



FOOD ALLERGY UPDATE

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Emerging Trends in Food Allergy



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In this issue of food allergy update, we examine evidence that the prevalence of food allergy has increased over the last decade, assess whether this increase is real or artefactual, and discuss techniques available for accurate diagnosis.

Case study

NATALIE

Natalie is an 8-month-old girl, born to parents with a family history of hay fever and asthma. She was exclusively breast-fed for 5 months. She developed severe atopic eczema at the age of two months and was noted to vomit frequently after feeds. On one occasion, at the age of 5 months, her father kissed her on the cheeks after he had been eating some cheese and Natalie developed mild contact urticaria on the face at the site of the kiss. Concerned about possible food allergies, her parents took her to a naturopath, who undertook 'hair analysis' and diagnosed multiple food 'allergies'.

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Her mother was advised to start herself on a strictly vegan diet, to avoid dairy products in her own diet and to substitute dairy products with unfortified soy milk instead. Despite feeling fatigued and having difficulties with milk supply, Natalie's mother was encouraged to continue breast-feeding because of initial improvement in the child's eczema. By the age of 6 months, Natalie appeared to be underweight, and was generally miserable. Assessment by her GP showed evidence of iron deficiency anaemia and a low albumin of 20 g/L (35-50 g/L).

Consider your response to the following questions:

- Q** How would you interpret the information provided by the patient's mother at this point?
- Q** Do you think that allergy might explain the child's symptoms?
- Q** What investigations would you recommend, if any?
- Q** What do you make of the initial diagnosis made by the naturopath?
- Q** How would you manage Natalie?

Clinical Progress

Her GP recommended ImmunoCap allergy testing, which showed positive reactions to cow's milk (specific IgE 17 kU/L), egg (1 kU/L) and peanut (10 kU/L). Based on these results, the GP recommended avoidance of cow's milk products, egg and peanut in the child's diet and that the child be weaned onto a soy formula. The GP referred her to an allergy specialist for further assessment. A few days after commencing soy formula, her eczema dramatically worsened. Her GP telephoned an allergy specialist for advice, who recommended commencement an extensively hydrolysed cow's milk formula [eHF] (or an amino acid based formula, if eHF not tolerated),¹ which led to significant improvement after a few days.



Allergic reactions to food: overview

Allergic reactions to food may be classified as IgE mediated, non IgE-mediated or mixed IgE/non-IgE mediated. The IgE-mediated food allergies are most common and best understood. The non IgE-mediated food allergies and the mixed IgE/non IgE mediated food allergies have been characterised more recently and our understanding of the pathogenesis of these syndromes continues to evolve.

IGE MEDIATED FOOD ALLERGIES

IgE-mediated food allergies result in 'immediate' (rapid onset) allergic reactions to food, are estimated to affect around 1 in 20 infants and are relatively easy to diagnose. Symptoms typically include hives, angioedema, and vomiting, usually within 30 minutes of food ingestion. More serious symptoms of anaphylaxis may include noisy breathing, difficulty breathing, hoarse voice, persistent cough, feeling drowsy, collapse or an infant going pale and floppy, usually occurring together with one or more of the symptoms listed above. IgE mediated reactions to foods are usually associated with positive allergy test results (skin prick testing or blood allergy testing), so that skin testing or blood allergy tests can help to confirm the diagnosis. This form of allergy is most common in young children. Hen's egg, cow's milk, peanut, tree nuts (most commonly cashew), soy, wheat, seeds or seafood account for over 90% of reactions.

NON-IGE MEDIATED FOOD ALLERGIES

Over the past decade, there has been increased recognition of the non-IgE mediated and mixed IgE/non-IgE mediated food allergy syndromes. These include the eosinophilic disorders (eosinophilic esophagitis, eosinophilic gastroenteritis) and the food protein induced disorders (food protein induced enterocolitis syndrome [FPIES], food protein induced enteropathy, food protein induced colitis). These conditions are distinct from the IgE-mediated food allergies in that they present with delayed onset of symptoms following ingestion of the food, involve predominantly gastrointestinal symptoms, and skin prick tests are usually negative.

