

in nasal washings in higher concentrations than expected on the basis of simple diffusion. These differences in ratio of specific-to-total IgE were considered evidence for a model in which IgE is produced locally but predominantly diffuses into serum.¹⁰ Our findings showing the presence of IgE localized in the germinal centers of ABPA lung support the concept of local IgE production.

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REFERENCES

1. Rosenberg M, Patterson T, Mintzer R, Cooper B, Roberts M, Harris K. Clinical and immunologic criteria for the diagnosis of allergic bronchopulmonary aspergillosis. *Ann Intern Med* 1977;86:405-14.
2. Greenberger PA, Smith LV, Hsu CC, Roberts M, Liotta JL. Analysis of bronchoalveolar lavage in ABPA: divergent responses of antigen-specific antibodies and total IgA. *J ALLERGY CLIN IMMUNOL* 1988;82:164-70.
3. Filley WV, Holley KE, Kephart GM, Gleich GJ. Identification by immunofluorescence of eosinophil granule major basic protein in lung tissues of patients with bronchial asthma. *Lancet* 1982;2:11-6.
4. Peters MS, Schroeter AL, Kephart GM, Gleich GJ. Localization of eosinophil granule major basic protein in chronic urticaria. *J Invest Derm* 1983;81:39-43.
5. Adolphson CR, Gleich GJ, Yunginger JW. Standardization of allergens. In: Rose NR, Friedman H, Fahey JL, eds. *Manual of clinical laboratory immunology*, 3rd edition. Washington, DC: American Society for Microbiology, 1986:652-9.
6. Krenik KD, Kephart GM, Offord KP, Dunnette SL, Gleich GJ. Comparison of antifading agents used in immunofluorescence. *J Immunol Methods* 1989;117:91-7.
7. Slavin RG, Bedrossian CW, Hutcheson PS, et al. A pathologic study of allergic bronchopulmonary aspergillosis. *J ALLERGY CLIN IMMUNOL* 1988;81:718-25.
8. Golbert TM, Patterson R. Pulmonary allergic aspergillosis. *Ann Intern Med* 1970;72:395-403.
9. Slavin RG, Fischer VW, Levine EA, et al. A primate model of allergic bronchopulmonary aspergillosis. *Int Arch Allergy Appl Immunol* 1978;56:325-33.
10. Nakajima S, Gillespie DN, Gleich GJ. Differences between IgA and IgE as secretory proteins. *Clin Exp Immunol* 1975;21:306-17.

Rhinitis and asthma caused by occupational exposure to carob bean flour

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Vegetable gums, such as acacia, tragacanth, and guar, are known causes of occupational rhinitis and asthma. Carob bean flour is another substance derived from a vegetable that grows in the Mediterranean region. It has been used industrially since the beginning of the twentieth century as a thickening agent in food products (ice cream, margarine, preserved foods, pet foods), and it is used in infant feeding for the control of diarrhea and vomiting.¹

CASE REPORT

The patient was a 48-year-old man who had been employed for 16 years in a jam factory. After 2 years in his

job, he complained of work-related rhinitis and irritated eyes; asthma appeared 3 years later. Rhinitis occurred during his work, and he had asthma during the night after work; symptoms disappeared on weekends or on holidays. During the last years, asthma tended to become more chronic. His job consisted of handling thickeners and hardeners in dry form (powder): carob bean flour (Idealgum; Idea, Milano, Italy); a blend of guar gum, carob bean flour, and carrageenan (Frimulsion N 40, Cesalpinia, Bergame, Italy); and pectin (Genu, Copenhagen, Denmark). He had no personal or familial history of atopy. IgE level was 31 IU/ml; lung function testing showed an obstructive syndrome (forced expiratory volume in one second [FEV₁] 68%, forced vital capacity [FVC] 93%, total lung capacity [TLC] 95% of predicted), with a baseline peak expiratory flow rate (PEFR) value of 500 L/min. Skin prick tests were performed with Idealgum 1/1000, Frimulsion N 40 1/1000, Genu 1/1000, and pure guar gum 1/1000 (Viscogum 3000A, Sanofi Bio-Industrie, Brussels, Belgium), diluted in phosphate-buffered saline. All were negative. Skin prick tests were also negative in five control subjects. A single-blind provocation test was performed (manipulation of carob bean flour for 15 minutes)

