

Letter to the Editor

Changes in peanut allergy prevalence in different ethnic groups in 2 time periods

To the Editor:

Peanut allergy (PA) is one of the most serious of the food hypersensitivities and appears to be increasing in prevalence. The prevalence of PA in industrialized countries, such as the United States and United Kingdom (UK),¹ appears to be higher than in the developing world, even among atopic children.²

There is recent evidence of ethnic variations in skin and respiratory allergy³ and that patterns of sensitization to foods differ among ethnic groups within the same geographic population,⁴ with such sensitization being more common in nonwhite minorities in the United States.⁵ However, there is also evidence that food allergy in North America is more prevalent in white than nonwhite groups, but the increase in food allergies over a 10-year period (based on hospital discharge diagnoses) was 3-fold higher in nonwhite groups.⁶

It has also been our impression that the proportion of children given a diagnosis of PA from nonwhite backgrounds has increased in our clinic over the past 15 years. Apart from the observation that Chinese American children appear to have a prevalence of PA similar to that of the general US population,⁷ relatively little attention has been paid to the ethnicity of those with PA in the context of the diverse populations found in urban Western settings.

Data from our recent food allergy work⁸ indicate that the household peanut consumption of nonwhite families is higher than that of white families, usually as a consequence of traditional dietary patterns. The influence of the recently withdrawn 1998 UK Department of Health guidance for mothers aimed at reducing PA on traditional patterns of peanut consumption in nonwhite families is unknown. The guidance stated, "Pregnant women who are atopic or have an atopic partner may wish to avoid eating peanuts during pregnancy and lactation, whilst their infants should avoid peanuts until 3 years." Similar guidance was issued in 2000 by the American Academy of Pediatrics but withdrawn by the UK Department of Health in 2009 because of the lack of evidence of efficacy after our data were collected.

We hypothesized that the move toward infant peanut avoidance since the publication of this guidance, coupled with the persistence of high levels of environmental peanut exposure, might have resulted in a disproportionate increase in the prevalence of PA among nonwhite populations in the UK.

We conducted an ethnicity-based study of children given a diagnosis of PA at a UK allergy clinic over a 14-year period between 1990 and 2004 ($n = 420$), with children with egg allergy ($n = 372$) as control subjects. Diagnosis of PA was based on either (1) a positive challenge result to peanut, (2) a convincing history of PA together with a positive skin prick or specific IgE test result to peanut, or (3) a specific IgE level of greater than 15 kU/L or an SPT response of greater than 7 mm in the absence of any history of peanut exposure. Further control data were obtained from attendees at general pediatric ($n = 1982$) and pediatric eczema ($n = 1864$) clinics between 1998 and 2004. The aim of this study was to establish the ethnicity of children with PA born over a 14-year period and compare any changes with those among relevant control subjects.

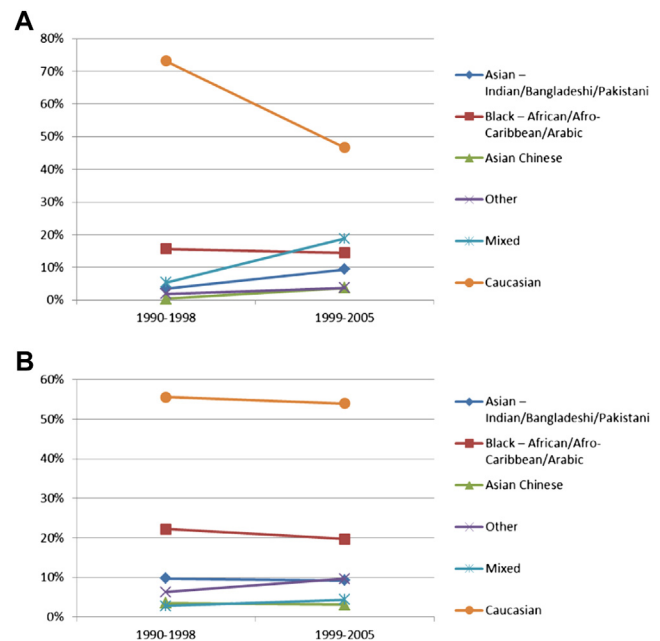


FIG 1. Proportion of children with PA (A) or egg allergy (B) from different ethnic groups over time.

The Fisher exact test was used to compare the proportions of children in different ethnic groups. Details were obtained from 420 children with PA and 372 children with egg allergy. Sex distribution was similar between the groups.