These forms of food allergy usually result in 'delayed' immune reactions and typically present with abdominal symptoms such as abdominal pain, chronic diarrhoea, severe recurrent vomiting, or failure to thrive/weight loss. The mixed IgE/non-IgE mediated food allergies may also lead to worsening eczema. Symptoms commence several hours or days after exposure to the food, and occur due to inflammation of the skin or gut. Routine skin prick testing and blood allergy testing is usually negative, making diagnosis more difficult, although so-called 'atopy patch tests' have shown some promise in recent studies. The most common triggers are cow's milk, soy and wheat, although other foods may also be the cause. The most common symptoms are one or more of:

- eczema/dermatitis
- frequent or persistent vomiting or diarrhoea
- irritability
- abdominal pain
- failure to put on weight
- blood loss from the bowel (sometimes).

Unfortunately, many of these symptoms may occur in other non-allergic conditions or with anatomical blockages in the bowel. It is, therefore, important to exclude other causes, while maintaining a high index of suspicion for the possibility of food allergy. Diagnosis usually rests on the history of reactions to food, improvement following elimination of the food and recurrence of symptoms following re-introduction/challenge with the food. Most of these 'delayed' allergic conditions resolve by the age of 3 years but occur occasionally in older children and adults.

Reactions usually occur with ingestion of larger quantities of the food than for the IgE-mediated food allergies; however, even small amounts of food allergen passing through breast milk can sometimes aggravate eczema or gut symptoms. Allergy testing of the infant may or may not be positive, depending on the underlying immune mechanism involved (mixed IgE/non-IgE vs non-IgE).

Eosinophilic oesophagitis is one of the more common forms of delayed food allergy. Eosinophilic oesophagitis is an inflammatory disease of the oesophagus, characterised by an eosinophilic infiltrate into the oesophagus mucosal lining and symptoms of severe acid reflux, dysphagia and sometimes food impaction. In infants, eosinophilic oesophagitis may present as gastroesophageal reflux disease. Skin tests may be positive as the underlying immune mechanisms are thought to involve both IgE and non-IgE mediated mechanisms. Atopy patch tests are suggested to be positive in the majority of patients.^{2,3} There is a high rate of response to allergen avoidance through initiation of an elemental diet with amino acid formulae (of the order of 90%), and a lesser response to directed diets that remove common 'allergenic' foods.⁴



How common is food allergy?

While the prevalence of food allergy is estimated to be approximately 5% for infants under 3 years,⁵ accurate assessment of food allergy prevalence is plagued by a variety of difficulties including a lack of reliable, population-derived estimates based on objective methods of assessment.^{5,6} Many studies of food allergy prevalence have utilised surrogate methods of identifying food allergy such as hospitalisation rates for anaphylaxis or patient/parent self-report of food allergy via, for example, telephone surveys.^{6,7}

Such self-reports of adverse food reactions generally overestimate the true prevalence of food allergy by 3-4 fold.^{8,9} For example, parent reported rates of food allergy are suggested to be between 6.8 and 11.8 % of children aged between 1 and 10 years.⁸⁻¹¹ However, more stringent studies using a combination of survey, investigator questioning, allergy testing and formal in hospital challenges have defined much lower rates of food allergy, between 2.3 and 3% of children aged 1-6 years.⁸⁻¹⁰ This overestimation of food allergy prevalence with self reporting may in part be because patients or parents of young children may mistake coincidental reactions to food or 'food intolerance' reactions (toxic reactions, food poisoning, enzyme deficiencies such as lactase deficiency leading to lactose intolerance, or irritation from skin contact with citrus, tomatoes or Vegemite) for true food allergy.

