



Letter

Allergic sensitization in American children of Middle Eastern ethnicity at age 2

Children of Middle Eastern ethnicity (MEE) born in the United States are seldom specifically examined in allergic disease research, and the prevalence of allergic sensitization in these children is unknown.¹ For analyses, these children can be combined with white children not of MEE or excluded entirely because of the small sample, as we have previously done.^{2,3} However, this subgroup could have a unique set of cultural norms and/or environmental exposures that distinguish them from white, black, non-Middle Eastern, and non-Hispanic children with respect to allergic disease risk factors.^{4,5} We previously observed disparities in allergic disease prevalence between 2-year-old black and white children in our unselected birth cohort study, the Wayne County Health, Environment, Allergy and Asthma Longitudinal Study (WHEALS), but had not investigated potential differences in MEE children.³

Our objective was to describe and compare allergic sensitization rates and total immunoglobulin E (IgE) levels between children who were identified as white, non-Middle Eastern, and non-Hispanic (WN) vs those identified as MEE (white, Middle Eastern, and non-Hispanic). Maternal report of child race and ethnicity was used. All WHEALS participants resided in the area of Detroit, Michigan, which includes one of the largest concentrations of Arabs in the world outside the Middle East.⁶

The MEE children ($n = 35$) were similar to WN children ($n = 132$) in maternal education, mode of delivery, household smoke exposure, sex, and, importantly, maternal history of allergy or asthma ($P \geq .1$ for all comparisons; data not shown). Maternal characteristics of age at delivery and marital status differed between the 2 groups, with MEE children having younger mothers who were more likely to be married (mean age 29.3 vs 31.4, $P = .010$; 100% married vs 89.4%, $P = .043$) compared with WN children. Breastfeeding practices also differed because MEE children were more likely to have ever been breastfed (94.1% vs 76.2%; $P = .020$) but less likely to have been exclusively breastfed during the first month of life (16.1% vs 29.2%; $P = .14$).

Household income, keeping a dog, and allergen exposure also were notably different. Despite similar maternal education levels, 25.7% of MEE households reported an income lower than \$40,000 vs 6.8% of WN households ($P = .008$). Only 2.9% of MEE households reported having a pet dog(s) compared with 45.5% of WN households, and consistently fewer MEE homes had detectable dog allergen in dust samples vacuumed from the infant's bedroom floor compared with WN households ($P < .001$ for the 2 comparisons). In

contrast, dust mite allergen (Der f) was detectable in 57.1% of MEE households vs 29.1% of WN households ($P < .001$).

Allergen-specific IgE (sIgE) and total IgE levels were measured from blood samples taken at approximately 2 years of age (mean 2.2 years old, SD 0.4); average age did not differ between MEE and WN children ($P = .084$). Total IgE and sIgE for up to 3 food (egg, milk, and peanut) and 7 inhalant (*Alternaria* species, cat, cockroach, dog, Der f, short ragweed, and timothy grass) allergens were measured. Atopic sensitization, or atopy, was defined as any sIgE level of at least 0.35 IU/mL. We report overall atopy (sIgE ≥ 0.35 IU/mL for ≥ 1 of the 10 allergens) and food and inhalant separately. Further, our previously identified predominantly multiply-sensitized atopic phenotype that was shown to be related to increased risk of atopic dermatitis and asthma is reported.^{7,8} Maternal inhalant atopy (prenatal or early postpartum blood sample) was defined by the same 7 inhalant allergens.

Relative risks (RRs) and 95% confidence intervals (95% CIs) express the association between MEE and atopy. Log-binomial regression was used to address confounding and for calculation of adjusted RR (aRR). Linear regression was used to associate MEE with total IgE (log-transformed). Confounding was assessed using available covariates based on the literature and included those mentioned in the preceding paragraph. Only covariates that resulted in a change of at least 15% in effect size for any outcome were included in the adjusted (final) model.

