Lifetime prevalence of childhood eczema and the effect of indoor environmental factors: Analysis in Hispanic and non-Hispanic white children

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ABSTRACT

Background: The prevalence of eczema varies markedly across the globe. It is unclear whether the geographic variation is due to race and/or ethnic differences, environmental exposures, or genetic factors.

Objective: We investigated the effects of ethnicity and environmental exposures on eczema in Hispanic white and non-Hispanic white children who participated in the Southern California Children's Health Study.

Methods: We performed a cross-sectional study with sociodemographic predictors and environmental exposures among Hispanic white and non-Hispanic white children ages 4–8 years enrolled in the Children's Health Study, 2002–2003.

Results: Eczema prevalence differed by ethnicity: Hispanic whites showed lower prevalence (13.8%) compared with non-Hispanic whites (20.2%), and adjustment for sociodemographic factors did not account for the ethnic difference (odds ratio [OR] 0.79 [95% confidence interval {CI}, 0.65–0.95]). Parental history of allergic disease had a larger effect in Hispanic whites than in non-Hispanic whites (p for interaction = 0.005). High maternal education level (OR 1.46 [95% CI, 1.14–1.87]), parental history of allergic disease (OR 2.21 [95% CI, 1.78–2.76]), and maternal smoking during pregnancy (OR 1.44 [95% CI, 1.06–1.95]) increased the risk of eczema. Indoor environmental factors (e.g., mold, water damage, humidifier use) increased the risk of eczema in non-Hispanic whites independent of a parental history of allergic disease, but, in Hispanic whites, increased risks were observed, primarily in children without a parental history of allergic disease.

Conclusion: Hispanic white children in southern California had a lower prevalence of eczema than non-Hispanic whites, and this ethnic difference was not accounted for by sociodemographic differences. The effects of a parental history of allergic disease and indoor environmental exposures on eczema varied by ethnicity, which indicated that the etiology of eczema may differ in Hispanic whites and in non-Hispanic whites.

(Allergy Asthma Proc 37:64–71, 2016; doi: 10.2500/aap.2016.37.3913)

Atopic eczema is one of the most common allergic diseases in children. Based on the International Study of Asthma and Allergies in Childhood epidemiologic research that was first reported in 1991, the prevalence of eczema varies from 10 to 30% worldwide and has shown a recent increase.^{1,2} In the United States, eczema prevalence in children ranged from 8.7 to 18.1%.^{3–5} The reasons for this wide range of eczema prevalence has not been established; several factors may influence disease occurrence, including genetic

variation, race and/or ethnicity, environmental exposures, and cultural factors. The role of race and/or ethnicity is supported by studies in the United States that showed that eczema prevalence is lower in whites compared with African Americans.⁴ A further understanding of the ethnic differences in prevalence is needed to support preventive intervention and individualized clinical management plans.

The developmental origins of health and disease hypothesis indicates that a child's environment from conception to age 2 greatly influences the child's risk for chronic disease.⁶ Atopic eczema is commonly diagnosed in children younger than the age of 5 years. Therefore, the environmental exposures during pregnancy or in early life could be important for the development of childhood eczema. Some of the previous studies reported that high mite allergen levels, mold in the kitchen during pregnancy,⁷ and maternal smoking in the child's first year of life^{8,9} were associated with an increased risk of childhood eczema. Among environmental factors, early life exposure to outdoor air pollution was reported to increase the risk of respiratory tract diseases but not for eczema.¹⁰

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This work was supported by a grant from the Research Year of Inje University (2014), the National Institutes of Environmental Health Sciences (grants P30ES007048, P01ES011627, R01ES021801, and R01ES023262), and the Hastings Foundation. The authors have no conflicts of interest to declare pertaining to this article. Supplemental data available at www.IngentaConnect.com

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Figure 1. Flowchart of the study participants.

Exposure to mold can trigger allergic reactions in subjects who are allergic to mold, and, furthermore, a damp indoor environment and mold exposure may cause respiratory and skin symptoms independent of allergic status.¹¹ There are many reports of investigations of the effect of indoor exposures in childhood on respiratory allergic diseases,^{12–14} but reports are limited for eczema.

In this study, we described the prevalence of eczema in southern California by using data from the Southern California Children's Health Study and investigated the factors that underlie variation in prevalence in children of Hispanic white and non-Hispanic white ethnicity focusing on socioeconomic factors and environmental exposures, especially those in the indoor environment, *e.g.*, mold-associated exposure.

