The Role of Food Allergy in Atopic Dermatitis

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KEYWORDS

• Atopic dermatitis • Food allergy • Sensitization • Dietary interventions • Immunologic mechanisms

KEY POINTS

- Many individuals are affected by both atopic dermatitis (AD) and IgE-mediated food allergy simultaneously.
- There is keen interest in whether there is a causal relationship between AD and food allergy.
- In general, AD is a risk factor for the development of food allergy, while food allergy is rarely a cause of AD.
- Sensitization to foods is common in patients with AD, and can lead to the overdiagnosis and/or misdiagnosis of food allergy.
- Dietary elimination of allergenic foods can have unintended harmful effects, including the development of immediate-type allergy to previously tolerated foods.

THE RELATIONSHIP BETWEEN ATOPIC DERMATITIS AND FOOD ALLERGY

Atopic dermatitis (AD) is a chronic inflammatory skin condition characterized by intense pruritus and recurrent eczematous skin lesions. It is the most common inflammatory skin disorder in childhood, affecting up to 20% of children and 3% to 10% of adults.[1,2] Any clinician who has encountered this disease has likely witnessed the immense negative consequences it can have for the quality of life, sleep quality, development, and functioning of affected patients and their families.[1] Despite the connotation "atopic," AD is not necessarily an allergic disease and occurs often in individuals who are not sensitized to food or environmental allergens.[3] However, rates of allergic disorders in patients with AD are substantial, with up to 80% of affected children going on to develop other atopic conditions, which include allergic rhinitis and asthma later in life, in a progression often termed the "atopic march."[4,5] Furthermore, the presence of early childhood AD is recognized as a strong risk factor for the development of food allergy.[6]

The relationship between AD and food allergy is complex, and whether one condition causes the other has been a topic of controversy.[7] An understanding of the interplay between skin inflammation and AD control, as well as cutaneous and oral exposure to foods, is crucial for evaluating and managing young patients with AD who may have food allergy. The onset of AD and the development of food allergy may coincide early in life. Atopic dermatitis often emerges in infancy just as a wide range of foods are being introduced in the diet. Because AD typically waxes and wanes, with exacerbations and periods of improvement, it may appear that the development of and severity of eczema is related to foods ingested by an infant or nursing mother. Often, parents of affected infants eliminate many foods in attempts to achieve control of AD, almost always without observed clinical improvement. Some foods such as acidic fruits and certain spices can act as irritants and cause rash in those with sensitive skin and flares

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Dermatol Clin 42 (2024) 527–535
https://doi.org/10.1016/j.det.2024.04.004

in those with AD, and may be mistaken by patients and families as food allergens. Immunoglobulin (Ig) E-mediated food allergy is present in a subset of patients with AD, and allergic reactions to foods can trigger AD exacerbations; however, food allergy is rarely the underlying or sole cause of AD, and there are risks posed by to elimination diets in young patients with AD.[7,8]

In examining the relationship between AD and food allergy, it is important to clarify the distinction between "sensitization" and clinically relevant "allergy" to foods.[9] Sensitization is the production of IgE to a specific food by the cells of the immune system; a person would be "sensitized" to milk if milk-specific IgE is detected in their serum or if percutaneous skin testing to milk produces a 3 mm or larger wheal. However, allergy is present if there is a clinical reaction upon exposure to the food. A person could be sensitized to a food, yet eat it regularly without issue—and in fact, many do. In the setting of sensitization to tolerated foods, food elimination, or avoidance may increase individual risk of developing food allergy that may not otherwise have manifested, a point that will be discussed in greater depth later in this section.

There are increased rates of food sensitization in individuals with atopic dermatitis.[8] The general elevation in total serum IgE often seen in this population, especially when eczema is in a highly inflamed state, can result in elevated allergen-specific IgE that typically has no clinical significance.[10,11] A recent systematic review and meta-analysis found that pooled prevalence of food sensitization, food allergy, and challenge-proven food allergy among 225,568 individuals with AD was 48.4%, 32.7%, and 40.7%, respectively.[12] This was significantly higher than the prevalence of these conditions among those without AD, who reported pooled prevalence of 17.9%, 9.4%, and 15.5%, respectively. Children with AD had higher pooled food sensitization and food allergy than adults (49.8% and 31.4%, compared with 28.6% and 24.1%), again suggesting a stronger link in the early years of life. The highest pooled prevalence of food sensitization and food allergy in individuals with AD was found at 0 to 2 years of age, to be 54.4% and 39.2%, respectively.

