

Table 1. Frequency of positivity (%) for the 11 foods tested in the patients: only Bet v 1+ (n=20), only Bet v 2+ (n=4), or both Bet v 1+ and Bet v 2+ (n°=6)

| Food        | Bet v 1+ | Bet v 2+ | Bet v 1+/v 2+ |
|-------------|----------|----------|---------------|
| Apple       | 90       | 100      | 100           |
| Peanut      | 35       | 100      | 100           |
| Carrot      | 40       | 100      | 100           |
| Tomato      | 15       | 75       | 100           |
| Potato      | 10       | 75       | 100           |
| Celery      | 35       | 75       | 83            |
| Hazel nut   | 45       | 75       | 67            |
| Orange      | 15       | 50       | 83            |
| Cucumber    | 10       | 50       | 50            |
| Kiwi        | 5        | 50       | 33            |
| Turkey meat | 5        | 25       | 17            |

alone, by 50% (2/4) positive for Bet v 2 alone, and by 50% for both recombinant allergens (3/6), but by none of the three negative patients. The univariate analysis by logistic regression indicated that six foods (apple, hazelnut, tomato, potato, peanut, orange) were significant predictive factors ( $P<0.05$ ) for the appearance of the 'oral allergy syndrome'. However, according to the Bonferroni correction only the  $P$ -value reached with apple should be considered significant ( $P=0.006$ ). The relative risk of developing 'oral allergy syndrome' was calculated by considering two threshold values of serum IgE levels (negative =  $<0.35$  kU/l and positive  $>3.5$  kU/l) against apple. This risk was estimated to be 1.44 in patients negative for apple, compared to that of apple-positive patients, and increased up to 1.99 in patients with IgE levels above 3.5 kU/l for apple.

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## Anaphylaxis to coconut

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**Key words:** allergic reaction; anaphylaxis; coconut; walnut.

• ALLERGIC reactions to coconut (*Cocos nucifera*) are very infrequent. To the best of our knowledge allergy to coconut has been reported in two patients allergic to walnut and almond (1).

A 28-year-old atopic man presented an immediate reaction after ingestion of a coconut ice-cream. The reaction consisted of oral pruritus, vomiting, and intense angioedema of the lips and uvula, which subsided in 24 h with symptomatic treatment in the emergency room. He had previously noticed oral pruritus with walnuts, but tolerated the ingestion of other nuts. He denied adverse reactions with other foodstuffs, and tolerated contact with latex materials.

The patient gave written consent to the following study.

### Prick tests with coconut ice-cream, fresh

A rare case of IgE-mediated food allergy. coconut were positive (wheal diameters of 9 and 13 mm, respectively). Specific immunoglobulin E (IgE) to coconut (CAP System, Pharmacia) was 18.30 kU/l. Total IgE was 572 kU/l.

Defatted homogenized coconut was extracted in 0.9% saline. After filtration and centrifugation, the supernatant was dialyzed, and finally passed through a 0.45 µm Millipore filter. The protein concentration was 3.6 mg/ml. A prick test performed with this extract elicited a strongly positive immediate reaction (20 mm) with an associated late response at 6 h (30 mm). This test was negative in five atopic controls.

The SDS-PAGE of coconut extract (Fig. 1; panel A) showed bands for molecular weights of 15, 20, 35, 45 and 200 kDa. In the IgE immunoblot intense reactivity to a protein of 78 kDa was observed, as well as weaker bands for 15–20, 22 and 30 kDa (Fig. 1; panel B, lanes 1 and 2). Bands of 20, 35 and 200 kDa could not be considered specific because they also appeared with the control serum (Fig. 1; panel B, lanes 3 and 4).

## References

- JOHANSSON SGO, O'B HOURIHANE J, BOUSQUET J, BRUIJNZEEL-KOOMEN C, DREBORG S, HAAHTELA T, KOWALSKI ML, MYGIND N, RING J, VAN CAUWENBERGE P, VAN HAGE-HAMSTEN M, WUTHRICH B. A revised nomenclature for allergy. An EAACI position statement from the EAACI nomenclature task force. *Allergy* 2000;**56**:813–824.
- VAN REE R, FERNANDEZ RIVAS M, CUEVAS M, VAN WIJNGAARDEN M, AALBERSE RC. Pollen related allergy to peach and apple: an important role for profilin. *J Allergy Clin Immunol* 1995 March;**95**:726–734.
- ROSSI RE, MONASTEROLO G, OPERTI D, CORSI M. Evaluation of recombinant allergens Bet v 1 and Bet v 2 (profilin) by Pharmacia CAP system in patients with pollen related allergy to birch and apple. *Allergy* 1996 December;**51**:940–945.
- FRITSCH R, BOHLE B, VOLLMANN U, WIEDERMANN U, JAHN SCHMID B, KREBITZ M, BREITENEDER H, KRAFT D, EBNER C. Bet v 1 the major birch pollen allergen and Mal d 1, the major apple allergen, cross react at the level of allergen specific T helper cells. *J Allergy Clin Immunol* 1998;**102**:679–686.

