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Occupational exposure to pectinase

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Key words: pectinase; proteolytic enzyme; sensitization.

● PROTEOLYTIC ENZYMES are used in many industrial processes, particularly in detergent production, the treatment of fruit, for agriculture, the baking industry, and wine-making. Enzyme allergy has been recognized since 1970; it can cause asthma, rhinoconjunctivitis, allergic contact dermatitis, and contact urticaria (1,2). The frequency of sensitization in exposed subjects has been reported at 4-60% (3), and the prevalence of respiratory disease 5-57% (2,4). Only three cases of allergic reaction due to pectinase were reported in the literature (5,6).

In the period between 1993 and 1999, 13 workers from a small factory selling pectinase and concentrated protopectinase came to the outpatient clinic of the Institute of Occupational Health of Brescia. Detailed work histories and information on work-related symptoms were collected for all the workers, who also underwent medical

**A potent sensitiser,
also of nonatopics.**

examination, respiratory function tests, chest X-rays, and measurement of total serum IgE and IgE antibody to pectinase (radioallergosorbent assay, RAST).

The subjects comprised seven men and six women, of mean age 39.2 years, with mean duration of employment of 9.5 years, and mean duration of exposure to pectinase of 4.3 years. Seven were smokers. Seven symptomatic workers referred latency of reaction, from the beginning of exposure to pectinase to the onset of dyspnoea and rhinitis, of between 4 and 24 months. No worker had any family history or personal history of atopy. Based on the clinical history (work-related symptoms 3/4 even past symptoms) and the results of allergologic tests, we diagnosed five cases of allergic occupational asthma, one of irritant occupational asthma, and one of irritant occupational rhinitis.

Among our population, the prevalence of sensitization to pectinase was 61%, while the prevalence of both sensitized and symptomatic subjects was 38%. These data are similar to those reported in studies on workers exposed to other proteolytic enzymes (2-4). No worker was atopic, which means that no workers had any family or personal history of past or present IgE-mediated allergic reactions to common allergens. In this population, the high frequency of sensitization and the short latency of the onset of work-related symptoms allow us to consider pectinase as a strong sensitizer, as are the other proteolytic enzymes (7).

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Do sex hormones influence airway mucosa?

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Key words: Asthma, sex hormones, estrogen, progesteron, and inflammation.

● In this study, we aimed to investigate the effects of sex hormones on bronchial tissues of mice.

Even though allergic mechanisms are of importance in about 80% of childhood asthma and in about 40-50% of the adult form, clinical observations and epidemiological studies show that the sex of the patient also may exert an important influence on the incidence, severity and progression of asthma (1). In early childhood wheezing and asthma are more common in boys than girls (2). In contrast, girls are more likely than boys to develop asthma in adolescence (3). In a new study, it is found

**No significant effects
were found on
bronchial epithelium of
mice**

that postmenopausal women present a significantly lower risk of developing asthma than premenopausal women of a similar age (4). In the same study, replacement therapy with estrogens increased the risk of asthma in a dose dependent manner. In animal models, sex steroid hormones have been shown to influence the immune system by various actions (5).

In this study, 74 BALB/c mice of three weeks old (in the preuberty) have been used. They were separated into four categories: Group 1: 11 males, 11 females; Group 2: 10 males, 10 females; Group 3: 10 males, 10 females; Group 4: six males and six females.

The mice in the Group 1 were injected intraperitoneally with 5 mg of progesterone every other day for two weeks. The mice in the Group 2 were injected intraperitoneally with 0.1 ml of estradiol benzoate 0.1 mg + estradiol phenylpropionate 0.4 mg + testosterone isocaproate 4 mg + testosterone propionate 2 mg + testosterone phenpropionate 4 mg, every other day for two weeks. Injections were repeated with 0.1 ml of estradiol benzoate 0.25 mg + progesterone 1.25 mg to the mice of Group 3 for the same period. Finally the mice of Group 4 were injected with 0.1 ml of serum physiological solution. The mice were dissected two days after the last injections. The cross-sections prepared from the lung tissues were investigated by the light microscopy.

The results were as follows. Group 1. Male mice: In only one of 11 mice there was a mild broncho-constriction with a heavy peribronchial cell infiltration consisted mostly of plasma cells, moderate lymphocytes and lesser mast cells. There were scattered areas of epithelial necrosis in the lumens of bronchioles. The remaining mice did not present any inflammatory changes. Female mice: There were not any inflammatory changes. Group 2. No remarkable histopathological features. Group 3. Male mice: No remarkable histopathological changes. Female mice: There were peribronchial mononuclear cell infiltrations in two of the mice. Group 4: No inflammatory changes.

In animal models, sex steroid hormones have been shown to influence the immune system by various actions: by affecting the structure and function of the thymus; modulating the activity of B and T cells,

natural killer cells and phagocytic cells (6). In human leukocyte studies (7,8) and in animal studies (9) estrogens have been shown to possess some anti-inflammatory properties. Physiological doses of estradiol significantly reduce the inflammatory responses in castrated mice although the percentage of polymorphonuclear cells in peripheral blood does not change (10). In our study, we intended to search whether if we could stimulate any inflammatory response in the bronchial tissues of prepubertal mice by giving them high doses of sex hormones. Our results do not allow making an interpretation about the inflammatory responses due to sex hormones in the studied mice. The inflammatory changes in three mice (one of Group 1 and two of Group 3) may be due to nonspecific environmental conditions. We conclude that we need further controlled investigations in this field.

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Allergy to peach

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Key words: bronchial provocation test; occupational asthma; peach; Rosaceae; Spain.

• A VARIETY OF high molecular weight allergens of vegetable origin, such as wheatflour or coffee bean, have been reported to cause allergic respiratory symptoms (1,2). As far as we know, occupational asthma due to peach inhalation, confirmed by specific bronchial challenge, has not been described in the medical literature. We report a case of occupational rhinitis and asthma in a factory worker, who handled