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Racial/ethnic and socioeconomic differences in self-reported food allergy among food-sensitized children in National Health and Nutrition Examination Survey III

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Over the past several decades, self-reported food allergy has increased rapidly among all racial/ethnic groups in the U.S., but it appears that this increase has been sharpest for non-Hispanic Black children.¹ Although non-Hispanic Black children have much higher rates of sensitization to common foods than children of other race/ethnicities,² changes in food sensitization are unlikely to be the reason for the recent increase in food allergy, as we recently demonstrated that IgE sensitization to common foods did not increase among any racial/ethnic group from the early 1990s to the mid-2000s.³ It is additionally possible that non-Hispanic black children who are food sensitized were historically less likely to report food allergy. However, to our knowledge, there have been no population-based studies in the U.S. that have directly examined this question. Here, using a nationally representative sample (the National Health and Nutrition Examination Survey [NHANES] III) in which we recently measured food specific IgE (sIgE), our goal was to determine whether there were racial/ethnic and socioeconomic differences in the relationship between sensitization to common foods and self-reported food allergy.

Participants aged 6–19 years from NHANES III part 2 (1991–1994) were included. Frozen stored sera was recently analyzed for peanut, milk, egg, and shrimp-specific IgE by ImmunoCap as previously described.³ Food sensitization was defined as peanut, milk, egg, or shrimp specific IgE ≥ 0.35 kU/L. Self-reported food allergy was defined as a positive response to: “within an hour after eating something, has [the subject] ever had a severe reaction, such as itching all over, trouble breathing, flushing, hives, or swelling of the face or hands or feet?” Race and/or ethnicity was obtained by self-report and was categorized as “non-Hispanic white” (“white”), “non-Hispanic black” (“black”) and “Mexican-American.” Household income was defined as the ratio of family income to the poverty threshold as defined by the US Census Bureau (Poverty Income Ratio [PIR]), and a PIR < 1 was considered “Poor.”

Overall, 2,673 children aged 6–19 had sIgE measured and questionnaire data regarding food reactions (eTable 1). A total of 23.5% (95% CI: 21.0 – 26.2) were sensitized to peanut, milk, egg or shrimp, 8.0% (95% CI: 6.2–10.3%) reported food allergy, and 2.7% (95% CI: 1.8–4.0) were both sensitized to food and reported food allergy (Table 1). Sensitization to foods was significantly higher among black than white or Mexican-American children (Table 1). In contrast, self-reported food allergy was significantly less common among black and Mexican American children than white children (Table 1, $p=0.001$ and $p=0.02$ respectively).

The relationship between sensitization to any food and self-report of food allergy was strongly dependent on race/ethnicity and income (eFigures 1 and 2). When examining the individual foods, peanut sensitization and self-reported food allergy were strongly associated in white children, but this was not seen among black or Mexican-American children (eFigure 1). Similarly, there was a relationship among these measures only among higher income children (eFigure 2). Race/ethnicity did not explain the poverty association completely, nor vice versa (data not shown). Similar results were seen for egg, but not milk or shrimp sensitization; shrimp sensitization had no relationship with report of food allergy overall or in any group (data not shown).

In conclusion, we found that the relationship between self-reported food allergy and food sensitization was strongly dependent on race/ethnicity and socioeconomic status in a population based sample from the early 1990s, suggesting historical under-recognition of food allergy among poorer and minority children, or alternatively over-recognition among high-income and non-minority groups. Although sensitization to foods is a poor marker for clinical food allergy,⁴ we would expect some relationship between food sensitization and clinical food allergy, and thus self-reported allergy, as we indeed found for certain groups here. The fact that this relationship was seen only among higher income and white children may explain why, historically, there were not disparities in reported food allergy despite higher levels of sensitization to common foods among non-Hispanic black children..

Other evidence to support differential recognition and diagnosis of food allergy by race/ethnicity and income includes Gupta and coworkers' finding that poor and minority children are less likely to receive a physician's evaluation when food allergy is suspected,⁵ and our recent observation that despite very high rates of clinical food allergy in inner-city children when rigorously assessed,⁶ children living in inner-city areas report less food allergy than their suburban and affluent counterparts after adjusting for race/ethnicity.⁷

Differences in recognition, however, may not solely explain these findings; it is also possible that the relationship between sensitization and food allergy varies among different racial/ethnic and socioeconomic groups. Differences in allergen introduction, for example, could lead to differences in the relationship between sIgE and food allergy symptoms. In a recent clinical trial, early introduction of allergenic foods did not affect IgE sensitization, whereas clinical allergy and skin prick wheal size were suppressed.⁸ It is possible that in response to expert opinion, higher income families delayed introduction of allergenic foods in the late 1980s, and that this trend spread to other groups later.

