

Letters

ALLERGY TO PUMPKIN AND CROSS-REACTIVITY TO POLLENS AND OTHER FOODS

IgE-mediated allergy to pumpkin flesh is rare, with 1 reported case describing a systemic reaction after ingestion of soup that contained pumpkin flesh.¹ In addition, 4 case reports also describe systemic reactions to pumpkin seed.^{2,3} The case involving pumpkin flesh demonstrated several IgE-binding bands shared among pumpkin, muskmelon, watermelon, cucumber, and zucchini, all of which are members of the Cucurbitaceae (gourd) family.¹ To our knowledge, no reports have assessed cross-reactivity of pumpkin flesh with either pollens or Rosaceae fruits. In this report, we describe the clinical and immunologic findings of 2 previously unreported cases of pumpkin flesh anaphylaxis: anaphylaxis after cutaneous-only exposure and in vitro inhibition of aeroallergens and peach with pumpkin.

Patient 1 was an 18-year-old, African American man with mild seasonal allergic rhinoconjunctivitis and claimed food allergy to strawberry, grape, and banana. The patient developed generalized pruritus, shortness of breath, and throat tightness after eating pumpkin cake made in a home economics class at school. He was administered epinephrine and admitted overnight for observation but required no further treatment except oral antihistamines. On his own volition, the patient subsequently ingested the nonpumpkin cake ingredients (wheat flour, egg, butter, sugar, and baking powder) without reaction. The serum level of specific IgE (sIgE; ImmunoCAP; Phadia, Portage, Michigan) for pumpkin was elevated (33.50 kU/L), and skin prick and sIgE tests for banana, strawberry, peach, cucumber, grape, house dust mite, cat, dog, Bermuda and timothy grasses, ragweed, Russian thistle, pigweed, oak, mountain cedar, elm, olive, and box elder produced positive results. Skin testing was not performed for pumpkin.

Antigen extract (excluding rind and seeds) was prepared from fresh pumpkin as previously described.⁴ Electrophoresis of the extract via sodium dodecyl sulfate–polyacrylamide gel electrophoresis (SDS-PAGE) and IgE immunoblot techniques were performed. SDS-PAGE revealed several protein bands, ranging from 6 to 225 kDa, and immunoblot revealed sIgE to 8 bands, ranging from 20 to 225 kDa (Figure 1). Preadsorption of the serum with pumpkin extract showed that pumpkin completely inhibited cucumber, peach peel, Bermuda grass, Russian thistle, and ragweed.

Patient 2 was an 8-year-old, white boy with moderate persistent asthma, perennial allergic rhinoconjunctivitis, and known food allergy to peach, nectarine, and soy. The patient developed nasocular pruritus, sneezing, eyelid swelling, and chest tightness while carving a pumpkin. He had no prior reactions to pumpkin. He had not ingested any of the pumpkin but rubbed his eyes after putting his hand inside the pumpkin. No other foods were ingested around this time. His symptoms were treated with a short-acting β -agonist and resolved without further sequelae. Percutaneous skin test results were positive to fresh pumpkin (10-mm wheal $\times$ 40-mm erythema) and pumpkin seed (4-mm wheal $\times$ 20-mm erythema). Skin test

results were also positive to peach, soybean, cucumber, watermelon, house dust mite, dog, Bermuda grass, Russian thistle, mugwort, ragweed, and *Alternaria*. sIgE test results were positive for zucchini, cantaloupe, and watermelon. Immunoblot showed sIgE to 6 bands, ranging from 15 to 100 kDa (Figure 1). Preadsorption of the serum with pumpkin extract showed that pumpkin did not inhibit cucumber, Bermuda grass, or ragweed sIgE bands.

Both of these highly atopic patients experienced anaphylaxis to multiple foods with the common food allergen: pumpkin flesh. Furthermore, both patients demonstrated sIgE to Rosaceae foods (peach), aeroallergens (Bermuda grass, Russian thistle, ragweed), and Cucurbitaceae (cucumber). Interestingly, patient 1 demonstrated similar cross-reactive molecular weight proteins for aeroallergens, Rosaceae, Cucurbitaceae, and pumpkin. In contrast, pumpkin did not inhibit sIgE bands (eg, cucumber, Bermuda grass, ragweed) for patient 2. It is noteworthy that for patient 2 exposure may have been inhalational in addition to cutaneous.⁵

