Race Disparities in Childhood Asthma: Does Where You Live Matter?

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Objective: This study investigates whether racial/ethnic disparities in childhood asthma prevalence can be explained by differences in family and neighborhood socioeconomic position (SEP).

Methods: Data were from the 2001 Rhode Island Health Interview Survey (RI HIS), a statewide representative sample of 2,600 Rhode Island households, and the 2000 U.S. Census. A series of weighted multivariate models were fitted using generalized estimating equations (GEE) for the logistic case to analyze the independent and joint effects of race/ethnicity and SEP on doctor-diagnosed asthma among 1,769 white, black and Hispanic children <18 years old.

Results: Compared with white children, black children were at increased odds for asthma and this effect persisted when measures of family and neighborhood SEP were included in multivariate models (AOR: 2.49; 95% CI: 1.30–4.77). Black children living in poverty neighborhoods had substantially higher odds of asthma than Hispanic and white children in poverty areas and children in moderate- and high-income neighborhoods (AOR: 3.20; 95% CI: 1.62–6.29).

Conclusion: The high prevalence of asthma among black children in poor neighborhoods is consistent with previous research on higher-than-average prevalence of childhood asthma in poor urban minority communities. Changing neighborhood social structures that contribute to racial disparities in asthma prevalence remains a challenge.

Key words: children/adolescents ■ asthma ■ racial/ethnic disparities ■ socioeconomic inequalities

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Asthma is the most common chronic childhood illness in the United States.[1] It is a major cause of childhood disability[2] and accounts for a substantial number of hospital admissions, physician visits and school absences.[3,4] As documented in several national studies, asthma prevalence is higher among non-Hispanic black children compared with non-Hispanic white children.[4-10] Among Hispanics, Puerto Rican children have a higher prevalence of asthma than Mexican-American or Cuban-American children,[11-13] but there are wide variations in asthma prevalence among U.S.-born and foreign-born Hispanic children.[14] In studies of symptomatic children, asthma is more severe among blacks than among whites.[2,4,15,16] In 1998–1999, for example, asthma-related hospitalizations for black children were three times that for white children.[4]

Eliminating racial/ethnic disparities in children's health is a national priority as outlined in Healthy People 2010,[17] but the factors responsible for part or all of the observed racial/ethnic disparities in childhood asthma prevalence and severity remain an enigma.[10] Given that blacks and many Hispanic subgroups are more likely to be poor than whites,[18,19] an important question is whether race and ethnicity are independent risk factors for childhood asthma or a confounder for persistent social and economic inequalities between racial/ethnic minorities and whites in the United States.

Previous studies have convincingly demonstrated that inequalities in socioeconomic position (SEP) interact with health status throughout the lifecycle and increase the risks of morbidity and mortality.[20,21] People with relatively low incomes have, on average, poorer health and shorter life expectancy than the most economically advantaged. A growing body of literature also has shown that the social and economic characteristics of neighborhoods affect health

independent of individual-level risk factors.[22,23] Studies have documented variations in health based on neighborhood socioeconomic characteristics for a wide range of outcomes, including low birthweight,[24] self-rated health,[25] healthcare use,[26] risk of domestic violence,[27] morbidity[28] and overall mortality,[29] above and beyond individual-level socioeconomic status. Some researchers have pointed out that given the reciprocal relationship between family- and neighborhood-level poverty, studies that only measure socioeconomic disparities at the individual-level may understate a neighborhood's overall contribution to health.[22,30]

The most frequently examined explanations for racial/ethnic disparities in childhood asthma prevalence have been family or household income, with prior research yielding conflicting results. Population-based studies analyzing national or local data have reported no, positive or inverse associations between income and childhood asthma, controlling for race and/or ethnicity.[4,6,9,10,15,31-36] In several of these studies, initial associations between black race and childhood asthma were diminished or reduced to a null effect when income was included in multivariate analyses.[9,35] Although some national studies have found a higher prevalence of asthma among black children than white children in poor and nonpoor families,[6,15] one population-based report found that black children were at substantially higher risk of asthma than white children only among children in families with incomes less than half of the federal poverty level, after adjustment for sociodemographic variables.[10]

