

Asthma caused by occupational exposure to pectin

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Pectin is a high-molecular-weight (20 to 400 kd) carbohydrate obtained from fruit rinds. It is used as a jelling agent in the jam and candy industries. Three cases of pectin-induced occupational asthma have been reported previously: one from Germany¹ and two from Canada.^{2,3} Both IgE- and IgG-mediated mechanisms have been suggested. In this report we present three cases to strengthen the evidence that occupational exposure to pectin may cause asthma and discuss potential mechanisms on the basis of our findings.

CASE REPORTS

History and symptoms

Patient 1. A 32-year-old man was a worker in the foodstuff industry. In 1981 he experienced nasal symptoms and dry cough when cooking plum jelly at work and was transferred to another workplace. After that, he was well until 1983, when pectin powder started to be used at his new workplace in the production of jelly sweets. He experienced symptoms of rhinitis and breathlessness when exposed to pectin at work and also during the night after, but he was free of symptoms when pectin was not used. Soon he experienced nasal and asthmatic symptoms when pectin containers were opened in his work area, although he was not handling pectin himself. He experienced similar symptoms when eating onion (which contains pectin). In April 1983 his spirometry showed obstructive airflow limitation. Diurnal peak expiratory flow (PEF) measurements showed falls in connection with pectin exposure, both immediately at work (from 600 to 400 to 450 L/min) and during the night after work (to 250 L/min). He was admitted to the Department of Pulmonary Medicine at the Turku University Central Hospital for further examination.

Patient 2. A 43-year-old man had worked since 1989 in his own company, selling berries and producing jam. He used pectin powder together with sugar in preparation of jam. Since 1992 he had experienced dry cough and nasal symptoms, and since 1993 he had also experienced dyspnea, which he linked to the handling of pectin powder. Bronchial asthma was diagnosed in 1993 at the local hospital on the basis of variability in the diurnal PEF values and a strong bronchial hyperresponsiveness in histamine challenge. He was admitted to the Finnish Institute of Occupational Health in Helsinki for further examination.

Abbreviations used

PEF: Peak expiratory flow
PRU: Phadebas RAST units

Patient 3. A 34-year-old woman had worked as a laboratory technician, analyzing sugars, since 1980. She had worked since 1992 for a company in which her main task was to analyze ingredients of jam sugar, including pectin powder. She had chronic rhinitis but was not atopic according to skin prick tests performed in 1986. Since 1993 her symptoms of rhinitis had become more severe, and she started to experience chest tightness at work. She connected her symptoms to dusts, especially pectin powder. She also had an erythematous rash on her hands when working with pectin. She was admitted to the Finnish Institute of Occupational Health in Helsinki for examination of asthma and rhinitis.

RESULTS

Descriptive characteristics of the patients and the main results are presented in Table I. Inhalation challenges were performed according to published guidelines.⁴

Pectin-specific IgE and IgG₄ antibodies were measured with ELISA⁵ in sera from patients 2 and 3 and two control subjects, but no antibodies were detected (Table I). For patient 1, Pharmacia (Uppsala, Sweden) measured specific IgE antibodies and found a positive value of 91 Phadebas RAST units (PRU)/ml for pectin and 1.85 PRU/ml for onion. RAST inhibition with pectin showed 99.7% inhibition for pectin IgE and 47% inhibition for onion.

DISCUSSION

We report three cases of asthma caused by occupational exposure to pectin powder. In our patients occupational sensitization was caused by an inhaled antigen. One of the patients also experienced asthmatic symptoms when eating onion, which contains pectin. This raises the question of the role of pectin in food allergies, for example, in citrus fruit and apple allergies.

Two patients (1 and 2) showed immediate responses in lung function measurements in pectin inhalation challenge. Both of them had positive skin test results for pectin, which indicates that an IgE-mediated hypersensitivity reaction is probably involved. Pectin-specific IgE

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TABLE 1. Descriptive characteristics of patients and main results

	Patient 1	Patient 2	Patient 3
Age/gender	32 yr/M	43 yr/M	34 yr/F
Profession	Worker in food-stuff industry	Producer of jam	Lab technician in sugar industry
Length of pectin exposure*	Not known	3 yr	1 yr
Smoking status	Current smoker	Lifelong nonsmoker	Lifelong nonsmoker
Family history of asthma	Two sisters & 1 brother had asthma	One sister had asthma as a child	No
Atopy†	House dust mite	No	Dermographism
Skin test with pectin	Positive‡	Positive§	Dermographism§
Total IgE	ND	107 kU/L	117 kU/L
Baseline FVC (% predicted)	6.10 L (112%)	5.97 L (121%)	3.98 L (94%)
Baseline FEV ₁ (% predicted)	4.95 L (114%)	4.78 L (120%)	3.26 L (91%)
Baseline FEV ₁ /FVC%	81	80	82
Nonspecific bronchial hyperresponsiveness	Mild	No	No
Pectin-specific IgE	91 PRU/ml	No	No
Pectin-specific IgG ₄	ND	No	No
Inhalation challenge tests with pectin¶			
Pectin + lactose FEV ₁	Immediate 45% fall; later not followed up	Immediate 25% fall; after 8 hr, 16% fall	No immediate fall; after 10 hr, 18% fall
Pectin + lactose PEF	Immediate 36% fall; after 6 hr, 31% fall	Immediate 22% fall; after 8 hr, 12% fall	No immediate fall; after 10 hr, 13% fall
Pectin alone FEV ₁	ND	ND	No immediate fall; after 10 hr, 23% fall
Pectin alone PEF	ND	ND	No immediate fall; after 10 hr, 22% fall

ND, Not done; FVC, forced vital capacity.