METHODS

Study Subjects

Subjects ($n = 5765$) were recruited from kindergarten or first grade classrooms (5–7 years old) in the Southern California Children's Health Study, from 2002 to 2003. A parent or guardian provided informed consent and completed a written questionnaire with demographic characteristics, ethnicity, personal and familial history of allergic disease, and environmental information of where they lived at baseline and each subsequent year of follow-up. Details about the study design and methods have been reported previously.¹⁵ Questionnaires were offered in English and Spanish. For the analysis, baseline information about age and sex, and parents who answered the English questionnaire for non-Hispanic white and Hispanic white children were included to perform a cross-sectional study to investigate the differences in response patterns for eczema between the English and Spanish version of the questionnaire ($n = 3302$) (Fig. 1).

Questionnaire

Personal demographic characteristics included age, sex, ethnicity, and socioeconomic status (parent's education level, annual household income, current health insurance coverage of the child), and maternal smoking while pregnant with the child. A history of any lifetime eczema was defined by the answer to the question "Has your child ever had eczema?" Parental history of allergic disease was defined when at least one of the parents reported having physician-diagnosed asthma or allergic rhinitis.

Information on exposures and household characteristics was collected by the questionnaire and included age of the house, carpeting, pets inside the house, water damage or flooding, mold or mildew in the house since the child has lived there, use of an air conditioner and/or humidifier, second-hand tobacco smoke, and specific combustion sources for nitrogen dioxide in the home.

Statistical Analysis

Descriptive statistics on demographics and socioeconomic status were examined. Logistic regression was performed to investigate the effect of demographic and of socioeconomic and environmental factors on eczema. In addition, multivariate logistic regression was used to investigate the effect of environmental exposure on eczema prevalence when adjusting for age, sex, maternal education level, and maternal smoking during pregnancy. Variables were included to control for confounding and to assess for heterogeneity of effects based on external information. The interactions between ethnicity and parental history of allergic disease were tested by including the interaction terms in the models. Stratified analysis was performed to test the effects of multifactors among children with or without a family history by

Table 1 Demographic characteristics of non-Hispanic white and Hispanic white children in the Southern California Children's Health Study at study entry 2002–2003

	Non-Hispanic White	Hispanic White	<i>p</i> Value
No. (%)	1616 (48.9)	1686 (51.1)	
Eczema, no. (%)	326 (20.2)	232 (13.8)	<0.001
Age, mean (SD), y	6.6 (0.7)	6.6 (0.7)	0.40
Sex, M:F	841:775	857:829	0.49
Health insurance, no. (%)	1513 (94.2)	1509 (91.7)	0.005
Household income, no. (%)			
<\$15,000	80 (5.5)	222 (14.8)	<0.001
\$15,000–49,999	330 (22.8)	570 (38.1)	
≥\$50,000	1035 (71.6)	704 (47.1)	
Maternal educational level, no. (%)			
High school or less	282 (19.0)	584 (39.3)	<0.001
Some college or more	1203 (81.0)	902 (60.7)	
Parental history of allergic disease, no. (%)	975 (65.3)	813 (53.4)	<0.001
Maternal smoking during pregnancy, no. (%)	209 (13.2)	111 (6.8)	<0.001

SD = standard deviation; *M* = male; *F* = female.

their ethnicity. Significance was defined as two-sided $p < 0.05$, and all analyses were conducted by using the Statistical Analysis System (SAS version 9.2; SAS Institute Inc., Cary, NC).

RESULTS

Characteristics of the Study Population

The study included 3302 children with 1616 non-Hispanic white and 1686 Hispanic white ethnicities (Table 1). The mean (standard deviation) age was 6.6 ± 0.7 years. There were no significant differences in age or sex between non-Hispanic whites and Hispanic whites ($p = 0.40$ and $p = 0.49$, respectively). The proportion of families with health insurance or higher education levels was lower in Hispanic whites compared with non-Hispanic whites. Household income was also lower in Hispanic white than in non-Hispanic white families ($p < 0.001$). A parental history of allergic disease and maternal smoking during pregnancy were less common in Hispanic whites compared with non-Hispanic whites.

