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The Associations between Eczema and Food and Inhalant Allergen Specific IgE vary between Black and White Children

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To the Editor

The Wayne Tri-county Health & Environment Allergy Study (WHEALS) birth cohort recruited pregnant women without regards to their allergic status. Prior WHEALS analyses demonstrated that by age 2 years Black children were more likely than White children to develop eczema/atopic dermatitis (AD); have higher total IgE; and, be atopic.(1) Black children were also more likely to be sensitized to each tested allergen (food and inhalant).(2) The goal of the present analyses was to investigate whether the associations between eczema/AD and IgE levels (total IgE and food and inhalant allergen-specific IgEs (sIgEs)) differed for Black children and White children in WHEALS.

At age 2 years, children underwent a clinical examination by a study-trained doctor. Blood was collected for measurement of sIgE for the following allergens: milk, egg, peanut, *Alternaria*, cat, cockroach, dog, *Dermatophagoides farinae* (Der f), short ragweed, and timothy grass. Physicians responded to the following question: “By your clinical evaluation do you believe that this child has or has had atopic dermatitis or eczema?” Hereafter, called eczema. Child race was based on maternal report. Further study details have been published. (1, 3) WHEALS participants included and excluded from these analyses are compared in Figure S1 and Table S1. Exclusions were due to incomplete data and our focus on the comparison of Black and White children.

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There were 550 children included in these analyses: n=390 Black children (n=108, 27.7% with eczema) and n=160 White children (n=19, 11.9% with eczema). Summary details appear in Table S2. Race modified the associations between IgE and eczema for all IgE variables (all interaction $p < 0.10$, Likelihood Ratio Test). (Table 1) Total IgE was higher in White children with eczema compared to those without eczema but this difference was not seen when Black children with and without eczema were compared. The sum of food sIgEs and of inhalant sIgEs were higher in those with eczema in both Black and White children. However, the sum of the sIgEs/total IgE ratios for both food and inhalant allergens (calculated to allow for simultaneous consideration of the sum of the sIgEs and total IgE) differed between Black and White children. Specifically, the ratios of both food and inhalant sIgEs were higher in Black children with eczema (versus no eczema) but lower in White children with eczema (versus no eczema). Adjusted odds ratios reflect similar patterns. (Table 1) Figure 1 shows Black children are more likely to have eczema than White children at lower levels of total IgE, whereas at higher levels of total IgE, data suggest that White children are equally or more likely to have eczema.

While we have reported in WHEALS that Black children had higher total IgE levels than White children, these analyses demonstrate this racial difference was only present among children without eczema.(1) Results in Figure 1 suggest that at lower concentrations, total IgE is associated with an increasing probability of eczema in Black but not White children. Other evidence suggests that the relationship between total IgE and asthma varies by race. Previous work from our group studying another cohort found that serum IgE was related to methacholine reactivity in European American (EA), but not African American (AA) children ages 6-8 years.(4) However, among children without an asthma diagnosis, the AA children had significantly higher total IgE than the EA children.

This body of evidence highlights the importance of the need to consider endotypes – phenotypes with different underlying etiologies – in allergic diseases.(5–10) Eczema is caused, in part, by defects in the skin barrier. These defects, which likely have multiple and various underlying etiological causes, then leave the child vulnerable to IgE sensitization.(5, 10) In data presented here, there are differences in total IgE and food sIgEs and inhalant sIgEs within those children with eczema and those differences vary by race.

A reliable assessment of severity was unavailable and could account for the observed racial differences. sIgE panels were limited to 13 allergens. Statistically significant differences in IgE levels may not indicate clinically important differences; however, this work provides a unique contribution to investigations of racial differences in allergic diseases. In future studies, it will be important to further identify the various endotypes associated with eczema/AD and determine whether they differ in frequency between race groups, as well as examine whether the observed patterns hold for severe eczema and more allergens.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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CLINICAL IMPLICATIONS

The relationships between eczema and IgE and allergen-specific IgEs differ between some Black children and White children at age 2 years. This difference suggests there may be different allergic disease phenotypes that emerge in very early life.

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Figure 1.

The graph demonstrates the modifying effect of race on the relationship between total IgE and the predicted probability of the child having eczema. The shaded areas are the 95% confidence bands. The dashed blue line is for White children. The dashed red line is for Black children. The solid lines represent 95% confidence bands. IgE units are in kU/L.

Table 1

Relationship of the sIgEs with eczema, by race of child with additional models adjusted for child gender and age at clinic visit. * Each interaction term between IgE/sIgE and race was $p < 0.10$, therefore separate models for Black children and White children are presented. IgE units in kU/L.

	Eczema (ever)			
	Yes	No		
	Geometric Mean (95% CI) Black: n=108 White: n=19	Geometric Mean (95% CI) Black: n=282 White: n=141	Adjusted* OR (95% CI)	Adjusted* p-value
Total IgE (in kU/L)				
Black Child	27.2 (19.2, 38.4)	22.3 (18.7, 26.7)	1.08 (0.91, 1.28)	0.39
White Child	45.0 (15.3, 131.7)	13.5 (10.5, 17.3)	1.61 (1.13, 2.31)	0.009
Sum of food sIgE (in kU/L)				
Black Child	0.86 (0.59, 1.26)	0.52 (0.42, 0.64)	1.21 (1.04, 1.42)	0.014
White Child	0.94 (0.29, 3.04)	0.28 (0.23, 0.34)	1.80 (1.20, 2.69)	0.004
Sum of inhalant sIgE (in kU/L)				
Black Child	0.97 (0.54, 1.77)	0.57 (0.51, 0.64)	1.40 (1.11, 1.77)	0.005
White Child	0.86 (0.63, 1.18)	0.47 (0.42, 0.52)	3.86 (1.70, 8.77)	0.001
Sum of food sIgE as a % of total IgE				
Black Child	3.35 (2.64, 4.24)	2.18 (1.86, 2.56)	1.43 (1.13, 1.81)	0.003
White Child	1.31 (0.61, 2.81)	1.94 (1.56, 2.40)	0.76 (0.46, 1.25)	0.28
Sum of inhalant sIgE as a % of total IgE				
Black Child	3.18 (2.32, 4.36)	2.37 (1.98, 2.82)	1.20 (0.98, 1.46)	0.071
White Child	1.15 (0.43, 3.10)	3.59 (2.70, 4.77)	0.56 (0.34, 0.92)	0.023
	Median (IQR)	Median (IQR)		
Total IgE (in kU/L)				
Black Child	26.5 (10.0, 65.6)	26.9 (8.2, 57.2)		
White Child	34.4 (11.4, 243.0)	10.7 (4.8, 41.4)		
Sum of food sIgE (in kU/L)				
Black Child	0.59 (0.15, 2.67)	0.35 (0.15, 1.03)		
White Child	0.50 (0.15, 2.49)	0.15 (0.15, 0.43)		
Sum of inhalant sIgE (in kU/L)				
Black Child	0.35 (0.35, 1.59)	0.35 (0.35, 0.65)		
White Child	1.07 (0.35, 1.62)	0.35 (0.35, 0.55)		
Sum of food sIgE as a % of total IgE				
Black Child	3.28 (1.53, 6.85)	2.26 (0.97, 4.85)		
White Child	1.96 (1.32, 2.57)	2.22 (1.09, 3.56)		
Sum of inhalant sIgE as a % of total IgE				
Black Child	3.27 (1.11, 9.78)	2.36 (0.94, 5.83)		
White Child	2.08 (0.26, 4.25)	3.80 (1.32, 9.00)		

* Models adjusted for age and gender.