Profilins and lipid transfer proteins are ubiquitous plant pan-allergens that may be a principal cause of cross-sensitization between pollens and plant-derived foods.⁶ Patient 2 in this series had an sIgE band to pumpkin at 15 kDa that potentially could represent a profilin or LTP homologue. Yet, this patient did not demonstrate cross-reactivity to cucumber or the pollens evaluated. Patient 1, on the other hand, demonstrated multiple cross-sensitizations but had no sIgE band to pumpkin smaller than 20 kDa. We theorize that pumpkin flesh allergy can result from different epitopes. In the first group, cross-reactivity between pumpkin flesh and pollens occurred with fruits from the Cucurbitaceae and Rosaceae families, whereas in the second group cross-reactivity with these allergens did not occur. The difference in the character of their reactions, one from ingestion of heated pumpkin flesh and the other through cutaneous and/or inhalational exposure of raw pumpkin, may be significant in this regard as well. We did not subject the patients to challenge testing given the severity of their reactions.

In conclusion, although pumpkin flesh allergy is rare, it can occur from a variety of exposure routes. Patients with pumpkin flesh allergy are highly atopic with multiple food and aeroallergen sensitivities. Some patients with pumpkin flesh allergy will have cross-sensitization to foods from the Rosaceae and Cucurbitaceae family and to pollens such as ragweed, Bermuda grass, and Russian thistle.

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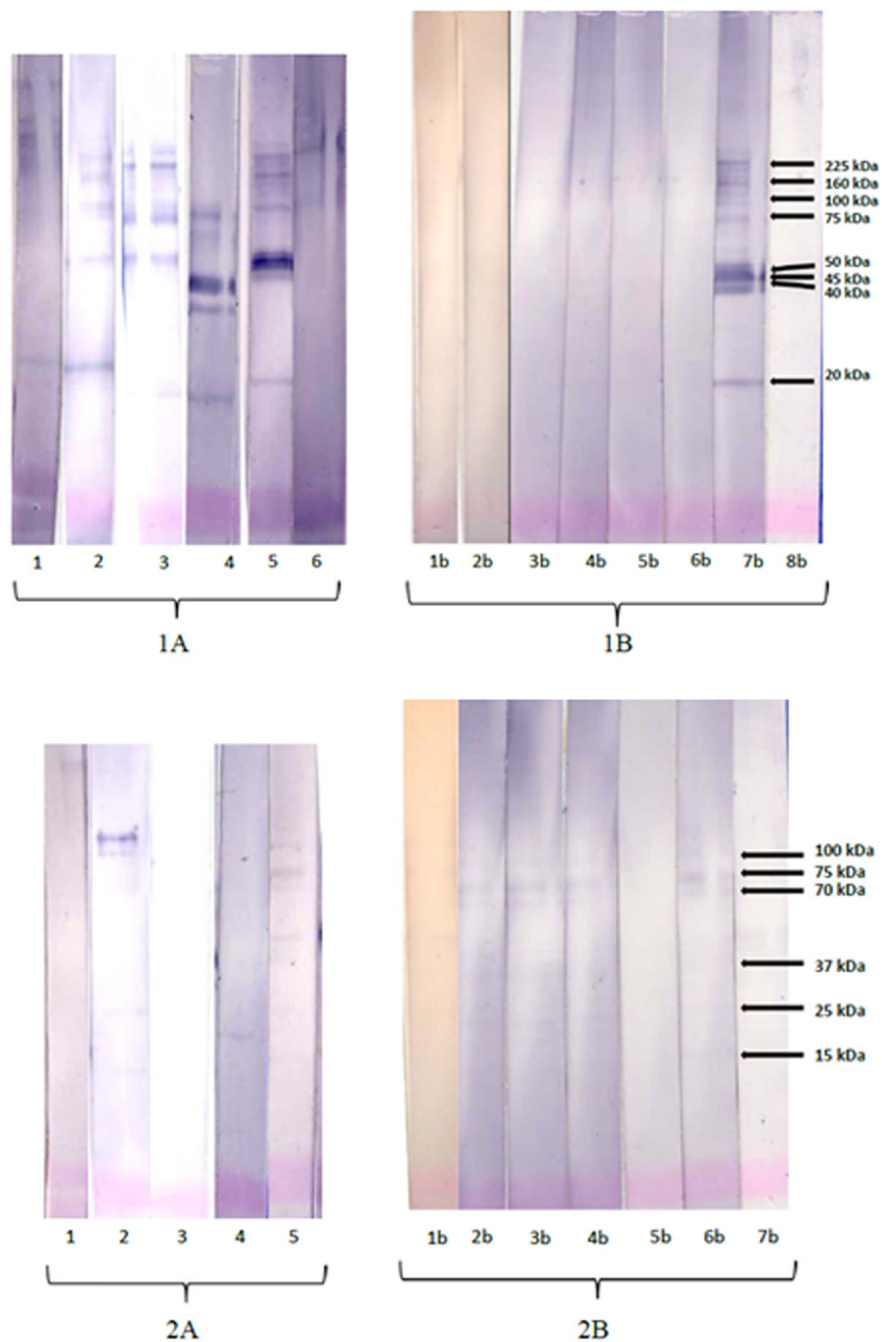


