Published in final edited form as:

J Epidemiol Community Health. 2011 October ; 65(10): 859–865. doi:10.1136/jech.2009.102806.

Social environment and asthma: Associations with crime and No Child Left Behind programs

Ketan Shankardass^{1,2}, Michael Jerrett³, Joel Milam², Jean Richardson², Kiros Berhane², and Rob McConnell²

¹Centre for Research on Inner City Health, St. Michael's Hospital, Toronto, Ontario, Canada

²Department of Preventive Medicine, Keck School of Medicine, University of Southern California, Los Angeles, California, United States

³School of Public Health, Division of Environmental Health Science, University of California, Berkeley, California, United States

Abstract

Background—The relationship between asthma and socioeconomic status remains unclear. We investigated how neighborhood, school and community social environments were associated with incident asthma in Southern California school children.

Methods—New onset asthma was measured over three years of follow-up in the Children's Health Study cohort. Multilevel random effects models assessed associations between social environments and asthma, adjusted for individual risk factors. Subjects resided in 274 neighborhoods and attended one of 45 schools in 13 communities. Neighborhoods and communities were characterized by measures of deprivation, income inequality and racial segregation. Communities were further described by crime rates. Information on schools included whether a school received funding related to the Title 1 No Child Left Behind program, which aims to reduce academic underachievement in disadvantaged populations.

Results—Increased risk for asthma was observed in subjects attending schools receiving Title I funds compared to those from schools without funding (adjusted hazard ratio 1.71, 95% CI 1.14–2.58), and residing in communities with higher rates of larceny crime (adjusted hazard ratio 2.02, 95% CI 1.08–3.02 across the range of 1827 incidents per 100,000 population).

Conclusions—Risk for asthma was higher in areas of low socioeconomic status, possibly due to unmeasured risk factors or chronic stress.

Corresponding author: Dr. Rob McConnell, Telephone: (323) 442-1593, Fax: (323) 442-3272, rmcconne@usc.edu.

COMPETING INTERESTS

None of the authors have any competing interests to declare.

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Keywords

asthma; multilevel models; socio-economic; air pollution

BACKGROUND

Higher rates of childhood asthma are often reported in areas of low socioeconomic status (SES).[1–4] Socioeconomic and spatial disparities in asthma are not fully explained by low individual SES or related risk factors, [5–9] and risk for asthma onset is likely influenced by complex relationships with social, economic and other conditions in the wider environment. [10] As a result, there is growing interest in the relationship between the social environment and asthma.[11, 12]

The social environment might be related to the development of asthma by influencing behaviors that lead to asthma (e.g. *in utero* tobacco smoke exposure), exposure to hazards of the physical environment (e.g. air pollution or indoor allergens), ability to access medical care, and by its effect on psychosocial stress.[3, 10, 12] These relationships may be reflected by four broad and overlapping types of low SES environments: high deprivation (i.e. a lack of economic wealth in an area), [5, 6, 8, 9, 13, 14] high income inequality (i.e. unevenness in the distribution of wealth within a population), [15, 16] high segregation (i.e. racial or ethnic separation in daily life), [17, 18] and low social capital (i.e. the structure and quality of social networks in an area does not result in mutual benefits).[9, 10, 19, 20]

Theoretically, measured associations between the social environment and childhood asthma onset may reflect causal pathways involving a complex mixture of *contextual effects* that have biological impact (e.g. community characteristics such as high crime that induce chronic stress) and *compositional effects* that are mediated by individual-level risk factors in the study population (e.g. smoking in the home).[21] Further, risk factors and biological processes related to childhood asthma onset could be determined by several aspects of one's social context. In particular, outside of their homes, children spend the bulk of their time in their residential neighborhood, at school or in the community at-large.[11] Multilevel models are useful for studying associations with the social environment because they can simultaneously adjust for environmental characteristics across multiple spatial scales, [5–9] while also adjusting for risk factors and “similarity by proximity” due to spatial clustering at the individual-level.[22]

In this paper, we examine contextual effects of the neighborhood, school and community social environment for new onset asthma, after accounting for a range of individual and household risk factors using multilevel models, in a prospective cohort of school children in Southern California.