Based on these abundance of data, it seems clear that atopic dermatitis, particularly when it emerges early in life, is a significant risk factor for food allergy.[7]

The gold standard for diagnosing food allergy is the double-blind, placebo-controlled oral food challenge (DBPCFC), and has been examined in studies to determine the true prevalence of IgE-mediated food allergy among infants and children with AD. IgE-mediated food allergy in this population ranges from 33% to 63% across several studies (though importantly, these studies excluded those who reported anaphylactic reactions or who had positive food-specific IgE elevated in a range typically diagnostic of allergy)[13–15]; These rates exceed the prevalence of food allergy in the general pediatric population, which was recently estimated at 7.6% based on a survey of 38,408 United States (US) households, and provides further evidence for AD as a risk factor for food allergy.[12,16] This finding was reiterated in HealthNuts, a large Australian study in which 20% of children with AD demonstrated allergy by oral food challenge (OFC) to peanut, egg white, or sesame seed, in comparison to 4% of children without eczema.[17]

Many of these studies highlight that age of onset and severity of eczema contributes to food allergy development, with earlier onset and more severe eczema augering greater allergy risk. Children with onset of AD before 3 months of age, for instance, have a significantly higher association with positive egg, milk, and peanut-specific-IgE.[18] In HealthNuts, one-year-old infants with eczema were 11 times more likely to have peanut allergy, and 6 times more likely to have egg allergy than children without eczema. Severity of disease is also a strong predictor of the development of both sensitization and allergy to foods, with more severe AD resulting in substantially greater rates of sensitization and allergy.[18,19]

IMMUNOLOGIC MECHANISMS LINKING ATOPIC DERMATITIS AND FOOD ALLERGY

The complex pathophysiology of AD involves the interplay of several factors including genetic predisposition, dysfunction of the epidermal barrier, and inflammation driven by T-cells.[20] While the immunologic mechanisms that link atopic and dermatitis are not fully understood, both involve a shared immunologic pathway that is associated with elevated serum IgE and a T-helper type 2 (Th2) response.[20–22] However, while AD has classically been considered a largely Th2-driven response, other T cells including Th1, Th17, and Th22 are now also known to contribute to AD pathogenesis, and different endotypes of AD are being identified with varying involvement of these cell classes.[23] Inflammation of the skin in AD is associated with the release of interleukin-4 (IL-4), interleukin-13(IL-13), and thymic stromal lymphopoietin (TSLP), all of which result in Th2 inflammation, Ig class switching, and higher IgE levels.[20,24] Among other atopic diseases, AD is associated more elevated average total serum IgE levels,

typically more than normal and in 100s to 1000s kU/L range. One hypothesis is that elevated total serum IgE in AD increases the likelihood of the production of food-specific IgE (sensitization) and the development of food allergy. Serum IgE levels have also been shown to be influenced by genetic modifications, in particular IL-13 variants.[25] IL-13 variants can result in higher total serum IgE levels, as well as sensitization to specific food allergens, most commonly hen's egg, in young children with AD.[26]

The epidermal barrier in AD demonstrates many functional defects including poor water retention, decreased ceramide lipid content, and reduced protective proteins such as filaggrin (FLG).[24] The dysfunction of the skin barrier in AD may in turn play a significant role in the development of food allergy and has been supported by several observations in studies. Individuals with a loss-of-function FLG mutation have a 3 to 5 times higher risk of AD, and are predisposed to develop aeroallergen sensitization, food allergy and asthma.[27–29]

The disrupted epidermal barrier may be particularly important in the development of food allergy in infants and young children with atopic dermatitis. While historically it was believed that sensitization to food allergens occurred primarily via allergen exposure in the gut, there is increasing evidence that sensitization can occur, and is significantly more likely to occur following primary allergen exposure on the skin. Moreover, this effect may be amplified in the setting of inflammation and impaired skin barrier that are characteristic of AD. One study supporting this found an increased rate of peanut sensitization with environmental exposure to peanut measured in living room dust in children with a history of AD.[30] Additional evidence of a skin route for peanut sensitization was demonstrated by a study in which the rate of peanut allergy was not affected by infant oral exposure to peanut but was increased in infants for whom topical creams containing peanut oil had been applied to inflamed skin before 6 months of age.[19] Another example of skin barrier dysfunction contributing to allergic sensitization is exemplified by the findings that mutations in the serine peptidase inhibitor Kazal type 5 genes are associated with asthma, AD, and with challenge-proven IgE-mediated food allergy, as noted in the Netherton Syndrome.[31] Further indication that skin barrier integrity may be a factor in food allergy development is illustrated by a retrospective study of infants under 1 year of age, for whom aggressive use of topical corticosteroid to shorten the course of eczema resulted in decreased food allergy development.[32]

