Published in final edited form as:

Ann Allergy Asthma Immunol. 2016 March ; 116(3): 219–224.e1. doi:10.1016/j.anai.2015.12.019.

Exploring racial differences in IgE-mediated food allergy in the WHEALS birth cohort

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Abstract

Background—Suspected food allergies are the cause of more than 200,000 visits to the emergency department annually. Racial differences in the prevalence of food allergy have also been reported, but the evidence is less conclusive. Researchers continue to struggle with the identification of food allergy for epidemiologic studies.

Objective—To explore racial differences in IgE-mediated food allergy (IgE-FA) in a birth cohort.

Methods—We used a panel of board-certified allergists to systematically identify IgE-FA to egg, milk, or peanut in a multiethnic birth cohort in which patient medical history, patient symptoms, and clinical data were available through 36 months of age.

Results—Of the 590 infants analyzed, 52.9% were male and 65.8% African American. Sensitization (serum specific IgE >0.35 IU/mL) to the food allergens was significantly higher for African American children compared with non-African American children as has been previously reported. No statistically significant racial/ethnic differences in IgE-FA were observed; however, a higher proportion of African American children were designated as having peanut allergy, and the percentage of African American children with an IgE level greater than 95% predictive decision points for peanut was 1.7% vs 0.5% for non-African American children. With the use of logistic regression, race/ethnicity was not significantly associated with IgE-FA (adjusted odds ratio, 1.12; 95% confidence interval, 0.58–2.17; $P = .75$) but was associated with sensitization to more than 1 of the food allergens (adjusted odds ratio, 1.80; 95% confidence interval, 1.22–2.65; $P = .003$).

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Disclosures: Authors have nothing to disclose.

Supplementary Data Supplementary data related to this article can be found at <http://dx.doi.org/10.1016/j.anai.2015.12.019>.

Conclusion—We did not observe an elevated risk of IgE-FA for African American children, although established differences in sensitization were observed. Racial/ethnic differences in sensitization must be taken into consideration when investigating disparities in asthma and allergy.

Introduction

Suspected food allergies are the cause of more than 200,000 visits to the emergency department annually.¹ Food allergies directly affect the quality of life of affected children and their families. There is compelling evidence that food allergy is increasing in the United States and other countries. Racial differences in the prevalence of food allergy have also been reported, but the evidence is less conclusive. Sicherer et al² found a higher prevalence of self-reported seafood allergy among African Americans responding to a telephone survey using random digit dialing; however, other studies have reported no significant differences³ or report differences only in sensitization.^{4,5} Variations in the definition of food allergy for these studies may be the reason for the inconsistencies.

Researchers continue to struggle with the identification of food allergy for epidemiologic studies. Investigators typically use surveys or clinical evidence of sensitization. A limitation of using surveys to identify food allergy is that respondents may confuse food intolerance with an allergic reaction to food allergens.⁶ Measuring IgE is also less than ideal in that a clinical diagnosis of food allergy is based on symptoms that occur on the ingestion of food, which cannot be accurately predicted by sensitization.⁷ Although sensitization is a major risk factor for IgE-mediated food allergy (IgE-FA), a substantial number of sensitized persons will not respond to an oral food challenge, which is still considered the gold standard for the clinical diagnosis of IgE-FA.^{7,8} However, double-blind, placebo-controlled oral food challenges (DBPCFCs) are seldom feasible for large studies, and there is a substantial tendency for false-negative results because the test may not reproduce what occurred when the patient actually experienced the adverse reaction(s).⁷ We did not find any studies that suggested a racial difference in food allergy based on evidence using oral food challenges, yet evidence of a racial difference in the prevalence of food allergy could help researchers explain persistent racial differences in asthma and allergy prevalence, morbidity, and mortality. As an alternative to self-report of food allergy, the use of sensitization to food allergens alone as a surrogate measure, or the conduct of DBPCFCs, we established a physician panel of allergists to systematically identify cases of food allergy in a multiethnic birth cohort in which a bundled portfolio of patient medical history, clinical data, and self-reported symptoms was available.⁹ The objective of the present analysis is to explore whether African Americans have more IgE-FA compared to other racial/ethnic groups in the cohort, using the results of the physician panel to identify cohort members with IgE-FA.

