model. The same was true for likely peanut allergy (2.10-fold; 95% CI, 1.2- to 3.67-fold, \( P < .01 \)). In subjects with a history of atopic dermatitis, the effect of EPE on peanut SPT sensitization was enhanced (odds ratio [OR], 1.97; 95% CI, 1.26–3.09; \( P < .01 \)) and even more so in those with history of severe atopic dermatitis (OR, 2.41; 95% CI, 1.30–4.47; \( P < .01 \)). The effect of EPE on likely peanut allergy was also increased in these children with a history of atopic dermatitis (OR, 2.41; 95% CI, 1.31–4.18; \( P < .01 \)), but the further increased effect was not seen with severe atopic dermatitis as it was with peanut SPT sensitization.

CONCLUSIONS. This study demonstrates a positive association between exposure to peanut protein in household dust and peanut sensitization and allergy in children with atopic dermatitis, providing further evidence that environmental exposure through an impaired skin barrier increases risk.

REVIEWER COMMENTS. These findings indicate that when young children with atopic dermatitis are exposed to peanut antigen through transcutaneous contact, the risk for peanut sensitization and allergy are increased. Results further support the dual-allergen exposure hypothesis, which proposes that oral exposure at young ages may lead to tolerance whereas transcutaneous exposure, particularly through an inflamed skin barrier, can lead to sensitization (Lack G. J Allergy Clin Immunol. 2012;129:1187–1197). Additional research is needed to determine if the same association is present with other food antigens. Furthermore, this study highlights the need for additional research to delineate the risks versus benefits of different routes of exposure (ie, oral vs transcutaneous) of peanut allergens and the timing and balance of that exposure to prevent or to reduce the risk of peanut allergy.

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Eczema in Early Childhood, Sociodemographic Factors and Lifestyle Habits Are Associated With Food Allergy: A Nested Case-Control Study

PURPOSE OF THE STUDY. The authors sought to determine whether certain atopic features, sociodemographic characteristics, and lifestyle habits were associated with childhood food allergy.

STUDY POPULATION. Through randomized telephone calls, study participants were asked to complete a questionnaire if they had a history of food allergy (cow’s milk, egg, peanut, tree nuts, fish, shellfish, wheat, soy, or sesame).

Controls were age-matched, nonallergic individuals either living in the same household or randomly selected.

METHODS. This was a case-controlled study nested within the SPAACE study (Surveying Prevalence of food Allergy in All Canadian Environments). A random telephone sampling of families were called between September 2010 and September 2011. Cases and controls were matched to 3 age groups: <5 years, 5 to 17 years, and >18 years of age. They aimed to have a ratio of at least 1:4 between cases and controls for each of 3 matched groups. Questionnaires looked at atopic history, sociodemographic patterns, and lifestyle habits.

RESULTS. Four hundred and eighty cases and 4950 controls completed the questionnaire. For all 9 food allergens, having a personal history of eczema in the first 2 years of life, asthma or hay fever, close family member with food allergy, and high household income (>20%) were all associated with a higher risk of probable allergy. Males and older individuals were less likely to have food allergy. Eczema in the first 2 years of life was the strongest risk factor for egg, peanut, tree nut, and fish allergy. Having older siblings and living on a farm were associated with lower risk of milk and egg allergy, respectively.

CONCLUSIONS. This large population-based, nested case-control study further confirms previously reported factors associated with increased allergy (higher household income, not having older siblings, and living in urban areas). In addition, eczema during the first 2 years of life, and not during the later years, is consistently associated with food allergies.

REVIEWER COMMENTS. Although we know there are lifestyle factors that play a role in atopic sensitization, less clear is whether we can establish prevention strategies to halt the progression of allergy. This large-scale study found that early childhood eczema is associated with food allergies, and the authors suggest that the disrupted skin barrier in atopic dermatitis leads to cutaneous allergenic exposure and may promote food allergy sensitization. Future studies should investigate whether using barrier repair treatments for eczema therapy in these youngsters may halt the development of food allergy.

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Which Infants With Eczema Are at Risk for Food Allergy? Results From a Population-Based Cohort

PURPOSE OF THE STUDY. To characterize the risk of challenge-proven food allergy among infants with eczema in the general population.
STUDY POPULATION. Infants were recruited from the general population at their immunization sessions in Melbourne, Australia. Recruitment took place between September 2007 and August 2011, and all infants were 11 to 15 months of age; 7134 infants were eligible for study, 5276 (74%) agreed to participate, and 4453 met criteria to be used in final study analysis.

