

Food Allergies

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Continuing Education Activity

Food allergies are a significant public health concern affecting millions of children and adults worldwide, with reactions ranging from mild symptoms to life-threatening anaphylaxis triggered by common allergens such as milk, eggs, peanuts, and shellfish. Accurate diagnosis relies on clinical history, skin prick testing, serum-specific immunoglobulin E measurement, and oral food challenges. Management focuses on strict allergen avoidance, patient education, and emergency preparedness, while emerging therapies, including immunotherapy and biologics, offer expanding treatment options. This activity provides an overview of etiology, epidemiology, clinical presentation, diagnosis, and current management strategies, including early allergen introduction, dietary modification, pharmacologic therapy, and acute treatment of anaphylaxis, while equipping healthcare professionals with practical tools to improve diagnostic accuracy, enhance patient safety, and optimize care through patient education and interprofessional collaboration.

Objectives:

- Identify common food allergens and recognize clinical manifestations of immunoglobulin E-mediated and non-immunoglobulin E-mediated allergic reactions.
- Screen patients with suspected food allergies using appropriate diagnostic tools based on clinical presentation and risk factors to ensure timely diagnosis and appropriate management.
- Select appropriate patients for referral to allergy specialists or for immunotherapy interventions based on diagnostic uncertainty or severity.
- Collaborate with the interprofessional healthcare team to educate, treat, and monitor individuals with food allergies, ensuring comprehensive care and improved patient outcomes.

Introduction

Food allergies are a significant public health concern affecting children and adults worldwide. The majority of allergic reactions are caused by 9 common food sources, including milk, soy, eggs, peanuts, fish, shellfish, wheat, tree nuts, and sesame. These immune-mediated responses, categorized as immunoglobulin E (IgE)-mediated or non-IgE-mediated, can range from mild to life-threatening and require timely recognition and appropriate management. Advancements in research and the ongoing development of clinical guidelines continue to refine evidence-based best practices for diagnosing, treating, and preventing food allergies.

Etiology

A complex interplay of genetic, environmental, and immunological factors drives the development of food allergies in infants and children. A strong genetic predisposition is evident, particularly in individuals with a family history of atopic conditions such as **asthma**, eczema, and allergic rhinitis. Research indicates that genetic variations affecting immune regulation and epithelial barrier function significantly contribute to the risk of developing food allergies. A notable example is the loss-of-function mutations in the filaggrin (*FLG*) gene, which encodes a protein essential for maintaining the integrity of the skin barrier. When this barrier is compromised, environmental allergens, including food proteins, can penetrate the skin and interact with immune cells, triggering sensitization. This mechanism is especially relevant in children with atopic dermatitis, who are at increased risk of food allergy development.[\[1\]](#)

In addition to *FLG* mutations, other genetic variants that affect immune system regulation also contribute to food allergy susceptibility. For example, variations in the interleukin-4 (IL-4) gene can lead to an exaggerated T-helper type 2 (Th2) immune response, promoting the development of IgE-mediated allergies. Furthermore, specific human leukocyte antigen (HLA) variants have been associated with an increased risk of peanut allergy, further underscoring the role of genetic factors in food allergy pathogenesis.[\[2\]](#)[\[3\]](#)

Environmental factors play a crucial role in modulating immune function and influencing the risk of developing food allergies. Factors such as mode of delivery (cesarean section versus vaginal delivery), early antibiotic exposure, and alterations in the gut microbiome can influence immune tolerance and the integrity of the intestinal barrier.[\[4\]](#) Children delivered via cesarean section face a higher risk of developing food allergies, possibly related to the lack of exposure to beneficial microbes encountered during vaginal delivery.[\[4\]](#) Early antibiotic use in childhood may increase the risk of developing food allergies, likely due to disruption of the gut microbiome and impaired immune regulation.[\[5\]](#) Additionally, the timing of introducing potentially allergenic solid foods to infants plays a significant role in the risk of food allergies, a topic further explored in the **section on Deterrence and Patient Education** below.

Finally, alterations in immune function are central to the pathogenesis of food allergies. A loss of immune tolerance to otherwise harmless food proteins can trigger inappropriate immune activation. Depending on the specific immunological pathways involved, this response may manifest as either IgE-mediated or non-IgE-mediated hypersensitivity reactions.

Epidemiology

Healthcare experts are increasingly recognizing food allergies as a growing global public health concern, with a rising prevalence in both high-income and resource-limited countries. An estimated 250 million people worldwide are affected by one or more food allergies.[\[6\]](#) In the United States, approximately 8% of children and up to 10% of adults are affected by this condition.[\[7\]](#) Notably, around 40% of affected children have multiple food allergies.[\[8\]](#) The most common allergenic foods include cow milk, eggs, peanuts, tree nuts, soy, wheat, fish, shellfish, and sesame.

