Mediators of the Socioeconomic Gradient in Outcomes of Adult Asthma and Rhinitis

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Objectives. We estimated the extent to which socioeconomic status (SES) gradients in adult asthma and rhinitis outcomes can be explained by home and neighborhood environmental factors.

Methods. Using survey data for 515 adults with either asthma or rhinitis, or both, we examined environmental mediators of SES associations with disease severity, using the Severity of Asthma Scale, and health-related quality of life (HRQL), using the Rhinasthma Scale. We defined SES on the basis of education and household income. Potential environmental mediators included home type and ownership, exposures to allergens and irritants, and a summary measure of perceived neighborhood problems. We modeled each outcome as a function of SES, and controlled for age, gender, and potential mediators.

Results. Gradients in SES were apparent in disease severity and HRQL. Living in a rented house partially mediated the SES gradient for both severity and HRQL ($P < .01$). Higher perceived levels of neighborhood problems were associated with poorer HRQL and partially mediated the income–HRQL relationship ($P < .01$).

Conclusions. Differences in home and neighborhood environments partially explained associations of SES with adult asthma and rhinitis outcomes. (*Am J Public Health.* 2013;103:e31–e38. doi:10.2105/AJPH.2012.300938)

Socioeconomic status (SES) gradients in health, in which lower SES is associated with poorer health status and outcomes, have been observed for many chronic illnesses, including asthma and rhinitis.[1–6] Asthma and rhinitis have prevalence estimates of 10% to 20% in the adult population,[6,7] with large impacts on morbidity, health care utilization, and quality of life in the United States and globally.[7–9] A recent analysis of National Health Interview Survey data found that a substantial portion of the racial/ethnic differences in asthma prevalence could be attributed to SES.[10] The specific mechanisms by which SES affects asthma and rhinitis outcomes, however, have not been fully elucidated.

The conceptual model underlying the present study posits that there are multiple pathways through which socioeconomic differences can account for variations in disease outcomes. Such pathways could include a differential burden of environmental exposures, differences in health access, or differences in capacity for optimal disease self-management because of education or other resources. Thus, this model holds that SES is a key explanatory variable upon which health outcomes are dependent, and that SES can work through different sets of intermediate factors that can be characterized as distinct "pathways."

In the current study, we focused on one pathway conceptualized in this model: the extent to which observed SES gradients in adult asthma and rhinitis can be explained by environmental factors in the home or surrounding neighborhood. Environmental exposures in the home that have been associated with asthma and rhinitis outcomes include irritants such as tobacco smoke and combustion products from heating and cooking sources,[11–13] and allergens from sources such as dust mites, pets, cockroaches, rodents, and mold.[14–21] Asthma morbidity and rhinitis symptoms have also been associated with ambient air pollution,[22,23] residential proximity to vehicular traffic,[24,25] occupational exposures,[26–28] neighborhood characteristics,[29,30] and stressful life events.[31]

Many of these risk factors may be more common among individuals with lower SES because of a lack of control over the home environment (for example, because of renting rather than owning), a paucity of resources to make modifications to the home, living in less desirable neighborhoods, or exposure to other kinds of adverse environmental exposures. Because of consistent differences seen in prevalence and outcomes of these conditions by SES, the observation that environmental exposures associated with these outcomes also vary with SES,[32] and the large burden of these conditions, asthma and rhinitis are important conditions in which to investigate the role of environmental factors in mediating the SES gradient in health disparities.

METHODS

The data analyzed here derived from the 2008–2009 cycle of the University of California, San Francisco (UCSF) Asthma Rhinitis Cohort (ARC), an ongoing study approved by the UCSF Committee on Human Research. Recruitment into the ARC occurred in 4 stages, as detailed previously.[33] Briefly, participants with confirmed asthma were initially recruited in 2 groups through community-based sampling of northern California physicians in 1992 and 1996. In 1999, a third group of participants who reported a physician's diagnosis of asthma or rhinitis (identified as allergic rhinitis, sinusitis, chronic postnasal drip, or hay fever) was recruited through random-digit-dial sampling in the same geographic region.[34] We drew participants in a fourth group from a completed study of Northern California Kaiser Permanente patients hospitalized for asthma between 2000 and 2004. All participants were aged 18 to 50 years at baseline. In 2008 to 2009, a total of 549 participants were interviewed, including 315 participants from the first 3 groups and 234 from the fourth group (representing 85% and 69% retention from previous interviews, respectively). Study participants completed

45-minute structured telephone interviews covering current symptoms and treatments, health-related quality of life (HRQL), health care utilization, occupation, smoking status, activity limitations, home environment, neighborhood conditions, and sociodemographic characteristics.