METHODS. One-year-old infants were examined for eczema on the face, back, and elbows by a trained nurse and underwent skin prick testing to peanut, egg, and sesame. Those with any detectable wheal to 1 of the test foods underwent an open oral food challenge to that food. Parents completed a questionnaire regarding age of onset of eczema, medication use, and environmental and demographic factors. The risk of food allergy stratified by eczema severity, age of onset, and medication usage was estimated using multivariate logistic regression with population sampling weights.

RESULTS. One in 5 infants with eczema had confirmed allergy to peanut, egg white or sesame by 12 months, compared with 1 in 25 infants without eczema (odds ratio = 6.2, 95% confidence interval [CI]: 4.9–7.9, P < .001). Infants with eczema were also 11 times more likely to develop peanut allergy (95% CI: 6.6–18.6) and 5.8 times more likely to develop egg allergy (95% CI: 4.6–7.4) than infants without eczema. The risk of peanut or sesame seed allergy was low in the absence of eczema (0.7% 95% CI 0.4–1.1). Fifty percent of infants (95% CI: 42.8–58.9) with early-onset eczema (<3 months) who required doctor-prescribed topical corticosteroid treatment developed challenge-proven food allergy by 12 months of age.

CONCLUSIONS. Infants with eczema were 6 times more likely to have egg allergy and 11 times more likely to have peanut allergy by 12 months than infants without eczema. The data suggested that a heightened awareness of food allergy risk is warranted among health care practitioners treating infants with eczema.

REVIEWER COMMENTS. This study confirms a strong association between eczema in infancy and the development of food allergy. Its strengths lie in the fact that it assesses a large general infant population, not a selection-biased group from a tertiary allergy or dermatology referral practice. It also uses food challenges to confirm the presence of food allergy. Because infants with eczema often have clinically irrelevant IgE antibodies to foods, a positive food challenge represents a more accurate determination of true food allergy. Currently National Institute of Allergy and Infectious Diseases-sponsored guidelines do not recommend screening infants with eczema for food allergies unless they have severe refractory eczema or a history of a reaction to a food. Fifty percent of infants with early-onset eczema (<3 months) and moderate to severe eczema (requiring prescription topical corticosteroids) may develop “real” food allergy by 12 months. Clinicians should be proactive to assess food allergy in this group and attempt prevention strategies by inducing primary oral tolerance with early introduction of foods or minimizing allergen exposure through weakened skin barriers. Although more research is needed to identify optimal prevention strategies, the development of food allergy appears to occur earlier than previously suspected.

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Peanut Avoidance and Peanut Allergy Diagnosis in Siblings of Peanut Allergic Children

PURPOSE OF THE STUDY. The purpose of this study was to evaluate familial behavioral patterns, clinical factors, and physician assessment by history and testing in siblings of peanut-allergic children.

STUDY POPULATION. Nine hundred and twenty-two siblings of peanut-allergic children who were part of the Canadian Peanut Allergy Registry (PAR) were evaluated by questionnaire between 2000 and 2012.

METHODS. Parents completed questionnaires on siblings’ sociodemographic characteristics, exposure and reactions to peanut, confirmatory tests performed, and whether peanut allergy had been confirmed. Univariate and multivariate logistic regression models were used to estimate associations between characteristics of the index child and family and specific outcomes in the sibling.

RESULTS. All PAR families were surveyed (n = 932), and 80% of the families responded (n = 748). Of the peanut allergic siblings, 13.6% (n = 125/922, confidence interval [CI], 11.4%–16%) had never been exposed to peanut, 764 had been exposed, and 33 were uncertain. Among those never exposed, the majority (n = 88/125) was born after the diagnosis of peanut allergy (PNA) in the index child. Of the sibling group, 8.7% (n = 80/922, 95% CI 7.0–10.7) reported having been diagnosed with peanut allergy by a physician. Among these 80 children, 41 (51.3%) had reacted to peanut, 34 (42.5%) had never reacted, and in 5 children, the parents were not sure. Among those who had never reacted, 29 had a confirmatory test, and 5 were diagnosed without any history or testing. In the multivariate regression analysis including only siblings aged 3 years or older, siblings born after the diagnosis of peanut allergy in the index child were more likely to have never been exposed.
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Desha Jordan and Angela Duff Hogan

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