food allergy

There is no consensus on the causes or prevention of food allergy, and the number of young patients presenting to GPs with parents concerned about food reactions is increasing. This article reviews the methods available to determine allergy and identify the possible food(s) responsible, and how to advise families to protect allergic children at risk of reactions – including the more severe effect of anaphylaxis – and what they may expect as the child ages. This article was written by Rohan Ameratunga, Adult and Paediatric Clinical Immunologist and Immunopathologist, Auckland Hospital and Clinical Associate Professor, Molecular Medicine and Pathology, School of Medical Sciences, University of Auckland.

Allergy increasing but causes unknown

Adverse reactions to foods are a common problem in general practice. Food allergy is a potentially severe adverse reaction to food proteins mediated by the immune system. There are, however, other forms of adverse reaction to foods, including toxic reactions (eg, ciguatera) and intolerances. Adverse food reactions of a non-toxic origin may be of one the following types:

- immune (food allergy)
  - IgE-mediated (eg, eczema)
  - non-IgE-mediated (eg, coeliac)
- non-immune
  - enzymatic (eg, lactase)
  - pharmacologic (eg, histamine).

Food aversion can occur in someone who already has allergies to foods. In some cases, the mechanism behind an adverse reaction to a food remains uncertain.

This article aims to help GPs with the very important task of identifying adverse reactions due to food allergy as the treatment and prognosis vary widely depending on the type of reaction.

For reasons that are unclear, the prevalence of food allergy appears to be increasing. Studies of a large group of children living on the Isle of Wight suggest a tripling of the prevalence of peanut sensitivity/allergy in less than 10 years.

The recent, well-conducted HEALTHNUTS community-based study from Melbourne suggests 10 per cent of all one-year-old children had a defined food allergy. Our own 2010 pilot study suggests food allergy in New Zealand may be at least as common as overseas.

The International Study of Asthma and Allergies in Childhood (ISAAC) has shown that up to 30 per cent of schoolchildren are allergic and up to 10 per cent of this group are likely to have severe allergy. Ideally, these children would be under the care of an allergy specialist. Currently, however, there is a worldwide shortage of allergy specialists. Some parts of Australia do not have specialist services. In New Zealand, there is an uneven distribution of specialists, with most located in Auckland.

For as long as the causes remain unclear the prevalence of food allergy is likely to continue increasing as there are no effective universally accepted prevention strategies.

Having a severe food allergy has profound social and lifestyle implications for patients and families. Eating out or visiting friends becomes potentially hazardous. Many children with severe food allergies have to take their own food when going to birthday parties. Having a child with severe food allergies can have a profound adverse effect on quality of life. One study suggests having a child with a food allergy has the same impact as having a child with type 1 diabetes.

Do you need to read this article? Try this quiz

1 An adverse reaction to a food does not necessarily imply an allergic mechanism. **True/False**
2 A defined food allergy is present in 10 per cent of one-year-olds. **True/False**
3 The strongest predictor of developing food allergy is the timing of introduction of foods. **True/False**
4 Goat's milk can be substituted in children with cow's milk allergy. **True/False**
5 Up to 80 per cent of children with egg allergy develop asthma. **True/False**
6 Two months is the minimum for a trial short-term elimination diet. **True/False**
7 The risk of dying from food allergy is highest during adolescence. **True/False**

