



Primary care management of food allergy and food intolerance

Abstract: *The incidence of food allergies is steadily increasing. Due to potentially life-threatening complications, it is important that primary care providers recognize and appropriately manage these disorders. This article includes a discussion of the current evidence-based guidelines for the diagnosis, screening, and management of food allergies.*

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Approximately 25% of Americans believe they have a food allergy (FA);¹ however, there are more people who believe they have FAs than people who actually do. The National Institute for Allergy and Infectious Disease (NIAID) defines an FA as an adverse effect caused by an immune response that occurs secondary to exposure to a food substance.² Over 90% of FAs result from exposure to milk, eggs, peanuts, tree nuts, fish, shellfish, soy, and wheat.^{3,4} *Food intolerances* (FIs) are nonimmunologic adverse effects caused by food exposure. Examples include milk, gluten, or wheat intolerance. Both FAs and FIs can cause significant distress

and illness in affected individuals, and the clinical similarities of these two can make distinguishing them difficult. However, because of their immunologic component, FAs can be life threatening. Therefore, primary care providers must be aware of the current clinical evidence that describes the identification and best management of FAs and FIs.

■ Epidemiology and pathophysiology

FAs occur after exposure to an allergy producing protein (allergen). In some individuals, this occurs the first time a food is eaten, while in others, dozens of exposures are needed for a

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response. The allergic response happens via immunoglobulin E (IgE) and non-IgE mechanisms. In IgE-mediated allergic responses, IgE molecules bind to food proteins (allergens) and cause mast cell degranulation and inflammatory mediator release. These inflammatory mediators (such as histamine, leukotrienes, and cytokines) cause increased capillary permeability, vasodilation, mucus secretion, and bronchial constriction. This results in various clinical symptoms and can include pruritus, dyspepsia, urticaria, wheezing, and anaphylaxis. Non-IgE responses occur when T cell and food antigen complexes form in the gastrointestinal mucosa and result in gastrointestinal distress. Cow's milk allergy is the most common form of non-IgE FA.⁵ In contrast, FI occurs when there is an inability to digest food due to a nonimmunologic reason such as an enzyme deficiency or metabolic disorder.^{6,7}

An estimated 4% of adults and 5% of children have FAs, and as many as 28% of children have FIs.^{8,9} However, these statistics are deceiving because although the prevalence rates are low, there was an 18% increase in FA rates from 1997 to 2007.³ The physical and financial burden of the growing FA problem is high. Approximately 9,500 children under the age of 17 years are hospitalized annually for FA-related illnesses.³

Although there is no clear predictor of which individuals will develop an FA or FI, there are clear risk factors for both conditions, especially age. The highest risk of FA occurs during childhood, specifically between 1 and 5 years of age.^{3,9} Infantile cow's milk allergy, in particular, is often "outgrown."¹ The prevalence of food sensitivity, measured by elevated IgE levels, peaks between the ages of 1 to 4 years (28.1%) and steadily decreases to 1.3% by 60 years of age.⁹ Other known risk factors for FA include male gender, and non-Hispanic Black race.^{3,9} People with asthma, eczema, or hay fever have two to four times the risk of FA compared to people without these conditions.^{2,3,9}

The most common FAs are peanut (0.34% to 0.75% of the population), shellfish (0.6%), milk (0.05% to 0.6%), and egg (0.12% to 0.39%).^{2,9} (see *Common protein-triggered FAs*). Milk allergies are most prevalent in individuals between 1 and 5 years old, and peanut allergies in those 6 to 19 years old. Other common FAs include soy (0.03% to 0.4%), wheat (0.4% to 2%), tree nuts (0.2% to 4.5%), fruits (0.02% to 4.2%), vegetables (0.01% to 2.7%), and fish (0.2%).^{2,10} Interestingly, heat breaks down many allergenic proteins, so people are more likely to have allergic responses to raw versus cooked products or pasteurized fruits and vegetables. Common FIs include lactose and gluten intolerance, although both are relatively rare in children.¹¹

■ Clinical presentation

One of the greatest challenges to the primary care diagnosis and management of FA and FI is that the clinical presentation

of these two disorders is very similar. In addition, clinical presentation varies among patients. Unfortunately, neither FA nor FI can be diagnosed based upon history and physical exam findings.

In most cases, individuals with FI present with gastrointestinal distress that occurs within 30 minutes to 1 hour after eating. Most food allergies occur within minutes to up to 2 hours after eating the allergenic food. In contrast to FI, FA may manifest with symptoms from multiple systems. The key to recognizing the association between FA and food intake is the timing of symptoms and determining which systems are involved.

