PATHOGENESIS OF ALLERGIC INFLAMMATION WITH PARTICIPATION OF IL-17 AND IL-23 CYTOKINES IN PATIENTS WITH CHRONIC EPSTEIN-BARR VIRUS PERSISTENCE IN ACTIVE AND LATENT PHASES

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Abstract. The biological properties of IL-23 are well-known for its ability to enhance Th17 cells functions, in particular their proliferation and production of IL-17. By affecting Th17 lymphocytes, IL-23 contributes to development and chronicity of inflammatory conditions and diseases, primarily autoimmune diseases. Allergic asthma is considered to be a Th2-dominant chronic inflammatory disease of the lungs. In patients with asthma are observed activation of Th2-cells, an increase in production of cytokines with subsequent pathogenetic effects. It is well known that viruses including the Herpesviridaeus family can be a trigger of different allergic conditions. It was suggested that the ability of IL-23 to induce the production of IL-17A and IL-17F may indicate its involvement in the pathogenesis of allergic diseases, including virus-induced. *The aim of our work* was to investigate the role of IL-17and IL-23 cytokines in the

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development of an allergic inflammation in chronic persistense of Epstein-Barr virus in active and latent phases. 38 people with allergic pathology and chronic persistence of the Epstein-Barr virus, mean age (32.7 ± 3.2) years, were included. General laboratory analysis, specific allergy and molecular genetic investigation was performed. Comparative analysis of IL-17 and IL-23 was performed in a group of people with confirmed allergy and chronic Epstein-Barr virus infection in active (first group, 20 people) and latent (second group, 18 people) phases. The control group consisted of 20 healthy people. Significant differences in levels of these cytokines were detected depending on latency phase of the Epstein-Barr virus and the control group. *Conclusions*. Obtained results demonstrate possible involvement of chronic EBV infection in pathogenesis of the allergic inflammation which depends on phase of the immunotropic virus persistence.

Key words: IL-17, IL-23, allergic diseases, Epstein-Barr virus.

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