METHODS

Study population

We selected 2497 subjects with no wheeze or asthma at baseline enrollment (2002–2003) from the Children's Health Study (CHS) cohort, which has been previously described.[23,

24] Forty-one additional subjects (1.6%) were excluded from the previous data set examining effects of traffic pollution effects, because traffic density on roads near participant residences were not available for dispersion modeled pollution exposure estimates (total oxides of nitrogen) used in the current analysis. Traffic-related pollution has been previously identified as an important risk factor for new onset asthma in this cohort, [25] and it may help explain geographic differences in the onset of asthma (see below). Therefore, the study population for the final analysis included 2456 subjects. Subjects resided in 274 census tracts (i.e. neighborhoods) and attended kindergarten or first grade in one of 45 schools distributed in 13 communities throughout Southern California (Figure 1). Informed consent was obtained from parents, and the study was approved by the University of Southern California Institutional Review Board.

Assessment of new onset asthma and covariates

Subjects with new onset asthma were identified using physician-diagnosed asthma as reported by one of the child's parents or guardians on annual questionnaires during three years of follow-up. Risk factors that might explain observed associations with measures of the social environment were assessed from responses given by parents or guardians on the study baseline questionnaire. Also, recent evidence from this cohort suggests that exposure to traffic-related pollution near the home and school may cause asthma.[25] Because high minority and low SES environments have been associated with higher exposure to air pollution at homes and schools in Southern California, [18, 26] we examined exposure to traffic-related pollution as a potential confounder. Figure 2 contains a full list of covariates considered in this analysis at the individual level, and a description of these variables can be found in the Online Supplement.

Measurement of the social environment

Figure 2 contains an overview of the hierarchical relationship between individual subjects and the three spatial levels of interest: neighborhoods, schools and communities. Data describing a range of social characteristics were available at each spatial level.

Neighborhood characteristics were defined by matching the census tract of residence to data from the U.S. 2000 Census. Characteristics included measures of deprivation, such as median household income, proportion of respondents over age 25 with low and high education (e.g. percent with no high school diploma), percent unemployed and percent living in poverty. Measures of community racial composition (e.g. percent African American) were also included, as they may be associated with the presence or absence of social capital, deprivation or segregation in an area. We also used the Gini coefficient to describe income inequality within neighbourhoods.[27] This commonly used measure of inequality can range from 0 to 1.0, with higher values indicating greater income inequality among neighborhood households. Finally, we examined population density as an indicator of unmeasured risk factors in more urban environments.

Schools were characterized by data reported to the California Department of Education for the 2002–2003 school year that describe the social environment.[28] The percent of students receiving free meals was used as an indicator of deprivation among students. Variables such

as the percent of students with minority ethnicity, the percent of students who were English-language learners, an indicator for whether or not the majority of students were of Hispanic ethnicity, and the Ethnic Diversity Index may uniquely reflect concepts related to social capital, segregation and deprivation. In particular, the Ethnic Diversity Index measured how evenly students were distributed among seven ethnic categories reported to the Department.

We also included variables describing school-level academic performance, which may reflect an aspect of social capital (e.g. social order, stress) that is unique to children. These included the Academic Performance Index, whether the school met academic “adequate yearly progress” criteria in the previous year, and a variable that described whether or not schools received funding related to Title I (‘Improving the Academic Achievement of the Disadvantaged’) under the No Child Left Behind Act of 2001. The purpose of this funding is to ensure that all children have access to high-quality education and achieve proficiency on State academic standards. Two types of funding are available to support academic improvement: Schools with >40% of student living in poverty are eligible to combine funding from Title I with other federal, state and local funds to implement school-wide programs aiming to improve conditions for all students, while schools with lower levels of poverty can apply for funding to support specific students of concern. Therefore, this measure may represent both academic performance and deprivation at schools.

The community social environment was estimated by similar variables used to describe neighborhoods (except for the Gini coefficient), based on data describing census block groups that were aggregated to the community level using a previously described method. [29] Also, we calculated two measures of the dissimilarity index to describe residential segregation of African Americans and Hispanics, respectively, compared with all other races.[30] The dissimilarity index ranges from 0 to 100 and can be interpreted as the percent of minority residents within the community who would need to move from their census block group to another to achieve even distribution across census block groups within the community.