Figure 1. Top, Patient 1. A, IgE immunoblot with cucumber (lane 1), ragweed (lane 2), thistle (lane 3), Bermuda grass (lane 4), pumpkin flesh (lane 5), and peach (lane 6). B, After preincubation of patient's serum with pumpkin flesh, inhibition immunoblot analysis of IgE antibody binding with cucumber (lane 1b), peach peel (lane 2b), ragweed (lane 3b), thistle (lane 4b), Bermuda grass (lane 5b), pumpkin flesh (lane 6b), uninhibited pumpkin flesh (lane 7b), and cord blood control (lane 8b). Bottom, Patient 2. A, IgE immunoblot with cucumber (lane 1), ragweed (lane 2), thistle (lane 3), Bermuda grass (lane 4), and pumpkin flesh (lane 5). B, After preincubation of patient's serum with pumpkin, inhibition immunoblot analysis of IgE antibody binding with cucumber (lane 1b), ragweed (lane 2b), thistle (lane 3b), Bermuda grass (lane 4b), pumpkin flesh (lane 5b), uninhibited pumpkin flesh (lane 6b), and cord blood control (lane 7b).

1. Figueredo E, Cuesta-Herranz J, Minguez A, et al. Allergy to pumpkin and cross-reactivity to other Cucurbitaceae fruits. *J Allergy Clin Immunol*. 2000;106:402–403.
2. Fritsch R, Ebner H, Kraft D, Ebner C. Food allergy to pumpkinseed: characterization of allergens. *Allergy*. 1997;52:335–337.
3. Casper R, Kurup VP, Lusting JV. Pumpkin seed anaphylaxis [abstract]. *Ann Allergy Asthma Immunol*. 2007;98:59.
4. Jordan-Wagner DL, Whisman BA, Goetz DW. Cross-allergenicity among celery, cucumber, carrot, and watermelon. *Ann Allergy*. 1993;71:70–79.
5. Ramirez DA Jr, Bahna SL. Food hypersensitivity by inhalation. *Clin Mol Allergy*. 2009;7:4.
6. Egger M, Mutschlechner S, Wopfner N, Gadermaier G, Briza P, Ferreira F. Pollen-food syndromes associated with weed pollinosis: an update from the molecular point of view. *Allergy*. 2006;61:461–476.

SERUM INTERLEUKIN 9 IN ALLERGIC RHINITIS

To the Editor: Various populations of effector and regulatory T cells have been shown to play a crucial role in allergic inflammation.

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tion.¹ Depending on the substances coexposed with the allergen and the status of the cells and cytokines in the milieu, CD4⁺ naive T cells can differentiate into T_H1, T_H2, T_H9, or T_H17 effector cells.² On the basis of their respective cytokine profiles, responses to chemokines, and interactions with other cells, these T-cell subpopulations can promote different types of inflammatory responses. In this regard, transforming growth factor β in the presence of interleukin (IL) 4 reprograms T_H2 cell differentiation and leads to the development of a new population of T_H9 cells that produce IL-9 and IL-10.³

T_H9 cells lack suppressive function and constitute a distinct population of effector T cells that promote tissue inflammation and mucous production.^{4,5} In particular, IL-9 is an important stimulus for tissue infiltration by mast cells. The cell sources of IL-9 include T cells, eosinophils, neutrophils, and mast cells. It has been reported that IL-9 is upregulated in the nasal mucosa during the pollen season and correlates with tissue infiltration by eosinophils.⁶ However, this study was based on nasal biopsy specimens; therefore, we aimed to measure serum IL-9 in a group of patients with allergic rhinitis (AR) to pollen.

