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CLINICAL PERSPECTIVES

Current perspectives on peanut allergy

Katie Frith ¹ and Constance H. Katelaris ²

¹Department of Immunology and Infectious Diseases, Sydney Children's Hospital and University of New South Wales, and ²Department of Immunology, Campbelltown Hospital and Western Sydney University, Sydney, New South Wales, Australia

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Correspondence

Katie Frith, Department of Immunology and Infectious Diseases, Sydney Children's Hospital, High Street, Randwick, NSW 2031, Australia.
 Email: catherine.frith@health.nsw.gov.au

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Abbreviations: Ara, *Arachis hypogaea*; CRD, component resolved diagnostics; DBPCFC, double-blind, placebo-controlled food challenges; FAIT, food allergen immunotherapy; IFN, interferon; IL, interleukin; nsLTP, non-specific lipid transfer proteins; OFC, oral food challenges; PAL, precautionary advisory labelling; PN-IgE, peanut-specific IgE antibodies; SPT, skin prick testing.

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Abstract

Peanut allergy is increasingly prevalent and for most patients is a life-long condition, with the potential to cause life-threatening reactions. Accurate diagnosis and appropriate management are essential to minimise risks due to accidental peanut exposure. Current management strategies focus on strict allergen avoidance and access to emergency medicines to treat potential reactions; however, active approaches are an area of intense research. Promising new methods of food allergen immunotherapy are set to change the approach to managing peanut allergic patients in the near future.

Introduction

Food allergies have become a significant problem in modern life, leading to large negative impacts on quality of life for those affected as well as for their families.¹ The threat of life-threatening reactions and high healthcare costs add to the overall burden.

The rise in food allergy is often referred to as the 'second wave' of the allergy epidemic as it follows the previous increase in asthma and allergic rhinitis that first started more than 50 years ago.¹

The causes for increasing rates of food allergy remain unclear, although some aspects of the modern, Western lifestyle are believed to be important contributors to the increase. The 'old friends' hypothesis proposes that a balanced immune system requires exposure to a diverse range of environmental and commensal microbes that were present during the evolution of the human immune system. Exposure to these microbes is thought to activate immunoregulatory mechanisms that reduce the risk of the inappropriate immune reactions that characterise allergic disease. Modern lifestyle practices are thought to disrupt these microbes through various mechanisms including dietary changes, early use of antibiotics and increasing rates of Caesarean births.¹

Egg, cow's milk and peanut are among the foods most commonly implicated in food allergic reactions in infants and young children. In most studies, estimates of prevalence of various food allergies have been based on positive skin or *in vitro* tests that indicate sensitisation, with or without a suggestive history, and rarely on a standardised food challenge, explaining the large heterogeneity seen in epidemiological studies. The Health Nuts study performed in Melbourne is one of the very few studies where young children were screened for food allergy by skin prick testing (SPT) and supervised food challenges (the gold standard for diagnosis of clinical allergy rather than sensitisation).² A very high rate of challenge-proven food allergy was described with 10.4% of participating children allergic to egg, peanut or sesame.² Although rates of anaphylaxis to foods are increasing, those of fatal anaphylaxis appear to be stable in most countries.^{3–6} However, Australia reported a significant increase, of 9.7% per year, in fatal food anaphylaxis between 1997 and 2013.⁶ In this cohort, peanut was the second most common cause of fatal food anaphylaxis, after seafood, and the most common cause in patients <20 years.⁶

This study will focus on the problems associated specifically with IgE-mediated peanut allergy because of its prevalence, persistence and importance as a cause of serious allergic reactions.

The average age at presentation of peanut allergy is 18 months, although peanut allergy can present later in childhood or adulthood, either as a primary food allergy or as part of the pollen-food allergy syndrome.⁷ The prevalence of childhood peanut allergy is estimated at around 1.4–5% with variability related to geography.⁸ Peanut allergy is unevenly distributed, and one of the possible explanations for the low prevalence of peanut allergy in some countries such as Israel, may be early

introduction of peanuts into the infants' diet. However, there are regions of the world such as Siberia where rates of peanut and tree nut allergy are low yet there is no early introduction of peanut into the infant diet. Such observations suggest that while timing of introduction is important, it is not the only factor influencing food allergy development.