Many studies have found higher-than-average prevalence of childhood asthma in poor urban communities, especially in minority neighborhoods.[16,31,34,36-40] Only four studies, however, linked children to their residential characteristics using U.S. census data to investigate whether a neighborhood's socioeconomic environment contributed independently to asthma outcomes after individual characteristics had been taken into account.[41-44] In two studies, asthma prevalence was higher in poverty neighborhoods, independently of individual characteristics, but Hispanic children remained at higher risk of having asthma compared with white children even after adjustment for area of residence.[41,43] Nevertheless, the influence of neighborhood characteristics on pediatric asthma is still in its infancy,[42] as the four previous studies that examined neighborhood-level variation in childhood asthma were limited to clinical samples[44] or samples drawn from a single city.[41-43] To our knowledge, no previous population-based national or statewide studies of racial/ethnic disparities in childhood asthma have taken family- and neighborhood-level SEP into account in a detailed manner.

This study builds on health disparities research and public policy efforts aimed at improving the health of racial and ethnic minorities by investigating the simultaneous effects of race and social disadvantage at the family- and neighborhood-levels on doctor-diagnosed asthma in a statewide population-based sample of children aged <18 years old. Current sociodemographic population data show that black children are more likely than either white or Hispanic children to live in neighborhoods of concentrated poverty, and low- and middle-income minority families are more likely to live in poverty neighborhoods than whites with the same incomes.[45] Therefore, we investigated the joint effect of race/ethnicity and residence in a poverty area on asthma prevalence.

METHODS

Data and Sample

Two sources of data are used in this study: the 2001 Rhode Island Health Interview Survey (RI HIS) and the 2000 U.S. census. The RI HIS is a random-digit-dial telephone survey administered every 3–5 years. The 2001 survey collected health-related information on children and adults in sampled Rhode Island households. Within each household, the adult with the most knowledge of the health and healthcare utilization of all household members reported for each household member. In 2001, the interviewed sample consisted of 2,600 respondents, which yielded information on 3,263 persons in family units and 6,877 household members. The response rate was 55%,[46] similar to the median response rate of 51.3% for the 2000 Behavioral Risk Factor Surveillance System (BRFSS) survey.[47] For the current study, we excluded children who were Asian (n=46), Hawaiian (n=2), Native American (n=30) or whose race could not be determined (n=20) because sample sizes for these race groups were too small to produce reliable estimates (n=98).

Outcome Variable: Asthma

A single item in the RI HIS assessed whether a child in the family had asthma. A child was considered to have asthma if the survey respondent answered "yes" to the question: "Did a doctor say that [name of child] has asthma?"

Independent Variables

Neighborhood SEP. 2000 census geography codes were used to link children to their neighborhood characteristics using five-digit census ZIP code tabulation areas (ZCTAs) from the 2000 U.S. census. Census ZCTAs were matched to the reported ZIP code of each household. All 70 ZCTAs for Rhode Island based on the 2000 census were included in this study. ZIP codes were classified as a poverty area if >25% households had incomes <200% of the federal poverty level (FPL). We

used the 200% rather than the 100% cutoff to include those individuals who qualify for state and federal means-tested entitlement programs (e.g., Special Supplemental Nutrition Program for Women, Infants and Children; Children's Health Insurance Program). In addition, we created a variable that combined a child's race/ethnicity and area of residence: 1) Hispanic children living in poverty neighborhoods, 2) black children living in poverty neighborhoods, 3) white children living in poverty neighborhoods, and 4) children in non-poor neighborhoods. Rhode Island has the highest percentage of Hispanic children living in poverty (47%) compared to the national rate (28%) and the highest percentage of black children living in poverty in New England (38%).[48] Three-fourths (75%) of children in poverty are concentrated in poor urban communities.[48]

Family-level SEP. Information on type of health insurance was available for each household member, which was categorized as none, publicly funded or private. Although health insurance is a proxy for access to care, it is also an economic indicator.[49] Respondents were asked their total family income before taxes from all sources during the past 12 months. For respondents who did not want to report a specific dollar amount, a series of questions determined if the income fell within a specific range. Income was classified according to 2001 poverty thresholds adjusted for household size from household income tables available from the U.S. Census Bureau.[50] Other SEP variables included the highest educational level attained by a child's parents, but the variable was highly correlated with family income and was not included in the multivariable models.

