

Original Article

Racial Differences in Food Allergy Phenotype and Health Care Utilization among US Children

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What is already known about this topic? There is a paucity of data in the epidemiology of food allergy (FA) as it relates to race and/or ethnicity. The limited existing data show that African American children are at an increased risk for FA and its associated morbidities, and there are no data on Hispanic children with FA.

What does this article add to our knowledge? We found that African American (AA) and Hispanic children had different food allergen profiles, higher rates of associated atopic conditions, and increased rates of FA-associated anaphylaxis and emergency department visits than white children.

How does this study impact current management guidelines? The higher rates of asthma and anaphylaxis among minority children are concerning, especially when considered in the context of increased anaphylaxis in AA children. These findings highlight the need for culturally sensitive educational programs to improve FA outcomes in these children.

BACKGROUND: Food allergy (FA) is a prevalent condition in the United States, but little is known about its phenotypes in racial minority groups.

OBJECTIVE: The objective of this study was to characterize disease phenotypes and disparities in health care utilization among African American (AA), Hispanic, and white children with FA.

METHODS: We conducted a large, 2-center, retrospective cohort study of children aged 0-17 years with FA seen in allergy/immunology clinics at 2 urban tertiary care centers in the United States. We used multiple logistic regression analyses adjusted for age, gender, and insurance.

RESULTS: The cohort of 817 children was composed of 35% AA, 12% Hispanic, and 53% non-Hispanic white. Compared with non-Hispanic white children, AA children had significantly higher odds of having asthma and eczema ($P < .01$), and significantly higher odds of allergy to wheat, soy, corn, fish, and shellfish ($P < .01$). Compared with non-Hispanic white children, Hispanic children had significantly higher odds of allergy to corn, fish, and shellfish ($P < .01$), and higher odds of eczema ($P < .01$), but a similar rate of asthma ($P = .44$). In this cohort, 55%, 18%, and 11% of AA, Hispanic, and white children were covered by Medicaid, respectively ($P < .00001$). Compared with whites, AA and Hispanic children had a shorter duration of follow-up for FA with an allergy specialist and higher rates of FA-related anaphylaxis and emergency department visits ($P < .01$).

CONCLUSIONS: FA phenotypes and health care utilization differ among children of different racial and/or ethnic backgrounds in the United States that put AA and Hispanic children at higher risks of adverse outcome than white children. These differences include coexistent atopic conditions, less well recognized food allergens, and higher rates of anaphylaxis. © 2016 American Academy of Allergy, Asthma & Immunology (J Allergy Clin Immunol Pract 2016;■:■-■)

Key words: Food allergy; Race; Ethnicity; African American; Hispanic; Asthma; Anaphylaxis

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Abbreviations used

AA- African American
 AD- Atopic dermatitis
 CCHMC- Cincinnati Children's Hospital Medical Center
 ED- Emergency department
 FA- Food allergy
 HDM- House dust mite
 NIH- National Institutes of Health
 RUMC- Rush University Medical Center

Food allergy (FA) is a major public health concern affecting approximately 8% of children in the United States¹ with an estimated economic burden of \$24.8 billion annually.² It has been well documented that the prevalence of FA has been increasing among children of all races and/or ethnicities^{3,4}; however, there is a paucity of data in the epidemiology of FA as it relates to race and ethnicity in the United States. Previous studies that have investigated FA among African American (AA) children found them to be at an increased risk for FA and its associated morbidities.^{5,6} An analysis of the National Health and Nutrition Examination Survey 2005-2006 showed that AA race is associated with higher odds of possible and likely FA compared with whites based on specific IgE measurements.⁵ Furthermore, an analysis of death certificates from the US National Mortality Database showed a higher rate of food-related fatal anaphylaxis among AAs than whites and a significant increase in the rate of fatal FA-related anaphylaxis among AAs from 1999-2001 to 2008-2010.⁷ Whether the increase in fatalities is due to differences in access to care, greater disease severity, and/or associated comorbidities is unclear and merits further research. It has been well documented that FA and food sensitization are risk factors for other common allergic conditions such as atopic dermatitis (AD) and asthma. Importantly, food-allergic reactions can result in life-threatening asthma attacks, leading to hospitalization, intubation, or death.^{8,9} AA and Hispanic children are known to be at higher risk for severe AD¹⁰ as well as uncontrolled severe asthma resulting in emergency department visits.¹¹ Therefore, understanding FA in these minority groups may decrease not only the burden of FA itself, but also its potential detrimental effects on AD and asthma in these children. This is especially important in the context of data that AA children with FA have lower odds of having a physician confirmed diagnosis of FA compared with whites.¹

