To determine the prevalence of self-reported peanut and tree-nut allergy among the general population of the United States in 2002 and compare it with prevalence rates obtained 5 years earlier.

**Study Population.** A total of 4855 households representing a census of 13 493 participated.

**Methods.** A nationwide, cross-sectional survey was administered by telephone to persons called by a random sampling of telephone numbers in the continental United States. Adults acted as surrogates for minors with peanut or tree-nut allergy. Differences in responses between groups were tested by $\chi^2$ analysis.

**Results.** Fifty-three percent of contacted homes participated. Peanut allergy, tree-nut allergy, or both was self-reported in 166 (1.2%); 95% confidence interval (CI): 1.0%, 1.4%) individuals in 155 (3.2%; 95% CI: 2.7%, 3.7%) households. These prevalence rates were similar to those reported in 1997. Any differences in prevalence rates between people of different race/ethnicity did not reach statistical significance. There was an overall male predominance of peanut or tree-nut allergy in children ($P = .02$) and a female predominance in adults ($P = .0008$). The prevalence of reported peanut allergy among children increased significantly from 0.4% in 1997 to 0.8% in 2002 ($P = .05$), but the rate of tree-nut allergy did not change significantly. The prevalence of peanut and tree-nut allergy in adults did not change significantly between 1997 and 2002. Overall, the adjusted prevalence rate taking into account individuals with reported allergy without convincing histories was 1.04% (95% CI: 0.9%, 1.2%). Of the reported reactions, 79% involved either respiratory symptoms or multiple organ systems. Only 74% of children and 44% of adults were evaluated by a physician for their allergic reactions, and self-injectable epinephrine was prescribed for 46% of the children and 23% of the adults.

**Conclusions.** The authors reported similar overall rates of peanut and tree-nut allergy in the United States, as was noted in 1997, but over this 5-year period the prevalence of peanut allergy in children doubled.

**Reviewers’ Comments.** The findings of increased prevalence of peanut allergy may be expected with the well-documented increase of atopic diseases in the past decades. Why the prevalence of tree-nut allergy would be unchanged during this same period will require additional investigation. A notable finding in this study is that >25% of children and 50% of adults who reported peanut or tree-nut allergy did not seek medical evaluation. Even more remarkable is that after medical evaluation for peanut or tree-nut allergy, self-injectable epinephrine was prescribed to approximately half of the children and less than one quarter of the adults. This underscores the need for continued improvement in the care of patients with food allergy, which is increasing in prevalence.

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**PEANUT ALLERGY: RECURRENCE AND ITS MANAGEMENT**


**Purpose of the Study.** To determine the rate of peanut allergy recurrence, identify risk factors for recurrent peanut allergy, and develop specific recommendations for the treatment of patients with resolved peanut allergy.

**Study Population.** Children >4 years old with prior diagnosis of peanut allergy who had undergone and passed an oral peanut challenge.

**Methods.** Children were evaluated by using questionnaires, skin tests, and peanut-specific IgE levels. Patients were invited to undergo a double-blind, placebo-controlled food challenge (DBPCFC) unless the history of a possible recurrence reaction was so convincing that a challenge would be potentially dangerous.

**Results.** Sixty-eight patients were evaluated. Forty-seven patients continued to tolerate peanut, of whom 34 ingested concentrated peanut products at least once per month and 13 ate peanut infrequently or in limited amounts but passed a DBPCFC. The status of 18 patients was indeterminate because they ate peanut infrequently or in limited amounts and declined to have a DBPCFC. After excluding 12 patients originally diagnosed with peanut allergy based solely on a positive skin-prick test or peanut-specific IgE level, 3 of 15 patients who consumed peanut infrequently or in limited amounts had recurrences, compared with no recurrences in the 23 patients who ate peanut frequently ($P = .025$). The recurrence rate was 7.9% (95% confidence interval: 1.7%, 21.4%).

