

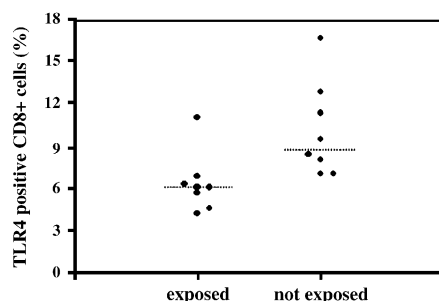


FIG 2. Percentage of TLR4⁺CD8⁺ cells in adenoids of children exposed to parental smoke compared with children not exposed. Medians are reported as horizontal lines. The statistical test used is the Mann-Whitney *U* test; *P* = .009.

the mechanisms involved either in the major susceptibility to upper airway infection or in the atopic risk observed in children exposed to ETS. Although obtained in a small number of subjects, these results suggest the crucial immunological function of TLRs in the upper airway.

Antonio Ricci, MD^a

M. Antonietta Avanzini, PhD^b

Cristina Scaramuzza, MD^a

Anna Maria Castellazzi, PhD^c

Massimo Marconi, PhD^c

Gian Luigi Marseglia, MD^c

^aDepartment of Pediatrics

Istituto di Ricerca a Carattere Scientifico Policlinico S. Matteo

P.le Golgi 19-27100 Pavia

Italy

^bResearch Laboratories

Istituto di Ricerca a Carattere Scientifico Policlinico S. Matteo

^cDepartment of Pediatrics

University of Pavia

Pavia, Italy

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Banana anaphylaxis with a negative commercial skin test

To the Editor:

The diagnosis of food allergy is becoming increasingly complex, but the skin prick test (SPT) is still at the center of the diagnostic work-up. Often the diagnosis of food allergy is based on skin tests alone. Fresh fruit, whether implicated in anaphylaxis or the oral allergy syndrome (OAS), poses many problems for traditional skin testing. As Ortolani et al¹ first reported in 1989, there is significant variability in the response to commercial extracts and fresh fruit applied by the prick-prick method. Other investigators have reported similar findings in food-induced anaphylaxis as well as OAS.² SPT has been compared with food challenge for the diagnosis of food allergy. In one study,³ only 58.8% of patients with a positive challenge had a positive commercial skin test result. However, 91.7% of patients with a positive challenge had a positive skin test result with fresh food.³

Because of the increasing association of banana allergy and latex allergy, the major allergens in banana have been characterized.⁴ Patients not allergic to latex have been confirmed to have anaphylaxis to banana.⁵ The major allergens are 32-kd and 34-kd proteins that have class I chitinase activity.⁴ More than half of patients allergic to banana respond to SPT performed with 1 of these 2 isolated proteins.⁴ Here, we report the case of a young boy with systemic symptoms and negative SPT result to commercial extract. This case is important because it is one of the first reported pediatric cases of systemic anaphylaxis to banana. Banana is generally thought to have low allergenic potential and is often appropriately introduced as part of the early infant diet. As food allergy becomes more prevalent, foods once believed not to cause systemic symptoms will increasingly require more in-depth clinical evaluation.

A 7-month-old boy presented to the allergy and immunology clinic at Duke University for evaluation of atopic dermatitis and possible food allergy. His atopic dermatitis started in the first several months of life. At 3 months of age, he had angioedema and erythema on his face and neck on his first introduction to a cow's milk formula. He was exclusively breast-fed until this point. At 5 to 6 months of age, he was started on solid foods. Approximately 2 hours after an isolated feeding of crushed banana, he awoke from a nap with generalized urticaria, vomiting, and respiratory symptoms, including wheezing. He was immediately taken to the emergency room for

appropriate treatment. He had been fed banana on one previous occasion, several weeks before this ingestion, without symptoms. He had no history of reaction to substances that cross-react with banana, including latex.

