Published in final edited form as:

Ann Allergy Asthma Immunol. 2017 November ; 119(5): 464–466. doi:10.1016/j.anai.2017.08.011.

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

Suzanne Havstad, MA¹, Alexandra R. Sitarik, MS¹, Christine Cole Johnson, PhD¹, Edward M. Zoratti, MD², Dennis R. Ownby, MD³, Albert M. Levin, PhD¹, and Ganesa Wegienka, PhD.¹

¹Department of Public Health Sciences, Henry Ford Hospital, Detroit, MI, USA

²Department of Internal Medicine, Division of Allergy, Henry Ford Hospital, Detroit, MI, USA

³Department of Pediatrics, Augusta University, Augusta, GA, USA

Keywords

Allergic sensitization; Middle Eastern ethnicity; IgE

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 may be combined with White children not of MEE or excluded entirely due to the small sample size, as we have previously done.(2, 3) However, this subgroup may have a unique set of cultural norms and/or environmental exposures that distinguish them from White/Black/non-Middle Eastern/non-Hispanic children with respect to allergic disease risk factors.(4, 5) We have previously observed disparities in allergic disease prevalence between 2-year old Black and White children in our own 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 IgE levels between children who were identified as White/non-Middle Eastern/non-Hispanic (WN) versus those identified as MEE (White/Middle Eastern/non-Hispanic). Maternal report of child race/ethnicity was used. All WHEALS participants resided in the Detroit, Michigan area – which includes one of the largest concentrations of Arabs in the world outside of the Middle East.(6)

Corresponding Author: Suzanne Havstad, MA, 1 Ford Place, 3E, Detroit, MI 48202, Phone: 313-874-6363 Fax: 313-874-6730, shavsta1@hfhs.org.

Trial registration: Not applicable

All authors have no conflict of interest to report.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

MEE children (n=35) were similar to WN children (n=132) in terms of maternal education, mode of delivery, household smoke exposure, gender, and, importantly, maternal history of allergy or asthma (all $p \geq 0.1$; data not shown). Maternal characteristics of age at delivery and marital status differed between the two groups, with MEE children having younger mothers that were more likely to be married (mean age 29.3 vs. 31.4, $p=0.010$; 100% married vs. 89.4%, $p=0.043$) as compared to WN children. Breast-feeding practices also differed as MEE children were more likely to have ever been breastfed (94.1% vs. 76.2%; $p=0.020$) but less likely to have been exclusively breastfed during the first month of life (16.1% vs. 29.2%; $p=0.14$).

Household income, dog-keeping and allergen exposure were also notably different. Despite similar maternal education level, 25.7% of MEE households reported income less than \$40,000 versus 6.8% of WN households, $p=0.008$. Only 2.9% of MEE households reported having a pet dog(s) compared to 45.5% of WN households, and consistently, fewer MEE homes had detectable dog allergen in dust samples vacuumed from the baby's bedroom floor compared to WN households (both $p<0.001$). In contrast, dust mite allergen (Der f) was detectable in 57.1% of MEE households versus 29.1% of WN households, $p<0.001$.

Allergen-specific IgE (sIgE) and total IgE levels were measured from blood samples taken at approximately two years of age (mean age=2.2 years (standard deviation=0.4); average age did not differ between MEE and WN children, $p=0.084$). Total IgE and sIgE for up to three food (egg, milk and peanut) and seven inhalant allergens (*Alternaria*, cat, cockroach, dog, Der f, short ragweed, timothy grass) were measured. Atopic sensitization, or atopy, was defined as any sIgE ≥ 0.35 IU/ml. We report overall atopy (≥ 1 sIgE ≥ 0.35 IU/ml for any of the 10 allergens) and food and inhalant separately. Further, our previously identified predominantly multiply-sensitized atopic phenotype (PM) that was shown to be related to increased risk of atopic dermatitis and asthma is also reported.^(7,8) Maternal inhalant atopy (prenatal or early postpartum blood sample) was defined by the same seven inhalant allergens.

Relative risks (RR) and 95% confidence intervals (95% CI) 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 $\geq 15\%$ in effect size for any outcome were included in the adjusted (final) model.

The risk of sensitization to at least one allergen for MEE children was almost twice that for WN children (aRR=1.94 (95% CI=1.14–3.33; $p=0.015$) (Table 1). This difference in risk is primarily appreciated when considering sensitization to at least one of the inhalant allergens (aRR=3.57 (95% CI = 1.44 – 8.86; $p=0.006$). MEE children also tended to have higher total IgE at age 2 ($p=0.051$) as compared to WN children (median value 21.5 vs. 12.0, respectively). All unadjusted analyses were non-significant ($p>0.05$); 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 PM atopy 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 may relate to cultural differences as 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 along with higher 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.

