

Food Allergy: A Review

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ABSTRACT

Food allergy is an important public health problem that affects children and adults, and it has been increasing in prevalence in the last 2 to 3 decades. The symptoms can vary from mild to severe, and in extreme cases food allergy can lead to anaphylaxis, which is a life-threatening allergic reaction. Currently, there is no cure for food allergy. Management of food allergy includes allergen avoidance or emergency treatment. The eight most common food allergens are eggs, milk, peanuts, tree nuts, soy, wheat, crustacean shellfish, and fish, all of which are frequently consumed in the US. Thus, patients and their families must remain constantly vigilant, which can often be stressful. Moreover, nonallergic food reactions, such as food intolerance, are commonly mistaken as food allergies. This article highlights risk factors, natural history, diagnosis, and management of food allergy. [*Pediatr Ann.* 2020;49(1):e50-e58.]

Food allergy (FA) is defined as an adverse reaction resulting from an immunoglobulin E (IgE)-mediated immune response to a given food.¹ Food is defined as any substance that can be safely consumed by humans, and includes food additives, dietary supplements, drinks, and chewing gum. Food allergens typically are proteins that are specific to a food. Substances that may be consumed but used only as medications, tobacco products, and cosmetics are not classified as food items.

In a genetically predisposed person, food allergens elicit a specific immune response that results in characteristic symptoms. Food allergens maintain their allergenicity even after they have been cooked or upon digestion in the gastrointestinal tract. Some allergens cause allergic reactions when they are consumed in raw form (most often fruits and vegetables).

Cross-reactivity is a phenomenon in which an antibody reacts with the original food allergen as well as another food or aeroallergen with which

it shares structural or sequence similarity. This may then result in an adverse reaction that is similar to that triggered by the original food allergen. For example, cross-reactivity is common among different shellfish and tree nuts.¹

Foods or food components can elicit some reproducible, nonimmunologic food reactions, which are termed food intolerances. These are not considered food allergens. Food intolerances include metabolic, pharmacologic, toxic, and undefined mechanisms. Adverse reactions to food additives, such as artificial colors (eg, Food, Drugs & Cosmetics yellow 5 [tartrazine]) and preservatives (eg, sulfites), do not have any defined immunologic mechanisms. Thus, adverse reactions to foods can be broadly classified as immune-mediated or non-immune-mediated mechanisms (Table 1).

EPIDEMIOLOGY AND NATURAL HISTORY

Prevalence

FAs are common and affect up to 10% of the population.² Prevalence of food allergy has been increasing over the last 2 to 3 decades.² They are more common in children (8%) compared with adults (3%).¹ The most common food allergens are peanut, tree nuts, fish, shellfish, egg, milk, wheat, soy, and sesame seeds, which account for the majority of reactions.¹

FAs are more prevalent in industrialized/westernized countries.³ A survey of US households showed that 8% of children have a food allergy. Among

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TABLE 1.

Adverse Reactions to Foods

Immune-Mediated Reaction		Non-Immune-Mediated Reaction	
Type	Reactions	Type	Reaction
IgE-mediated	Anaphylaxis, oral allergy syndrome	Metabolic	Lactose intolerance
Non-IgE-mediated	FPIES, celiac disease	Pharmacologic	Caffeine
Mixed IgE- and non-IgE-mediated	Eosinophilic gastrointestinal disorders	Toxic	Scombroid fish poisoning
Cell-mediated	Allergic contact dermatitis	Idiopathic	Sulfite

Abbreviations: FPIES, food protein-induced enterocolitis syndrome; IgE, immunoglobulin E.

these, 2.4% have multiple food allergies, and up to 3% experience severe reactions.⁴

In a study conducted by Mahdavinia et al.⁵ of 817 children with FA, it was noted that black children have higher rates of anaphylaxis and emergency department visits compared to white children. They were also more likely to have allergy to wheat, soy, corn, fish, and shellfish; similar rates of peanut, milk, and egg allergy; and lower rates of tree nut allergy.⁵

Risk Factors

Multiple factors influence the susceptibility to FA or sensitization. These include race/ethnicity (higher among Asian and black children compared to white children), sex (male sex during childhood), genetics, atopic diseases (comorbid atopic dermatitis [AD]), hygiene, vitamin D deficiency, fat intake (reduced consumption of omega-3 polyunsaturated fatty acids), decreased intake of antioxidants, use of antacids (increased use reduces digestion of allergens), obesity (an inflammatory state), and the timing and route of exposure to foods.³ When oral intake of allergens is delayed, it can result in sensitization by exposure through other routes such as cutaneous or inhalation. The dual allergen exposure

hypothesis suggests that in patients with eczema, environmental exposure occurs through the impaired skin barrier, which leads to sensitization and FA even in the absence of oral exposure.^{3,6} The gut microbial environment is also associated with food sensitization and allergy.⁷ Certain microbiota such as *Lactococcus* and *Citrobacter* may be underrepresented in patients with FA.⁷

