

IgE running interference for rhinovirus?

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Abstract

Anti-IgE treatment in pediatric allergic asthmatics reduces rates of symptomatic rhinovirus infection.

As asthma prevalence increases worldwide, we still lack a mechanistic understanding of the increased severity of respiratory viral infections in asthmatics. Both allergic sensitization and respiratory viral infections are clear risk factors for asthma exacerbations. There is a surge in exacerbations in the fall, which is likely multifactorial, including seasonal viral and allergen exposures. In the initial publication from the PROSE study, initiating therapy with omalizumab, an anti-immunoglobulin E (IgE) monoclonal antibody, in the mid-summer led to reduced rates of fall asthma exacerbations and improved in vitro antiviral activity from peripheral blood mononuclear cells. However, there was not a direct link between decreasing levels of free serum IgE and human respiratory viral infection susceptibility.

In this study, which extends the prior PROSE study, pediatric atopic asthmatics were run-in with guidelines-based asthma care, randomized to receive omalizumab (or not) before the fall season, and performed weekly online symptom counts and nasal swabs. Omalizumab decreased symptomatic respiratory illnesses (based on symptom counts). Rhinovirus (RV) is the most common virus implicated in pediatric asthma exacerbation, and all nasal swabs were assessed for RV species, regardless of symptoms. Though there was no effect of omalizumab on the frequency of RV infections (defined by positive swabs), there were fewer symptomatic days with a given RV infection. Most importantly, omalizumab reduced the frequency of RV+ symptomatic respiratory illnesses, with a trend toward reducing non-RV+ symptomatic respiratory illnesses.

This study links prior in vitro evidence of the negative effect of IgE on antiviral responses with clinical evidence that both atopy and viral infections are risk factors in asthma exacerbations. Here, they link the in vivo decrease in free serum IgE levels with omalizumab therapy with small but significant reductions in rates of symptomatic RV respiratory infections. Broadening the assessment of nasal swabs to non-RV viruses and bacterial pathogens, and replication in other cohorts, will deepen our understanding of the breadth of applicability of this connection, and these data streams will contribute to ongoing investigations into mechanisms that link elevated IgE (and the “type 2 high” inflammation asthma phenotype generally) to poor antiviral responses, suggesting novel therapeutic strategies.