Answers on page 33

Food allergens vary

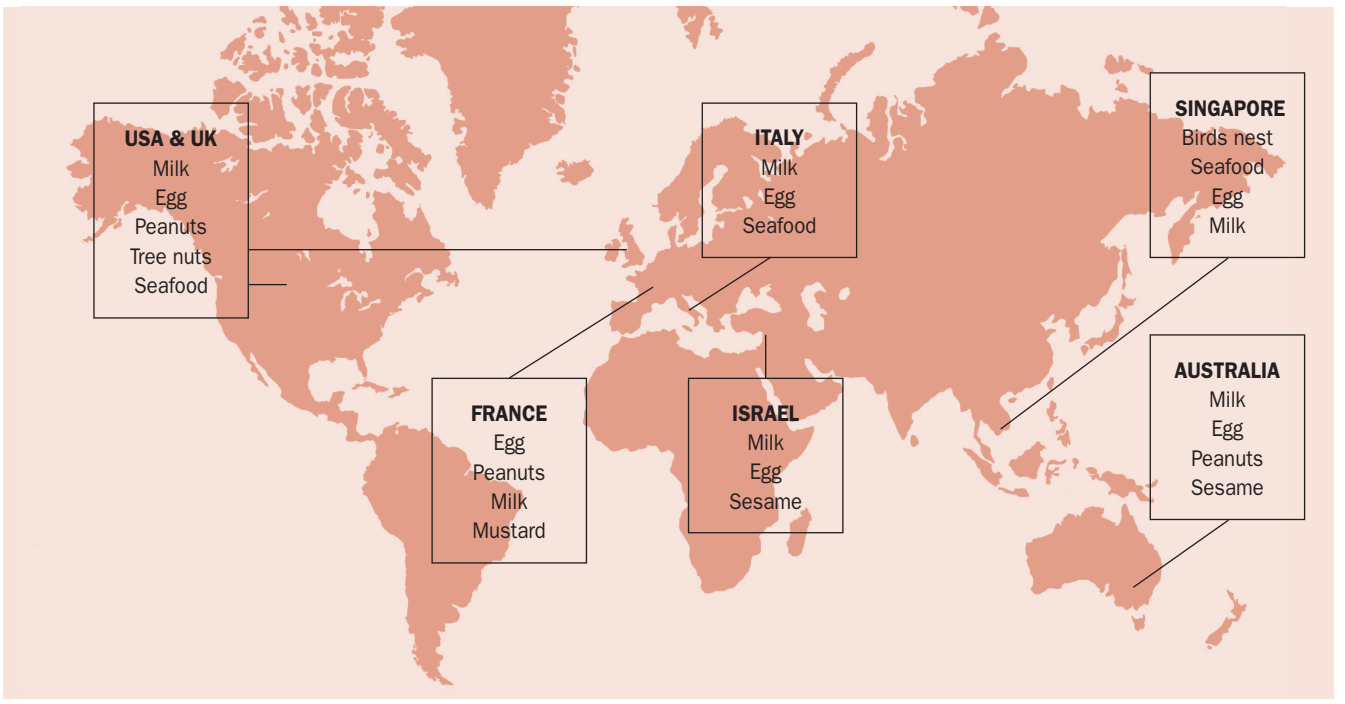


Figure 1. International ethnic patterns of food allergy

In children, the most common food allergens are cow's milk, egg, soy, wheat, peanut, tree nuts and fish. In adults, tree nuts and shellfish are also important.

There are ethnic and cultural differences in food allergy and this is largely a reflection of diet (Figure 1). In Japan, rice allergy is common. In the Middle East, sesame allergy is an increasingly recognised problem. Mustard allergy is common in France. Sesame allergy is also well recognised in Australia, which has a large Middle Eastern immigrant population. Fish and shellfish allergy are common in Singaporean children eating a traditional Chinese diet. With increased immigration, the pattern of food allergy may change in New Zealand because of changes in dietary intake.

Food cross-reactivity

IgE antibodies formed by exposure to specific foods can cross-react with closely related foods (Table 1). This is usually a result of structurally similar allergenic proteins.

Cross-reactions can closely follow phylogenetic relationships (eg, the cross-reaction between cow's and goat's milk). Most infants allergic to cow's milk react to goat's milk. Goat's milk is not a substitute for children with cow's milk allergy.

IgE antibodies formed by exposure to specific foods can cross-react with closely related foods

It is also clear that up to half of patients who react to one tree nut react to other tree nuts. Similarly, there is cross-reactivity among shellfish. Generally, patients who react to one crustacean (eg, crab) react to other crustaceans (eg, lobsters, crayfish, prawns) but are less likely to react to bivalves (eg, oysters, scallops).

Other forms of cross-reactions (eg, between cow's milk and beef and between chicken and eggs) are recognised but rare. Panallergens (eg, storage proteins) are thought to be structurally conserved and may be responsible for IgE cross-reactions. There are some unusual cross-reactions (eg, between pollens and food or latex and food) due to structurally similar allergens, even though botanically unrelated.

When patients are convinced they have multiple food allergies, this is sometimes due to cross-reacting IgE antibodies. In other situations, apparent allergy to multiple foods may be due to allergen contamination.

There are, however, an increasing number of children who have multiple food allergies. These children face the twin hazards of allergic reaction(s) and potential nutritional deficiency from a restricted diet. Such children should be under the care of a team specialising in food allergy.

Food cross-reactivity (Table 1)

Food	Cross-reaction	Percentage (%)
Egg	Chicken	<5
Cow's milk	Goat's milk	>90
Cow's milk	Beef	10
Fish	Other fish	50
Peanut	Legumes	<10
Wheat	Other cereals	25
Tree nuts	Other nuts	>50

Nutritional risks of long-term elimination diets (Table 2)

Allergen	Vitamins and minerals
Milk	Vitamin A, vitamin D, riboflavin, pantothenic acid, vitamin B_{12}, calcium and phosphorus
Egg	Vitamin B_{12}, riboflavin, pantothenic acid, biotin and selenium
Soy	Thiamin, riboflavin, pyridoxine, folate, calcium, phosphorus, magnesium, iron and zinc
Wheat	Thiamin, riboflavin, niacin, iron and folate if fortified
Peanut	Vitamin E, niacin, magnesium, manganese and chromium

Clinical evaluation and tests required

An accurate diagnosis is essential in patients presenting with adverse reactions to food. If the causative food is not identified, the child risks ongoing allergic reactions to the food. In contrast, inappropriate elimination diets can place the child at risk of nutritional deficiencies (Table 2).

