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Short Communication
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Chitinases and Chitinase-Like Proteins in Pathogenesis of Asthma and Respiratory Diseases

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Recent experimental studies provide strong evidence in support of crucial involvement of Chitinases and Chitinase-Like Proteins (CLPs) in the pathogenesis of asthma. Particularly, mutant genetic variants of *CHIT1*, *CHIA*, and *CHI3L1* (YKL-40) and their increased protein levels have been associated with predisposition to asthma, as well as its severe form [1]. Chitin is a biopolymer and a key component of the exoskeleton of insects, fungi, parasitic nematodes, and house dust mites. Humans express chitinases (*CHIT1* and *CHIA*) and CLPs, including *CHI3L1* (chitinase 3-like 1), *CHI3L2* (chitinase 3-like 2), *OVGP1* (oviduct-specific glycoprotein), and *CHID1* (stabilin-1–interacting chitinase-like protein), which modulate innate immune responses against chitin-containing pathogens. Thus, chitinases confer a protective function against pathogens, but a self-damaging role after a response to allergens has been documented [2].

Chitin particles are inhaled into the airway as remains of dust mites and other organisms. In the airway wall, they initiate an innate immune response by stimulating the production of cytokines, chitinases (*CHIT1* and *CHIA*), and CLPs (*CHI3L1*) in macrophages, neutrophils, and eosinophils. Chitinases produced in the airway wall degrade chitins (polymers) into fragments, or these chitins are bound by CLPs, such as *CHI3L2*, which alter the biological effects of the chitin fragments on the immune response [1]. They also induce Th2-dominated immune responses, which are associated with allergic inflammation, including the increased production of proinflammatory cytokines, serum IgE, and prominent tissue eosinophilia. These cytokines and profibrotic factors, in turn, drive inflammation and remodeling in the bronchial wall [3].

In our study we genotyped patients with bronchial asthma and healthy volunteers. We estimated the frequency of mutations in the *CHIA* gene (rs3806448 (G→A) and rs12033184 (C→A)) in 445 samples of residents of the city of Novosibirsk, Russia (population control) and in 91 blood samples of children with asthma identified by the ISAAC questionnaire. The frequencies of polymorphic variants are rs3806448 G -0.5272:A -0.4728 for population control and G -0.5055:A -0.4945 for children with asthma; for rs120033184 G -0.5528:A -0.4472 for population control and G -0.5281:A -0.4719 for children with signs of asthma. There is no association with the disease for the chosen polymorphic variants of *CHIA* gene (OR=0.92 for rs3806448 G; OR=0.91 for rs120033184).

The lack of association of the *CHIA* gene mutations with asthma, despite previous studies showed the association of mutations in *CHIA* gene with asthma, is due to the poor choice of SNP (both substitutions rs3806448 and rs12033184 are located in upstream, and not in the structural part of the gene) and the small sample size of asthma.

References

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