DERMATOLOGIC CLINICAL MANIFESTATIONS OF FOOD ALLERGY

In an IgE-mediated allergic reaction to food, cutaneous symptoms may include acute (within minutes to hours) onset of urticaria, pruritus, angioedema, erythroderma, and flushing, and may or may not be associated with respiratory, gastrointestinal, circulatory or neurologic symptoms of a systemic allergic reaction.[33] Neither AD-onset and flares are typical of an acute allergic reaction nor is AD a hallmark of anaphylaxis. AD can develop following acute pruritus and scratching within 2 hours of an immediate allergic food reaction, or may not manifest until 6 to 48 hours later.[34,35] In 1985, Sampson *and colleagues* established that AD is linked to IgE-mediated food allergy in a study of 101 positive DBPCFCs in 113 children with severe AD.[36] While older studies of DBPCFCs have demonstrated that AD in children can be exacerbated by a food, it is not clear how often AD was observed as the only symptom.[7] Nonetheless, the most commonly implicated foods linked to worsening eczema, have been cow's milk, egg, and peanut.[7,37] One European study implicated birch pollen-related foods such as apple, carrot, hazelnut, and celery as AD triggers.[38] A more recent Dutch study looking at 1186 DBPCFCs in 682 children, did not identify AD or eczema flares to be common manifestations of food reactions.[35] These investigators found that children with AD were more frequently sensitized to food without clinical reactions compared with those without AD. Furthermore, they concluded that a child is unlikely to have an allergy to a food if the only symptom observed or reported following food ingestion or food challenge is AD. For adults, a causal link between AD and food allergy has not been well-studied or described.

In summary, the relationship between food allergy and AD is likely more impacted by AD being a risk factor for developing food allergy and not that food allergy is a trigger for developing or exacerbating AD. An understanding of this relationship has significant implications for the clinical management of AD, in particular with regard to food allergy testing, skin care, and dietary management, and has informed recently published practice parameters.[39]

To illustrate the diagnostic challenges of these conditions, the authors have prepared the following hypothetical cases, based on commonly encountered clinical scenarios.

- Case 1: A 9-month-old has a history of AD that initially developed around 2 to 3 months of age involving the cheeks, abdomen, and

neck. The infant was initially introduced to scrambled eggs around 6 months of age without immediate adverse reactions, and continued to eat eggs every 2 to 3 days for the next month. However, the infant's parents noticed worsening of eczema beginning around 6 months of age, despite treatment with prescription topical steroid. Allergy evaluation conducted at 7 months of age demonstrated egg sensitization, with negative skin prick testing but serum egg white specific IgE of 0.44 kU/L. The family decided to eliminate egg from the diet because of concerns about egg as possible triggers for eczema. Over the 3 months during which egg was avoided, the eczema significantly improved. However, at 9 months of age, within minutes of trying egg in the form of French toast, the patient experienced acute respiratory distress, facial swelling, and hives, requiring prompt treatment by family with intramuscular epinephrine, and transport to an emergency department for continued observation. Subsequent allergy testing at 10 months of age demonstrated egg white serum specific IgE of 12.2 kU/L and ovomucoid IgE of 9.4 kU/L. The family was advised to strictly avoid all forms of egg and to carry an epinephrine auto-injector at all times.

Case 2: A 3-year-old presented in early infancy with AD and their primary care provider tested the child and documented multiple food sensitizations by serum IgE laboratory testing. Based on this testing, any foods with positive tests were not introduced, and the family had been avoiding dairy, soy, wheat, peanuts, and tree nuts. In part due to these restrictions, the child demonstrated poor weight gain, picky eating, and developed vitamin D deficiency. At 3 years, the eczema was moderately well-controlled on a regimen of topical steroids and frequent emollient use. Testing for avoided foods were repeated, and over the course of 2.5 years, the patient passed allergist-supervised OFCs to soy, wheat, dairy, peanut, and almond, prompting introduction of the previously avoided foods, and dramatically improving the options for a diverse diet. There are plans to complete food challenges for the remaining tree nuts. The patient's parents, in collaboration with a pediatric allergist and dietician, continue to implement nutritional supplementation, feeding therapy, and offer a balanced diet to address his low weight.