We further found that the association between food sensitization and self-reported food allergy was different for peanut, egg, milk, and shrimp. Whereas there was an overall correlation between sensitization to peanut, egg, or milk and self-reported severe reactions to food, this was not seen with shrimp, for which there was no relationship between sensitization and self-reported allergy in any group. This finding suggests that alternative exposures, such as dust mite or cockroach, may be driving shrimp sensitization, as has been previously hypothesized,⁹ and that much of shrimp sensitization may be clinically meaningless. In terms of the other foods, the relationship between peanut and egg sensitization and self-reported allergy was most strongly affected by race/ethnicity and income. This may reflect the fact that all participants were over 6 years of age, when most milk allergy has resolved, or may reflect different patterns of introduction and recognition of symptoms for the milk allergens.

Limitations of this analysis include the fact that food allergy is defined based on self-report, which has been shown to overestimate food allergy when defined by positive oral food challenges.¹⁰ However, as the question used in this survey assessed acute reactions, positive responses are more likely to represent true IgE-mediated food allergy. Furthermore, sIgE was only measured to peanut, milk, egg, and shrimp, which do not comprise all of the known food allergens. These allergens, however, account for approximately 80% of food allergy in children.¹¹ In addition, these are historical data, with no comparison available to current times. Because self-reported food allergy has increased among non-Hispanic blacks in recent times, we might expect that a current study would show more correlation between sensitization and self-reported food allergy among all racial/ethnic groups.

These limitations are balanced by the large representative sample, rigorous procedures within NHANES, and measurement of sIgE in an unselected population. To our knowledge, there are no other population-based studies that have examined the contribution of demographic features to the relationship between self-reported food allergy and markers of sensitization.

In summary, our findings support a hypothesis that there was uneven recognition of food allergy in the past, with more recognition among higher income and non-minority groups. Combined with our recent finding that sIgE to peanut, milk, egg, and shrimp did not change across a period where self-reported food allergy increased, this finding suggests that changes in recognition of food allergy may underlie recent increases in the report and diagnosis of this condition. However, we cannot exclude the possibility that environmental exposures correlated with socioeconomic status modify the relationship between sensitization and clinical food allergy.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Distribution of food sensitization and self-report of food allergy by race and sex (n=2,673).

	All Foods sIgE	p	Peanut sIgE	p	Egg sIgE	p	Milk sIgE	p	Shrimp sIgE	p	FA	p	FA + sIgE*	p
Total	23.5 (21.0–26.2)	N/A	10.1 (8.1–12.3)	N/A	3.0 (2.2–4.1)	N/A	8.5 (7.1–10.2)	N/A	11.3 (9.7–13.2)	N/A	8.0 (6.2–10.3)	N/A	2.7 (1.8–4.0)	N/A
Race/Ethnicity														
White	17.2 (14.4–20.4)		9.0 (6.9–11.7)		3.0 (1.9–4.8)		5.6 (4.1–7.5)		6.6 (4.8–9.1)		9.0 (6.7–12.0)		2.5 (1.5–4.2)	
Black	36.0 (33.4–38.6)	<0.001	13.0 (10.9–15.4)	0.39	4.9 (3.8–6.2)	0.005	12.8 (11.0–14.8)	<0.001	20.6 (18.2–23.3)	<0.001	4.5 (3.4–5.9)	0.09 [†]	2.1 (1.5–3.0)	0.05
Mexican-American	28.5 (23.9–33.5)		9.8 (7.2–13.1)		2.2 (1.3–3.5)		12.2 (9.9–14.9)		12.5 (10.3–15.1)		4.9 (3.3–7.3)		0.9 (0.4–2.1)	
Sex														
Male	25.6 (22.2–29.3)	0.04	11.9 (8.9–15.7)	0.06	3.2 (2.1–5.0)	0.43	9.6 (7.5–12.3)	0.17	12.0 (9.7–14.7)	0.40	8.6 (6.2–11.7)	0.57	3.6 (2.2–6.1)	0.09
Female	21.2 (18.3–24.4)		8.1 (6.1–10.8)		2.7 (1.9–3.8)		7.4 (5.6–9.7)		10.7 (8.7–13.0)		7.5 (5.2–10.7)		1.7 (0.9–3.3)	

* Food-specific IgE to peanut, egg, milk or shrimp. Values listed as percentages (95% Confidence Intervals); FA: Food Allergy, PN: Peanut, N/A: not applicable; P values obtained by chi-squared test.

[†] p<0.001 for Black versus White and p=0.02 for Mexican-American versus White.