Peanut allergy has different clinical and immunologic patterns in different areas of the world, possibly because of differing environmental pollen exposures and culinary traditions of the populations. There is evidence to suggest that allergenicity is affected by different modes of processing (see below). Peanut-allergic individuals may react to different proteins within the peanut and these differences can account for differing clinical patterns of reactivity.

Peanut allergens

Peanut (*Arachis hypogaea*) belongs to the legume family (Leguminosae). Peanuts are most commonly grown in China, followed by India and USA. In many parts of the world such as Europe or USA, peanuts are primarily consumed in roasted form. They might be eaten as whole peanuts, peanut butter or as an ingredient in many products. Peanuts can be roasted in the shell or they are shelled, blanched, oven roasted either dry or in oil, and ground for the production of peanut butter. In other parts of the world such as Asia and Africa, raw peanuts are used more commonly as a cooking ingredient. Peanuts have a high protein content of 24–29% and contain various allergens. Allergenicity may depend on methods of processing; roasting at high temperatures appears to promote the formation of compact globular protein aggregates that can increase the allergenicity of the major allergens, Ara h 1 and 2, whereas boiling may reduce allergenicity.

To date, 17 peanut allergens have been identified. Many of these proteins have protective functions or are seed storage proteins. Peanut allergens belong to diverse protein families leading to IgE-mediated cross-reactions among different members of the legume families but also other plant foods such as tree nuts.⁹

Three peanut-specific storage proteins (Ara h 1, 2, 3) and two cross-reactive proteins (Ara h 8 and 9) are used in diagnostic testing. The storage protein Ara h 2 is the most useful for measuring sensitisation frequency and in eliciting clinical symptoms in peanut-allergic patients. Ara h 6 is also a major allergen and is similar to Ara h 2. They share 58% homology at the amino acid level; both are storage proteins of the 2S albumin type; both are heat stable and resistant to digestion; both are associated with the potential for severe clinical reactions. A

95% probability of a positive peanut oral food challenge (OFC) was estimated for an Ara h 2-specific IgE.¹⁰ In contrast, Ara h 8 was associated with either no symptoms or only mild oral symptoms. Several studies have demonstrated that when used together for diagnosis, Ara h 2 and Ara h 6 provide the highest diagnostic accuracy for predicting clinical peanut allergy.

Sensitisation to stable peanut allergens (Ara h 1, 2, 3, 6, 7) usually occurs early and probably through the cutaneous route, particularly in those infants with atopic dermatitis and impaired skin barrier. These patients are at greatest risk of subsequent severe immediate clinical reactions following exposure to peanut, a risk probably linked to the high stability of these allergens and their high concentration in peanut. Sensitisation to Bet v1 homologues (Ara h 8) and profilins (Ara h 5) is caused by cross-reactions to various pollen allergens. In parts of Europe where Birch pollen levels are high, sensitisation to Ara h 8 is seen whereas areas with high grass pollen levels are associated with Ara h 5 sensitisation.¹¹ These peanut proteins are heat labile and susceptible to digestion. As peanuts are generally consumed roasted or cooked, only mild, predominantly oropharyngeal symptoms are experienced. Another clinical pattern is seen with sensitisation to non-specific lipid transfer proteins (nsLTP; Ara h 9, 16, 17). Ara h 9 is stable to heat and digestion and while it is classed as a secondary food allergen particularly in Mediterranean countries, it can cause systemic symptoms in those sensitised to it.¹¹

Clinical manifestations of peanut allergy

IgE-mediated peanut allergy causes reactions of varying severity, including life threatening anaphylactic reactions. Symptoms occur rapidly, usually within 2 h of exposure, due to the release of vasoactive cytokines from mast cells and basophils (Table 1). Allergic reactions to peanut are unpredictable and may vary in severity depending on factors such as dose, route of exposure, food preparation techniques, and the presence of modifying cofactors such as asthma, infection, exercise and alcohol.

