

meadow fescue, timothy, meadow grass, *Penicillium* mix, *Candida*, cereal mix, elm, and plantain were found to be the predominant allergens. Grass pollens appear to be the most important allergen in seasonal rhinitis, whereas weeds were a more common cause of perennial rhinitis than grass pollens.

Our study has included the first skin prick test findings in children with respiratory diseases (asthma and/or rhinitis) in Ankara, Turkey. Atopy is a very important risk factor for respiratory allergic diseases. Therefore, skin tests may be useful to detect children with a risk of developing respiratory allergic diseases.

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References

1. International Study of Asthma and Allergies in Childhood (ISAAC) Steering Committee. Worldwide variation in prevalence of symptoms of asthma, allergic rhinoconjunctivitis, and atopic eczema: ISAAC. *Lancet* 1998;**351**:1225–1232.
2. Türktaş İ, Selçuk ZT, Kalyoncu AF. Prevalence of asthma and wheezing in Turkish children. *Eur Respir J* 1998;**12 Suppl 29**:52s.
3. Kalyoncu AF, Karakoca Y, Demir AU, et al. Prevalence of asthma and allergic diseases in Turkish university students in Ankara. *Allergol Immunopathol (Madr)* 1996;**24**:152–157.
4. Sly R. Changing prevalence of allergic rhinitis and asthma. *Ann Allergy Asthma Immunol* 1999;**82**:233–248.
5. Kalyoncu AF. Perennial and seasonal rhinitis in Ankara, Turkey. *Allergy* 1997;**52**:1040–1041.
6. Mungan D, Çelik G, Sin B, Baybek S, Demirel Y, Mısırlıgil Z. Characteristic features of cockroach hypersensitivity in Turkish asthmatic patients. *Allergy* 1998;**53**:870–873.

Occupational asthma to perfume

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Key words: challenge test; occupational asthma; perfume.

● PATIENTS with airway diseases often experience asthma-like symptoms upon contact with perfumes. Up to now, they were

mainly assumed to be due to the irritative effects of perfumes in patients with pre-existing unspecific bronchial hyperreactivity.

A 44-year-old woman worked in a drugstore from 1970 until 1996. Since 1988, she had noticed dyspnea while presenting customers with various perfume brands. In 1996, she experienced a severe asthma attack. Because of the respiratory problems, she could no longer work in her profession. Her case history revealed neither previous respiratory disorders nor an atopic disposition. She had smoked 10–15 cigarettes per day for 18 years.

Total serum IgE was 65 kU/l and within the normal range. Skin prick tests with 20 common environmental allergens revealed no type I sensitization. The epicutaneous tests with the European standard reagents panel indicated a delayed type-IV reaction to a mixture of odorous substances (HAL, Düsseldorf, Germany). The medical examination did not provide evidence for lung disorders. Lung-function tests showed a

moderate and reversible obstructive ventilation pattern (airway resistance R_{aw} =0.52 kPa*s/l; FEV₁%VC 62%; intrathoracic gas volume and residual volume were within normal ranges).

Workplace-related specific challenges with nine different brands of perfumes as well as with one individual perfume resulted in increased nasal airflow resistance and in significant bronchial obstruction (Fig. 1).

Cases of airway irritation by perfumes without clinical symptoms or occupational exposure have been described (1, 2), but the question of whether perfumes alone could cause the development of bronchial asthma has not been answered. There are contradictory opinions about the pathogenetic role of perfumes. Some authors could not find a correlation between bronchial hyperreactivity, as verified by methacholine, and perfume-related asthma (3, 4), while others (5, 6) reported a clear correlation between the methacholine response and the severity of airway obstruction caused by perfumes.

In this otherwise healthy woman, case history and clinical findings suggest obstructive airway disease as a result of occupational exposure to perfumes over a period of 26 years. The long-term exposure to perfumes in the workplace obviously initiated the manifestation of chronic asthma, which persisted even after the patient avoided further contact. The causative role of perfumes was confirmed by specific challenges when she showed immediate-type asthmatic reactions. However, it should be considered that perfumes are a complex mixture of natural and

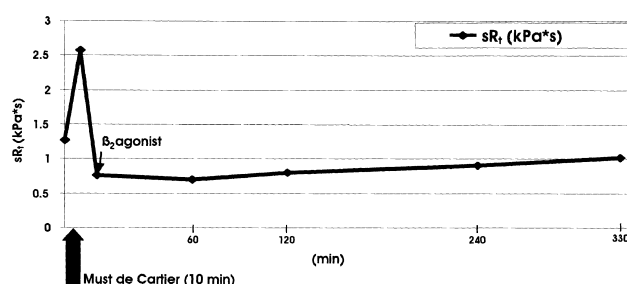


Figure 1. Inhalative challenge with perfume "Must de Cartier".

synthetic substances, including volatile oils, aldehydes, ethanol, and other ingredients, so that an exact identification of the asthma-triggering substance(s) cannot be made. Nevertheless, such hypersensitive reactions are relevant not only to workers' claims of compensation but also to prevention, because an increasing number of air-fresheners used in homes and stores may also contain perfumes, making primary prevention quite difficult.