The diagnosis of food allergy is heavily dependent on the clinical evaluation as well as laboratory testing. Neither can be interpreted in isolation. It is very important to determine if there is a close temporal relationship between the consumption of a specific food and development of symptoms. There can be pitfalls with this approach as patients who have eczema as their main manifestation of food allergy may not identify a close link between their rash and a specific food.

Patients with eczema are often sensitised or allergic to multiple foods. It is important to distinguish between sensitisation and allergy; sensitisation reflects the presence of IgE antibodies but not necessarily clinical allergy.

Children with specific IgE antibodies to a food, and who are tolerating this food, should continue to consume this food. Stopping it may, paradoxically, result in allergic reactions when the food is reintroduced after an interval.

In contrast, a young child with high levels of food-specific IgE, and who has never been exposed to the food, may be at risk of allergic reactions. Such children should be evaluated by a specialist to determine if the elimination diet should continue or whether food challenges be undertaken.

Identification of food triggers requires testing and may require elimination and food challenges to confirm the relevance of food-specific IgE antibodies (see later).

The physical examination helps to determine if there have been any complications from food allergy, such malnutrition or growth retardation, as well as the presence of eczema.

Immunological mechanisms behind food allergy

Food allergy can be mediated by a variety of immunological mechanisms. These vary from severe anaphylaxis caused by IgE antibodies to malabsorption caused by cytotoxic T cells in coeliac disease (Figure 2). Some forms of food allergy appear to be caused by multiple mechanisms.

IgE antibodies mediate most forms of immediate food allergy. In this situation, some individuals, for genetic reasons, generate IgE antibodies to harmless antigens (allergens). When the person is re-exposed to the food (or other allergen), mast cell and basophil-bound IgE cross-links the food (or other) allergen. This leads to degranulation of mediators leading to clinical manifestations. Immediate reactions mediated by IgE antibodies are among the most severe and can be life-threatening.

There is likely to be a strong genetic element in IgE-mediated food allergy, as the strongest predictor of developing food allergy is family history. This is helpful in identifying high-risk individuals who may need to be closely monitored.

The natural history of food allergy

Most children with egg and cow's milk allergy have symptoms that remit by middle childhood. Cow's milk and egg allergy tend not to recur once an individual has achieved a remission. Some data suggest cow's milk allergy can extend into adolescence and even early adulthood.

Of children who have well-documented peanut allergy, data suggest 20 to 30 per cent undergo remission. The highest rates of remission are seen in children who had a low to moderate (2KIU) specific IgE test.

Other forms of food allergy, such as shellfish allergy in adults, tend to persist. Most research on food allergy, however, has been conducted in children. Adults who develop food allergy later in life are increasingly being recognised.

Formula substitutes

In children with a proven allergy to cow's milk, a hypoallergenic formula (ie, Pepti-Junior) can be used. Some children allergic to cow's milk may react to the hypoallergenic formula and require an elemental formula, such as Neocate or Elecare (currently funded).

In older children, soy formula can be used as a milk substitute. There are unfounded theoretical concerns with soy, such as those regarding phyto-oestrogens and genetic engineering. In children under six months, soy should not be used as there is a significant risk of soy sensitisation.

Goat's milk formula is not a satisfactory alternative for children with cow's milk allergy given that the allergens are almost identical. In one study of children allergic to cow's milk, almost all reacted adversely to goat's milk.

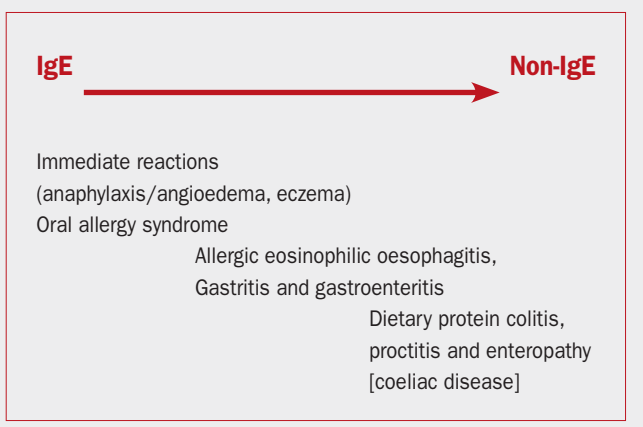


Figure 2. Pathogenesis of food allergy

Testing for specific food allergy

IgE-mediated food allergy can be identified with skin prick testing or specific IgE testing from blood (Figure 3).