Methods

Study Cohort

All aspects of this research were approved by institutional review boards responsible for the conduct of research in human subjects at Henry Ford Health System (HFHS) and Georgia Regents University. Eligibility and recruitment for the Wayne County Health, Environment, Allergy, and Asthma Longitudinal Study (WHEALS) birth cohort are described in a

previous publication.¹⁰ The WHEALS cohort was established to identify environmental factors related to the development of allergy and asthma in infancy and childhood. Recruited participants were pregnant women aged 21 to 45 years who were seen for prenatal care in 1 of 5 HFHS obstetric clinics from September 1, 2003, through November 26, 2007. The women resided in the city or western suburbs of Detroit as defined by zip code, spoke English well enough to provide written informed consent, and had plans to stay in the Detroit area for 2 years after delivery. Collection of blood and interview data for the cohort has been described previously and is summarized below.^{9,10}

Blood Collection

Placental cord blood specimens were collected after delivery and analyzed for total serum IgE level. In addition, research field staff collected venous blood samples from infants during home visits at 1, 6, and 12 months of age for total serum IgE measurements and during a 24-month clinic visit to measure total and allergen specific serum IgE. Specific serum IgE measurements were determined for 11 allergens, including the food allergens milk, egg, and peanut. Serum IgE measurements were conducted using the Pharmacia UniCAP system (Phadia; Thermo Fisher Scientific Inc, Waltham, Massachusetts). Food sensitization was defined as a food specific IgE level of 0.35 IU/mL or higher.

Skin Prick Tests

During a 24-month clinic visit, infants underwent skin prick tests (SPTs) for food allergens to milk, egg, and peanut. SPTs were performed using a Duotip-test device (Lincoln Diagnostics Inc, Decatur, Illinois). A positive SPT result to a food allergen was defined as having a wheal diameter 3 mm or greater than the saline control test. The allergy testing was performed under the guidance of a board-certified allergist (C.N., R.M., E.Z.). SPT results were discussed with the patient's guardian at the time of the testing. At the parent/guardian's request, a letter with the testing results was sent to the child's pediatrician. A standardized assessment sheet was completed by the allergist at the 2-year visit and sent to the study's principal investigator, who reviewed the results to see whether there was a positive allergic reaction and/or history that required further follow-up as noted by the evaluating allergist.

Parent Interviews

Field staff interviewed parents when the infant was 1, 6, 12, and 24 months of age. These interviews addressed infants' episodes of illness, atopic dermatitis or eczema, allergy and allergic reactions, and rashes and also collected information related to family medical history. Parents were also asked about any prescriptions or medical visits for the infant. A follow-up parent interview was administered when the child was 3 to 6 years of age (mean age, 4.3 years), including specific items on infant food avoidance, gastrointestinal symptoms, and onset and duration of reactions to food. Data collected during these interviews were supplemented by a review of the infant's medical record. For infants born outside the HFHS, medical records were requested and reviewed. Medical record reviews included assessments of all clinical encounters, with an emphasis on any diagnoses of food allergy, possible allergic disease diagnoses for related conditions (eg, eczema), any reports of foods causing symptoms or health problems, and the infant age at time of diagnosis or parental report of symptoms.

Determination of Food Allergy

A physician panel of 2 board-certified generalist allergists (C.N., R.M.) was established to classify cohort participants as to the presence or absence of IgE-FA to milk, egg, and peanut.⁹ The physician panel was presented with data collected from the parent interview, which included information on infant gastrointestinal symptoms suggestive of food intolerance or food allergy; other potential allergic reactions, such as rash, respiratory difficulty, wheezing, or colic; any report of food avoidance; and clinical assessments (serum total and specific IgE and SPT results). For efficiency, infant data were forwarded to the panel only if 1 or more of the following criteria were met for milk, egg, or peanut allergens: (1) 1 or more specific IgE levels of 0.35 IU/mL or higher, (2) a positive SPT result, or (3) parental report of infant symptoms potentially related to food allergy *plus* at least one specific IgE level of 0.10 IU/mL or higher (eTable 1 and eTable 2). This level of IgE was not used to define sensitization but was used to ensure that those infants with even a low probability of IgE-FA would be reviewed by our physician panel.