Although cow's milk and egg allergies are among the most commonly reported food allergies worldwide, geographic variations exist, likely determined by cultural feeding patterns.[\[9\]](#) Many

children eventually outgrow allergies to milk, eggs, or soy; however, allergies to peanuts, tree nuts, and shellfish are more likely to persist into adulthood.[\[10\]](#)[\[6\]](#) Additionally, individuals with a history of allergies to bee venom, medications, or latex have an elevated risk of developing food allergies later in life.[\[8\]](#)

The prevalence of food allergies varies widely and is influenced by factors such as geography, dietary habits, environmental exposures, access to healthcare, and the diagnostic criteria used.[\[11\]](#) Higher rates are observed in Westernized countries, particularly in urban areas, where food allergies affect up to 10% of infants.[\[7\]](#) In contrast, prevalence tends to be lower in rural and resource-limited regions. These regional differences may be attributed to variations in early-life microbial exposure, air pollution, ingestion of microplastics, and contact with natural environments, including exposure to tree pollen, insects, and animals.[\[5\]](#)[\[12\]](#)[\[13\]](#)[\[14\]](#)[\[15\]](#)

Racial and ethnic differences in food allergies are also observed within countries. For instance, Black children in the United States have higher rates of peanut and shellfish allergies compared to White children.[\[7\]](#) The impact of food allergies extends beyond the individual, contributing to increased emergency department visits, hospital admissions, healthcare costs, and significant psychosocial stress for affected individuals and families.[\[16\]](#) The incidence of food-induced anaphylaxis has also risen, particularly among children and adolescents.[\[17\]](#) Understanding these epidemiological patterns is crucial for clinicians to guide screening, patient education, and preventive strategies tailored to the needs of diverse populations.

Pathophysiology

Food allergy results from an abnormal immune response to dietary antigens, leading to either IgE-mediated or non-IgE-mediated hypersensitivity reactions. In sensitized individuals, exposure to specific food proteins triggers immune activation rather than tolerance, leading to the release of inflammatory mediators and the development of allergic symptoms. In IgE-mediated food allergies, initial sensitization begins with antigen presentation by dendritic cells, which activates Th2 lymphocytes. This leads to the production of allergen-specific IgE antibodies by B cells. These antibodies then bind to IgE receptors on mast cells and basophils.

Re-exposure to the offending food allergen triggers the release of histamine and other pro-inflammatory mediators, leading to smooth muscle contraction, vasodilation, and increased mucus secretion. Symptoms of immediate hypersensitivity can range from urticaria to anaphylaxis, the latter being a severe, potentially life-threatening systemic allergic reaction. Anaphylaxis develops rapidly and typically involves at least 2 organ systems or presents with hypotension, airway compromise, or significant respiratory distress.[\[18\]](#)[\[13\]](#)

Oral allergy syndrome, also known as pollen food allergy syndrome, is an IgE-mediated allergic reaction caused by cross-reactivity between structurally similar proteins in pollen and certain raw fruits, vegetables, and nuts. In sensitized individuals—typically those with pollen allergies—the immune system misidentifies specific food proteins as allergens because they resemble pollen antigens. Upon ingestion of these raw foods, IgE antibodies bound to mast cells and basophils in the oral mucosa recognize the allergens, initiating the release of histamine, prostaglandins, and leukotrienes. This immune response results in localized symptoms, including itching, tingling, and

swelling of the lips, tongue, mouth, and throat. These symptoms usually resolve within minutes to hours and rarely progress to systemic reactions or anaphylaxis.[\[19\]](#)[\[20\]](#)

Cooking denatures the proteins responsible for cross-reactivity with pollen allergens, rendering them unrecognizable to the immune system. As a result, most individuals with oral allergy syndrome can tolerate cooked forms of these foods. Apples, peaches, cherries, and bananas are among the most common foods associated with oral allergy syndrome.

In contrast, non-IgE-mediated food allergies, including food protein-induced enterocolitis syndrome (FPIES), food protein-induced enteropathy, and allergic proctocolitis, are driven by cell-mediated immune mechanisms. These conditions involve delayed hypersensitivity (type IV) reactions, characterized by T-cell-mediated inflammation, epithelial barrier dysfunction, and immune pathways unrelated to IgE-mediated responses.[\[20\]](#) Affected individuals typically present with delayed-onset gastrointestinal symptoms, such as vomiting and diarrhea.[\[21\]](#)

In summary, the pathophysiology of food allergies occurs when the immune system loses tolerance to typically harmless dietary proteins. Genetic predisposition, impaired epithelial barrier function, and environmental factors collectively contribute to the breakdown of immune regulation, triggering a dysregulated immune response. As a result, exposure to specific food allergens can provoke either IgE-mediated or non-IgE-mediated allergic reactions.