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References

1. Bell IR, Schwartz GE, Peterson JM, Amend D. Self-reported illness from chemical odors in young adults without clinical syndromes or occupational exposures. *Arch Environ Health* 1993;**48**:6–13.
2. Eriksson NE, Löwhagen O, Nilsson JE, Norrlind K, Wihl JÅ. Flowers and other trigger factors in asthma and rhinitis – an inquiry study. *Allergy* 1987;**42**:374–381.
3. Balder B, Bäcker U, Löwhagen O. Clinical significance of bronchial hyperreactivity. Abstract no. 44 in Proceedings of XVth Nordic Congress in Allergology, 1984.
4. Millqvist E, Löwhagen O. Placebo-controlled challenges with perfume in patients with asthma-like symptoms. *Allergy* 1996;**51**:434–439.
5. Kumar P, Caradonna-Graham VM, Gupta S, Cai X, Rao PN, Thompson J. Inhalation challenge effects of perfume scent strips in patients with asthma. *Ann Allergy Asthma Immunol* 1995;**75**:429–433.
6. Shim C, Williams MH. Effect of odors in asthma. *Am J Med* 1986;**80**:18–22.

Unusual egg allergy in an adult

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Key words: egg allergy; food allergy; heat lability.

● HEN'S egg is one of the most common causes of food-allergic reactions, particularly in children. Egg white, which is generally considered more allergenic than egg yolk, contains over 20 proteins, four of which, namely, ovomucoid (Gal d 1), ovalbumin (Gal d 2), ovotransferrin (Gal d 3), and lysozyme (Gal d 4), have been identified as major egg allergens (1–3). Allergic patients react almost invariably to both raw and cooked egg products, and it has been shown that egg white retains its IgE-binding properties upon heat treatment (4). We report an unusual case of IgE-mediated egg allergy in an adult.

A 47-year-old man presented at this allergy center because of a 10-year history of immediate, severe oral and throat itching and gastric pain after the ingestion of various foods containing raw or underdone eggs. The offending foods included fresh pastries, fresh cream, cream puffs, ice cream, and underdone omelettes. In contrast, the patient could eat hard-boiled eggs, well-done omelettes, and

pasta containing eggs. He did not report a familial history of atopic diseases, and never suffered from atopic eczema or respiratory allergy.

The patient did not show any reactivity in SPT with a large panel of airborne allergens (Allergopharma) present in this geographic area. SPT with a large panel of commercial food-allergen extracts (Dome-Hollister/Stier 1:20 w/v) including egg white, egg yolk, milk, meats, fish, crustaceans, and several fruits and vegetables were carried out. A strong skin reactivity to both egg white and egg yolk (3+ by comparison with histamine 10 mg/ml) was found.

In view of clinical findings, immunoblot analysis with a self-prepared extract of hen's egg white was carried out. Egg white was homogenized and extracted 10% w/v by overnight incubation with 0.15 M phosphate-buffered saline (PBS), pH 7.2. Particulate material was removed by centrifugation at 18 000 g for 30 min; the supernatant was exhaustively dialyzed against PBS. Final protein content (according to Bradford) was 3 mg/ml. Electrophoresis was carried out with a 10–20% polyacrylamide gradient gel. The

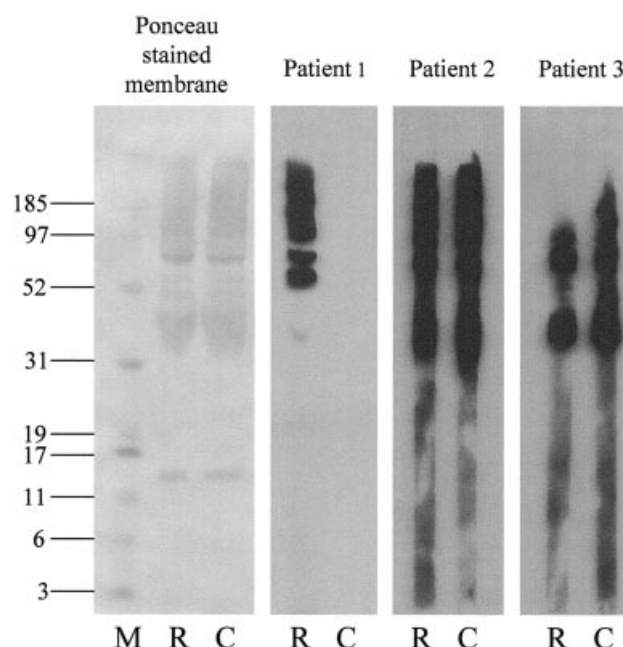


Figure 1. Immunoblot analysis: patient 1 showed IgE reactivity to proteins at 67, 78, and >100 kDa in raw egg-white extract, but no reactivity to cooked egg. Two egg-allergic controls (patients 2 and 3) showed no difference in IgE reactivity to raw or cooked egg white. R: raw; C: cooked.