

Recent advances in peanut allergy

Jonathan O'B. Hourihane

Peanut remains preeminent as the food allergen most associated with severe and fatal allergic reactions. Reactions are frequent despite patients' best efforts to avoid peanut. In the future, better information sharing and communication between families and both schools and restaurants may lead to a decrease in the rate of severe reactions induced by exposure to peanut outside the home. Reaction severity may increase over time but up to 25% of young peanut allergic individuals may outgrow their peanut allergy. Personalized care plans and education programmes may have an impact on avoidance of peanut and on the appropriate responses of caregivers. Peanut's allergenicity may be affected by the method of cooking, with roasted peanuts appearing more allergenic than boiled or fried peanuts. Immunotherapy with modified peanut allergens and DNA based vaccines may soon move from animal studies to clinical trials. *Curr Opin Allergy Clin Immunol* 2:227–231. © 2002 Lippincott Williams & Wilkins.

Division of Infection Inflammation and Repair, Southampton University Hospitals NHS Trust, Southampton, UK

Correspondence to Dr Jonathan O'B. Hourihane, Division of Infection Inflammation and Repair, Mailpoint 218, Southampton University Hospitals NHS Trust, Tremona Road, Southampton SO16 6YD, UK
Tel: +44 2380 79 4939; fax: +44 2380 79 5023; e-mail: j.hourihane@soton.ac.uk

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Abbreviations

IgE	immunoglobulin E
IgG	immunoglobulin G
PCP	personalized care plan
SPT	skin prick test

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Introduction

There are many factors that contribute to a serious or fatal allergic reaction in the community. These factors may be either human factors such as asthma, exercise, medication use, risk-taking or delay of epinephrine administration, or they may be non-human, such as the dose of allergen, the location of reaction and particularly what is the specific food involved.

Peanut dominates the list of foods implicated in fatal allergic reaction to foods. Bock *et al.* [1•] have shown that peanut caused 14/21 food allergy fatalities for which they had full data and more than 50% of fatalities with incomplete data. This dominance has understandably led to greater fear of peanut than other foods when assessing the community care needed for peanut allergic individuals. The reasons for this dominance are still not clear.

Peanut is immunogenic and highly allergenic

Animal models for food allergy and atopic dermatitis have been established over the last few years [2,3]. Their use suggests that peanut is more allergenic than cow's milk in the mouse models established. Li and colleagues [4•] showed it is possible to sensitize weanling mice to milk but not adult mice, even using the mucosal cholera toxin. It was possible, however, to sensitize adult mice with peanut and cholera toxin.

In a separate series of experiments the same group have shown that serum immunoglobulin E (IgE) levels were higher in mice sensitized with lower doses of peanut and cholera toxin (5 versus 25 mg peanut extract + cholera toxin). Intragastric challenge with 10 mg of peanut extract produced higher anaphylaxis scores in the mice sensitized with the lower dose. Both the above findings were made using a particular strain of mice, C3H/HeJ [4•]. Previous work with a different focus has shown that different strains of mice respond differently to immunization with peanut plasmid DNA, some showing increases and others decreases in serum levels of IgG1 and IgG2a [2]. The transfer of DNA based or protein based immunotherapy strategies for treatment of peanut allergy from animal models to clinical practice awaits the development of post-exposure immunomodulatory strategies.

Peanut induces the production of significant amounts of immunoglobulin G (IgG) and its subtypes, as well as IgE, without any predominant pattern in groups of patients with differing levels of clinical severity [5•]. This suggests that the poly-isotypic response may account for the difficulty in inducing desensitization with conventional

immunotherapy based on native peanut protein. In non-allergic individuals, IgG antibodies to milk and peanut appear to reflect dietary exposure, though milk IgG levels were much higher than peanut IgG. Milk and peanut allergic individuals demonstrate similar patterns of IgE, G and G4 to the relevant allergens, suggesting the immune response is not exclusively committed to IgE production. The authors of this report hypothesize that sequential isotype switching may exist, sensitive to a common trigger such as interleukin-4.

Peanut allergens

The major allergen Ara h2 has been sequenced, with a deduced structure of 207 amino acid residues [6•]. The use of modified allergens for immunotherapy depends on, among other factors, decreasing IgE binding capacity while retaining the capacity to stimulate T cells, because tolerance is an active immunological process, rather than simply the absence of sensitization [7]. Peanut antigen, but not phytohaemagglutinin, induces oligoclonal responses of T cell receptor V β expression in HLA matched siblings discordant for peanut allergy, despite phytohaemagglutinin inducing a vigorous proliferative response [8•].

Bannon *et al.* [9•] have used site directed mutagenesis to produce modified peanut allergens which have significantly reduced capacity to bind IgE and still retain T cell stimulatory capacity. The modified Ara h2 allergen induced lower clinical scores of anaphylaxis and lower plasma histamine levels when tested using the mouse model of peanut anaphylaxis described above.

Early life origins of peanut allergy

Peanut allergy has been shown to affect 1% of French respondents to a questionnaire survey [10] and in keeping with British and American studies [11,12] peanut allergy was more prevalent in children than in adults.