History and Physical

The approach to evaluating a patient with possible food allergies depends on whether the clinician investigates a known reaction following food ingestion or considers food allergy as one of several potential diagnoses. When a food allergy is suspected based on a reaction, the clinical history should detail the exposure and characterize the timing and nature of the symptoms. Key questions include:

- What did the patient consume? In what quantity?
- Was the food cooked or raw?
- Did it contain a known allergen such as peanuts, tree nuts, milk, egg, fish, shellfish, wheat, soy, or sesame?
- How much time elapsed before symptom onset?

In IgE-mediated food allergies, symptoms typically develop within minutes to 2 hours of exposure to the allergen. Although individuals may initially present with mild symptoms, subsequent exposures can result in more severe and potentially life-threatening reactions.

Clinicians should gather a detailed account of the reaction, including the severity, progression, and duration of symptoms. Presentations may involve multiple organ systems, such as the skin (urticaria, swelling), gastrointestinal system (nausea, vomiting), respiratory system (wheezing, respiratory distress), or cardiovascular system (tachycardia, syncope). Notably, it is essential to determine whether the patient has previously reacted to this food or a related one, and whether the severity of their response has changed over time. Clinicians should also assess for contributing factors, such as physical exertion, medications, alcohol intake, or underlying medical conditions, that may have

influenced the reaction. Finally, details about any treatments used, including antihistamines, epinephrine, or emergency care, should be documented.

When food allergy is one of several potential explanations for a patient's symptoms, the goal of the history is to assess whether it is the most likely cause. The clinician should investigate the patient's typical diet, with particular focus on common allergenic foods, and identify any consistent patterns between food intake and symptom onset. A food diary can be invaluable in pinpointing food-related triggers, especially in patients with chronic or recurrent symptoms. Timing and reproducibility are key factors; the clinician should ask whether symptoms occur regularly after eating specific foods. Recurrent hives, vomiting, diarrhea, abdominal pain, or respiratory issues following meals should raise suspicion for food allergies.

Additionally, clinicians should inquire about any personal or family history of atopic conditions such as **asthma**, eczema, or allergic rhinitis, as these increase the likelihood of food allergies. A feeding history is crucial for young children, including information on breastfeeding and the introduction of solid foods and potential allergens. Considering other possible causes, such as environmental or medication exposures, non-allergic food intolerances, or gastrointestinal conditions that may mimic food allergies, is also essential.

Non-IgE-mediated food allergies, including irritability, vomiting or frequent spitting up, diarrhea, and poor weight gain, commonly present with gastrointestinal symptoms in infants and young children. In FPIES, affected infants typically experience vomiting 1 to 3 hours after ingestion of the triggering food. With continued exposure, symptoms may progress to include abdominal distention, bloody diarrhea, lethargy, anemia, and failure to thrive. The most frequently implicated foods in FPIES include cow's milk, soy, oats, and rice.

Food protein-induced allergic proctocolitis (FPIAP) typically presents as blood-streaked stools in otherwise healthy infants during the first few months of life. Approximately 60% of cases occur in exclusively breastfed infants. Cow's milk protein is the most common trigger, although other allergens such as soy, egg, and wheat may also be involved. FPIAP is generally a benign, self-limiting condition, with most cases resolving spontaneously within the first year of life.[\[22\]](#)[\[23\]](#)

Patients with food protein-induced enteropathy often present with chronic steatorrhea, protein-losing enteropathy, malabsorption, and poor weight gain during the first few months of life. The malabsorption can lead to anemia, hypoalbuminemia, protein-losing enteropathy, and vitamin deficiencies. While grossly bloody stools are typically absent, occult blood may be present. Cow's milk is the most common trigger for FPE, followed by soy, wheat, and rice. Symptoms generally resolve in individuals by the age of 2 to 3.[\[24\]](#)

In IgE-mediated allergic reactions, cutaneous symptoms such as acute urticaria, flushing, pruritic eyes, and angioedema typically appear within minutes to 2 hours after exposure to the allergen. Common triggers include milk, eggs, peanuts, tree nuts, sesame, and certain fruits, such as kiwi. Notably, about 30% of children with moderate-to-severe atopic dermatitis also have coexisting food allergies, highlighting a potential link between skin barrier dysfunction and allergic sensitization.

Breathing-related symptoms, including nasal congestion, sneezing, coughing, and wheezing, can also manifest, particularly in IgE-mediated reactions. However, isolated respiratory symptoms are rare. Wheezing occurs in approximately 25% of patients with IgE-mediated food allergy reactions, although only 10% of individuals with **asthma** experience food-induced respiratory symptoms.

