Eur Respir J 2006; 27: 85–94
DOI: 10.1183/09031936.06.00061205

Area-level socio-economic status and health status among adults with asthma and rhinitis

P.D. Blanc*,#, I.H. Yen*, H. Chen*,#, P.P. Katz*,¶, G. Earnest*, J.R. Balmes*,+, L. Trupin*, N. Friedling*, E.H. Yelin*,¶ and M.D. Eisner*

ABSTRACT: **Socio-economic status (SES) may affect health status in airway disease at the individual and area level.**

In a cohort of adults with asthma, rhinitis or both conditions, questionnaire-derived individual-level SES and principal components analysis (PCA) of census data for area-level SES factors were used. Regression analysis was utilised to study the associations among individual- and area-level SES for the following four health status measures: severity of asthma scores and the Short Form-12 Physical Component Scale (SF-12 PCS) (n=404); asthma-specific quality of life (QoL) scores (n=340); and forced expiratory volume in one second (FEV1) per cent predicted (n=218).

PCA yielded a two-factor solution for area-level SES. Factor 1 (lower area-level SES) was significantly associated with poorer SF-12 PCS and worse asthma QoL. These associations remained significant after adding individual-level SES. Factor 1 was also significantly associated with severity of asthma scores, but not after addition of the individual-level SES. Factor 2 (suburban area-level SES) was associated with lower FEV1 per cent predicted in combined area-level and individual SES models.

In conclusion, area-level socio-economic status is linked to some, but not all, of the studied health status measures after taking into account individual-level socio-economic status.

KEYWORDS: **Adults, asthma epidemiology, asthma quality of life, asthma with chronic sinusitis, economic aspects of asthma**

AFFILIATIONS
*Dept of Medicine,
#Cardiovascular Research Institute, and
¶Institute for Health Policy Studies, University of California San Francisco, San Francisco, and
+Division of Environmental Health Sciences, School of Public Health, University of California Berkeley, Berkeley, CA, USA.

CORRESPONDENCE
P.D. Blanc
350 Parnassus Avenue Suite 609
San Francisco
CA 94117
USA
Fax: 1 4154766426
E-mail: blancp@itsa.ucsf.edu

Received:
May 26 2005
Accepted after revision:
September 05 2005

SUPPORT STATEMENT
This work was supported by grant R01 ES 10906 (National Institute for Environmental Health Sciences, Research Triangle Park, NC, USA).

European Respiratory Journal
Print ISSN 0903-1936
Online ISSN 1399-3003

Socio-economic status (SES) is accepted as playing a major role in determining health status [1], but there is no consensus on a standard approach to its measurement in health research [2, 3]. SES can be ascertained at the individual level by assessment through questionnaire items that directly quantify personal or family income, items that delineate markers of social status such as education and occupation (which are also surrogates of economic status), or survey measures that estimate wealth or financial assets. SES can also be measured at an ''area level'', that is, the status of the surrounding neighbourhood or community. Area-level SES is often captured through population-based surveys, especially census sampling data. Area-level SES typically incorporates income measures, education patterns and employment rates, but may also include measures of wealth and deprivation, including average home values and rates of social-assistance provision.

There is increasing interest in analysing health status in relation to individual- and area-level measures of SES considered simultaneously, thus addressing the question of whether living in a disadvantaged area confers additional risk of poor health beyond low individual-level SES [4–8]. Examining the relationships between individual-level SES, area-level SES, and health status among persons with airway diseases such as asthma or rhinitis is particularly relevant to this question. While SES is linked to many chronic diseases, it is especially germane to asthma because there are specific mechanisms by which SES-related variables could adversely affect asthma and rhinitis. At the individual level, examples of exposures that may be linked to SES include: lower-paying occupations with higher exposures to irritants and allergens, compromised housing stock with poorer indoor air quality, environmental tobacco smoke (ETS) exposure, and gas cooking stove use. In terms of linkages to SES at the area level, those with airway disease in particular may be at an increased risk of adverse health effects from poor ambient air quality due to traffic density, point-source pollution from proximity to industrial sites, and social-community stressors.

The current authors hypothesised that area-level SES influences general and disease-specific health status in adult asthma and rhinitis, even after controlling for individual-level SES and taking into account potential confounding variables that might explain apparent area-level SES associations. This would be consistent with a model of health determinants in which the surrounding community, which includes both the social and the physical environment, has important effects on persons with these chronic health conditions. This hypothesis was tested among persons in a cohort of adults with asthma and rhinitis by combining detailed individual-level SES data from interviews and home visits with linkage to census data for area-level measures of SES.

METHODS

Overview

Interview data from one wave of a multiwave longitudinal study of adults with asthma and rhinitis, including measures of general health status, disease severity and quality of life (QoL), were analysed. A subset of subjects also underwent spirometry. The subjects' residential addresses were linked to census data at the block-group level. Using principal components analysis (PCA), two area-level SES factors derived from census variables were developed. These were tested as independent variables in models of health status, disease severity, QoL and lung function, with and without concurrent measures of individual SES measures garnered at the individual level at the time of interview. The study was carried out following University of California San Francisco Committee on Human Research approval.

Initial subject recruitment

The current cohort of persons with asthma and rhinitis was first established in 1992, following the recruitment of adults aged 18–50 yrs with asthma whose names were recorded on out-patient clinic visit logs. These logs were maintained by a random sample of northern California (USA) adult pulmonary specialists, allergy immunologists and family practice physicians [9, 10]. The participation rates of the sampled physicians were as follows: 57 out of 92 (62%) pulmonary specialists; 17 out of 19 (89%) allergists; and 34 out of 74 (46%) family practice physicians. Physicians logged in potential study subjects prospectively over a 4-week period (increased to 8 weeks in cases of low visit frequencies), and 751 out of 869 (86%) eligible patients were recruited successfully. To address subject attrition over time, the original cohort was augmented in 1999 by recruitment of an additional group of subjects, some with asthma and others with rhinitis alone [11].These subjects were recruited by random-digit telephone dialling, which was also limited to northern California. For this recruitment by telephone, the eligibility criteria for subjects were to be aged 18–50 yrs and to report the physician-diagnosed condition. For the random-digit dialling sample, 300 out of 455 (66%) health condition-eligible subjects participated. Since 2000, the region from which the sample was drawn had a population aged 18–65 yrs of ~6 million persons.

