

1. Chang TW, Wu PC, Hsu CL, Hung AF. Anti-IgE antibodies for the treatment of IgE-mediated allergic diseases. *Adv Immunol.* 2007;93:63–119.
2. Spector SL, Tan RA. Advances in allergic skin disease: omalizumab is a promising therapy for urticaria and angioedema. *J Allergy Clin Immunol.* 2009;123:273–274.
3. Sheinkopf LE, Rafi AW, Do LT, Katz RM, Klaustermeyer WB. Efficacy of omalizumab in the treatment of atopic dermatitis: a pilot study. *Allergy Asthma Proc.* 2008;29:530–537.
4. Spector SL, Tan RA. Effect of omalizumab in patients with chronic urticaria. *Ann Allergy Asthma Immunol.* 2007;99:190–193.
5. Kaplan AP, Joseph K, Maykut RJ, Geba GP, Zeldin RK. Treatment of chronic autoimmune urticaria with omalizumab. *J Allergy Clin Immunol.* 2008;122:569–573.
6. Corren J, Shapiro G, Reimann J, et al. Allergen skin tests and free IgE levels during reduction and cessation of omalizumab therapy. *J Allergy Clin Immunol.* 2008;121:506–511.
7. Slavin RG, Ferioli C, Tannenbaum SJ, Martin C, Blogg M, Lowe PJ. Asthma symptom re-emergence after omalizumab withdrawal correlates well with increasing IgE and decreasing pharmacokinetic concentrations. *J Allergy Clin Immunol.* 2009;123:107–113.

ALLERGY TO PRAIRIE DOG LIPOCALINS

Prairie dogs are mammals that belong to the *Cynomys* genus of the Scuridae family. They are small, sociable, short-tailed, ground-dwelling creatures that live in colonies that typically inhabit short-grass prairies. Prairie dogs are not really dogs but rodents closely related to ground squirrels, chipmunks, and marmots. They are given the name prairie dog because of their habitat and warning call, which sounds similar to dogs barking. Native of North America, they are usually found in the United States, Canada, and Mexico. Their popularity is progressively increasing, and they are becoming a common pet. The role of the other members of the Scuridae family as a cause of allergic symptoms is known, but no cases of demonstrated IgE-mediated allergy to prairie dogs have been reported.

A 41-year-old man sought care for a 6-month history of perennial asthma and rhinitis. The patient related these symptoms as coinciding with the admission of 2 black-tailed prairie dogs to his house and referred to aggravation when he had contact with the bedding material of the animals.

Prairie dog hair was homogenized and extracted. Small, round balls of prairie dog feces with urine were obtained from the bedding material. Protein extracts, hair, and feces-urine were prepared by homogenization in phosphate-buffered saline, dialyzation, and lyophilization. Skin prick tests to commercial common aeroallergens were performed with positive results (wheal ≥ 3 mm) to *Olea europaea* pollen, dust mites, and cat, dog, horse, and hamster epithelium. No skin prick tests were performed with any of the prairie dog extracts (PDEs) for sanitary reasons. Serum specific IgE levels against PDEs were measured with the enzyme allergosorbent test, revealing 1.7 kU/L to prairie dog hair and 2.4 kU/L to feces-urine. Both PDEs were analyzed by sodium dodecyl sulfate–polyacrylamide gel electrophoresis (SDS-PAGE) as described by Laemmli,¹ showing protein bands ranging from 120 to 6 kDa (Fig 1). SDS-PAGE IgE immunoblotting assays with the patient's serum were performed and revealed IgE reactivity with a 21-kDa protein (PP1) in both PDEs and with proteins of 18 kDa (PP2) and 17 kDa (PP3) only in the feces-urine extract (Fig 1). The bands of the feces-urine extract were manually excised from the gel, digested with trypsin, and analyzed by matrix-assisted laser desorption/ionization time of flight and liquid chromatography coupled to tandem mass spectrom-