Skin testing for food allergy

For skin testing, a drop of allergen extract (or fresh food in some cases) is placed on the skin. The skin is then gently pricked or scratched to allow a small amount of the extract to enter the epidermis. If the patient has IgE antibodies to that allergen, a wheal and flare response is seen. It is essential there is a positive (usually histamine) and a negative (saline) control to allow interpretation of the results of skin testing.

Skin testing for IgE-mediated food allergy has acquired a poor reputation. Most positive skin tests (and low-level specific IgE tests) reflect the presence of IgE antibodies (sensitisation) but not necessarily clinical allergy. Many positive skin tests cannot be confirmed by double-blind, placebo-controlled food challenges. In contrast, false-negative skin test results for IgE-mediated food allergy are rare, if performed appropriately.

Therefore, the positive predictive value of skin testing is lower but the negative predictive value is high. This underscores the importance of skilled performance and interpretation of food allergy test results. Inappropriate restrictive diets based purely on skin test results (or low levels of specific IgE to foods) can result in nutritional adverse consequences.

It is also important that medications blocking the histamine H_1 receptor are stopped at least three days before skin testing. Allergy testing (skin testing and specific IgE) must not be undertaken within one month of a severe allergic reaction, as a false negative result may occur.

Technical issues and nationwide variability

Although deceptively simple, there are major technical issues with performing and interpreting skin tests. Reproducibility is poor in some studies. In New Zealand, skin testing reagents are not registered by Medsafe and are imported under Section 29 of the Medicines Act. LabPlus at Auckland Hospital routinely obtains patients' written consent for skin tests. In the event of a serious adverse reaction, ACC would probably cover the event.

In many parts of New Zealand, there are problems with skin testing. In some areas, community testing for food allergy is not available. Some laboratories refuse to test young infants and some report results in a semi-quantitative scale (+ to ++++), which cannot be interpreted.

There is also a small but significant risk of a systemic allergic reaction to skin testing. Skin testing should be undertaken where there is access to immediate medical assistance. As a result, many private labs do not offer skin testing for peanut. Testing for common food allergens, such as tree nuts or sesame, is unavailable through most community laboratories.

There is also variation in interpreting results of skin testing. In children, a 2mm wheal may be significant in comparison with 3mm one for adults. Pressing on the wheal with a ruler may give a different result from using calipers. There is currently no training programme for technologists or nurses undertaking skin testing.

Quality control is another major issue. Deterioration of skin testing reagents can be difficult to detect and may result in false-negative results, placing the patient at risk of an allergic reaction through misdiagnosis. Skin testing is not accredited by IANZ, the New Zealand laboratory testing programme, and there is no external or internal proficiency programme.

Specific IgE blood testing

Specific IgE testing (formerly known as RAST testing) is an in vitro method for identifying IgE antibodies against food (and other) allergens. Collected blood is sent to a reference laboratory for quantitation of specific IgE antibodies. Specific IgE testing is more expensive than skin testing. In many circumstances, specific IgE testing (especially the new component testing) has significant advantages over skin testing for food allergy, for example:

- in very young children
- in combative or extremely distressed children
- in the presence of dermatographia, which can lead to uninterpretable results as all the tests (including saline control) may react
- where antihistamines cannot be discontinued
- where there is no skin testing service in the area
- in severe dermatitis with little skin left unaffected
- where there is a small risk of systemic reaction with skin testing, especially with fresh foods
- in pregnancy, which is a relative contraindication to skin testing because of the small risk of systemic reaction.

Studies undertaken by Professor Hugh Sampson have shown – for egg, milk, peanut and fish allergy – specific IgE levels measured on the Phadia ImmunoCAP system appear to correlate with results of double-blind, placebo-controlled food challenge (Table 3). Other studies have suggested some variation in the exact thresholds for predicting reactivity in such challenges.

There have been major advances in specific IgE testing since 2010. Tests of specific food allergen components (eg, Ara h 2 from peanuts) are superior to skin testing and specific IgE to peanuts. Testing for ovomucoid may predict whether a child is likely to pass a baked egg challenge. Similarly, component testing for soy and wheat allergy appears to be superior to skin testing.

It is likely that some of the latest tests on the new Immuno Solid-phase Allergy Chip (ISAC) platform will result in further improvements in diagnostic accuracy for food allergy. The emerging evidence is they may substantially reduce the number of food challenges performed and, therefore, contribute to patient safety.

Quality control is superior for specific IgE testing. There are external proficiency programmes for specific IgE testing. Internal controls are also routinely included. This reduces the risk of a false-negative result. All laboratories in New Zealand use the same instrument, allowing for comparison of results. Results are highly reproducible when repeated on the same sample or when exchanged with other laboratories. Results are comparable for patients moving cities or countries.

On the other hand, the range of commercial allergens for skin testing is greater than for specific IgE testing. Similarly, skin testing is superior in testing for the oral allergy syndrome (see later), where fresh fruit and vegetables are used for the test. The labile allergens in fruits and vegetables may degrade during commercial preparation. Generally, testing with fresh fruits and vegetables is undertaken in the specialist or hospital setting. It is thus essential for both modalities of testing to be available depending on the clinical circumstances.