Onset of gastrointestinal symptoms can be immediate (beginning within minutes of food ingestion to up to 2 hours later) in both FA and FI. However, gastrointestinal FI symptoms can also be delayed, and the onset of symptoms begins 12 to 24 hours after food ingestion. Common gastrointestinal symptoms include nausea, reflux, bloating, cramping, vomiting, diarrhea, and flatulence. Sudden onset of forceful vomiting is a well-documented symptom of IgE responses.² Dermatologic symptoms include eczema exacerbation, facial flushing, pruritus, angioedema, and urticaria (the latter two are signs of anaphylactic response). Dermatologic symptoms, with the exception of facial flushing, primarily occur with FA and not FI. Respiratory symptoms occur only in FA and are often a sign of anaphylaxis. These include tearing, rhinorrhea, sneezing, oropharyngeal tingling, wheezing, and cough.

Anaphylaxis is a rare and life-threatening complication of FA that results in cardiovascular shock and/or bronchospasm

Common protein-triggered FAs^{1,2}

Pediatric FAs

- Milk: cow
- Eggs
- Peanuts
- Soybean (soya)
- Wheat
- Tree nuts
- Fish

Adult FAs

- Shellfish: shrimp, lobster, crab, clams, crawfish, mussels, oysters, prawns, snails, scallops, squid, octopus
- Tree nuts: walnuts, pecans, almonds, beech nut, butternut, cashews, chestnuts, coconut, macadamia, pistachios
- Fish
- Peanuts
- Fruit: melons, strawberries
- Soybean (soya)
- Wheat

Cross-reactions associated with OAS^{2,12,13}

Pollen allergen	Food cross-reactivity
Birch pollen	Apples, carrots, celery, hazelnuts, peaches, pears, raw potatoes, plums
Ragweed pollen	Bananas, melons (cantaloupe, honeydew, watermelon), tomatoes, cucumbers
Grasses	Kiwis, tomatoes, peaches
Mugwort pollen	Apples, carrots, celery, kiwi fruit, peanuts, spices (caraway seeds, parsley, coriander, anise seeds, fennel seeds)
Fruit pollen	Kiwi, pineapple, orange

and airway obstruction. It is most common in adolescents and young adults, and is associated with allergies to a specific food like peanuts.² Diagnosis of an anaphylactic reaction is not based on severity of symptoms, but rather the presence of symptoms that affect more than one body system (severe vomiting and urticaria, wheezing, and diarrhea). Early signs of anaphylaxis include tearing, rhinorrhea, tingling of the lips and tongue, and a feeling of warmth or “something isn’t right.” If not detected at this point, symptoms progress to wheezing, difficulty breathing and/or swallowing, hoarseness, light-headedness, vomiting, and eventually hypotension, angioedema, and bradycardia. These symptoms typically get worse with repeated exposure. Although rare, anaphylaxis can occur with a first-time exposure to a food.

The most common sign of FA in adults is oral allergy syndrome (OAS).¹² OAS is the result of an IgE-mediated type I hypersensitivity cross-reaction between food and inhaled tree, weed, or grass pollens (see *Cross reactions associated with OAS*). It is more common during the months when seasonal pollen levels are highest. Affected individuals present with tingling or numbness of the lips, palate, or oropharynx within minutes of mucosa contact with the offending food. Less frequently, individuals may complain of throat tightness or lip/tongue edema. Anaphylaxis from OAS is rare. Symptoms rapidly decrease once the food is swallowed or spit out.

Unfortunately, approximately 20% to 30% of the population reports having an FA, yet reliable estimates of FA prevalence are between 3% and 4%.^{1,2,4,13} The mistaken belief that an individual has an FA can result in the inconvenience of unnecessary dietary restrictions and cause undue anxiety for patients and their families. The challenge for primary care providers is that a history and physical exam alone cannot diagnose FA and FI. Additionally, many providers are uncomfortable suggesting that patients with suspected

allergies continue to eat potentially offending foods because of the risk of anaphylaxis. Removing the offending food from the diet will prevent symptoms in both FA and FI.

■ Clinical diagnosis

Food elimination diets are important to the diagnosis of FA and FI, especially for non-IgE-mediated conditions.² FA diagnosis is appropriate in cases with significant clinical symptoms that resolve following food elimination. However, it is important to note that patients who develop food tolerance to allergens that result in minimal symptoms prior to food elimination may be at risk for anaphylaxis if foods are reintroduced after tolerance wanes.

