Ethnic Differences in the Effect of Asthma on Pulmonary Function in Children

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Rationale: The impact of asthma on chronic lung function deficits is well known. However, there has been little study of ethnic differences in these asthma-associated deficits.

Objectives: To examine whether there are ethnic differences in the effects of asthma on children's lung function.

Methods: We evaluated the impact of asthma on lung function in 3,245 Hispanic and non-Hispanic white school children (age 10–18 yr) in a longitudinal analysis of the Southern California Children's Health Study. Sex-specific mixed-effects regression spline models were fitted separately for each ethnic group.

Measurements and Main Results: Large deficits in flows were observed among children with asthma diagnosed before age 4 years regardless of ethnicity. Hispanic girls with asthma had greater deficits in flows than non-Hispanic girls and were largest for maximal mid-expiratory flow (–5.13% compared with –0.58%, respectively). A bigger impact of asthma in Hispanic girls was also found for FEV₁, FEF₇₅, and PEF (*P* value 0.04, 0.07, and 0.005, respectively). These ethnic differences were limited to girls diagnosed after age 4 years. In boys, asthma was also associated with greater deficits in flows among Hispanic than in non-Hispanic white children (differences that were not statistically significant). Ethnic differences in prevalence of pets and pests in the home, health insurance coverage, parental education, and smoking did not explain the pattern of lung function differences.

Conclusions: Larger asthma-associated lung function deficits in Hispanics, especially among girls, merit further investigation to determine public health implications and to identify causes amenable to intervention.

Keywords: Hispanics; non-Hispanic whites; pulmonary function; asthma

Asthma is the most common chronic disease in children. In addition, the prevalence in the United States has been increasing since 1980, and approximately 5 million children less than 18 years of age are now affected (1–6). Reduced airway flows are a hallmark of the disease and are useful in assessing asthma severity and airway remodeling (7–9). Asthma may be exacerbated by common environmental exposures, including pet allergen, tobacco smoke, and air pollution, resulting in reduced lung function (10–13). Mold and pests are common in substandard housing, and poverty associated with these asthma triggers may also be associated with poor asthma control because of restricted access to appropriate medical care including controller medications (14, 15). These social and envi-

AT A GLANCE COMMENTARY

Scientific Knowledge on the Subject

Although there have been numerous studies on the effect of asthma on lung function deficits, ethnic differences in the impact of asthma on lung function have not been well characterized.

What This Study Adds to the Field

Larger obstructive deficits in lung function were associated with asthma among Hispanic than among non-Hispanic white children, especially among girls. Further investigation of reasons for these differences is warranted to identify potential targets for intervention.

ronmental conditions vary by race and ethnicity, and it has been suggested that such social disparities explain part of the disproportionate severity of asthma in inner cities in the United States (13, 16). However, although both pulmonary function (PF) and asthma prevalence vary by race and ethnicity (14, 15, 17–33), there has been little study of ethnic differences in the impact of asthma on lung function.

We examined the relationship between ethnicity, asthma, and lung function, and potential environmental explanations for ethnic differences in the effect of asthma on lung function, in a longitudinal follow-up of participants in the Southern California Children's Health Study (34, 35). We focused on Hispanics and non-Hispanic whites, because Hispanics in this region, especially newer immigrants, as a group are less affluent and more likely to encounter associated environmental exposures, including those common to substandard housing (36, 37).

METHODS

The Southern California Children's Health Study is a longitudinal cohort study of lung function growth in children 10–18 years of age in 12 southern California communities. The research protocol was approved by the Institutional Review Board of the University of Southern California. Details regarding selection of communities, cohort recruitment, questionnaire-based assessment of health and covariates, including socioeconomic status (SES), access to care, and environmental exposures, and PF testing have been reported elsewhere (34, 35, 38). For this analysis, we used longitudinal data from 4,824 fourth and seventh grade children enrolled in 1993 and a second fourth grade cohort enrolled in 1996 (median age was 10 for fourth graders, 13 for seventh graders in 1993, and 9.9 for fourth graders in 1996). Each cohort was evaluated annually up to high school graduation at age 18. There was retesting of a 10% random sample of children each year for quality control purposes. The average number of tests was 7.4 per subject (range, 2–13). Based on a parent-completed questionnaire at study entry, children who had cystic fibrosis, history of severe chest injury, or incomplete information on asthma at baseline were excluded from analysis. Only non-Hispanic white and Hispanic children were