To the best of our knowledge, this is the first study to report allergic sensitization rates in pre-school aged American MEE children. These data suggest differing 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 one racial group, as meaningful associations may be obscured.

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

Acknowledgments

Funding: This work was funded by the National Institutes of Health, USA

R01 AI051598 and P01 AI089473

References

1. Bergmans R, Soliman AS, Ruterbusch J, et al. Cancer incidence among Arab Americans in California, Detroit, and New Jersey SEER registries. *Am J Public Health.* 2014; 104(6):e83–91.
2. Wegienka G, Havstad S, Joseph CL, Zoratti E, Ownby D, Johnson CC. Allergic sensitization frequency and wheezing differences in early life between black and white children. *Allergy Asthma Proc.* 2012; 33(6):493–9. [PubMed: 23394507]
3. Wegienka G, Havstad S, Joseph CL, et al. Racial disparities in allergic outcomes in African Americans emerge as early as age 2 years. *Clin Exp Allergy.* 2012; 42(6):909–17. [PubMed: 22909162]
4. Jessri M, Farmer AP, Olson K. A focused ethnographic assessment of Middle Eastern mothers’ infant feeding practices in Canada. *Matern Child Nutr.* 2015; 11(4):673–86. [PubMed: 23795644]
5. El-Sayed AM, Tracy M, Scarborough P, Galea S. Ethnic inequalities in mortality: the case of Arab-Americans. *PloS one.* 2011; 6(12):e29185. [PubMed: 22216204]
6. Nriagu J, Senthamarai-Kannan R, Jamil H, Fakhori M, Korponic S. Lead poisoning among Arab American and African American children in the Detroit metropolitan area, Michigan. *Bull Environ Contam Toxicol.* 2011; 87(3):238–44. [PubMed: 21725626]

7. Havstad S, Johnson CC, Kim H, et al. Atopic phenotypes identified with latent class analyses at age 2 years. *The Journal of allergy and clinical immunology*. 2014; 134(3):722–7. e2. [PubMed: 24636082]
8. Fujimura KE, Sitarik AR, Havstad S, Lin DL, Levan S, Fadrosh D, et al. Neonatal gut microbiota associates with childhood multisensitized atopy and T cell differentiation. *Nat Med*. 2016; 22(10): 1187–91. [PubMed: 27618652]
9. Herzog HA. Biology, culture, and the origins of pet-keeping. *Animal Behavior and Cognition*. 2014; 1(3):296–308.
10. Al-Fayez G, Awadalla A, Templer DI, Arikawa H. Companion animal attitude and its family pattern in Kuwait. *Society and Animals*. 2003; 11:17–28.

Table 1

Allergic sensitization and total IgE at age 2

	White, Middle Eastern (MEE) N=35 ¹	White, non-Hispanic, non- Middle Eastern (WN) N=132 ¹	N (%) or Median (25 th –75 th percentiles)	Unadjusted Relative risk or β (95% CI)	Adjusted ² Relative risk or β (95% CI)	Adjusted p-value ²
Sensitized to any allergen³ at age 2						
--Yes	15 (50.0%)	44 (38.6%)		1.30 (0.85, 1.98)	1.94 (1.14, 3.33)	0.015
--No	15 (50.0%)	70 (61.4%)				
Sensitized to Food³ allergen						
--Yes	10 (31.3%)	34 (28.1%)		1.11 (0.62, 2.00)	1.58 (0.84, 2.99)	0.15
--No	22 (68.8%)	87 (71.9%)				
Sensitized to Inhalant³ allergen						
--Yes	9 (31.0%)	19 (16.8%)		1.85 (0.94, 3.64)	3.57 (1.44, 8.86)	0.006
--No	20 (69.0%)	94 (83.2%)				
PM Phenotype⁴						
--Yes	6 (18.2%)	15 (11.8%)		1.54 (0.65, 3.66)	2.00 (0.68, 5.92)	0.21
--No	27 (81.8%)	112 (88.2%)				
Total IgE at age 2	21.5 (8.5–42.9)	12.0 (5.8–42.2)		0.16 (–0.26, 0.69) [*]	0.61 (0, 1.22) [*]	0.051

¹ Due to some low blood volumes, not all specific IgE tests were completed for every child, the N represents the total number of children with at least 1 specific IgE measure or a total IgE;² Adjusted for detectable Can f 1 in living room floor dust and household income;³ Sensitization to any allergen defined as sIgE 0.35 for at least one of 10 allergens: egg, milk and peanut (food) allergens; and dog, cat, der f, grass, ragweed, cockroach and Alternaria (inhalant) allergens;⁴ Multiply-sensitized phenotype;^{*} beta estimate (95% CI) from linear regression on log-transformed total IgE