The standard for the diagnosis of FA is the oral food challenge. However, many providers do not use food challenges because of personnel and time requirements, and because of the risk of inducing anaphylaxis. Skin prick testing and food-specific antigen testing are problematic because of the likelihood of false positives, false negatives, and the risk of identifying a serologic reaction that does not actually result in clinical symptoms.^{2,4,8} Positive skin or serum food-specific IgE testing in individuals with mild or nonspecific symptoms (rash, gastrointestinal complaints) is associated with a less than 50% chance of having actual FA.⁴ As a result, there are no clear recommendations for one diagnostic test over another. Skin prick testing has a positive predictive value of 50% and a greater than 95% negative predictive value, and requires antihistamine discontinuation for a number of days before testing.¹ Total serum IgE levels, atopy patch testing, and intradermal testing are not recommended due to the lack of scientific evidence to support these practices.^{2,4} Radioallergosorbent testing is no longer recommended as this has been replaced with the more sensitive fluorescence enzyme-labeled assays.^{2,14} Although there is clear evidence supporting the use of food allergen specific IgE tests, especially in instances where skin testing is challenging or contraindicated, clinicians should avoid using food allergen panels and should order single food-specific levels to avoid the diagnosis of a food sensitivity that is not clinically significant.

Diagnostic testing should be reserved for those with significant risk of FA. These individuals include children and adults with eosinophilic esophagitis (EE), moderate-to-severe atopic dermatitis, enterocolitis, those with suspected or risk for anaphylaxis, and allergic proctocolitis.^{2,8} The first step to a proper diagnosis of FA is reviewing the patient’s history and conducting a physical exam. Food diaries can help identify temporal relationships with food, reproducibility of the reaction, and symptom patterns. Because an estimated 50% to 90% of presumed FAs are not actually allergic reactions, but more likely FIs, neither

patient history nor physical exam alone is sufficient to diagnose FA.²

No single testing approach works for all patients. Clinicians must use a tailored approach to best match patient needs with diagnostic limitations (see *Primary care action plan for FA diagnosis*).

■ Differential diagnoses

There are various differential diagnoses that merit consideration when FA and FI are suspected (see *Differential diagnoses for FA*). EE is a disorder that results from a combination of allergic and immune responses.⁷ Risk factors for EE include male gender, age (infants, children, and adolescents), and history of atopy (asthma, allergies, and eczema). The cardinal sign of EE is the subjective feeling of food “sticking.” Reflux, painful swallowing, cough, and food aversion are also common. Diagnosis is made by documenting the presence of eosinophils in the esophagus.⁷ This diagnosis should be considered in individuals where regurgitation is the primary symptom and there is unclear temporal relationship between symptoms and food ingestion.

Allergic proctocolitis is a non-IgE-mediated, benign condition that causes blood-streaked bowel movements in an otherwise healthy infant. This disorder occurs in both formula- and breast-fed infants, although up to 60% of cases occur in breast-fed infants.¹⁵ The classic history in allergic proctocolitis includes increasingly frequent bloody or mucous stools without nausea or discomfort, beginning in the first few weeks of life. Growth and development in affected individuals is normal, and physical exam is normal.

Symptoms generally resolve within 48 to 72 hours of eliminating milk products from the infant or breastfeeding mother’s diet.

Food protein-induced enterocolitis syndrome (FPIES) is a non-IgE-mediated disorder that causes sudden, repeated, forceful vomiting and diarrhea in formula-fed infants. Unlike allergic proctocolitis, this condition can cause significant morbidity secondary to electrolyte imbalances, hypotension, dehydration, and leukocytosis.^{2,15} If not promptly diagnosed, chronic FPIES can cause failure to thrive and hypoalbuminemia. Most cases are caused by exposure to cow’s milk or soy, and as many as half of patients who react to milk will also react to soy.¹⁵ There are also documented cases following rice, oat, or cereal grain ingestion.² Symptoms appear within minutes to up to 2 hours after eating, and often mimic anaphylaxis. Because this reaction does not involve IgE mediation, epinephrine has no clinical effect on symptoms. Treatment of FPIES involves switching to a casein hydrolysate formula or breastfeeding. Interestingly, there are no published reports of FPIES in breast-fed infants.¹⁵

A rare non-IgE-mediated reaction to cow’s milk, Heiner syndrome causes upper and lower respiratory tract illness, gastrointestinal symptoms, iron deficiency, and failure to thrive. Symptoms appear following cow’s milk protein ingestion that rapidly disappears once cow’s milk is discontinued and includes cough, wheezing, nasal congestion, vomiting, fever, and pulmonary infiltrates. Lab findings may indicate elevated IgG, IgA, IgM, IgE, and eosinophil levels, and decreased hemoglobin and hematocrit.¹⁶

Primary care action plan for FA diagnosis^{2,4}

Step 1: History and physical exam

Thorough patient history

- Family history of atopy or FAs
- Patient medical history of atopy (including eczema, asthma, rhinitis)
- What symptoms occur?
- When do the symptoms occur in relation to foods eaten?
- Do the symptoms occur every time the food is eaten?
- Do the symptoms happen at any other time?
- What treatments have been tried in the past?