Respiratory compromise may indicate progression toward anaphylaxis and warrants immediate recognition and intervention. In the United States, food allergy reactions are the leading cause of anaphylaxis presenting to hospital emergency departments. Patients often report generalized malaise, a sense of impending doom, and lethargy, along with symptoms affecting multiple organ systems, including the skin, respiratory tract, gastrointestinal system, and cardiovascular system. Cardiovascular involvement typically manifests as hypotension, tachycardia, pallor, dizziness, syncope, or collapse.[\[8\]](#)

Evaluation

Specific IgE-mediated food allergies can be confirmed or ruled out using 3 principal tests: skin prick testing (SPT), serum-specific IgE testing (sIgE), and an oral food challenge (OFC). Evaluation should be reserved for patients with a high pre-test probability of food allergy, based on a history of allergic symptoms following the consumption of a particular food. Both SPT and sIgE testing have a high negative predictive value, helping to rule out food allergies and prevent unnecessary dietary restrictions when results are negative. However, since SPT and sIgE testing only identify sensitization rather than clinical allergy, positive results without a corresponding clinical history have limited diagnostic value. When the history and initial test results—either SPT or sIgE—fail to provide clarity, an oral food challenge remains the diagnostic "gold standard."

Several factors determine the choice of the initial test. Patients at high risk for anaphylaxis, those with **asthma** symptoms, or individuals who cannot tolerate SPT due to dermatological conditions may be better suited for sIgE testing. The RadioAllergoSorbent Test (RAST), developed in the 1970s, was the original method for detecting allergen-specific IgE antibodies in serum using radioactive labeling. However, newer methods that directly measure allergen-specific IgE antibodies offer higher sensitivity and reproducibility, and have largely replaced RAST. Despite this, "RAST" is still commonly used colloquially to refer to any sIgE assay.

An advantage of sIgE testing over SPT is that primary care clinicians can order the assay and interpret the results without consulting an allergy specialist. Although high concentrations of sIgE are typically associated with an increased likelihood of a clinical reaction, the levels do not always correlate with the severity of symptoms. A negative result can help rule out a food allergy. Still, it may also reflect a lack of prior exposure to the allergen, underscoring the importance of testing only when there is a clear history of symptoms following ingestion of a likely allergen.

Allergists typically perform SPT as part of a comprehensive food allergy evaluation. Although SPT is more sensitive than sIgE testing, it must be conducted in a setting that recognizes and manages anaphylaxis. During the procedure, a small drop of allergen extract is placed onto the skin's surface, usually on the forearm or back, and a tiny prick or scratch introduces the allergen into the epidermis. In patients sensitized to the allergen, a localized wheal, resembling a mosquito bite, typically forms within 15 minutes. The size of the wheal generally correlates with the severity of the allergy. However, children aged 2 or younger and adults aged 70 or older may experience smaller dermal reactions. Certain systemic medications, such as antihistamines and tricyclic antidepressants, can interfere with test results and should be discontinued approximately 1 week before testing.[\[8\]](#)

Intradermal testing involves injecting a small amount of allergen extract into the dermis. Although this technique is more sensitive than SPT, it is less specific, resulting in a higher rate of false positives and an increased risk of systemic reactions. For these reasons, allergists typically perform intradermal testing to evaluate environmental or drug allergies, and it is not recommended for diagnosing food allergies.

When the clinical history and initial testing are inconclusive, an OFC may be necessary to confirm a diagnosis of food allergy. This procedure involves the supervised ingestion of the suspected allergenic food in gradually increasing amounts, with close monitoring for allergic reactions. Similar to the SPT testing, the OFC must be conducted in a setting equipped to treat anaphylaxis. Patients should avoid the suspected food for at least 2 weeks before the challenge and discontinue medications that could interfere with the test, such as antihistamines and beta-adrenergic bronchodilators.[\[25\]](#)

The OFC can be conducted as a double-blind placebo-controlled challenge to minimize bias, or as an open or single-blind test if the clinician believes the risk of bias is low. If the blinded challenge result is negative, it can be repeated with an open, supervised feeding of a typical serving of the food to rule out a false-negative result. In addition to confirming a food allergy diagnosis, allergists use the OFC to determine the threshold dose that triggers a reaction and to assess whether a patient has outgrown a previously diagnosed food allergy. Although the OFC is effective, it is costly, resource-intensive, and carries a low but significant risk of anaphylaxis and potentially fatal outcomes.[\[26\]](#)

When individuals present with a clear history of an immediate allergic reaction to a specific food and have a positive SPT or elevated sIgE, an OFC is often unnecessary to confirm the diagnosis. If symptoms resolve following the elimination of the suspected food, clinicians and families can reasonably conclude a food allergy without proceeding to an OFC. However, patients with uncontrolled **asthma**, recent anaphylaxis, acute illness, or those in settings lacking immediate access to emergency care should not undergo an OFC, as it carries a higher risk of severe allergic reactions compared to skin and serum testing.

Currently, no reliable testing exists to diagnose non-IgE-mediated food allergies, such as FPIES, food protein-induced enteropathy, and FPIAP. These conditions are diagnosed clinically, based on a detailed history and physical examination findings. A supportive history includes the timing and pattern of symptoms following food exposure, symptom improvement after eliminating the suspected allergen, and symptom recurrence upon reintroduction of the food.