Socioeconomic Factors and Eczema Prevalence

The overall prevalence of eczema was 16.9%. High maternal education level and maternal smoking during pregnancy were associated with eczema (adjusted odds ratio [aOR] 1.46 [95% confidence interval {CI}, 1.14–1.87], and aOR 1.44 [95% CI, 1.06–1.95], respectively) (Table 2). Parental history of allergic disease was also significantly associated with eczema (aOR 2.21 [95% CI, 1.78–2.76]).

We observed that lifetime prevalence of eczema was lower in Hispanic whites (13.8%) than in non-Hispanic

whites (20.2%) (OR 0.63 [95% CI, 0.53–0.76]). Age, sex, health insurance, household income, maternal education level, parental history of allergic disease, and maternal smoking during pregnancy did not explain this ethnic difference (aOR 0.79 [95% CI, 0.65–0.95]). In addition, health insurance and household income were not associated with eczema in models that included ethnicity and other factors.

We found that the effect of parental history of allergic disease on eczema was greater in Hispanic white (OR 3.44 [95% CI, 2.45–4.83]) than in non-Hispanic white children (OR 1.83 [95% CI, 1.38–2.43]) (p for interaction = 0.005). When the subjects were stratified by ethnicity, the only sociodemographic factor that significantly increased the risk of eczema in non-Hispanic whites was parental history of allergic disease (aOR 1.72 [95% CI, 1.29–2.30]). In contrast, a high maternal education level, parental history of allergic disease, and maternal smoking during pregnancy were associated with increased risk in Hispanic whites (aOR 1.50 [95% CI, 1.07–2.12], aOR 2.99 [95% CI, 2.11–4.22], and aOR 1.86 [95% CI, 1.13–3.08], respectively) (Supplemental Table 1).

We considered the overall role of genetic differences that may explain the ethnic variation by investigating the relationship of genetic admixture with eczema among Hispanic whites. We found that variation in genetic contributions of founding populations (Native American and European) was not associated with eczema and did not explain the differences in prevalence in Hispanic whites and non-Hispanic whites (Supplemental Table 2).

Table 2 Association of selected socioeconomic factors with eczema in Southern California Children's Health Study participants at study entry 2002–2003

	No Eczema, <i>n</i> = 3221	With Eczema, <i>n</i> = 681	OR (95% CI)	aOR (95% CI)
Age, mean (SD), y	6.7 (0.7)	6.5 (0.6)	0.93 (0.82–1.06)	1.00 (0.86–1.15)
Sex, M:F	1660:1561	348:333	0.98 (0.83–1.16)	1.00 (0.83–1.20)
Ethnicity, no. (%)				
Non-Hispanic white	1290 (47.0)	326 (58.4)	1.00	1.00
Hispanic	1454 (53.0)	232 (41.6)	0.63 (0.53–0.76)	0.79 (0.65–0.95)
Health insurance, no. (%)	2910 (92.3)	646 (96.1)	1.02 (0.81–1.29)	1.45 (0.93–2.27)
Household income, no. (%)				
<\$15,000	362 (12.8)	54 (8.7)	1.00	1.00
\$15,000–49,999	897 (31.8)	167 (26.9)	1.25 (0.90–1.74)	1.19 (0.79–1.80)
≥\$50,000	1566 (55.4)	400 (64.4)	1.71 (1.26–2.33)	1.33 (0.90–1.98)
Maternal educational level, no. (%)				
High school or less	900 (31.9)	129 (20.4)	1.00	1.00
Some college or more	1926 (68.2)	504 (79.6)	1.83 (1.48–2.25)	1.46 (1.14–1.87)
Parental history of allergic disease, no. (%)	1601 (55.1)	481 (76.4)	2.63 (2.16–3.20)	2.21 (1.78–2.76)
Maternal smoking during pregnancy, no. (%)	302 (9.7)	78 (11.8)	1.23 (0.95–1.61)	1.44 (1.06–1.95)

OR = odds ratio; CI = confidence interval; aOR = adjusted odds ratios (models were adjusted for all variables shown in the table); SD = standard deviation.