The respondents were interviewed at the time of initial enrolment, with follow-up interviews at 18–24-month intervals thereafter. Subjects from the random-digit telephone recruitment joined the initial cohort after that group had had three rounds of interviews; the combined cohort was interviewed twice prior to the wave of interviews that were analysed here. Thus, at the time of the interview used in this analysis, all subjects had participated in 2–5 previous interviews. Findings from the initial recruitment and earlier interviews have been reported previously [9–19].

Subject interviews

In total, 416 (76%) interviews were completed in 2002–2003 from among 548 eligible subjects who had completed the most recent interview 2 yrs before. Two interviews were conducted in Spanish; all others were in English. Subjects with annual family incomes <US$40,000 or with a high school level of education or less were less likely to participate ($p<0.01$). Those successfully followed were somewhat older than those who were not (mean difference 2.6 yrs; 95% confidence interval (CI) 0.8–4.3 yrs). There were no statistically significant differences based on sex or race/ethnicity.

Other details of subject retention through this interview wave have been reported previously [20]. Interviews used computer-assisted telephone interview software and averaged 45 min in duration. Core individual SES items included annual household income, employment status and education; in addition, standard demographics and clinical data related to asthma and rhinitis were collected.

Lung function

Spirometry was completed in a subset of subjects participating in a home visit assessment (229 out of 390 (59%) eligible subjects) [20]. There were no significant individual SES differences between subjects who did and did not undergo lung function testing. Spirometry was performed using an EasyOne™ spirometer (ndd Medical Technologies, Chelmsford, MA, USA) that met American Thoracic Society (ATS) 1994 spirometry standards [21], and a standard protocol that conformed to ATS performance guidelines [22].

Individual-level SES based on survey responses

Individual SES measures were derived from responses to the structured telephone interviews. All of the principal survey items had been pre-tested and used in previous interview waves, and were pilot tested again in the context of the current questionnaire. For education, subjects were asked to report the highest level of education achieved. Since the current study group was relatively well educated, three separate categories for higher education were created, defined by some college education or an associate degree, completion of undergraduate studies or graduate training. Less than a high school education and high school completion were collapsed together into a single category. Annual family income was elicited as a categorical rather than continuous variable, with a maximum open-ended category of ≥$100,000 per annum. Income was assigned for 12 subjects who did not provide these data at this interview based on previous responses, and family income was assigned for two other subjects who were single (no spouse or partner) based on the USA median earnings for their current reported occupation.

Occupation and employment status were elicited using items based on standard USA labour market definitions. Open-ended responses were coded according to the 2000 USA Census [23] job classifications, and then collapsed into broad occupational groups defined *a priori* as likely to have greater homogeneity in SES. Based on specific interview questionnaire items probing disability status, subjects who were not working were subdivided into two groups, as follows: those not working, at least in part, due to health reasons (based on specific survey responses ascertaining this); and those not working due to all other reasons, for example, keeping house, studying or retirement. The rationale for this was that not working due to health reasons is likely to be associated with substantial income loss, whereas the latter category is likely to be heterogeneous in terms of associated SES.

Health status, disease severity and disease-specific QoL

General health status was assessed using the Short Form (SF)-12, yielding the Physical Component Scale (PCS; normative score of 53 ± 7 among USA adults aged 18–44 yrs without chronic morbidity) [24, 25]. Disease severity was quantified using the severity of asthma score, a validated measure including symptoms, medications and healthcare utilisation [26, 27]. A maximum score of 28 reflects the greatest asthma severity. Persons with rhinitis alone can also be scored, typically achieving lower values based on symptoms and medications. The Marks Asthma QoL questionnaire, a validated, asthma-specific instrument using a 20-item Likert-type scale adapted for telephone administration [28, 29], was administered in 345 individuals with asthma. Possible scores range 0–60; higher scores reflect poorer asthma-specific QoL.

Other interview-based and home visit-derived covariates

In addition to individual-level SES variables, selected subject covariates were analysed for smoking exposure and the use of gas cooking, since these could play a role as confounders linked to area-level SES, yet were not captured by the specific individual-level SES items. Pet ownership was also examined, because a dog-related effect had been observed in a previous analysis in the current cohort and such ownership might also track with SES. Smoking exposure status was classified into the following six separate categories: current active smoking (n=31); former smokers with current regular ETS exposure (n=27); former smokers without regular home or work ETS exposure (n=83); never-smokers with regular home or work ETS exposure (n=46); and never-smokers without such ETS exposure (n=217; referent category). Where available from home visits [20], serum cotinine values were used to confirm or exclude ETS exposure (n=199) and, in two cases, to identify active smokers who denied such activity. Subjects were categorised dichotomously for each of the following variables: gas cooking stove use (n=207; 51%); pet cat ownership (n=154; 38%); and dog ownership (n=190; 47%). Each was established through direct home inspection or, in subjects without a home visit, by survey responses.

Geocoding for census linkage

Latitude and longitude coordinates were assigned to each subject's address using electronic street map databases, a process referred to as geocoding [30–34]. Geocoding was carried out by Sonoma Technology (Petaluma, CA, USA) using the TeleAtlas MultiNet™ USA (TAMN) roadway database (Tele Atlas, Lebanon, NH, USA), which contains detailed roadway and address information and high positional accuracy. TeleAtlas Eagle Geocoding Technology was used to locate addresses in the TAMN database, yielding a corresponding latitude and longitude coordinate pair [35]. When necessary, addresses were verified using data sources, such as aerial photography [36], from the USA Geological Survey and online address location services such as Yahoo!® and MapQuest®. Analyses were performed using the Environmental Systems Research Institute ArcGIS software.

Area-level SES measures derived from census data

Subjects were linked to the corresponding 2000 USA Census data at the block-group level. Out of 404 subjects analysed, 22 pairs (44 subjects; 11%) fell within the same census-block group. On an *a priori* basis, the current authors selected a series of variables from the census that represented area-level measures of SES, which included: measures of income and poverty status; employment status; education; home value, age and ownership; family configuration; and population density. Categorical variables were reported as the proportion with a given characteristic in the block group, and continuous variables as the median value across all observations. Thus, a distribution of values was generated whose units comprised a set of proportions or medians. Census demographics such as distributions of age, sex, race or immigrant status were not treated as area-level surrogates of SES.