Physical exam

- Although physical exam findings are not diagnostic, findings such as eczema, urticaria, or wheezing may be suggestive of atopy, increasing the risk of FA.

Step 2: Food allergen specific serum IgE (sIgE) testing

sIgE

- If H&P are suggestive of FA and possible trigger foods are identified, sIgE can confirm causative foods but is not diagnostic.

Step 3: Referral

Referral to allergy specialist

- If H&P are highly suggestive of FA or sIgE testing is positive.
- Oral food challenge is the gold standard for diagnosis of FAs.
- Skin Prick Test may be helpful to identify allergens.

Differential diagnosis for FAs^{2,6,10,12,15}

Differential	Clinical presentation
Eosinophilic esophagitis	Dysphagia, feeling that food “gets stuck,” abdominal/chest pain, vomiting, and regurgitation. More common in males and peaks in age between 1-4 and 10-14 years.
Eczema flare from nonfood exposure	Decreased humidity, dry skin, bacterial infections, and skin friction may cause worsening of eczema. Symptoms include maculopapular eruptions of the flexural skin folds and face, generalized skin dryness, and lichenification.
Food chemical effect	Facial flushing with spicy food, phototoxic reaction between lemon juice and sunlight causing contact dermatitis.
Infection from contaminated food	Nausea, vomiting, diarrhea, abdominal cramping, and flatulence. Food poisoning from <i>Staphylococcus aureus</i> infection typically begins with 6 hours of food ingestion
Food exacerbated conditions	Migraine symptoms (unilateral, throbbing headache with or without aura) following ingestion of foods containing vasoactive amines (chocolate, aged cheese, red wine, beer). Spicy foods can worsen gastroesophageal reflux.
Protein-induced proctocolitis/enterocolitis	Allergic proctocolitis causes blood streaking in an otherwise asymptomatic infant (usually breast-fed). Protein-induced enterocolitis causes blood in stool, vomiting, failure to thrive in a formula-fed infant. Symptoms disappear within 2 to 3 days of cow's milk protein removal from diet.
Food intolerance	Abdominal pain, nausea, bloating, flatulence, and diarrhea that begin within 30 minutes to 2 hours of ingesting milk products.
Heiner syndrome	Rare disease that affects infants and causes pulmonary disease characterized by pulmonary infiltrate, wheezing, cough, rhinorrhea, fever, hematochezia, anemia, and failure to thrive.

■ Clinical management

Patient and family education is the cornerstone of FA and FI management. The most effective method for managing FA and FI is food avoidance. Although there is no clear evidence that children with FA and FI are more likely to have impaired growth and nutritional deficiencies, routine dietary counseling and growth monitoring is recommended especially during infancy.^{1,2} Food avoidance can be challenging for school age children and adolescents who often eat meals that are not prepared by parents or family members. Older children and adolescents are more likely to be compliant with FA and FI dietary restrictions when they understand the reasons why foods have to be avoided and when they are allowed to be actively involved in food substitution decisions. Patients and family members must learn to read food labels carefully, and to be especially cautious about wording that prepared foods “may contain trace amounts of.” Education on how to recognize each person's early signs of allergic reaction as individual symptoms vary widely and is important. Delays in anaphylaxis treatment are potentially life threatening yet many patients report fears of “over using” autoinjectable epinephrine.

Education is also essential for those who mistakenly believe they have an FA. It is important that primary care providers don't discount the patient's beliefs, but rather explore the patient's definition of FA. Primary care providers

can help patients understand why symptoms present the way they do by explaining the physiology of symptoms, when known. Individuals who have food chemical reactions, infection from contaminated food, or food-exacerbated conditions may benefit from reassurance that their real physical symptoms are not actually due to FA or FI. Primary care providers should educate these patients about avoiding or diminishing reactions.

The NIAID guidelines do not recommend routine medications for the management of FA.² However, antihistamines help decrease OAS and dermatologic symptoms.¹⁴ Short-acting antihistamines like diphenhydramine and longer-acting antihistamines like loratadine and cetirizine have wide safety margins and are generally well tolerated with PRN dosing. In some cases, FI can be managed through dietary supplementation with enzymes like lactase. Those with a history of or significant risk for anaphylaxis require prescribed autoinjectable epinephrine. True anaphylactic reactions may require more than one dose of epinephrine, so it is recommended that patients keep at least two injectors on hand.² Education with nonactive demo autoinjectors is particularly helpful as many patients do not realize the force required to activate an autoinjector, and are usually unaware and startled by the loud sound made as the needle ejects.