Infant data were presented to the panel in a standardized, web-based format. To enhance standardization in classifying infants as to the presence of IgE-FA, physicians were asked to combine professional experience with investigator-developed protocols based on the Guidelines for the Diagnosis and Management of Food Allergy in the United States.^{11,12} Infants were classified as highly probable, likely, and unlikely to have had IgE-FA. A third allergist (R.E.) independently reviewed and ruled on discordant decisions.

Statistical Analysis

The study population for these analyses includes cohort children with 6- or 12-month interview data, data from the clinic visit at 2 to 3 years, and specific IgE measures at 2 to 3 years. For this analysis, atopic phenotypes included food allergy as determined by physician panel (highly probable or likely); allergen specific IgE to egg, milk, or peanut; positive SPT results to egg, milk, or peanut; and the 95% predicted decision (PD) points for IgE established by Sampson et al.¹³ These are derived from “specific IgE antibody levels recommended for identifying patients with a >95% probability of reacting to a food challenge” (pg 895).¹³ The χ^2 tests were used to make subgroup comparisons of participant characteristics for binary and categorical variables, and *t* tests were used for comparison of continuous variables.

Logistic regression was used to calculate odds ratios (ORs) and corresponding 95% confidence intervals (CIs) to describe the association between race and food allergy and between race and sensitization, adjusting for potential confounders. Independent variables included in the model were reported before delivery and included yearly household income less than \$40,000, maternal educational level of high school or less, and unmarried status. In addition, from the 1- and 6-month survey, we collected mother's report of infant race, infant sex, breastfeeding for less than 6 months, infant exposure to environmental tobacco smoke during mother's pregnancy, and current smoking in child's household. Independent variables were retained in the final model if their inclusion changed the estimate of effect between race and food allergy or race and sensitization by more than 15%.¹⁴

Results

Of the 1258 infants enrolled in the study, 590 (46.9%) were included in the analysis (Fig 1). Demographic characteristics of included participants are provided in Table 1. The mean (SD) maternal age at infant birth was 29.8 (5.2) years, and 52.9% of the infants included in the analysis were male. A total of 65.8% of the study sample self-reported non-Hispanic black/African American race. The remaining 34.2% were categorized as non-black/African American and included 138 white infants (68.3%), 30 Hispanic/Latino infants (14.9%), and 34 infants of other races (16.8%), which included 1 American Indian/Alaska Native infant, 17 Asian infants, 12 Middle Eastern infants, and 4 who reported more than one race. Compared with the 668 not included in the analysis, those included in the analysis were significantly more likely to be male ($P = .03$) and have parents who were married ($P = .02$) with higher levels of education and household income (both $P < .001$).

Table 2 gives the estimated prevalence of IgE-FA, by race, as determined by the allergist panel (C.N., R.M.). The panel reached consensus for 238 (85.0%) of 280 infants, whereas 42 decisions (15.0%) were finalized with input from a third allergist (R.E.). Of the 41 children found to have IgE-FA (Table 2), 12 (29.3%) had at least one medical encounter documented in the medical record with an International Classification of Diseases, Ninth Revision (ICD-9), code for food allergy according to retrospective medical record review.

Prevalence for milk and egg IgE-FA was similar by race (Table 2). A higher proportion of African American infants were designated as having peanut allergy, but this finding was not significantly different by race. An IgE level of 0.35 IU/mL or greater to each of 3 food allergens was significantly higher for African American children compared with non-African American children. There was no difference in the percentage of children meeting IgE 95% PD points for milk and egg by race. Although not statistically significant ($P = .43$), the percentage of African American children with IgE levels greater than 95% PD points for peanut, however, was 3 times higher than that of non-African American children (1.7% vs 0.5% for African Americans and non-African Americans).

Table 3 gives the results of logistic regression analyses to determine the independent association between ethnicity and the outcomes of IgE-FA (by physician panel review) and by sensitization, adjusting for potential confounders. For the model using IgE-FA as the dependent variable, no variables changed the estimate between race/ethnicity and IgE-FA by more than 15%. A final model included only race/ethnicity with an adjusted OR of 1.12 (95% CI, 0.58–2.17; $P = .75$). For an IgE level of 0.35 IU/mL or higher, the adjusted OR was 1.80 (95% CI, 1.22–2.65; $P = .003$) for a model containing race/ethnicity and marital status.