We included 2 commonly used measures of SES, income and educational attainment,[35] each grouped into 6 levels in the questionnaire, as shown in Table 1. Education is considered a stable measure of SES across the adult lifespan and income is indicative of current access to resources. Asking about categories of income in surveys rather than absolute levels reduces item nonresponse[36] and measuring education in terms of credentials rather than years better captures its effect on socioeconomic position.[37] We modeled both measures as categories to allow for nonlinear effects on health outcomes.[37] In sensitivity analyses, we combined both variables into a single measure, giving a point for each level of education and category of income and dividing the resulting score into quintiles, an approach that we have used previously.[38]

Potential Environmental Mediators

We examined 4 categories of self-reported environmental exposures that might account for the SES gradient in asthma and rhinitis outcomes: housing, allergens and irritants, neighborhood, and occupation. Our conceptual model of a distinct environmental pathway of SES effects informed our overall study focus. In our choice of the specific variables analyzed, we relied upon measures known to be associated with asthma and rhinitis outcomes and that could be reasonably expected to vary with SES, consistent with the model as posited.[11–31]

Housing. We combined type and ownership of housing into a multicategory variable, defined as mobile home (most of which were owner-occupied), rented apartment, rented single-family home, or owner-occupied home or apartment. This variable was intended to capture potential SES-linked differences in control over one's living environment with regard to potential exposures and the capacity to make modifications to reduce disease symptoms.

Allergens and irritants. Variables reflecting environmental allergen exposures in the home included carpeted bedroom, presence of mold and mildew with water damage, and owning a pet against the advice of a physician (to focus on atopic individuals). Variables capturing potential home exposure to environmental irritants included gas heat from a stand-alone unit or kitchen stove (not a natural gas furnace), using a fireplace or wood-burning stove for heat at least 5 days per week in winter, and active or passive tobacco smoke exposure. For several of the potential exposures, we investigated more inclusive definitions: mold or mildew without necessarily observing water damage, gas for either cooking or heating, and pet ownership (without regard to medical advice about living with a pet). None of these sensitivity analyses revealed any substantial differences from the presented results.

Neighborhood. To evaluate the contribution of the neighborhood environment, we used a previously validated survey-based scale in which participants rated the degree to which each of the following was a problem (ranging from 0 = no problem to 5 = severe problem): traffic, trash, odors, smoke from fires, noise, and violence.[29,30] These perceived problem categories are intentionally wide-ranging, subsuming elements of ambient air pollution (i.e., traffic, smoke, odors) as well as the social environment (i.e., trash, noise, violence). Nonetheless, the scale manifested a high degree of internal consistency (Cronbach's $\alpha = 0.76$), indicating that it measures a single construct reflecting perceived neighborhood

TABLE 1—Characteristics of Study Participants (n = 515): University of California, San Francisco Asthma Rhinitis Cohort, 2008–2009

Characteristic	Distribution, No. (%) or Mean ±SD (Range)
Female	386 (75)
Race/ethnicity	
African American	72 (14)
Latino	64 (12)
Asian/Pacific Islander	34 (7)
Other or multiple race	11 (2)
White	334 (65)
Age, y	53 ±9 (25–67)
Annual family income, $	
< 20 000	53 (10)
20 000–< 40 000	91 (18)
40 000–< 60 000	71 (14)
60 000–< 80 000	83 (16)
80 000–< 100 000	74 (14)
≥ 100 000	143 (28)
Education	
No high-school degree	22 (4)
High-school graduate	68 (13)
Some college, no degree	133 (26)
Associate degree or trade school	86 (17)
Bachelor's degree	126 (24)
Postgraduate education	80 (16)
Employment status	
Employed	301 (58)
Unable to work	99 (19)
Other (seeking work, retired, attending school, keeping house)	115 (22)
Disease onset before age 18 y	199 (39)
Condition group	
Asthma alone	84 (16)
Asthma with rhinitis	411 (80)
Rhinitis alone	20 (4)

conditions. We therefore calculated a summary score for each respondent based on the mean problem rating over the 6 items, with a potential range of 0 to 5.