Individuals of different racial backgrounds have diverse diets based on their cultural background; these differences may affect rates of sensitization to different foods as well as the impact of FA on their daily lives. Therefore, it is important to study the profile of sensitization and allergies to different foods in association with race and ethnicity. Limited existing data suggest that there are differences between ethnic and racial groups with regard to sensitization to specific foods. In a study of FA-related ambulatory care visits, AA children were found to have higher rates of sensitization to peanut, milk, and shrimp than white children.⁴ However, to date, there have been no studies evaluating differences in allergen profiles among racial and/or ethnic groups in physician confirmed FA. Furthermore, most previous FA studies have focused on limited lists of foods. Many common allergens that have not been studied are ubiquitous that can make strict avoidance harder. Therefore, it is crucial to have a comprehensive understanding of the differences

of FA between different racial and ethnic groups in association with other patient-related variables.

The objective of this study was to characterize racial and/or ethnic differences in fFA phenotype and health care utilization among food-allergic children. Specifically, we aimed to determine the prevalence of severe allergy, atopic comorbidities, and allergy to a more comprehensive list of allergens (the aforementioned foods as well as fin fish, soy, wheat, and corn) among AA, Hispanic, and white children. We also aimed to describe differences in rates of subspecialist visits among these groups.

METHODS

Subjects

The study was approved by the Institutional Review Boards of Rush University Medical Center (RUMC), Northwestern University Feinberg School of Medicine, and Cincinnati Children's Hospital Medical Center (CCHMC). A search of the electronic medical records of RUMC and CCHMC was performed by their respective information technology departments to identify children aged 0-17 years with a diagnosis of FA who were evaluated in RUMC or CCHMC Allergy clinics between October 2008 and December 2014. All data were deidentified for the statistical analysis. In both institutions, a standard electronic history and physical examination form (including results of skin prick tests and laboratory testing) specific for FA was completed during clinical encounters. This information was entered into Research Electronic Data Capture databases. Identical Excel files were created from these databases; data for each clinic were first analyzed separately and then pooled for further analyses. Patients for whom all main data points for the study were completed and retrievable were included in this analysis. Specific precaution was implemented to improve the accuracy of the data; all data were reviewed by 2 separate investigators to ensure accuracy and reliability of FA and atopic diagnoses and other health-related information. Diagnosis of FA required convincing symptoms (cutaneous, respiratory, gastrointestinal, or systemic) of an IgE-mediated reaction to a specific food and either an elevated serum-specific IgE measured by the ImmunoCAP assay (Phadia AB, Uppsala, Sweden) or a positive skin test measured by the standard skin prick test using Greer allergen extracts (Greer Laboratories Inc., Lenoir, NC) to that specific food(s). In case of tree nuts, fish, and shellfish, that are food groups, evidence of allergy to one or more types of food allergens within the group was considered a positive allergy history to that food group. For example, allergy to shrimp was considered shellfish allergy and allergy to almond was considered tree nut allergy. Diagnostic criteria for comorbidities are detailed in Table E1, available in this article's Online Repository at www.jaci-inpractice.org. In terms of food-related anaphylaxis and emergency department (ED) visits, the ED visit and/or the subsequent follow-up outpatient visits in the chart were carefully reviewed. This was to ascertain that the FA-related reaction was the primary diagnosis for that ED visit and determined as the cause of anaphylaxis and/or other severe reactions at discharge. Two centers have similar processes for follow-up appointments. At both centers, patients are called before their follow-up appointment as a reminder; both clinics will refer acute severe food-related reactions to ED and are accommodative in terms of adding patients to the clinic for follow-up of FA-related reactions.