It is important to be aware that the level of specific IgE to foods alone does not predict the potential severity of a clinical reaction. The severity is likely to be determined by multiple factors, including:

- the level of specific IgE antibodies
- the amount of food consumed
- the rate of absorption of food
- co-factors such as exercise or alcohol, and
- other factors, including the presence of asthma.

We have shown that the allergenicity of the food may also be an important factor in determining the severity of a reaction.

The food-specific IgE level can be monitored every six to 12 months to help identify patients who may be in the process of achieving remission. Younger children should be tested every six months, while children and adults with high IgE levels can be tested yearly. Current data suggest 20 to 30 per cent of patients with a true peanut allergy undergo remission.

When the specific IgE decreases to a low level, a food challenge can be undertaken to confirm remission. Challenges are performed where there are facilities to resuscitate the patient. There are data to suggest successful challenge and regular consumption of baked egg and milk products can speed recovery from these allergies.

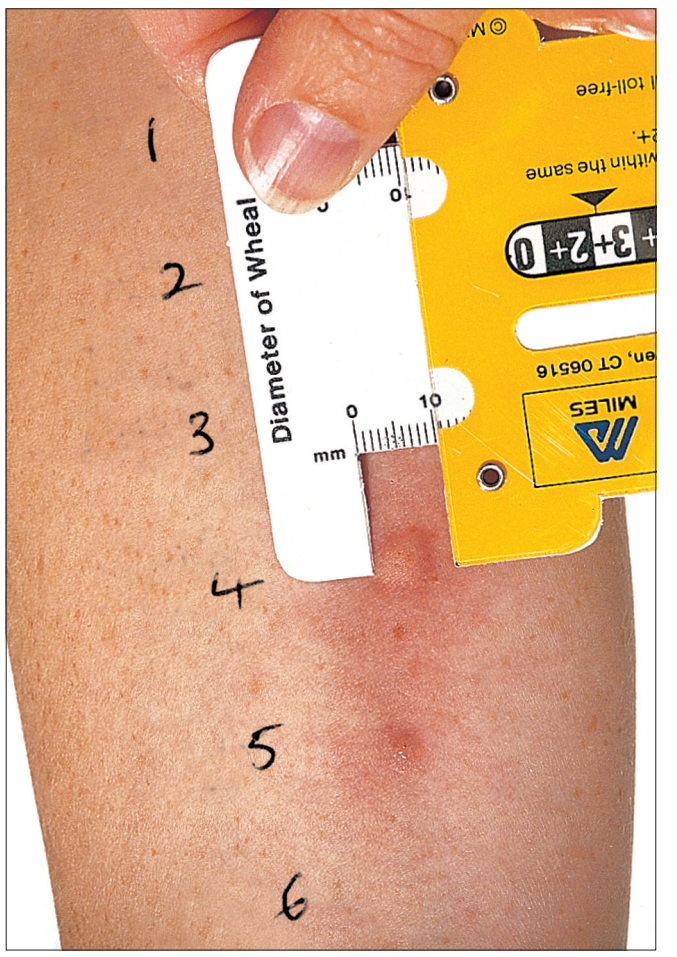


Figure 3. Skin prick tests – positive (histamine) and negative (saline) controls are placed on the skin in addition to the allergens

Specific IgE: correlation with positive food challenge
(Table 3)

Allergen	IgE (KIU)	Positive predictive value
Egg	7	98
– infant < 2 years	2	95
Milk	15	95
– infant < 2 years	5	95
Peanut	14	100
Fish	20	100
Tree nuts	15	95
Soy	30	73
Wheat	26	74

Food-specific IgE level correlates with the probability of a positive double-blind, placebo-controlled food challenge. The threshold values indicate 95 per cent of children who have a food-specific IgE level greater than this value will have a positive challenge. The thresholds vary for each food. The correlation between wheat and soy-specific IgE is not as close as for milk, eggs, peanuts and fish. (Information derived from children presenting to the Johns Hopkins Children's Center with suspected food allergy and eczema.)

Other diagnostic methods for identifying food allergy

If a specific food is thought to contribute to a food allergy, short-term elimination diets can be helpful. The food is eliminated for a period of two to three weeks. A substitute can be used. Where cow's milk allergy is suspected, a substitute such as soy could be used in an older child or a hypoallergenic formula in a younger child.

Food challenges can sometimes be helpful in children with food allergy. There are several options, including open food challenges and double-blind, placebo-controlled challenges (the gold standard for food allergy diagnosis). Food challenges for diagnosis are not undertaken if there is a history of anaphylaxis, and they should generally be undertaken in a specialist or hospital setting with facilities to treat a severe reaction.

