

Dermatologic Allergy

The Role of Food Allergy in Atopic Dermatitis

VINCENT S. BELTRANI, M.D.

*Department of Dermatology
Columbia University
New York, New York*

*Department of Medicine
Division of Allergy & Rheumatology
UMDNJ, Newark, New Jersey*



Vincent S. Beltrani, M.D.
*Department of Dermatology
Columbia University
New York, New York
UMDNJ, Newark, New Jersey*

DR. VINCENT S. BELTRANI, M.D., is Associate Professor of Dermatology at Columbia University's College of Physicians & Surgeons, and also at the Department of Allergy and Rheumatology at the University of Medicine and Dentistry of New Jersey in Newark. He received his M.D. degree from Columbia in 1957. He completed an internship at King's County Hospital in Brooklyn, NY, and a surgical residency at the Brooklyn VA Hospital. From 1959 to 1964 he was involved in Family Practice in Pleasant Valley, NY. In 1964 he entered a Dermatology Training Program, followed by an allergy fellowship from 1964 to 1967. He completed the boards in dermatology in 1968, and also again in 1975 when the AAAAI boards separated from those of internal medicine and pediatrics. He was at the time one of the five doubly boarded physicians in dermatology/allergy. Dr. Beltrani is the author of at least 50 publications and textbook chapters regarding dermatologic allergy.

In this column, "Dermatologic Allergy," Dr. Beltrani will be discussing various specific skin diseases and will also be inviting contributions from other guests. These commentaries on allergic skin disorders will be a part of each issue for the coming year.

ONE OF THE PRO-CON SESSIONS at the last Academy meeting in Miami included the topic "Food Allergy Plays a Major Role in Atopic Dermatitis." Because the dermatologists were attending their annual Academy meeting concomitantly in San Francisco, our intended adversary (Jon Hanifin) could not present his "con" point of view. Therefore, I was volunteered to represent him (and the supposed dermatologist's stance). How ironic, (I felt) since I recently was asked to represent the "pro" view of the role of "allergy" in atopic dermatitis at a recent Academy of Dermatology Meeting.

For those who were present at our (AAAAI) session, it should have been readily evident that my "pro" opponent failed to present her assignment of "food playing a *major* role in AD" and presented instead her experience that food *can* be a factor in AD! Neither I nor most dermatologists disagree. Our "controversy" was to debate food allergy as a "minor" or "major" factor.

Needless to say, there was virtually no confrontation. I therefore reviewed my (and the majority of dermatologists') position, of neither trivializing nor highlighting food allergy as a special cause of AD. I reminded the audience that Sulzberger and Wise were the first to recognize the dermatologic syndrome as "atopic eczema" (in 1921) when they acknowledged the association of positive skin prick tests with this group of eczematous patients. Simpson and Hanifin recently published that "the atopic appellation was unfortunate because it resulted in a conceptually misdirected focus on allergy causing the pruritic eczematous lesions of AD."¹ This impression is, perhaps self-servingly, unaccepted by some allergists. Dr. Amy Paller, a pediatric dermatologist who authored the chapter on atopic dermatitis in Hurwitz's textbook on *Clinical Pediatric Dermatology* states that "IgE-mediated food allergy is common in children with atopic dermatitis, but positive SPT and RAST tests, especially to foods, often show no correlation with clinical manifestations of AD, while negative SPT or RAST tests are highly predictive at eliminating potential allergens."² She later affirms that "food allergens have been noted to induce rashes in 40% of children with moderate to severe atopic dermatitis, many of which are urticarial or maculo-papular in character, and not eczematous. Despite the frequency of reactivity, pediatric dermatologists estimate that up to 10%–15% of children with moderate to severe atopic dermatitis have food allergies that may be relevant to their AD."

The above statements are virtually in complete accordance with a recent publication by Hugh Sampson, the well recognized "food allergy" maven: "Nowhere does the discrepancy between a positive SPT and the absence of clinical symptoms seem more apparent than in the various tests used to diagnose food allergy. The positive SPT does *not* mean the patient will experience an allergic reaction to that food," and "Negative tests are very reassuring that the food can be ingested safely—except in disorders that are not due to IgE antibodies."³ In my preparation of a set of slides on atopic dermatitis, I asked Dr. Sampson to contribute a section on "Food Allergy and AD," which clearly states that "food allergy should be considered a 'trigger' in the early infancy group of patients with AD, when the eczema is generalized, moderate to severe, and poorly or unresponsive to avoidance of the 'other' triggers and/or topical anti-inflammatory therapy." In his studies of unselected children with moderate to severe AD evaluated by blinded challenges, he has reported that 33%–37% have clinically significant food allergy, and the severity of the AD correlates positively with the risk for food hypersensitivity. Food allergy appears to be much less prevalent in adults with AD.