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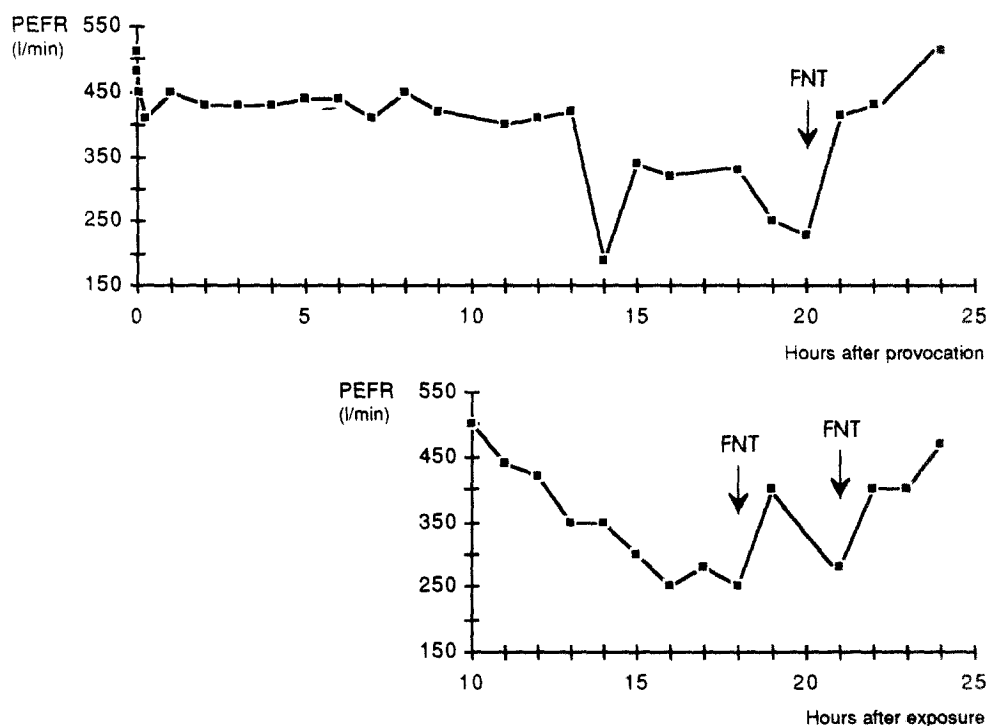


FIG. 1. PEFR after provocation (manipulation of carob bean flour for 15 minutes) (*top*) and after a typical work day (*bottom*): in both cases the patient had delayed asthma approximately 14 to 18 hours after exposure, and a second fall of PEFR some hours later, resolving after nebulization of fenoterol (FNT).

(Fig. 1). The patient had cough within 3 minutes and rhinitis with sneezing within 20 minutes, with a slight increase in nasal pressure: from 10 to 25 mm H₂O in left nostril and from 10 to 30 mm H₂O in right nostril (passive anterior rhinomanometry; Heyer-PAR, Carl GmbH, Bad Ems, Germany). Symptoms lasted for 3 hours, without significant changes in spirometric values (prechallenge: FEV₁ 72% of predicted, FVC 87%, PEFR 510 L/min; slight decrease 3 hours after manipulation: FEV₁ 57%, FVC 77%, PEFR 430 L/min). Fourteen hours after provocation the patient had wheezing on physical examination and a sizable decrease in PEFR (from 510 to 190 L/min), with spontaneous recovery, then a second fall 6 hours later (PEFR 230 L/min), resolving after nebulization of fenoterol. PEFR was followed during 5 consecutive days, after stopping work for 2 weeks: PEFR measurements ranged from 500 to 370 L/min, without symptoms of asthma. Finally, the patient was hospitalized for monitoring of PEFR after a typical work day: he reported rhinitis within a few minutes after exposure at work, and he had asthma approximately 18 hours after exposure, with wheezing and a very significant fall of PEFR (from 500 to 250 L/min), similar to that obtained after provocation (Fig. 1). PEFR was also followed in three control subjects during 24 hours after manipulation of carob bean flour for 15 minutes: no reaction was evidenced. RAST was performed to carob bean flour and guar gum (Pharmacia, Uppsala, Sweden); both results were positive, carob bean flour 0.3 to 0.4 kU/l and guar gum 0.7 to 0.8 kU/l (CAP system). The patient completely stopped handling

these substances and had no more asthma attacks. He received inhaled beclomethasone dipropionate 1000 µg/day. Pulmonary function tests performed 1 year later were normal.