Patients with elevated food-specific IgE tests but no history of exposure may need food challenges to distinguish between food allergy and sensitisation. This is a common problem in children with eczema.

Conditions linked with food allergy

Eczema

There is strong link between eczema and food allergy. The work of Professor Hugh Sampson has clearly shown up to 40 per cent of infants with eczema have allergies as a significant trigger. In many cases, the link might not be obvious, as even a small amount of the allergenic food may be sufficient to trigger eczema. Furthermore, there may be a time lag between exposure and the development of eczema.

However, it should be noted, in many of these children, other trigger factors are also present (Figure 4). Therefore, failure to address these triggers results in suboptimal control of eczema. This may explain the controversy surrounding the role of food allergy as a trigger for eczema, between dermatology and allergy specialists.

Respiratory tract symptoms

Large cohort studies suggest a significant proportion of children with food allergy develop asthma. Up to 80 per cent of children with egg allergy develop asthma. Often, food-allergic children who are destined to develop asthma experience an improvement in their eczema at the time they develop respiratory tract allergies. This is sometimes called the "allergic march" and is a reflection of the inherited tendency to react to common food and environmental allergens.

Asthma and rhinitis symptoms, in contrast, are unusual symptoms of food allergy. In a small number of very young children, respiratory symptoms can be triggered by food allergy. In most children, however, other manifestations are present (eg, eczema) in addition to respiratory symptoms.

Oral allergy syndrome

A recently recognised form of food allergy is the oral allergy (food-pollen) syndrome. As indicated, there is cross-reactivity between IgE antibodies to pollens (most commonly birch pollen) and allergens of fresh fruits and vegetables.

Patients suffer itching and irritation of the oral mucosa after consuming fresh fruits and vegetables.

Testing for the oral allergy syndrome requires skin prick testing with fresh fruits and vegetables. Standard laboratory reagents for skin testing and specific IgE testing are less helpful as the allergens triggering the oral allergy syndrome are labile. In most, but not all, cases the oral allergy syndrome is not associated with systemic allergic reactions. Oral allergy syndrome is more common in the South Island, reflecting a higher prevalence of birch pollen allergy.

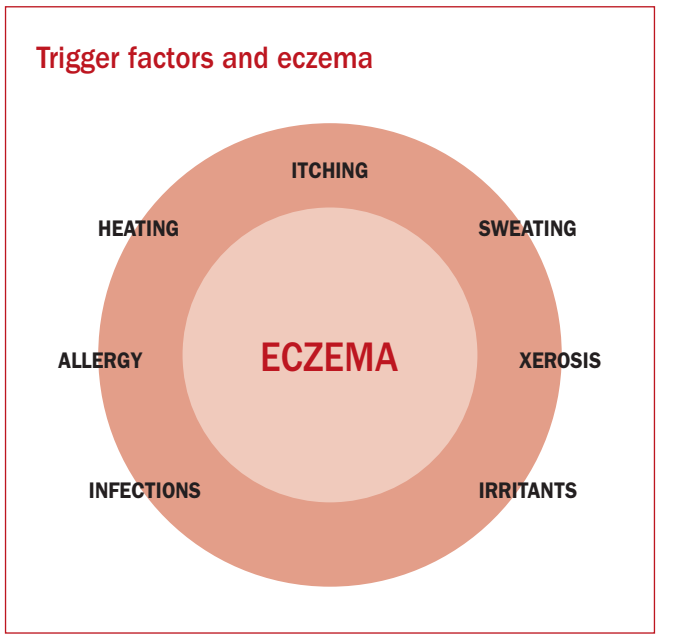


Figure 4. Eczema can be triggered by multiple factors including food allergy

Unusual manifestations of food allergy

Food allergy can manifest in more unusual ways, for example, in aggravating gastro-oesophageal reflux. A careful history can help identify such children. Rarer manifestations of food allergy including eosinophilic oesophagitis and colitis, among others, which are not discussed here. If there is concern a child or adult has an unusual manifestation of food allergy, the patient should be referred to an allergy specialist.

Managing food allergy requires plan

Anaphylaxis

Anaphylaxis is the most frightening manifestation of food (and other) allergy. Foods commonly responsible include peanuts, tree nuts, fish and shellfish, but any food can potentially cause anaphylaxis.

Any patient who has previously had anaphylaxis should have access to an adrenaline autoinjector (eg, EpiPen, EpiPen Jr, Anapen 300 or Anapen 150). These are not funded in New Zealand. Older children and adults should have a MedicAlert emblem and an anaphylaxis action plan (Figure 5); these are available online at www.allergy.org.au

There are several plans depending on the clinical situation and type of autoinjector device used. An ACC form should also be completed if the trigger can be identified.

Teachers and caregivers should be familiar with managing a severe reaction. Public health nurses are able to visit schools to teach how to manage this.