Allergic reactions to peanut occur through different routes of exposure. Contact reactions to peanut are

common and may produce local cutaneous signs, for example, urticaria in peanut-allergic individuals. Despite being a cause of significant anxiety, contact with peanut has not been shown to cause systemic reactions even in people with a history of severe reactions to peanut.¹² In infants, an isolated contact reaction is equivalent to a positive skin prick test (sensitisation), and some infants may be able to tolerate peanut; however, the decision to trial peanut should be made by a paediatric immunologist. Inhalation reactions to peanut are also a cause of anxiety but are extremely uncommon outside of occupational exposures to airborne peanut dust or flour, or aerolised peanut protein in cooking. In contrast, the smell of peanut, which releases volatile compounds but not peanut protein, does not cause allergic reactions but may trigger nausea or symptoms of anxiety that might be misinterpreted as an allergic reaction.¹³

Systemic reactions occur most frequently after ingestion. Food-induced anaphylaxis occurs most commonly in young children, with peanut being the commonest cause worldwide, along with tree nuts, cow's milk and egg.^{14,15} Fatal food anaphylaxis is reported to account for 0.03–0.3 deaths per million inhabitants yearly worldwide, with peanut being the leading causative allergen, peanut, along with tree nuts, account for 55–87% cases of fatal food anaphylaxis.⁵ Most fatal food-induced reactions occur in adolescents and young adults most likely due to increased risk taking behaviours in this age group, although a possible physiological predisposition to more severe reactions in this age group cannot be excluded.^{3,5,6} Other risk factors for fatal outcome include asthma and delayed administration of adrenaline.^{3,5,6}

Patients with peanut allergy are frequently allergic to other foods. In a cohort of challenge proven peanut allergic patients aged 4–20 years, 70% reported another IgE-mediated food allergy, most commonly to egg 39%, cow's milk 30% and tree nuts 39%.¹⁶ Most peanut-allergic patients can tolerate other legumes including soy, lupin and lentils. Although high rates of cross-sensitisation (positive SPT or *in vitro* tests) are seen with other legumes clinical allergy due to cross-reactivity with other legumes is much lower, with rates of allergies to soy and other legumes, 6% and 3%, respectively, in the same peanut-allergic cohort.¹⁶ Cross-reactivity with other legumes as well as tree nuts occurs due to similar epitopes within proteins, although cross-sensitivity has been reported with phylogenetically different species.¹⁷

In general, the majority of children become tolerant to (or outgrow) milk, egg, soy or wheat allergies, whereas peanut and tree nut allergies are typically more persistent.¹⁸ Estimates of tolerance are considered to be between 10% and 20%. Persistence is more likely if the reaction occurred with a low threshold dose eliciting the

Table 1 Signs and symptoms of an IgE-mediated allergic reaction

Mild to moderate reaction	Cutaneous: urticaria, angioedema Gastrointestinal: abdominal pain, vomiting
Severe reaction	Respiratory: wheeze, persistent cough, stridor, chest tightness, difficulty breathing, difficulty swallowing, vocal changes (hoarse voice) Cardiovascular: dizziness, collapse, pallor, floppiness/lethargy (young children)

reaction or if there were severe symptoms with a reaction. Other factors associated with poorer prognosis for tolerance development include earlier age at diagnosis; presence of other comorbid allergic diseases such as allergic rhinitis, asthma and atopic dermatitis; and larger SPT size or higher food-specific IgE levels.

Investigations

Accurate diagnosis of food allergy with correct identification of the culprit food or foods is essential because there is a significant burden on quality of life that accompanies such a diagnosis. Avoidance is difficult, and accidental exposure that results in allergic reactions is frequent; disruption to family life and social restrictions are significant.