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included in the analysis, because sample size was limited for other ethnic groups. A child was categorized as Hispanic if the parent answered "Yes" to a question on Hispanic ethnicity, regardless of race. Only 22 of these recorded a specific race other than white (13 Asian and 9 African American), but most identified race as "other" (52.8%) or "mixed" (15.6%). Children of white race but not Hispanic ethnicity were defined as non-Hispanic whites. When a parent or legal guardian answered "yes" to the question "Has a doctor diagnosed your child with asthma?" in the baseline questionnaire, or the child answered "yes" to the question "Has a doctor ever said you had asthma?" in the annual questionnaire at time of PF testing, the child was classified from then on as having asthma. Children with a lifetime history of asthma were further categorized into those with diagnosis reported to have been made before 4 years of age and 4 years or older, based on known age-related phenotypic differences in asthma and on differences in the impact of early life onset on asthma severity (39, 40). Newly diagnosed children with asthma based on a new report during follow-up were grouped into the late-onset category. Five lung function measurements were derived from yearly PF testing using procedures described elsewhere (34): (1) FVC, (2) FEV₁, (3) maximal midexpiratory flow (MMEF), (4) FEF₇₅, and (5) PEF.

Based on previous reports that effects of asthma on lung function vary by sex (2, 41), which were consistent with results from preliminary analyses of our data (38), all models were stratified by sex. We used a flexible mixed-effects regression spline model (38, 42–45), with a general form given by

$$E(\log[PF_{cij}]) = \mu + a_{ci} + f_1(AGE_{cij}) + f_2(AGE_{cij}) \times \log(HEIGHT_{cij}) + X_{cij}\gamma + \alpha_g I_g, \quad (1)$$

where $f_1(AGE_{cij})$ and $f_2(AGE_{cij})$ represent the age-dependent intercept and slope of $\log[PF_{cij}]$ on logged height, and a_{ci} is a random subject-specific intercept. Adjustment covariates X_{cij} included in all models were community, school grade, technician, spirometer, room temperature, barometric pressure, body mass index (BMI), BMI², respiratory infection at PF testing, and severe chest illness before age 2 years. The parallel percent difference $[\exp(\alpha_g) - 1] \times 100$ for the g^{th} group with asthma was the primary value of interest.

These sex-specific models were fit separately for non-Hispanic (white) and Hispanic ethnic groups. Models for both groups combined were also fitted, adjusted for ethnicity, with non-Hispanic participants as the reference group. To test for different effects of asthma on PF between Hispanics and non-Hispanics, an interaction term between each asthma group variable I_g and the Hispanic indicator was added into the combined models. Based on results of these analyses, we also examined differences in the proportion of children with clinically relevant obstructive airway deficits defined as the lower fifth percentile of predicted lung function at age 18 (46). The interaction between age and asthma group variable I_g was also explored to test age-related trends of asthma effect on lung function. Residuals analysis was performed to check the goodness of fit of models. The jackknife method was used to test the robustness of the estimates. All analyses were performed using SAS 9.1 (SAS Institute Inc., Cary, NC) and the Splus (TIBCO Software Inc., Palo Alto, CA) statistical software package (47, 48).

RESULTS

Of the 4,824 participants enrolled in the study, 937 were in the seventh grade cohort, 1,806 in the first fourth grade cohort, and 2,081 in the second fourth grade cohort. There were 470 children excluded because of incomplete information on asthma, 13 for serious chest injury, and 5 for cystic fibrosis. Of those remaining, there were 2,405 non-Hispanic white and 1,242 Hispanic children available for analysis. An additional 202 boys (140 non-Hispanic and 62 Hispanic) and 200 girls (119 non-Hispanic and 81 Hispanic) were excluded because they did not have at least two PF tests. The final data set used for analysis consisted of 3,245 children, among whom 2,146 were non-Hispanic (1,079 boys and 1,067 girls) and 1,099 were Hispanics (525 boys and 574 girls).

