ITGB3 (integrin β3) and TLR2 (Toll-like receptor 2) are candidate genes for asthma and mold allergy. Integrin β3 forms a complex with TLR2, and is required for the response of monocytes to TLR2 agonists such as fungal glucan. We therefore hypothesized that interaction between genes encoding the TLR2-ITGB3 complex may enhance susceptibility to mold sensitization.

The study was conducted in 1243 adults (514 with asthma) who participated in the follow-up of the Epidemiological Study on the Genetics and Environment of Asthma (EGEA). Sensitization to mold allergens was determined by skin prick test. Association of mold sensitization with 15 single nucleotide polymorphisms (SNPs) in ITGB3 was tested under an additive model. Interactions between the TLR2/+596 SNP, previously shown to be associated with asthma, and ITGB3 SNPs were tested.

Mold sensitization was found in 115 asthmatics (22.0%), and in 61 non-asthmatics (8.5%). The ITGB3 rs2056131 T minor allele was associated with a lower risk of mold sensitization in asthmatics with an odds ratio (OR) of 0.60 (95% confidence interval 0.43-0.83, P=0.001). Significant gene-gene interaction between nine ITGB3 SNPs and the TLR2/+596 SNP was found in asthmatics (P interaction=0.05 to 0.001). These ITGB3 SNPs were significantly associated with mold sensitization in carriers of the TLR2/+596 TT genotype (OR 1.85 to 2.79; P=0.03 to 0.002), whereas much weaker associations were found in carriers of the TLR2/+596 C allele (OR 0.65 to 0.91; P=0.04 to 0.60).

Interaction between innate immunity genes ITGB3 and TLR2 may influence susceptibility to mold sensitization in adults with asthma.

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