Exclusions and missing data

Of 416 interviewees potentially available for analysis, geocoding was not possible for one subject, and seven additional subjects who had recently moved were also excluded due to discordance between residence at interview and at spirometry. Four other subjects were excluded due to insufficient income or occupational data to allow income classification, resulting in a final study sample of 404 subjects.

Data analysis

Health status measures were compared between subjects with asthma (with or without concomitant rhinitis) and those with rhinitis alone using the unpaired t-test. A correlation matrix was calculated for a series of SES-related variables from the 2000 USA Census linked to subjects' geocoded location at the block-group level. Of the 404 subjects analysed, 400 (99%) were geocoded to a level of precision within a single street block or its equivalent; the geocoded location of residence was approximated as the centre point of a five-digit postal code for only four subjects. Due to substantive intercorrelations among these area-level variables, PCA was carried out to develop integrated measures of SES that would not be collinear. Exploratory analyses yielded two variables (*i.e.* median rental costs and per cent commuting $\geqslant$30 min) not retained in final models due to poor factor solutions. The weights of orthogonally transformed factors from the PCA were used to generate two area-level SES measures. Correlations for each census variable were calculated from the PCA against each of four health status measures: SF-12 PCS; severity of asthma scores; forced expiratory volume in one second (FEV_1) % predicted; and asthma-specific QoL. With the four health status measures as dependent variables, three multiple linear regression analyses were tested. The first used individual SES variables as independent variables, the second used the two area-level SES measures together, and the third used both individual- and area-level SES measures combined. The present authors retested the final models including demographic covariates and additional individual-level variables related to cigarette smoke exposure, gas cooking stove use, and cat and dog ownership. In a separate sensitivity analysis addressing the possible effects of differently scaled census variables, the current authors retested a separate set of PCA factors weighted by z-scores derived from the distributions of the census variables. For the calculation of the z-scores, duplicate census block-group observations were excluded. The data were also re-analysed using generalised estimating equation (GEE) modelling to take into account the potential effect of clustering by shared census block groups (n=22 pairs of clustered subjects only).

RESULTS

Subject descriptive data

The descriptive, self-reported demographic and SES data for the 404 subjects analysed are summarised in table 1. More than two-thirds of the subjects were female, consistent with the general female sex predominance in adult asthma. Overall, the group was largely White, non-Hispanic, well-educated and middle-to-upper income, although minorities, those with lower levels of education and persons with lower income were well represented. For example, with regards to minority groups, Hispanics comprised 11% and Blacks 6% of subjects. Of 88 subjects with annual family incomes ≤US$40,000, 19 (22%) were ≤125% of the poverty level, a measure of lower SES.

Table 2 presents the outcome measures of study interest without stratification by diagnosis. Out of 404 subjects, 341 (84%) had a physician's diagnosis of asthma, with or without concomitant rhinitis, whereas 63 (16%) reported a diagnosis of rhinitis alone. The subset of subjects with rhinitis alone had significantly better general health status as measured by the SF-12 PCS (mean score difference 3.5; 95% CI 0.5–6.4), lower severity of asthma scores (mean score difference -6.6; 95% CI -5.3– -7.9), and higher FEV1 % pred (mean difference 14%; 95% CI 7–20%). Asthma QoL, which was a disease-specific measure ascertained only among those with this condition (n=340 due to one missing observation), yielded a mean value of 16.2±14.7 with an SEM of 3.22.

Development of independent predictive variables for area-level SES

Table 3 presents descriptive statistics for the census variables included in the PCA and summarises the two-factor solution that resulted. The initial two Eigen values were 6.0 and 2.1, with a subsequent rapid fall off (<1.0). Out of the 13 variables included, 10 weighted ±0.66 or greater on an initial factor (factor 1), six in a positive and four in a negative direction. Three factors weighted ≥±0.66 on the second factor (factor 2), two positively and one negatively. The range of observed values for factor 1, based on the mix of subjects' covariates, was -2.3–4.9 (10th to 90th percentile range -1.1–1.3). The range of observed values for factor 2 was -4.1–1.6 (10th to 90th percentile range -1.4–1.2). Table 4 presents correlation coefficients for each of the 13 census SES variables included in the two-factor PCA solution against the four health status measures of interest previously mentioned. Eight out of the 13 were significantly correlated ($p \leq 0.05$) with SF-12 PCS; all but one weighted ≥±0.66 on factor 1. The remaining SES variable (proportion of owner-occupied homes) manifested the highest degree of cross-weighting for factor 1 (-0.42). Four variables were significantly correlated ($p<0.05$) with severity of asthma

TABLE 1 Descriptive characteristics for adults with asthma or rhinitis undergoing structured telephone interviews

Subject characteristics	
Subjects n	404
Age yrs	46±9
Females	287 (71)
White, non-Hispanic	294 (73)
Education	
High school graduate or less	63 (16)
Some college, associate degree or trade school	137 (34)
College graduate	130 (32)
Graduate school or more	74 (18)
Smoking status	
Never	263 (65)
Former	110 (27)
Current	31 (8)
Annual family income US$#	
<40000	88 (22)
40000–60000	67 (17)
60000–80000	77 (19)
80000–100000	67 (17)
>100000	105 (26)
Employment status	
Managerial	161 (40)
Technical/service	34 (8)
Clerical/sales	84 (21)
Manufacturing/labourer/construction/agricultural	29 (7)
Not working, health reasons	33 (8)
Not working, other reasons	63 (16)

Data are presented as mean±SD and n (%). #: >100% due to rounding.

TABLE 2 Outcome measures of health status, lung function and quality of life

Outcome measure	Subjects n	Mean±SD
SF-12 PCS	404	44.8±10.9
Asthma severity score	404	6.9±5.4
FEV1 % predicted#	218	85.1±18.1
Asthma QoL score¶	340	16.2±14.7

SF-12 PCS: Short Form-12 Physical Component Scale; FEV1: forced expiratory volume in one second; % pred: % predicted; QoL: quality of life. #: only available among subjects completing a study home visit; ¶: available for subjects with asthma only (one subject missing data).

