



# Community Paediatric Review

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### Editorial Note:

Suggestions about parent information about food allergy can be accessed from

[www.rch.org.au/ccch/pub](http://www.rch.org.au/ccch/pub)

## Food allergy

### Definition

There has been much confusion in recent years regarding the terminology used to describe allergic reactions particularly food reactions. The World Allergy Organisation Nomenclature Review Committee have proposed the following nomenclature. The term food hypersensitivity is used to describe any adverse reaction to a food and encompasses the two distinctly different pathologies of:

1. Food Allergy. **Food Allergy** is defined as a reaction that is due to an immunological mechanism and can be further characterised as IgE (allergy antibody) mediated or non-IgE mediated.
2. Food Intolerance. **Food Intolerance** in contrast is a food hypersensitivity that is not immunologically mediated. It may occur as a result of factors present in the food such as toxins (eg, histamine in scombroid fish poisoning) or pharmacologic (eg, tyramine in cheeses or red wine). These reactions can occur in most healthy subjects when given in high enough doses. Alternatively food intolerance may occur as a result of a metabolic disorder (eg, lactase deficiency resulting in lactose intolerance). Food intolerance will not be discussed further in this article.

### Prevalence

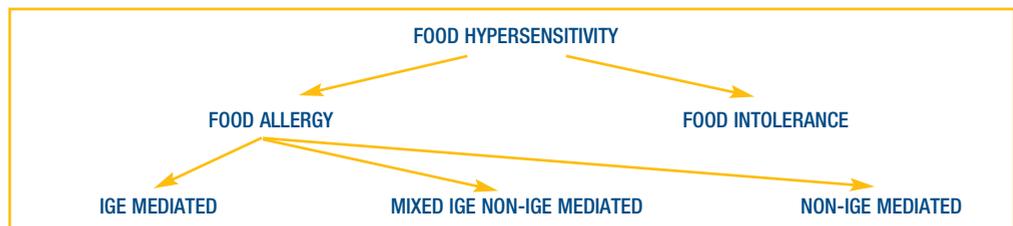
Food allergy is more common in children than adults with the incidence increasing in recent decades.

IgE mediated food allergy affects about 6% of children under 3 years of age and about 2% of adults. Children with allergic diseases such as eczema or asthma are at greater risk of having food allergy. More than 95% of food allergic reactions occur to 8 foods. In children the common food allergens are egg, milk, peanut, wheat, soy, and fish. Most children 'outgrow' their allergy to egg, milk, wheat and soy by the age of 5 yrs however nut and seafood allergy is ongoing into adult life in up to 80%. Thus in adults most food allergic reactions are due to peanut, tree nuts, fish and shellfish.

### Clinical presentation

#### IgE mediated food allergy

The diagnosis of IgE mediated food allergy can usually be made on the description of presenting symptoms obtained from a detailed history. The onset of the reaction occurs typically within 30 minutes of ingestion of the offending allergen and may involve cutaneous (erythema, urticaria, angioedema), gastrointestinal (vomiting, diarrhoea, abdominal pain), respiratory (cough, hoarse voice, stridor,



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wheeze) and/or cardiovascular (hypotension/collapse) systems. When the reaction involves the respiratory and/or cardiovascular systems it is termed 'Anaphylaxis'.

### *Non IgE mediated food allergy*

Under this heading there are a number of distinct syndromes that typically have a delayed (hours to days) presentation following food exposure:

- *Food protein induced enterocolitis syndrome*  
A disorder of young infants typically due to cow or soy milk but also seen in older infants due to other foods such as rice cereal. Patients present with delayed onset (1– 3 hrs after allergen ingestion) of protracted vomiting that frequently becomes bilious. Hypotension occurs in up to 15% of cases typically manifesting as pallor and floppiness with these patients not infrequently misdiagnosed as having overwhelming sepsis. It is not uncommon for these patients to have several episodes before the food trigger is identified.
- *Food protein induced enteropathy*  
Encompasses a spectrum of disorders that may present in the first months of life with diarrhoea, vomiting and failure to thrive. Most commonly due to cow milk protein but can also be secondary to soy, egg, wheat, rice, chicken and fish.