Data from Federal Bureau of Investigation Uniform Crime Reports were used to characterize crime in the community, [31] which may be a marker for high relative deprivation or low social capital in communities, [32] and stress related to exposure to violence at the individual level.[10, 33] These data are reported annually by city, county and state law enforcement agencies, and describe rates of various types of violent (e.g. murder, rape, robbery and aggravated assault) and property (e.g. burglary, larceny, motor vehicle theft) crime. Effects related to crime were found to be primarily driven by the larceny crime rate, which was by far the most prevalent type of crime, e.g., the mean larceny crime rate (1908 per 100,000) was similar to the mean rate for all other types of crime combined (1904 per 100,000). Therefore, in this analysis we focus on results related to larceny crime, which is defined as theft involving the taking and carrying of personal property. Because the recruitment of study subjects was not completed until partway through 2003, data for 2004 were used in this analysis to reflect crime occurring near study baseline that may have affected all subjects in the cohort.

Statistical methods

Contextual effects for asthma onset were assessed using multilevel Cox proportional hazards models.[34] All models contained age and gender stratification of the baseline hazard, adjustment for race and ethnicity, and a 2-level independent random effects structure, which allowed for clustering around schools and communities, and assessment of residual variation in time to asthma onset. Random effects for neighbourhoods, rather than schools, were included in a sensitivity analysis, which resulted in a similar pattern of effects; therefore, only models featuring a school-community random effects structure are presented. Letting u_i and u_{ij} denote community and school (within community) level random effects, the model takes the following general form:

$$h_{ijl}(t) = h_{0s}(t) u_i u_{ij} \exp(\beta X_{ijl} + \delta^T Z_{ijl})$$

$h_{ijl}(t)$: hazard function for the l th subject in the i th community, and j th school;

$h_{0s}(t)$: the baseline hazard function for stratum s (i.e., age at study entry and gender);

X_{ijl} : characteristics of the social environment for the l th subject in the i th community, and j th school; and

Z_{ijl} : covariates (e.g., race and ethnicity, traffic-related pollution) for the l th subject in the i th community, and j th school.

In this model, the school-level random effects are assumed to be positive and independent conditional on the community level random effects (see Hughes, 2007 for details).[35] Analogous models could be developed for neighborhood level random effects. All analyses were conducted using R software [36] and software designed to run within R for implementing random effects Cox proportional hazards models.[34, 37]

Variables describing the neighborhood, school and community social environment were initially tested as main effects on time to incident asthma. All variables which were statistically significant at an alpha level of 0.10 were then systematically co-adjusted to determine whether these variables had independent effects on incident asthma. To determine the extent to which effects of the social environment may be attributable to compositional rather than contextual pathways, the model containing all statistically significant, independent effects of the social environment was adjusted for hypothesized individual-level risk factors for asthma (Figure 2). We further adjusted the final contextual effects model for residential and school traffic-related pollution and exposure to wildfires in 2003 during follow-up to examine potential confounding.[38] Indicators of missing data for individual-level risk factors were also included in models adjusted for these covariates to allow all 2456 subjects into these models.[39]

RESULTS

The characteristics of subjects in this cohort have been described previously. [40] There were 118 cases of new onset asthma resulting in a crude incidence rate of 1.9 per 100 person-years. Subjects ranged from 5–9 years of age at baseline and 47.7% were male. The majority of subjects were of either Hispanic ethnicity (55.3%) or (non-Hispanic) white race

(36.2%), and the remainder were either African-American (3.1%) or of other (5.4%) race/ethnicity. Low parental education was reported for 21.2% of subjects, while the majority of subjects (55.1%) were of medium parental education, and 23.7% were of high parental education. Individual level risk factors for asthma have been previously examined, [25, 41] and effects mostly took the expected sign. Significant positive associations were found for African-American race, underweight, parental history of asthma, musty odor in the home and residential traffic-related pollution.