Discussion

Obtaining accurate food allergy prevalence estimates for epidemiologic studies is challenging. The gold standard of DBPCFC is seldom feasible in a large-scale study. Potential study limitations can result from the use of questionnaires. For example, recall bias is a potential limitation to this study. We believe this type of bias has been minimized due to

the collection of clinical and objective data periodically at key time points that span the child's lifetime (birth through 3–6 years of age). In addition, physician panel members took into consideration clinical data and report of symptoms. A major limitation of questionnaires is the difficulty respondents may have in being able to distinguish a variety of adverse food reactions from an IgE-mediated response to ingested foods. The National Health Interview Survey asks about “food or digestive allergy,” but without report of a physician diagnosis, the problem of misclassification remains. In light of these problems, we used a physician panel to obtain an estimate of IgE-FA for research purposes. The question of an increased risk of food allergy for persons of African ancestry has been difficult to assess because of problems with the outcome selected and the way the outcome is defined. It is clear that food allergy and sensitization are not the same. Because sensitization is a very important and quantifiable component of food allergy, investigators often use this outcome as an indicator of the risk of food allergy. Most studies finding racial differences in sensitization have inferred that there may be racial differences in food allergy as well. Using multivariate logistic regression, we did not observe a statistically significant elevated risk of IgE-FA for African Americans, although the previously established elevated risk of sensitization was observed.

There is no consensus in the literature regarding racial differences in food allergy. Although early reports^{3,16} cite a higher rate of shellfish allergy among African American respondents compared with European Americans, the overall prevalence of any food allergy was similar by race. Previous studies^{5,17} used the National Health and Nutrition Examination Survey to explore patterns of food allergy and reported an increased risk for “non-Hispanic blacks,” males, and children. In this study, likely food allergy and probable food allergy were determined using previously published cut points for serum specific IgE. Using surveys from the National Center for Health Statistics, Branum et al⁴ reported that *increases* in food allergy from 1997 through 2007 were evident across 3 racial/ethnic groups (non-Hispanic white, non-Hispanic black, and Hispanic), but non-Hispanic white children had the highest reported prevalence of food allergy using the National Health Interview Survey. Sicherer et al¹⁵ examined medical records of children seen in an urban hospital-based pediatric clinic and reported that black children were significantly more likely than other racial/ethnic groups (consisting primarily of Hispanic and multiracial children) to be allergic to peanut and shellfish and to be allergic to more than one food. Authors noted that confirmation of food allergy was evident for less than half of the children. Gupta et al¹⁸ found that non-Hispanic black and Asian children were more likely to have parental report of food allergy than non-Hispanic white children but were less likely to have it confirmed. Hannaway et al,¹⁹ however, reported a lower prevalence of food allergy among nonwhite students compared with white students using prescriptions for injectable epinephrine on file in school records. Systematic reviews on the topic also differ, with some indicating there is a racial difference²⁰ and others reporting no evidence.²¹ Problems cited in these reviews included use of different definitions for food allergy, including sensitization, self-report of a physician diagnosis, and the results of a food challenge.

Joseph et al²² previously published on racial differences in physiologic parameters related to asthma. They noted that the association between spirometric factors, such as response to methacholine, and IgE appeared to differ by race. In that analysis conducted among youth of

a middleclass suburb, the authors described how total IgE increased with increasing methacholine reactivity for European American children, as might be expected, but they did not observe a similar trend for African American youth. They concluded that the association of IgE to asthma indicators differed by race. In the present analysis, we have come to a similar conclusion. IgE is influenced by factors related to ancestry, and therefore, its association with certain outcomes (asthma and allergy) must be described in that context.²³ The known tendency for African Americans to have higher IgE levels must come into play when investigating racial disparities in biological parameters for asthma and allergy.²³ When using cut points beyond the traditional 0.35 IU/mL or higher, vestiges of difference still persist but are difficult to interpret without larger sample sizes. For example, in Table 2, IgE levels for peanut are 77.9% and 240% higher using cut points of 0.35 IU/mL or higher and 95% PD points, respectively, for African Americans vs non-African Americans. This finding is similar to what was reported for the Boston cohort, in which African Americans were found to have a higher prevalence of sensitization to peanut. Kumar et al,²⁴ however, focused only on sensitization, although for peanut, the investigators dichotomized serum IgE levels using greater than 5 kUA/L as a cutoff, which is associated with a higher likelihood of clinical reactivity.