*From the beginning of exposure to the onset of lower respiratory tract symptoms.

†According to skin tests with common aeroallergens.

‡Skin scratch testing. Test result in one control subject was negative. With onion powder there was also a positive reaction.

§Skin prick testing. Test results in 16 control subjects were all negative.

||At baseline in histamine or metacholine challenge.

¶Control challenges with lactose showed no significant lung function changes.

antibodies were not detected in patients 2 or 3, but a positive value of 91 PRU/ml was found in patient 1. Patient 3 showed a late response 10 hours after the challenge with pectin, which indicates that other mechanisms are also involved. Patients 1 and 2 also showed falls in lung functions 6 to 8 hours after the challenge, but because they had received bronchodilating medication after immediate reactions (patient 1, two inhalations of rimeterol; patient 2, two inhalations of terbutaline), it is more difficult to interpret these late reactions. Because a previous report had suggested that IgG₄ antibodies may play a pathogenetic role,² we studied pectin-specific IgG₄ antibodies in patients 2 and 3 but did not detect such antibodies. In patient 3 a dose-response relation was demonstrated between the amount of pectin exposure and the magnitude of the lung function fall, which supports a causal link between pectin and asthma.

In conclusion, the three cases reported here strengthen the evidence that occupational exposure to pectin may cause asthma. Our findings suggest that type I hypersensitivity has a role in the development of pectin-induced asthma, but other mechanisms are likely

to be involved. Total avoidance of pectin powder is recommended for patients with pectin-induced asthma, because our patients continued to experience symptoms and PEF falls in connection with occasional pectin exposure even when receiving inhaled steroid medication regularly. The role of pectin in food allergies would be an interesting topic for future studies.

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Allergy to chicken meat without sensitization to egg proteins: A case report

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Allergy to chicken may be considered uncommon in comparison with other foods such as fish, egg, and milk in subjects without professional exposure.¹ In the last few years increasing attention has been given to the immunologic and clinical aspects of the so-called "bird-egg syndrome."^{2,3} Patients with this allergy exhibit cutaneous and respiratory symptoms after the ingestion of egg yolk and/or inhalation of bird- and chicken-derived materials (e.g., feathers) caused by a cross-reactive allergen (α -livetin, a chicken serum albumin).³

In this report we describe an unusual case of chicken meat allergy with severe cutaneous and respiratory symptoms after consumption of small quantities of this food and/or inhalation of chicken-derived materials in a subject without egg allergy.

CASE REPORT

We have observed a 23-year-old woman who experienced allergic symptoms (urticaria-angioedema and sometimes severe bronchial obstruction) after the ingestion of chicken and after breathing the air near a poultry pen.

The age of onset of cutaneous manifestations was about 3 years, and until this age the patient regularly ate chicken without problems. At the age of 6 years she experienced the first bronchial obstruction after breathing the air near a poultry pen. At the age of 12 years she had a severe crisis of asthma associated with a cutaneous involvement (urticaria-angioedema) near a poultry pen, the presence of which was unknown to the patient and was recognized only after the onset of symptoms.

In recent years, the patient experienced recurrent cutaneous and respiratory symptoms after unknowingly eating small quantities of chicken meat. During the same period she experienced asthmatic symptoms of varying degrees after inhalation of fumes originating from cooked chicken (even though she did not eat this food). Moreover, on one occasion the patient experienced intense edema and itching on her hands after contact with a sponge used to clean a pot in which a chicken had been cooked.

The patient does not have a bird in her indoor environment, and the ingestion of fresh or cooked eggs does not induce symptoms. Bronchial and/or cutaneous symptoms are not elic-

Abbreviations used

PPT:	Prick-prick test
SDS-PAGE:	Sodium dodecylsulfate-polyacrylamide gel electrophoresis
SPT:	Skin prick test

ited by exposure to other allergens, either by ingestion or inhalation.

METHODS

The examination of the patient included cutaneous serologic tests and nonspecific bronchial provocation challenge with commercially available extracts and reagents. Informed consent was obtained from the patient.

Skin prick test (SPT)

SPTs were performed according to the method of Aas and Belin⁴ with the use of a standardized pricker and extracts of common inhalant allergens and the most important foods (Dome Hollister Stier, Bayer Group, Milan, Italy).

A prick-prick test (PPT) with both fresh uncooked and cooked food was also done, on different days according to the method of Dreborg and Foucard,⁵ by using chicken legs and the same standardized pricker. This lancet was used for pricking first the muscle of the chicken leg and then the skin of the patient's forearm. Wheal areas obtained from both the SPT and PPT were also calculated.

Serologic IgE determinations

A serum sample was obtained for total and specific IgE determinations (Pharmacia CAP system FEIA; Pharmacia Diagnostics, Uppsala, Sweden).

Sodium dodecylsulfate-polyacrylamide gel electrophoresis (SDS-PAGE) and IgE immunoblotting

Protein extracts were purchased from three suppliers: soluble chicken feather and budgerigar feather from Hollister-Stier Laboratories (Spokane, Wash.); chicken meat, chicken serum albumin, and chicken egg albumin from ALK (Denmark); and egg yolk from Allergon AB (Engelholm, Sweden). Extracts were prepared according to the method of Ipsen and Löwenstein.⁶ The air sample taken from the area near a poultry pen was processed as previously described.⁷ Before use for electrophoresis, extracts were dissolved in distilled water and diluted in concentrated Laemmli sample buffer.⁸ SDS-PAGE and IgE immunoblotting were carried out as previously described.³ A serum pool from patients with bird-egg syndrome was applied for comparison.

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