Covariates. Other variables included (◆ indicates reference group): race/ethnicity (Hispanic, black, white◆); age (0–5,◆ 6–11, 12–17 years); sex (boys, girls◆); routine medical visit in past year (yes, no◆); whether the child had a parent with asthma (yes, no◆); and whether the home had asthma triggers,[51] including exposure to passive smoking in the home (yes, no◆); and mildew, mold or excess dampness in the home in the past 12 months (yes, no◆). The question on whether a parent had asthma was identical to the question asked of children. Doctor-diagnosed asthma in a parent served as a proxy for a genetic predisposition to asthma since a family history of allergy is strongly associated with persistent asthma during childhood.[52]

Analysis

The analytic goal was to estimate excess risk of asthma among children in relation to neighborhood and housing characteristics as well as racial/ethnic group. Using SUDAAN (version 9.0) statistical software programs,[53] we developed a series of logistic multivariable models. We estimated excess risk from odds ratios derived from generalized estimating equations (GEE). The GEE procedure accounts for hierarchical clustering, where children are nested in families, families are nested in households, and households are nested in neighborhoods.[54,55]

In order to better understand the relative importance of race/ethnicity in relation to childhood asthma, we sought to determine which variables might cause race to "drop out" of the model, after controlling for other factors. We started with a core set of demographic, healthcare and household environmental variables (Model 1), and then added family income and health insurance variables to see if these measures of SEP diminished the risks associated with race and ethnicity (Model 2). We then included neighborhood-level SEP to determine whether neighborhood characteristics diluted race/ethnicity and family-level SEP estimates (Model 3). The final model focused on the joint effect of race and living in a poverty neighborhood on childhood asthma in a model controlling for family income and other covariates (Model 4).

In this study, we did not include interaction terms in our final logistic regression model. Our interest was not to parameterize and estimate an average effect of being minority, plus an average effect of being poor, plus the statistical interaction of these terms. Our goal was to estimate effects of deprivation on asthma risk. We expressly conceptualized measurement for children in relation to social and economic deprivation; thus, a child who was both a minority and living in a poverty area received a "yes" for exposure to social and economic deprivation, i.e., operationalized as a minority child in a poor neighborhood. Thus, our model was saturated with respect to our conceptual definitions of the covariates under study. All analyses were weighted to account for unequal probabilities of selection, oversampling, nonresponse and to allow for calculation of population-based statewide estimates.

RESULTS

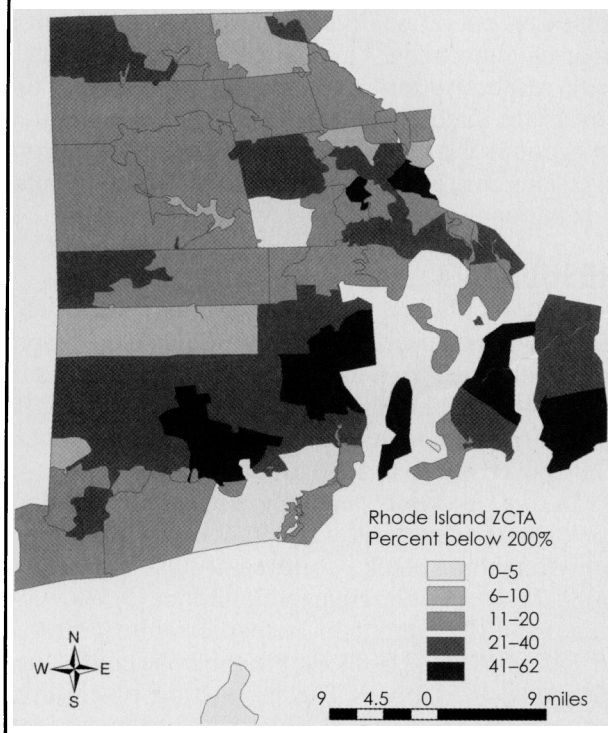
Figure 1 illustrates the distribution of ZCTAs by neighborhood poverty according to sociodemographic data obtained from the decennial 2000 U.S. census. Areas with the highest concentration of poverty lie within one of Rhode Island's six core cities, where child poverty rates in 2000 exceeded 15%.

In this statewide sample, the prevalence of doctor-diagnosed asthma was 10%. It was higher for non-Hispanic black children (20%, 95% CI: 10.9–29.2) than for Hispanic children (9.5%, 95% CI: 6.1–12.8), and non-Hispanic white children (9.5%, 95% CI: 7.6–11.4), but the 95% confidence intervals overlapped. The proportion of children whose parent had asthma varied (but not significantly) by race/ethnicity, with the highest rate (15%) for

Hispanic children and the lowest rate (9%) for non-Hispanic black children. Living in a home that was damp or had mildew and mold was more common among non-Hispanic black and non-Hispanic white children and less common among Hispanic children. Although there were fewer Hispanic children than non-Hispanic black or white children exposed to secondhand smoke in the home, this difference was not statistically significant. Compared with non-Hispanic white children, minority children were significantly more likely to have a parent with a high-school education or less, a family income 200% of FPL, no health insurance or publicly funded health insurance, and to live in a poverty area.