DISCUSSION

Vegetable gums are known causes of IgE-mediated allergy. Some of them (guar gum, carob bean flour) are widely used in foods as thickening, hardening, stabilizing, suspending, or gelling agents. Guar gum (extracted from the seeds of a Leguminosae, *Cyamopsis tetragonolobus*, growing in India and Pakistan) has been shown to cause occupational allergic rhinitis and asthma, and obstructive sleep apnea syndrome in one exceptional case.²⁻⁴ Carob bean flour is extracted from another Leguminosae, *Ceratonia siliqua*, that grows in the Mediterranean region. This is the first reported case of occupational allergy caused by carob bean flour. Specific IgE to carob bean, but also to guar gum and tragacanth, have been found in the sera of some patients allergic to soy bean,⁵ suggesting some degree of cross-reactivity in the family Leguminosae. The molecular structures of carob bean and guar gum are very similar, consisting of a high molecular weight polysaccharide (a galactomannan, composed of galactan and mannan units, with a ratio of 1:4 for carob and 1:2 for guar, respectively). RAST

results suggest sensitization to both substances in this case, or a possible cross-reactivity between carob bean and guar gum. Specific IgE levels, though significant, were too low to perform a RAST-inhibition test. Carrageenans (galactose polymers, extracted from red seaweeds) and pectin (a glucuronic acid polymer, originating from fruits) were also handled by the patient, but nothing has shown that IgE-mediated mechanisms might be involved with these substances. Attempts to know whether they could induce specific IgE were unsuccessful (Pharmacia).

In summary, we describe the first case of occupational allergy to carob bean flour, with evidence of an IgE-mediated mechanism. The patient had immediate rhinitis and delayed asthma, which resolved after he stopped working.

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REFERENCES

1. Reynolds JEF. Stabilising and suspending agents. In: Martindale, ed. *The extra pharmacopoeia*. London: The Pharmaceutical Press, 1989:1432-8.
2. Kanerva L, Tupasela O, Jolanki R, Vaheri E, Estlander T, Keskinen H. Occupational allergic rhinitis from guar gum. *Clin Allergy* 1988;18:245-52.
3. Malo JL, Cartier A, L'Archevêque J, et al. Prevalence of occupational asthma and immunologic sensitization to guar gum among employees at a carpet-manufacturing plant. *J ALLERGY CLIN IMMUNOL* 1990;86:562-9.
4. Leznoff A, Haight JS, Hoffstein V. Reversible obstructive sleep apnea caused by occupational exposure to guar gum dust. *Am Rev Respir Dis* 1986;133:935-6.
5. Yman L, Rolfsen W, Malmheden-Iman I. Food additives from the legume family (*Leguminosae/Fabacae*). A potential allergy risk. *Allergy* 1988;43:81.

The true story of the pilot who was itching to fly

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The difficulty of finding a specific cause for chronic urticaria/angioedema has prompted some treating physicians to recommend multiple screening tests in addition to a comprehensive history and physical examination to identify an undetected infection or inflammation.¹ However, some controversy exists as to how many screening tests should be ordered in cases in which the history does not direct the physician toward a specific problem. Recommendations for screening tests range from routine laboratory tests (or none at all),^{2,3} to chest radiography, hemography, sedimentation rate measurement, serology, tests for antinuclear antibody, rheumatoid factor, and ova and

parasites in the stool, serum protein electrophoresis, and skin biopsy.⁴ We present a patient with chronic urticaria/angioedema whose ardent desire to return to work prompted us to pursue an exhaustive workup.

CASE REPORT

H.S. is a 28-year-old Air Force pilot with a 2-year history of progressively worsening chronic urticaria/angioedema whose symptoms and subsequent use of medications had prevented him from flying for more than 6 months. Initially his urticaria was episodic, lasting less than 7 days and responding rapidly to antihistamines. The frequency and duration of his symptoms gradually increased until he was having symptoms daily. Ultimately, his urticaria/angioedema could not be controlled, even with 200 mg hydroxyzine and 100 mg doxepin daily, or with several other combinations of high-dose H₁ and H₂ antihistamines. Prednisone was the only medication that controlled his symptoms. Before being seen at our medical center, he had an extensive workup including normal complement studies and tests for antinuclear antibody, rheumatoid factor, RPR hepatitis serology, thyroid functions, and thyroid antibodies. Skin testing for aeroallergens and foods and a trial of a salicylate-free diet all yielded negative results. On initial examination, he ap-

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