Wide variation existed in the distribution of characteristics describing neighborhood, school and community social environments (Table 1). Students attending schools that received any Title I funding were 1.66 times more likely to be diagnosed with asthma during follow-up (95% CI 1.09–2.52). A likelihood ratio test comparing a model which distinguished type of Title I funding, i.e., targeted assistance program versus school-wide program, to one which did not was non-significant ($p=0.59$), so we focused on the overall effect of Title I funding for parsimony. The community larceny crime rate was also positively associated with asthma (HR 1.23, 95% CI 1.02–1.48, across the inter-quartile range of 570 per 100,000), and there was a clear dose-response relationship with the crude asthma rate across the 13 study communities (Figure 3).

The school Ethnic Diversity Index and neighborhood population density initially had borderline positive significant associations with asthma (HR for Ethnic Diversity Index 1.31, 95% CI 1.01–1.70 across the inter-quartile range of 17; HR for population density 1.19, 95% CI 1.00–1.43 across the inter-quartile range of 2340 per sq km); however, when these variables were simultaneously co-adjusted for Title I funding and the larceny crime rate, their effects were greatly reduced and became non-significant (see co-adjusted HR in Table 1; HR for Ethnic Diversity Index reduced by 26%, $p=0.14$; HR for population density reduced by 35%, $p=0.26$). On the other hand, effects for Title I funding and the larceny crime rate remained largely unchanged in the co-adjusted model. Collinearity was not a concern in co-adjusted models because the Pearson correlation coefficients between these variables were generally low (i.e. below 0.3).

When co-adjusted in a single model, effects for the larceny rate and Title I funding remained significant and increased in size (see Model 1 in Table 2). Effects for the larceny crime rate and Title I funding were robust to adjustment for hypothesized risk factors for asthma describing individual characteristics and the indoor household environment. This was true when we ran a series of tests adjusting individually for each potential confounder (data not shown), and when all of these variables were simultaneously entered into the model (see Model 2a in Table 2).[39] Exposure to wildfires also did not confound these contextual effects.

Residential traffic-related pollution had a weak positive correlation with the larceny crime rate ($R=0.24$, $p<0.001$), and a weaker positive correlation with Title I funding ($R=0.09$, $p<0.001$). When the model containing the larceny crime rate and Title I funding was adjusted for traffic-related pollution, the effect of the larceny rate was reduced by 20% but it remained significant (HR 1.25, 95% CI 1.02–1.52; Model 2b in Table 2). There was a 25% increase in risk for incident asthma across the inter-quartile range and a 202% increase in

risk across the full range (1827 per 100,000; data not shown). In this model, effects for school Title I funding (HR 1.64, 95% CI 1.08–2.47) and traffic-related pollution (HR 1.44, 95% CI 1.19–1.76) were relatively stable compared with their unadjusted values. Adjustment for exposure to traffic-related pollution at school, rather than the household, resulted in a similar but weaker pattern of confounding (data not shown).

DISCUSSION

We used a multilevel model to evaluate effects of the social environment on asthma incidence in a cohort of Southern California school children. Subjects living in communities with high larceny crime rates, or attending schools receiving Title I funds, had increased risk for new onset asthma compared with subjects living in communities with lower larceny crime rates, or attending schools without Title I funding, respectively. These contextual effects were independent of one another and not explained by a wide range of individual and household characteristics, including SES (i.e. highest educational attainment in parent), race and ethnicity, indicators of indoor allergens, second hand and *in utero* tobacco smoke, BMI, medical insurance coverage and parental stress, or by residential exposure to traffic-related pollution.

Community crime rates and school Title I funding may both be markers of areas with high deprivation. However, SES did not explain increased risk for asthma at either the individual (i.e. parental education) or population levels (e.g. percent poverty in communities, percent free meals in schools). Thus, there may be physical characteristics correlated with these variables that are poorly measured in households and schools that may be risk factors for asthma. For example, Title I schools may be older or more poorly maintained and thus have higher concentrations of indoor allergens (e.g. mold secondary to water damage) that cause respiratory infections.[42, 43] Also, parents in areas of high crime could be more likely to keep their children indoors out of concern for their safety, thus increasing exposure to indoor environmental toxins.[44] While we did not have school-level assessments of the indoor physical environment, we did examine some of these characteristics at the household level. In a sensitivity analysis, we further adjusted Model 2B (in Table 2) for the time spent outdoors per week by subjects (as reported by their parents) and the contextual effect were unchanged (results not shown).