Thirty-eight patients with AR to pollen were evaluated during the pollen season, when they were symptomatic. Skin prick test and

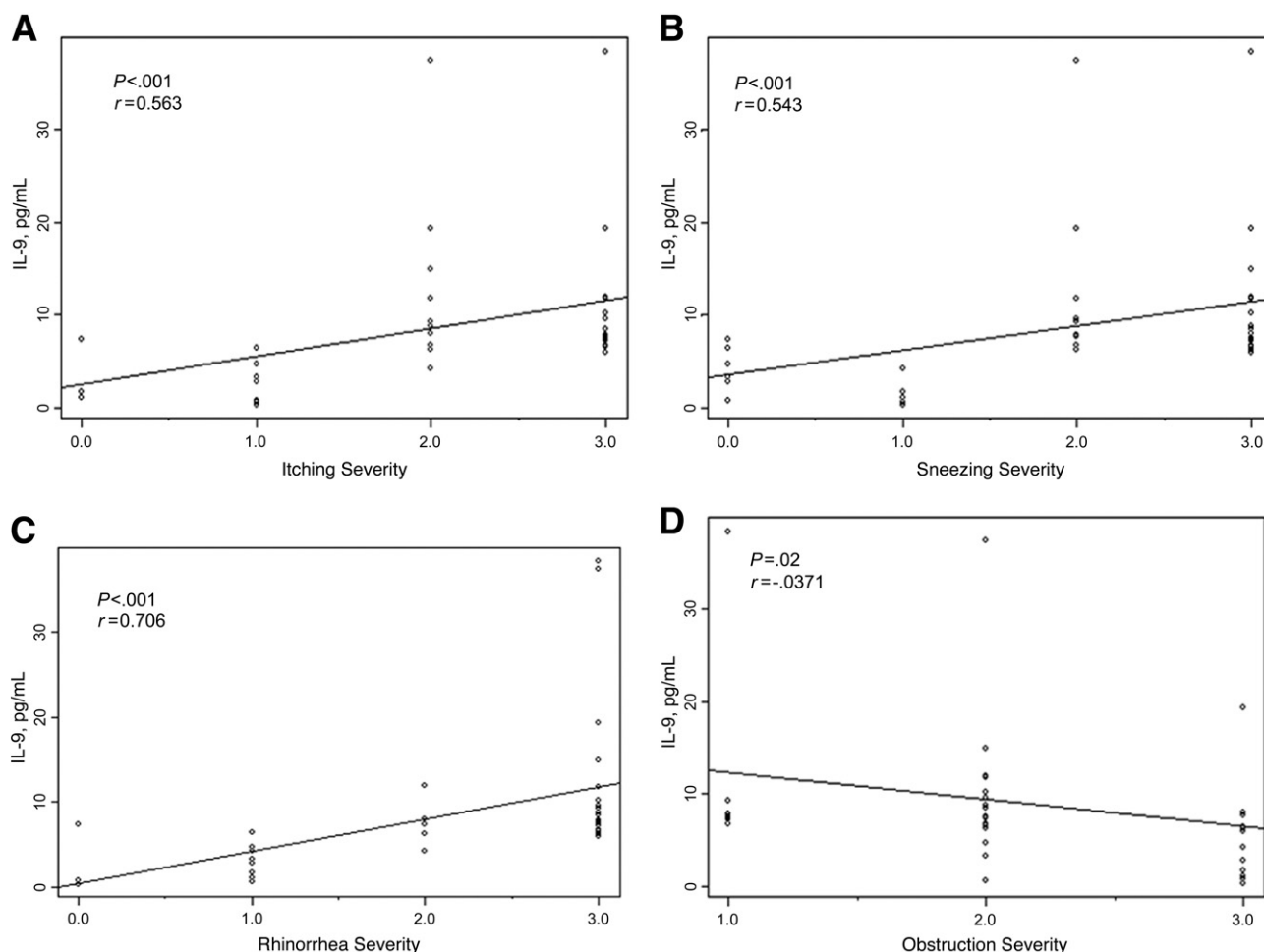


Figure 1. Correlation, expressed with the Spearman correlation coefficient (r) and P value, between serum interleukin 9 (IL-9) and nasal symptoms in allergic patients: itching (A), sneezing (B), rhinorrhea (C), and obstruction (D).