Studies have evaluated the risk of FA in siblings of patients with food allergy. Gupta et al.⁸ evaluated 1,120 children with FA with at least one sibling and found that 66.6% of the siblings were sensitized but only 13.6% were clinically symptomatic.

NATURAL HISTORY

The natural history of childhood FA suggests a high rate of resolution for some allergies in childhood, such as milk (>50% by age 5-10 years), egg (50% by age 2-9 years), wheat (50% by age 7 years), and soy (45% by age 6 years).⁹ This resolution continues into adolescence. However, other FAs tend to persist or have low rates of resolution, such as peanut allergy (only 20% of patients resolved by 4 years) and tree nut allergy (resolved in only 10% of patients). Allergy to seeds, fish, and shellfish is usually persistent.⁹ Resolution of clinical allergy is usually

lower when the initial level of food-specific IgE (sIgE) is high.

In children, a decrease in sIgE levels over time often suggests onset of tolerance to the food. In patients with egg allergy, those who are able to tolerate baked egg tend to outgrow their egg allergy at a much faster rate as compared to those who cannot tolerate baked eggs (tolerance induction).¹⁰

A study of patients with egg allergy showed that resolution of egg allergy was more common in patients who had lower initial egg sIgE levels and when the initial reaction was urticaria or angioedema instead of atopic dermatitis.¹¹

In patients with peanut allergy, resolution is seen more commonly prior to age 6 years. Resolution is much slower after age 10 years.¹²

FA in adults can be a continuum of childhood FAs (eg, milk, peanut, and tree nuts) or may reflect development of new sensitization to food later in life. Kamdar et al.¹³ identified 171 cases of adult-onset food allergy. They found that shellfish (54%), tree nut (43%), fish (15%), soy (13%), and peanut (9%) were the most common adult-onset allergies. The peak age of onset was in the early 30s, and women were more likely to be affected. Anaphylaxis was reported in one-half of the cases.¹³

PATHOGENESIS/MECHANISMS

Normal physiologic response of the human body toward ingested antigens is oral tolerance. A breakdown of this process results in an increased prevalence of sensitization and food allergies, both IgE-mediated and non-IgE-mediated. Sensitization to food allergens also can occur through other routes, such as the skin.¹⁴

Role of Skin Barrier

Skin barrier disruption can be caused by inflammation (as in AD) or genetic defects (such as filaggrin gene mutations). This results in increased rates of sensitization through damaged skin.¹⁵ A study has shown that impaired skin barrier function at birth is associated with food sensitization or allergy at age 2 years. Thus, an early barrier defect can contribute to increased food allergy.¹⁵ Egg, milk, and peanuts were the most common allergens identified in the study.

NATURAL HISTORY OF CONDITIONS COEXISTING WITH FOOD ALLERGY

FA may coexist with atopic conditions such as asthma, AD, eosinophilic esophagitis (EoE), and exercise-induced anaphylaxis.

Asthma

Patients who have FA with coexisting asthma are at an increased risk for severe asthma exacerbations requiring emergency room visits, hospitalization, and steroid use.¹⁶ Studies have shown that almost all patients who died due to fatal or near-fatal anaphylaxis due to food allergy also had asthma.¹⁷

Atopic Dermatitis

FA is a common comorbidity in patients with AD. IgE-mediated FA is more common in children younger than age 5 years with moderate to severe AD resistant to topical steroid therapy, affecting

up to 35% of patients with AD.¹⁸ Milk, peanut, and egg are most likely to produce positive food challenges in children with AD.¹⁹ Elimination of culprit food allergens results in improvement of pruritus and other symptoms.²⁰ In one study, reintroduction of the food in the diet did not cause any recurrence or worsening of the skin lesions when the patients developed tolerance to that food.²⁰

Eosinophilic Esophagitis

EoE is a chronic condition associated with sensitization to food allergens. Sensitization is mainly to eight common food allergens as well as uncommon foods such as beans, peas, and mustard. Studies suggest that dietary changes benefit symptoms²¹ as well as histopathologic changes²² in the esophagus of patients with EoE.