Work exposures. Because there may be an SES gradient in occupational exposures to irritants or sensitizers, we also examined reported exposure to vapors, gas, dust, or fumes (VGDF) at work. To elicit this, we used a single validated questionnaire item.[39] We analyzed occupational exposure limited to the subset of participants who were employed at the time of interview.

Outcome Measures

The primary outcome measures in this study were disease severity and disease-specific HRQL. We quantified disease severity using the Severity of Asthma Scale, a well-validated measure that incorporates current symptoms and standard asthma medications plus longer-term indicators of severity, including previous hospitalization or intubation and chronic corticosteroid use.[40–44] Potential scores ranged from 0 to 28, with higher scores reflecting greater severity. Participants with a physician's diagnosis of chronic rhinitis without asthma also completed the Severity of Asthma Scale battery, typically yielding scores of 5 points or lower, consistent with subclinical asthma, within a spectrum of airway disease.[34,45]

To capture HRQL, we used a validated English-language version of the Rhinasthma Questionnaire, a measure designed for both rhinitis and asthma.[33,46] The questionnaire consists of 28 questions rated on a 5-point Likert scale, summed and transformed to a 0 to 100 scale in which higher scores indicate poorer HRQL.

Statistical Analysis

A total of 515 (94%) participants had complete data on all variables in the current analysis. After ascertaining that the 33 participants with missing data did not differ from included participants with respect to demographics, outcomes, or exposures, we deleted them from all subsequent analyses. We compared scores for both the severity of asthma and rhinasthma HRQL by condition status and across the 6 levels of each measure of SES (income and education), using the ANOVA *F*-test and the pairwise *t* test comparing each level of education and income to the highest (referent) category. We then examined the relationships between the environmental exposure variables of interest and the SES measures, using the χ^2 test for categorical and the *t* test for continuous measures. For these analyses, we dichotomized annual household income as less than $40 000 versus at least $40 000 and education as less than a bachelor's degree versus bachelor's degree or higher, using the cut points apparent in the distribution of the outcome variables across the 6-level variables. We investigated the relationships among exposures and the 6-level SES measures but, finding no additional relevant associations, we present only the results using the dichotomous SES variables.

We entered all exposure variables that were at least moderately ($P < .1$) associated with either SES measure into ordinary least squares regression models of disease severity and HRQL; we also included age and gender in these models a priori, along with 1 SES measure at a time. Regression diagnostics, including assessment of multicollinearity, indicated that all models were well within acceptable limits. To account for possible clustering among participants in the 4 recruitment groups, we estimated robust standard errors for the final models presented.

We tested statistically significant parameter estimates ($P < .05$) from the multivariable models for statistical mediation of the relationships between SES and either asthma severity or HRQL. Mediation analysis is used to quantify the degree to which an intervening variable (here, an environmental exposure) accounts for the relationship between a predictor variable (e.g., SES) and the outcome in question. In a situation of complete mediation, the parameter estimate for the predictor will no longer be significant after adding the mediating variable to the model. More often, partial mediation occurs; that is, the parameter estimate is reduced, but may still be significant. In this situation, the significance of the mediation is assessed with a statistical test. In the current analysis, we used a modification of the Sobel test for statistical mediation that allows for dichotomous intervening variables and the simultaneous control of other variables.[47]

