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 al[6] 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.

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Available online December 22, 2004.
doi:10.1016/j.jaci.2004.10.034

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 dorsalgy. 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

described elsewhere.[5] During the OPT, an anaphylaxis occurred at a cumulative dose of 36.1 mg celecoxib (actual doses administered were 0.1, 1, 10, and 25 mg over a 3-hour period) with generalized pruritus, rhinoconjunctivitis, and gastrointestinal pain, rapidly followed by tachycardia (heart rate, 148 bpm) and a drop of 65 mm Hg in systolic blood pressure. The shock subsided after 2 consecutive intramuscular injections of 0.25 mg epinephrine together with 100 mg intravenous methylprednisolone and 5 mg dexchlorpheniramine.

Six weeks later, a second OPT was performed with rofecoxib to find an alternative treatment to celecoxib. No symptoms occurred after the full therapeutic dose administration. Because of the sulfonamide structure of celecoxib,[3] we performed a third OPT with cotrimoxazole (sulfametoxazole + trimetoprime). There was no reaction.

Cases of anaphylaxis to celecoxib have been described[1-4,6,7] but appear to be rare. Indeed, a recent postmarketing surveillance study was undertaken in England,[6] and only 2 episodes of anaphylaxis have been reported in a cohort of 17,458 patients (0.01%). Drug responsibility was proven by OPT in 1 case only[2] and by clinical history in the other cases.[1,3,4,6] In another case,[7] a patient with 3 episodes of anaphylaxis to diclofenac underwent an in-hospital open challenge to rofecoxib. This patient experienced an isolated drop in blood pressure 40 minutes later, which was more likely a vasovagal reaction.[8] Allergic mechanisms were proven by using lymphocyte transformation tests in 2 cases,[2,4] but apparently not IgE-mediated, as demonstrated by negative skin tests results[2,4] (including our case) and ELISA.[1] Cross-reactions between sulfonamides and the sulfur moiety of celecoxib have been suggested in 1 case[4] but this seems to be an independent and coincidental sensitization to the 2 drugs. Our case report excludes cross-reactions between celecoxib and sulfamethoxazole and also between celecoxib and rofecoxib. Therefore, it underlines the importance of a thorough drug allergy work-up, allowing the reintroduction of other coxibs.

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Available online December 22, 2004.
doi:10.1016/j.jaci.2004.10.045

IgE-mediated asthma associated with a unique allergen from Angelim pedra (*Hymenolobium petraeum*) wood

To the Editor:

Angelim pedra *(Hymenolobium petraeum)* is a tree found in the Amazon basin belonging to the Fabaceae family and is harvested for its timber. It can grow as tall as 60 m, and its wood is used in construction, furniture, and carpentry.[1]

Exposure to sawdust from a variety of woods has been shown to cause occupational asthma and contact urticaria, according to published scientific reports.[2,3]

The following is a description of a case of occupational asthma caused by Angelim pedra in a 38-year-old male carpenter who was asymptomatic when harvesting Angelim pedra wood in Brazil for 20 years. He has lived in Spain since 1998. In 2000, he began to have cough, rhinorrhea, wheezing, dyspnea, and ocular itching within a few minutes of each new exposure on initating new work with this imported wood in Spain in 1999. He reported no symptoms with other woods.

Skin prick tests were performed by using the extracts of a variety of wood sawdust (iroko, mansonia, oak, cedar, pine, teak, obeche), which were extracted (10% wt/vol) in PBS and shaken for 24 hours at room temperature. The suspension was centrifuged at 8000 rpm for 30 minutes, the pellet was discarded, and the remaining solution was filtered through a 0.22-μm pore size membrane. All skin tests with extracts of various kinds of woods were tested in 3 atopic and nonatopic unexposed carpenters.

Nonspecific bronchial challenge testing with methacholine was performed 24 hours before bronchial challenge with Angelim pedra wood. Bronchial challenge with Angelim pedra sawdust was performed in a 3-m^3 challenge chamber. The wood dust aerosol was generated by asking the patients to tip the wood dust from 1 tray to another for increasing periods (1, 5, 15 minutes) as previously described.[4] The concentrations of total dust particles during the challenges were measured. A maximum concentration of 2.5 mg/m^3 was not exceeded. The patients wore protective clothing and vinyl gloves during the challenge. On a control day, the subject was challenged with formaldehyde to ensure that fluctuations of FEV_1 were <10% and to exclude a sensitization to formaldehyde. Bronchial challenge with formaldehyde