Cat is a major allergen in patients with asthma from west Siberia, Russia.

E. S. Gusareva, E. J. Bragina, E. V. Deeva, N. V. Kazakevich, V. P. Puzyrev, L. M. Ogorodova, E. S. Gusareva, E. J. Bragina, E. V. Deeva, N. V. Kazakevich, V. P. Puzyrev, L. M. Ogorodova, M. Lipoldová*

Key words: asthmatic patients; cat, and mite allergens; Russia; sensitization; specific IgE.

Asthma is the most severe allergic disease. The majority of cases are associated with atopy, which is characterized by hyperproduction of total and specific immunoglobulin E (IgE) against common environmental allergens (1, 2). Major allergens and prevalence of sensitization in atopic patients vary in different populations, providing cues about the pathogenic effects of environment and lifestyle. In Russia, measurement of sensitization to allergen mixtures is a common clinical practice in patients with severe symptoms of allergic disorders. The data on the less common tests of individual allergens have not been systematically collected or published in international journals and they are not accessible to scientists and clinical allergologists.

We present the first data on allergic sensitization in asthma patients from Russia.

We therefore estimated the specificity and intensity of sensitization to 20 different airborne allergens in patients with...
No evidence of tumor necrosis factor-α release in blood of patients with chronic urticaria

A. Tedeschi*, M. Lorini, C. Suli, R. Asero

Key words: autologous serum skin test; chronic urticaria; tumor necrosis factor-α.

Mast cells play a key role in chronic urticaria as they release histamine and other inflammatory mediators and cytokines causing urticarial symptoms. In about 30% of chronic urticaria patients, mast cell activation is triggered by autoantibodies directed against the α-subunit of the high affinity IgE receptor; in the other patients the stimuli inducing mast cell activation have not been defined, but an immunological mechanism is also suspected. From a practical point of view, the presence of circulating histamine-releasing factors is demonstrated by in vivo autologous serum skin test (ASST), which is considered as a screening test for histamine-releasing autoantibodies (1). Positivity of ASST allows distinguishing patients with chronic autoimmune urticaria from patients with an idiopathic disorder. Tumor necrosis factor (TNF)-α is released by human skin mast cells and other inflammatory cells which can be found at the site of urticarial lesions (2). Therefore, TNF-α is a candidate mediator of urticaria. Tillie-Leblond et al. (3) found TNF-α release during a systemic reaction occurring after cold immersion test in two patients with cold urticaria, and an increased immunoreactivity for TNF-α and IL-3 was detected on endothelial and perivascular cells of the upper dermis in skin lesions from chronic urticaria patients (4). We measured serum TNF-α levels in patients with chronic autoimmune and chronic idiopathic urticaria. Sera were drawn from 62 adult patients diagnosed as having chronic urticaria on the basis of recurrent hives for more than 6 weeks. In all cases, known causes of chronic or recurrent urticaria were ruled out by appropriate investigations. Physical urticarias were excluded as well. All patients had active urticaria at the time of the study. Five days after anti-histamine therapy (ceftizine, loratadine, or fexofenadine in all cases) was stopped, an intradermal test with 0.05 ml of fresh autologous serum (ASST) was carried out. ASST was performed and read at 30 min following the method by Sabroe et al. (1). Intradermal injection of saline solution (0.9% weight/volume NaCl) was performed as negative control and skin prick test with 10 mg/ml histamine as positive control. Patients showing a wheal with a diameter at least 1.5 mm greater than the control saline solution were considered positive. Forty patients were strongly positive on ASST and 22 were negative. TNF-α was assayed by a sensitive immunoenzyme method (R & D Systems, Minneapolis, MN, USA) using the same serum samples employed for ASST. Sera from 12 normal adult subjects were used as control. TNF-α concentration was below the sensitivity of the assay (15.6 pg/ml) in all serum samples from chronic urticaria patients and normal subjects. These results indicate that TNF-α is not released in measurable amounts in blood of patients with chronic urticaria, either autoimmune or idiopathic. Although a role for TNF-α as mediator of urticarial lesions cannot be excluded, its release in the skin microenvironment does not lead to a substantial increase in blood concentration.

*Internal Medicine 2
Ospedale Maggiore Policlinico
Mangiagalli e Regina Elena
Via Pace 9, Milano
Italy
Tel: +39 02 55033596
Fax: +39 02 50320723
E-mail: albited@alice.it

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Via Pace 9, Milano
Italy
Tel: +39 02 55033596
Fax: +39 02 50320723
E-mail: albited@alice.it

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