Communities with higher rates of larceny crime and schools with Title I funding may be more stressful for children in Southern California. Exposure to violence and perception of neighbourhood safety have been associated with increased asthma morbidity and higher risk for lifetime and new onset asthma in other settings, [19, 20, 45–49] and crime in the community can lead to post-traumatic stress in residents.[3, 10, 33, 50] High crime and Title I funding may also be a proxy for areas with low social capital, where children are less able to rely on social support to cope with stressors.[51, 52] Finally, because schools apply for Title I funds to support academic improvement, students attending these schools may be more likely to experience stress related to academic difficulties.[53, 54] In addition to possible direct effects on the development of asthma, accumulating evidence suggests that chronic stress may increase vulnerability to environmental exposures associated with asthma.[19, 55] For example, increased inflammation of the airways in college students with

asthma exposed experimentally to allergens has been associated with stressful examination periods, [56] and we previously showed that children in this cohort with higher levels of parental stress were more susceptible to the effects of traffic-related pollution on new onset asthma [41]. We adjusted contextual effects in this analysis for parental stress, traffic-related pollution and for interactions between these two variables, and saw little evidence that stress was mediating contextual effects on asthma (data not shown). However, while parental stress may be a good proxy for stress experienced by subjects in the household, it is unlikely to capture variation in stress among children attributable to, for example, the school environment or exposure to crime in the community.

These results suggest that low SES environments that are also stressful may increase the risk for new onset asthma. This is supported by some multilevel studies of low SES environments, [6, 7, 9, 14] while others suggest the opposite relationship.[5, 8] Contradictory results may partly reflect differences in methodological approach across studies.[57] Previous analyses have been primarily cross-sectional, while our study uses a prospective design. Because asthma in children may cause stress for the parents, cross-sectional observations may be problematic. Also, whereas past studies have examined effects related to a single spatial level, we simultaneously examined characteristics across three spatial levels and show that multiple levels of influence may affect risk for asthma. For example, because effects for both Title I funding and the larceny crime rate appeared to be biased towards the null in unadjusted models compared with co-adjusted effects (see change in estimates from Table 1 to Model 1 in Table 2), previously reported contextual effects may be confounded by effects at other spatial levels.

Although population disparities in asthma have been found using census-based measures to characterize residential areas, [7–9] some argue that such arbitrary boundaries may not capture meaningful variation in health outcomes.[29] While associations between census-based measures of neighborhoods and asthma generally took the expected direction in our study, we found significant effects using variables not based on census data. Because children spend around a third of their waking hours at school, the role of the school social environment in childhood asthma warrants further examination. In general we observed elevated risks for many school characteristics that were larger than effects from other contextual levels. Further, since the quality of the school social environment may be related to the residential areas they serve, effects related to residential and individual characteristics have the potential to be confounded by school variables.

We observed partial confounding of contextual effects by a measure of traffic-related pollution; more so than by individual or household characteristics. In study areas where exposure to traffic-related pollution is higher in low SES neighborhoods, previously reported contextual effects for residence in a low SES area may be positively biased, and future studies should attempt to control for exposure to such pollution.

To our knowledge, this is the first multilevel study to simultaneously examine the relationship between multiple levels of social environment and asthma incidence. Increased risk for incident asthma was associated with schools characterized by academic failure and poverty, and communities with high rates of larceny crime, and these effects were not

explained by a wide range of individual and household characteristics. These results indicate that further investigation of the school and community social environment could elucidate the role of risk factors in the physical environment or stress in mediating contextual effects and identify new avenues for disease prevention.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

FUNDING

This work was supported by the National Institute of Environmental Health Sciences [grant numbers 5R03ES014046-02, 1R01 ES016535, 5P01ES009581, 5P01ES011627, and 5P30ES007048]; the U.S. Environmental Protection Agency [grant numbers R831845, RD831861 and R826708]; the National Cancer Institute [grant number 1U54CA116848-01]; the Hastings Foundation; and the Canadian Institutes of Health Research.

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WHAT THIS PAPER ADDS

What is already known on this subject?