As these cases demonstrate, it can be difficult to tease out the complex interplay of cause and effect between AD and food allergy, and hard to navigate the diagnostic and management challenges these conditions pose. The potential harms of allergy testing in the absence of prior clinical allergic reactions must also be carefully considered in this patient population.

Food-specific serum IgE levels and skin prick tests to individual foods cannot distinguish between food sensitization and clinical allergy, whereas observing an individual ingesting a food, either through home introduction or supervised OFC, provides the best evidence of tolerance versus allergy. During an OFC, skin manifestations are one of several objective clinical signs of allergy, which should be monitored. In a patient with underlying eczematous dermatitis, however, some skin symptoms can be difficult to interpret. Skin symptoms during a food challenge that consist only of worsening eczema or AD, possibly manifesting as increased erythema or pruritus of existing eczematous patches of skin, in the absence of urticaria, angioedema, or other immediate-type symptoms, can be confounding. If a patient's reaction during an OFC is limited to an eczema flare, is this a sufficient indicator of a positive OFC, and should the patient continue strict avoidance of a food? One early landmark study of children with severe AD found that 84% demonstrated cutaneous symptoms during oral food challenge, making this far from theoretic.[36] Also complicating the interpretation of eczema flares as a sign of food allergy is the phenomenon of skin erythema and pruritus that can result from the acidity of citrus fruits, tomato, and strawberries, as well as from any food having direct skin contact with the perioral region for many infants. Knowledge of the aforementioned confounders and pitfalls, as well as familiarity with the nature of a particular patient's eczema, should be considered during observation and interpretation of an OFC for a patient with underlying or active AD.

PRENATAL DIET, INFANT FORMULA, AND SUPPLEMENTS

There has been keen interest regarding whether the maternal diet during pregnancy and lactation, as well as whether formula choice and vitamin or probiotic supplementation may play a role in preventing or treating atopic dermatitis.[40] A 2008 Association of American Publishers (AAP) report, and 3 follow-up systematic reviews have concluded from published studies that exclusion of foods such as milk, egg, peanut, tree nuts, and fish during pregnancy or while breastfeeding do not prevent AD, including AD, in infants.[41] Furthermore studies of the effects of maternal dietary elimination during breastfeeding on infants who already

have AD are limited and have shown conflicting results, thus further research is needed.

Even without considering the maternal diet, breast milk by itself has been investigated for potential protective and beneficial effects with respect to all childhood atopic diseases, including AD. The evidence is not strong, but does suggest that there is an AD prevention benefit to exclusive breastfeeding for 3 to 4 months (but not beyond 4 months) for those with a first-degree family history of AD.[42–44] Numerous studies have also examined whether formula choice may impact the development of atopic disease, and results have been conflicting. Current consensus is that there is insufficient evidence that hydrolyzed or partially-hydrolyzed formulas prevent AD even in those who are considered high-risk for atopy, and this is reflected in the most recent AAP clinical report.[40] Furthermore, soy formula has not been shown to prevent atopy, and amino-acid elemental formulas have not been studied for their preventative effects.[45] Regarding essential fatty acids, which have anti-inflammatory properties; studies have not yet demonstrated convincing benefit for infant AD by maternal supplementation with omega-3, omega-6 fatty acids or evening primrose oil during lactation.[46]

Many studies have focused on supplementation for infants at risk of developing AD, as well as for children with AD, but so far results are not conclusive. A meta-analysis has shown that serum vitamin D is lower in patients with AD, and in particular for those with more severe AD, and vitamin D supplementation has been examined for potential therapeutic benefit in AD.[47] Furthermore, there has been considerable exploration of probiotics, in particular *Lactobacillus* and *Bifidobacteria*, and prebiotics, for their microbiome modulating and anti-inflammatory effects, and possible role in preventing or treating AD. A 2018 Cochrane review of 39 randomized controlled trials did not find that probiotics reduced the severity of eczema,[48] while there is some evidence from studies that prenatal and postnatal probiotics may reduce the risk of infants developing AD.[49] Given the heterogeneity of these studies, however, it is difficult to make conclusions about the preventative or therapeutic effects on AD, and there is no evidence-based recommendation about vitamin D supplementation, probiotics, or prebiotics at this time for breastfeeding parents and infants.