Subcutaneous therapy, sublingual immunotherapy, and oral immunotherapy are not recommended by NIAID, but there is evidence that immunotherapy can desensitize people with FA.^{2,4} What is not known is how long this desensitization will last and if the benefit outweighs the risk of anaphylaxis. More scientific evidence is needed to determine the safety and efficacy of immunotherapy in practice.

■ FA prevention

Because of the morbidity and mortality associated with FA and FI, there is significant interest in preventing these disorders, especially for those at risk for FA. NIAID defines at-risk individuals as those who have a biologic parent or sibling with a history of allergic rhinitis, asthma, atopic dermatitis, or FA.² The desire for primary prevention of food sensitivities led to the historical development of multiple recommendations that have no clear scientific justification.

For example, the American Academy of Pediatrics and NIAID no longer recommend delaying the introduction of allergenic foods such as egg whites, berries, and wheat beyond 4 to 6 months of age.^{2,17} Other recommended FA prevention strategies are not scientifically supported. There is no value to allergy testing before introducing “highly allergenic foods” such as milk, peanuts, and egg in high-risk infants and children.²

Despite the evidence that antigens pass in breast milk and some studies indicating that the risk of peanut allergy can be minimized by diet restrictions during pregnancy and lactation, these findings are not consistent.^{14,17} Thus, the American Academy of Pediatrics and NIAID do not recommend diet modification during pregnancy and lactation to prevent FA and FI.^{2,17} Lastly, there is no evidence that soy formula is preferable to cow’s milk formula in high-risk infants without clinical symptoms.^{2,17}

There are only three clear FA preventive strategies with scientific evidence and include the following:^{2,4,14}

1. Infants who are exclusively breast-fed for at least 4 months have a significantly lower risk of atopic disease than their formula-fed peers.
2. The delay of complementary solid introduction (such as baby foods) until at least 4 to 6 months of age correlates with decreased FA, asthma, and eczema.
3. High-risk infants, as defined above, may benefit from hydrolyzed formula with the aim of preventing milk allergy. However, it is important to note these hydrolyzed formulas are significantly more expensive than traditional infant formulas, and they are not as readily available in retail stores.

■ Scenarios

Scenario 1: Mr. J is a 47-year-old who presents with complaints of tingling of the lips, tongue, posterior pharynx, and throat after eating melons, walnuts, and bananas. He has a history of seasonal ragweed allergies. None of these episodes were accompanied by rash, wheezing, or difficulty swallowing. Mr. J was referred to an allergist and had positive skin tests to ragweed, dust mites, and walnuts. This patient was placed on a food avoidance diet, daily cetirizine for his seasonal and perennial allergies, and was educated to take oral diphenhydramine in case of accidental ingestion. He has had no further food-related problems.

Scenario 2: KF is a 17-year-old who presents with a complaint of abdominal pain, bloating, nausea, and diarrhea after eating whole-wheat products. She has a personal and family history of asthma, allergies, and atopic dermatitis. Wheat specific IgE assay does not indicate an allergy. She later complained of symptoms with pasta and other gluten containing foods. Her symptoms resolved after going on a gluten-free diet.

At-risk individuals are those who have a parent or sibling with a history of allergic rhinitis, asthma, atopic dermatitis, or FA.



Scenario 3: MS is an 18-month-old who presents with severe ocular swelling, conjunctival redness, and excessive tearing after getting peanut butter in his eye during a snack about 15 minutes ago. This was his first exposure to peanuts. He has no history of allergies. Because of the child’s age and the severity of the reaction, MS was referred to a pediatric allergist for further evaluation. He was diagnosed with peanut allergy after a food challenge test. His parents were given an autoinjectable epinephrine pen, educated about its use and how to administer it, and advised to get a medical alert bracelet for MS. He remains healthy and has had no further peanut or food-related reactions.

■ Conclusion

FA and FI can cause significant physical discomfort, and FA can be life threatening. Despite the public’s tendency to incorrectly report having FA or FI, prevalence estimates indicate that both these conditions occur at greater rates today than in the past. It is important for primary care providers to be familiar with the nuances of FA and FI clinical presentation. Many patients benefit from a multidisciplinary approach that includes collaboration between primary care and allergy, dermatology, gastrointestinal, and pulmonary care

specialists. There is limited scientific evidence for FA prevention, and the most effective management strategies include food avoidance and prompt anaphylaxis treatment. NP

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