

EDITORIAL

Atopic disease and food allergy

Our understanding of the aetiology of the most important atopic diseases of childhood remains extremely limited. A role for foods has been contemplated both in atopic eczema and in atopic asthma. Such a role has been most strongly advocated in the case of atopic eczema, though it continues to be a source of considerable controversy [1]. The supporting evidence can be divided into four main categories:

(1) *Evidence of a prophylactic effect from modification of the diet in early infancy.* Though it is widely accepted that a modest prophylactic effect can be obtained by early dietary modification in genetically at-risk infants, this has not been established as clearly as one would wish. Neither could such an effect in any case be regarded as direct evidence that atopic eczema is ever caused by food.

(2) *Evidence of immediate hypersensitivity responsiveness (e.g. on intracutaneous testing), or of circulating food-specific IgE antibodies to specific foods.* Data in this category is compatible with the clinical observation that immediate hypersensitivity reactions to foods are common in children with atopic eczema, of which perhaps the most frequent is contact urticaria. However, the assumption that such reactions establish a role for foods in the provocation of these patients' eczematous skin lesions is not justified by currently available evidence, though it is widely held [2]. Indeed, the results of skin prick tests and the IgE RAST are used in many centres as a basis for the selection of foods to be excluded from patients' diets with the aim of improving their eczema, though there is little evidence that this approach is an effective one.

(3) *Evidence of provocation of eczema by oral food challenge.* Evidence in this category has been largely anecdotal, and challenges undertaken under properly controlled conditions have not resulted in unequivocal eczematous responses, more typically in responses of an immediate hypersensitivity type [3].

(4) *Evidence of therapeutic benefit following dietary exclusion of specific foods.* This would be the most convincing type of evidence, but very few adequately controlled trials have been reported. On the basis of the study we undertook several years ago [4], we do use empirical elimination diets in a proportion of young children with atopic eczema, where simpler therapeutic measures have failed. However, in practice it is our experience that such treatment is of limited value except in infants.

Much less investigative effort has been directed towards a

possible role for foods in the provocation of atopic asthma, perhaps because of a general feeling that inhaled allergen was *a priori* more likely to be relevant. It has been suggested that foods could play a provocative role in about 2–6% of atopic asthma patients [5,6]. Although not carefully studied, it is a clinical impression that immediate hypersensitivity reactions to foods, such as contact urticaria, angioedema and vomiting, are less frequent in asthmatic than in eczematous children, and this may also be a reason for less intense interest in an aetiological role for foods.

The paper from Adler *et al.* (pp. 683–688) in this issue, describes a study in which parental perceptions of food-related symptoms in children with asthma were compared with the results of skin prick tests and *in vitro* tests for IgE antibodies to specific foods. Parental perceptions are of course liable to substantial bias and therefore perhaps to exaggerated estimates of the frequency of food-induced symptoms. It is therefore of interest that parents of only 15% of children in this study reported that they had observed food provocation of their child's asthma. This cannot of course be taken to confirm that foods play no more than a small aetiological role; it may indicate merely that the onset of worsening of the asthma generally occurs so long after the ingestion of the responsible item that the relationship is obscured. Thus, parental perceptions may be exceedingly misleading in cases other than those in which the provocation of symptoms occurs rapidly after ingestion.

Adler *et al.* point out that a rapid increase in airway sensitivity has previously been demonstrated objectively in some asthmatic children following ingestion of tartrazine [7], benzoate and sulphur dioxide [7] and of cola drinks [8]. In none of these is it probable that the provocative factors act as antigens or haptens in a classical type I hypersensitivity reaction; they are perhaps more likely to be amplifying an immunological response or an inflammatory pathway by pharmacological or other means. In the 15% of cases whose parents had observed provocation of asthma by a food in the study by Adler *et al.*, 'additives' or 'fizzy drinks' had been implicated as the cause in about two-thirds. Only in the remaining 5% were 'other foods' (unspecified) incriminated which might have been acting as true antigens. Although the authors did not specifically investigate the level of agreement between the alleged asthmatic responses to these foods with the results of SPTs and the *in vitro* tests for IgE antibodies, previous studies have indicated that this was

likely to have been poor [5]. Of course, the most likely explanation for a lack of such agreement is that the symptom was not mediated by type 1 hypersensitivity.

If, in this study, one considers the non-asthmatic clinical reactions that are likely to have reflected type 1 hypersensitivity responses, one finds that parents reported urticaria/angioedema in 19% and unclassified gastrointestinal reactions in 8%. One might wonder whether skin prick and IgE antibody test results would have shown a clearer agreement with these symptoms than they did with all symptoms pooled.

A major portion of the paper by Adler *et al.* concerns the relative values in clinical practice of SPTs, the IgE RAST and the multiple chemiluminescence assay (MAST-CLA), and the authors are not able to reach conclusions on this issue. It is of course difficult to approach this issue in a study in which most of the alleged clinical reactions to foods were almost certainly not mediated by IgE antibodies. The conclusion of the authors that none of these tests proved useful in validating parental allegations of unspecified symptomatic reactions to foods is nonetheless an important one, and one that is still widely ignored, particularly in some of the less reputable but lucrative allergy clinics operating outside the NHS.

References

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