Treatment / Management

Managing food allergies requires strict avoidance of allergenic foods, patient-specific education, and regular follow-up to reassess tolerance and adjust dietary plans as necessary.[\[27\]](#) An exception to strict avoidance is seen in patients with oral allergy syndrome, in which heat-induced protein denaturation allows them to tolerate certain allergenic foods when cooked or baked, despite experiencing symptoms when consuming the same foods raw. The American College of Allergy, **Asthma** & Immunology and the National Institute of Allergy and Infectious Diseases provide comprehensive guidelines, including counseling families on proper food preparation, reading ingredient labels, and understanding the risks of cross-contamination and hidden allergens. Over time, many children and some adults with food allergies may develop tolerance, making regular

reassessment critical for determining whether the allergy persists.[20] If symptoms persist despite adherence to an elimination diet, food allergies are unlikely to be the cause.

Prompt administration of epinephrine is the first-line treatment for anaphylaxis. Patients or caregivers should administer an epinephrine autoinjector (EAI) at the first sign of anaphylactic symptoms, then immediately transfer to an emergency facility for further evaluation and treatment.[10] to epinephrine's short half-life, a second injection is often required 5 to 15 minutes after the initial injection. The recommended dose of epinephrine is 0.1 mg/kg, with a maximum dose of 0.3 mg for children and 0.5 mg for adults. In the United States, epinephrine autoinjectors are available in 3 strengths: 0.1 mg, 0.15 mg, and 0.3 mg. A 0.5 mg dose is also available in Canada and some European countries

The FDA approved inhaled epinephrine (Neffy; ARS Pharmaceuticals) in 2025 for the emergency treatment of type I allergic reactions, including anaphylaxis, in patients aged 4 and older with a body weight of at least 15 kg. This needle-free alternative is beneficial for patients who fear injections or struggle to use an EAI correctly.[29][30] Inhaled epinephrine also offers a longer shelf life than EAI, providing additional convenience for patients. In March 2026, the FDA approved an update removing the age criteria for the 1 mg dose of epinephrine nasal spray, allowing all patients weighing at least 15 kg to access the therapy.[ARS Pharmaceuticals. [ARS Pharmaceuticals Receives FDA Approval to Remove Age Requirement From neffy® 1 mg \(epinephrine nasal spray\) Label](#). March 27, 2026]

Clinicians may administer adjunctive medications, such as antihistamines, corticosteroids, and inhaled beta-agonists, to reduce symptoms of anaphylaxis. However, these medications should never replace epinephrine as the initial treatment of choice.[31] In community settings, patients frequently attempt to manage systemic allergic reactions with antihistamines before administering an EAI, delaying appropriate intervention and increasing the risk of anaphylaxis.

Allergen immunotherapy, including oral, sublingual, and epicutaneous approaches, aims to desensitize individuals with food allergies to specific antigens, thereby reducing the severity of reactions and improving their quality of life.[32] This approach induces tolerance by gradually increasing exposure to allergenic extracts over weeks to months.[8][33] Newer biological therapies, such as omalizumab (an anti-IgE monoclonal antibody), target key pathways involved in systemic allergic responses and have demonstrated efficacy in raising the reactivity threshold to food allergens, particularly when used in conjunction with oral immunotherapy. In 2024, the FDA approved omalizumab for the treatment of food allergies. Additional biologics are currently under investigation to evaluate their safety and effectiveness.[34][35]

The American College of Allergy, **Asthma** & Immunology's 2023 Practice Parameter Update emphasizes eliminating food allergens from the diet, using epinephrine, and ensuring education and preparedness for managing allergic reactions in community settings. These evolving approaches, from allergen avoidance and emergency preparedness to immunotherapy and biological therapies, reflect a comprehensive and increasingly personalized approach to treating patients with food allergies.[28]

Differential Diagnosis

The differential diagnosis of food allergies encompasses both immune-mediated and nonallergic disorders that present with a range of symptoms similar to those of food allergies. Eosinophilic gastrointestinal disorders, such as eosinophilic esophagitis, eosinophilic gastroenteritis, and eosinophilic colitis, are chronic, immune-mediated conditions characterized by eosinophilic infiltration of the gastrointestinal tract. These disorders are associated with Th2 cell activity and are often triggered by food allergens.[\[36\]](#) Eosinophilic esophagitis, the most common of these disorders, typically presents in school-age children to midlife and is frequently observed in individuals with a personal or family history of atopic conditions, including **asthma**, eczema, rhinitis, and IgE-mediated food allergies. Patients commonly experience dysphagia, vomiting, and reflux-like symptoms.