### *Mixed IgE and non-IgE mediated food allergy*

Up to half of these patients with allergic eosinophilic syndromes have other allergic disease.

- *Allergic eosinophilic oesophagitis*  
Presents in infancy through to adolescence with symptoms of chronic gastroesophageal reflux that fails to respond to antireflux therapy. These patients exhibit vomiting, food refusal, abdominal pain, and irritability.
- *Allergic eosinophilic gastritis*  
Can present from infancy through to adolescence. These patients present with post-prandial symptoms including nausea, vomiting, abdominal pain plus food refusal and in more severe cases symptoms of gastric outlet obstruction
- *Allergic eosinophilic gastroenteritis*  
Can present at any age with failure to thrive or weight loss plus symptoms of oesophagitis or gastritis.
- *Eosinophilic proctocolitis*  
Presents in the first few months of life due to the passage of food proteins passed in maternal breast milk or due to cow milk or soy milk formulas.

Affected infants present with gross or microscopic blood in their stools but are otherwise well and thriving.

## **Diagnosis**

### *IgE mediated food allergy*

The presence of food specific IgE antibodies can be detected by skin prick test (SPT) or blood test (RAST – Radioallergosorbent test) where allergen specific IgE antibodies can be quantified in the skin or blood, respectively. Skin prick testing is simple, rapid and inexpensive. Skin prick testing should only be undertaken by practitioners experienced with the methodology and interpretation of the results as false positive results are relatively common. The negative predictive value however is excellent thus enabling near exclusion of significant immediate hypersensitivity to such foods in the event of a negative SPT.

RAST testing is more expensive with a limitation on the number of allergens that can be ordered at any one time. Results of testing can take up to a week to become available.

The definitive diagnosis of food allergy is made by eliciting an immediate reaction on graded food challenge. This should not be performed at home if there is a suspicion of IgE mediated food allergy.

A number of techniques for the diagnosis of food allergy have been adopted by alternative practitioners eg.vega (electrodermal) testing, *cytotoxicity testing, the provocation neutralisation procedure, applied kinesiology, reagenic pulse testing* and *chemical analysis of body tissues, such as hair analysis*. These techniques have no scientific basis, are expensive and can lead to inappropriate dietary restriction.

### *Non IgE mediated food allergy*

There are no specific diagnostic tests available for non IgE mediated food hypersensitivity syndromes. Thus an elimination diet followed by food challenge is the only means of confirming diagnosis of these intestinal syndromes. It is important that any elimination diet be supervised by practitioners with appropriate expertise in the field and to ensure nutritional adequacy is maintained.

In the mixed IgE/non IgE syndromes SPT may be of some help. Endoscopy and biopsies can support the diagnoses of the various intestinal syndromes.

## Management

There is no cure for food allergy. The only proven therapy available is strict avoidance of the implicated food allergen(s). For this to be done effectively patients and their families need to be educated to read food labels and to identify alternative terminologies for any given food allergen. The involvement of a dietician can be invaluable to assist with this.

Patients with IgE mediated food hypersensitivity should also be provided with a management plan in the event of an immediate reaction following inadvertent exposure to an offending food allergen. For some patients this may include the prescription of an automated injectable adrenaline device (Epipen®/Epipen junior®). This requires assessment by a Paediatrician, Allergist/Immunologist or a Respiratory Physician and must include appropriate education and a written anaphylaxis management plan.

## Follow-up

Most food allergies (with the exception of nuts and shellfish) resolve with time, patients should therefore be reviewed regularly to monitor for the development of tolerance and to review emergency management plans.

It is also important that the nutritional adequacy of each patient's diet is assessed with nutritional supplements provided as required eg. the addition of a calcium supplement over 12 months in patients avoiding cow milk.