Table 3 Association of individual indoor environmental factors with eczema between non-Hispanic white and Hispanic white children in Southern California Children's Health Study at study entry 2002–2003

	All Participants		Non-Hispanic White	Hispanic White
	OR (95% CI)	aOR (95% CI)#	aOR (95% CI)*	aOR (95% CI)*
Home first constructed, year				
1970s or later	1.00	1.00	1.00	1.00
Before 1970s	1.23 (1.03–1.47)	1.14 (0.94–1.39)	1.32 (0.99–1.78)	1.05 (0.75–1.47)
Use air conditioning	1.21 (1.01–1.45)	1.09 (0.88–1.34)	0.90 (0.54–1.48)	1.28 (0.83–1.99)
Water damage or flooding	1.41 (1.14–1.73)	1.36 (1.09–1.71)	1.39 (1.04–1.87)	1.22 (0.84–1.77)
Mold or mildew on the walls, ceilings, or floors	1.55 (1.29–1.87)	1.51 (1.22–1.86)	1.41 (1.06–1.88)	1.68 (1.22–2.30)
Musty odor	1.15 (0.75–1.75)	1.08 (0.67–1.74)	1.35 (0.69–2.66)	0.86 (0.42–1.75)
Ever used humidifier or vaporizer	1.69 (1.42–2.01)	1.44 (1.18–1.75)	1.47 (1.14–1.90)	1.34 (0.97–1.86)
Carpeting	1.20 (0.84–1.70)	1.19 (0.78–1.80)	1.30 (0.70–2.43)	1.02 (0.57–1.83)

OR = odds ratio; CI = confidence interval; aOR = adjusted odds ratio.

*Adjusted for age, sex, mother's educational level, parental history of allergic disease, and maternal smoking during pregnancy.

#Adjusted for age, sex, ethnicity, mother's educational level, parental history of allergic disease, and maternal smoking during pregnancy.

Effect of Indoor Environmental Factors on Eczema

The presence of water damage or flooding (aOR 1.36 [95% CI, 1.09–1.71]); mold or mildew on the walls, ceilings, or floors (aOR 1.51 [95% CI, 1.22–1.86]); and ever used a humidifier (aOR 1.44 [95% CI, 1.18–1.75]) increased the risk of eczema after adjusting for age, sex, ethnicity, maternal education level, parental history of allergic disease, and maternal smoking during pregnancy (Table 3). When the sub-

jects were stratified by ethnicity; water damage or flooding (aOR 1.39 [95% CI, 1.04–1.87]); mold or mildew on the walls, ceiling, or floors (aOR 1.41 [95% CI, 1.06–1.88]); and ever used humidifier (aOR 1.47 [95% CI, 1.14–1.90]) significantly increased the risk of eczema in non-Hispanic whites, whereas only mold or mildew on the walls, ceilings, or floors significantly increased the risk in Hispanic whites (aOR 1.68 [95% CI, 1.22–2.30]).

Table 4 Association of socioeconomic factors and individual indoor environmental exposures with eczema stratified by non-Hispanic white and Hispanic white ethnicity and parental history of allergic disease

	Non-Hispanic White Parental History of Allergic Disease		Hispanic White Parental History of Allergic Disease	
	–	+	–	+
N	519	975	711	813
Age	1.08 (0.75–1.56)	1.02 (0.81–1.28)	0.81 (0.53–1.26)	0.92 (0.71–1.19)
Sex (female)	1.37 (0.83–2.25)	0.94 (0.70–1.26)	1.65 (0.91–2.99)	0.84 (0.60–1.19)
Health insurance, no. (%)	2.85 (0.67–12.14)	1.06 (0.53–2.12)	2.76 (0.66–11.62)	1.31 (0.60–2.85)
Household income				
<\$15,000	1.00	1.00	1.00	1.00
\$15,000–49,999	0.37 (0.11–1.20)	1.75 (0.94–4.01)	3.16 (0.92–10.82)	1.56 (0.75–3.26)
≥ \$50,000	0.72 (0.26–2.04)	1.56 (0.72–3.42)	3.25 (0.95–11.10)	1.77 (0.87–3.59)
Mother's educational level				
High school or less	1.00	1.00	1.00	1.00
Some college or more	1.33 (0.71–2.48)	1.48 (0.94–2.31)	1.53 (0.83–2.82)	1.86 (1.21–2.86)
Maternal smoking during pregnancy	0.68 (0.30–1.56)	0.94 (0.59–1.51)	3.33 (1.20–9.21)	1.00 (0.52–1.93)
Home first constructed years				
1970s or later	1.00	1.00	1.00	1.00
Before 1970s	0.89 (0.53–1.49)*	1.21 (0.89–1.64)*	1.72 (0.87–3.42)*	0.93 (0.64–1.36)*
Use air conditioning	1.78 (0.96–3.31)*	0.89 (0.64–1.24)*	1.02 (0.55–1.88)*	1.03 (0.71–1.51)*
Water damage or flooding	1.25 (0.68–2.30)*	1.49 (1.05–2.10)*	2.44 (1.14–5.24)*	1.07 (0.69–1.65)*
Mold or mildew on the walls, ceilings, or floors	1.60 (0.91–2.82)*	1.43 (1.03–2.00)*	3.66 (1.93–6.93)*	1.35 (0.92–1.97)*
Musty odor	1.48 (0.40–5.49)*	1.39 (0.63–3.08)*	1.42 (0.30–6.66)*	0.74 (0.32–1.70)*
Ever used humidifier or vaporizer	1.78 (1.06–2.99)*	1.59 (1.18–2.14)*	1.91 (0.97–3.77)*	1.18 (0.81–1.73)*
Carpeting	1.83 (0.54–6.20)*	1.24 (0.61–2.53)*	1.55 (0.46–5.23)*	0.89 (0.43–1.84)*