Although food allergy is increasing, the condition remains difficult to study, and questions remain regarding racial differences in prevalence. More research is needed that can provide guidance in the appropriate interpretation of serum specific IgE and corresponding PD points, given African ancestry. Because only 3% to 6% of infants will develop food allergy in childhood, larger ethnically diverse cohorts are also needed to fully explore food allergy etiology and risk.

Acknowledgments

We acknowledge the families that have participated in WHEALS. We appreciate your trust. We also thank the research staff who labored diligently at data collection. We appreciate your hard work.

Funding Sources: The studies reported in this publication were supported by the grants R01AI50681 and 1R21AI080066-01A1 from the National Institutes of Health.

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Figure 1. Study population. SPT indicates skin prick test; WHEALS, Wayne County Health, Environment, Allergy, and Asthma Longitudinal Study.

Table 1Characteristics of the 1,258 study participants^a

Characteristic	Included in analysis (n = 590)	Not included in analysis (n = 668)	P value
Maternal age at infant birth, mean (SD), y	29.8 (5.2)	29.4 (5.3)	.18
Race/ethnicity			.11
Non-Hispanic black/African American	388 (65.8)	434 (65.0)	
All others ^b	202 (34.2)	234 (35.0)	
Infant sex ^c			.03
Male	312 (52.9)	312 (46.8)	
Female	278 (47.1)	355 (53.2)	
Medicaid			.72
Yes	187 (31.7)	170 (32.7)	
No	403 (68.3)	350 (67.3)	
Marital status			.02
Married	382 (64.7)	391 (58.5)	
Unmarried	208 (35.3)	277 (41.5)	
Maternal education level greater than bachelor's of science			<.001
Yes	196 (33.2)	155 (23.2)	
No	394 (66.8)	513 (76.8)	
Annual household income <40,000			<.001
Yes	190 (32.2)	287 (43.0)	
No	322 (54.6)	308 (46.1)	
Refused/don't know	78 (13.2)	73 (10.9)	
Parental history of asthma or allergy			.89
Yes	246 (45.4)	279 (45.8)	
No	296 (54.6)	330 (54.2)	
Breastfed for >6 months			.008
Yes	156 (26.8)	97 (19.9)	
No	427 (73.2)	391 (80.1)	
Maternal smoking during pregnancy			.06
Yes	59 (10.0)	90 (13.5)	
No	531 (90.0)	578 (86.5)	
Current smoker in household ^d			.48
Yes	120 (20.3)	38 (18.1)	
No	470 (79.7)	172 (81.9)	
Complementary food <4 months			.83
Yes	233 (39.5)	176 (38.9)	
No	357 (60.5)	277 (61.1)	

^aData are presented as number (percentage) unless otherwise indicated.^bIncludes 138 white infants (68.3%), 30 Hispanic/Latino infants (14.9%), and 34 infants of other races (16.8%).

^cOne infant with missing data.

^dCurrent at 2- to 3-year interview.

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Estimated prevalence of food allergy and sensitization to egg, milk, or peanut and IgE PD points¹⁵ for each food allergen, restricted to infants with measures of specific IgE

Table 2

	Milk (n = 566) ^a			Egg (n = 567)			Peanut (n = 560)			Any(n = 558) ^b		
	African American, no. (%)	Non-African American, no. (%)	P value	African American, no. (%)	Non-African American, no. (%)	P value	African American, no. (%)	Non-African American, no. (%)	P value	African American, no. (%)	Non-African American, no. (%)	P value
IgE-FA ^c	6 (1.6)	3 (1.5)	.99	20 (5.4)	11 (5.6)	.95	17 (4.7)	7 (3.6)	.54	27 (7.4)	14 (7.2)	.91
IgE 0.35 IU/mL	128 (34.8)	51 (25.8)	.03	101 (27.4)	38 (19.2)	.03	50 (13.7)	15 (7.7)	.03	157 (43.2)	63 (32.3)	.01
IgE 95% PD points	3 (0.8)	0 (0)	.56	8 (2.2)	5 (2.5)	.78	6 (1.7)	1 (0.5)	.43	13 (3.6)	6 (3.1)	.75

Abbreviations: IgE-FA, IgE-mediated food allergy; PD, predictive decision.

