

A complicated relationship between peanut environmental exposure and the development of allergic sensitization to peanuts

In case-controlled studies, peanut allergy has been associated with high household peanut consumption.^{1,2} Case-control studies are though very vulnerable to unrecognized confounding. So, Brough et al have looked at whether post-natal environmental peanut exposure is associated with later allergic sensitization to peanut in a population-based cohort.³ Maternal bed dust was collected post-natally in the BAMSE cohort. There was a significant association between peanut exposure and sensitization at age 4 years (odds ratio 1.41, 95% confidence interval: 1.05-1.90, $P = .02$) and 8 years (2.11, 1.38-3.22, $P = .001$) compared to sex and parental atopy-matched controls (Figure 1). Interestingly, an association was not seen when the whole BAMSE cohort was assessed.

Having pointed out the problem of bias with case-controlled studies, this is not an issue in the nested case-control design used by the authors as information about the cases and controls is collected prospectively before the outcome is assessed.³ So why is there a difference between the case-control analysis and the cohort one? I think the comparator is the key thing here. In the case-control analysis, the comparator was children with a family history of atopy, whereas in the cohort analysis, they were children with and without

such a family history. If high environmental exposure to peanut only results in peanut sensitization in genetically predisposed children, many of children with high exposure in the case-control analysis will have developed peanut sensitization as all had a family history of atopy. This contrasts with the cohort analysis where many of those with high peanut exposure will not have been genetically predisposed and so will not have developed peanut sensitization. So in this second analysis, high exposure will not seem to be associated with the development of sensitization. I would be interested in seeing what happens when the cohort analysis is repeated after splitting all the participants into those with and without a family history of atopy? My prediction is that a relationship is seen but only in those with a family history.

Children with asthma are vulnerable to rhinovirus-induced exacerbations of their disease.^{4,5} Looi et al have investigated the mechanism underlying this observation within a bronchial epithelial model.⁶ Bronchial brushing samples were obtained from children with and without asthma undergoing a general anaesthetic. Epithelial cells were grown ex vivo into a confluent, differentiated air-liquid interface (ALI) culture. Tight junction integrity was disrupted with

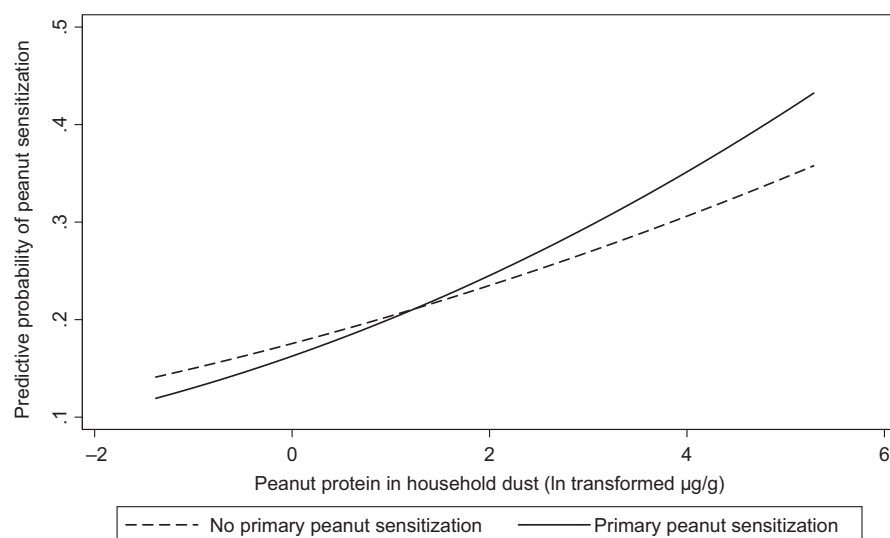


FIGURE 1 Predictive probability of peanut sensitization in children with and without primary peanut sensitization with different levels of peanut exposure [see figure 1 in H. A. Brough et al. (pp. 586–593)]

Caption to cover illustration: The cover image, by P. Korošec et al., is based on the Review *Important and specific role for basophils in acute allergic reactions*, <https://doi.org/10.1111/cea.13117>.