*Adjusted odds ratio: adjusted for age, sex, mother's educational level, and maternal smoking during pregnancy.

A parental history of allergic disease showed ethnic-specific modulation of environmental associations. In analyses that stratified children by their parental history of allergic disease within each ethnicity group (Table 4), water damage or flooding and mold or mildew on the walls, ceiling, or floors increased the risk of eczema in the subjects with a parental history of allergic disease in non-Hispanic white (aOR 1.49 [95% CI, 1.05–2.10] and aOR 1.43 [95% CI, 1.03–2.00], respectively), whereas the same exposures increased eczema in the subjects without a parental history of allergic disease in Hispanic whites (aOR 2.44 [95% CI, 1.14–5.24] and aOR 3.66 [95% CI, 1.93–6.93], respectively). Humidifier use increased the risk of eczema prevalence regardless of a parental history of allergic disease in non-Hispanic whites (aOR 1.59 [95% CI, 1.18–2.14] and aOR 1.78 [95% CI, 1.06–2.99], respectively) but did not have statistically significant effects in Hispanic whites (aOR 1.18 [95% CI, 0.81–1.73] and aOR 1.91 [95% CI, 0.97–3.77], respectively). The year when the house was constructed, use of air conditioning, musty odor, and carpeting did not show statistically significant associ-

ations with a risk of eczema that depended on ethnicity or a history of parental allergic disease.

DISCUSSION

In our Southern California Children's Health Study, the eczema prevalence was 16.9% in 5- to 7-year-old children. Hispanic white children showed a significantly lower prevalence than non-Hispanic white children. The prevalence was higher in children who had a history of parental allergic disease or whose mothers had a high educational attainment, or who smoked during pregnancy. Exposure to indoor mold or humidity during childhood also affected eczema development, and the effects of these exposures differed by ethnicity, depending on the presence of a parental history of allergic disease. Genetic admixture did not explain the ethnic differences in eczema occurrence.

The overall lifetime eczema prevalence of 16.9% in our study was similar to other epidemiologic studies. The German Multicenter Atopy Study found that 17.2–20.5% of 5- to 7-year-old children had eczema. Similarly, other global studies found that 0.9–22.5% of 6- to

7-year-old children had eczema.^{16,17} Also, a study of the general population in the United States reported that the prevalence of empiric eczema was 17.1%.³ In our study, we found that eczema prevalence was 20.2% in non-Hispanic white children and 13.8% in Hispanic white children. Most previous studies that reported eczema prevalence rarely described the difference among Hispanic whites and non-Hispanic whites in the study population. Ethnicity appeared to be an important factor that should be considered in studies of eczema etiology.

Although the causes of eczema are still poorly understood, genetic predisposition with the incitement of environmental exposures is thought to be a main factor in eczema etiology. Our study demonstrated that maternal education level, parental history of allergic disease, and maternal smoking history during pregnancy increased the risk of childhood eczema. Similarly, two Polish population studies that searched for the risk factors of atopic dermatitis reported that atopic dermatitis was more common in subjects with parental atopy, higher education, and higher economic status.^{18,19} In studies based in Korea, a parental history of allergic disease was the most important risk factor for atopic dermatitis, but the other socioeconomic factors, such as parental education levels, were not.^{20,21} Based on results from these studies, a parental history of allergic disease would be the most important factor for the development of eczema, but some other studies showed an association only with a maternal history of allergic disease and not with a paternal history.^{22,23} The variability of the results may be due to the difference in the ethnicity of the enrolled populations, differences in eczema phenotype, or other factors. Ethnicity and a parental history of allergic disease indicate a genetic influence on the development of eczema; however, differences in genetic admixture do not support a major role for genetics in explaining the ethnic differences on the development of eczema.