TABLE 3 Census SES variables included in a principal components analysis

Census variables in model	Frequency		Factor weights	
	Mean±SD	Range	1	2
Income variables				
Annual household income median US$ (thousands)	60±25	11–166	↓	
Income below the poverty level %	9.6±8.8	0–65	↑	
Households receiving SSSI %	4.3±4.3	0–28	↑	
Households on public assistance %	3.6±4.9	0–37	↑	
Housing variables				
Home values median US$ (thousands)	294±194	60–1000	↓	
Owner-occupied homes %	66±23	2–98		↑
Home construction yr median	1969±16	1939–1999		↑
Room occupancy of one person or less %	92±9	43–100	↓	
Population density thousands·mile^{-2}	6.4±7.3	0.001–53		↓
Other variables				
Managerial occupations %	40±16	4–81	↓	
Unemployed %	4±3	0–24	↑	
Some high school or less %	16±13	0–71	↑	
Single parent households %	14±9	0–58	↑	

Two items were cross-weighted at greater than ±0.40 as follows: factor 1, owner-occupied home, weight=-0.42; and factor 2, median home value, weight=-0.46. SES: socio-economic status; SSSI: supplemental social security income; ↑: positive factor weighting after orthogonal rotation ⩾0.66; ↓: negative factor weighting after orthogonal rotation ⩽-0.66.

TABLE 4 Correlation of area-level measures of SES with health status measures

Census measures	SF-12 PCS#		Severity of asthma score#		FEV1 % pred¶		QoL score+	
	r	p-value	r	p-value	r	p-value	r	p-value
Factor 1 variables								
Annual household income median US$	0.14	0.004	-0.10	0.04	0.004	0.96	-0.17	0.002
Income below poverty level %	-0.21	<0.0001	0.07	0.17	0.03	0.68	0.21	0.0001
Households receiving SSSI %	-0.18	0.0002	0.15	0.002	0.01	0.87	0.25	<0.0001
Households on public assistance %	-0.18	0.0002	0.05	0.35	0.02	0.74	0.21	<0.0001
Home value median US$	0.10	0.05	-0.09	0.06	0.14	0.05	-0.16	0.003
Room occupancy of one person or less %	0.10	0.06	-0.03	0.59	-0.05	0.50	-0.15	0.004
Managerial occupations %	0.15	0.002	-0.12	0.02	0.09	0.20	-0.23	<0.0001
Unemployed %	-0.16	0.002	0.06	0.22	-0.07	0.29	0.17	0.001
Some high school or less %	-0.15	0.003	0.07	0.14	0.02	0.73	0.22	<0.0001
Single parent households %	-0.23	<0.0001	0.13	0.01	-0.01	0.89	0.19	0.0005
Factor 2 variables								
Owner-occupied homes %	0.16	0.001	-0.04	0.42	-0.15	0.03	-0.13	0.01
Home construction year median	0.09	0.08	-0.04	0.37	-0.14	0.04	-0.01	0.89
Population density thousands·mile^{-2}	-0.03	0.59	0.06	0.23	0.15	0.03	0.004	0.94

SES: socio-economic status; SF-12 PCS: Short Form-12 Physical Component Scale; FEV1: forced expiratory volume in one second; % pred: % predicted; QoL: quality of life; SSSI: supplemental social security income. #: n=404; ¶: n=218; +: 340.

score, all of which also strongly weighted on factor 1. Four variables were significantly correlated ($p<0.05$) with FEV1 % pred; three weighted ⩾±0.66 on factor 2, whereas the remaining variable (median home value) manifests the highest degree of cross-weighting (-0.46) for factor 2. Eleven out of the 13 variables (all except housing construction year and population density) were significantly correlated ($p<0.05$) with asthma-specific QoL scores.

Individual-level SES variables as independent predictors of health status

Table 5 contains the multiple linear regression analyses for three groups of individual SES variables (education, income and employment status) combined as independent predictors in four separate models of health status end-points. The overall models were statistically significant ($p<0.001$) for severity of asthma score, SF-12 PCS and asthma-specific QoL, but not for FEV_1 % pred ($p>0.2$). Not working due to health reasons was the only individual SES variable consistently predictive of greater asthma severity, poorer health status and worse QoL. Taking into account the other variables in the model, annual family income did not appear to be related to any of the health status measures, nor was there a consistent trend in the point estimates of the parameter effects over increasing income levels. The three predictors were interrelated: income and education (Chi-squared test for trend, $p<0.001$), income and occupational status ($p<0.001$), and education and occupational status ($p<0.001$).

Combined models of area- and individual-level SES

The results of further multiple linear regression analyses are shown in table 6. These analyses study the same end-points as table 5, but are stratified. In models that included area-level SES only, factor 1 was a statistically significant predictor of lower SF-12 PCS (poorer heath status) within the whole group, after excluding those with rhinitis alone, and with and without individual-level SES (table 6). Taking into account individual-level SES, the difference spanning the lower to upper decile of observed area-level SES scores accounts for a 3.3-point decrement in the SF-12 PCS, slightly less than one half the SD of normative values [23, 24]. In modelling that included area-level SES only, factor 1 was a statistically significant predictor of severity of asthma score (leading to greater severity). After adjustment for individual-level SES, factor 1 was no longer statistically associated with the severity of asthma score. Excluding subjects with rhinitis alone did not substantively alter these findings. Factor 1 was also a predictor of asthma-specific QoL (leading to poorer QoL). This relationship remained statistically significant ($p=0.001$) after inclusion of individual-level SES. Taking into account the latter, an interdecile change in factor 1 score predicts a difference of 6.2 points in QoL score, which is approximately twice the observed SEM for the QoL (3.2).

Among all subjects, factor 2 was a significant ($p=0.02$) predictor of lower FEV_1 % pred after inclusion in the model of individual-level SES variables, which did little to change the overall explanatory power of the model. The point estimates of effect were similar after exclusion of subjects with rhinitis alone, although in the model including individual-level SES, the 95% CIs for factor 2 were wider and did not exclude the no-effect level ($p=0.06$). Based on the model, an interdecile change in area-level SES represented by factor 2 would account for a 7.7% decrement in FEV_1 % pred value (*e.g.* a change from 86% to 79% pred).