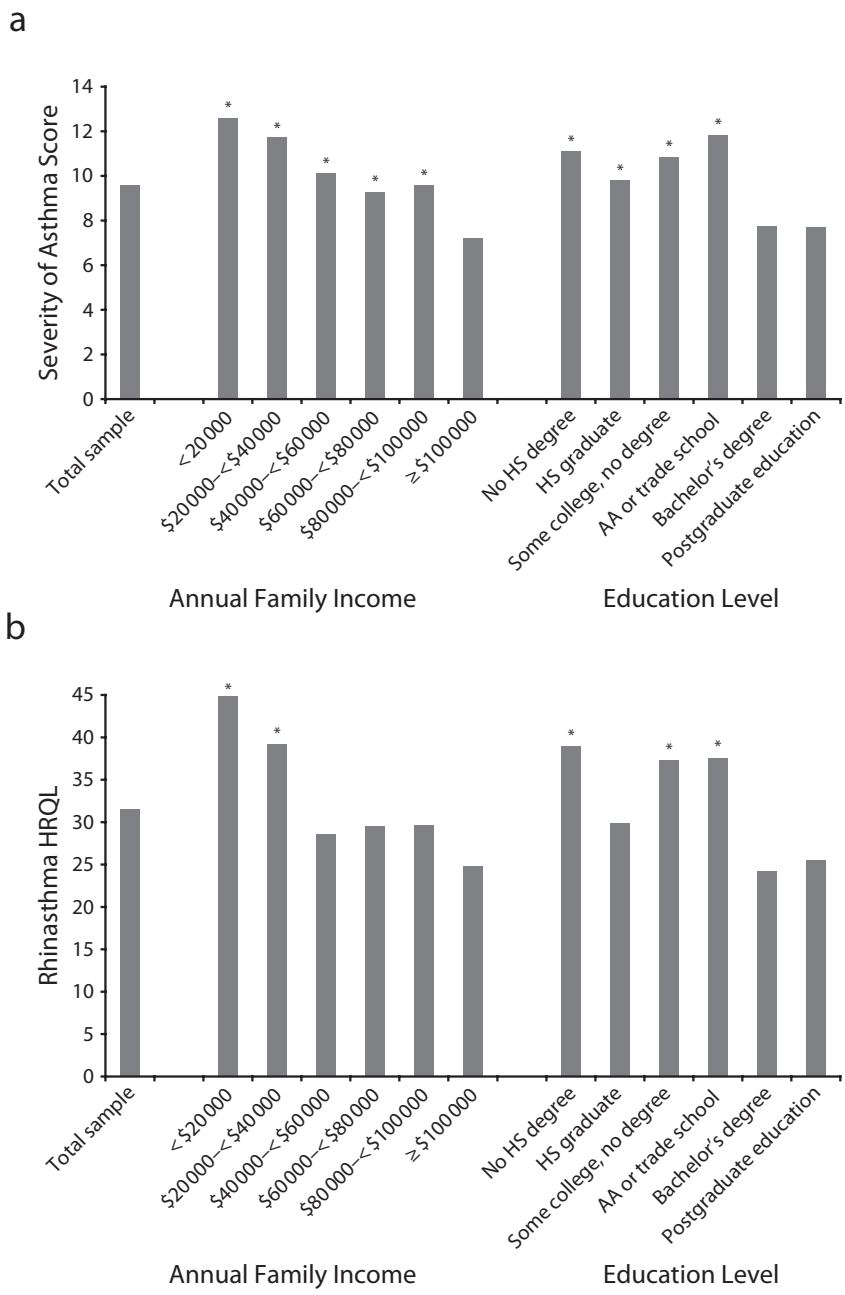
We undertook several sensitivity analyses in the current study. First, we anticipated that the relationship between home type or ownership and disease outcomes would be influenced by different types of housing available in urban compared with rural areas, which in turn might influence the type and degree of pollutant exposure. We therefore investigated the relationship between home type or ownership and residence in rural versus urban areas, using residential zip code to categorize participants according to Rural Urban Commuting Area codes.[48] Second, we added a variable for child- versus adult-onset disease to the final models to investigate potential differences in the effect of long-standing (childhood-onset) disease on the SES–environment–disease outcomes relationships. Lastly, we limited the sample to include only currently employed individuals, to assess the potential mediating effects of occupational exposures. We conducted analyses in SAS version 9.2 (SAS Institute, Cary, NC) and Stata/SE version 12 (StataCorpLP, College Station, TX).

RESULTS

Study participants ranged from 25 to 67 years of age; 75% were women. The sample was racially and ethnically diverse (Table 1). There was a substantial representation of participants at the lower end of the income distribution (28% reported annual household incomes < $40 000), and although only 17% reported no postsecondary education, more than 1 in 4 participants attended some college but had not obtained a bachelor's degree. Nearly 60% of participants were currently employed, with the remainder divided between those unable to work (19%) and not working for other reasons (22%), including seeking employment, retired, attending school, or keeping house. Childhood-onset disease was common, with 39% of participants diagnosed before age 18 years. Most participants (80%) had asthma with concomitant rhinitis; 4% reported rhinitis alone and 16% asthma only.