The etiology of childhood asthma and the specific role of socioeconomic status in causing asthma remain unclear. Examination of the relationship between the social environment and asthma may inform our understanding about why social disparities in asthma occur and provide support for plausible etiologic pathways.

What does this study add?

In this paper we found a higher risk for new onset asthma in under-achieving schools that serve low-income students and in communities with high rates of crime. A growing number of studies demonstrate a relationship between environmental violence and childhood asthma, which supports a body of evidence that stress can play a role in the development of asthma. Our results also motivate a closer examination of schools as potentially asthmagenic environments and as sites preventive intervention. Finally, exposure to traffic-related air pollution was identified as a partial mediator of social disparities in asthma, suggesting a need to integrate risk factors from the social and biophysical environments in future asthma research.

Figure 1.
Map of study communities

Figure 2.
Diagram of data hierarchy and covariates

Figure 3.
Scatterplot of community larceny crime rate by crude community asthma rate

Table 1
Characteristics of CHS neighborhoods, schools and communities, with bivariate and co-adjusted associations with incident asthma

Level	N	Variable	N (%)	Mean (Standard deviation)	Range (Interquartile range)	Bivariate HR (95% CI) ¹	Co-adjusted HR (95% CI) ¹
Neighborhood	274	Median income (\$)		48,287 (17,286)	13,506–106,596 (20,968)	0.84 (0.68–1.05)	
		Education below high school diploma (%)		25.7 (16.1)	3.7–68.6 (23.1)	1.08 (0.81–1.42)	
		Education at or above graduate degree (%)		6.9 (5.8)	0–27.6 (7.0)	0.96 (0.78–1.18)	
		Below poverty (%)		14.6 (10.8)	0.9–56.7 (12.5)	1.18 (0.96–1.46)	
		Total unemployment		7.4 (4.4)	0.9–23.4 (5.1)	1.16 (0.93–1.44)	
		White residents (%)		48.9 (23.9)	9.0–91.4 (39.6)	0.82 (0.58–1.15)	
		Hispanic residents (%)		36.4 (20.0)	5.9–89.8 (30.7)	1.13 (0.83–1.52)	
		African American residents (%)		6.6 (7.4)	1.9–54.5 (7.5)	1.17 (0.92–1.49)	
		Asian residents (%)		6.2 (5.8)	0.1–44.4 (5.5)	1.12 (0.89–1.40)	
		Other race/ethnicity residents (%)		1.9 (1.4)	5.4–17.3 (0.7)	0.96 (0.80–1.15)	
		Population density (per sq km)		2550 (2780)	5–19121 (2340)	1.19 (1.00–1.43) ³	1.12 (0.92–1.37)
		Gini coefficient		0.40 (0.05)	0.27–0.52 (0.06)	1.12 (0.85–1.47)	
		Title I funding (Yes/no)	27 (60.0)			1.66 (1.09–2.52) ⁴	1.62 (1.07–2.47) ⁴
School	45	Targeted assistance program (Yes/no) ²	14 (31.1)			1.77 (1.12–2.80) ⁴	
		School-wide program (Yes/no) ²	13 (28.9)			1.49 (0.89–2.52)	
		Free meals (% of students)		45.3 (28.8)	3.7–98.3 (48.0)	1.25 (0.89–1.75)	
		English learners (% of students)		20.7 (20.6)	1.3–78.9 (25.9)	1.03 (0.80–1.31)	
		Ethnic diversity index		34.4 (12.6)	4–58 (17.0)	1.31 (1.01–1.70) ⁴	1.22 (0.94–1.59)
		Minority ethnicity (% of students)		54.0 (26.2)	19.7–98.4 (54.0)	1.43 (0.92–2.21)	
		Majority of students are Hispanic ethnicity (Yes/no)	20 (44.4)			1.35 (0.90–2.02)	
		Academic performance index, base score		756.5 (73.9)	582–875 (100)	0.89 (0.69–1.15)	
		Academic performance index, state rank		6.3 (2.4)	1–10 (3)	0.88 (0.69–1.12)	
		School met “Adequate Yearly Progress” criteria (Yes/no)	36 (81.8)			0.91 (0.57–1.46)	
Community	13	Median income (\$)		46,127 (8,729)	29,891–56,690 (12,678)	1.05 (0.78–1.42)	
		Low education, below high school diploma (%)		23.4 (9.6)	9.0–37.4 (15.9)	1.00 (0.91–1.10)	
		High education, at or above graduate		5.7 (3.4)	2.1–13.7 (4.2)	1.06 (0.85–1.32)	