Exercise-Induced Anaphylaxis

Foods can trigger exercise-induced anaphylaxis in about one-third of adults. Crustacean shellfish, cheese, celery, alcohol, and tomatoes are the common triggers.²² In these cases, the food can be tolerated well when the patient is not doing any exercise. The most common symptoms are pruritus, urticaria, angioedema, flushing, and shortness of breath.

DIAGNOSIS OF FOOD ALLERGY

A detailed medical history and physical examination are the cornerstones in the diagnosis of FA.

Medical History

A detailed medical history should be obtained for all patients who present with food allergic reactions. This includes patients who present with anaphylaxis or symptoms outlined in **Table 2**. Symptoms usually occur within minutes to hours of exposure to the food.

Medical history helps to identify the causative allergens as well as the mech-

anism of reaction, either IgE mediated (immediate) or non-IgE-mediated (delayed). However, the history alone is not sufficient in some cases to make a diagnosis of FA.²³ Further evaluation by laboratory studies or oral food challenges may be required to confirm a diagnosis of FA. Questions that should be asked to patients are listed in **Table 3**.

Patients and their families should be given information regarding allergen avoidance and emergency management of allergic reaction as well as anaphylaxis. Families should also be educated regarding preparing allergen-free food that is age and culturally appropriate. This helps improve patient and caregiver quality of life as well decrease anxiety.

Physical Examination

Physical examination of the patient may reveal urticaria, angioedema, and other signs of an allergic reaction. However, there are no physical findings that are consistent solely with a diagnosis of FA; thus, a physical examination may not be diagnostic of FA in the absence of an acute allergic reaction.

Diagnostic Tests

Diagnostic tests should be selected based on medical history provided by the patient. The tests used should aim to identify the culprit allergen as suggested by history and not randomly test for a large panel of foods. The history can help the clinician understand the underlying mechanism for the allergic reaction (ie, IgE-mediated or non-IgE-mediated) and then select the diagnostic test.

Skin prick/puncture tests. Skin prick/puncture tests (SPT) are the most commonly used tests to evaluate a patient with food allergy.²⁴ When the patient history suggests an IgE-mediated mechanism, SPT can be done to identify the culprit food. A positive SPT result suggests the presence of food sIgE pres-

TABLE 2.

Symptoms of Food-Induced Allergic Reactions

Organ System	Symptoms/Signs
Cutaneous	Pruritus, urticaria, erythema, angioedema
Ocular	Conjunctival erythema, pruritus, tearing, periorbital edema
Respiratory	Sneezing, nasal itching, hoarseness, cough, chest tightness, dyspnea, wheezing, stridor
Cardiovascular	Tachycardia, hypotension, dizziness
Gastrointestinal	Oral pruritus, angioedema of lips, tongue, or palate, colicky abdominal pain, vomiting

ent on the surface of cutaneous mast cells. Usually, the size of the response correlates with the extent of clinical reactivity to that particular food. In a patient with confirmed FA, the SPT helps in identifying the food(s) responsible for IgE-mediated FA.

Occasionally, SPTs can be negative in patients with IgE-mediated FA. In such patients, other mechanisms such as locally secreted IgE may be responsible for the allergic reactions. In such patients in whom history is significant, diagnosis should be confirmed by tests such as physician-supervised oral food challenge.

There are some pitfalls associated with SPT in patients with food allergy. SPTs tend to have low specificity and positive predictive value as compared with oral food challenges. Also, the reagents and methods used for SPTs are not standardized. SPTs effectively detect the presence of sIgE; however, many patients have sIgE in the absence of clinical symptoms of FA. Thus, use of SPTs alone in the clinical setting is not diagnostic of FA and may result in overdiagnosis.²⁵

Allergen-specific serum immunoglobulin E. Allergen-specific sIgE tests aim at detecting the presence of sIgE antibodies in the serum, which represents allergic sensitization rather than clinical

reactivity. Specified cut-off levels are suggestive of clinical reactivity (usually defined as 95% predictive values) and are predictive of clinical reactivity.²⁶ Originally, radioallergosorbent tests were used to measure sIgE, but now more sensitive fluorescence enzyme-labeled assays are available that have sensitivity comparable to that of SPTs. sIgE levels may correlate directly with the probability of a clinical reaction. Thus, high levels of sIgE may suggest ingestion of the food in question will lead to an allergic reaction.²⁶

Different laboratories or different assay systems are available to measure IgE;²⁷ however, results are not comparable between different laboratories. Different forms of antigens are used by each system (eg, skim milk vs freeze-dried milk vs whole milk). Different measurements of sIgE are provided by each system for the same serum samples. Thus, the predictive values for one assay test cannot be applied to other test methods.²⁷

Occasionally sIgE levels may be undetectable in patients with IgE-mediated FA. Thus, confirmatory tests such as oral food challenge must be done if the history is highly suggestive of food allergy.

