

Is the Prevalence of Food Allergy Not on the Rise After All?



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The prevalence of food allergy is thought to be increasing. The highest figures reported in the general population come from the Healthnuts Study in Australia, where 8.9%, 3%, and 0.8% of infants had a reaction to raw egg, peanut, and sesame, respectively, during oral food challenge.¹ The temporal increase in food allergy is best described for peanut allergy. In sequential cohorts from the same geographical location in the United Kingdom, peanut allergy increased from 0.5% in a cohort of 3–4-year-old children born in 1989 to 1.4% in a cohort of children with similar age born between 1994 and 1996. In the latter cohort, food allergy was diagnosed by challenge or by a combination of history and the presence of allergen-specific IgE as measured by skin prick testing. In the former cohort, food allergy was defined as parent-reported food allergy, which could have involved some degree of overdiagnosis and would make the observed increase in the prevalence of peanut allergy even more significant.^{2,3}

In this issue of the journal, McGowan et al⁴ report the results of a large cross-sectional study of 6- to 19-year-olds in the National Health and Nutrition Examination Survey looking at specific IgE levels to common foods, including cow's milk, egg, peanut, and shrimp, measured in serum samples collected 18 years apart. Surprisingly, overall specific IgE levels did not increase over time and there was a significant decrease in specific IgE to shrimp during the same period. This is in contrast with the increase in the prevalence of clinical food allergy reported in the United States and other Western countries in the recent decades. The authors provide 2 possible explanations for the discrepancy between the changes in the prevalence of sensitization and the changes in the prevalence of food allergy: either the rise in food allergy is not due to a rise in food sensitization or the

reported increase in the prevalence of food allergy is factitious and due to increased awareness of food allergy.

Allergic sensitization does not equate to clinical allergic disease and means tolerance in most cases. In various food allergy studies, only a small proportion of sensitized patients developed allergic reactions when exposed to the food during an oral food challenge.^{1,5,6} This discrepancy between sensitization and allergy has also been described for atopic diseases other than food allergy. For instance, asthma, allergic rhinitis, and atopic eczema were 7 to 10 times more common in the United Kingdom than in Albania despite similar rates of sensitization, in a report published in the early 2000s.⁷ On the contrary, in 2 successive cohorts of school children in Sweden, there was a 50% increase in sensitization over a period of 10 years but no increase in allergic symptoms.⁸ In these 2 successive cohorts, the risk factor profile for sensitization changed dramatically, suggesting that the risk factors for sensitization may be different from the risk factors for allergic disease. Indeed, the underlying changes driving the increase in food allergy observed in the recent decades may not be the same as the factors driving the production of IgE. The lack of accompanying clinical data precludes a direct comparison between the prevalence of food allergy and the prevalence of food sensitization in the National Health and Nutrition Examination Survey population.⁴ It is possible that the observed increase in the proportion of children with high levels of IgE to foods, particularly to peanut, during the studied period could potentially reflect a rise in food allergy.

Interestingly, non-Hispanic black children had higher levels of sensitization to all foods tested than did Hispanic and non-Hispanic white children at both time points.⁴ In the Learning Early About Peanut Allergy screening study,⁹ black race was associated with a significantly higher risk of sensitization to peanut as measured by serum peanut-specific IgE level and was protective against cutaneous sensitization, as shown by a positive skin prick test result to peanut, which later in the Learning Early About Peanut Allergy study was shown to be more closely related to clinical challenge—proven peanut allergy than serum specific IgE.¹⁰ In a previous cross-sectional study of US children, the prevalence of food sensitization was also highest in non-Hispanic blacks and it was lowest in non-Hispanic white children, who in turn had the highest prevalence of food allergy.¹¹ Of note, the increase in food allergy was 3-fold higher in nonwhite than in white populations during the 10-year period. In a systematic review of studies performed in the pediatric US population, the increase in self-reported food allergy per decade was highest in non-Hispanic blacks followed by Hispanics and non-Hispanic whites.¹² Although the increase in self-reported food allergy paralleled the increase in allergic sensitization in non-Hispanic blacks, the diagnosis of food allergy was not confirmed by oral food challenge. A significant increase in peanut allergy (but not

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egg allergy or eczema) in nonwhite children was also observed in a study performed in the United Kingdom over a 14-year period; however, this was except for the group of black-African descent in whom the prevalence of peanut allergy remained stable.¹³ Taken together these data support the concept that factors driving sensitization and factors driving clinical allergy may not be the same and may have different effect on patients from diverse ethnical backgrounds. Whether this is due to genetic (or epigenetic) differences or due to environmental and cultural differences remains unknown and deserves further research. For example, higher levels of clinically irrelevant IgE in those of black-African descent could be a result of higher exposure to parasites in previous generations.¹⁴ The larger rise in peanut allergy in nonwhite populations could be a result of avoidance of peanut in infancy in the new home country combined with higher exposure to peanut allergens in the home environment as a result of a culture of high peanut consumption in the household.¹³ The fact that the prevalence of other atopic conditions, such as eczema, did not change together with food allergy in some studies¹³ and the fact that some of the data were generated from surveys of the general population using standardized methodology^{4,15} suggest that differences in food allergy between patients of different ethnicity are not (at least entirely) due to increased awareness about food allergy or due to differences in access to health care. Migrants often adopt the prevalence of food allergy of their new home country and certain ethnicities may confer a higher degree of susceptibility. In the Healthnuts Study, infants of first-generation migrants from East Asia to Australia were at a higher risk of peanut and other food allergies compared with infants of Australia-born parents.¹⁵ Food allergy studies in migrants raise fascinating questions and can potentially provide even more fascinating answers about the gene-environment interactions involved in the development of food sensitization and food allergy.

In conclusion, although immediate-type food allergies are mediated by IgE, the presence of IgE is not sufficient for developing clinical allergic disease. Diagnosing allergy on the basis of sensitization alone may lead to overdiagnosis. Allergy and sensitization are 2 different phenomena. Risk factors driving the increase in food allergy are probably distinct from risk factors driving the production of IgE. There is an unmet need to better understand IgE, the factors involved in its ability to elicit allergic reactions and the role of IgE in nonwhite populations, to

improve the diagnosis and possibly to develop novel approaches to prevent food allergy.

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