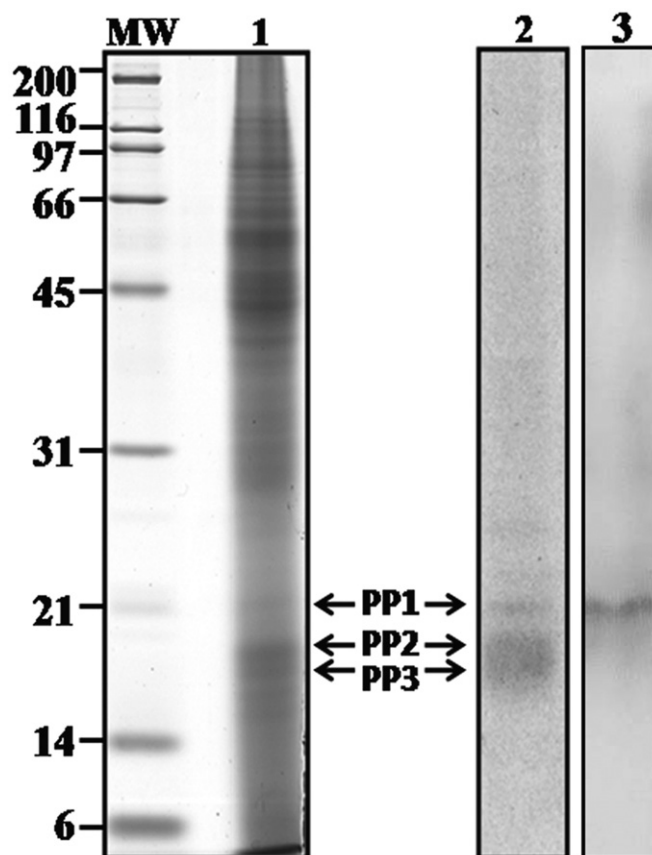


Figure 1. Identification of prairie dog proteins. Lane 1, Sodium dodecyl sulfate–polyacrylamide gel electrophoresis after Coomassie blue staining with the feces-urine extract. Lane 2, IgE immunoblot of feces-urine extract. Lane 3, IgE immunoblot of hair extract. MW indicates molecular weight marker; PP1, 21-kDa protein; PP2, 18-kDa protein; PP3, 17-kDa protein.

etry (MC/MC) following the methods of Pastor et al.² The proteomic analysis was performed in the Proteomic Service of the Complutense University of Madrid, a member of the ProteoRed Network. Sequencing of the resulting peptide (TVIIAADNVEKIEKDGDIR) in PP1 and PP2 by MS/MS (Fig 1) showed high homology with the corresponding regions in the primary structure of different lipocalins (with the mouse major urinary protein [73%] and with aphrodisin from a female hamster [78%]). The analysis of PP3 (Fig 1) did not match any previously reported allergens when compared with the databases. However, 2 peptides of this band were analyzed by MS/MS, obtaining the sequences DPAELTGNWR and EELTGAWR, which contained the motif glycine-X-tryptophan (GXW), characteristic of the lipocalin family.

The lipocalin protein family is a large group of small extracellular proteins that exhibit great functional diversity with roles in retinol transport, invertebrate cryptic coloration, olfaction and pheromone transport, and prostaglandin synthesis. Despite the high structural homology of the group, the sequence identity in the family could be as low as 20%. However, the sequence motif GXW is present in all of them.³ Thus, we believe that PP3 could be a member of the lipocalin family. Most of the mammalian-derived major allergens belong to this group^{4,5} like the major urinary protein (17.8 kDa) and

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hamster aphrodisin (17 kDa), and their allergenicity has been demonstrated.^{3,4}

There is one case of anaphylaxis after a prairie dog bite reported in the literature; allergens derived from the saliva were speculated to be involved in the reaction, but no immune mechanism was demonstrated.⁶ To our knowledge, we present the first demonstrated case of IgE-mediated allergy to prairie dog that suggests lipocalins as the relevant allergens.

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1. Laemmli UK. Cleavage of structural protein during assembly of head of the bacteriophage T4. *Nature*. 1970;227:680–685.
2. Pastor C, Cuesta-Herranz J, Cases B, et al. Identification of major allergens in watermelon. *Int Arch Allergy Immunol*. 2009;149:289–290.
3. Flower DR. The lipocalin protein family: structure and function. *Biochem J*. 1996;318:1–14.
4. Virtanen T, Kinnunen T. Mammalian allergens. *Clin Allergy Immunol*. 2008;21:201–218.
5. Virtanen T, Zeiler T, Mäntytjärvi R. Important animal allergens are lipocalin proteins: why are they allergenic? *Int Arch Allergy Immunol*. 1999;120:247–258.
6. Onaka A, Watase J, Matusaka M, et al. A case of anaphylaxis after a prairie dog bite. *Chudoku Kenkyu*. 2004;17:155–158.