Mast cell activation syndrome is an immune-mediated condition characterized by the inappropriate activation of mast cells, leading to the release of mediators such as histamine and tryptase. This release causes a variety of symptoms across multiple organ systems. The symptoms can resemble those of food allergies, including gastrointestinal distress, rashes, and anaphylaxis.[\[28\]](#)

Patients with celiac disease experience an autoimmune reaction to gluten. Symptoms often include abdominal pain after gluten ingestion, chronic diarrhea, weight loss, fatigue, and anemia. Celiac disease is typically diagnosed through serological testing or a small bowel biopsy. Clinicians must also differentiate allergic reactions from non-food causes, such as medications or insect stings, which can produce symptoms coinciding with food ingestion.

Nonallergic conditions that present with symptoms similar to those of food allergies include lactose intolerance, fructose malabsorption, gastroesophageal reflux disease (GERD), irritable bowel syndrome, histamine intolerance, and panic or anxiety-related reactions. For instance, lactose intolerance may cause bloating and diarrhea after consuming dairy products, while GERD-induced regurgitation may be mistaken for food allergy-related vomiting. Infants with symptoms such as vomiting, lethargy, and poor weight gain may have inborn errors of metabolism, such as galactosemia. Panic attacks can lead to throat tightness, dizziness, and hyperventilation, further complicating the differential diagnosis.

Histamine intolerance, a less common condition, can mimic the symptoms of food allergy reactions. Affected individuals may experience headache, flushing, and urticaria after consuming histamine-rich foods such as aged cheeses, processed meats, fermented products, and alcohol. Additionally, the ingestion of foods containing additives or spices can lead to irritant or vasomotor responses that resemble symptoms of food allergy, including flushing, nasal congestion, headache, and a burning sensation in the mouth.

Other nonallergic conditions that can cause urticaria include infections, such as viral illnesses or reactions to enterotoxins produced by *Staphylococcus aureus*. Foodborne gastroenteritis caused by pathogens such as *Salmonella* or norovirus may be mistaken for food allergies, as symptoms like vomiting and diarrhea can occur after consuming contaminated food. Because both immune-mediated and nonallergic conditions can mimic food allergies, thorough history-taking and diagnostic evaluation are essential. Important differential diagnoses include eosinophilic gastrointestinal disorders, mast cell activation syndrome, celiac disease, food intolerances, infections, and anxiety-related symptoms.

Prognosis

The prognosis for patients with food allergies varies based on several factors, including the specific allergen, age of onset, severity of reaction, and the presence of comorbid conditions such as **asthma** or atopic dermatitis. Children with allergies to soy, wheat, milk, and eggs often outgrow them. Most develop tolerance to soy and wheat by school age, and approximately 80% outgrow milk and egg allergies by adolescence. Notably, about 75% can tolerate these foods in baked form, even among those who remain allergic to milk and egg. In contrast, allergies to peanuts, tree nuts, fish, and shellfish are more likely to persist into adulthood. Only about 20% of individuals with a peanut allergy eventually outgrow it, and the likelihood is even lower for those with tree nut or shellfish allergies, which are typically lifelong.^[8] Non-IgE-mediated food allergies usually resolve within the first few years of life.

Patients at increased risk for persistent or severe food allergies include those with elevated allergen-specific IgE levels, a history of anaphylaxis or severe systemic reactions, and children who have had delayed introduction of allergenic foods. The presence of **asthma**—especially when combined with a history of anaphylaxis—further increases the risk of life-threatening reactions. Importantly, individuals who have previously experienced only mild reactions may still develop systemic responses or anaphylaxis upon future exposures, underscoring the unpredictable nature of food allergy severity.

Reports reveal significant disparities in food allergy outcomes, with racial and ethnic minorities and individuals living in high-poverty areas experiencing higher rates of emergency department visits, hospitalizations, and fatalities due to food-induced anaphylaxis. Multiple studies have documented increased diagnostic coding for food-induced anaphylaxis among Black, Hispanic, and Asian children compared to White children. Black individuals, in particular, are disproportionately affected by comorbid **asthma**—a known risk factor for fatal anaphylaxis. Mortality data further reveal a greater burden of fatal reactions among Black individuals, especially males.^[37] These findings underscore the urgent need for targeted public health strategies to address inequities in the diagnosis, management, and outcomes of food allergy.

With strict avoidance of known food allergens and appropriate emergency preparedness, most individuals with food allergies can lead full, healthy lives. For those who do not outgrow their allergies, immunotherapy and emerging biological treatments offer promising options for desensitization. However, food-related anxiety and overly restrictive diets can adversely affect a child's prognosis, leading to poor nutrition, impaired growth, and diminished quality of life. Elimination of foods based on unconfirmed allergies may deprive children of essential nutrients. Moreover, anxiety surrounding food can contribute to social isolation and emotional distress for both children and their families, underscoring the importance of psychosocial support in improving long-term outcomes.