Atopy patch test. An atopy patch test (APT) is generally used when history is suggestive of delayed or non-IgE-

TABLE 3.

Questions That Should Be Asked Regarding a Diagnosis of Food Allergy

- What were the presenting symptoms?
- What was the food that precipitated the reactions, and has the patient had reactions to this food more than once?
- What was the amount of food ingested?
- What was the reaction to baked (extensively heated) or uncooked food?
- How much time after consuming the food did symptoms occur?
- Can the food ever be eaten without the occurrence of symptoms?
- Was the presence of confounding factors such as activity, alcohol, or use of aspirin or nonsteroidal anti-inflammatory drugs associated with reactions?
- Has the patient ever developed similar symptoms after being exposed to another food?
- What was the duration of the symptoms?
- What treatment was provided to treat the symptoms?

mediated mechanism for the reaction. APT is an investigational tool for diagnosing FA and there are no standardized reagents available for patch testing. The sensitivity and specificity of APTs are highly variable as compared with oral food challenges. APT may be helpful for diagnosing FA in some cases such as patients with AD and EoE.

Use of skin prick tests, sIgE tests, and atopy patch tests in combination. There is no significant benefit in using all three tests (SPTs, sIgE tests, and APTs) for the evaluation of FA rather than using SPTs or sIgE tests alone. Using these tests in combination may provide higher positive and negative predictive values but may not be clinically relevant. Moreover, this may be time-consuming for the patient and families and thus inconvenient; however, using two tests may

sometimes be more helpful for identifying the culprit food allergen.

Food elimination diets. In some patients, omitting one or more foods as suggested by their medical history may be useful in the diagnosis of FA. This is shown to be helpful in diagnosing some non-IgE-mediated food-induced allergic disorders such as food protein-induced enterocolitis syndrome, and some mixed IgE- and non-IgE-mediated food reactions such as EoE.²⁸ However, this can result in nutritional deficiencies, especially when multiple foods are omitted from the diet.²⁸

Oral food challenges. The gold standard for diagnosing FA is an oral food challenge. The double-blind, placebo-controlled food challenge reduces bias and is the most specific challenge; however, it is often time consuming. Single-blind or open-food challenges are often done to diagnose food allergy. The food challenge is negative if the patient does not develop any symptoms, and this helps to rule out FA. A definitive diagnosis of FA can be made when the challenge is positive (patient develops objective symptoms during the challenge) and results correlate with medical history and laboratory tests.

An oral food challenge should begin with a dose that is lower than the dose required to elicit a reaction.²⁹ The dose is gradually increased during the challenge while monitoring for any allergic symptoms until target dose is reached. The food challenge should be performed only under supervision of medical personnel who have experience in carrying out a food challenge so that immediate treatment can be administered in case of allergic reaction or anaphylaxis.

Molecular or component-resolved diagnostic tests. These tests aim at identifying IgE against specific proteins in a food and can provide more specific

diagnostic information. For example, Ara h 2 is a major peanut protein that is associated with clinical reactions, whereas Ara h 8 is a birch tree pollen (Bet v 1 allergen) homolog and is usually not associated with significant clinical reactions. Ara h 1, 2, 3, and 9 are stable proteins whereas Ara h 8 is labile. Thus, a patient with undetectable Ara h 1, 2, 3, and 9 but positive Ara h 8 would be more likely to develop tolerance.³⁰ On the contrary, increasing concentrations of sIgE to Ara h 2 signify increasing risk of reaction to peanut. Many component-resolved diagnostic tests have become commercially available and have been considered promising in the diagnosis of food allergy.

Routine FA testing should not be done prior to the introduction of highly allergenic foods (such as milk, egg, and peanut) in children who are at high risk of reacting to such foods.¹ These include children who have severe allergies and/or significant family history of FA. Widespread testing for large panels of foods has poor predictive value and is not recommended. False-positive results are common and result in unnecessary dietary restrictions. There are no advantages in testing for FA prior to introduction of highly allergenic foods, especially when there is no personal history of allergies or family history.