In the full sample, the mean ±SD severity of asthma score was 9.6 ±6.1 (higher scores indicate greater severity). The mean ±SD rhinasthma HRQL score was 31.4 ±21.4, (higher scores indicate poorer HRQL). As expected, participants with rhinitis alone had severity scores (mean ±SD = 1.9 ±1.2) much lower than those with asthma alone (10.5 ±6.0) or with both conditions (9.8 ±6.0). By contrast, participants with asthma and concomitant rhinitis had higher HRQL scores (34.5 ±21.4) than those with either asthma alone (19.6 ±16.8) or rhinitis alone (18.0 ±16.3).

Figure 1 shows the mean asthma severity and rhinasthma HRQL scores by income and



Note. AA = associate degree; HRQL = health-related quality of life; HS = high school. Mean ±SD severity of asthma score = 9.6 ±6.1. Higher scores indicate greater severity. Mean rhinasthma HRQL score = 31.4 ±21.4. Higher scores indicate poorer HRQL.
*$P < .05$ for comparison with highest level of income or education.

FIGURE 1—Mean scores at each level of family income and education for (a) severity of asthma and (b) rhinasthma HRQL: University of California, San Francisco Asthma Rhinitis Cohort, 2008–2009.

education gradients. Overall, greater severity and worse HRQL were associated with lower SES, whether measured by income or education ($P < .001$). However, the patterns of effect differed for the 2 SES measures and both outcomes. For asthma severity (Figure 1a), a monotonic gradient was evident, in which higher levels of income were associated with reduced severity. By contrast, there was an apparent threshold for SES defined by education, in that participants without a 4-year college degree had similarly elevated severity (ranging from 9.8 to 11.8) compared with college graduates and higher education levels (mean scores = 7.7 in both groups). For HRQL (Figure 1b), there was an apparent threshold for both SES measures, although participants with high school degrees had distinctly lower scores (that is, better HRQL) than did groups with either more or less education.

Lower income was associated with home type and ownership, heating with gas stove or space heater, tobacco smoke exposure, and the summary measure of neighborhood problems including traffic, trash, odors, fire smoke, noise, and violence (Table 2). Lower education was similarly associated with these variables, as well as with having a carpeted bedroom.

Annual family income remained associated with severity and HRQL after we adjusted for the potential mediator variables, although the magnitude of the SES effect was reduced at each level of income, particularly in the HRQL model (Table 3). These multivariable models explained 15% and 16% of the variation in severity and HRQL, respectively. Living in a rented single-family home was associated with an increase of 3.6 points in the severity of asthma score and 6.5 points in the rhinasthma score (indicative of poorer health status compared with those in owner-occupied residences). Furthermore, the Sobel test indicated that this variable acts as a partial mediator of the SES gradient on asthma severity ($P < .01$) and HRQL ($P = .01$). The neighborhood-level exposure score was associated with HRQL (but not severity) and also appeared to be a significant mediator of the income–HRQL relationship (Sobel $P = .01$). Secondhand smoke exposure, although independently associated with disease severity, was not a significant mediator tested in the same manner.

The pattern of effects for the income-based SES gradient models, described previously, is very nearly replicated in the education-based SES gradient models (Table 3). Once again, the magnitude of the parameters for each level of education was reduced for both outcomes. The shape of the distribution manifest in Figure 1 remains, however, with high-school graduates having comparable scores to those

with the highest levels of education. As with the income analysis, living in a rented single-family home and exposure to secondhand smoke were associated with greater disease severity, and living in a rented single-family home and a higher neighborhood assessment score predicted poorer HRQL. Home type was a highly significant partial mediator of the association between education and both outcomes. Secondhand smoke exposure and neighborhood assessment were less robust mediators of disease severity and HRQL, respectively ($P = .06$). The analyses of the combined income–education variable were consistent with the results for income and education considered separately (data not shown).