Complications

Complications of food allergies are diverse and can significantly impact health and development. The most severe complication of IgE-mediated food allergy is life-threatening anaphylaxis, which requires prompt intramuscular administration of epinephrine. Peanuts are the leading cause of food-induced anaphylaxis.^[8] Risk factors for severe reactions include a history of systemic reactions, comorbid **asthma**, and delayed use of epinephrine. In infants with FPIES, severe vomiting and diarrhea can lead to hypovolemic shock. Food allergies may exacerbate atopic dermatitis, particularly

in response to allergens such as milk, eggs, and peanuts. In patients with **asthma**, ingestion of food allergens can also trigger wheezing and other respiratory symptoms.

Children with multiple food allergies are at increased risk for nutritional deficiencies, growth delays, and feeding challenges due to the dietary restrictions required to avoid allergens. These limitations can also contribute to heightened food-related anxiety, diminished quality of life, and social isolation in both children and adults. Adolescents, in particular, may be vulnerable to developing selective eating patterns or eating disorders. Early intervention, combined with psychosocial and nutritional support, is crucial for promoting healthy development and preventing long-term complications.

Consultations

Primary care clinicians often make the initial diagnosis of food allergy and collaborate with specialists, such as allergists, immunologists, registered dietitians, dermatologists, gastroenterologists, and pulmonologists, for comprehensive evaluation and management. In cases of anaphylaxis, emergency physicians provide acute stabilization. They may involve intensive care specialists or anesthesiologists when airway management, hemodynamic monitoring, or vasopressor support is needed due to severe or refractory symptoms.

Deterrence and Patient Education

Deterrence and comprehensive patient education are essential for minimizing the risk of anaphylaxis in individuals with known food allergies. Key strategies include strict avoidance of identified allergens, ensuring access to and correct use of epinephrine, and educating patients, families, and caregivers on recognizing and responding promptly to allergic reactions. In contrast, prevention efforts aim to lower the overall incidence of food allergies by promoting early dietary interventions, such as the timely introduction of allergenic foods during infancy.

Patients and their caregivers must be able to accurately read and interpret food labels to prevent accidental exposure to allergens. In the United States, the Food Allergen Labeling and Consumer Protection Act of 2004 requires that packaged foods clearly disclose the presence of any of the 8 major allergens, including milk, soy, egg, wheat, fish, shellfish, nuts, and peanuts. In 2021, legislators added sesame to the list of major food allergens. The act mandates that food labels must present allergen information in plain language, either within the ingredient list or in a separate "Contains" statement. Manufacturers may also include voluntary advisory warnings, such as "may contain trace amounts of nuts" or "produced in a facility that also processes nuts." In the European Union, the Food Information for Consumers regulation requires listing 5 additional allergens, including gluten, celery, mustard, sulfites, and lupin.[38]

Families managing food allergies must navigate grocery shopping and dining out. Patient education should emphasize the importance of thoroughly reviewing restaurant menus, inquiring about potential allergens in dishes, and clearly communicating food allergies to restaurant staff when placing an order. Food Allergy Research & Education (FARE) is a valuable resource for families, offering reliable and up-to-date information at www.foodallergy.org. [39]

When individuals are diagnosed with a food allergy, patient and caregiver education should include how to identify the symptoms of a systemic reaction or anaphylaxis. A systemic reaction involves more than one organ system and may present with symptoms such as urticaria, nausea, or sneezing, ranging from mild to moderate in severity. Anaphylaxis, on the other hand, is a severe, potentially life-threatening reaction that typically has a rapid onset and involves 2 or more organ systems, often including hypotension and airway compromise.

Patients and caregivers must be able to recognize these reactions and understand when and how to use EAls.[20] Proper EAI use reduces the risk of accidental injection and enhances treatment effectiveness. Clear instructions should emphasize injecting into the anterolateral thigh using the "place and press" technique to avoid finger injuries. There are documented cases of individuals accidentally injecting epinephrine into their thumbs after holding the autoinjector upside down.[28] Demonstration autoinjectors are valuable training tools that enable users to practice key steps—such as removing the safety cap, holding the device properly, and simulating the injection—without administering medication.