TABLE 5 Multiple linear regression analyses for individual socio-economic status (SES) variables and health status

Individual SES variables	Health status measures#							
	SF-12 PCS¶		Severity of asthma score+		FEV_1 % pred§		Asthma QoLƒ	
	β±SE	p-value	β±SE	p-value	β±SE	p-value	β±SE	p-value
Education								
High school graduate or less (referent)								
Some college, associate degree or trade school	-0.7±1.5	0.64	1.4±0.8	0.07	-0.3±3.9	0.94	-2.0±2.2	0.37
College graduate	2.0±1.6	0.22	-1.3±0.8	0.14	5.1±4.2	0.22	-7.3±2.4	0.002
Graduate school or more	1.6±1.9	0.41	-0.2±1.0	0.85	4.1±5.0	0.42	-5.2±2.8	0.07
Annual income US$								
<40000 (referent)								
40000–60000	-0.2±1.7	0.91	0.4±0.9	0.68	-6.5±4.4	0.13	-1.6±2.4	0.52
60000–80000	-0.8±1.6	0.62	0.7±0.8	0.38	-3.4±4.1	0.40	-0.7±2.4	0.77
80000–100000	0.2±1.7	0.89	1.6±0.9	0.08	0.0±4.3	0.99	2.4±2.5	0.35
>100000	0.3±1.6	0.87	0.8±0.8	0.35	-3.7±4.0	0.35	-1.6±2.4	0.52
Employment status								
Manufacturing/construction/agricultural (referent)								
Managerial (referent)	0.0±2.1	0.99	-0.8±1.1	0.47	5.1±5.5	0.35	2.8±3.0	0.35
Technical/service	1.3±2.5	0.61	0.3±1.3	0.81	12.0±6.1	0.05	0.8±3.5	0.83
Clerical/sales	-1.0±2.1	0.63	-1.2±1.1	0.29	4.4±5.6	0.44	2.9±3.0	0.33
Not working, health reasons	-17.8±2.6	<0.0001	5.7±1.4	<0.0001	-1.4±6.4	0.83	23.0±3.6	<0.0001
Not working, other reasons	-1.0±2.3	0.67	-0.7±1.2	0.54	8.3±5.8	0.16	5.4±3.2	0.09

SF-12 PCS: Short Form-12 Physical Component Scale; FEV_1: forced expiratory volume in one second; % pred: % predicted; QoL: quality of life; #: overall model p-value for FEV_1 $p=0.23$, all other models $p<0.0001$; ¶: n=404, model $R^2=0.20$; +: n=404, model $R^2=0.13$; §: n=218, model $R^2=0.02$; ƒ: n=340, model $R^2=0.21$.

TABLE 6 Area-level socio-economic status (SES) predictors of health status: multiple linear regression analyses with and without individual SES variables

Health outcomes#	Subjects n	Multiple logistic regression models¶									
		Area-level SES only					Area-level and individual SES				
		R^2	Factor 1		Factor 2		R^2	Factor 1		Factor 2	
			$\beta \pm$ SE	p-value	$\beta \pm$ SE	p-value		$\beta \pm$ SE	p-value	$\beta \pm$ SE	p-value
Asthma or rhinitis											
SF-12 PCS	404	0.04	-2.28 ± 0.53	<0.0001	0.84 ± 0.53	0.11	0.22	-1.36 ± 0.55	0.01	0.79 ± 0.50	0.12
Severity of asthma score	404	0.01	0.61 ± 0.27	0.02	-0.13 ± 0.27	0.62	0.13	0.21 ± 0.29	0.46	-0.13 ± 0.26	0.62
FEV_1 % pred	218	0.03	-0.35 ± 1.12	0.76	-3.59 ± 1.17	0.002	0.03	0.21 ± 1.28	0.87	-2.95 ± 1.24	0.02
Asthma alone											
SF-12 PCS	341	0.05	-2.28 ± 0.59	0.0001	1.16 ± 0.59	0.05	0.22	-1.40 ± 0.61	0.02	1.04 ± 0.56	0.06
Severity of asthma score	341	0.01	0.63 ± 0.29	0.03	-0.31 ± 0.29	0.27	0.14	0.31 ± 0.30	0.30	-0.33 ± 0.28	0.24
FEV_1 % pred	186	0.03	-0.44 ± 1.22	0.72	-3.41 ± 1.30	0.01	0.04	-0.13 ± 1.37	0.92	-2.62 ± 1.37	0.06
Asthma QoL score+	340	0.06	3.83 ± 0.78	<0.0001	-0.24 ± 0.78	0.76	0.23	2.58 ± 0.80	0.001	-0.06 ± 0.73	0.94

SF-12 PCS: Short Form-12 Physical Component Scale; FEV_1: forced expiratory volume in one second; % pred: % predicted; QoL: quality of life. #: a higher SF-12 PCS equates with a better health status, a higher severity of asthma score and asthma QoL score equate with poorer status; ¶: reported R^2 is adjusted to take into account number of predictors in model, models with individual SES include all of the variables listed in table 5; +: one asthma subject with missing data for QoL score.

Additional analyses

Using z-weighted scores to calculate the weighted factors yielded substantively similar results in the area-level modelling; moreover, re-estimating the models using area- and individual-level SES combined with age, sex and race (white, non-Hispanic compared to all others) did not change these findings (data not shown). There were 44 subjects (22 pairs) from within the same census block group. GEE modelling, taking into account clustering, did not substantively impact the estimated parameter coefficients or SE estimates (data not shown). In order to consider the potential confounding effects of ETS and gas stove exposure, the models were also re-estimated including these variables. In these linear regression models, factor 1 remained a statistically significant predictor of poorer SF-12 PCS ($p=0.01$) and asthma-specific QoL ($p=0.001$), and factor 2 remained a statistically significant predictor of lower FEV_1 % pred ($p=0.02$). Furthermore, there was no increase in the adjusted model R^2 values. Adding cat and dog ownership to this model did not substantively impact the models for SF-12 PCS or asthma QoL, but the area-level SES association between factor 2 and FEV_1 % pred weakened ($\beta \pm$ SE=-2.0 ± 1.4; $p=0.14$). In this model, dog ownership was an independent predictor of lower FEV_1 % pred ($\beta \pm$ SE=-6.2 2.8; $p=0.03$), an association that has been reported previously in this cohort [20].