It has been known for many years that most peanut allergic individuals react to their first overt exposure to peanuts, implying sensitization by unidentified or occult exposure to peanut before their first overt exposure [11,12]. Allergens are detectable in breast milk. These two independent facts have led to the concept of sensitization to peanut (and other allergens) via in-utero exposure or via breast milk [13–15]. Vadas and colleagues [16•] have now shown, conclusively, that peanut allergens are easily detectable in breast milk, from 6 h after ingestion of 50 g of peanut. Only 11 of 23 participants were shown to express peanut in breast milk. It is intriguing that only half of the participating mothers expressed peanut, and one mother expressed peanut in breast milk more slowly and for longer than the other 10 participants who expressed peanut. Atopy and post-partum age did not differ between secretors and non-secretors of peanut, but this important small study poses as many questions as answers.

International differences in prevalence of peanut allergy: are they due to how peanut is cooked?

It has long been a point of comment that peanut allergy appears to be a disease of developed countries. This has been attributed to the general epidemic of allergic diseases in such populations and the hygiene hypothesis [14,17]. The nature of the allergen may also be a significant difference because peanut is a staple food in many developing countries, with similar volumes of consumption to those in North America [18•]. The 'American' pattern of peanut consumption is to eat peanut raw or roasted, not boiled or fried, as it is in African (unpublished personal observation by author, 2000) and Asian populations, where peanut allergy is very uncommon [19,20].

Roasting of peanuts increases IgE binding [21]. Natural ageing of nuts [22] or cooking may cause production of advanced glycation end products [23•] that render proteins more resistant to acid digestion. Ara h 1 is resistant to heat induced changes in allergenicity, forming stable multimers [24]. Two groups have examined the effect of roasting and other methods of cooking [18•,23•] on peanut allergenicity. Roasting appears to increase production of advanced glycation end products, increase IgE binding (abolished by anti-advanced glycation end product antibody) and decrease acid digestion of proteins. In contrast, boiling or frying decreases IgE binding. This is intriguing work, applying basic biochemistry to an area of fundamental epidemiological uncertainty.

Peanut consumption in Sweden has only increased by 5% between 1996 and 1999, but the number of specific IgE tests requested for peanut has nearly trebled [25•]. This is probably due, at least partly, to increased awareness and ascertainment bias. Positive results were more likely in children less than 10 years old, with 42% of infants tested aged less than 2 years having positive results. Only 60% of positive results were associated with clinical reactivity, with symptoms more common with higher CAP class results, as expected. No data are given about maternal or feeding practices in Sweden or about the nature of the peanut products that are most common in Sweden, though it appears that roasted peanuts are the most common form and peanut butter is very rare (personal communication, G. Johansson, December, 2001). Overall peanut consumption may not have increased much in Sweden but young children are again identified as the group most affected by peanut allergy.

Clinical severity can increase over time ...

A major point of anxiety for parents and uncertainty for clinicians has been what to advise for children with mild reactions to peanut. Cross-sectional questionnaire re-

views have suggested symptoms may worsen with time [11]. This has been supported by a further cross-sectional study from New York [26••] and confirmed in a longitudinal study from Denver [27••], though a recent British study has contrasting findings [28•].

The Peanut Allergy Register of the Food Allergy Network demonstrated again the early life origins of peanut allergy and that most first reactions occur at home [26••,29••]. The location of exposure predictably changes with time, with restaurants and other non-home locations increasingly common with subsequent exposures [26••]. The Peanut Allergy Register report of increased severity and concomitant increased use of epinephrine with subsequent exposure may be confounded slightly by increased awareness of symptoms and reaction strategies and the availability of epinephrine (which is usually not available for a first reaction).

Vander Leek [27••] followed annually a group of 102 children whose peanut allergy was diagnosed before the age of 4 years. Mild initial reactions were followed by further reactions in 70% of cases, with 44% being more severe. Even mild skin reactions to oral contact followed the same pattern on re-exposure. Finally, this study confirmed previous findings that severe peanut allergic reactions predict future severe allergic reactions.

... but peanut allergy can resolve

The bad news for peanut allergy sufferers about progressively increasing severity of reactions is offset by the confirmation that peanut allergy can resolve [30]. It may be that the increased severity with age actually partly reflects resolution in childhood of more minor degrees of allergy to peanuts. Vander Leek's [27••] longitudinal study identified 4/102 young children who lost sensitivity to peanut. Spergel *et al.* [31••] reported the first case of a child reverting from a positive challenge to peanut to a negative challenge 1 year later and no further reactions after 3 years of follow up. Further negative challenges resulted in those with a history of urticaria after peanut exposure (9/17 cases), a history of refusing to eat peanut but found to have a positive skin prick test (SPT) (8/13 cases), and flares of atopic dermatitis after peanut exposure (4/10). No individual with severe initial reactions achieved tolerance. Zimmerman and Urch [32•] reported tolerance achieved by 10% of patients after a minimum of 1 year between tests.