... globalisation and adoption of foods from different countries has led to reduction in geographical differences

To some extent, the wide geographical ranges reported for food allergy prevalence may represent different prevalence or different triggers in different populations, and different methodologies employed to ascertain prevalence. For example, rice and seafood allergy are more common in Japan than other countries and fish allergy is more common in Scandinavia. In countries such as Australia and the USA, peanut allergy is common and is also increasing in Asia, consistent with a globalisation of dietary trends.

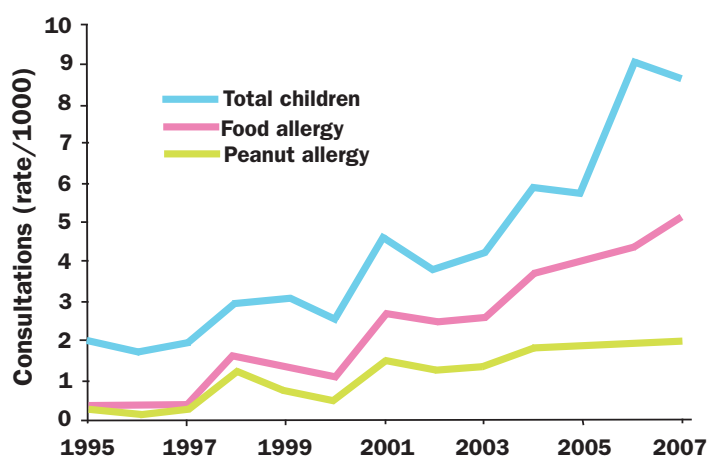
IS FOOD ALLERGY INCREASING?

To determine whether food allergy is truly increasing in prevalence, one requires an accurate estimate of food allergy prevalence in the same population at different time points using identical methodology, preferably using a combination of patient survey, questioning by skilled personnel, and confirmatory allergy testing as well as challenge.

In the absence of such studies in Australia or elsewhere, surrogate markers have been used as indicators of such an increase. One author of this article (RJM) examined changing demand for specialist food allergy services for children aged less than 4 years from 1995 to 2006 as a surrogate measure for changes in prevalence. Demand for food allergy assessment increased 12 fold from 1995 to 2006.¹² Over the same period, Australian hospital admissions for all anaphylaxis doubled, while admissions for food induced anaphylaxis in children aged 0-4 years increased 5 fold.^{12,13} (Updated data derived in part from this study is shown in Figure 1). These data are consistent with studies from the UK,¹⁴ and New York¹⁵ over the last decade, indicating similar anaphylaxis hospitalisation trends. Moreover, recent general practice database studies in the UK reflect the same trends, independent of the potential artefacts induced by relying upon data from hospital anaphylaxis admission rates, which could be confounded by lower threshold for admission or a lower threshold to diagnose anaphylaxis instead of milder allergy-like reactions.¹⁶



FIG. 1. CONSULTATION RATES FOR CHILDREN AGED LESS THAN 6 YEARS WITH FOOD ALLERGY (OVERALL) AND PEANUT ALLERGY (SPECIFICALLY). AGE ADJUSTED POPULATION RATES FOR ACT RESIDENTS ONLY AGED LESS THAN 6 YEARS, (UPDATED FROM MULLINS RJ, MJA 2007).



The best evidence of recent increases in food allergy is derived from studies examining the prevalence of peanut allergy.¹⁷⁻¹⁹ Examining children aged 3-4 years old on the Isle of Wight using a combination of population surveys, skin testing and challenge, clinical allergy to peanut increased from 0.5 to an estimated 1.5%, while sensitisation to peanut trebled from 1.1 to 3.3% between 1989 and 1994-6.^{17,19} A similar increase in prevalence of self reported peanut allergy from 0.6 % to 1.2 % between 1997 and 2002 was derived from a random digit telephone survey of American children, although without confirmatory allergy testing or challenge.¹⁸

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These data taken together with longer allergy clinic waiting lists in Australia and New Zealand, are consistent with a recent marked increase in the prevalence of food allergy, that closely follow the previously noted increases in asthma, eczema and allergic rhinitis prevalence.