Level	N	Variable	N (%)	Mean (Standard deviation)	Range (Interquartile range)	Bivariate HR (95% CI) ¹	Co-adjusted HR (95% CI) ¹
		Below poverty (%)		15.6 (6.8)	8.2–31.2 (6.8)	1.04 (0.84–1.29)	
		Total unemployment		7.7 (2.3)	5.4–13.3 (7.4)	1.05 (0.85–1.29)	
		White residents (%)		49.6 (17.9)	23.5–83.5 (22.2)	0.85 (0.64–1.13)	
		Hispanic residents (%)		37.8 (14.3)	12.4–61.5 (14.2)	1.08 (0.87–1.34)	
		African American residents (%)		5.7 (5.3)	0.9–18.3 (4.7)	1.13 (0.94–1.35)	
		Asian residents (%)		5.2 (3.3)	1.0–13.2 (5.2)	1.17 (0.86–1.60)	
		Other race/ethnicity residents (%)		1.7 (0.5)	1.2–2.9 (0.6)	0.91 (0.72–1.15)	
		Population size		124,712 (142,053)	452,588 (156,473)	1.00 (1.00–1.00)	
		Population density (per sq km)		1756 (1310)	261–4305 (1360)	1.10 (0.89–1.36)	
		Dissimilarity index (African Americans vs. Others)		27.2 (6.0)	19.6–41.6 (5.6)	0.89 (0.74–1.09)	
		Dissimilarity index (Hispanics vs. Others)		30.9 (8.3)	18–847.0 (9.2)	1.04 (0.84–1.29)	
		Total crime rate (per 100,000)		3757 (1252)	2283–7051 (1369)	1.20 (0.98–1.49)	
		Larceny crime rate (per 100,000)		1908 (580)	1005–2832 (570)	1.23 (1.02–1.48) ⁴	1.26 (1.03–1.54) ⁴
		Non-larceny crime rate (per 100,000)		1904 (829)	914–4264 (583)	1.07 (0.93–1.23)	

¹ Hazard ratios were scaled across the interquartile range of exposure. All models contained age and gender stratification of the baseline hazard, adjustment for race and ethnicity, and a 2-level independent random effects structure including schools and communities. The co-adjusted model included all variables that had $p < 0.10$ in bivariate models.

² Sub-categories of Title I funding were modeled together; therefore hazard ratios and 95% confidence intervals were mutually co-adjusted.

³ $p < 0.10$

⁴ $p < 0.05$

Table 2
Associations between incident asthma and characteristics of the community and school adjusted for individual-level covariates

Level	Effect	Model 1	Model 2a	Model 2b
		HR (95% CI)	HR (95% CI)	HR (95% CI)
Community	Larceny crime rate (per 100,000) [/]	1.32 (1.08–1.60)	1.29 (1.06–1.57)	1.25 (1.02–1.52)
School	Title I funding	1.71 (1.13–2.57)	1.68 (1.10–2.56)	1.63 (1.08–2.46)

All models contained age and gender stratification of the baseline hazard, adjustment for race and ethnicity, and a 2-level independent random effects structure including schools and communities.

Model 2a was adjusted for individual and household characteristics, including race/ethnicity, parental history of asthma, underweight/overweight, medical insurance coverage, parental education, parental stress, second hand smoke in utero and in the home, musty odor, mildew, cockroaches, water damage, pets and gas stove in the home, and carpet in child's bedroom. Indicators of missing data were also included to allow all 2456 subjects into this model; all missing data indicators were non-significant, except for mildew.

Model 2b was adjusted for race/ethnicity and modeled traffic-related air pollution (NOx) at households.

[/] Hazard ratios and 95% confidence intervals were scaled across the interquartile range of the larceny crime rate in all communities (570 per 100,000).