Definitions of race used in this study were based on the National Institutes of Health (NIH) recommendation to divide individuals into 5 racial categories (American Indian or Alaska Native, Asian,

black or African American, white, and Native Hawaiian or Other Pacific Islander) and 2 ethnic categories (Hispanic or Latino and non-Hispanic or Latino). This is based on the NIH policy notice NOT-OD-01-053. As this study focuses on comparing AA, Hispanic, and white children, only data for patients in these 3 categories are reported. In this study, children of white race and Hispanic ethnicity were categorized as Hispanic, whereas children of white race and non-Hispanic ethnicity were categorized as whites. Children who were identified as AA and non-Hispanic were categorized as AA. The few children who were identified as AA race and Hispanic ethnicity were excluded because of very low number.

Statistical analysis

To examine unadjusted associations between the 2 institutions and outcomes, we used χ^2 -square tests for categorical variables and *t*-tests for continuous variables. We used a similar approach to examine the unadjusted differences in outcomes between AA, Hispanic, and white children with the addition of analysis of variance tests for the continuous variables. To calculate adjusted odds ratios and regression coefficients to compare the 3 groups of children, we estimated multiple regression models that controlled for age, insurance status, and gender. We used logistic regression models for binary outcomes and linear regression models for continuous outcomes. All statistical analyses were performed using Stata 14.0. (Stata Corp. 2009. Stata Statistical Software: Release 14. College Station, Tex).

RESULTS

Eight hundred and seventeen AA, Hispanic, and white children with a diagnosis of FA were included; 354 patients were seen at RUMC and 463 at CCHMC. Demographic and clinical characteristics are detailed in Table I. The 2 cohorts had similar age and gender distributions; however, the racial distribution at the 2 sites was different, as AA and Hispanic children were seen more frequently at RUMC than CCHMC.

The total sample consisted of 285 AA (35%), 99 Hispanic (12%), and 433 white (53%) children. The 3 racial and/or ethnic groups had similar mean ages and gender distribution (Table II). The rate of Medicaid coverage was significantly higher among African American than Hispanic and white children.

Race and/or ethnicity and types of foods associated with FA

Peanut was the most common food allergen in all 3 groups. Rates of allergy to peanut, egg, and milk were similar among the groups (Table III). Rates of allergy to corn, shellfish, and fish were significantly higher in both AA and Hispanic children than whites. Rates of allergy to wheat and soy were significantly higher in AA children than whites. The only allergen more common among white children than AA and Hispanic children was tree nut.

Comorbid allergic conditions

After adjusting for age, gender, and insurance type, AA children had significantly higher odds of comorbid asthma than white children (Table IV). Odds of eczema were significantly higher for both AA and Hispanic children in comparison with whites. Fifty-four percent of patients were evaluated for allergic rhinitis by skin prick test or serum sIgE to a standard panel of aeroallergens specific to the geographic location. The panels included trees, grasses, weeds, dust mites, molds, dog, cat, and

TABLE I. Demographic and clinical characteristics of 817* patients with IgE-mediated food allergy visited at Rush University Medical Center and Cincinnati Children Hospital Medical Center between 2008 and 2014

Characteristic	RUMC Number or mean n = 354	CCHMC Number or mean n = 463
Race		
White	99 (28.9%)	334 (72.1%)
African American	173 (48.8%)	112 (24.2%)
Hispanic	82 (23.2%)	17 (3.7%)
Gender		
Male	198 (56.2%)	305 (65.9%)
Female	154 (43.7%)	158 (34.1%)
Age in years at time of chart review (mean $\pm$ SD)	7.97 $\pm$ 4.38	7.70 $\pm$ 4.18
Asthma (%)		
Yes	146 (41.2%)	206 (44.5%)
No	208 (58.8%)	257 (55.5%)
Allergic		
Rhinitis (%)		
Yes	239 (67.9%)	259 (55.9%)
No	110 (31.0%)	204 (44.1%)
Eczema (%)		
Yes	239 (67.7%)	129 (27.9%)
No	110 (31.1%)	334 (72.1%)
Insurance (%)		
Medicaid	173 (49.0%)	72 (15.5%)
Private	180 (50.9%)	391 (84.5%)

CCHMC, Cincinnati Children's Hospital Medical Center; RUMC, Rush University Medical Center; SD, standard deviation.