DISCUSSION

It was found that SES at the area level predicts general and asthma-specific health status in adult asthma and rhinitis. A group of such variables (factor 1) consistent with lower SES (*e.g.* measures of income, education, occupational status and housing) was associated with greater asthma severity measured by the severity of asthma score, poorer general health status measured by the SF-12 PCS, and worse asthma QOL measured by the Marks disease-specific instrument. In contrast, a group of other variables (factor 2) consistent with "suburban" SES (*e.g.* more recently constructed, owner-occupied homes in less densely populated census-block groups) was associated with lower lung function, but not health status by other measures. The effects were clinically relevant when SES was scaled from the uppermost to lowest decile of change (10th to 90th percentile), a common metric of income disparity [4], and measured in terms of effect against 0.5 SD (SF-12 PCS) or 1 SEM unit (asthma-specific QoL), both accepted indicators of a "minimal important difference" [37, 38]. This was also the case in terms of a substantive difference in FEV_1 % pred.

It is important to note that, in the case of severity of asthma score, the relationship with area-level SES did not remain statistically significant after taking into account the effect of individual-level SES. Thus, although other general health status (SF-12 PCS) and asthma-specific health measures (asthma QoL and FEV_1 % pred) were related to area-level SES over and above individual-level SES, the current study findings were mixed.

Relevant studies of asthma and area-level SES have focused on childhood disease. A study in Boston (MA, USA), which combined survey responses with census-derived income data at the postal-code level, found that the area level was not statistically related to childhood asthma prevalence after taking into account individual education and income [39]. A study in Great Britain linked survey data for asthma symptoms to census data, showing that lower SES was associated with higher childhood asthma prevalence, taking into account sample area and ethnic group [40]. Although individual subjects' social class was studied separately, individual- and population-level measures of SES were not included in the same analysis. An Italian study of childhood asthma prevalence analysed individual SES data (using educational level) combined with a census-based approach for SES, including educational level, occupational category, unemployment, one-person families, large families, persons per room, and rental *versus* ownership [41]. The latter study also used census-tract income as a separate, population-level measure. After taking into account individual-level SES, there was a statistically significant link between one or both of the area-level SES measures for general asthma prevalence, the prevalence of severe asthma, and asthma hospitalisations. A recent analysis of childhood asthma studied 215 incident cases from a birth cohort of 3,970 in Rochester (MN, USA) falling within 17 census tracks [42]. In the study by JUHN *et al*. [42], area-level income and the transportation variable were related to asthma incidence, taking into account the limited individual-level SES surrogate of maternal education.

Studies of adult asthma also provide evidence of the relationship to area-level SES, although this research is more limited than that in childhood asthma. An analysis of adult asthma prevalence, using data from the European Respiratory Heath Survey, found that study centre-level SES based on educational level was an independent risk factor for disease, after taking into account individual-level SES (based on occupation or education) [43]. A series of studies conducted in the USA have analysed hospitalisation rates for asthma (children and adults), whilst considering census-derived data, but none of these studies has linked to individual-level survey data [44–48]. A recent study of the prevalence of adult asthma and other breathing problems (including bronchitis and emphysema) that considered both individual- and area-level SES found that area-level SES was not related to disease, but that an area-level measure including social cohesion and informal social control was related to this outcome, taking into account SES [49].

A study that was particularly relevant to the impact of individual- and area-level SES among those with established disease, rather than prevalence or incidence, analysed data on β-agonist use among 202 adults with asthma in Vancouver (BC, Canada) and linked this to SES based on Canadian census data at the postal-code level for median household income, unemployment rate, and education [50]. Each of these measures was significantly associated with β-agonist bronchodilator use in separate analyses, remaining statistically significant after taking asthma severity into account. However, only one SES measure, either individual- or area-level but not both, was included in each analysis. Also of note, geocoding at the postal-code level in the USA yields poorer area-level SES measures compared to data linked at either the census-block group or census-tract level. Census-block group and census-tract data (the latter are a larger aggregate of the former) have been shown to function similarly, providing geocoding success rates are comparable [51, 52]. In the current study, only four out of 404 subjects that were analysed had a match at less than a one-street block level of accuracy, for an overall precision at the block-group level of 99%.

The potential limitations of the present study should be recognised. Despite increasing use of geocoding, there is no single accepted approach for choosing the census-based area-level SES measures to analyse [5, 6, 8, 30, 51–54]. Although the complete list of census variables the present authors chose to include is not identical to other studies of chronic disease, it does have substantial overlap with them by including standard measures of income, wealth (assets) and social deprivation, while excluding demographic variables (such as age, sex and race/ethnicity) that may be indirect surrogates of SES but which subsume multiple other attributes as well. The current authors have attempted to minimise the overinterpretation of data, both in the inferences that are drawn from the ''factors'' themselves and by also providing data on the individual variables that comprise them. Since the subjects were drawn from northern California and are predominantly well educated with middle-to-upper income, the current findings should be generalised with caution, but this limitation should be balanced against the overall paucity of similar data from any other USA region or SES mix. Furthermore, since the current study subjects were dispersed over a fairly large area (few were even paired by census-block group), there was no opportunity to evaluate a single neighbourhood or cluster of small areas. Conversely, this argues against any theoretical concerns of clustering effects, that is, findings due to subjects with shared local influences.

In the current analysis, the inclusion of persons with allergic rhinitis but without asthma also might be considered a limitation, but this provides an advantage of a greater spectrum of disease severity, and the analysis excluding subjects with rhinitis alone yielded substantively similar results to those in the entire group. Finally, the present authors acknowledge that the apparent area-level SES associations may be explained by unmeasured individual-level SES not captured by personal education, income and employment status. For example, although these are good measures of income, they may not reflect accumulated wealth (such as home ownership), some of which was captured in the area-level measures. A composite individual-level SES measure may also have yielded additional data; one of the most common of these, however, the Hollingshead index, is based only on education and occupation, the latter of which does not score those not working for health or other reasons [55]. It is also possible that other, non-SES individual-level factors that track with area-level SES account for unexplained confounding leading to the current observations. This may explain the effect of dog ownership and suburban SES with regards to lung function. It is also possible that dog ownership is simply a marker for other individual-level factors that might indeed be linked to area-level SES (for example, larger home gardens with more allergenic plants). It has been previously reported that neither specific immunoglobulin E sensitisation nor endotoxin appears to account for the link between dog ownership and lower lung function in the current authors' data set [20].