ARE ALL ALLERGIES RISING EQUALLY?

Not all allergic conditions have risen simultaneously, suggesting that differential aetiological factors may be contributing. In Australia, rising rates of asthma were the first to be noted.²⁰ Review of asthma prevalence rates 10 years later showed that asthma amongst school children aged 6-7 years had plateaued and fallen by 23%, while eczema and allergic rhinitis continued to rise.²⁰ A study from the UK¹⁴ highlighted these disparities in rising prevalence for the different allergic conditions, showing that while admissions for allergic rhinitis had fallen by 40% and eczema-related admissions had slowed, admissions for anaphylaxis and food allergy had risen by 700% and 500%, respectively.

Evidence remains less clear as to whether the delayed type non-IgE mediated and mixed IgE/non-IgE mediated food allergies are increasing in parallel with the IgE mediated food allergies. Increases have been observed for eosinophilic oesophagitis in children with prevalence rising from 0.05 to 0.89 cases/100,000 children in Western Australia between 1995 and 2004,²¹ and similar changes noted in the USA,²² but retrospective studies abroad have cast some doubt on whether this food allergy disorder is increasing in prevalence, or whether it is simply being more accurately diagnosed.

WHY THE RISE IN FOOD ALLERGY?

There are more questions than answers in this area. While the hygiene hypothesis (whereby lessened exposure to microbial stimuli in early childhood is postulated to be associated with an increased risk of allergy) has been proposed as one possible explanation, this has not been studied specifically in food allergy and there is no information whether this might play a role in food allergy despite provisional evidence of its possible role in asthma and atopic eczema. Caesarean section delivery has been reported to be associated with a 1-2% rise in the rate of allergic disorders, possibly related to alterations of the infant intestinal microbiota,²³ but would not explain the more dramatic increases in food allergy diagnoses noted in recent studies.

Increased exposure to allergenic foods such as peanut, tree nuts or sesame seed, has been proposed as one potential explanation. Whilst superficially this seems to make sense, this would not explain the increase in allergic reactions to other common allergens such as egg or milk, which have occurred despite these being ingested at similar levels in western diets over the same time period. Furthermore, while peanut allergies in US children have reportedly doubled from 1997 to 2002,¹⁸ consumption of peanuts or awareness of food allergies did not appear to increase to this extent over the same period.

Food processing methods have been shown to increase the allergenicity of foods.⁶ For example, dry-roasting peanuts, which is common in Western countries including Australia is known to increase allergenicity compared with boiling or frying peanuts, and may be a contributor beyond simple food exposure alone.⁶

There is no evidence that exposure to preservatives in food or vaccination has a role to play. Sensitisation to food via topical exposure (such as in some moisturisers, or by kissing)²⁴ has been proposed as one potential contributor and would be consistent with observations of contact sensitisation followed by systemic allergic reactions to food that have been described, particularly in those working in the food industry.²⁵ There is some human and animal evidence that the use of antireflux medication might have some role to play,²⁶ but as the majority of young infants with food allergy have not had gastroesophageal reflux disease (GORD) nor exposure to these medications, this is unlikely to play a major role.

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Sensitisation to allergens through the breast milk was proposed over 10 years ago as a potential risk factor particularly with regard to peanut.²⁷ Subsequent studies have failed to support this as a contributor and there is no evidence that dietary restrictions during pregnancy or breast-feeding are of any benefit in reducing the risk of food allergy, and with some evidence of an adverse impact on maternal and child nutrition.²⁸

TABLE I. MECHANISMS PROPOSED TO EXPLAIN THE RISE IN FOOD ALLERGY PREVALENCE

MECHANISM
Reduced exposure to microbial stimuli in early childhood (Western lifestyle)
Increased exposure to potential food allergens
Infantile colic
Food processing techniques
Topical sensitisation to allergens
Prolonged dietary allergen avoidance strategies
Other (eg antacid medication)

IS AVOIDANCE THE ANSWER?