The sum of the numbers for each variable does not always add up to the cohort total because of missing data. The frequency of incomplete data did not vary between the 2 centers or among different races for any of the variables.

*Initially 867 cases were qualified; however 50 cases were not included in the analyses as more than 2 data points were missing.

TABLE II. Demographic variables among different races in all food-allergic patients visited at Rush University Medical Center and Cincinnati Children Hospital Medical Center

Race group	Whites n = 433	African American (AA) n = 285	Hispanic n = 99	χ^2 -square or ANOVA P-value
Male, frequency (%)	279 (64.58)	162 (56.84)	62 (63.27)	0.107
Age in years at the time of chart review, mean $\pm$ SD	7.71 $\pm$ 4.31	8.15 $\pm$ 4.37	7.31 $\pm$ 3.72	0.187
Medicaid insurance, frequency (%)	46 (10.62)	156 (54.93)	43 (43.43)	0.0001

ANOVA, Analysis of variance; SD, standard deviation.

cockroach. There was no statistical difference among racial groups in rates of evaluation for allergic rhinitis. Among those who were evaluated, rates of allergic rhinitis were similar among racial groups (Table IV).

TABLE III. Types of food allergens among different races in food allergy patients in study at both Rush University Medical Center and Cincinnati Children Hospital Medical Center

Race group	White	African American (AA)	Hispanic
Peanut (%)	65.1	65.6	52.5
Odds ratios (95% CI)*	ref	1.12 (0.79-1.60) <i>P</i> = .521	0.65 (0.41-1.02) <i>P</i> = .061
Tree nuts (%)	45.5	18.6	25.3
Odds ratios (95% CI)	ref	0.31 (0.21-0.46) <i>P</i> = .000	0.49 (0.29-0.82) <i>P</i> = .007
Milk (%)	24.9	29.5	25.3
Odds ratios (95% CI)	ref	0.98 (0.67-1.43) <i>P</i> = .903	0.82 (0.49-1.41) <i>P</i> = .485
Egg (%)	38.3	44.6	45.5
Odds ratios (95% CI)	ref	1.27 (0.89-1.80) <i>P</i> = .185	1.19 (0.75-1.91) <i>P</i> = .457
Wheat (%)	7.4	23.2	11.1
Odds ratios (95% CI)	ref	2.95 (1.78-4.88) <i>P</i> = .000	1.31 (0.62-2.77) <i>P</i> = .478
Soy (%)	7.4	27.0	12.1
Odds ratios (95% CI)	ref	3.43 (2.10-5.62) <i>P</i> = .000	1.40 (0.67-2.09) <i>P</i> = .368
Fish (%)	3.5	34.39	16.16
Odds ratios (95% CI)	ref	11.66 (6.38-21.03) <i>P</i> = .000	4.82 (2.25-10.33) <i>P</i> = .000
Shellfish (%)	7.39	23.5	15.2
Odds ratios (95% CI)	ref	3.34 (2.02-5.55) <i>P</i> = .000	2.26 (1.14-4.49) <i>P</i> = .019
Corn (%)	2.1	15.1	7.1
Odds ratios (95% CI)	Ref	6.60 (3.00-14.51) <i>P</i> = .000	3.28 (1.16-9.28) <i>P</i> = .025

CI, Confidence interval.

*The reported odds ratios (95% CIs) were calculated in comparison with whites by logistic regression adjusting for age, gender, and insurance.

Severe food-related allergic reactions

AA and Hispanic children had significantly higher rates of food-induced anaphylaxis than white children (odds ratio [95% confidence interval]: 2.34 [1.55-3.52] and 2.88 [1.71-4.87], respectively). Furthermore, AA and Hispanic children also had higher odds of ED visits for FA-related reactions than white children (Table IV).