Hispanic children were more likely to be of lower SES and to have housing conditions reported to be associated with asthma exacerbation, with the exception of second-hand tobacco smoke (SHS) exposure at home, which was more common among non-Hispanic children (Table 1). Hispanic parents were less likely to have completed high school (26% compared with 7% of non-Hispanics), and their children were less likely to have insurance (72% vs. 89%). Hispanics were also more likely to report mold, mildew, or cockroaches, and less likely to have a dog or cat in the home. They were also less likely to have been exposed to tobacco smoke *in utero* from a smoking mother (10% compared with 21%). There were few children who smoked at study entry. In general, there was more missing information for Hispanic children.

The proportion of children with asthma (including those with a reported lifetime physician diagnosis or diagnosis during follow-up) was over 20% in all sex-race subgroups, and most first reported a diagnosis after age 4 years (Table 2). Boys were more likely than girls to have early onset asthma, regardless of ethnicity. There was little difference between Hispanic and non-Hispanic participants in the proportion diagnosed early or later in childhood.

In both racial and ethnic groups combined, there were small but significant deficits among children with asthma in small airway flows and in PEF (Table 3). However, the pattern of sex-specific asthma effects differed by ethnicity. Among girls, there was little impact of asthma on lung volume (FVC), regardless of ethnicity (Table 3). Hispanic girls with asthma had large deficits in small airway flows (−5.13% in MMEF compared with asthma-associated deficits of −0.58% in non-Hispanic girls; interaction P value 0.009) (Table 3 and Figure 1). Asthma-related FEF₇₅ deficits were −4.89% in Hispanic girls compared with −0.57% in non-Hispanic girls (interaction P value 0.07); PEF deficits were −4.33% compared with −0.62% (interaction P value 0.005). Hispanic girls also had larger asthma-associated deficits in FEV₁ (−1.14%) compared with those in non-Hispanic girls (0.39%; P value for interaction 0.04). Deficits in flows were also consistently larger in Hispanic boys (e.g., −1.90% compared with 0% for FEV₁ in non-Hispanics), but the differences were not statistically significant. Hispanic boys had a small deficit in FVC (−1.3%) that was marginally different from the impact of asthma in non-Hispanic whites (0.63%; P value for interaction 0.05) (Figure 1).

We examined potential additional confounders of the differences between Hispanic and non-Hispanic children by adding the social and housing characteristics shown in Table 1 one at a time to the relevant model. The effect of asthma on PF was insensitive to the inclusion of these potential confounders (data not shown), and hence they were not included in the final models. In a sensitivity analysis to see if access to care could explain the pattern of asthma effects, we restricted the analysis to participants with health insurance. The pattern of effects of ethnicity were similar, although the asthma-associated deficit in FEV₁ in Hispanic boys (−2.42%) became larger relative to the deficit in non-Hispanic boys (0%), (interaction P value 0.02; see Table 3 for comparison). We also restricted the analysis to participants born in the United States (85% of Hispanics and 98% of non-Hispanics) to see whether ethnic difference could be related to birth and early life influences in another country. Although the magnitude of the differences between Hispanics and non-Hispanics became slightly smaller for United States born participants, the overall pattern of effects was very similar (results not shown). Finally, we excluded the 22 Hispanics from the analysis who reported race as African American or Asian. The pattern of differences between Hispanics and non-Hispanics was not substantially changed. However, the differ-

TABLE 1. COMPARISON OF SOCIAL AND ENVIRONMENTAL EXPOSURES OF NON-HISPANIC WHITE AND HISPANIC CHILDREN AT STUDY

Baseline Characteristics	White (n = 2,146)	Hispanic (n = 1,099)
Second-hand smoking exposure (%)		
Yes	789 (36.77)	285 (25.93)*
No	1,297 (60.44)	723 (65.69)
Missing	60 (2.80)	91 (8.28)
Personal smoking (%)		
Yes	4 (0.19)	0 (0)
No	2,142 (99.81)	1,099 (100)
History of mother smoking during pregnancy (in utero) (%)		
Yes	442 (20.60)	110 (10.01)*
No	1,657 (77.21)	942 (85.71)
Missing	47 (2.19)	47 (4.28)
Exercise during the 30 min before pulmonary function testing (%)		
Yes	436 (20.32)	229 (20.84)
No	1,710 (79.68)	870 (79.16)
Insurance (%)		
Yes	1,907 (88.86)	793 (72.16)*
No	206 (9.60)	271 (24.66)
Missing	33 (1.54)	35 (3.18)
Parental education (%)		
< High school	147 (6.85)	284 (25.84)*
= High school	408 (19.01)	234 (21.29)
> High school	1,561 (72.74)	496 (45.13)
Missing	30 (1.40)	85 (7.73)
Cat (%)		
Yes	1,116 (52)	274 (24.93)*
No	1,030 (48)	825 (75.07)
Dog (%)		
Yes	1,447 (67.43)	523 (47.59)*
No	699 (32.57)	576 (52.41)
Mold or mildew (%)		
Yes	642 (29.92)	250 (22.75)*
No	1,450 (67.57)	779 (70.88)
Missing	54 (2.52)	70 (6.37)
Cockroach (%)		
Yes	191 (8.90)	166 (15.10)*
No	1,955 (91.10)	933 (84.90)
Inhaler use for asthma (%)		
Yes	181 (8.43)	71 (6.46)
No	58 (2.70)	26 (2.37)
Participants with no asthma	1,907 (88.86)	1,002 (91.17)
Severe wheeze (%)		
Yes	285 (13.28)	117 (10.65)*
No	1,775 (82.71)	923 (83.99)
Missing	86 (4.01)	59 (5.37)