To further explore the relationship between living in a rented single-family home and both outcomes, we investigated the extent to which these participants lived in more rural areas, where such living situations may be more common and which might alter the risk profile for asthma and rhinitis outcomes. There was no statistically significant difference in the proportions of rural and urban residents living in rented single-family homes (12.5% and 14.5%, respectively; $P = .67$), nor did inclusion of that variable change the relationships seen in Table 3 (data not shown). Similarly, an investigation of the impact of childhood- versus adult-onset disease indicated that age of diagnosis was not associated with either measure of SES, nor did including this in the final models have any appreciable effect on the relationships of interest (data not shown).

In an analysis of the 301 currently employed participants, those with lower levels of education were more likely to report exposure to VGDF at work, but there was no statistically significant gradient by income. Exposure to VGDF itself was not associated with either asthma severity or HRQL. Rerunning the models described previously including only currently employed participants reduced the magnitude of the income effect, but the pattern was similar. In multivariable models, VGDF exposure was not associated with either severity or HRQL (data not shown).

TABLE 2—Relationships Between Exposure Variables and Socioeconomic Measures: University of California, San Francisco Asthma Rhinitis Cohort, 2008–2009

		Annual Family Income			Education		
Exposures	Total Sample (n = 515), No. (%) or Mean ±SD	< $40 000 (n = 144), No. (%) or Mean ±SD	≥ $40 000 (n = 371), No. (%) or Mean ±SD	*P*	< Bachelor's Degree (n = 309), No. (%) or Mean ±SD	≥ Bachelor's Degree (n = 206), No. (%) or Mean ±SD	*P*
Indoor environment							
Home type and ownership				< .01[a]			< .01[a]
Mobile home	18 (3)	12 (8)	6 (2)		17 (6)	1 (0.5)	
Apartment, rented	65 (13)	35 (24)	30 (8)		45 (15)	20 (10)	
Single family home, rented	70 (14)	39 (27)	31 (8)		57 (18)	13 (6)	
Home or apartment, owned	362 (70)	58 (40)	304 (82)		190 (61)	172 (83)	
Carpet in bedroom	385 (75)	113 (78)	272 (73)	.23	240 (78)	145 (70)	.06[a]
Gas heat (space heater or stove)	55 (11)	26 (18)	29 (8)	< .01[a]	40 (13)	15 (7)	.04[a]
Wood-burning heat (≥ 5 d/wk)	46 (9)	12 (8)	34 (9)	.77	26 (8)	20 (10)	.61
Mold and water damage	51 (10)	15 (10)	36 (10)	.81	30 (10)	21 (10)	.86
Have pet, despite MD advice	87 (17)	17 (12)	70 (19)	.05	48 (16)	39 (19)	.31
Smoke exposure				< .01[a]			< .01[a]
Active smoking	50 (10)	25 (17)	25 (7)		44 (14)	6 (3)	
Secondhand smoke only	49 (10)	20 (14)	29 (8)		39 (13)	10 (5)	
None	416 (81)	99 (69)	317 (85)		226 (73)	190 (92)	
Neighborhood-level exposures							
Any problems with:							
Traffic	249 (48)	74 (51)	175 (47)		157 (51)	92 (45)	
Trash	126 (24)	46 (32)	80 (22)		72 (23)	54 (26)	
Odors	121 (23)	41 (28)	80 (22)		81 (26)	40 (19)	
Smoke from fires	232 (45)	69 (48)	163 (44)		145 (47)	87 (42)	
Noise	241 (47)	75 (52)	166 (45)		156 (50)	85 (41)	
Violence	129 (25)	49 (34)	80 (22)		82 (27)	47 (23)	
Summary score[b]	0.8 ±0.9	1.1 ±1.1	0.7 ±0.8	< .01[a]	0.9 ±0.9	0.7 ±0.8	< .01[a]

[a]Associated with lower socioeconomic status ($P < .1$).
[b]Neighborhood problems rated on a scale of 0 (no problem) to 5 (severe problem). Cronbach's α on 6 items = 0.76.