PREVENTION AND MANAGEMENT OF ATOPIC DERMATITIS AND FOOD ALLERGY

Optimization of skin care practices, including avoidance of irritants, aggressive moisturizer application, and the use of topical anti-inflammatory medication, is a cornerstone of the management of AD, and is recommended as the first-line therapy before considering food allergy testing or intervention.[7] However, it is unclear if aggressive treatment of AD may aid in preventing food allergy. Given the evidence for epicutaneous sensitization through a dysfunctional skin barrier, it has been hypothesized that moisturizer used to restore the skin barrier in AD, may reduce allergic sensitization.[2] In one study, early aggressive application of topical corticosteroids to shorten the duration of eczema followed by frequent emollient use in infants with AD was associated with a decrease in later development of food allergies.[32] A subsequent prospective, randomized controlled trial failed to replicate this finding, and in fact demonstrated the opposite finding: In the Enquiring About Tolerance study population, regular application of moisturizers to young infants' skin was associated with increased development of food allergy.[50] The reasons for this are unclear, and could potentially reflect increased likelihood of cutaneous exposure if food proteins are also present during moisturizing, or may indicate inappropriate moisturizer selection. However, studies have not produced conclusive evidence regarding whether moisturizer type, frequency, and age at application can contribute to preventing the development of food allergies.[2]

The recently published 2023 AD guidelines recommend against the use of elimination diets as an intervention for treatment of AD.[39] This stems from the potential risks of food elimination causing the development of IgE-mediated allergy, particularly for infants, as well as the risk of causing malnutrition as a result of dietary restriction. A systematic meta-analysis of dietary elimination for AD included 10 trials (599 patients, mean age of 1.5 years) that examined the harms and benefits of elimination diets for the treatment of AD.[51] The investigators concluded that elimination diets may lead to a slight but unimportant improvement in eczema severity, pruritus, and sleep disorder in those with mild to moderate AD. They also noted indirect evidence of an increased risk of development of peanut allergy when peanut was eliminated from the diets of infants with severe AD until age 5.[52] In a review of 298 pediatric patients with suspected food-triggered AD who underwent elimination diets, 19% went on to develop new immediate-type food reactions and 30% of these reactions were anaphylaxis.[53] In addition to the potential for an elimination diet to increase the likelihood of IgE-mediated food allergy, the 2023 AD guidelines panel proposed that additional harms of an elimination diet could include malnutrition

and undue burden for patients and caregivers.[39] They also stated that more research is needed to clarify the interplay between dietary elimination and the immunologic mechanisms that result in eczema flares because of food allergens. Evidence and expert guidance overall suggest that elimination of food from the diet to treat AD should be discouraged.

By contrast, early introduction of allergenic foods such as peanut and egg has been shown to significantly reduce the risk of FA in young infants, and many food allergy prevention guidelines now recommend this approach.[54] The data on AD with respect to food introduction are more mixed. One case-control study found that early solid food introduction before 4 months of age was associated with lower risk of AD reported via parent questionnaire.[55] However, other prospective cohort studies have found no difference, positive or negative, in rates of AD associated with timing of solid food introduction.[56,57] Overall, early food introduction remains a mainstay of FA prevention and should be recommended in accordance with current guidelines.

Finally, biologic medications have emerged as promising therapeutic options for the treatment of many allergic conditions, including asthma, chronic urticaria, nasal polyposis, eosinophilic gastrointestinal disease, atopic dermatitis, and food allergy. Rather than targeting individual allergens, these medications target specific molecular mechanisms that drive allergic reactions, in particular by inhibiting T_H2 T-cell, and some non-T_H2 cell, activation pathways. As a result, biologic agents offer a more targeted approach compared with traditional treatments, as well as the potential to treat multiple coexisting disease states at once.[58] With the recognition that the pathogenesis of both AD and food allergy involve skin barrier defects and type 2 allergic inflammation, some of the same agents are being investigated for both conditions.[59] In particular, biologics targeting IgE, IL-4, and IL-13, and alarmins (TSLP, IL-33, and IL-25), appear to be closest to clinical implementation.[58]

Of these, omalizumab, an anti-IgE monoclonal antibody, has been investigated the most for its role in improving reaction thresholds and as an adjunct to oral immunotherapy for patients with IgE-mediated food allergies.[60] By binding to circulating IgE antibodies, and downregulating antigen recognizing FCϵRI receptors on basophils and mast cells, omalizumab can prevent the allergic cascade and reduce the severity of allergic reactions when an individual encounters a known allergen. While not shown to be an efficacious treatment as a therapy for AD, numerous clinical trials have demonstrated the safety and efficacy of omalizumab in treating food allergies, both as an adjunct to oral immunotherapy, as well as monotherapy.[61–63] Very recently, omalizumab was approved by the FDA for individuals 1 year and older to reduce the risk of harmful reactions for those who have IgE-mediated food allergy.[64] This approval was based on results of a multicenter randomized placebo-controlled clinical trial that demonstrated that 16 weeks of treatment with omalizumab significantly improved the reaction threshold for reacting to peanut and other common food allergens.[65]