These patients should also receive their routine childhood immunisations as scheduled. Measles Mumps Rubella immunisation is safe in children with significant egg allergy however influenza vaccination may be contraindicated.

## Prevention of food allergy

For high risk infants (ie: where there is a significant family history of allergy) the current recommendation is that exclusive breast feeding be continued for at least the first 4-6 months. Maternal dietary restriction during breastfeeding is not recommended for disease prevention. If formula feeding is necessary a hydrolysed formula (where milk proteins have been broken down) is recommended in high risk infants. Partially hydrolysed formula (eg NAN HA®) is available without prescription however extensively hydrolysed formula (eg Peptijunior®,

Alfare®) is only available on authority prescription with restricted use for infants with proven cow's milk and soy allergy. Although there is no evidence regarding the timing for introduction of solid foods, it is often recommended to delay the introduction of solid foods until after 6 months and to avoid nuts and shellfish until after 3-4 years in high risk infants.

## Common misconceptions

Behavioural changes such as hyperactivity are commonly ascribed to food allergy but proof of causation is lacking.

There is no evidence that milk increases mucous production and elimination of milk and wheat is only of benefit in patients who have confirmed allergy to these allergens.

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## Reflection Questions

- 1. Would you be able to explain to a parent the difference between food allergy and intolerance?*
- 2. If a parent told you that their child was allergic to milk, how would you react? What information would you give to them?*
- 3. Do you have established relationships with a range of professionals to assist families in addressing their concerns about food allergies?*
- 4. How would you encourage parents to have their child's food allergy reviewed?*

## Headlice update

With the current increase in headlice infestation, the following general advice may support your discussion with parents.

Myths surrounding headlice are many and varied:

Myth	Fact
Children with lice scratch or itch.	Only 50% display any itching.
Lice live in carpets, beds, clothes and school buses.	Lice cannot live and lay eggs away from a warm human scalp.
One treatment is enough. If dead lice are found the product has worked.	Re-treatment is required in 7-10 days to catch nymphs (eggs) that have emerged from unhatched eggs not affected by the treatment as no product has been shown to kill eggs.
Everyone in the family should be treated.	By treating family members all together whether they are infected or not you may be contributing to the problem of the lice developing resistance to a chemical treatment. Family members should only be treated if infected to prevent cross infection.
Some products prevent head lice.	There is no proven preventative treatments available.

There are two broad treatments options available:

### 1. Insecticide products

Head lice insecticide products fall into four groups on the basis of their active ingredient:

1. Pyrethrins
2. Synthetic pyrethroids (permethrin, bioallethrin)
3. Organophosphates (maldison or malathion)
4. Herbal products

Parents should be reminded to check the products for registration with the Therapeutic Goods Administration – there will be a Aust L or Aust R number on the product. It is also important to check for any age restrictions (not recommended for children under 2 years), or cautionary notes for pregnant/breastfeeding women, or other allergies.

### 2. Wet combing

- Comb hair conditioner on to dry, brushed hair – this stuns the lice for 20 minutes and makes it difficult for them to grip the hair or crawl around.
- Comb sections of the hair with a fine toothed, head lice comb, starting at the root and working to the tip of the hair.

- Wipe the conditioner from the comb onto a tissue. Look at tissue and comb for lice and eggs.
- Repeat the combing for every part of the head at least 5 times.

There are several reasons for treatment failures including:

1. **Inadequate application of product** – is the product being applied correctly? Some treatments need to be left on for at least 8 hours, while some only need a few minutes
2. **Lice are resistant to the insecticide/product used** – are the head lice sensitive to the product being used? If so use product from another group. In Australia resistance to maldison has been reported (Goldsmid JM. Head louse treatment: is there an insecticide resistance problem? [letter]. Med J Aust 1990;153:233-4.
3. **Failure to re-treat to kill nymphs newly emerged from eggs** – has a complete treatment regime of at least 2 applications (7-10 days apart) been given?
4. **Reinfection** – is reinfection occurring from school or family contacts?

Further information and fact sheets can be accessed from all state government websites.



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