TABLE 3—Association of Severity and Health-Related Quality of Life With Socioeconomic Status, With and Without Adjustment for Potential Mediators: University of California, San Francisco Asthma Rhinitis Cohort, 2008–2009

Models and Covariates	Severity of Asthma Score			Rhinasthma HRQL		
	b (SE)	*P*	$P_{(Sobel)}$[a]	b (SE)	*P*	$P_{(Sobel)}$[a]
Income model (unadjusted)						
Annual family income, $ (Ref: ≥ 100 000)						
< 20 000	5.39 (0.83)	< .01		20.04 (1.81)	< .01	
20 000 to < 40 000	4.52 (0.26)	< .01		14.33 (1.99)	< .01	
40 000 to < 60 000	2.95 (0.94)	< .01		3.73 (0.85)	< .01	
60 000 to < 80 000	2.08 (0.78)	< .01		4.71 (1.76)	< .01	
80 000 to < 100 000	2.37 (0.38)	< .01		4.84 (2.15)	.03	
Income model (adjusted)[b]						
Annual family income, $ (Ref: ≥ 100 000)						
< 20 000	3.25 (1.10)	< .01		13.41 (2.53)	< .01	
20 000 to < 40 000	3.41 (0.18)	< .01		10.11 (2.50)	< .01	
40 000 to < 60 000	2.43 (0.87)	< .01		1.65 (1.19)	.17	
60 000 to < 80 000	1.30 (0.85)	.13		2.00 (2.41)	.41	
80 000 to < 100 000	1.95 (0.25)	< .01		3.06 (1.96)	.12	
Type of home (Ref: owner occupied)						
Mobile home	−1.17 (0.93)	.21		−3.26 (2.58)	.21	
Apartment, rented	1.01 (0.53)	.06		2.04 (1.34)	.13	
Single family, rented	3.60 (0.72)	< .01	< .01	6.52 (3.21)	.04	.01
Carpet in bedroom	0.24 (0.46)	.61		0.38 (2.03)	.85	
Gas heat (kitchen stove or space heater)	0.40 (1.77)	.83		0.30 (5.44)	.95	
Smoke exposure (Ref: none)						
Active smoking	−0.62 (1.01)	.54		−2.12 (2.12)	.32	
Secondhand smoke only	2.65 (0.39)	< .01	.25	3.78 (4.39)	.39	
Neighborhood-level exposures (0–5 score)	0.33 (0.20)	.1		4.73 (1.02)	< .01	.01
Education model (unadjusted)						
Education (Ref: postgraduate education)						
No high school degree	3.40 (1.89)	.07		13.46 (5.57)	.02	
High school graduate	2.12 (0.80)	< .01		4.31 (3.88)	.27	
Some college	3.16 (0.97)	< .01		11.72 (2.13)	< .01	
Associate degree or trade school	4.13 (1.23)	< .01		11.98 (1.79)	< .01	
Bachelor's degree	0.05 (0.38)	.9		−1.34 (3.30)	.69	
Education model (adjusted)[c]						
Education (Ref: postgraduate education)						
No high school degree	1.49 (1.76)	.4		7.07 (4.46)	.11	
High school graduate	1.24 (0.71)	.08		1.91 (3.77)	.61	
Some college	2.14 (0.52)	< .01		8.39 (1.81)	< .01	
Associate degree or trade school	3.29 (0.93)	< .01		10.08 (2.56)	< .01	
Bachelor's degree	−0.07 (0.17)	.67		−1.46 (3.18)	.65	
Type of home (Ref: owner occupied)						
Mobile home	−0.08 (0.80)	.92		1.02 (2.88)	.73	
Apartment, rented	1.86 (0.85)	.03		4.90 (1.64)	< .01	
Single family, rented	4.11 (0.50)	< .01	< .01	8.92 (2.17)	< .01	.01

Continued

DISCUSSION

We observed SES gradients in adult asthma and rhinitis disease severity and HRQL. This relationship was nuanced: the income-based SES gradient was monotonic; for education, it was more bimodal. The type and ownership of one's dwelling (specifically, single-family rented home), was associated with more severe disease and worse HRQL, and partially mediated the SES relationship to these outcomes. This relationship was not explained by an urban–rural gradient. Perceived neighborhood-level exposure was significantly associated with HRQL (although not severity), also partially mediating the SES gradient for HRQL.