Follow-up with an allergist

The duration of follow-up was calculated by subtracting the age at the first visit from the age at the last visit. Although age at the first visit to an allergist was similar among racial groups, AA and Hispanic children had a shorter duration of specialist follow-up than whites (Table III). Children insured through Medicaid had significantly lower durations of follow-up (*P* = .0002) (Table E1, available in this article's Online Repository at www.jaci-inpractice.org).

DISCUSSION

To our knowledge, this is the first study designed to assess and characterize racial and ethnic differences in FA in the United States. Both AA and Hispanic children had significantly higher rates of corn, shellfish, and fish allergies compared with white children confirming unique allergen profiles by race and/or

ethnicity. Similarly, AA children had higher rates of atopic conditions including both asthma and eczema. This study also highlights important differences in FA-related health care utilization between racial and/or ethnic groups in the United States. Both AA and Hispanic children had higher rates of ED visits for FA-related anaphylaxis. Understanding differences by race and ethnicity is critical for improving management and care for all children.

The differences in rates of allergy to various foods observed in this study are likely multifactorial. The role of food introduction in infancy in the development of FA is an area of active investigation, but most experts agree that the age of introduction can influence food sensitization and allergy. In a study by Taveras et al,¹² solid foods were introduced in 34% and 41% of AA and Hispanic infants below 4 months of age, respectively, versus 13% of white infants. In the same study, rates of exclusive breastfeeding at 6 months were significantly lower in AA and Hispanic infants than white infants. A recent study investigating the timing of introduction of allergenic foods found that urban population, which were largely publicly insured and AA, introduced cow milk, egg, fish, and peanut at an earlier age than the private insurance-based, largely non-Hispanic white suburban population.¹³ Therefore, the observed racial variation in food allergen profiles may be due to differences in food introduction practices as well as familial and cultural differences in diets.

In our series, AA children had a significantly higher rate of wheat allergy than did whites. Wheat-based bread is a staple in the United States and consumed on a daily basis in most households, with the average consumption more than 130 pounds per person per year.¹⁴ Wheat allergy can therefore be a significant financial burden, especially for low-income families. It is possible that although a food-allergic child is instructed to avoid wheat, wheat-based products cannot be eliminated from his or her household, increasing the chance of accidental exposure. The higher rate of wheat allergy among AA children may explain the higher rates of anaphylaxis and ED visits observed among this population. Similarly, the rate of corn allergy was found to be significantly higher in children of Hispanic ethnicity than in whites; this could result in similar problems for families of Mexican origin, for whom corn is the main food staple.¹⁵ A prospective study capturing a detailed history during the ED visits of food ingestions at the time of the food-related reaction is needed to test this hypothesis. Shellfish allergy is a life-threatening allergy that is commonly underdiagnosed.¹⁶ Tropomyosins from shellfish and fish have a high degree of homology with house dust mite (HDM) and cockroaches.¹⁷ Shrimp tropomyosin (Pen a 1) has more than 80% amino acid sequence similarity with HDM (Der p 10),¹⁷ and cockroach (Per a 7).¹⁸ The high rate of FA to shellfish seen in some Asian countries has been attributed to the higher exposure to inhaled HDMs in these tropical regions.¹⁷ High exposure to cockroach at home has been found to correlate with higher shrimp and cockroach IgE levels.¹⁹ AA and Hispanic children from inner city areas are exposed to high levels of HDM and cockroach allergens.^{20,21} Previous studies have established that increased amount of these allergens at home is a strong risk factor for sensitization to them.²² It is possible that inhaled tropomyosins from cockroach and HDMs are the primary sensitizer for shellfish and fish allergy seen in AA and Hispanics seen in our series.