**Conclusions.** Children who outgrow peanut allergy are at risk for recurrence, and this risk is significantly higher for patients who continue largely to avoid peanut after resolution of their allergy. It is recommended that patients eat peanut frequently and carry epinephrine indefinitely until they have demonstrated ongoing peanut tolerance.

**Reviewer’s Comments.** Recent studies reported that up to 20% of peanut-allergic children may outgrow this condition, giving hope to many patients. Follow-up of the children who passed an oral peanut challenge showed that some children experienced acute allergic reactions to peanut some time after having passed a challenge. Children avoiding peanut were more likely to have recurrence of their peanut allergy than those ingesting peanut on a regular basis. The possibility of recurrence of peanut allergy and importance of regular dietary peanut intake should be discussed with patients and their parents when considering oral peanut challenges. It should be noted that recurrence has been reported solely for peanut and fish allergy, whereas recurrence of other food allergies such as those to cow’s milk, egg, soy, or wheat have not been described in the literature.

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**DISTRIBUTION OF PEANUT ALLERGEN IN THE ENVIRONMENT**


**Purpose of the Study.** To determine the amount of peanut protein detectable in a variety of common exposure settings and examine the effectiveness of measures used to clean peanut from tables and hands.

**Methods.** A monoclonal-based enzyme-linked immunosorbent assay was used to detect 1 of the major peanut proteins (Ara h1) from surface-wipe samples of hands, tables, and other surfaces and from air samples.

**Results.** After purposeful handling of a teaspoon of peanut butter, hand-washing with liquid soap, bar soap, or commercial wipes resulted in no detectable Ara h1. However, using plain water without soap or an antibacterial hand sanitizer left detectable Ara h1 on 3 of 12 and 6 of 12 hands, respectively. Purposeful placement of a teaspoon of peanut butter on 1 square foot of tabletop followed by
cleaning resulted in no detectable Ara h1 when common household cleaning agents (such as Formula 409 or even plain water) were used; however, dishwashing liquid left Ara h1 on 4 of 12 tables (possibly by leaving a film). Six schools were assessed without special prior preparation (2 used peanut-free tables/preparation areas, and 1 was totally peanut-free). Of the 6 preschools and schools evaluated, Ara h1 was found on 1 of 13 water fountains (130 ng/mL), 0 of 22 desks, and 0 of 36 cafeteria tables. Airborne Ara h1 was undetectable in simulated real-life situations when participants consumed peanut butter, shelled peanuts, and unsalted peanuts.

Conclusions. The major peanut allergen, Ara h1, is relatively easily cleaned from hands and tabletops with common cleaning agents and does not seem to be widely distributed in preschools and schools. Airborne Ara h1 was not detectable in many simulated environments.

Reviewer’s Comments. A major concern for those with peanut allergy is the potential for reactions to casual contact. This study is reassuring in that areas tested without obvious peanut contamination generally had no detectable Ara h1, and eating surfaces and hands, purposefully smeared with peanut, were cleaned adequately with simple, available methods. A limitation of the study is that the assay detected only 1 of several peanut allergens, so the total amount of peanut allergen on these items is unknown. Nonetheless, in most cases no peanut was detectable, providing a level of reassurance for families. On the other hand, it is known that a very small amount (although typically a visible amount) of peanut, if ingested, could cause a severe reaction in some children. Therefore, caution would still be advised about exposure to peanut and the need for careful cleaning practices, particularly with young peanut-allergic children. These children may be around messy eaters and may be inclined to place contaminated fingers and other objects into their mouths.

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FOOD ALLERGY AND ATOPIC DERMATITIS IN INFANCY: AN EPIDEMIOLOGIC STUDY


Purpose of the Study. To examine the relationship between atopic dermatitis and IgE-mediated food allergy in infancy.

Study Population. A birth cohort of 620 infants from the Melbourne Atopy Cohort Study, a cohort with a family history of eczema, asthma, hay fever, or immediate food allergy in a parent or sibling.