* Chi-square tests for associations between baseline characteristics of Hispanics and non-Hispanics with $P < 0.05$ (after excluding those with missing values from analysis).

ences between Hispanic and non-Hispanic boys in the impact of asthma on FVC was somewhat less (-1.09% in Hispanics, 0.63% in non-Hispanics, compared with -1.3% and 0.63% , respectively, in Table 3) and was no longer statistically significant (interaction P value 0.09). There was no age-related trend in the impact of asthma on lung function in either ethnic group.

For girls, large effects of asthma on small airway flows and PEF occurred in those with early onset asthma (Table 4), regardless of ethnicity. For example, girls with early onset asthma had a -10.88% deficit in MMEF and those with late onset had only a -1.16% deficit. However, the deficits in flows among Hispanic girls whose asthma was diagnosed after 4 years of age were significantly larger than those for non-Hispanic girls for whom asthma had little impact on PF testing. MMEF, for example, was reduced by -4.04% in Hispanic girls with asthma, compared with 0.07% for non-Hispanic girls (interaction P value 0.02). Significantly larger deficits in late-onset diagnoses

were also observed for FEV₁ and PEF among Hispanic compared with non-Hispanic girls.

In boys, effects of asthma on all lung functions, except FVC, were also considerably larger in those with early onset asthma than in those with late onset, regardless of race or ethnicity (Table 5). For example, boys with early onset asthma had a -13.66% deficit in MMEF compared with -1.50% for those with late-onset asthma. As for boys, deficits in flows associated with late-onset asthma were larger in Hispanic than non-Hispanic boys (except for PEF), but these differences were not statistically significant. The deficit in FVC in Hispanic boys was largely observed in those diagnosed early in life (-3.78%), and no deficit was observed in non-Hispanics (0.86%).

Residual analysis did not reveal any departure from the normality assumption in the models and there were no gross outliers. Moreover, a jackknife approach was used to test the robustness of our findings and we found that there was little bias ($0-3\%$) for the most significant findings on flows in girls.

TABLE 2. ASTHMA CHARACTERISTICS OF THE NON-HISPANIC WHITE AND HISPANIC PARTICIPANTS

Characteristics	Groups	Girl		Boy	
		Non-Hispanic White n (%)	Hispanic n (%)	Non-Hispanic White n (%)	Hispanic n (%)
Asthma status	Asthmatic	259 (24.3)	117 (20.4)	290 (26.9)	125 (23.8)
	Nonasthmatic	808 (75.7)	457 (79.6)	789 (73.1)	400 (76.2)
Age at first diagnosed asthma	<4	55 (21.3)	26 (22.6)	91 (31.5)	41 (33.3)
	≥4	203 (78.7)	89 (77.4)	198 (68.5)	82 (66.7)
	Missing	1	2	1	2
Total		1,079	1,067	574	1,079

DISCUSSION

There has been little study of ethnic differences in the impact of asthma on lung function. The Harvard Six-Cities Study found no differences between blacks and whites in the effect of asthma or wheeze on the levels of FEV₁ and MMEF in the preadolescent and adolescent years (41). To our knowledge, differences in the impact of asthma among Hispanics and non-Hispanic whites have not been reported previously. We found larger asthma-associated deficits in Hispanic than in non-Hispanic white children, especially among girls. Hispanic girls had significantly larger deficits in flows (FEV₁, MMEF, and PEF) than non-Hispanic girls, differences that were limited to those diagnosed after 4 years of age. A similar pattern of effects was observed in boys, although the differences in flows were significant only for FEV₁ (in a sensitivity analysis restricted to children with health insurance). There was little, if any, effect of asthma on lung function in non-Hispanic children with asthma diagnosed after age 4 years, regardless of sex.

