

Positive skin prick test reactivity to milk was detected in 7/114 (6%) infants. A total of 15/114 (13%) infants had challenge-proven cow's milk allergy. The mean age at diagnosis of CMA was 7.2 (5.9-8.5) months. The mean duration of breastfeeding was exclusively 3.3 (3.0-3.6) and overall 7.7 (7.1-8.3) months and was comparable in infants with and without positive SPT or challenge.

The mean energy intake of the mothers was 8.0 (7.7-8.3), MJ/day. Maternal intake of cow's milk protein ranged from 1.9 to 63.1 g/day, mean 26.2 (24.0-28.3) g/day and, adjusted for energy intake, 3.3 (3.0-3.5) g/MJ/day. The mean proportion of energy derived from fat was 36.6 (35.6-37.6) E% and from saturated fatty acids 15.8 (15.2-16.3), E%. Maternal intake of cow's milk protein was not associated with SPT reaction to milk or CMA in the infant (Table 1). In contrast, high intakes of total fat and saturated fatty acids were associated with sensitization to milk.

Our result indicates that sensitization to food allergens may not depend directly on the absolute amount of allergenic proteins in the diet, but rather on a complexity of dietary determinants. The Western diet typically contains an abundance of fat, especially saturated fatty acids, and the prevalence of allergic disease is highest in

Western societies. It would appear that breastfeeding mothers should perhaps reduce their fat intake rather than eliminate cow's milk from their diet to prevent allergy in the infant.

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Table 1. Maternal dietary intake during breastfeeding associated with cow's milk allergy and sensitization to milk in the infant

| Maternal intake            | CMA +    | CMA -    | p-value * | SPT milk + | SPT milk - | p-value * |
|----------------------------|----------|----------|-----------|------------|------------|-----------|
| Milk protein (g/MJ)        |          |          |           |            |            |           |
| Low (<3.3)<br>n = 57       | 9 (16%)  | 48 (84%) | 0.58      | 3 (5%)     | 54 (95%)   | 0.72      |
| High (= 3.3)<br>n = 57     | 6 (11%)  | 51 (89%) |           | 4 (7%)     | 53 (93%)   |           |
| Total fat (E%)             |          |          |           |            |            |           |
| Low (<36.6)<br>n = 59      | 6 (10%)  | 53 (90%) | 0.41      | 0 (0%)     | 59 (100%)  | 0.005     |
| High (= 36.6)<br>n = 55    | 9 (16%)  | 46 (84%) |           | 7 (13%)    | 48 (87%)   |           |
| Saturated fatty acids (E%) |          |          |           |            |            |           |
| Low (<15.8)<br>n = 56      | 4 (7%)   | 52 (93%) | 0.09      | 0 (0%)     | 56 (100%)  | 0.01      |
| High (= 15.8)<br>n = 58    | 11 (19%) | 47 (81%) |           | 7 (12%)    | 51 (88%)   |           |

\*. Categories defined on basis of mean values, Fisher's exact test  
CMA+, child with cow's milk allergy; CMA -, child without cow's milk allergy  
SPT milk +, positive skin prick test reaction to milk; SPT milk -, no skin prick test reaction to milk

## Allergenic similarities of 2S albumins

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**Key words:** 2S albumins; anaphylaxis; cross-reactivity; mustard; sunflower seed.

● A 54-YEAR-OLD man with a history of more than 10 years of pellitory allergy experienced an anaphylactic episode 15 min after the ingestion of a piece of bread 'baked with five cereals' purchased at a supermarket. Skin prick tests (SPT) with commercial extracts of cereals (wheat, rye, barley, rice, oats) and soybean (Dome-Hollister/Stier 1:10 w/v), and with both the crust and crumb of the offending bread roll were negative. In contrast, some fragments included in the crust of the roll induced a strong skin reaction (wheal diameter 10 mm); these turned out to be sunflower seeds (not declared among the ingredients of the roll). The patient did not recall any sunflower seed intake before, but had bred parrots and reported that their food contained sunflower seeds. In view of possible cross-reactivity between the major allergen of sunflower seed, a 2S albumin (1,2), and similar proteins in other vegetable foods (3-7), SPT were performed with extracts of peanut, walnut, Brazil nut (DHS, 1:10 w/v), sesame (Stallergenes 1:3 w/v), and with a commercial mustard (Louit Freres brand; Dijon type). The patient reacted to the mustard only.

