

inhalation challenge by pouring sunflower seeds (200 g) for 5 min provoked marked symptoms of rhinoconjunctivitis associated with a 39% fall in FEV₁ (Fig. 1). On the next day, exposure to flour for 75 min resulted in a 42% fall in FEV₁ (data not shown).

Skin prick testing (SPT) with a battery of 10 common inhalant allergens revealed a positive reaction to *Dermatophagoides pteronyssinus* and *D. farinae*. SPT with extracts of wheat, barley, rye, and oats were negative, while α -amylase elicited a 4-mm response (histamine control: 4 mm) (ALK, Zaventem, Belgium). SPT were performed with an extract of sunflower-seed dust prepared by stirring sunflower seeds with phosphate-buffered saline (20% w/v) for 10 min at room temperature. SPT with a 1/10 dilution of this extract elicited a 10-mm wheal reaction. SPT gave negative results with pollen of sunflower and pollen of other Compositae. Specific IgE antibodies were assessed by the CAP method (Pharmacia CAP System, Uppsala, Sweden). The subject's serum exhibited a significant level of specific IgE against sunflower seeds (9 IU/ml), while specific IgE against sunflower pollen was not detected.

Inhalation challenges and immunologic tests demonstrated that sunflower-seed dust was the agent that caused rhinoconjunctivitis and asthma at work in our subject. The persistence of work-related respiratory symptoms despite avoidance of exposure to sunflower seeds probably resulted from sensitization to α -amylase. To the best of our knowledge, this is the first account of occupational asthma and rhinitis caused by sunflower seeds. This observation further confirms that sensitization to sunflower seeds can result from inhalation of seed dust, as already suggested by Axelsson et al., who found an association between food allergy to sunflower seeds and exposure to pet birds (2).

The results of SPT and assessment of specific IgE in our patient suggest that he could be sensitized to allergens that are present in sunflower seeds, but not in pollen grains. Such a discordance between the results of immunologic tests for pollen and seeds has been reported in two patients (4, 5). Immunologic cross-reactivity between sunflower pollen and seeds should be further investigated.

This report shows that sunflower-seed dust should be considered a potential cause of respiratory allergy among exposed workers. Further investigations are needed to investigate the prevalence of hypersensitivity reactions among subjects processing or ingesting sunflower seeds.

Key words: asthma; bronchial provocation tests; occupational diseases; sunflower.

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Is walnut really a birch-pollen-related fruit?

PATIENTS with birch pollinosis frequently develop adverse reactions to fruits and vegetables due to cross-reactivity between pollen and food allergens (1, 2). Several clusters of food hypersensitivity have been identified in patients with birch-pollen allergy, one of the most important being nut (3-5); in effect, recent studies have shown common allergenic structures in hazel pollen and hazelnuts (6). In clinical practice, patients with birch pollinosis and oral allergy syndrome (OAS) often include walnuts among causative foods. However,

A study of 36 birch-pollen-allergic patients failed to show any relation to walnut.

skin prick tests (SPT) with commercial walnut extracts are almost invariably negative in these subjects: in a recent study carried out at this allergy center

(7), only 1/149 birch-pollen-hypersensitive patients with OAS after the ingestion of nuts was positive on SPT with a commercial extract of walnuts; notably, all these patients were clearly positive on SPT with fresh hazelnut. The blame for the poor sensitivity of commercial extracts of vegetable foods has been generally laid on allergen instability,

as in the case of apple or other Rosaceae (8). However, such an explanation may be too simplistic, since the sensitivity of SPT with commercial extracts of other nuts, such as almond, hazelnut, or peanut, in patients with birch-pollen allergy is quite high. This study focused on the relationship between walnut allergy and birch pollinosis.

The study was performed on 36 birch-pollen-hypersensitive adult patients (M/F 12/24) reporting OAS after the ingestion of nuts. All underwent SPT with fresh hazelnut and walnut by the prick-prick technique (8) and SPT with two commercial extracts of both walnut and hazelnut (Stallergènes, France, and Allergopharma, Germany, for hazelnut; Dome/Hollister-Stier, USA, and Allergopharma for walnut). All SPT were performed on the volar side of the forearm with standard disposable lancets (1-mm tip; DHS) (8); results were assessed by comparison with the wheal induced by histamine hydrochloride 10 mg/ml. Only wheals equal to or greater than 2+ (i.e., half the average of the histamine wheal) were considered positive. Moreover, sera from 10 randomly selected patients underwent detection of IgE specific for hazelnut and walnut by the immunoenzymatic CAP System (Pharmacia, Sweden).

No patient was positive on SPT with fresh walnut, and only two patients showed weak (++) skin reactivities with both commercial extracts. In contrast, all patients were positive on SPT with fresh hazelnut; skin reactivity was moderate or strong (i.e. >++) in 35/36 patients. Thirty-two (89%) and 26 (72%) patients were positive on SPT with commercial hazelnut extracts by Stallergènes and Allergopharma, respectively; however, wheals induced by commercial extracts were weaker than those induced by fresh hazelnut in all cases. IgE specific for hazelnut was detected in 9/10 sera (class 2 in five cases; class 3 in four cases), whereas no serum was positive for walnut-specific IgE.

It is well known that cross-reactivity between birch pollen and vegetable foods is mostly caused by labile allergens (8–10), and this is the main reason that commercial food extracts for SPT are generally less reliable than fresh material for diagnostic purposes (8). This was clearly the case for hazelnut sensitivity in this study. By contrast, findings with walnut were rather surprising. At first glance, the poor diagnostic sensitivity of both SPT with commercial walnut extracts and *in vitro* methods might be considered the consequence of allergen loss during extraction procedures (8).

However, unexpectedly, patients did not show any reactivity on SPT with fresh walnut either. On the basis of these findings, if walnut really were a birch-pollen-related food, the only way left definitely to diagnose walnut hypersensitivity in these patients would be the double-blind, placebo-controlled, food challenge, a procedure which is extremely difficult to carry out in patients with OAS. However, another possible explanation is that walnut does not express any Bet v 1-like allergen; thus, it cannot be considered a birch-pollen-related food. The findings of previous studies of SPT with other vegetable foods (8, 11) seem to support this hypothesis.

Key words: birch pollen; CAP System; oral allergy syndrome; RAST; skin prick test; walnut.

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