The dermatologists are in complete accordance with Dr. Sampson's emphasis that "although certain allergy tests are very accurate at detecting and quantitating IgE antibodies, they must be interpreted by someone who is highly skilled in the diagnosis of food allergy and who understands the clinical limitations of these tests. Positive SPT or RAST tests to a food someone is eating regularly with no allergic symptoms does not justify eliminating those foods from their diet." Dr. Paller avers that the relationship between reactivity to foods and AD itself is complex. She agrees "that food allergens have been noted to induce rashes in 40% of children with moderate to severe AD, but many of these rashes are urticarial, or maculopapular, and not eczematous. When Dr. Sampson biopsied the skin of those DBPC patients with positive food reactions, the histology did not reveal spongiosis ("eczema"). His assumption that continued ingestion of the putative allergen and further scratching would "later" result in an eczematous eruption has never been verified (nor would it be allowed by an IRB).

Not surprisingly there are no specific publications addressing the specific role of food in AD. Only the (international) textbook *Pediatric Dermatology*, has a detailed chapter entitled "The Role of Diet in Atopic Dermatitis," authored by David, Patel, and Ewing, and states: "There is no evidence that food hypersensitivity causes AD, *e.g.*, randomized, controlled studies. It is, however, one of a number of triggers which can exacerbate pre-existing AD in a subset of patients."⁴ I have published a list of all the referenced "triggers" of itch,⁵ and IgE-induced reactions are included but are not recognized as having a greater importance than any of the other triggers, (certainly not as important as scratching, xerosis, and heat/sweating). The latter textbook also points out that "too often, strict avoidance of putative foods are not associated with improvement," and it should be readily apparent to most practitioners that "parental reports to food intolerances are notoriously unreliable." Lastly they report, "Controlled studies have demonstrated that improvement of AD on an elimination diet are associated by a marked placebo effect." In their experience,

DERMATOLOGIC ALLERGY

“Although dietary elimination may be associated with immediate improvement, the long-term outcome appears to be unaffected by dietary success or failure.” From the above dermatologic authorities, we can only conclude that IgE-induced food reactions can certainly cause an exacerbation of the eczema in a subset of patients with moderate to severe AD, especially in their first year of life. The following summary lists the relative indications for eliminating a food (or foods) from the diet of patients with moderate to severe AD:

- History of food intolerance
- Extensive body surface area involved (>20%)
- Children younger than two years of age (especially <12 months)
- Parents very enthusiastic to try a diet
- Presence of other symptoms possibly attributable to food hypersensitivity, *e.g.*, diarrhea, asthma, etc.

In conclusion, the “con” argument regarding the role of food being a “major” factor is a very clear negative, with a corollary which recognizes a subset of those moderate to severe atopic dermatitis patients in whom a limited number of foods may be one of several other triggering factors. The lack of any “pro” discussion following my presentation leaves me with the impression that no one in the audience disagreed with me strongly enough to comment. Therefore, could this controversy essentially involve just the “over-zealous advocates” versus the “more skeptical adversaries”?

REFERENCES

1. Simpson EL, Hanifin JM. Atopic dermatitis. *Med Clin N Am* 2006;90(1):149–167.
2. Hurwitz S, ed. In: *Clinical pediatric dermatology*, 2nd ed., New York: Elsevier, 2006.
3. Sampson HA. Food allergy testing: When, why, and what does it mean? *Food Allergy News*, 2006;15:3–6.
4. David , Patel , Ewing . The role of diet in atopic dermatitis. In: J Hopper, A Oranje, N Prose, eds. *Pediatric dermatology*. Oxford, UK: Blackwell Science, 2000.
5. Beltrani, VS. The clinical spectrum of atopic dermatitis. *J Allergy Clin Immunol* 1999;104:S87–S98.

Address correspondence to:
Vincent S. Beltrani, M.D.
29 Fox Street
Poughkeepsie, NY 12601

E-mail: alderm31@earthlink.net

This article has been cited by:

1. 2006. LiteratureWatchLiteratureWatch. *Pediatric Asthma, Allergy Immunology* **19**:3, 190-194. [[Citation](#)] [[PDF](#)] [[PDF Plus](#)]