Advice to avoid potentially allergenic food during pregnancy, breast-feeding and early infancy is commonly recommended and practised in our community. However, there is no evidence that dietary restrictions during pregnancy or breast-feeding are of benefit in reducing the risk for the infant developing allergic disease. There is evidence that early introduction of complementary foods or cow's milk formula in the first 3 months of life is associated with an increased risk of developing allergic disease in infancy including food allergy, and that exclusive breastfeeding for the first 3-6 months of life is associated with a reduced risk for the development of allergic disease compared to feeding with cow's milk formula; therefore allergen avoidance in the first 4-6 months of life can be recommended at this time for the prevention of allergic disease in early childhood. There is, however, no evidence that food allergen avoidance in the infant diet beyond the age of six months is of any benefit in reducing the development of allergic disease including food allergy.²⁹ Two recent studies have provided provisional evidence that such strategies might even promote sensitisation and food allergy rather than tolerance.^{30,31}

In the last few months, both the American Academy of Paediatrics and the European Paediatric Gastroenterology Society have reversed previous recommendations to restrict infant diets beyond the age of 6 months.^{32,33} These changes reflect current Australasian Guidelines developed by the

Australasian Society of Allergy and Clinical Immunology (<http://www.allergy.org.au/content/view/182/1/>) that do not recommend food restrictions beyond the age of 4-6 months as a strategy to reduce the risk of new food allergy developing (unless allergy to a specific food is already established). Overall, more evidence is emerging regarding the most appropriate stage to introduce solids in allergy prevention.

Accurate diagnosis of food allergy

Accurate diagnosis is dependent on a detailed history, a determination of whether an allergic mechanism is likely to be playing a role, the results of allergy testing (either skin prick testing or blood allergy testing), and in some cases withdrawal of potential allergenic foods followed by challenge.

HISTORY

As in any condition, a detailed history should be undertaken to determine whether allergy is likely to be playing a role. Symptoms that should arouse a strong suspicion of food allergy in infants include anaphylaxis; generalised allergic reaction with urticaria, angioedema and/or severe vomiting (within 1–2 hours of ingestion); persistent vomiting; failure to thrive; or bloody diarrhoea.^{5,34} However, there are a large number of other symptoms that may also be caused by food allergy. These include gastro-oesophageal reflux, atopic dermatitis unresponsive to conventional treatments, severe infantile colic presenting in the first weeks of life, and persistent constipation following introduction of cow's milk formula. Establishing the temporal relationship between ingestion of a food and onset of the symptoms can help ascertain the likely mechanisms causing the food allergy (IgE or non-IgE-mediated immune mechanism), and can support the allergic nature of reactions (if reactions are consistently reproducible after ingestion of the food). Reactions that occur immediately or less than 1 hour from food ingestion are generally IgE-mediated whereas delayed reactions (onset several hours to >24 hours after ingestion) are non-IgE mediated. An exception is FPIES, which can cause profuse vomiting within several hours of ingestion.

MEASUREMENT OF FOOD-SPECIFIC IGE

Measurement of food-specific IgE (by skin prick testing or a CAP-RAST blood test) may be of use when IgE-mediated food reactions are suspected. Single food allergen tests should be requested directed against the most common triggers; food mixes should not be ordered as these frequently give misleading or difficult to interpret results. The significance of such results (and diagnostic decision points) is discussed below. Food specific IgE may be negative in the setting of non-IgE mediated food allergy and mixed IgE/non-IgE mediated food allergy. Total serum IgE is of no use in evaluating a child with potential food allergy and should not be requested.