At the time of his initial evaluation in our clinic, he had a normal physical examination except for moderate atopic dermatitis. Initial SPT result with a commercial milk extract was positive. The SPT result with commercial banana extract (Greer Laboratories, Lenoir, NC) was negative, histamine control was 4+, and saline was not reactive. Because of his convincing history, a prick-prick test with fresh banana was performed, resulting in a 20-mm × 20-mm wheal. We used a Greerpick (Greer Laboratories, Lenoir, NC), first puncturing a peeled banana and then, with the same pick, placing the skin test in the usual fashion. Further testing included a CAP-FEIA (Pharmacia, Uppsala, Sweden) to banana, with a level of 4.70 kU/L. For banana, specific IgE levels predictive of clinical reactivity have not been established. However, 2 patients with food challenge–confirmed systemic symptoms or OAS had values of 5.21 kU/L and 5.24 kU/L.⁵ Our patient was sent home on a food-avoidance diet, with injectable epinephrine for use in case of an accidental ingestion. Despite occasional clinical cross-reactivity, we did not recommend avoidance of latex or other foods (kiwi, avocado, figs) or further testing.

This patient's case illustrates one of the pitfalls in the evaluation of fruit allergy. Commercial extracts for food and fruit testing are not standardized. Akkerdaas et al⁶ found significant variation in the protein concentration of 9 commercially available hazelnut extracts for SPT. One can infer that all nonstandardized extracts have similar, if not greater, variability. The use of prick-prick skin testing was used as part of a large study of melon allergy and found to have a sensitivity of 79% (54%-93%).⁷

Many clinicians are using SPT with fruit extracts as a method to diagnose food allergy. On the basis of food challenge data,⁸ the presumed negative predictive value of a nonreactive skin test is greater than 95%. Our case illustrates that fresh fruit–induced anaphylaxis may be missed if commercial extracts alone are relied on for diagnosis in the setting of a suggestive clinical history. Prick-prick testing with fresh fruit is a quick and inexpensive method to increase the diagnostic yield of SPT. Any patient with a convincing history and negative commercial extract SPT should have prick-prick testing. In addition, CAP-FEIA RAST testing and oral food challenge should be part of the complete evaluation.

David W. Hauswirth, MD

A. Wesley Burks, MD

Department of Pediatrics

Division of Allergy and Immunology

Duke University Medical Center

Box 2898

Durham, NC 27710

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Anaphylactic shock caused by a selective allergy to celecoxib, with no allergy to rofecoxib or sulfamethoxazole

To the Editor:

Coxibs are selective inhibitors of the COX-2 enzyme. They have been developed as powerful, nonsteroidal anti-inflammatory drugs with a good gastric tolerance. Very few hypersensitivity reactions have been described with the coxibs, particularly anaphylactic reactions.¹⁻⁴

A 39-year-old woman had been treated regularly with celecoxib for severe mechanical dorsalgia. The treatment lasted for almost 2 years, and then she stopped for 2 months. In November 2003, the patient took a 200-mg capsule. Thirty minutes later, she developed a severe, life-threatening anaphylactic reaction with urticaria, facial and tongue angioedema, bronchospasm, and malaise, followed by hypothermia. She recovered fully after intramuscular epinephrine and intravenous methylprednisolone and dexchlorpheniramine.

The patient had no other medical history, in particular no other drug allergy, apart from a typical latex work-related allergy, with rhinoconjunctivitis and asthma. She was a cleaner in a surgical clinic and had also experienced severe gastrointestinal pain with noncoxib nonsteroidal anti-inflammatory drugs. Skin prick test results to common aeroallergens were negative. Pulmonary function tests were normal. She was not taking any daily medication.

A skin prick test result to celecoxib (one 200-mg pill resuspended in 1 mL saline) was negative. To confirm the presence of anaphylaxis caused by celecoxib, an oral provocation test (OPT) was performed under medical surveillance (6 doses: 0.1, 1, 10, 25, 50, and 150 mg given at 30-minute intervals were planned). Precautions are