Patients should receive written action plans for managing allergic reactions and regularly review them with nurses or healthcare providers.[20] In addition, it is more common for individuals to fail to use epinephrine when needed than to use it unnecessarily. Many primary care clinicians advise, "If there's more than skin, Epi goes in," emphasizing prompt use of an autoinjector for any allergic symptoms beyond urticaria. Epinephrine is only effective in treating anaphylaxis if it is readily accessible; therefore, patients must carry it at all times, including at school, work, and while traveling. Guidelines from the American Academy of Allergy, **Asthma**, & Immunology and the American College of Allergy, **Asthma** & Immunology recommend that patients using epinephrine for anaphylaxis seek prompt medical evaluation and consider transport to an emergency department for monitoring and care.[28]

Schools and daycare programs play a crucial role in preventing anaphylactic reactions. In 2019, New York was the first state in the United States to pass Elijah's Law, named after a child (aged 3) with a known milk allergy who tragically died after being served a grilled cheese sandwich at daycare, despite staff being aware of his allergy. The law mandates comprehensive allergy management protocols in schools and childcare facilities, including staff training, clear communication of allergy information, safe food service practices, access to epinephrine, and individualized emergency care plans. Since its enactment, several other states have adopted similar legislation to enhance protection for children with food allergies. [**Asthma** and Allergy Foundation of America, Elijah-Alavi Foundation, (2022). *Child Care Policies for Food Allergy: Elijah's Law Report for U.S. States and Territories*. Retrieved from aafa.org/ElijahsLaw.]

Although genetic predisposition contributes to the development of food allergies, adopting specific dietary strategies may help reduce the risk in young children. Exclusive breastfeeding has a protective effect, likely because breast milk contains essential immune factors that support the maturation of the infant's immune system. This may reduce the risk of developing food allergies, as well as other allergic conditions such as atopic dermatitis and **asthma**. The AAP recommends exclusive breastfeeding for the first 6 months of life.[40] Additionally, current evidence does not support avoiding potentially allergenic foods during pregnancy or lactation as a strategy to prevent the development of food allergies in infants.

The AAP also recommends offering allergenic foods, such as peanuts and eggs, to infants at high risk of food allergies, starting around 4 to 6 months of age, including those with severe atopic dermatitis or a strong family history of allergies. This advice represents a significant shift from earlier guidelines that advised delaying the introduction of such foods.[\[41\]](#)[\[42\]](#) Clinical studies, including the Learning Early About Peanut Allergy (LEAP) trial, have demonstrated that early introduction to peanuts significantly reduces the risk of developing a peanut allergy. In the LEAP trial, infants who began regular peanut consumption between 4 and 11 months had an 81% lower incidence of peanut allergy at age 5 compared to those who avoided peanuts..[\[43\]](#) Similarly, the early introduction of eggs has been associated with a reduced incidence of egg allergy.[\[44\]](#)

The AAP's guidance on the timing of complementary food introduction aims to balance the benefits of exclusive breastfeeding with reducing the risk of food allergies in high-risk infants. The early introduction of allergenic foods before 6 months is recommended for specific high-risk groups, rather than as a universal strategy. While most clinicians recognize that many healthy, low-risk infants begin complementary foods before 6 months, the AAP advises introducing one food at a time to monitor for adverse reactions and identify specific allergens. The Enquiring About Tolerance (EAT) study, however, demonstrated that introducing multiple allergenic foods early is feasible and safe, without negatively affecting breastfeeding.[\[45\]](#)

Enhancing Healthcare Team Outcomes

Effective management of food allergies relies on a coordinated, interprofessional approach to ensure comprehensive care for both children and adults. Primary care clinicians and nurses play a key role in educating patients and caregivers about allergen avoidance, emergency preparedness, and proper use of EAls.

Approximately 25% of first-time allergic reactions occur in schools. Nurses are present in about 79% of public schools in the United States and play a vital role in training staff to recognize anaphylaxis and administer epinephrine, as well as promoting a culture of shared responsibility, encouraging teachers and cafeteria staff to help prevent allergic reactions by cleaning surfaces, checking snack labels, and ensuring EAls are accessible on playgrounds and during field trips.[\[46\]](#) Physicians, school nurses, teachers, and support staff must collaborate to create and maintain individualized allergy action plans that include strategies to reduce the risk of allergen ingestion, as well as procedures for recognizing and treating allergic reactions and anaphylaxis. Physicians provide medical expertise, prescriptions, and documentation, while school nurses translate these into daily practice, coordinate staff training, and serve as on-site clinical leaders. This coordinated approach ensures that children with food allergies are kept safe, supported, and included in school activities.[\[47\]](#)

Both schools and daycare centers should implement standard allergy protocols, maintain current individualized allergy action plans, and stock non-designated EAls for emergency use. The U.S. School Access to Emergency Epinephrine Act supports these efforts by encouraging states to mandate the stocking of epinephrine in schools and granting legal immunity to those who prescribe or administer it in good faith.[\[48\]](#)

Pharmacists play a crucial role in educating caregivers on the proper administration of medications, the safe storage of epinephrine, and identifying potential drug-food interactions. Registered dietitians guide patients and families in reading food labels, spotting hidden allergens, and creating safe,

nutritionally balanced diets. Additionally, allergist specialists such as allergists, immunologists, and other medical professionals contribute to long-term care by advising on prevention strategies and treatment of reactions. Close coordination among healthcare and educational professionals is vital to deliver comprehensive, evidence-based care tailored to each patient's needs.

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