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ATOPY PATCH TESTING

Unfortunately, skin prick testing and measures of food-specific IgE have a limited role to play in patients with delayed immune reactions to food as discussed above. Atopy patch testing (APT) has been proposed as a potential method for confirmation of at least some forms of delayed food allergy. This test is based on cutaneous T-cell mediated responses to food allergen that is applied to the skin under occlusion; a positive reaction is demonstrated by examination for the appearance of localised dermatitis 48 hours after application. Criticisms aimed at APT include a lack of standardisation of preparations and signs diagnostic of a positive result, the time-consuming nature of this technique and the relatively small additional positive predictive value they offer over SPT and specific IgE antibody tests.³⁵ While commercial test preparations are available in some markets, the exact role of this technique remains to be defined and validated.

Finally, endoscopy or colonoscopy with biopsy may be required to prove or disprove the presence of allergic disease in patients with predominantly gastroenterological symptoms.

DIAGNOSTIC DECISION POINTS

It is important to note that when evaluating patients with IgE mediated hypersensitivity to food, the magnitude of the response measured on skin prick testing or by in vitro assay for serum allergen specific IgE can provide an indication of the likelihood that there is true clinical allergy – the larger the skin test wheal or higher the serum level of food specific IgE, the more likely there is clinical allergy. Food-specific cut-offs have been proposed for the diagnosis of clinical allergy to cow's milk, egg, peanut and fish.³⁶⁻⁴³ Importantly, however, the size of the SPT wheal or level of food specific IgE does NOT provide a guide to the likely severity of any clinical reactions that might occur upon ingestion of the food. Furthermore as described above, skin prick tests and serum levels of food specific IgE will be of little use in the diagnosis of patients with delayed immune reactions to food.

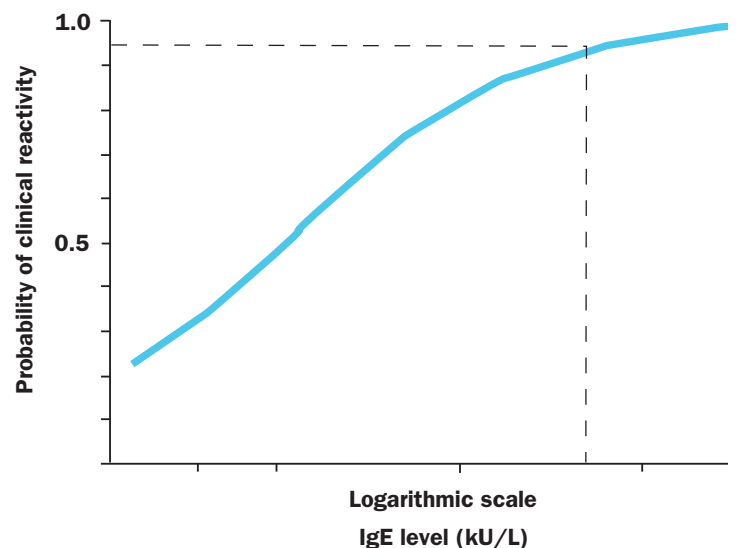
Several authors have established decision points for specific IgE tests that predict clinically relevant food allergy

Several authors have established decision points for specific IgE tests that predict clinically relevant food allergy. These have been defined in various studies for both SPT and serum food specific IgE levels.^{5,44,45} For SPT, diagnostic decision points are based on wheal diameters that predict a positive food challenge result with over 95% accuracy. Australian investigators have previously determined SPT wheal diameters that were highly predictive for allergy to cow's milk (≥ 8 mm), egg (≥ 7 mm) and peanut (≥ 8 mm).⁴⁵ However, it should be pointed out that predictive SPT wheal diameters are often smaller in young children under 2 years of age and that variation in published diagnostic decision points exists, likely due to the variability of skin testing technique, skin prick test device used, extracts and operators.⁵

Diagnostic decision points for food-specific IgE levels measured by in vitro testing have also been defined for several foods including cow's milk, egg, peanut and fish.⁴⁴ As with cut-off points for SPT, the decision points for cow's milk and egg-specific serum IgE in infants under 2 years of age are lower than in older patients. Also, as shown in figure 2, there is

a continuous relationship between IgE concentration and risk of clinical allergic reaction. False negative results may occur more frequently with CAP-RAST testing than with SPT, and therefore, infants with a low food specific IgE concentration and high clinical suspicion of allergy should have allergy ruled out by a negative SPT and food challenge.

