



Understanding the key issues in the treatment of uncontrolled persistent asthma with type 2 inflammation

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This review covers the pathophysiology of type 2 inflammation in asthma, its influence on type 2 comorbidities, and ways in which type 2 biomarkers can be harnessed to improve diagnosis and further personalise treatments in the age of biologic medicines <https://bit.ly/2MSOI20>

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Abstract

Asthma is a complex respiratory disease that varies in severity and response to treatment. Several asthma phenotypes with unique clinical and inflammatory characteristics have been identified. Endotypes, based on distinct molecular profiles, help to further elucidate the heterogeneity within asthma. Type 2 inflammation, involving both the innate (type 2 innate lymphoid cell) and adaptive (T-helper type 2 cells) immune systems, underpins the complex pathophysiology of chronic inflammation in asthma, as well as the presence of comorbid disease (*e.g.* chronic rhinosinusitis with nasal polyps, allergic rhinitis and atopic dermatitis). Type 2 inflammation is characterised by upregulation of the type 2 cytokines interleukin (IL)-4, IL-5 and IL-13, IgE-mediated release of immune mediators and dysfunction of epithelial or epidermal barriers. Targeting these key proximal type 2 cytokines has shown efficacy in recent studies adopting a personalised approach to treatment using targeted biologics. Elevated levels of biomarkers downstream of type 2 cytokines, including fractional exhaled nitric oxide, serum IgE and blood and sputum eosinophils, have been linked to mechanisms involved in type 2 inflammation. They have the potential to aid diagnosis, and to predict and monitor response to treatment. The objective of this review is to summarise the current understanding of the biology of type 2 inflammation in asthma, examine its influence on type 2 inflammatory comorbidities, and discuss how type 2 inflammatory biomarkers can be harnessed to further personalise treatments in the age of biologic medicines.