We evaluated possible reasons for these differences based on our hypothesis that they could be explained by social factors, SHS exposure, and housing characteristics, but information available did not explain the ethnic differences in the impact of asthma on lung function. Hispanic children were less likely to have insurance (Table 1), and this might have explained the observed differences in flows, especially in girls, if lack of access to care resulted in delayed diagnosis of early onset asthma (which was associated with larger deficits in both ethnic groups) or suboptimal treatment. However, one might expect this explanation to have resulted in attenuated effects after statistical adjustment for health insurance or restriction to children with insurance, which was not the case. Lack of access to care might also be reflected in a lower proportion of symptomatic Hispanic children being diagnosed with asthma early in life, but we found little evidence for this because most children were diagnosed after 4 years of age, regardless of sex or ethnicity. Hispanic children with wheeze in the previous year were slightly more likely to have a doctor diagnosis of asthma at study entry (54%) than were non-Hispanic children (50%). Therefore, delayed diagnosis seems an unlikely explanation for the ethnic differences in flows in girls. In an additional sensitivity analysis, the larger deficits in flows in Hispanic girls were generally limited to those who had late-onset asthma and medication (inhaler) use (data not shown). Information on type of inhaler (controller or rescue medication) was not available. It is possible that the differences in lung function were explained by treatment with controller medications, if lack of insurance resulted in fewer Hispanic children being treated. However, although some studies have suggested that inhaled corticosteroids in older children and adults improved lung flow rates (49), several randomized trials in children found no long-term beneficial effects of controller medication on lung function (50, 51). Other investigators have found that lung function was lower in children of lower SES (52). Hispanic children's lower prevalence

of health insurance coverage and lower parental educational attainment (Table 1), compared with non-Hispanic children, are markers for lower SES, but adjustment for parental education also did not explain the pattern of lung function differences.

Indoor allergen exposure associated with poor housing conditions and *in utero* and SHS exposure may modulate the severity of asthma and the impact on lung function (10, 11, 13). However, adjustment for mold or mildew and for cockroaches in the home did not alter the pattern of ethnic-specific effects of asthma on lung function. Tobacco smoke exposure was uncommon among Hispanics compared with rates in non-Hispanics (Table 1), and adjustment for *in utero*, SHS exposure and for personal smoking in the year before each lung function test also did not explain the ethnic differences.

The impact of asthma on FVC in boys was marginally statistically different from the effect in non-Hispanics. However, this difference was partly caused by the larger FVC (albeit not significantly so) in non-Hispanic children with asthma compared with non-Hispanic children without asthma (Table 3 and Figure 1). The difference between Hispanics and non-Hispanics also was reduced and was no longer significant in the sensitivity analysis excluding African Americans and Asians from the analysis. Thus, this pattern of ethnic difference may have been caused by chance.

TABLE 3. SEX- AND ETHNIC-SPECIFIC EFFECTS OF ASTHMA ON PULMONARY FUNCTION*

Sex	PFT	NH&H	Percent diff. in Level		
			Non-Hispanic White	Hispanic	Interaction P Value†
Girls	FVC	0.4	0.51	−0.14	0.25
	FEV ₁	−0.03	0.39	−1.14	0.04
	MMEF	−2.00‡	−0.58	−5.13§	0.01
	FEF ₇₅	−1.90	−0.57	−4.89‡	0.07
	PEF	−1.78§	−0.62	−4.33§	0.01
Boys	FVC	0.01	0.63	−1.30‡	0.05
	FEV ₁	−0.62	0	−1.90§	0.1
	MMEF	−2.91§	−2.79§	−3.03‡	0.78
	FEF ₇₅	−3.93‡	−3.24‡	−5.03§	0.69
	PEF	−1.04	−0.97	−1.13	0.7

Definition of abbreviations: MMEF = maximum midexpiratory flow; NH&H = non-Hispanics and Hispanics combined group; PFT = pulmonary function test.