The risk of sensitization to at least 1 allergen for MEE children was almost twice that for WN children (aRR 1.94, 95% CI 1.14–3.33, $P = .015$; Table 1). This difference in risk is primarily appreciated when considering sensitization to at least 1 of the inhalant allergens (aRR 3.57, 95% CI 1.44–8.86, $P = .006$). MEE children also tended to have higher total IgE at 2 years of age ($P = .051$) compared with WN children (median 21.5 vs 12.0, respectively). All unadjusted analyses were nonsignificant ($P > .05$); however, adjustment for the confounders of having detectable dog allergen and household income augmented effect sizes and yielded statistical significance. Rates of all types (food and inhalant) of sensitization for the MEE children were consistently higher than for the WN children. The risks of food sensitization and multiply-sensitized atopic phenotype were in the same direction but were not statistically significant with or without adjustment.

Very few households of MEE children had pet(s), which could relate to cultural differences because there are references to the “uncleanliness” of dogs in the Qu’ran.^{9,10} The combination of similar parental history and lower pet-keeping rates in addition to the higher level of dust mite allergen might suggest the “path” to allergy for MEE children has a strong environmental component. However, larger studies are needed to test this hypothesis.

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Table 1
Allergic Sensitization and Total IgE at 2 Years of Age

	White MEE (n = 35) ^a	WN (n = 132) ^a	Unadjusted RR or β (95% CI)	Adjusted ^b RR or β (95% CI)	Adjusted ^b P value
Sensitized to any allergen ^c at 2 y of age					
Yes	15 (50.0)	44 (38.6)	1.30 (0.85–1.98)	1.94 (1.14–3.33)	.015
No	15 (50.0)	70 (61.4)			
Sensitized to food allergen ^c					
Yes	10 (31.3)	34 (28.1)	1.11 (0.62–2.00)	1.58 (0.84–2.99)	.15
No	22 (68.8)	87 (71.9)			
Sensitized to inhalant allergen ^c					
Yes	9 (31.0)	19 (16.8)	1.85 (0.94–3.64)	3.57 (1.44–8.86)	.006
No	20 (69.0)	94 (83.2)			
PM					
Yes	6 (18.2)	15 (11.8)	1.54 (0.65–3.66)	2.00 (0.68–5.92)	.21
No	112 (88.2)	112 (88.2)			
Total IgE at 2 y of age	21.5 (8.5–42.9)	12.0 (5.8–42.2)	0.16 (–0.26 to 0.69) ^d	0.61 (0–1.22) ^d	.051

Abbreviations: CI, confidence interval; IgE, immunoglobulin E; MEE, Middle Eastern ethnicity; PM, multiply-sensitized atopic phenotype; RR, relative risk; WN, white, non–Middle Eastern, non-Hispanic.

^aBecause of some small blood volumes, not all specific IgE tests were completed for every child; number represents the total number of children with at least 1 specific IgE measure or a total IgE. Data are represented as number (percentage) or median (25th–75th percentiles).

^bAdjusted for detectable Can f 1 in living room floor dust and household income.

^cSensitization to any allergen was defined as specific IgE level of at least 0.35 IU/mL for at least 1 of 10 allergens: egg, milk, and peanut (food) allergens and dog, cat, Der f, grass, ragweed, cockroach, and *Alternaria* (inhalant) allergens.

^dThe β estimate (95% CI) from linear regression on log-transformed total IgE.

To the best of our knowledge, this is the first study to report allergic sensitization rates in preschool-age American MEE children. These data suggest different early life sensitization rates for MEE and WN children, particularly with respect to inhalant allergen sensitization. Therefore, we recommend thoughtful consideration before combining these children into 1 racial group, because meaningful associations can be obscured.

There are limitations to this work. The sample was not large; however, we believe the data are unique. Residual confounding by “dog keeping” could exist as only 1 MEE child lived with a dog. Furthermore, we did not assess whether effects of dog ownership other than allergen level, for example, the home microbiota, could explain the observed differences. These findings illustrate a gap in knowledge about the health of this minority ethnic population and highlight the need for further research focused on this group. This work also can assist health care providers in highlighting information that could be central to the care of this potentially susceptible and understudied American ethnic group.

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