Recent studies that searched for the developmental origins of allergic disease focused on early life risk factors.^{6,22,24,25} The influence of environmental exposure that occurred during critical developmental periods of the immune systems may increase the likelihood of allergic disease. Prenatal exposures may influence the programming and development of neonatal immune response.^{26,27} Maternal smoking is a frequently cited early life exposure that has been shown to have an effect on the development of atopic disorders.^{9,28,29} Tobacco smoke exacerbates the T-helper 2 response by increasing production of interleukin 4, interleukin 5, and other proinflammatory cytokines that result in increased allergic responses, whereas it reduces the T-helper 1 response by altering natural killer T-cell function and suppressing interferon- γ production.^{30,31} There are a few studies that do not show an association

between maternal smoking during pregnancy and eczema.^{9,32} In our study, maternal smoking during pregnancy increased the risk of eczema more in Hispanic white children, especially those who did not have a parental history of allergic disease. The discrepancies between the results may have occurred because it is difficult to pinpoint the critical window of exposure and because many prenatal exposures, *e.g.*, heavy maternal smoking in pregnancy, tend to coexist with other environmental and socioeconomic factors.^{33,34}

Environmental factors that relate to the hygiene hypothesis are thought to have protective effects on atopic dermatitis.³⁵ The parental education level may be considered as one of the factors of hygiene hypothesis, such as daycare attendance or the number of siblings.³⁶ As in previous epidemiologic studies, we found that high maternal education level was associated with eczema,^{35–37} which may reflect, in part, a bias; mothers with a high education level are more informed about the health of their children and visit the hospital then this may increased opportunities for eczema diagnosis.³⁸

Exposures in the indoor environment during pregnancy and early life may increase risks of allergic disease. Many studies reported that housing condition, dampness, and mold growth in the house are associated with respiratory symptoms, but studies related to eczema are scarce.^{11,39–43} The mechanism that mediates damp housing effects on asthma could be its relation to indoor mold, which serves as an allergenic protein or through its relation to house-dust mite.^{39–42} Besides acting as an allergen, dampness and/or mold may also operate through nonallergic mechanisms. Studies compared the effects of dampness between subjects with atopy and subjects without atopy, and observed the association of dampness with respiratory symptoms in subjects without atopy as well as subjects with atopy.^{11,43} Furthermore, an international study reported the association between a damp house condition and/or visible molds and eczema as well as respiratory allergy in both children with atopy and children without atopy.⁴⁴ In the present study, we found that water damage or flooding, mold exposure in the house, and humidifier use increased the risk of eczema in the non-Hispanic white children with a parental history of allergic disease, whereas water damage or flooding and mold exposure increased the risk of eczema in Hispanic whites with no parental history of allergic disease. For the development of eczema, dampness or mold exposure may have different mechanisms, depending on the ethnicity and genetic background.

Our results must be interpreted in light of the study design. This study was a cross-sectional study, so it had limitations in establishing a cause-and-effect relationship between indoor exposures and eczema development. Socioeconomic and environmental factors that

occurred in early life exposures may be affected by recall bias, which attenuated the reported effects. Therefore, further prospective observation of eczema and measurement are needed. Another limitation may be our case definition for eczema. We based our questions about eczema on the International Study of Asthma and Allergies in Childhood questionnaire, commonly used worldwide for epidemiologic studies, but we condensed it to make it short and simple for the convenience of the participants. A case definition that uses more detailed questions or with a physical examination by physicians would likely improve the definition of eczema. However, we think it unlikely that the misclassification of eczema is related to exposure status in a manner that would produce a major bias in our results.

CONCLUSION

Eczema prevalence was lower in Hispanic white children compared with non-Hispanic white children. Sociodemographic and genetic admixture did not explain the ethnic differences in prevalence. The effects of indoor environmental exposures on eczema varied by ethnicity as well as parental history of allergic disease. Further studies are needed to determine why eczema varied by ethnicity and to determine whether environmental factors have difference effects in Hispanic white and non-Hispanic white children.

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