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Accepted for publication 29 May 1998
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ALLERGY 1998;53:905-907

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Occupational asthma caused by sunflower-seed dust

SUNFLOWER (*Helianthus annuus*) belongs to the Botanic family of Compositae. Sunflower seeds are widely used in the production of margarine and cooking and salad oils, as well as livestock, bird, and poultry food. Anaphylactic reactions have been reported after ingestion of sunflower seeds (1, 2), sunflower oil (3), and honey containing sunflower pollen (4). Rhinitis and asthma have been documented in workers exposed to sunflower pollen (5, 6) and in people living in sunflower-growing areas (6). We herein present the case of a baker who developed rhinitis and asthmatic reactions when exposed to sunflower-seed dust at work.

A 31-year-old man had been working as a baker between 1981 and 1990 without experiencing respiratory symptoms. He had smoked 25 cigarettes a day since the age of 15 and reported a history of mild and transient asthma during childhood. In 1990, he was hired to work in a bakery where various "natural health" breads were produced, including bread flavored with sunflower seeds. About 3 months later, he developed rhinoconjunctivitis and asthma when exposed to dried sun-

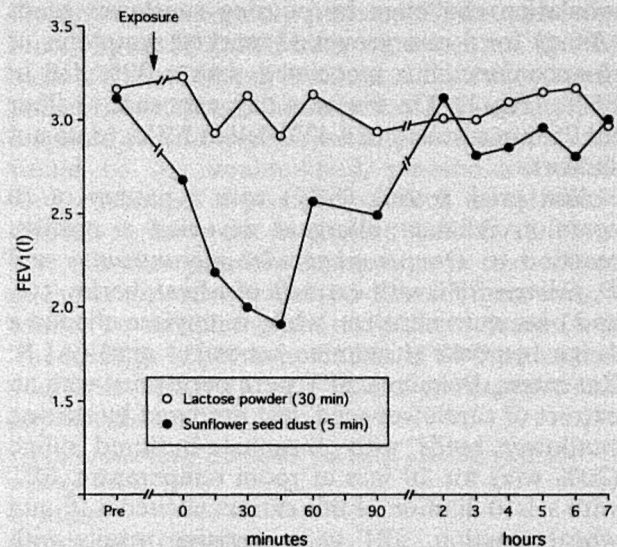


FIGURE 1 Results of inhalation challenges showing changes in FEV₁ after exposure to lactose powder for 30 min (open circles) and to sunflower-seed dust for 5 min (closed circles).

flower seeds, even when the seeds were handled only by his coworkers. The subject reported having experienced an anaphylactic reaction after eating about half a sunflower seed. He noticed that his respiratory symptoms worsened progressively and became less specifically related to exposure to sunflower seeds. In February 1995, the subject decided to work in another bakery where he was no longer exposed to sunflower seeds. He still experienced rhinitis and asthma at work, as well as nocturnal asthma attacks. In April 1996, he had to stop working, because he was experiencing severe asthma symptoms despite treatment with inhaled fluticasone propionate and salbutamol.

A baker developed rhinitis and asthma after workplace exposure to sunflower-seed dust.

Inhalation challenge tests were carried out in the laboratory 8 months after avoidance of workplace exposure. Baseline lung function testing showed an FEV₁ of 3.17 l (72% predicted value) with an FEV₁/forced vital capacity ratio of 62%. As a control challenge, the subject tipped lactose powder from one tray to another for 30 min without eliciting significant changes in FEV₁ (Fig. 1). At the end of this control day, the provocative concentration of histamine causing a 20% fall in FEV₁ was 0.18 mg/ml, reflecting marked nonspecific bronchial hyperresponsiveness. On the next morning,

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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Accepted for publication 3 June 1998

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ALLERGY 1998;53:907-908

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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,

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