Sunflower seeds (raw and roasted) and mustard seeds were homogenized and extracted (5%) in 0.1 M phosphate-buffered saline (PBS; pH 7.4), and shaken overnight at 4°C. Extracts showed final protein concentrations of 3.4, 2.3, and 0.9 mg/ml, respectively. Immunoblot analysis of sunflower seed extract was carried out by conventional methods. To investigate the cross-reactivity between allergens of sunflower seed and mustard, 0.3 ml of the patient's serum was absorbed with 0.8 ml of mustard extract for 3 h at 25°C before immunoblotting.

**Sunflower seed-induced anaphylaxis with cross-reactivity to mustard.**

Results are shown in Fig. 1. Raw sunflower seed (lane 1) and roasted sunflower seed (lane 2) showed a very similar SDS-PAGE profile. The patient's serum showed IgE reactivity to at least three distinct protein bands at about 13–15 kDa and 43–67 kDa in roasted sunflower extract (lane 3). Pre-absorption of serum with mustard extract caused a total loss of IgE reactivity at low molecular weights, but no change of the 43–67 kDa reactivity (lane 4). The immunoblot profile resembled that found by Kelly et al. (1,2). The low molecular weight allergens (13–15 kDa) probably correspond to the 2S albumin fraction. In sunflower seed, this fraction is made up of different albumins, one of which (SFA-8/SSA) is particularly rich in methionine and cysteine (8) and is an IgE-binding protein (1,2). The immature and mature (cleaved) forms of this protein show a molecular mass of 16–17 kDa and 12 kDa, respectively (1). Our inhibition experiments demonstrated complete cross-reactivity between sunflower seed (*Compositae*) and mustard (*Brassicaceae*) 2S-albumins. No cross-reactivity between allergens from these families has been described before. SPT with mustard should be always carried out in sunflower seed-allergic patients. The

remaining IgE-binding protein at 43–67 kDa has not yet been characterized and is likely to be a sunflower seed-specific allergen.

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## Interleukines in nasal lavage

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**Key words:** allergen-specific provocation; interleukin-5; seasonal allergic rhinitis; tryptase.

• THE biological markers of allergic inflammation are characteristic of different phases of the IgE-mediated reaction. In the present study levels of tryptase (early phase) and of interleukin-5 (IL-5) (late phase) were measured in nasal lavage fluid (NLF) from children with intermittent, seasonal allergic rhinitis (SAR). Non-allergic children served as a control group.

Tryptase (RIA Pharmacia Tryptase RIAct) and IL-5 (ELISA R & D Systems) levels in the NLF were measured in 15 allergic and 15 non-allergic children. The mean ages were 13.0 years (range: 8–16) and 12.6 years (range: 6–15), respectively.

**Increased levels of tryptase and IL-5 after specific allergen provocation.**

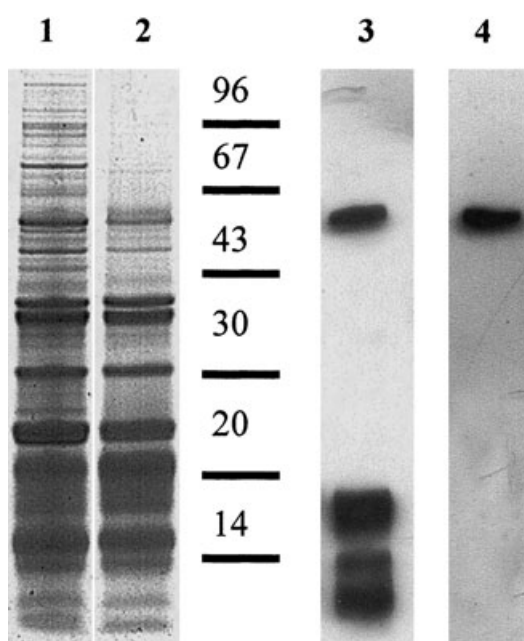


Figure 1. SDS-PAGE profile of raw (lane 1) and roasted (lane 2) sunflower seed extracts; lane 3 shows patient's IgE reactivity to different proteins in roasted sunflower seed extract; lane 4 shows IgE reactivity to roasted sunflower seed after absorption of serum with mustard seed extract.