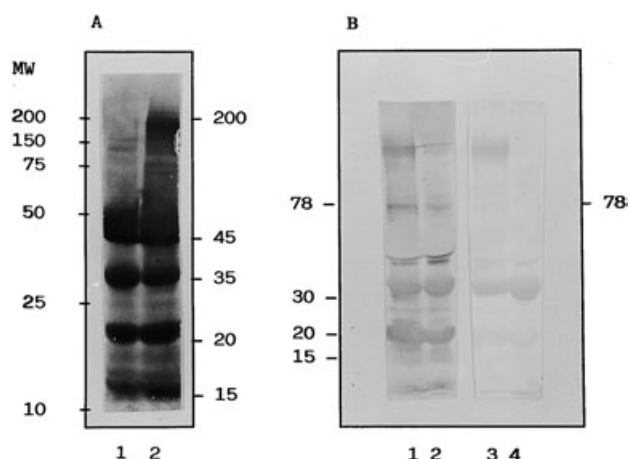


Figure 1. A) Coconut SDS-PAGE, without (lane 1) or with (lane 2)  $\beta_2$ -mercaptoethanol. B) Coconut IgE immunoblotting: lanes 1 and 3 reduced, lanes 2 and 4 unreduced; lanes 1 and 2 patient serum, lanes 3 and 4 control.

Allergy to walnut was ruled out by prick test and CAP. An open oral challenge with walnut was negative. Skin prick test and CAP to latex were both negative.

In conclusion, we present an exceptional case of anaphylaxis induced by the ingestion of a coconut ice-cream due to an IgE-mediated reaction to coconut, which was demonstrated by both *in vivo* skin tests and *in vitro* tests. In the two previously reported cases of combined allergy to nuts and coconut the main allergens involved had molecular weights of 35–36 kDa (1). A different allergen pattern was identified in our patient probably because he was monosensitized to coconut.

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#### Reference

1. TEUBER SS, PETERSON WR. Systemic allergic reaction to coconut (*Cocos nucifera*) in 2 subjects with tree nut and demonstration of cross-reactivity to legumin-like seed store coconut and walnut food allergens. *J Allergy Clin Immunol* 1999;103:1180–1185.

## Unusual NSAID hypersensitivity

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**Key words:** aspirin; conjunctivitis; NSAID intolerance.

• THE appearance of conjunctivitis as the sole manifestation of an adverse reaction to a drug administered by the oral route is an uncommon phenomenon (1). We report a case of conjunctivitis induced by non-steroidal anti-inflammatory drugs (NSAIDs). To the best of our knowledge, no similar selective ocular reactions have been previously reported. A non-atopic 53-year-old female reported several episodes of ocular pruritus, eye redness, and lacrimation, always affecting her left eye. All the episodes started in the first 2 h following the intake of NSAIDs, such as aspirin, metamizol, ibuprofen, and dextetoprofen, and resolved spontaneously after 2–6 h. She denied involvement of the right eye, as well as rhinitis, bronchial or skin symptoms. A complete ophthalmologic examination, including slit lamp biomicroscopy and conjunctival impression cytology (2) of both eyes, was normal in basal conditions.

### Isolated conjunctivitis after oral intake.

The patient gave written informed consent to the reintroduction of NSAIDs in order to study her reported adverse reactions. All drugs were given following a single-blind placebo-controlled procedure. Two hours after the administration of aspirin 125 mg she presented pruritus, hyperemia of the conjunctiva and lacrimation of her left eye. An impression cytology taken at that moment revealed an important neutrophil infiltrate. No reaction was observed (ophthalmologic examination, impression cytology) of the right eye. The reaction subsided spontaneously in 4 h, and the infiltrate in the impression cytology also disappeared. Two weeks later the patient received diclofenac, an NSAID she had never taken before, and 2 h after the intake of 25 mg she presented a left eye conjunctivitis with a neutrophil infiltrate. Two hours after the application of fluorometolone 1 mg/ml eye-drops the inflammatory response of the left eye subsided and the infiltrate in the impression cytology disappeared. Skin, nasal or bronchial involvement were absent in these two positive challenges, and no significant changes of basal  $FEV_1$  were observed. Diclofenac eye-drops (1 mg/ml) were applied to both eyes. Thirty minutes later conjunctival hyperemia and lacrimation of the left eye appeared, but no reaction was observed in the right eye. In contrast, paracetamol (up to 1000 mg) and nimesulide (up to 300 mg) were well tolerated.

In an attempt to prevent the NSAID-induced inflammatory response of the left eye, several pre-medication strategies were assayed. Disodium cromoglycate (40 mg/ml), sodium nedocromil (20 mg/ml), levocabastine (0.5 mg/ml) and fluorometolone (1 mg/ml) eye-drops were given separately at therapeutic doses for 2 days prior to the administration of aspirin. No protective effect was observed with any of them, although the eliciting dose of aspirin increased to 250 mg after the pre-medication with fluorometolone.

In conclusion, we present an exceptional case of isolated left eye conjunctivitis after the oral (and local) administration of NSAIDs, in which a local idiosyncratic reaction to inhibition of the cyclo-oxygenase (COX) pathway seems to be involved (3). The inflammatory response induced by NSAIDs was mild, transient, and self-