FA evaluation may be helpful prior to introduction of foods in some cases. An oral food challenge can be done in some people with certain risk factors, such as having a sibling who has peanut allergy or having another coexisting FA (eg, testing for tree nut allergy in a child with peanut allergy).³¹

Patients with FA should undergo follow-up testing to reevaluate if the food allergies are outgrown. Patients who are allergic to milk, egg, soy, and wheat can be tested annually, whereas patients who are allergic to peanut, tree nut, fish, and

shellfish can be evaluated every 2 to 3 years. However, in the event of a recent FA reaction, repeat testing can be deferred for several years.

Vaccinations in Patients with Egg Allergy

Administration of vaccines containing egg protein is associated with risk of anaphylaxis in patients who have IgE-mediated egg allergy.

Influenza vaccine. Egg-based inactivated influenza vaccine has been shown to be safe in people with egg allergy.³² US allergy guidelines from the American Academy of Allergy, Asthma, and Immunology/American College of Allergy, Asthma, and Immunology Joint Task Force on Practice Parameters state that influenza vaccines can be safely administered to people with egg allergy of any severity and that no special precautions are required.³³ The guidelines suggest that screening questionnaires do not need to ask about the egg allergy prior to administration of influenza vaccine.³³ Live-attenuated influenza vaccine can also be safely administered to patients with egg allergy of any severity.³⁴ Similar recommendations have been approved by American Academy of Pediatrics, the United States Centers for Disease Control and Prevention Advisory Committee on Immunization Practices, and the National Institute of Allergy and Infectious Diseases.³⁵

Measles, mumps, rubella (MMR) vaccine and varicella vaccine (MMRV). These vaccines can be safely administered to children with egg allergy of any severity.³⁶

Yellow fever vaccine. This vaccine is contraindicated in patients who are allergic to eggs; however, it can be safely administered if allergy evaluation and testing is done with the vaccine.³⁷

Rabies vaccines. This vaccine is contraindicated in patients who are allergic to eggs; however, it can be safely administered if allergy evaluation and testing is done with the vaccine.³⁷

PREVENTION OF FOOD ALLERGY

Maternal Diet During Pregnancy and Lactation

Food allergen avoidance/restriction either during pregnancy or breastfeeding has not been shown to prevent FA.³⁸ Studies do not show any significant reduction in severity of eczema in infants if the mother avoided any dietary antigens. The authors did not notice any significant difference in the incidence of AD, asthma, and positive SPTs to egg or milk.³⁸

Exclusively breast-feeding is recommended until age 4 to 6 months. Although breast-feeding has not been conclusively shown to have a protective role in prevention of allergic diseases, all infants, including those who have a family history of atopic disease, should be exclusively breast-fed until 4 to 6 months of age if possible.

Introduction of Solid Foods

Introduction of solid foods should not be delayed beyond age 4 to 6 months. Allergenic foods should be introduced early on in life without delay.¹ In the Learning Early About Peanut (LEAP) trial, infants age 4 to 11 months at high risk for peanut allergy were those with severe eczema with or without egg allergy. Infants with peanut SPT results of 4 mm or less were randomized to consume or avoid peanut to age 5 years. Peanut allergy was significantly lower in infants who were sensitized to peanut and randomized to consumption (10.6%) compared to the avoidance group (35.3%). Among infants who were not sensitized, peanut allergy was 13.7% in the avoidance group and 1.9% in the

ingestion group.¹ The LEAP trial recommends that peanut should be introduced as early as 4 to 6 months in infant-safe forms. This reduces sensitization and peanut allergy later in life.³⁹

Infant Formulas

Soy infant formula⁴⁰ or hydrolyzed infant formulas⁴¹ do not offer any advantage over cow's milk infant formula in prevention of development of FA.

MANAGEMENT OF FOOD ALLERGY

Currently, there is no cure for FA. Effective management of food allergy requires strict avoidance of food allergens as well as prompt treatment of allergic reaction.

Allergen avoidance is presently the safest strategy for management of FA. This should be done for both IgE-mediated and non-IgE-mediated FA. Patients should avoid cross-reactive foods that hold clinical significance. Cross-reactivity is common among different shellfish and different tree nuts (**Table 4**).