ALLERGY TO PUMPKIN WITH CYCLOPHILIN AS THE RELEVANT ALLERGEN

Pumpkin (*Cucurbita pepo*) is a plant that belongs to the Cucurbitaceae family, genus *Cucurbita*. Similar to some squash, it creeps on the ground and is robust, and its fruit can vary greatly in shape and color. Pumpkins are cultivated all around the world and their growing ranges from agricultural to ornamental purposes. Although allergy to fruits belonging to the Cucurbitaceae family, especially melon, have been reported as 1 of the most frequent causes of food allergy,¹ there is only 1 indexed reported case concerning allergy to pumpkin, which described an IgE-mediated mechanism and cross-reactivity with other Cucurbitaceae fruits.²

A 24-year-old woman presented with a history of itching sensation in her lips, mouth, and pharynx within minutes of eating pumpkin and melon. She did not have any symptoms with other

foods belonging to the Cucurbitaceae family. She reported a history of seasonal rhinoconjunctivitis attributable to pollens as the only additional atopic symptom.

Pumpkin extract (PE) and melon extract (ME) were prepared by homogenization in phosphate-buffered saline, dialysis, and lyophilization. Skin prick tests to commercial common aeroallergens were performed with positive results (wheal ≥ 3 mm) to *Olea europaea*, grass pollen, dust mites, and cat epithelium. The results of skin prick tests with commercial fruit allergens were positive to melon and peach and negative to kiwi, grape, and banana. The results of skin prick-prick tests performed with raw pumpkin, melon, and cucumber were all positive, showing wheals of 7, 5, and 5 mm, respectively. Serum specific IgE levels were 0.6 kU/L against pumpkin and 0.4 kU/L against melon of a total IgE level of 372 IU/mL. The PE and ME were analyzed by sodium dodecyl sulfate–polyacrylamide gel electrophoresis (SDS-PAGE) as described by Laemmli,³ showing protein bands ranging from 116 to 18 kDa for the PE (Fig 1A) and 66 to 17 kDa for the ME (Fig 1B). SDS-PAGE IgE immunoblotting assays were performed, and the results revealed IgE reactivity with a 18-kDa protein in the PE (Fig 1A) and with proteins of 66, 54, 52, 42, 35, 33, 20, and 17 kDa in the ME (Fig 1B). SDS-PAGE immunoblotting inhibition assays with ME as the inhibitor and PE in solid phase and with PE as the inhibitor and ME in solid phase did not show any IgE-binding inhibition (Fig 1A and B). Enzyme-linked immunosorbent assays were also performed with PE and ME in solid phase, using serum previously inhibited with ME and PE, respectively. No inhibition was observed in both assays. To identify the IgE-binding protein of the PE, the 18-kDa band was manually excised from the gel, digested with trypsin, and analyzed by matrix-assisted laser desorption/ionization time of flight and liquid chromatography coupled to tandem mass

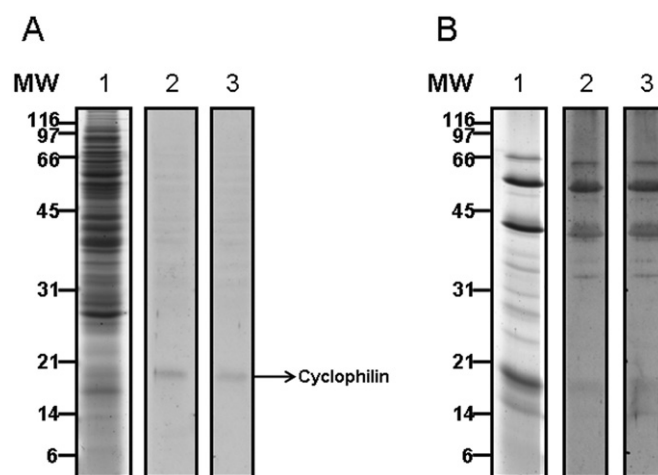


Figure 1. Identification of proteins in the pumpkin and melon extracts. A, Pumpkin. Lane 1, Sodium dodecyl sulfate–polyacrylamide gel electrophoresis (SDS-PAGE) after Coomassie blue staining with the pumpkin extract. Lane 2, SDS-PAGE IgE immunoblotting of the pumpkin extract. Lane 3, SDS-PAGE immunoblotting inhibition with melon extract as inhibitor and pumpkin extract in solid phase. B, Melon. Lane 1, SDS-PAGE after Coomassie blue staining with the melon extract. Lane 2, SDS-PAGE IgE immunoblotting of the melon extract. Lane 3, SDS-PAGE immunoblotting inhibition with pumpkin extract as inhibitor and melon extract in solid phase. MW indicates molecular weight marker.

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