In this cohort, AA and Hispanic children had higher rates of FA-related anaphylaxis and ED visits. This finding is consistent

TABLE IV. Disease characteristics and comorbidities among different races in food allergy patients in study at both Rush University Medical Center and Cincinnati Children Hospital Medical Center

Race group	White	African American (AA)	Hispanic
Percent of cases with asthma	35.3	58.6	27.7
Odds ratios (95% CI)*	ref	2.35 (1.63-3.40) <i>P</i> = .000	0.82 (0.49-1.36) <i>P</i> = .441
Percent of cases with allergic rhinitis	57.7	67.4	56.6
Odds ratios (95% CI)	ref	1.20 (0.83-1.75) <i>P</i> = .331	0.89 (0.54-1.44) <i>P</i> = .627
Percent of cases with eczema	35.1	56.5	56.1
Odds ratios (95% CI)	ref	1.80 (1.28-2.53) <i>P</i> = .001	1.86 (1.17-2.95) <i>P</i> = .009
Percent of cases with anaphylaxis to foods	16.4	33.9	35.4
Odds ratios (95% CI)	ref	2.36 (1.59-3.521) <i>P</i> = .000	2.80 (1.70-4.64) <i>P</i> = .000
Percent of cases with ED visit for food allergy	18.2	39.7	34.3
Odds ratios (95% CI)	ref	2.68 (1.93-3.93) <i>P</i> = .000	2.14 (1.30-3.53) <i>P</i> = .003
Age in years at first allergist visit (mean ± standard deviation)	4.28 ± 3.71	4.86 ± 3.83	4.04 ± 3.28
Regression coefficients (95% CI)†	ref	0.14 (−0.15 to 0.44) <i>P</i> = .333	0.02 (−0.37 to 0.42) <i>P</i> = .908
Duration of follow-up (y)‡ (mean ± standard deviation)	3.20 ± 2.12	2.32 ± 2.38	2.22 ± 2.25
Regression coefficients (95% CI)	ref	−0.69 (−1.04 to −0.34) <i>P</i> = .000	−0.72 (−1.19 to −0.25) <i>P</i> = .003

CI, Confidence interval; ED, emergency department.

*The reported odds ratios (95% CIs) were calculated in comparison with whites by logistic regression adjusting for age, gender, and insurance.

†Regression coefficients for all numeric variables in different races were calculated in comparison with whites by linear regression adjusting for age, gender, and insurance.

‡Duration of follow-up was calculated by subtracting the age at the first visit from the age at the last visit.

with a previous population-based report showing an increased rate of fatal FA-related anaphylaxis in AA patients.⁷ The study by Jerschow et al⁷ showed that AA girls are at a nearly 2-fold greater risk of FA-related fatal anaphylaxis than white girls and that AA boys are at a 3-fold greater risk relative to their white counterparts. They reported a more than 3-fold increase in the FA-related ED visits among AA males,⁷ which is in agreement with a recent study showing that ED visits and hospital admissions for food-induced anaphylaxis increased by nearly 30% per year from 2008 to 2012 among children of all race and/or ethnicity groups in Illinois.²³ There was a significant association between ED visits and anaphylaxis in our series. Thus, the higher rate of ED visits among minorities may be indicative of increased FA severity in these groups. Furthermore, these increased rates among AA relative to white children may be linked to the increased asthma rate seen in our study. Exposure to food allergens in children with FA and asthma can result in uncontrolled asthma attacks, which could make the incident more life threatening.^{8,9} However, prospective studies are needed to determine whether rates of FA-related ED visits in these populations are affected by other factors in the system (eg, access to primary health care providers) and/or individual (eg, home management practices) levels.

Minority children in our study had a shorter duration of follow-up with an allergist than white children in our tertiary care systems. The higher rate of Medicaid coverage among minority patients is a proxy for lower income. Medicaid coverage was associated with a shorter duration of follow-up even after adjusting for race (Table E2, available in this article's Online Repository at www.jaci-inpractice.org). A recent

study showed that low-income children incur higher costs for ED visits and hospitalization, and spend less on specialty care.²⁴ A shorter follow-up duration can negatively affect FA outcomes in that the highest rates of FA-related anaphylaxis and death occur during the teenage years and early adulthood.^{25,26} For minority children at increased risk of anaphylaxis at baseline, earlier detachment from specialty care when approaching high-risk ages may be particularly detrimental. The above findings point to the need for implementation of strategies to provide better access to outpatient specialist care for all children with FA, especially those from lower income families with lower access to care. Furthermore, it calls for development and implementation of individualized programs designed to educate families with food-allergic children tailored to their cultural and socioeconomic background.

