult SES had
no influence on adult asthma, and a diagnosis of asthma in
childhood had no impact on educational achievement or
adult SES. Our findings with regard to asthma contrast with
those for other health problems in this cohort in which
significantly worse adult health occurred in those who grew
up in a low SES background.46

There was a significant trend to increased atopy with
higher childhood SES. This was not statistically significant
after adjusting for potential confounding factors, but is
consistent with the findings of other studies.53 We did not
find any significant association between serum IgE and SES.

This study addresses some of the problems which limit the
interpretation of previous studies. Firstly, we have prospect-
ively collected data throughout childhood into early adult-
hood, allowing a search for consistent associations between
SES and asthma at different ages. Secondly, objective
measures of lung function and airway responsiveness were
performed as well as subjective measures of asthma such as
symptoms, doctor diagnosis, and treatment. We can therefore
discount apparent differences in the prevalence of asthma
due to selective reporting of symptoms, differences in
diagnostic practice, or access to care. Thirdly, detailed
information was available on known and potential confoun-
ders such as smoking during pregnancy, parental asthma,
parental smoking (indicating likely exposure to environ-
mental tobacco smoke), birth order, and breast feeding.

Although we have identified no relationship between SES
and asthma from childhood to early adulthood, this does not
preclude an association during later life. There was a trend to
a reduced prevalence of asthma and less asthma treatment in
those from advantaged socioeconomic backgrounds by age 26
(fig 1). However, this trend was not apparent for wheeze,
airway responsiveness, or lung function and was in the
opposite direction to the trend observed for atopy.

The generalisability of our findings to other populations
is unclear. In common with any study of social conditions and
health, the findings may not apply to societies in which social
conditions differ markedly. Although the Dunedin cohort
represents the entire socioeconomic spectrum of New
Zealand, the range and nature of the socioeconomic
disparities may be less or greater in other countries. In
addition, issues relating asthma to SES may differ across
countries. For example, poor housing in the USA may expose
children to high levels of cockroach allergen. In this cohort
cockroach allergy was less common than allergy to house
dust mite (unpublished). Exposure to house dust mite is
likely to be universal in a city such as Dunedin.47

Nevertheless, we believe that this study is the most
comprehensive examination of the relationship between
asthma and socioeconomic conditions to date. Data from

Figure 1 Prevalence of asthma, wheeze, asthma treatment, airway
responsiveness, atopy, and spirometric values in high (mean Elley
Irving score <2, ▲), medium (mean score 2–4, ■) and low (mean score
>4–6, ●) childhood socioeconomic status (SES) groups.
surveys in other countries should be interpreted in the light of the methodological issues that we have raised.

ACKNOWLEDGEMENTS

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REFERENCES


Table 1  Odds ratios, coefficients and 95% confidence intervals from cross sectional time series analyses of asthma, wheeze, asthma treatment, time off school/work due to asthma, bronchial responsiveness, atopy, FEV1/FVC ratio, and log serum IgE on childhood socioeconomic status (SES; 1 = professional, 6 = manual labourer)

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<th>Adjusted for sex only</th>
<th>Fully adjusted</th>
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<tbody>
<tr>
<td></td>
<td>n OR 95% CI</td>
<td>n OR 95% CI</td>
</tr>
<tr>
<td>Current asthma</td>
<td>6104 1.07 (0.94 to 1.21)</td>
<td>4939 1.07 (0.92 to 1.25)</td>
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<tr>
<td>Wheeze</td>
<td>6096 1.04 (0.96 to 1.13)</td>
<td>4937 1.05 (0.96 to 1.16)</td>
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<tr>
<td>Asthma treatment</td>
<td>6099 1.05 (0.92 to 1.19)</td>
<td>4936 1.05 (0.90 to 1.22)</td>
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<td>Time off school/work</td>
<td>5278 1.08 (0.93 to 1.26)</td>
<td>4220 1.11 (0.94 to 1.30)</td>
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<tr>
<td>Bronchial response*</td>
<td>5656 1.07 (0.94 to 1.21)</td>
<td>4632 1.03 (0.88 to 1.20)</td>
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<tr>
<td>Atopy</td>
<td>1593 0.90** (0.80 to 1.00)</td>
<td>1299 0.89 (0.78 to 1.02)</td>
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<th>n Coefficient 95% CI</th>
<th>n Coefficient 95% CI</th>
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<tr>
<td>FEV1/FVC ratio</td>
<td>5860 –0.13 (–0.46 to 0.20)</td>
<td>4792 –0.03 (–0.42 to 0.37)</td>
</tr>
<tr>
<td>Log IgE</td>
<td>1350 0.03 (–0.06 to 0.12)</td>
<td>1095 0.01 (–0.10 to 0.11)</td>
</tr>
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</table>

n = number of observations in each analysis (several observations made for each subject at different ages); OR = odds ratio (change in odds of the outcome for each point change in the SES scale); coefficient = change in prevalence of outcome for each point change in the SES scale; FEV1 = forced expiratory volume in 1 second; FVC = forced vital capacity.

*Bronchial responsiveness means a response to methacholine or salbutamol as defined in the text. Fully adjusted figures are adjusted for sex, breast feeding, parental asthma, parental smoking, birth order, and smoking during pregnancy. **p<0.05.
Abnormalities of fatty acid metabolism in CF

This study found low tissue levels of the fatty acid docosahexaenoic acid (DA) and a raised ratio of arachidonic acid (AA) to DA in patients with cystic fibrosis (CF) compared with healthy controls in nasal and rectal biopsy specimens (p<0.001 and p = 0.009 for the ratios, respectively). In cells from nasal scrapings the AA:DA ratio was also significantly higher in subjects with CF than in controls (p<0.001), with CF carriers showing intermediate levels between the two (p<0.001). Intermediate levels similar to CF carriers were observed in patients with asthma or acute upper respiratory tract infections (URTIs), but lower levels were observed in those with inflammatory bowel disease. The findings in CF were independent of pancreatic sufficiency status. No difference between plasma carriers of two essential amino acids, linoleic acid and eicosatrienoic acid, or AA were noted between CF patients, carriers, and healthy controls. The authors suggest that the imbalance may be a result of cystic fibrosis transmembrane conductance regulator gene expression in these tissues rather than a consequence of malabsorption, although “intermediate” levels in those with asthma or an URTI suggest that other factors are involved. Fatty acids such as DA can be converted into potent anti-inflammatory mediators and the low levels observed in this study may lead to the excessive inflammatory response observed in CF.

This study confirms a tissue abnormality of fatty acid metabolism in CF, previously only demonstrated in CF knockout mice. Correction of this imbalance could be a potential treatment target in CF.

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Relationship between socioeconomic status and asthma: a longitudinal cohort study

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