In bivariate logistic regression analysis, non-Hispanic black children had an increased risk of doctor-diagnosed asthma compared with non-Hispanic white children [odds ratio (OR): 2.29; 95% CI: 1.38–4.10; not shown]. This relationship persisted in models adjusted for other covariates. In the multivariate model that included baseline variables and that controlled for the correlations between children in the same household, non-Hispanic black children were nearly three times as likely as non-Hispanic white children to have doctor-diagnosed asthma [adjusted odds ratio (AOR): 2.87; 95% CI: 1.59–5.20; Table 2, Model 1]. Adding family income diminished the risk associated with race and ethnicity, but black race remained a significant correlate of asthma (AOR: 2.53; 95% CI: 1.35–4.73; Table 2, Model 2). When family income and area of residence were included together with baseline variables, the risk estimates associated with black race did not decline appreciably (AOR: 2.49; 95% CI: 1.30–4.77; Table 2, Model 3). Other factors associated with an increased risk of asthma (not shown) included ages 12–17 (AOR: 1.73; 95% CI: 1.10–2.70), having publicly funded health insurance (AOR: 2.08; 95% CI: 1.22–3.56), having a parent with asthma (AOR: 2.91; 95% CI: 1.82–4.67), and living in a home with excessive dampness and mildew (AOR: 1.90; 95% CI: 1.24–2.91). Living in a poverty neighborhood was not associated with an increased risk of asthma (AOR: 1.10; 95% CI: 0.69–1.75; Table 2, Model 3), but in additional analyses, non-Hispanic black children living in a poverty neighborhood had a three-fold excess risk of doctor-diagnosed asthma relative to children residing in nonpoverty communities (AOR: 3.20; 95% CI: 1.62–6.29; Table 2, Model 4).

Figure 1. Percent of Rhode Island residents below 200% poverty by year 2000 U.S. census ZCTA boundaries



DISCUSSION

The findings in this study not only serve as prevalence data for Rhode Island but also as a way to estimate racial/ethnic and socioeconomic inequalities in relation to asthma in children and adolescents. Additionally, this line of inquiry may help to elucidate potential pathways associated with the etiology of illness and disease as we age.

In this statewide representative sample of white, black and Hispanic youth age <18 years, an estimated 21,589 Rhode Island children have been diagnosed with asthma, yielding an overall prevalence rate of 10% (95% CI: 8.3–11.7%, not shown). Our estimate of 10% is higher than the 6% of children with physician-diagnosed asthma in the 1988–1994 National Health and Nutrition Examination Surveys (NHANES III)[56] but lower than the 12% of children that have ever been diagnosed with asthma in the 2002 National Health Interview Surveys (NHIS)[1] and lower than that reported in some area studies that have described prevalences of 18–20% for definite and probable asthma.[37,40,57] There is no universal definition of childhood asthma, making it difficult to measure the true burden of disease in U.S. children and adolescents.[58] Additionally, asking a proxy respondent whether a doctor diagnosed a child with asthma potentially eliminates those children who experience respiratory difficulties but do not have a clinical diagnosis. Certainly, a clinical diagnosis of asthma does not mean that all children are free of breathing problems—just that the adult respondent did not have a confirmation from a doctor.

Our analyses were undertaken to more fully explore the effect of SEP on childhood asthma and to examine the extent to which racial/ethnic differences in asthma prevalence could be explained by poverty in a representative sample of Rhode Island households. Previous nationwide surveys of racial/ethnic disparities in asthma prevalence in U.S. children could not adequately adjust for neighborhood SEP because the surveys lacked geographic codes to link respondents to their residential charac-

Table 1. Characteristics of children ages 0–17 years by race/ethnicity in the Rhode Island Health Interview Survey 2001, linked to the 2000 U.S. Census