A DVD for families and caregivers on managing anaphylaxis can be either borrowed or purchased from Allergy New Zealand. Internet-based training is also available on the Australasian Society of Clinical Immunology and Allergy (ASCIA) website at www.allergy.org.au

Factors associated with food allergy fatalities

In the US, at least 125 people die each year from food allergy. This is an underestimate, as many cases likely go unreported. Often, the cause of death can be recorded as asthma with the antecedent food reaction unreported. In Australia, there were several fatalities in New South Wales. As a result, EpiPen was funded by the federal government.

The cost of an adrenaline autoinjector is a significant barrier for many patients and parents of highly allergic children

The cost of an adrenaline autoinjector is a significant barrier for many patients and parents of highly allergic children. Whole or part subsidy is a high priority for Pharmac. A syringe and vial of adrenaline is not a safe option for most patients. Studies have shown the difficulty in drawing the correct dose of adrenaline and giving a deep intramuscular injection of adrenaline.

Careful study of patients who died from food allergy suggests patient-related as well as food-related factors are important. Peanuts, tree nuts, fish and shellfish are associated with most deaths. Any food, however, may be associated with a fatal reaction. The mode of death appears to be intractable asthma in most cases. In most but not all cases, there was a delay in administering adrenaline.

The risk of dying from food allergy is highest during adolescence, with its increased risk-taking behaviour. In other fatal cases, there was poor understanding of food allergy and these patients did not have an anaphylaxis action plan. Deaths from food allergy are very rare in infants.

Studies from the UK have emphasised the importance of lying flat during anaphylaxis. Fatalities have been recorded in standing or sitting patients as a result of poor venous return and peripheral pooling of blood. Patients who have experienced anaphylaxis should be observed for at least six hours before discharge.

QUIZ ANSWERS

1. True. **2.** True. **3.** False. **4.** False. **5.** True. **6.** False. **7.** True

Avoidance a central part of management

Currently, there is no safe, widely available treatment for food allergy apart from avoidance. It is also essential for an accurate diagnosis of food allergy to be made. Even highly allergic children are not likely to be allergic to more than three or four foods. An overly restrictive diet runs the risk of nutritional complications.

Strict elimination diets carry nutritional risks and should be supervised by an experienced dietitian. There are many issues, including alternative naming of food allergens as well as undeclared allergens in foods. Allergy New Zealand offers an email alert service to its members of inadvertent contaminations of foods at the time of manufacture.

The Australia and New Zealand Food Safety Authority is responsible for implementing new food labelling laws. Defensive labelling of foods with "may contain traces of peanuts" is a problem for families.

This is a difficult area and a dietitian interested in food allergy is of great assistance to families. The dietitian plays a key role in ensuring the child eats a nutritionally sound diet and avoids foods he or she is allergic to.

It is important for the child to be evaluated periodically to determine if he or she has undergone remission. Growth parameters need to be closely monitored to ensure no nutritional sequelae arise from the diet.

Allergy prevention advice remains controversial

High-risk children can be identified from their family history of atopy. The European Academy of Allergy and Clinical Immunology and the American Academy of Pediatrics (AAP) allergy prevention guidelines differ slightly. For obvious ethical reasons, clinical research in this area is difficult.

Current evidence suggests the risk of sensitisation appears to be minimal during pregnancy. Food allergens from the maternal diet do cross into breast milk.

The AAP previously recommended (in high-risk children) introducing:

- wheat and soy after age one year
- eggs at two years, and
- fish and nuts at three years.

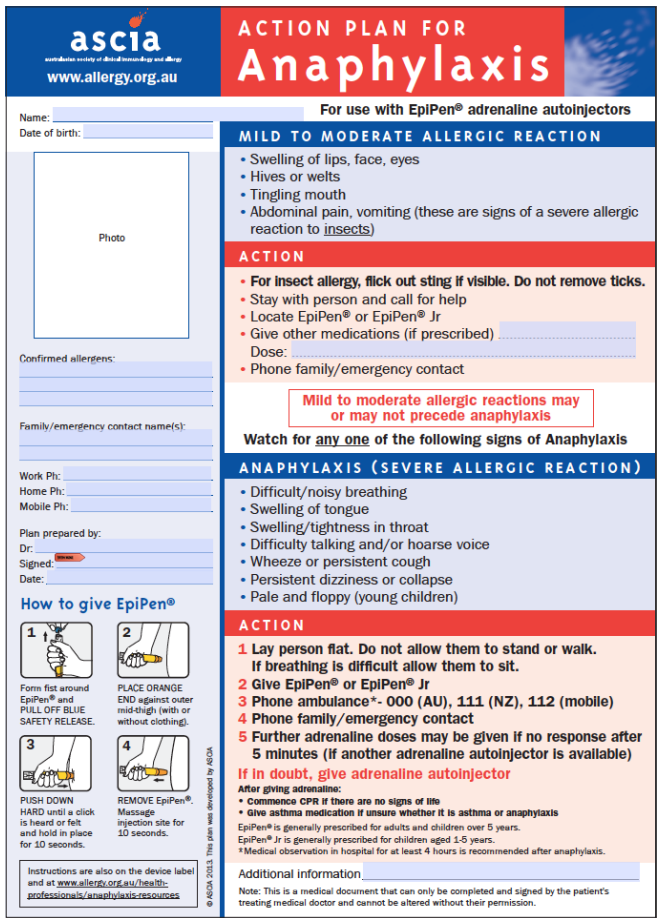
Mothers choosing an elimination diet should be under the care of an experienced dietitian. High-risk children should be exclusively breastfed for at least six months. The European academy recommends using a hypoallergenic formula for high-risk children whose mothers cannot breastfeed. Currently, in New Zealand, these formulae are only subsidised for children who have already developed a food allergy. For most families, the cost is prohibitive.