FIG. 2. PROBABILITY OF CLINICAL REACTIVITY ACCORDING TO IGE CONCENTRATION



Finally, the measurement of food-specific IgE as a 'screening test' may result in positive results of uncertain significance if the child has not consumed the food and no reaction has occurred, as there is high rate of sensitisation (positive SPT or CAP-RAST) that is not associated with clinical allergy (50-60%).^{46,47} Under these circumstances, specialist assessment and consideration of medically supervised challenge may be indicated.

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UNORTHODOX ALLERGY TESTING

The use of unproven diagnostic techniques for 'allergy diagnosis' is common in our community, most commonly Vega testing, cytotoxic food testing, IgG food allergy testing as well as kinesiology and hair analysis. These practices are based on concepts of disease pathogenesis very different to those underlying Western medicine, have no scientific rationale, and have not been shown to be reliable or reproducible when subjected to formal study. ASCIA advises against the use of these tests for diagnosis or to guide medical treatment. No Medicare rebate is available in Australia for these tests, and their use is not supported in New Zealand. Adverse consequences may also arise from unorthodox testing and treatments. Treatment based on inaccurate, false positive or clinically irrelevant results is not only misleading, but can lead to ineffective and at times expensive treatments, and delay more effective therapy. Sometimes harmful therapy may result, such as unnecessary dietary avoidance and risk of malnutrition, particularly in children (<http://www.allergy.org.au/content/view/322/271/>).

The trends to increased prevalence of allergic disease have important implications for public health, medical workforce planning, costs of health care and the availability of specialist allergy services in Australia and New Zealand

IMPLICATIONS OF AN INCREASED PREVALENCE OF FOOD ALLERGY

The trends to increased prevalence of allergic disease have important implications for public health, medical workforce planning, costs of health care and the availability of specialist allergy services in Australia and New Zealand. There is an urgent need to invest in training of allergy/immunology specialists and other health professionals with an interest in allergy and increase the skills of our general health workforce to provide evidence based advice.



Case study answers

Do you think that allergy might explain the child's symptoms?

A role for food allergy should be considered in infants with severe eczema not responding to initial skin care measures. Nevertheless, while food allergy is common in young children, the majority of infants with eczema do not have food allergy as a major contributor. Natalie has severe eczema and a history that raises the possibility of cow's milk allergy (hives on skin contact with cow's milk). She may or may not have additional food allergies. Reduced albumin may be the result of intestinal loss due to intestinal inflammation, or loss through the skin due to severe eczema.

What investigations (if any) would you recommend, if any?

It would be reasonable to perform single allergen skin prick or blood testing for IgE to cow's milk and soy. Skin prick or blood testing for IgE to other common food allergens to which Natalie has not been exposed, such as egg, peanut, sesame seed, cashew and wheat may also be considered. In this case, the skin test to cow's milk is likely to be positive given the history of urticaria following cutaneous contact with cow's milk. Nevertheless, a negative skin prick test or CAP-RAST would not exclude the diagnosis.

What do you make of the initial diagnosis made by the naturopath?

Unproven diagnostic tests are potentially misleading, may delay appropriate diagnosis and lead to unnecessary dietary restrictions with nutritional consequences, as in this case.

What other issues are raised by this case?