Fig 1 shows the ethnic breakdown of the children with peanut and egg allergy in group 1 (born 1990-1998) and group 2 (born 1999-2004). Among the patients with PA (group 1, $n = 261$; group 2, $n = 159$), there is a highly significant increase of 23.5% (from 26.82% to 50.31%, $P = .001$) in the proportion of nonwhite subjects given a diagnosis of PA over time. A statistically significant increase is seen in all ethnic subgroups of the nonwhite group, with the exception of those of black-African descent (Table I). Among children with egg allergy (group 1, $n = 144$; group 2, $n = 228$), there is a less than 1% change in the proportion of nonwhite children, from 44.4% in 1990-1998 to 45.2% in 1999-2004, with no statistically significant change in any of the ethnic subgroups.

Among the general pediatric clinic attendees, the proportion of nonwhite subjects increased from 386 (67.8%) of 570 in 1998-1999 to 898 (69.4%) of 1294 in 2003-2004. Among the pediatric eczema clinic attendees, the proportion of nonwhite subjects decreased from 705 (70.3%) of 1003 in 1998-1999 to 680 (69.5%) of 979 in 2003-2004. Neither of these changes between the 2 time points was statistically significant. During this time period, the percentage of nonwhite subjects among the children with PA increased from 42.2% in 1998-1999 to 50.9% in 2003-2004, which is statistically significant.

In summary, our data reveal that between 2 distinct time periods over 14 years, the proportion of children with PA from nonwhite backgrounds increased from 26.82% to 50.31%, a highly statistically significant increase of 23.5%. An increase was seen over time in almost all of the nonwhite subgroups. This

TABLE I. Proportions of ethnic groups among the children with PA and egg allergy in groups 1 and 2

Ethnic group	Group 1, 1990-1998		Group 2, 1999-2005		Percentage change	P value
	No. of patients	Percentage	No. of patients	Percentage		
PA						
White	191	73.18	79	49.69	−23.49	.0001
Nonwhite	70	26.82	80	50.31	23.49	.0001
Asian-Indian/Pakistani	9	3.45	15	9.43	5.99	.016
Black-African/Arab	41	15.71	23	14.47	−1.24	NS
Asian Chinese	1	0.38	6	3.77	3.39	.013
Other	5	1.92	6	3.77	1.86	NS
Mixed	14	5.36	30	18.87	13.50	.0001
Total	261	100	159	150		
Egg allergy						
White	80	55.56	123	53.95	−1.61	NS
Nonwhite	64	44.44	105	46.05	1.61	NS
Asian-Indian/Pakistani	14	9.72	21	9.21	−0.51	NS
Black-African/Arab	32	22.22	45	19.74	−2.49	NS
Asian Chinese	5	3.47	7	3.07	−0.40	NS
Other	9	6.25	22	9.65	3.40	NS
Mixed	4	2.78	10	4.39	1.61	NS
Total	144	100	228	100		

NS, Not significant.

increase appears to be specific to PA because no such change was seen among children with egg allergy over the exact same time period. Our data cannot be explained by changing or improved awareness of atopic disorders among the nonwhite population because no significant changes were seen over a similar time period in referrals to the eczema clinic. Furthermore, there is no evidence to suggest that significant changes have occurred to the ethnicity of the overall potential referral base of the hospital over time.

The dual-allergen exposure hypothesis posits that early exposure to peanut induces oral tolerance, whereas exposure to environmental peanut in households results in transcutaneous sensitization and allergy development. The outcome of tolerance or allergy to a particular food in an atopic subject depends on the balance and timing of these 2 different routes of exposure. We explain the increase in PA in nonwhite subjects by using this hypothesis. Before 1998, we hypothesized that infants from nonwhite households were fed peanut products in early infancy and thus were likely to be protected against PA. Indeed, there is evidence that household peanut consumption is generally higher in nonwhite than white families,⁸ and thus we would expect that peanut consumption in nonwhite infants would have been higher. However, after 1998, by following the guidelines, nonwhite infants would no longer be protected by early oral exposure to peanut. In the face of high environmental exposure to peanut, transcutaneous exposure and allergic sensitization would have occurred because of a lack of oral exposure and high environmental exposure, thus resulting in an increase in PA. However, this assumes a high degree of adherence to the guidelines and equally similar uptake among different ethnic groups. There is no evidence to support this, and indeed, there is some evidence to suggest a possible differentially higher uptake of the guidance by nonminority groups.⁹

It is worth considering the inherent limitations of any research that attempts to ascribe any outcome to ethnicity. The lack of prospective data collection related to ethnicity in this study has resulted in a lack of precision, especially in our nonwhite

grouping. The ethnic categories we have used are broad and overlapping, and use of broad groupings can obscure significant within-group heterogeneity.

Our findings demand that the prevalence of PA among the various ethnic groups be examined more closely to establish whether there are racial disparities. This will allow targeting of prevention strategies at the ethnic groups most at risk of allergy development.

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