The tools available for diagnosis include a detailed medical history, SPT, determination of serum peanut-specific IgE antibodies (PN-IgE), and medically supervised OFC. The latter are time consuming, costly in terms of resources, and are not without significant risk so ideally, non-invasive tests with high diagnostic accuracy are needed to give certainty around the need for avoidance without the risk of having to perform a challenge.

Numerous studies, almost exclusively in children, have correlated clinical outcomes against SPT and PN-IgE with informative results. The diagnostic utility of SPT and PN-IgE increases with the strength of positivity correlated with the features of the medical history. More recently, component testing, where specific IgE levels to specific individual proteins in peanut are measured, may prove useful in assisting with the decision to perform an OFC.

History

The history should include a careful assessment of the symptoms, timing in relation to peanut ingestion, consistency of symptoms, amount ingested, and eliciting cofactors such as exercise, alcohol use, or medications, and comorbid conditions.

Skin testing

Skin tests are subject to several variables including test reagent quality and potency, device used for performing the test, and method of reading/recording results. SPT and not intradermal tests are recommended because of their greater safety and specificity. Intradermal tests, while having greater sensitivity, correlate less well with OFC and carry the risk of inducing a systemic reaction.

Two important features of SPT should be noted; increasingly larger wheal sizes are correlated with increasing risk of clinical allergy and secondly, reactions

can occur in patients with a 'negative' skin test. False negative SPT are more common in infants <6 months of age.

PN-specific IgE

Similar to studies using SPT, there are variations in results on the basis of population characteristics, but increasing IgE concentrations are associated with higher risks of clinical allergy. Thus, levels of 15 kU_A/L are usually associated with clinical reactions. Importantly, reaction rates of approximately 20% have been noted among children with 'undetectable' (<0.35 kU_A/L) PN-IgE. Of note, sIgE nut mix panels are not recommended in the diagnosis of peanut allergy as they provide limited clinical information and often lead to unnecessary dietary exclusions.

Relevance of positive tests

Specific IgE testing by SPT, serum tests or both, for diagnostic purposes when food allergy is suspected, should be guided by a history suggestive of an IgE-mediated reaction focusing on the food or foods suspected of provoking the reaction. When positive, these tests provide evidence of sensitisation to a particular protein; clinical allergy can only be confirmed through an OFC. Data that further emphasise this important point comes from the NHANES study where 17% of people tested demonstrated sensitisation to a major food allergen but only 2.5% had clinical reactivity consistent with a diagnosis of food allergy.¹⁹ In the Melbourne study by Osborne *et al.*²⁰ only 33% of peanut-sensitised and 55% of egg-sensitised infants had positive responses to an OFC with the relevant food.

Component resolved diagnostics

As discussed in the previous section, peanut, like any food, is composed of many proteins. An immune response directed toward one or another protein (component of peanut) may have different implications, depending on characteristics of the protein. Specific IgE to the various proteins can now be measured (component resolved diagnostics, CRD). Sera from children with clinical peanut allergy primarily bind Ara h 2 and Ara h 6. Various studies have suggested that IgE binding to Ara h 2 is particularly useful for diagnostics.²¹

The pattern of binding to peanut proteins varies regionally, presumably on the basis of differences in environmental exposures, so it is important that the utility of CRD be examined in the different geographical regions before using the results to determine whether or not an OFC should be conducted.²²

Oral food challenge

Food challenges are used either to confirm a diagnosis of food allergy or to demonstrate acquisition of tolerance to the food. Double-blind, placebo-controlled food challenges (DBPCFC) are the gold standard for research studies but are difficult to arrange and are rarely performed in routine practice. Open or single blind challenges are far more commonly used in clinical practice and, providing objective end-points are defined for a positive challenge, are a very useful investigation. A typical OFC is performed by introducing the food in gradually increasing amounts at time intervals between 20 and 30 min, until a 'standard helping' of the food is ingested. Severe symptoms may be induced during an OFC so patients and parents must be fully informed of the risks and have a clear understanding about the reason for performing the challenge which should be conducted in a medical facility with expertise in the procedure and where staff have appropriate resuscitation skills for early recognition and management of anaphylaxis. Risk factors for severe reactions include a previous history of anaphylaxis, a high degree of sensitivity on specific IgE testing and comorbid conditions such as asthma.