In evaluating cases of eczema where parents are concerned about the role of allergy (a very common concern), it is important to educate parents on the routine management

of this common condition (ensuring adequate use of emollient moisturisers, topical corticosteroids, wet wraps, staphylococcal reduction measures), to ensure adequate nutrition of infant and mother if any dietary restrictions are considered, and to seek skilled dietetic advice if undertaken. In Natalie's case, she was unfortunate enough to suffer from delayed immune reactions to soy as well, which resolved with a hypoallergenic formula. Around 10% of infants with delayed type non-IgE mediated or mixed IgE/non-IgE mediated reactions to cow's milk will also develop allergy to soy, and the first line treatment for the delayed forms of cow's milk allergy would be hydrolysed cow's milk formula or amino acid based formula.

Where food allergy is suspected, in vitro testing for common single food allergens (milk, soy, egg, peanut for example) may help guide initial management pending specialist advice. The results of in vitro testing in this case, however, also raised other concerns. She had high levels of specific IgE to cows milk consistent with her history, but also had high levels of peanut specific IgE, which while suggestive of her probably being allergic to peanut, would not be definitive until accidental or deliberate exposure occurred. By contrast, low levels of egg-specific IgE were measurable, which may or may not be indicative of egg allergy. Clarification of whether Natalie was clinically allergic to egg would require a formal challenge to egg performed in a hospital setting. These results underline the problems associated with 'screening tests' for food allergy in the absence of a definitive allergic reaction to that one food. The results of allergy testing may sometimes correlate with the history, but may at times provide data of uncertain clinical significance, which may one day need to be defined by deliberate challenge.

FAQs

If you would like a question related to any aspect of food allergy diagnosis or management answered, write to: Food Allergy Update, Nutricia Australia Pty Ltd, Talavera Corporate Centre, Level 4, Building D, 12-24 Talavera Road, North Ryde NSW 2113 or email: nccl@nutricia.com.au

I have heard that eosinophilic oesophagitis can be caused by inhalation of allergens. Is this true?

Immunological interaction between the airways and the gut certainly appears a real phenomenon. Animal studies have demonstrated the presence of eosinophilic oesophagitis in animals in response to intranasal allergen challenge. This suggests that inhalant allergens are able to elicit eosinophilic inflammation in the gastrointestinal tract and highlights a potential link between pulmonary and oesophageal eosinophilic inflammation. Observations in humans support this concept by demonstrating increased numbers of duodenal eosinophils and mucosal IgE-positive cells during the pollen season among patients with, for example, birch pollen allergy.



Netting the Net

ASCIA (AUSTRALASIAN SOCIETY OF CLINICAL IMMUNOLOGY AND ALLERGY) EDUCATION RESOURCES

www.allergy.org.au

Make this site your first place to look for information regarding all clinical aspects of allergy and immunology. With numerous links to a variety of conditions and information arranged for both health professionals and patients, the site has the advantage of having locally relevant content compiled by the peak body of Clinical Allergists and Immunologists in Australia and New Zealand.

ACT AGAINST ALLERGY

www.actagainstallergy.com.au

www.actagainstallergy.co.nz

An initiative of Nutricia, the Act Against Allergy website contains a plethora of information for both health professionals and parents of infants and children with food allergies. The healthcare professionals section allows access to clinical literature, congress and meeting reports, tools, useful links and more. Please log on and register.

ANAPHYLAXIS AUSTRALIA

www.allergyfacts.org.au

www.allergyfacts.org.au is the website of Anaphylaxis Australia, whose stated aim is 'developing anaphylaxis awareness through education, research & support'. This site contains a wide range of literature and resources to answer many questions about living and coping with anaphylaxis and would be an ideal website referral for parents of infants newly diagnosed with this condition.



**IN OUR NEXT NEWSLETTER, WE EXAMINE THE USE OF LONG-CHAIN
POLYUNSATURATED FATTY ACIDS IN INFANT FORMULA**

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Declaration of interest

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