Once again, it should be noted that the severity of asthma score was not related to area-level SES after taking into account individual-level SES. If area-level SES is indeed linked to the other general health status (SF-12 PCS) and disease-specific measures (asthma QoL, FEV_1 % pred) that were analysed among adults with asthma, rhinitis or both conditions, even after taking into consideration individual-level SES, the question of what underlying mechanisms may account for this relationship remains. For example, the current analysis does not include measures of outdoor environmental exposures that may correlate with area-level SES. One particularly relevant set of factors includes the proximity of a subject's residence to traffic and the density of the traffic flow. The census data that were used in the present analysis do not provide such information, but the current authors intend to explore this question further by employing other data sets linked through geocoding. Similarly, the present authors have yet to study area-level SES juxtaposed with measures of community in its social context, such as neighbourhood cohesion or support that may mediate asthma through multiple pathways, including stress modification or access to healthcare.

One challenge to such analyses is the complex nature of the potential interrelationships among these variables. Clearly, the ties between macrosocio-economic status and microhealth status in asthma and rhinitis need to be explored using multiple analytic approaches in order to more fully understand other potential explanatory mediators, to study possible regional differences in these relationships, and to better elucidate effects within socio-economic status strata.

ACKNOWLEDGEMENTS

The authors wish to thank F. Lurmann of Sonoma Technology, Inc. (Petaluma, CA, USA) for geocoding. The authors would also like to acknowledge the assistance of S. Shiboski for statistical review; the home visit study nurses for spirometry (C. Elago, A. Ervin-King, W. Kryda and J. Sansone); the study telephone interviewers (M. San Pedro and K. Van der Meulen); research assistant M. San Pedro-Salcedo; and the study coordinator C. Archea.

REFERENCES

1 Isaacs SL, Schroeder SA. Class – the ignored determinant of the nation's health. *N Engl J Med* 2004; 351: 1137–1142.
2 Duncan GJ, Daly MC, McDounagh P, Williams DR. Optimal indicators of socioeconomic status for health research. *Am J Public Health* 2002; 92: 1151–1157.
3 Cirino PT, Chin CE, Wolf M, Lovett M, Morris RD. Measuring socioeconomic status. Reliability and preliminary validity of different instruments. *Assessment* 2002; 9: 145–155.
4 Stafford M, Marmot M. Neighbourhood deprivation and health: does it affect us all equally? *Int J Epidemiol* 2003; 32: 357–366.
5 Winkleby MA, Cubbin C. Influence of individual and neighbourhood socioeconomic status on mortality among black, Mexican-American, and white women and men in the United States. *J Epidemiol Community Health* 2003; 57: 444–452.
6 Steenland K, Henley J, Calle E, Thun M. Individual- and area-level socioeconomic status variables as predictors of mortality in a cohort of 179,383 persons. *Am J Epidemiol* 2004; 159: 1047–1056.
7 Jaffe DH, Eisenbach Z, Neumark YD, Manor O. Individual, household and neighborhood socioeconomic status and mortality: a study of absolute and relative deprivation. *Soc Sci Med* 2005; 60: 989–997.
8 Henderson C, Diez Roux AV, Jacobs DR Jr, Kiefe CI, West D, Williams DR. Neighbourhood characteristics, individual level socioeconomic factors, and depressive symptoms in young adults: the CARDIA study. *J Epidemiol Community Health* 2005; 59: 322–328.
9 Blanc PD, Cisternas M, Smith S, Yelin E. Asthma, employment status, and disability among adults treated by pulmonary and allergy specialists. *Chest* 1996; 109: 688–696.
10 Blanc PD, Eisner MD, Israel L, Yelin EH. The association between occupation and asthma in general medical practice. *Chest* 1999; 115: 1259–1264.
11 Blanc PD, Trupin L, Eisner M, *et al.* The work impact of asthma and rhinitis: findings from a population-based survey. *J Clin Epidemiol* 2001; 54: 610–618.
12 Eisner MD, Blanc PD. Environmental tobacco smoke exposure during travel among adults with asthma. *Chest* 2002; 122: 826–828.
13 Katz PP, Yelin EH, Eisner MD, Blanc PD. Perceived control of asthma and quality of life among adults with asthma. *Ann Allergy Asthma Immunol* 2002; 89: 251–258.
14 Eisner MD, Yelin EH, Katz PP, Earnest G, Blanc PD. Exposure to indoor combustion and adult asthma outcomes: environmental tobacco smoke, gas, stoves, and woodsmoke. *Thorax* 2002; 57: 973–978.
15 Blanc PD, Trupin L, Earnest G, *et al.* Effects of physician-related factors on adult asthma care, health status, and quality of life. *Am J Med* 2003; 114: 581–587.
16 Cisternas MG, Blanc PD, Yen IH, *et al.* A comprehensive study of the direct and indirect costs of adult asthma. *J Allergy Clin Immunol* 2003; 111: 1212–1218.
17 Snyder L, Blanc PD, Katz PP, Yelin EH, Eisner MD. Leukotriene modifier use and asthma severity: how is a new medication being used by adults with asthma. *Arch Int Med* 2004; 164: 617–622.
18 Katz PP, Yelin EH, Eisner MD, Earnest G, Blanc PD. Performance of valued life activities reflected asthma-specific quality of life more than general physical function. *J Clin Epidemiol* 2004; 57: 259–267.
19 Yelin EH, Trupin L, Earnest G, Katz PP, Eisner M, Blanc P. The impact of managed care on health care utilization among adults with asthma. *J Asthma* 2004; 41: 229–242.
20 Blanc PD, Eisner MD, Katz PP, *et al.* Impact of the home indoor environment on adult asthma and rhinitis. *J Occup Environ Med* 2005; 47: 362–372.
21 Mortimer KM, Fallot A, Balmes JR, Tager IB. Evaluating the use of a portable spirometer in a study of pediatric asthma. *Chest* 2003; 123: 1899–1907.
22 American Thoracic Society. Standardization of spirometry. *Am J Respir Crit Care Med* 1994; 152: 1107–1136.
23 US Census Bureau. Industry and Occupation 2000. www.census.gov/hhes/www/ioindex/download.html. Date last accessed: February 8, 2005.
24 Ware JY, Kosiniski M, Keller SD. How to score the SF-12 health survey. 2nd Edn. Boston, MA, The Health Institute, New England Medical Center, 1995.
25 Gandek B, Ware JE, Aaronson NK, *et al.* Cross-validation of item selection and scoring for the SF-12 health survey in nine countries. *J Clin Epidemiol* 1998; 51: 1171–1178.

