

The uncertainties and anxieties around food allergy

A report in the European Respiratory Journal now provides an explanation for how obesity and asthma may be linked.¹ Margaglione et al examined the airway wall for adipose tissue in individuals with and without asthma. Adipose tissue was found only in the outer wall of large airways. They found that the amount of adipose tissue was associated with body mass index, wall thickness and the number of inflammatory cells. These latter two phenomena could readily give rise to an adiposity-related asthma presentation (Figure 1).

In this issue of the journal, Knibb et al argue that there is a need for better access to psychological services to support families to cope with food allergy.² Food allergy has a pervasive impact on patients and their families. With the issues around precautionary labelling,^{3,4} those affected experience continued uncertainty and therefore anxiety about whether what they about to eating food that is contaminated with their triggering allergen. This can seriously impact on their activities and quality of life. The authors describe psychological approaches that may be helpful to patients who are particularly affected.⁵ Working in an allergy service multidisciplinary team that includes a psychologist, I can provide first-hand support for their argument.

Continuing on the theme of precautionary labelling, I should mention one of my papers.⁶ Within the iFAAM project, we surveyed consumers across Europe with food allergy. We aimed to understand how they assess risk with precautionary allergen labelling and

this could be improved. Our focus was on the use of a Quantitative Risk Assessment framework. Within this evidence-based approach would mean that only products likely to cause a reaction would have a precautionary allergen label.⁷ This would deliver certainty for consumers with allergies and help healthcare professionals give better advice.^{4,8}

Finally, Smeekens et al have looked at whether indoor dust promotes airway sensitization to peanut and peanut allergy development in mice, as they do for aeroallergens.⁹ In this model, inhalation of low levels of peanut only caused peanut allergy when combined with indoor dust (Figure 1). So environmental adjuvants in indoor dust may be important determinants of the development of peanut allergy in childhood.

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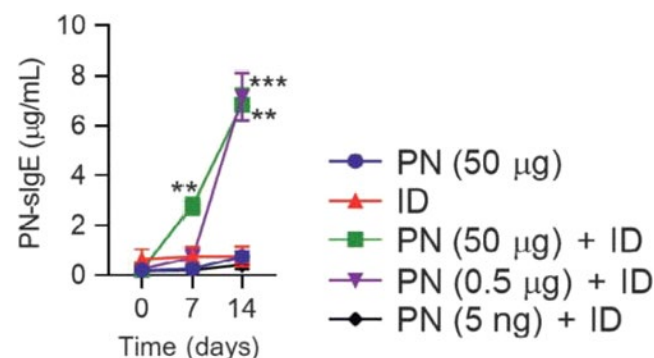


FIGURE 1 Indoor dust induces sensitization to low levels of inhaled peanut. Mice were sensitized with varying amounts of peanut (PN) (50 µg, 0.5 µg, 5 ng) with or without indoor dust (ID). Peanut-specific IgE (PN-sIgE) was measured on 0, 7 and 14. For more details, see Figure 2, Smeekens et al (pp. 1498-1509)

Caption to cover illustration: The cover image is based on the Review Article *Hereditary angioedema: Looking for bradykinin production and triggers of vascular permeability* by Margaglione et al, DOI: 10.1111/cea.13506.

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