

age difference of the patient populations, the most obvious difference is in the allergen preparations that were used. From a practical point of view, we chose to use the same aqueous allergen solutions for the SPT as well as the APT, whereas petrolatum-based allergen preparations were used in most recent adult studies. Based on current knowledge, these petrolatum-based preparations are preferable for further standardization of the APT (8). Despite this reservation, we believe the data described above add valuable information to our current knowledge of the APT with aeroallergens in children.

In conclusion, we found a substantial number of clear-cut positive APT reactions to three common aeroallergens, with a relatively high percentage of urticarial reactions, in our patient population of young children with AD. Although the APT seems promising as a diagnostic and possibly even prognostic skin test, its clinical value still appears limited at the moment (8, 16). Further studies aimed at standardization, reproducibility and clinical validation in children as well as adults are needed before the APT with aeroallergens can be used in routine daily clinical practice (8). For future use, we would like to prompt increased interest in the immediate type urticarial reactions in the APT, especially in children.

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Occupational asthma caused by chamomile

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Key words: *Asteraceae*; asthma; bronchoprovocation tests; chamomile; occupational disease.

Chamomile flower (*Matricaria chamomilla* L. or *recutita*

L., botanical family of *Asteraceae* or *Compositae*) is widely used as a medicinal agent because of its sedative and anti-inflammatory properties. Immunoglobulin E-mediated urticaria and anaphylaxis have been reported following topical application (1), ingestion of herbal tea (2), and administration of chamomile-containing enema (3). Herein, we describe a tea-packing plant worker who developed occupational asthma and rhinitis caused by inhalation exposure to chamomile dust.

A 43-year-old man developed work-related rhinoconjunctivitis and asthma

Inhalation of chamomile dust can cause IgE-mediated rhinitis and asthma among herbal tea workers.

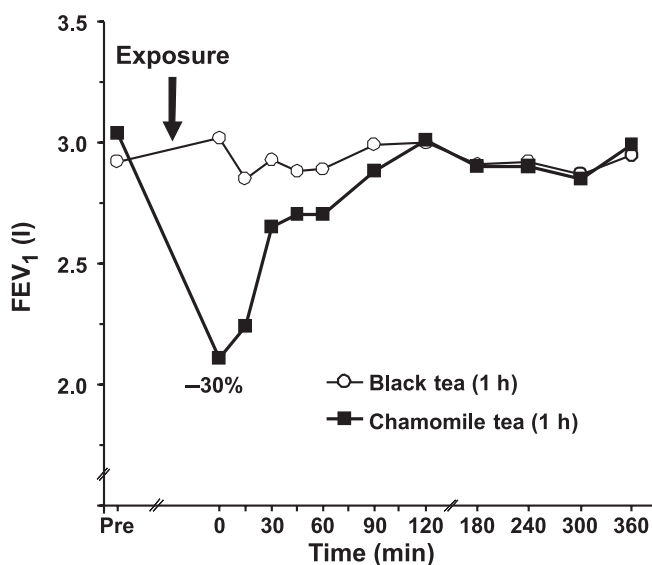


Figure 1. Results of specific inhalation challenges illustrating the changes in FEV₁ after exposure to black tea (open circles) and to chamomile tea (300 g) tipped for 60 min (closed circles).

symptoms 11 years after starting his employment as a maintenance worker in a tea-packing plant. The facility processed black tea (*Camellia sinensis*) as well as various herbal teas, including chamomile, lime (*Tilia cordata*), and dog rose. The subject noticed that his symptoms occurred electively when chamomile tea was packaged. He became completely symptom-free when the production of herbal teas was transferred to another factory.

Specific inhalation challenges were performed 8 months after complete cessation of exposure to herbal teas. These challenges were completed by tipping plant components from one tray to another for gradually increasing periods of time. Baseline spirometry showed a forced expiratory volume in 1 s (FEV₁) of 2.92 l (82% pred) and an FEV₁/forced vital capacity ratio of 75%. As a control challenge, the subject was exposed to black tea (300 g) for 1 h without inducing significant changes in FEV₁ (Fig. 1). At the end of this control day, histamine PC₂₀ was within the nonasthmatic range (20.0 mg/ml). The analysis of induced sputum revealed a total cell count of 0.56×10^6 cells/ml in the absence of eosinophils. On the next day, tipping chamomile tea (300 g) for 1 h resulted in a 30% fall in FEV₁ without any late bronchial response (Fig. 1). Six hours after the end of exposure, histamine PC₂₀ was measured at 8 mg/ml. The sputum

sample collected at that time showed a substantial increase in sputum eosinophils (7.5% of 3.36×10^6 cells/ml) as compared with control day assessment. On the following day, exposure to lime tea for 2 h did not elicit any functional change (histamine PC₂₀ value, 28 mg/ml). The subject denied having ever ingested herbal tea and did not agree to perform an oral challenge.