* All models adjusted for community, school grade, technician, spirometer, room temperature, barometric pressure, body mass index (BMI), BMI², respiratory infection at PFT, and severe chest illness before age 2 years. Asthma effects were reported as comparison with participants with no asthma.

† P value for testing whether effects were different between Hispanics and non-Hispanics.

‡ P < 0.05.

§ P < 0.01.

|| P < 0.1.

Figure 1. Selected asthma-specific lung function growth trajectories by ethnicity and sex. MMEF = maximal midexpiratory flow.

Other environmental exposures and cultural characteristics, especially dietary differences, and genetic variation merit further investigation as possible causes for the observed ethnic differences. Genetic variation has been shown to modulate the effect of asthma on lung function among Hispanics (53), and we have previously shown that genetic variation in susceptibility to tobacco smoke may affect lung function deficits among children with asthma (12). The use of race and ethnicity in studies such as ours is controversial (54, 55); Hispanics may not even be aware of their precise racial background and may report ancestry based on physical appearance and family national origin (37). Populations similar to our southern California population, which is primarily of Mexican origin with a smaller proportion from Central America, have been reported to have on average 52% Native American and 45% European ancestry (37, 56). The ethnic pattern of effects of asthma was not substantially affected in the sensitivity analysis excluding those reporting African American or Asian ancestry. Our results could not be generalized to Puerto Ricans, who have different racial ancestry, higher prevalence of lifetime physician-diagnosed asthma and active physician-diagnosed asthma, earlier onset of asthma, and lower lung volume than Mexicans (36), or to other Hispanic groups. Nevertheless, distinguishing genetic from poorly characterized envi-

ronmental causes that are correlated with genetic ancestry requires consideration of ethnicity (55).

Our results should be interpreted in light of some limitations to the study. First, the lack of adequate sample size from other racial and ethnic groups precluded their evaluation in this study. Second, variation in loss to follow-up based on characteristics, such as SHS exposure at study entry (*see* Table E1 in the online supplement), could limit the generalizability of our results to those who were lost to follow-up. However, controlling for these factors did not explain our results, so bias caused by loss to follow-up seems unlikely to explain the results. Third, asthma is a complex chronic inflammatory disease for which there is no universally recognized definition. Case ascertainment was done by parental report of physician-diagnosed asthma without clinical examination, which is widely used in epidemiologic studies (57), is reproducible (58, 59), and is a valid measure of what physicians actually report to patients (60, 61). The rates of asthma were high, partly because we included both lifetime cases at study entry (Table 1) and cases reported at yearly follow-up (Table E1). This design may also help explain why most cases were diagnosed after age 4 years, although it is commonly believed that most asthma originates in earlier childhood (2). Because we did not follow the cohort from birth, we cannot

TABLE 4. ETHNIC-SPECIFIC EFFECTS OF ASTHMA ON PULMONARY FUNCTION BY AGE AT DIAGNOSIS FOR GIRLS*

PFT	Asthma by Age at Diagnosis	NH&H	Percent Diff. in Level		Interaction P Value [†]
			Non-Hispanic White	Hispanic	
FVC	<4	1.67	1.68	1.66	0.94
	≥4	0.33	0.45	-0.31	0.2
FEV ₁	<4	-1.56	-1.24	-1.68	0.93
	≥4	0.08	0.49	-1.11	0.03
MMEF	<4	-10.88 [‡]	-9.56 [‡]	-11.84 [‡]	0.67
	≥4	-1.16	0.07	-4.04 [§]	0.02
FEF ₇₅	<4	-12.42 [‡]	-11.53 [‡]	-11.92 [§]	0.93
	≥4	-0.77	0.34	-3.59	0.08
PEF	<4	-7.10 [‡]	-6.41 [‡]	-7.66 [§]	0.79
	≥4	-1.28 [§]	-0.18	-3.90 [‡]	0.01

Definition of abbreviations: MMEF = maximum midexpiratory flow; NH&H = non-Hispanics and Hispanics combined group; PFT = pulmonary function test.

* All models adjusted for community, school grade, technician, spirometer, room temperature, barometric pressure, body mass index (BMI), BMI², respiratory infection at PFT, and severe chest illness before age 2 years. Asthma effects were reported as comparison with participants with no asthma.

[†] P value for testing whether effects were different between Hispanics and non-Hispanics.