Dupilumab, a monoclonal antibody that inhibits the action of IL-4 and interleukin-13, has been at the forefront of AD treatment since its approval by the FDA for this indication in 2017. Dupilumab has also been approved for treatment of eosinophilic gastrointestinal disease in 2022. By targeting cytokines involved in the inflammatory processes associated with AD, dupilumab therapy results in significant improvements in skin symptoms, including itchiness, redness, and skin lesions, and has been found to reduce serum specific IgE to many allergenic foods.[66,67] Currently, trials are underway to evaluate its effect on food allergy, and this medication also appears to offer promise as an adjunct therapy with oral immunotherapy.[58] An understanding of overlapping immunologic mechanisms in the pathogenesis of AD and food allergy, has led to current investigation of the novel agent abrocitinib, a Janus-kinas-1 inhibitor approved for treatment of AD, for use as a potential food allergy therapeutic.[68]

While extremely promising and exciting, the use of biologic medications for the treatment of FA and AD is not without challenges. These medications can be extremely costly, and their long-term safety profiles are still being studied. However, collectively, these medications represent a potential paradigm shift in the treatment of atopic conditions, including but not limited to AD and food allergy, and will undoubtedly become more widely used therapies in the future.

SUMMARY

While there is clearly an association between AD and food allergy, the conundrum is whether there is a causal relationship between these atopic conditions. Does food allergy cause AD or does AD present a risk factor for the development of food allergy? As this article has attempted to demonstrate, the answer is not straightforward and there are still gaps in our knowledge. Children with moderate-to-severe persistent AD are at an increased risk of developing food allergy. A defective skin barrier and inflammatory milieu are likely

serving as the entry point for sensitization to food allergens. However, far more children are "sensitized" to foods than are clinically allergic, and skilled allergy evaluation is crucial for establishing an accurate food allergy diagnosis. There is some danger to removing foods that are tolerated from the diet but to which a patient may be sensitized. Furthermore, delayed introduction and prolonged avoidance of allergenic foods can result in the development of IgE-mediated food allergies.

Elimination diets are not generally recommended for the treatment of atopic dermatitis, and subjecting all children with AD to unscrupulous food elimination is discouraged by expert panels and in several published guidelines. Additionally, dietary interventions, including maternal elimination diets and supplements during pregnancy, lactation, and the early infant diet, have not yet been proven to prevent or treat AD in infants and toddlers. Biologic agents used to manage many atopic conditions, are being examined in clinical trials for their therapeutic potential for both AD and food allergy. Given the evident relationship between atopic dermatitis and food allergy, these modalities are an area of continued interest and active research.

CLINICS CARE POINTS

- Typical cutaneous findings in IgE-mediated allergy are acute onset of urticaria, pruritus, angioedema, erythroderma, or flushing. AD onset and flares are not typical manifestations of food allergic reactions.
- Frequent moisturization and the use of topical anti-inflammatory medications are first-line therapies for AD, and should be optimized before considering food allergy testing in the absence of a history of acute food reactions.
- Food elimination diets are not recommended in the management of atopic dermatitis. Delayed introduction or elimination of foods from children's diets may increase the risk of food allergy development or malnutrition.
- Early introduction of allergenic foods in infancy has been shown to decrease the risk of food allergy development.
- "Sensitization" to foods (ie, positive serum IgE or skin prick testing) is far more common than clinical allergy. Skilled allergy evaluation is crucial for establishing an accurate food allergy diagnosis, particularly among children with AD.

DISCLOSURES

B. Trogen: Nothing to disclose. M. Verma: Nothing to disclose. A. Cox: Nothing to disclose. S. Sicherer: Reports royalty payments from UpToDate and from Johns Hopkins University Press; grants to his institution from the National Institute of Allergy and Infectious Diseases, United states, from Food Allergy Research and Education, United states, and from Pfizer, United states; and personal fees from the American Academy of Allergy, Asthma and Immunology as Deputy Editor of the Journal of Allergy and Clinical Immunology: In Practice, outside of the submitted work.

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