Skin prick tests were carried out using increasing concentrations of black tea, chamomile, and lime extracts prepared by stirring the commercial teas in sterile saline. Skin prick test with chamomile extract at 10 mg/ml elicited a 6-mm weal response while remaining negative with black tea and lime tea extracts. Skin prick tested with common allergens provided a positive response to mugwort (*Artemisia vulgaris*) and a mixture of grass pollen (Stallergènes, Antony, France). The subject's serum exhibited a significant level of specific IgE against the pollen of chamomile (3.12 IU/ml), mugwort (1.12 IU/ml), and timothy grass (1.53 IU/ml) using the ImmunoCAP method (Phadia Diagnostics, Uppsala, Sweden).

To the best of our knowledge, this is the first report of chamomile-induced asthma documented through specific inhalation challenges. The possibility of respiratory allergic reactions caused by chamomile has been previously reported in cosmeticians, who developed dermati-

tis and rhinitis, while preparing herbal beauty face masks containing chamomile and lime flowers (4). Patch, skin prick, and nasal provocation tests were positive to both chamomile and lime. High prevalence rates of sensitization to chamomile as well as other herbal plants have been documented among herbal tea workers, although these immunological findings did not correlate with work-related changes in lung function indices (5, 6).

This observation demonstrates that the inhalation of chamomile dust can induce IgE-mediated rhinitis and asthma among exposed workers. Appropriate workplace hygiene measures should be implemented in order to minimize airborne exposure to medicinal plants.

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Occupational rhinitis to sodium alendronate

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Key words: alendronate; allergy; bisphosphonate; occupational rhinitis; sensitization.

Sodium alendronate (SA) is a low-molecular-weight (LMW) compound inhibiting bone resorption, used to treat osteoporosis, recognized as an irritant of the respiratory system and skin.

A few cases of skin reaction after oral intake of bisphosphonate have already been reported (1, 2). To our knowledge, no report exists on occupational respiratory allergy caused by this compound.

A 40-year-old woman was referred to our department for suspected work-related rhinitis and asthma. Since 1995 she had been employed in a pharmaceutical company, assigned to the tablets packaging department of SA (about 1 week each month) and other drugs, and particularly during the 3 months before our observation she was employed exclusively on the SA line. The product line was enclosed, provided with local exhaust fans, and the subject wore a mask without a particle filter. One month after being moved exclusively to the SA line, she started to complain of nasal itching,

rhinorrhoea and dry cough. Symptoms appeared every Monday, 1 h after starting her work, improved during weekends and resolved during holidays. Symptoms were more severe during day shifts than during night shifts, when only less number of lines was involved in the production. No similar symptoms were reported by other employees.

The patient came to our observation 1 month after the symptoms' onset; she had worked until the day before our consultation.

The clinical history reported wheals after strawberry ingestion, palmo-plantar erythema after intake of an unspecified antibiotic, eczema after use of latex gloves and sneezing upon exposure to cats. She had never taken SA.

Lung function tests were normal, skin prick tests (SPTs) were positive to cat dander and grass pollens, serum IgE levels were 36.8 kU/l and peripheral eosinophilia was 4.3%.

Both SPTs and patch tests with SA in saline (1 : 100 and 1 : 10 w/v) were positive, and showed negative results when performed on 10 nonexposed subjects.

A specific Inhalation Challenge (SIC) was performed, out of grass pollens period, with occupational method (3). After a control day, exposure for 60 min to SA 10 mg dissolved in lactose provoked a significant increase in nasal symptoms' score and a significant decrease in peak nasal inspiratory flow (PNIF) at 15, 30 and 240 min, but no change in forced expiratory volume in 1 s (FEV₁). After SA exposure, provocative dose of methacholine producing a 20% fall in FEV₁ (PD₂₀ FEV₁) decreased from 1636 µg before challenge to 704 µg and induced sputum (IS) eosinophils increased from 1.2% to 7.9% (4).

The SIC repeated after premedication with oral levocetirizine induced no significant change in PNIF. SIC was also performed in a healthy control with no significant change.

A diagnosis of allergic occupational rhinitis (OR) caused by SA was made (5). During follow-up visit, the patient had ceased exposure to SA and was completely free of symptoms.

Among LMW-chemicals, several drugs are well-known to induce work-related respiratory allergy (6). To our

knowledge, this is the first case, described in the literature, of OR caused by SA. The patient's history, SPTs, patch tests and the SIC results fulfilled the criteria for the diagnosis of OR caused by SA (5). The presence of a latency period, the dual nasal response to SIC, the absence of symptoms in other exposed workers suggest an allergic mechanism.

Interestingly, sensitization occurred despite the enclosure of the production process and the use of a personal protective device (mask). It may be suggested that during the loading phase of tablets, some powder disperses into the environment and the mask proved inadequate. The observed decrease in PD₂₀ FEV₁ and increase in IS eosinophils may be related to lower airways' inflammation induced by the nasal response and/or may have represented an early, preclinical marker of occupational asthma (OA). The negative response to SIC after levocetirizine suggests that mast cells may play a role in the response.

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