The future for food allergy management

There is currently exciting research under way in the treatment of food allergy. Recombinant antibodies have been used to block IgE responses. The first study using talizumab (TNX 901) was very successful, with the majority of peanut allergic patients being able tolerate up to eight peanuts without reacting. A trial of omalizumab was discontinued after severe adverse reactions. Other approaches include genetic modification of foods to alter their allergenicity.

It is likely that effective forms of treatment for many forms of food allergy will become available within the next decade.

Peanut desensitisation has been successfully undertaken the UK and the US. This is regarded as a research procedure and is likely to carry serious risks. There are unanswered questions about the effect of missing doses as well as sudden exercise during the course of treatment. Similarly, milk and egg desensitisation may be successful in a minority of allergic children in the research setting.


ascia
www.allergy.org.au

ACTION PLAN FOR
Anaphylaxis

For use with EpiPen® adrenaline autoinjectors

Name:
Date of birth:

Photo

Confirmed allergens:

Family/emergency contact name(s):

Work Ph:
Home Ph:
Mobile Ph:

Plan prepared by:
Dr:
Signed:
Date:

How to give EpiPen®

1 Form fist around EpiPen® and PULL OFF BLUE SAFETY RELEASE.
2 PLACE ORANGE END against outer mid-thigh (with or without clothing).
3 PUSH DOWN HARD until a click is heard or felt and hold in place for 10 seconds.
4 REMOVE EpiPen®. Massage injection site for 10 seconds.

Instructions are also on the device label and at www.allergy.org.au/health-professionals/anaphylaxis-resources

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MILD TO MODERATE ALLERGIC REACTION

- Swelling of lips, face, eyes
- Hives or welts
- Tingling mouth
- Abdominal pain, vomiting (these are signs of a severe allergic reaction to insects)

ACTION

- **For insect allergy, flick out sting if visible. Do not remove ticks.**
- Stay with person and call for help
- Locate EpiPen® or EpiPen® Jr
- Give other medications (if prescribed) Dose:
- Phone family/emergency contact

Mild to moderate allergic reactions may or may not precede anaphylaxis

Watch for any one of the following signs of Anaphylaxis

ANAPHYLAXIS (SEVERE ALLERGIC REACTION)

- Difficult/noisy breathing
- Swelling of tongue
- Swelling/tightness in throat
- Difficulty talking and/or hoarse voice
- Wheeze or persistent cough
- Persistent dizziness or collapse
- Pale and floppy (young children)

ACTION

1 **Lay person flat. Do not allow them to stand or walk. If breathing is difficult allow them to sit.**
2 **Give EpiPen® or EpiPen® Jr**
3 **Phone ambulance*- 000 (AU), 111 (NZ), 112 (mobile)**
4 **Phone family/emergency contact**
5 **Further adrenaline doses may be given if no response after 5 minutes (if another adrenaline autoinjector is available)**

If in doubt, give adrenaline autoinjector

After giving adrenaline:
- **Commence CPR if there are no signs of life**
- **Give asthma medication if unsure whether it is asthma or anaphylaxis**

EpiPen® is generally prescribed for adults and children over 5 years.
EpiPen®Jr is generally prescribed for children aged 1-5 years.
*Medical observation in hospital for at least 4 hours is recommended after anaphylaxis.

Additional information

Note: This is a medical document that can only be completed and signed by the patient's treating medical doctor and cannot be altered without their permission.

Figure 5. Action plan for anaphylaxis for use with EpiPen or EpiPen Jr

Further reading

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Full list of references available on request

Conclusions

Food allergy is becoming increasingly common. This creates a significant problem because of the shortage of allergy specialists in New Zealand. Clinical history and testing play a critical role in identifying these children. Any child with identified food allergy should consult a paediatrician or allergy specialist. Dietitians play an important role in the management of these patients. Children with multiple, severe food allergies should be under the care of team specialising in food allergy. **D**