The mediation we observed supports our study hypothesis that the SES gradients in asthma and rhinitis outcomes may work partially through environmental exposures. Some of the exposures previously identified as risk factors for poor outcomes in asthma and rhinitis do not appear to mediate the SES–outcomes relationship in this study. It is notable that the exposures subsuming multiple environmental factors, (i.e., home rental and neighborhood condition) were identified as mediators in this analysis, whereas the more narrowly cast exposure measures (such as carpeting or heating with gas stove or space heater) were not. Other researchers[32] have argued that the SES gradient in health outcomes is mediated by the conjoint effect of multiple adverse environmental exposures. Our findings support this view.

The present analysis does have potential limitations. This survey-based study relies on self-reported data, not direct exposure measurements. Thus, we tested the relationship of a carpeted bedroom, not dust mite concentrations, to severity and HRQL. Similarly, we used reported mildew and water stains as evidence for potential mold, not spore count. It is likely that nonsystematic misclassification of exposure results from this approach, which could be enough to account for a lack of observed associations. By contrast, systematic reporting bias could result in an overestimation of the relationship between neighborhood conditions and HRQL, if those with poorer HRQL tended to overreport adverse neighborhood conditions.

A potential limitation to our approach is consideration of income and education, but not

TABLE 3—*Continued*

Carpet in bedroom	0.29 (0.44)	.5		0.29 (2.07)	.89	
Gas heat (kitchen stove or space heater)	0.43 (1.50)	.78		0.61 (5.00)	.91	
Smoke exposure (Ref: none)						
Active smoking	−0.61 (1.00)	.54		−2.16 (1.69)	.21	
Secondhand smoke only	2.48 (0.05)	< .01	.06	3.42 (2.63)	.19	
Neighborhood-level exposures (0–5 score)	0.45 (0.24)	.06		4.96 (1.00)	< .01	.06

Note. HRQL = health-related quality of life. Multiple linear regression models included age, gender, and all variables shown in tables. The sample size was n = 515.
[a]Sobel tests for mediation conducted only on exposure variables that were significant ($P < .05$) in these models.
[b]Model R^2 for severity of asthma score = 0.15; model R^2 for rhinasthma HRQL score = 0.16.
[c]Model R^2 for severity of asthma score = 0.16; model R^2 for rhinasthma HRQL score = 0.17.

other measures of SES, such as wealth or assets. These measures, however, would have overlapped with a key measure of interest, home residential status, which we view as an environmental exposure surrogate. Another limitation we faced was that of unmeasured explanatory variables. For this reason, we believe our findings should be viewed in the context of highlighting pathways for further analyses, in particular through longitudinal study. Such mediating factors could include social stressors, access to care, ambient air quality, early life exposures to irritants or allergens, and previous occupational exposures (especially among those not currently working). The poorer outcomes seen in the group with vocational and associate degrees may be attributable to previous workplace exposures.

Although the study uses an established cohort, the full set of variables included in the present analysis is only available cross-sectionally. This raises the possibility of reverse causality (i.e., lower SES caused by disease, rather than vice versa). For this reason, we used education as an alternate indicator of SES, as it is more stable over the adult lifetime. It is less likely that severe asthma would result in lower educational attainment than in lower current income. The relative similarity of results for education and income is consistent with the direction of effect leading from SES to adverse disease outcomes and not the reverse, a finding observed in the general literature on SES and health as well.[49] Nonetheless, we cannot completely exclude the possibility that underlying, unmeasured factors could explain the observed associations among SES, current environmental exposures, and disease outcomes. Longitudinal analyses can more fully address some of these outstanding questions.

Despite the limitations noted, this study represents a novel exploration of the potential mechanisms underlying the SES gradient in adult asthma and rhinitis. To build on our findings, further delineation of the partial mediation of the SES gradient that we observed should include additional measures quantifying environmental exposures. In particular, such investigation should focus on personal-level exposures linked to housing type and neighborhood-level exposures represented by the perceived factors studied here. ■

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Contributors

L. Trupin led the analysis, carried out all statistical analyses, and drafted the article. P. P. Katz, J. R. Balmes, H. Chen, E. H. Yelin, and T. Omachi participated in the design of the study and the interpretation of the results, and helped to draft the article. P. D. Blanc conceived of the study, helped to draft the article, and directed and designed the project that led to the present analysis. All authors read and approved the final article.

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Human Participant Protection

The protocol for this study was approved by the University of California San Francisco Committee on Human Research and all participants gave their written informed consent.

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