	Total n (%)	Hispanic n (%)	Non-Hispanic Black n (%)	Non-Hispanic White n (%)	P Value[1]
Number in sample	1,769 (100)	353 (20.0)	142 (8.0)	1,274 (72.0)	
Doctor Diagnosis of Asthma					
Child					
Yes◆	187 (10.0)	37 (9.5)	27 (20.0)	123 (9.5)	0.1194
Parent					
Yes	237 (13.6)	46 (15.1)	12 (8.8)	179 (13.6)	0.3486
Sex					
Female	867 (47.6)	152 (44.2)	69 (44.2)	646 (48.4)	0.3461
Male	902 (52.4)	201 (55.8)	73 (55.8)	628 (51.6)	
Age (Years)					
0–5	541 (31.6)	109 (31.8)	51 (37.8)	381 (31.3)	0.7776
6–11	666 (37.6)	132 (38.0)	52 (36.2)	482 (35.5)	
12–17	562 (30.8)	112 (30.2)	39 (26.0)	411 (31.2)	
Routine MD Visit Past Year					
Yes	1,574 (90.2)	289 (82.6)	124 (90.2)	1,161 (91.5)	0 .0119
Health Insurance (Child)					
None	64 (3.5)	18 (5.2)	12 (1.5)	34 (3.3)	<0.0001
Public	409 (20.0)	188 (54.3)	63 (46.2)	158 (12.5)	
Private	1,290 (76.5)	144 (40.4)	75 (52.3)	1074 (84.2)	
Parent Education[2]					
Less than high school	161 (7.9)	91 (27.9)	16 (12.3)	54 (4.1)	<0.0001
High-school graduate	494 (26.6)	133 (35.9)	50 (35.2)	311 (24.5)	
At least some college	1,101 (65.5)	122 (36.2)	75 (52.5)	904 (71.4)	
Family Income, %FPL[3]					
≤200%	630 (31.8)	267 (74.6)	77 (56.6)	286 (22.8)	<0.0001
201–400%	545 (33.4)	56 (17.5)	39 (25.0)	450 (36.7)	
>400	594 (34.8)	30 (7.9)	26 (18.4)	538 (40.5)	
Mildew/Mold/Excessive Dampness in Home ≤12 Months					
Yes	363 (21.5)	46 (13.0)	24 (20.8)	293 (23.1)	0.0200
Exposure to Passive Smoking in Home[4]					
Yes	217 (13.0)	25 (8.9)	15 (10.3)	177 (13.8)	0.2655
Neighborhood Characteristics					
Poverty area[5]	712 (34.1)	296 (84.9)	116 (83.2)	300 (22.2)	<0.0001

1: All estimates (except for sample sizes) were calculated by using the sampling weights to provide population estimates; 2: Highest level of educational attainment of parent or caregiver; 3: FPL: federal poverty level; 4: smoker ages ≥18 smokes in home; 5: Poverty area: Census ZCTA where ≥25% households have incomes ≤200% FPL; ◆ yes versus no

teristics using census data.[4,6,9-10,15,31,33,35] At least two earlier studies[41,43] found a positive association between neighborhood poverty and asthma prevalence, whereas two other studies[42,44] found no association. Studies conducted in noninner-city settings[42] or in primary care clinics[44] are likely to produce very different results than cohort studies that include inner-city neighborhoods with high concentrations of poverty.[43] Furthermore, efforts to disentangle the effects of individual- and neighborhood-level SEP on health outcomes is not simple, and multivariate analyses that adjust for neighborhood characteristics may still miss the true picture of a community.[22]

In the current study, childhood asthma was concentrated among non-Hispanic black children living in Rhode Island's poor neighborhoods—a finding consistent with the National Cooperative Inner City Asthma Study (NCICAS).[59] Although not shown, nearly all Hispanic and non-Hispanic black children in families with incomes ≤200% FPL (91% and 85%, respectively)—but only 30% of non-Hispanic white children in families with incomes ≤200% FPL—resided in one of Rhode Island's six core cities, where as noted, child poverty rates exceed 15%. Unfortunately, the variable measuring race and neighborhood poverty rates used in the dataset did not allow a comparison between black and Hispanic children living in high- versus low-poverty communities due to insufficient information in low-poverty areas for minority children and the drawing of the ethnic minority over sample from telephone exchanges where the percentage of Hispanics and African Americans was higher than the state norm,[46] primarily from the city of Providence, a majority "minority" city with the third highest child poverty rate among U.S. cities with populations of 100,000 or more.[60]