Unproven techniques

Unfortunately, food allergy is an area where application of procedures with no scientific basis flourishes particularly in the hands of so called 'alternative' practitioners. Such procedures include provocation-neutralisation, applied kinesiology, vega testing, cytotoxic food testing and allergen-specific IgG₄ tests. These tests have no scientific validity but are offered at great cost to patients, bear no relationship to the real situation and must be discouraged.

Prevention

Studies have not shown that maternal dietary allergen elimination during pregnancy or breastfeeding reduces the risk of infant food allergy, and maternal dietary exclusions are not recommended.¹ Infants at high risk of peanut allergy include those with severe eczema and, or egg allergy.²³ The LEAP study established that in high risk infants, introduction of peanut into the diet before 11 months of age, and regular ingestion over the first 5 years of life significantly reduced peanut allergy, by 86% in those with negative SPT and 70% in those sensitised to peanut (SPT: 1–4 mm).²³

Management

Management of peanut allergy requires strict avoidance of peanut and readily available access to rescue

medications to treat accidental reactions. While research has demonstrated that early introduction and regular consumption of peanut can significantly reduce the risk of developing a peanut allergy in high risk infants, once an individual has developed a peanut allergy strict avoidance is usually recommended.²³ Peanut is commonly consumed in many countries and may also be an ingredient in cosmetics and emollients. Peanut is a common ingredient in many Asian and African foods, as well as being commonly found in certain foods such as confectionary, breakfast cereals/muesli and bakery goods. Peanut-allergic patients and their families need education to ensure they can identify foods containing peanut, which may be listed with alternative names, for example, arachis oil. Dietary factsheets are available and are a valuable resource (see Useful resources). Many countries, including Australia, European Union, USA and Canada have legislation requiring manufacturers to clearly label foods containing specified allergens; however, legislation does not apply to possible cross-contamination with peanut. Precautionary advisory labelling (PAL) was established in 2003 and is a voluntary way for manufacturers' to alert food-allergic patients to the possibility of cross-contamination. Almost half packaged foods in supermarkets in Australia, USA and UK contain PAL, however, studies have shown that PAL is confusing for patients and health professionals.²⁴ Also, as PAL is voluntary and not standardised it is difficult for consumers to determine the risk from foods that do not carry a PAL and anaphylaxis to undeclared allergens in foods, both with and without PAL is not rare, with the biggest risk from unlabelled foods eaten outside the home.²⁵

Emergency treatment

Despite best efforts at avoidance, accidental exposures to peanut occur mostly through contact or ingestion. Individuals with peanut allergy must therefore be adequately prepared to manage allergic reactions. Adrenaline is essential in the treatment of anaphylaxis, and should be administered early if there are signs of a severe allergic reaction. Anti-histamines alleviate IgE-mediated skin and gastrointestinal symptoms but do not treat or prevent anaphylaxis. Reactions may vary in severity due to several factors; it is a common misconception that allergic reactions always escalate in severity with subsequent reactions. Currently no test, including OFC, can predict the likelihood of anaphylaxis. Anaphylaxis risk in peanut-allergic individuals should be based on a thorough assessment including age, history of reactions, for example, frequency and severity, SPT or peanut-specific IgE level to help predict the likelihood of a reaction, the