This study has several limitations inherent in retrospective studies; some of the results including ED visits or anaphylaxis might have been missed or underreported. And it is possible that the missing data for these variables are varied among different race and/or ethnicity groups. However, using emergency departments outside of the RUMC system was not different among the 3 race and/or ethnicity groups. There is significant heterogeneity among individuals of Hispanic ethnicity living in United States,²⁷ which needs to be investigated in a separate large study evaluating these groups. It is noteworthy that the current study is conducted on children with FA visited at allergy and/or immunology specialist clinics and may not represent US children with FA in general. These 2 centers were different in terms of some of the variables and racial distribution; however, as there are only 2 centers in this study, we could not apply analytical

approaches such as fixed- and random-effects modeling to completely rule out the effect of site differences on results.

In conclusion, we found that AA and Hispanic children had different food allergen profiles, higher rates of associated atopic conditions, and increased rates of FA-associated anaphylaxis and ED visits than white children. Minority children were also more likely to have Medicaid and were lost to follow-up at earlier ages; the latter finding may reflect differences in access to health care between racial and/or ethnic groups. The higher rates of asthma and anaphylaxis observed among minority children are particularly concerning, especially when considered in the context of the previously reported higher rate of FA-related fatal anaphylaxis in AA children.⁷ These findings call for future investigation of these issues; they also highlight the need for culturally sensitive educational programs to improve FA outcomes for all children.

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ONLINE REPOSITORY

TABLE E1. Description of the main variables used in the study

Variable	Definition used in the study	Variable type	Response
Asthma	Physician's diagnosis of asthma based on respiratory symptom documented in the chart	Binary	Yes/No
Allergic rhinitis	Positive skin prick test or elevated sIgE to aeroallergens documented in the chart (fixed aeroallergen panel including trees, grasses, weeds, dust mites, cockroach, molds, cat, and dog)	Binary	Positive ≥ 1 or more aeroallergens/negative to all aeroallergens
Eczema	Physician's diagnosis of atopic dermatitis	Binary	Yes/No
Food allergy	Physician's diagnosis of food allergy based on a positive history of reaction plus positive skin prick test or elevated sIgE to food allergen documented in the chart	Binary	Yes/No
FA-related anaphylaxis	History of reported immediate systemic reaction to foods that based on severity and timing symptoms is indicative of an IgE-mediated immune release of potent mediators from tissue mast cells and peripheral basophils as defined by Sampson et al*	Binary	Yes/No

The diagnosis for allergic comorbidities was considered positive if such diagnosis was made or confirmed at the first allergy visit or during the follow-up period.

*Sampson HA, Munoz-Furlong A, Bock SA, Schmitt C, Bass R, Chowdhury BA, et al. Symposium on the definition and management of anaphylaxis: summary report. J Allergy Clin Immunol 2005;115:584-91.

TABLE E2. Impact of insurance status on food allergy-related outcomes

Insurance group	No Medicaid	Medicaid
Percent of cases with anaphylaxis to foods	21.4	32.4
Odds ratios (95% CI)*	ref	1.17 (0.80-1.70) $P = .423$
Percent of cases with ED visit for food allergy	23.1	38.0
Odds ratios (95% CI)	ref	1.33 (0.92-1.92) $P = .128$
Age in years at first allergist visit (mean $\pm$ standard deviation)	4.35 $\pm$ 3.70	4.66 $\pm$ 3.73
Regression coefficients (95% CI)†	ref	0.17 (−0.13 to 0.46) $P = .263$
Duration of follow-up (y) (mean $\pm$ standard deviation)	3.20 $\pm$ 2.12	2.32 $\pm$ 2.38
Regression coefficients (95% CI)	ref	−0.54 (−0.90 to −0.19) $P = .002$

CI, Confidence interval; ED, emergency department.

*The reported odds ratios (95% CIs) were calculated by logistic regression adjusting for age, race/ethnicity, and gender.

†Regression coefficients for all numeric variables were calculated by linear regression adjusting for age, race/ethnicity, and gender.