It is important to educate patients and their families about reading all food labels. The Food Allergen Labeling and Consumer Protection Act mandates that the manufacturers should disclose the presence of the eight major food allergens (milk, egg, peanut, tree nuts, soy, wheat, fish, and crustacean shellfish) on food labels whenever they are used as ingredients because these allergens are responsible for most of the severe FA reactions in the United States.¹ People with a FA should avoid consuming foods with warning labels that state "this product may contain trace amounts of allergen" if they are allergic to those foods. However, despite ingredient labelling and reading of food labels, unintentional exposure to allergen still occurs.

Epinephrine is the first-line treatment in all cases of anaphylaxis. Intramuscular (IM) epinephrine is more effective com-

pared to subcutaneous injection as it results in a rapid increase in concentrations of epinephrine in the plasma and tissue.⁴² The IM dose should be administered in the anterolateral thigh in vastus lateralis muscle. Children who weigh less than 25 kg should receive a 0.15-mg dose of epinephrine from an auto-injector, whereas children and adults who weigh 25 kg or more should receive a 0.3-mg dose from an auto-injector.⁴³ Onset of action of epinephrine is within minutes but it is rapidly metabolized; thus the effect is short-lived and some patients may require repeated doses. Epinephrine dosing can be repeated 5 to 15 minutes after the previous dose if symptoms worsen or the patient does not improve.⁴⁴

An epinephrine auto-injector should be prescribed to all patients with FA, and their use should be reviewed with the family. A written action plan for anaphylaxis management should also be provided to the family along with Internet resources (<http://www.foodallergy.org>, <http://www.cofargroup.org>, <http://www.aaaai.org>, <http://www.acaai.org>, <http://www.aafa.org>, <http://www.allergyready.com>).

Specific food allergens should be avoided by patients who have other comorbid conditions such as AD, asthma, or EoE if they have a documented FA. However, there is no benefit to avoiding allergenic foods (milk, egg, peanut, tree nuts, wheat, soy, seafood) as a means of managing any coexisting AD,⁴⁵ asthma, or EoE if the food allergy is not proven. Unnecessary food avoidance can result in nutritional deficiencies and growth deficits.

There are no medications currently available to prevent IgE-mediated food-induced allergic reactions from occurring in a person with an existing FA.

Future Therapies

Allergen avoidance is an effective strategy to prevent FA, but accidental

TABLE 4.

Clinical Cross-Reactivity Among Different Foods

Food	Related Food	Rate of Clinical Reaction
Peanut	Legumes	5%
Tree nut	Another tree nut	35% ^a
Fish	Another fish	50%
Shellfish	Another shellfish	75%
Cow's milk	Goat's or sheep's milk	>90%
	Beef	10%

^aMore common cross-reactions include walnut and pecan, almond and hazelnut, cashew and pistachio.

exposures still happen. Allergen immunotherapy or desensitization temporarily increases the threshold for reacting to the food allergen and thus provides a measure of safety, but this requires continuous exposure to the allergen. Currently, oral immunotherapy (OIT), epicutaneous immunotherapy (EPIT), and sublingual (SLIT) immunotherapy are most common areas of interest. A systematic review of studies of allergen immunotherapy, mostly in children, reported that desensitization has multiple beneficial effects but is associated with systemic (anaphylaxis) and local adverse reactions (oropharyngeal/gastrointestinal/perioral rash).⁴⁶ The balance of benefit and risk underscores the clinical equipoise required for these treatments. Studies have compared different routes of immunotherapy and have shown that although OIT is more efficacious than SLIT and EPIT, it is associated with a higher risk of side effects (allergic reactions and EoE).⁴⁷ SLIT and EPIT have fewer side effects but their efficacy is lower compared to OIT. These therapies increase the reactive thresholds and thus decrease the risk of anaphylaxis.⁴⁸ One study explored the benefit of using omalizumab (anti-IgE antibodies) in combination with OIT. The study showed that patients had fewer side effects, but it did

not change the efficacy profile⁴⁹ and also increased costs of treatment.

Additional strategies that might not be allergen specific are under investigation and include traditional Chinese medicine,⁵⁰ dupilumab and other biologics,⁵¹ DNA-based vaccines,⁵² and probiotics.⁵³

CONCLUSION

The prevalence of FA is high and affects up to 10% of the population. Diagnosis of FA requires understanding the pathophysiology and epidemiology and then combining it with patient history and test results. It is possible to have sensitization without clinical reactivity and vice versa. Currently, management requires allergen avoidance and emergency treatment. An epinephrine auto-injector should be prescribed to all patients with food allergy. Numerous resources are available to patients and physicians to promote education and counseling to improve safety and quality of life. Future therapies such as OIT, EPIT, and SLIT are underway.

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