presence of risk factors such as asthma, and social circumstances, for example, remote location, or likelihood of accidental exposure. Patients with a history of anaphylaxis, and those considered to be at high risk of anaphylaxis should be prescribed an adrenaline autoinjector. In Australia EpiPen is the only adrenaline autoinjector currently available. EpiPens are available in two sizes 150 mcg and 300 mcg. Product information recommends EpiPen 300 mcg be prescribed for patients over 30 kg, however, most immunologists would prescribe this dose for patients ≥ 20 kg, as it more closely approximates the recommended dose 0.1 mL/kg of 1:1000 adrenaline. EpiPen Junior (150 mcg) is recommended for patients weighing ≥ 10 kg. The decision to prescribe one to a child weighing < 10 kg should be made in discussion with a paediatric immunologist. Education is essential to ensure patients and their families know when and how to administer correctly the autoinjector to minimise delays in life saving treatment. Anaphylaxis action plans are a helpful resource and provide clear step by step instructions to treat a person with an allergic reaction regardless of severity (see Useful resources). Both plans and rescue medication should be readily available to the patient or carer at all times, as accidental exposures can be difficult to predict.

Immunotherapeutic approaches

In contrast to allergen avoidance food allergen immunotherapy (FAIT) offers an active approach to managing patients with food allergy. Peanut has been a major focus of FAIT research given its prevalence, persistence and importance as a cause of serious allergic reactions. Currently, general expert consensus does not recommend FAIT in clinical practice due to safety concerns and insufficient data regarding long-term efficacy. Peanut FAIT involves administering gradually increasing doses of peanut allergen over a prolonged period of between 6 and 36 months. The goal of FAIT is to increase the threshold dose of allergen needed to cause an allergic reaction, both on active treatment, termed desensitisation, and also, ideally after a period of no allergen exposure, which is referred to as tolerance, or sustained unresponsiveness. To date, there is no consensus on the safest and most effective protocol for peanut FAIT and mechanisms of tolerance are incompletely understood. Success of FAIT is assessed clinically, with DBPCFC to assess threshold dose, in conjunction with various markers of tolerance including SPT size, PN-IgE, IgG4, basophil activation, regulatory T-cell induction and cytokine production ($\downarrow$ IL4 and IL13, $\uparrow$ IL10 and $\uparrow$ IFN γ).²⁶

Various routes of administration and peanut products, as well as several immunomodulatory adjuncts, such as probiotic *Lactobacillus rhamnosus* and monoclonal antibody Omalizumab, have been investigated for their ability to improve efficacy and safety of FAIT (Table 2).^{26–32} Epicutaneous immunotherapy (EPIT) and subcutaneous immunotherapy (SCIT) have been less frequently studied, but have been shown to be effective in achieving desensitisation.^{31–33} The smaller doses of peanut used in EPIT (100–500 mcg) make it extremely safe, and therefore an option for patients whose dose threshold is too low to tolerate oral immunotherapy (OIT).^{26,32,33} Limited SCIT studies have been performed using either native or modified peanut allergen in humans, with current trials using modified hypo-allergen.^{26,31} Newer techniques such as LAMP-DNA peanut vaccine are currently under investigation in phase I/II trials. These vaccines contain DNA encoding peanut allergen and LAMP1 within bacterial plasmid vectors given by injection. DNA is translated into allergen and LAMP1 protein, which enhances immunogenicity, and elicits an immune response.²⁶

FAIT is associated with a significant risk of local and less commonly, systemic allergic reactions, and therefore should only be undertaken in experienced centres with adequate resources to administer this therapy.^{26,33,34} FAIT requires strict adherence to therapy over a prolonged period of time to achieve desensitisation, and to reduce the increased risk of reactions associated with irregular dosing. To ensure both short- and long-term patient safety, careful patient selection is paramount. Considerations include patient and family motivation and ability to deliver rescue medications, comorbidities including asthma, eosinophilic oesophagitis (EoE), chronic spontaneous urticaria and eczema.³³