26 Blanc PD, Jones M, Besson C, Katz P, Yelin E. Work disability among adults with asthma. *Chest* 1992; 45: 461–472.
27 Eisner MD, Katz PP, Yelin EH, Henke J, Smith S, Blanc PD. Assessment of asthma severity in adults with asthma treated by family practitioners, allergists, and pulmonologists. *Med Care* 1998; 36: 1567–1577.
28 Marks GB, Dunn SM, Woolcock AJ. A scale for the measurement of quality of life in asthma. *J Clin Epidemiol* 1992; 45: 461–472.
29 Katz PP, Eisner MD, Henke J, Shiboski S, Yelin EH, Blanc PD. The Marks Asthma Quality of Life Questionnaire: further validation and examination of responsiveness to change. *J Clin Epidemiol* 1999; 52: 667–675.
30 Krieger N, Waterman P, Lemieux K, Zierler S, Hogan JW. On the wrong side of the tracts? Evaluating the accuracy of geocoding in public health research. *Am J Public Health* 2001; 91: 1114–1116.
31 Maantay J. Mapping environmental injustice: pitfalls and potential of geographic information systems in assessing environmental health and equity. *Environ Health Perspect* 2002; 110: 161–171.
32 McElroy JA, Remington PL, Trentham-Dietz A, Robert SA, Newcomb PA. Geocoding addresses from a large population-based study: lessons learned. *Epidemiology* 2003; 14: 399–407.
33 Cayo MR, Talbot TO. Positional error in automated geocoding of residential addresses. *Int J Health Geogr* 2003; 2: 10.
34 Bonner MR, Han D, Nie J, Rogerson P, Vena JE, Freudenheim JL. Positional accuracy of geocoded addresses in epidemiologic research. *Epidemiology* 2003; 14: 408–412.
35 TeleAtlas Eagle Geocoding Technology. www.geocode.com. Date last accessed: June 30, 2004.
36 UCLA GIS Database and Map Server. SPOT satellite 10-meter panchromatic data for California (used for the validation of roadway systems). www.gisdb.clluster.ucla.edu. Date last accessed: June 30, 2004.
37 Norman GR, Sloan JA, Wyrwich KW. Interpretation of changes in health-related quality of life: the remarkable universality of half a standard deviation. *Med Care* 2003; 41: 582–592.
38 Wyrwich KW, Tierney WM, Wolinsky FD. Further evidence supporting an SEM-based criterion for identifying meaningful intra-individual changes in health-related quality of life. *J Clin Epidemiol* 1999; 52: 861–873.
39 Litonjua AA, Carey VJ, Weiss ST, Gold DR. Race, socioeconomic factors, and area of residence are associated with asthma prevalence. *Pediatr Pulmonol* 1999; 28: 394–401.
40 Duran-Tauleria E, Rona RJ. Geographical and socioeconomic variation in the prevalence of asthma symptoms in English and Scottish children. *Thorax* 1999; 54: 476–481.
41 Cesaroni G, Frachi S, Davoli M, Forastiere F, Perucci CA. Individual and area-based indicators of socioeconomic status and childhood asthma. *Eur Respir J* 2003; 22: 619–624.
42 Juhn YJ, St Sauver J, Katusic S, Vargas D, Weaver A, Yunginger J. The influence of neighborhood environment on the incidence of childhood asthma: a multilevel approach. *Soc Sci Med* 2005; 60: 2453–2464.
43 Basagnia X, Sunjer J, Kogevinas M, *et al.* Socioeconomic status and asthma prevalence in young adults: the European Community Respiratory Health Survey. *Am J Epidemiol* 2004; 160: 178–188.
44 Carr W, Zeitel L, Weiss K. Variations in asthma hospitalizations and deaths in New York City. *Am J Public Health* 1992; 82: 59–65.
45 Gottlieb DJ, Beiser AS, O'Connor GT. Poverty, race, and medication use are correlates of asthma hospitalization rates. A small area analysis in Boston. *Chest* 1995; 108: 28–35.
46 Ray NF, Thamer M, Fadillioglu B, Gergen PJ. Race, income, urbanicity, and asthma hospitalization in California: a small area analysis. *Chest* 1998; 113: 1277–1284.
47 Yuen EJ. Severity of illness and ambulatory care-sensitive conditions. *Med Care Res Rev* 2004; 61: 376–391.
48 Chen Y, Stewart P, Dales R, Johansen H, Scott G, Taylor G. Ecological measures of socioeconomic status and hospital readmissions for asthma among Canadian adults. *Respir Med* 2004; 98: 446–453.
49 Cagney KA, Browning CR. Exploring neighborhood-level variation in asthma and other respiratory diseases. *J Gen Intern Med* 2004; 19: 229–236.
50 Lynd LD, Sandford AJ, Kelly EM, *et al.* Reconcilable differences: a cross-sectional study of the relationship between socioeconomic status and the magnitude of short-acting β-agonist use in asthma. *Chest* 2004; 126: 1161–1168.
51 Krieger N, Chen JT, Waterman PD, Soobader M-J, Subramanian SV, Carson R. Geocoding and monitoring of US socioeconomic inequalities in mortality and cancer incidence: does the choice of area-based measure and geographic level matter? *Am J Epidemiol* 2002; 156: 471–482.
52 Krieger N, Chen JT, Waterman PD, Soobader M-J, Subramanian SV, Carson R. Choosing area based socioeconomic measures to monitor social equalities in low birth weight and childhood lead poisoning: The Public Health Disparities Geocoding Project (US). *J Epidemiol Community Health* 2003; 57: 186–199.
53 Krieger N, Waterman PD, Chen JT, Soobader M-J, Subramanian SV. Monitoring socioeconomic inequalities in sexually transmitted infections, tuberculosis, and violence: geocoding and choice of area-based socioeconomic measures – the Public Health Disparities Geocoding Project (US). *Pub Health Rep* 2003; 118: 240–260.
54 Diez Roux AV, Stein Merkin S, Arnbett D, *et al.* Neighborhood of residence and incidence of coronary heart disease. *N Engl J Med* 2001; 345: 99–106.
55 Deonandan R, Campbell K, Ostbye T, Tummon I, Robertson J. A comparison of methods for measuring socio-economic status by occupation or postal area. *Chronic Dis Can* 2000; 21: 114–118.