Research has shown that much of the large asthma burden seen among black children stems from more severe morbidity rather than higher prevalence, which raises the question about variations in environmental exposures associated with the development of asthma. Although we cannot demonstrate this with the RI HIS data, we hypothesize that race may be capturing a variety of environmental exposures associated with asthma onset in poverty neighborhoods (e.g., above-average exposure to dust mite allergen, cockroach allergen and air pollution) as well as unmeasured stressors linked to everyday bur-

Table 2. Adjusted odds ratios (AOR) and 95% confidence intervals (CI) for association of race/ethnicity and socioeconomic position with asthma among children in the Rhode Island Health Interview Survey 2001 linked to the 2000 U.S. Census

	Model 1[a] AOR (95% CI)	Model 2[b] AOR (95% CI)	Model 3[c] AOR (95% CI)	Model 4[d] AOR (95% CI)
Race/Ethnicity				
Hispanic	1.08 (0.66, 1.77)	0.98 (0.55, 1.74)	1.02 (0.57, 1.84)	
Black	2.87 (1.59, 5.20)	2.53 (1.35, 4.73)	2.49 (1.30, 4.77)	
White	1.0	1.0	1.0	
Family Income, %FPL				
≤200%		0.64 (0.36, 1.13)	0.55 (0.32, 0.96)	
201–400%		0.68 (0.42, 1.08)	0.67 (0.42, 1.07)	
>400%		1.0	1.0	
Neighborhood Characteristics				
Poverty area			1.10 (0.69, 1.75)	
Nonpoverty area			1.0	
Race/Ethnicity and Neighborhood Characteristics				
Hispanic race and lives in poverty area				1.07 (0.56, 2.03)
Black race and lives in poverty area				3.20 (1.62, 6.29)
White race and lives in poverty area				1.04 (0.61, 1.75)
Children in nonpoverty areas				1.0

a: Model 1 adjusted for age (0–5,◆ 6–11, 12–17 years), sex (boys, girls◆), routine medical visit past year (yes, no◆), whether the child had a parent with asthma (yes, no◆), whether the home had asthma triggers, including exposure to passive smoking in the home (yes, no◆), and mildew, mold or excess dampness in home in past 12 months (yes, no◆); ◆ = reference group; b: Model 2 adjusted for Model 1 variables and health insurance (none/publicly funded versus private◆); c: Model 3 adjusted for Model 1 and 2 variables; d: Model 4 adjusted for age (0-5,◆ 6-11, 12-17 years), sex (boys, girls◆), routine medical visit past year (yes, no◆), whether the child had a parent with asthma (yes, no◆), whether the home had asthma triggers, including exposure to passive smoking in the home (yes, no◆), and mildew, mold or excess dampness in home in past 12 months (yes, no◆), health insurance (none/publicly funded versus private◆) and family income as a percentage of the federal poverty level (≤200%, 201–400% and >400%◆).

dens of racial prejudice and discrimination. One consequence of residential racial segregation, for example, is the intensification and concentration of poverty in aging suburban and urban communities characterized by a low tax base; deteriorating infrastructure; substandard housing; and less access to economic, social and physical resources essential for health.[61] It is surprising that we did not find that poor Hispanic children, like poor non-Hispanic black children, had an elevated risk of asthma. We speculate that our racial category of "Hispanic" was too heterogeneous to have been meaningful. Rhode Island's Hispanic population is small but diverse, with Puerto Ricans comprising 28% of the Hispanic population, followed by those from the Dominican Republic (20%), Central America (12.5%), South America (9.5%) and Mexico (6.5%).[60] Studies have shown a lower prevalence of asthma among foreign- than U.S.-born Hispanic children, which has been attributed to social and cultural factors—notably, patterns of migration, number of years in the United States and the level of acculturation.[12,14] The RI HIS can be used to evaluate racial/ethnic differences in health outcomes, but the survey does not include questions on immigration status and thus important differences in the risk of childhood asthma among Hispanic children may have been masked.

