

Towards safer oral immunotherapy for food allergy and allergic asthma is still increasing in prevalence

In October's issue of *Journal of Allergy and Clinical Immunology*, Frischmeyer-Guerrero et al published a study aiming to identify which patients are likely to benefit from omalizumab while undergoing oral immunotherapy (OIT) for milk allergy.^{1,2} Within a randomized, double-blind, placebo-controlled trial, omalizumab was demonstrated to reduce adverse effects during OIT but does not increase the efficacy of this intervention. They found that milk-induced basophil CD63 expression was transiently reduced to a greater extent in omalizumab than placebo-treated subjects. Pre-OIT basophil reactivity was positively associated adverse effects during OIT with much fewer symptoms seen with concurrent omalizumab therapy. The authors suggest that raised baseline basophil reactivity and a low allergen-specific IgE/total IgE ratio might help identify subjects who have less adverse effects and greater likelihood of sustained unresponsiveness with concurrent omalizumab therapy. Now, we need a prospective study to confirm this prediction.

Asthma and chronic obstructive pulmonary disease (COPD) share many features with some patients presenting with clinical features of both. In these issues, Hirai et al³ have used a clustering approach to identify which clinical and pathophysiological features best identify patients with this asthma-COPD overlap phenotype. They identified four biological clusters: 1, predominantly COPD; 2, asthma-COPD overlap phenotype; 3, non-atopic and late-onset asthma; and 4, early-onset atopic asthma. Features of both asthma and COPD identified the overlap phenotypes: raised total serum IgE, elevated blood eosinophil count, higher TBX21/GATA3 ratio, low

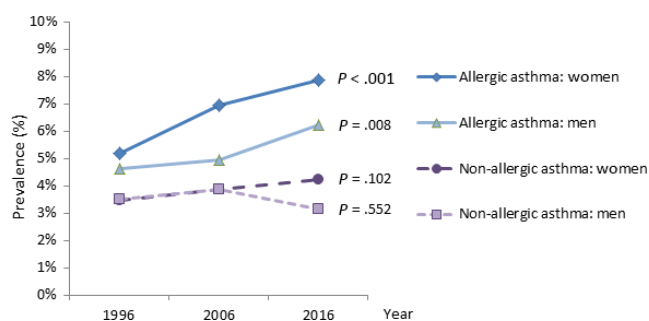


FIGURE 1 Prevalence of current asthma across 3 surveys [(see Figure 1 in Backman et al (pp. 1426–1435))]

Caption to cover illustration: Structures of saposins and Pru p 3. [see figure 5 in et al. (pp. 1398–1408)].

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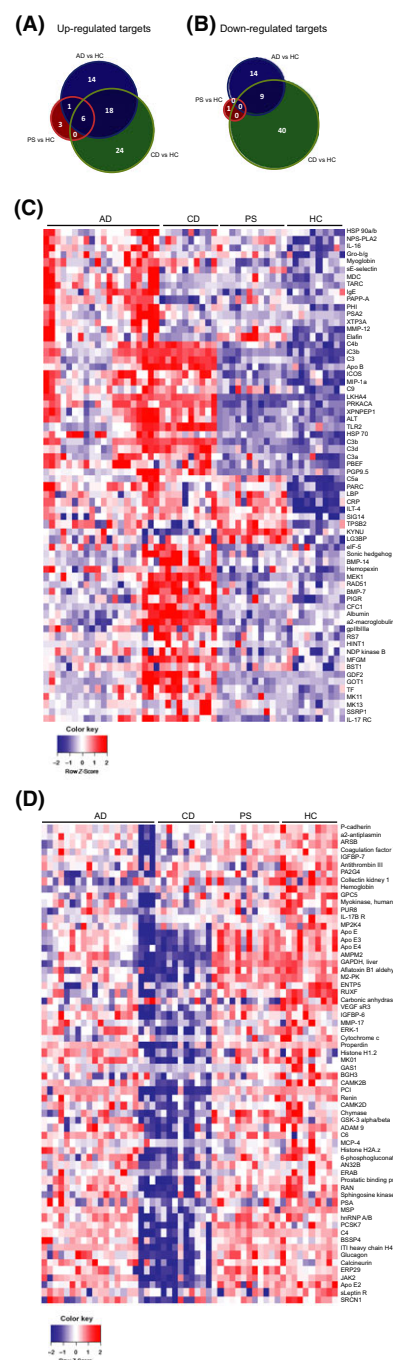


FIGURE 2 Heat map of proteins up-regulated in one or more of AD, PS or CD compared to healthy controls [see Figure 1 in Wang et al (pp. 1456–1467)]

FEV1/FVC ratio and smoking. Related features have been seen in other papers.⁴ Now, we need to see some replication data in another cohort and some other pathobiological data to understand whether or not each of these clusters has distinct pathophysiologies.

We all have our favourite data to quote about changes in asthma prevalence.⁵ Is prevalence still increasing or have we reached the ceiling? Backman et al⁶ have looked at how asthma prevalence has changed from 1996 to 2016 using questionnaire data from three cross-sectional Swedish samples from 1996, 2006 and 2016. Prevalence of current asthma increased significantly from 8.4% in 1996 to 9.9% in 2006 and to 10.9% in 2016. Allergic asthma (defined as co-existing reported allergic rhinoconjunctivitis) increased significantly from 5.0% in 1996 to 6.0% in 2006 and to 7.3% in 2016 (Figure 1). Non-allergic asthma was static at 3.4%-3.8% while smoking decreased from 27.4% in 1996 to 12.3% in 2016. So, at least in Sweden, asthma prevalence is still increasing with atopy perhaps driving this change.

Atopic dermatitis (AD), psoriasis (PS) and contact dermatitis (CD) have common features in terms of barrier disruption and systemic inflammation. Wang et al⁷ have aimed to characterize proteomic signatures that might differ between these skin conditions. They identified proteins that are modulated in all three conditions and others whose production is only modified in individual ones. The authors comment that these novel signatures may help to provide a better understanding of the pathophysiology of these conditions (Figure 2).

Finally, the European Academy of Allergy and Clinical Immunology (EAACI) Guidelines on Allergen Immunotherapy were launched at the Pediatric Allergy and Asthma Meeting (PAAM) in London at the end of October. They are available to download at the EAACI website.

G. Roberts^{1,2,3}

¹Faculty of Medicine, Clinical and Experimental Sciences and Human Development and Health, University of Southampton, Southampton, UK

²NIHR Southampton Respiratory Biomedical Research Unit, University Hospital Southampton NHS Foundation Trust, Southampton, UK

³The David Hide Asthma and Allergy Research Centre, St Mary's Hospital, Isle of Wight, UK

Correspondence

G. Roberts, Paediatric Allergy and Respiratory Medicine (MP803), Clinical & Experimental Sciences & Human Development in Health Academic Units University of Southampton Faculty of Medicine & University Hospital Southampton, Southampton, UK.

Email: g.c.roberts@soton.ac.uk

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