[‡] P < 0.01.

[§] P < 0.05.

directly determine whether a new diagnosis during follow-up truly represented a late-onset incident case or was a second occurrence of asthma that first occurred during infancy but was not reported by a parent on the baseline questionnaire. Rates of new onset asthma in this cohort, which we have previously reported (62, 63), are high compared with reported incidence rates of childhood asthma in earlier decades (64–66). However, the rates in this cohort reflect the increasing rates of prevalence of asthma and of incidence measured more recently in similar study designs around the world (67–75). Although differences in design preclude comparison with rates in many other studies, reported age of onset has been validated and has provided reliable estimates in questionnaire-based longitudinal studies (76). We also found that girls were more likely to develop asthma during later childhood than boys, results consistent with previous studies (77–80). Although misclassification of

asthma might affect the observed impact on lung function, this is an unlikely explanation for the pattern of observed results unless misclassification was differential with respect to sex and ethnicity.

Finally, deficits in flows are an indicator of asthma severity (81), but acutely these deficits may reflect reversible bronchospasm and chronically they may reflect remodeling and fixed obstructive defects (8, 9). We had limited ability to distinguish these two interpretations of the data, for example by evaluating the individual response to bronchodilators. However, the ethnic differences in asthma-associated lung function deficits did not change after adjustment for use of an inhaler at the time of PF testing or for severe wheeze (as defined in the online supplement), both of which may have been markers for disease exacerbation. These sensitivity analyses suggest that the observed lung function deficits were chronic.

TABLE 5. ETHNIC-SPECIFIC EFFECTS OF ASTHMA ON PULMONARY FUNCTION BY AGE AT DIAGNOSIS FOR BOYS*

PFT	Asthma by Age at Diagnosis	NH&H	Percent Diff. in Level		Interaction P Value [†]
			Non-Hispanic White	Hispanic	
FVC	<4	-0.73	0.86	-3.78 [‡]	0.02
	≥4	0.08	0.6	-1.06 [§]	0.15
FEV ₁	<4	-4.49 [§]	-3.48 [§]	-6.07 [§]	0.2
	≥4	-0.13	0.43	-1.38	0.18
MMEF	<4	-13.66 [§]	-14.16 [§]	-11.78 [§]	0.55
	≥4	-1.50	-1.3	-1.87	0.91
FEF ₇₅	<4	-16.28 [§]	-17.09 [§]	-13.49 [§]	0.45
	≥4	-2.12	-1.23	-3.73	0.5
PEF	<4	-4.25 [§]	-3.89 [‡]	-4.36	0.83
	≥4	-0.46	-0.51	-0.38	0.54

Definition of abbreviations: MMEF = maximum mid-expiratory flow; NH&H = non-Hispanics and Hispanics combined group; PFT = pulmonary function test.

* All models adjusted for community, school grade, technician, spirometer, room temperature, barometric pressure, body mass index (BMI), BMI², respiratory infection at PFT, and severe chest illness before age 2 years. Asthma effects were reported as comparison with participants with no asthma.

[†] P value for testing whether effects were different between Hispanics and non-Hispanics.

[‡] P < 0.05.

[§] P < 0.01.

^{||} P < 0.1.

A major strength of this study is the large sample size and yearly assessment of asthma and lung function from childhood through adolescence, which made it possible to identify the larger effects of asthma on lung function among Hispanics, especially on indicators of obstruction (MMEF and FEF₇₅) in girls diagnosed after age 4 years. Although the ethnic differences in mean flow rates were small, large differences were observed in the proportion of children with late-onset asthma with abnormal FEV₁ and MMEF (each with 14.8% who were abnormal in Hispanic girls with asthma, compared with 5.1% abnormal in non-Hispanic white girls with asthma). These differences were statistically significant (interaction P value = 0.06 for ethnic differences), despite limited power caused by relatively small sample size in these subgroups. The largest deficits in flows were associated with diagnosis at early age and did not vary between ethnic groups, results that were consistent with an earlier analysis of a sample of this cohort with shorter follow-up (38) and with other studies (82, 83). Further investigation is warranted to determine the implications of these ethnic differences for lung function and associated cardiorespiratory disease, including mortality, in later adult life, and to identify potentially preventable causes (84). Ethnic differences in dietary and genetic characteristics, for example, might explain the observed differences.

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