An intriguing observation from our data is the higher prevalence of asthma in nonpoor families than in poor families. In contrast, children with publicly funded insurance had a higher prevalence of doctor-diagnosed asthma than children with private healthcare coverage. Differences in asthma prevalence between children with public health insurance and those with private health insurance may exist because families that have children with asthma and cannot afford private healthcare coverage may be more likely than other low-income families to enroll in a publicly funded health insurance plan in order to access the healthcare their children need. Like many other states, Rhode Island provides health insurance to income eligible children. RIte Care, Rhode Island's Medicaid Managed Care Program, covers children up to age 19 in families with incomes up to 250% of the federal poverty level. Clearly, health insurance and household income are correlated. By controlling for these two variables simultaneously, one effect of this multicollinarity was that one proxy for economic well-being (health insurance) was associated with an increased risk of asthma, and this reduced all the coefficient estimates for income. Data on asthma severity would allow for refinement of this interpretation by estimating severity of asthma across socioeconomic and racial/ethnic characteristics. An analysis controlling for both race (black versus white) and family income (as a percentage of the federal poverty index) found that poor white children had nearly double the prevalence of asthma-related activity limitations compared with nonpoor white children (13.2% vs. 7.2%), although poverty appeared to have less of an effect on activity limitations due to asthma for black children.[15]

Another potential explanation for the higher prevalence of asthma in nonpoor families than in poor families is potential differential bias in inclusion of poor families. Since our data were collected using random-digit dialing, there is the possibility that the poorest families (in terms of family income) did not have telephones and thus were excluded from participating. Whether children in these families were at higher risk for asthma than their less poor neighbors has been suggested by one study that showed that childhood asthma was concentrated only among non-Hispanic black children living at less than half the poverty level relative to other poor children and children at other income-to-FPL levels.[10] Future work may provide more definitive conclusions.

The moderately increased risk of asthma associated with living in a house that was damp or had mold/mildew reported in our study, while not very impressive, raises important questions about which environmental factors are associated with asthma onset and which environmental exposures contribute to active asthma. The Institute of Medicine (IOM) report, *Clearing the Air: Asthma and Indoor Air Exposures*, concluded that there was insufficient evidence of an association between exposure to mold and mildew and asthma onset.[62] Whether prolonged exposure to mold and mildew elevates the risk of asthma is a question that warrants further investigation. The IOM report also concluded that exposure to house dust mites and to environmental tobacco smoke was associated with asthma onset in preschool children. We did not find an association between exposure to passive smoking and asthma among younger- or older-aged children in our study, but the 2001 RI HIS did not assess how often children were exposed to smoking in the home; thus, the smoking variable only approximates a child's exposure to secondhand smoke. The IOM report also suggested the need for additional research on gene-environment interactions related to the development of asthma. Although our study was not designed to examine this issue, it did demonstrate that childhood asthma was associated with having a parent that had asthma, but we were unable to determine if the underlying cause of this relationship was genetics or a family's exposure to asthma triggers in the environment.

The generalizability of our results may be affected by the way we measured neighborhood SEP. Census ZCTAs include a larger population size (average population: 30,000) and therefore greater socioeco-

nomic heterogeneity than census tracts (average population: 4,000), favoring the use of census tracts as the measure of geographical classification. While the choice of the geographical classification (census block group, census tract, census ZIP code) affects the degree of clustering of poor health by area, overall, the level of geography selected has only a modest effect on the size of health differences by neighborhood deprivation.[63] In the 2001 RI HIS, only ZIP codes were available to link children and adolescents to their neighborhood characteristics. This will change with the next release of the RI HIS, which asks respondents to identify the streets closest to their home address in order to link households to their residential characteristics by census tract. Obtaining such geographical information will also allow for more complex spatial analysis using tools such as geographical information systems (GIS), which are becoming increasing popular in public health surveillance.

Our study of childhood asthma used GEE for analysis of correlated data, which cannot be used to partial out the amount of variance attributable to each block of variables in a model. Thus, we were unable to demonstrate how much of the variance in childhood asthma was explained by the joint effect of race and neighborhood SEP, controlling for other factors. There are no multilevel software packages for modeling population-based clustered data, but these software packages will be developed in the near future as multilevel modeling becomes more widely used.

The RI HIS has been and will continue to be essential to our understanding of chronic disease prevalence and associated risk behaviors at the individual, family and household levels. Yet the RI HIS and national datasets of importance to surveillance and research of chronic conditions in children can be improved. As David Williams and others point out,[64-68] there is a need to rethink what is meant by racial and ethnic disparities in health. Residential racial segregation, institutionalized racism and immigration status have decisive consequences for social mobility, educational opportunities and employment, and profound implications for individual and population health over the life course. Surveillance datasets that include measures of indoor and outdoor environmental exposures related to the development of childhood asthma and the social and economic factors that adversely affect the health of racial/ethnic minorities will help us understand the role of risk factors in the pathogenesis of asthma.

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