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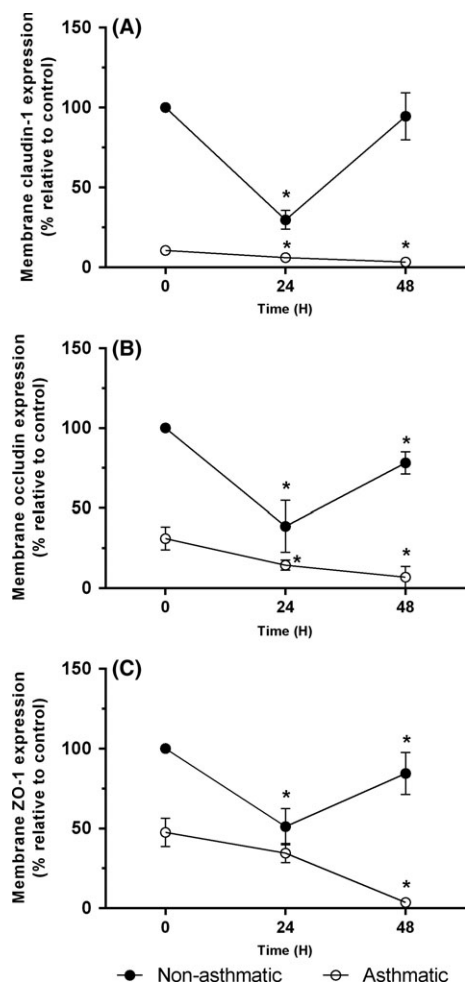


FIGURE 2 Expression of proteins from the membrane tight junction over time in airway epithelial cultures from children with and without asthma following rhinovirus infection [see figure 5 in K. Looi et al. (pp. 513–524)]

rhinovirus infection in all children but ones with asthma had more prolonged problems with increased permeability (Figure 2). Can we do anything to protect these tight junctions during a rhinovirus

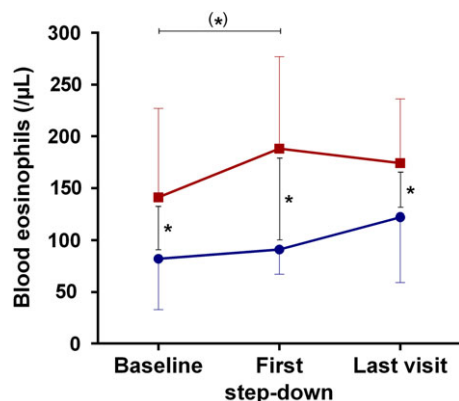


FIGURE 3 Absolute blood eosinophil count at baseline, after the first step-down and at the last visit in patients with a successful and unsuccessful withdrawal of inhaled corticosteroids [see figure 3 in S. Demarche et al. (pp. 525–535)]

infection and might this prevent viral infections exacerbations of asthma?

It is very easy to increase the dose of an inhaled corticosteroid in a patient with asthma. It is much more difficult to reduce the dose. Inhaled corticosteroids are not equally effective in all forms of asthma; non-eosinophilic asthma in particular is not thought to be responding well. So, Demarche et al have looked to see whether inhaled corticosteroids can be safely withdrawn in non-eosinophilic asthma.⁷ In 36 patients, the dose was weaned every 3 months regardless of asthma control unless there was a reduction in asthma control or an increase in exacerbations. In 39%, inhaled corticosteroids were completely withdrawn and in a further 28% they were reduced with no deterioration in asthma control (Figure 3). Predictors of successful reduction were younger age and low blood eosinophils. There are interesting parallels with predictors of response to oral corticosteroids.⁸

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