Rhinoviruses are a major cause of asthma exacerbations. Asthmatics may have increased susceptibility to rhinoviral (and bacterial) infections but the mechanisms involved are not known. This study aimed to identify a possible mechanism of susceptibility involving induction of type III interferon, interferon-λ.

Primary bronchial epithelial cells from asthmatic and normal subjects were infected with rhinovirus and levels of viral RNA expression were measured. Viral replication was increased in cells from asthmatic subjects compared with normal subjects. Furthermore, both the expression of mRNA encoding type III interferons as well as the level of interferon-λ protein was decreased in asthmatics compared with normal volunteers. Levels of mRNA encoding the type III interferon and levels of interferon-λ protein were inversely related to levels of viral RNA. The production of interferon-λ by bronchoalveolar lavage (BAL) cells (mainly macrophages) from asthmatic and normal subjects infected with rhinovirus was also measured. Production of interferon-λ was again lower in cells from asthmatics than in cells from normal subjects.

After the ex vivo studies the same volunteers were inoculated with rhinovirus 2. Significant inverse correlations were observed between the ex vivo production of interferon-λ and severity of symptoms, BAL fluid virus load, and airway inflammation. The increased risk of bacterial infection was assessed by incubating BAL cells from the same subjects with lipopolysaccharide and measuring type III interferon production. Significant induction of interferon-λ was noted, but this was impaired in asthmatic subjects.

This study highlights a possible mechanism in the pathogenesis of asthma exacerbations involving type III interferon.

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Deficient innate immunity and susceptibility of asthmatics to infection

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