^aNumber with specific IgE measures for this food allergen, including 24, 23, and 30 infants with missing specific IgE data for milk, egg, and peanut, respectively.

^bDenominator is persons with specific IgE measures for all 3 food allergens and excludes 32 infants missing at least one food specific IgE.

^cAs determined by physician panel and combines highly probable and likely.

Table 3

Results of logistic regression for the association of race to food allergy and to sensitization to milk, egg, or peanut among children in the WHEALS birth cohort^a

Variable	Black/African American	
	aOR (95% CI)	P value
IgE-mediated food allergy ^b	1.12 ^c (0.58–2.17)	.75
IgE 0.35 IU/mL	1.80 ^d (1.22–2.65)	.003

Abbreviations: aOR, adjusted odds ratio; CI, confidence interval; WHEALS, Wayne County Health Environment Allergy and Asthma Longitudinal Study.

^aVariables available for logistic regression analysis were those listed in Table 1. Variables were retained in the final model if their inclusion changed the estimate of effect between race and the dependent variable (food allergy as determined by physician panel or food sensitization defined as specific IgE level 0.35 IU/mL) by 15% or more.¹³

^bAs determined by physician panel.

^cNo variables changed the aOR by 15% or more for food allergy as determined by physician panel.

^dAdjusted for mother's marital status.

eTable 1Geometric mean total IgE by race for subgroups of study sample by race^a

Status	Sample size	Geometric total IgE, mean (SE)			<i>P</i> value ^b
		All	African American	Non-African American	
Final determination is IgE-FA	52	101.8 (4.5)	98.4 (4.4)	110.7 (5.3)	.99
Final determination is no IgE-FA					
No potential for food allergy	310	11.3 (3.2)	12.1 (3.2)	10.3 (3.2)	.12
Met criteria for panel review	280	52.9 (3.3)	51.8 (3.3)	55.8 (3.3)	.61
Panel consensus is no IgE-FA	228	46.7 (3.0)	45.7 (3.0)	48.9 (2.8)	.42

Abbreviation: IgE-FA, IgE-mediated food allergy.

^aThese subgroups correspond to Figure 1.^b*P*value for African American vs non-African American comparison.

eTable 2

Guidelines Provided to Physician Panel for the Determination of IgE-Mediated Food Allergy in a Birth Cohort

Allergen	Highly probable	Likely	Unlikely
Milk	Specific IgE 15 IU/mL ^a AND a SPT negative control OR a convincing medical history AND SPT >3 mm OR specific IgE >0.1 IU/mL	Convincing medical history AND specific IgE 0.1 IU/mL	No, equivocal, or weak medical history AND specific IgE <2 OR SPT 3 mm
Egg	Specific IgE 7 IU/mL ^a AND a SPT negative control OR a convincing medical history AND SPT >3 mm OR specific IgE >0.1 IU/mL	Equivocal or weak medical history AND specific IgE 2 IU/mL <95% PD points OR SPT >4 mm	No, equivocal, or weak medical history AND specific IgE <2 OR SPT 3 mm
Peanut	Specific IgE 14 IU/mL ^a AND a SPT negative control AND a convincing history AND SPT >3 mm OR specific IgE >0.1 IU/mL	Equivocal or weak history AND specific IgE 2 IU/mL <95% PD points OR SPT >4 mm	No, equivocal, or weak history AND specific IgE <2 OR SPT 3 mm

Abbreviations: PD, predictive decision; SPT, skin prick test.

^a Adapted from Adverse Reactions to Food Committee of American Academy of Allergy, Asthma & Immunology. Work Group Report: Oral Food Challenge Testing. *J Allergy Clin Immunol.* 2009; 123(6 suppl):S365–S383.