Over the past decade, an increasing number of small studies has reported benefit in desensitisation to peanut from FAIT.³⁴ A recent systematic review and meta-analysis of peanut OIT in over 1000 paediatric patients confirmed OIT was effective at inducing desensitisation, but also significantly increased the likelihood and frequency of allergic reactions, including anaphylaxis and adrenaline use, compared with peanut avoidance.³⁴ There are limited data on long-term efficacy for peanut FAIT.^{28,33,34} Most FAIT data come from studies in children or mixed populations and the benefit of FAIT in adults remains unclear.³³ Given that a significant proportion of people with peanut allergy are also allergic to other foods, multiple food FAIT would be desirable, although the vast majority of trials to date have been with single foods.

Peanut immunotherapy remains an important area of active research as questions remain regarding the most

Table 2 Peanut immunotherapy: an overview of available modalities

	OIT ^{26,27,29,30,34}	SLIT ^{26,30}	SCIT ^{26,31}	EPIT ^{26,32}	LAMP-DNA vaccine ²⁶
Modality	Native or modified peanut protein ingested daily. OIT uses higher allergen doses than other forms of FAIT. Adjuncts used in some OIT trials to assess impact on safety and/or efficacy	Daily sublingual application of liquid peanut protein extract	Trials have used native or modified peanut allergen. Current trials underway using modified hypo-allergen given by subcutaneous injection weekly	Peanut protein is sprayed onto patches, which are applied daily to the skin on back or upper arm	Vaccine containing DNA encoding peanut allergen and LAMP1 within bacterial plasmid vectors given by injection. DNA is translated into allergen and LAMP1 protein, which enhances immunogenicity and elicits an immune response
Strengths	Low cost; ease of administration; more OIT data compared with other FAIT modalities	Good safety profile; ease of administration	Supervised dosing may improve compliance	Safe and well-tolerated; useful in people with very low reaction threshold who may not tolerate other forms of FAIT	Supervised dosing may improve compliance; potentially short duration of treatment
Weaknesses	Benefit not established in adults; risk of anaphylaxis and EoE	Less efficacious compared with OIT	Higher risk of anaphylaxis; limited data from human studies; administered by injection	Lower threshold doses achieved compared with other FAIT modalities	Limited data from human studies; administered by injection

EPIT, epicutaneous immunotherapy; FAIT, food allergen immunotherapy; LAMP1, lysosomal associated membrane protein1; OIT, oral immunotherapy; SCIT, subcutaneous immunotherapy; SLIT, sublingual immunotherapy.

efficacious protocol, efficacy in adults, cost effectiveness, long-term efficacy and impact on quality of life.

Conclusion

In recent decades peanut allergy has emerged as an important health condition given its increasing prevalence and risk of severe reactions. Peanut allergy is no longer solely the domain of paediatricians, as an increasing number of adolescents transition into the care of adult physicians with life-long peanut allergy. Currently, the management of peanut allergic patients focuses on strict allergen avoidance as well as access to emergency medications to treat reactions from accidental exposures. In the near future peanut immunotherapy will be available, offering active strategies to desensitise and reduce the risk of accidental exposures to people with peanut allergy.

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Useful resources

- ASCIA, the peak professional body of clinical immunology and allergy in Australia and New Zealand: allergy.org.au
- Anaphylaxis action plans: <https://www.allergy.org.au/hp/anaphylaxis/ascia-action-plan-for-anaphylaxis>
- Diet sheets: <https://www.allergy.org.au/patients/food-allergy/ascia-dietary-avoidance-for-food-allergy>
- Allergy prevention: <https://www.allergy.org.au/patients/allergy-prevention>
- E-learning modules: <https://www.allergy.org.au/hp/hp-e-training>
- Anaphylaxis Australia, a charitable, not-for-profit organisation for allergy support: allergyfacts.org.au
- Nip Allergies in the Bub for food allergy prevention: www.preventallergies.org.au
- 250K website for teenagers and young adults with severe allergies: www.250K.org.au

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