Methods. A total of 487 children had complete data including skin-prick tests to evaluate IgE-mediated food allergy to cow’s milk, egg, and peanut at the age of 1 year. Participants were grouped as no atopic dermatitis (group 0) or in quartiles of increasing severity of atopic dermatitis (groups 1–4) quantified by days of topical steroid use as reported in monthly interviews. Adverse reactions to foods were recorded.

Results. The cumulative prevalence of atopic dermatitis was 28.9% by 12 months of age (10.3% of the cohort of moderate severity). As atopic dermatitis severity increased, so did the prevalence of IgE-mediated food allergy (group 0: 40 of 346; group 1: 6 of 36; group 2: 8 of 35; group 3: 12 of 35; group 4: 24 of 35; \( \chi^2 = 76; P < 10^{-6} \)). The frequency of reported adverse food reactions was as follows: group 0, 43 of 346; group 1, 4 of 36; group 2, 8 of 35; group 3, 5 of 35; group 4, 13 of 35 (\( \chi^2 = 17; P = .002 \)). The relative risk of an infant with atopic dermatitis having IgE-mediated food allergy is 5.9 for the most severely affected group.

Conclusions. Atopic dermatitis is common in infancy. There is a strong association between IgE-mediated food allergy and atopic dermatitis in this age group.

Reviewer’s Comments. Studies using double-blind, placebo-controlled food challenges show that nearly 40% of infants and young children with severe atopic dermatitis have food allergy. This epidemiologic study focusing on children <1 year of age supports the association of IgE-mediated food sensitization with a history of atopic dermatitis. Therefore, evaluation for food allergies should be considered in infants with severe atopic dermatitis.

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LATE ECZEMATOUS REACTIONS TO FOOD IN CHILDREN WITH ATOPIC DERMATITIS


Purpose of the Study. To evaluate the frequency of late-phase atopic dermatitis (AD) reactions to foods during double-blind placebo-controlled food challenges (DBPFCs) and correlate the results with food-specific IgE and patch tests.

Study Population. Sixty-four children aged 1 to 10 years (median: 2 years) with mild to severe AD evaluated in an outpatient dermatology and allergy department in Germany.

Methods. The inclusion criterion was suspicion of food-related AD by parents and/or a referring physician. The children underwent testing for food-specific IgE (n = 64). Allergen patch testing (APT) to suspected foods was performed if they did not have a rash on their back (n = 41). The first day was an incremental food challenge up to a full serving as tolerated, and on the second day the children were given a full dose of the food/placebo. The children were observed for 48 hours after the challenges. Reactions occurring within 6 hours were considered immediate, and those occurring after >6 hours were considered late reactions.

Results. A total of 106 food challenges were performed to milk, egg, wheat, or soy, with 64% of the children reacting to at least 1 food; of those who reacted, 83% reacted to only 1 food, 15% reacted to 2 foods, and 1 child reacted to 3 foods. The most common trigger was egg (62%), followed by milk (47%) and wheat and soy (35% each). Immediate reactions occurred in 88% of the challenges. Late AD reactions were seen in 28 of 49 (57%) of positive challenges. Sensitivity of history in predicting immediate reactions was only 54%, and for late reactions only 25%; reactions to milk had the highest sensitivity (50–67%), and soy had the lowest (0%). In general, sensitivity (77% vs 68%), specificity (60% vs 50%), and positive predictive values (57% vs 33%) were higher for immediate reactions versus late reactions. Diagnostic accuracy of food-specific serum IgE was greater for children <2 years old. There was no difference in sensitivity (67%) or specificity (38%) of APT for predicting immediate or late AD reactions. The positive predictive value of APT was greater for immediate reactions (38% vs 24%). It is notable that 19% of patients reacted on day 2 of the challenge, having previously tolerated the food on day 1.

Conclusion. Food allergy should be considered in any child with AD who is not responding to standard therapy.
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