Skolnick *et al.*'s [33••] large study of 223 children diagnosed as peanut allergic suggests up to 22% of young children with peanut allergy may achieve tolerance. These study participants have similar peanut specific IgE at diagnosis but significantly lower levels when the repeat challenge was performed and found to

be negative. Only 2/54 children with the highest levels (>10 kU_A/l) achieved tolerance. The presence of a specific IgE of less than 2 kU_A/l at the time of the repeat challenge was associated with negative challenges in 67% of cases. As in other studies, the prevalence of other atopic diseases was lower in those who tolerated peanut than in those with persistent disease. Smaller studies have shown differing results about initial reaction severity [30,31••]; however, the larger study in Baltimore, Maryland and elsewhere [33••] suggests initial minor skin reactors are more likely to outgrow peanut allergy.

Skin tests, specific immunoglobulin E and food challenges

Several years ago values of SPT and food allergen specific IgE were promulgated as predictive of the outcome of food challenges, based on a retrospective review of highly selected populations [34,35]. The same research groups and others have now brought forward prospective studies on less selected children that confirm the earlier reports. SPT responses to peanut of 4 mm in children less than 2 years old and 8 mm in children older than 2 years [36••,37] predict a positive challenge result. In an older group of patients, a specific IgE value of greater than 15 kU_A/l using the CAP system also suggests a challenge will be positive [38••]. All the results for SPTs were produced in open challenges, not double-blind challenges, but these are acceptable data due to the young age of the children studied, when an open challenge is easily used. Nearly all the data for specific IgE were generated by interpretation of a convincing or suggestive history rather than challenges. Differences between the studies may be accounted for by the use of different systems for measuring specific IgE, different reagents for SPT and age groups of children studied. It must be remembered also that there may be reasons to challenge individuals with higher levels of peanut SPT or specific IgE (e.g. never exposed to peanut). Similarly, the low sensitivity of the values above which a positive challenge is likely (the 'diagnostic decision point') means that positive challenges are possible at any level of specific IgE, whether measured directly in serum or indirectly in the skin by SPT. The diagnostic decision points do not help in ruling out a specific food allergy, for which a challenge is needed. A food challenge is not negative until the food has been reintroduced in its native form [39]. The above studies and others [40•] support the widely held practice that a negative SPT with peanut virtually eliminates the likelihood of a positive reaction to peanut during a challenge.

Reactions in schools

Recent studies of preschool and school age children have shown that most reactions in children, which are usually

not fatal, occur outside a school environment – at home or, increasingly, in restaurants (the frequency of the latter increases with age) [41••]. An allergic reaction in school can, however, represent the first reaction in 25% of peanut allergic individuals [26••].

Rhim and McMorris [42••] surveyed the strategies of 273 elementary schools in Michigan for supporting food allergic pupils. Fifty-five percent of responding schools reported more than 10 identified food allergic pupils enrolled in their school. Only 16% of schools had a written individual care plan for each food allergic pupil. The situation is slightly better if the food implicated is peanut: a small proportion of the 73 schools with peanut allergic pupils provided most of the examples of designated classrooms, use of Medic Alert identification and personalized care plans (PCPs).

There is an interesting difference between what schools reported as their intended response to an allergic reaction and what others found when they looked at the problem from the ‘consumer’ point of view [26••]. Ninety-four percent of schools reported that they would transfer the child to a medical facility and only two of 109 schools said they would contact the child’s parents for further assistance [42••]. In fact, schools contacted parents to ‘find out what to do’ after 60% of actual reactions and in 32% the parents noticed signs of reaction simply when picking their child up from school [26••].

Personalized care plans

Schools cannot be accountable if a child is not identified as food allergic or if a child is identified as being allergic to one food but then reacts to another. The availability of emergency plans varies from 33% of peanut and tree nut individuals nationwide in the USA [29••] to 86% of pupils in Baltimore [43••].

Data from recent studies imply that the mere presence of an information pack and action plan decreases the incidence of allergen exposure at home and in school and day care facilities [28•,44••]. PCPs appear to benefit the majority of those so equipped. The higher rate of reaction in children without a PCP in place [44••] must force us to reconsider the prospective assessment of risk and the withholding of PCPs, or their local equivalent, from those who are currently considered low risk, based on retrospective assessments of previous reactions.

A PCP can decrease the rate of re-exposure to peanut and other food allergens. Personalized care plans should be reviewed regularly. Schools must have programmes for training new members of staff and for retraining of existing staff. Doctors and allied professionals should provide these programmes rather than parents.

Conclusion

Clinical data from community surveys and registers confirm the predominance of peanut as the allergen most often associated with severe and fatal allergic reactions. Increased care needs to be paid to education of families, schools and caterers about hazard identification and responses to exposure. The finding that there may be a worsening of reaction if peanut allergy persists past early childhood is offset by the confirmation that peanut allergy can resolve in about 20–25% of young children affected. Significant progress is being made using animal models and identification and modification of peanut allergen structure, which may soon lead to human trials of immunomodulation using modified proteins or DNA based vaccines.

References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:

- of special interest
- of outstanding interest

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