Food Allergies: Diagnosis, Treatment, and Prevention

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In the United States, approximately 2% to 3% of adults and 8% of children have a food allergy. Allergic reactions range from minor pruritus to life-threatening anaphylaxis. These allergies often lead to significant anxiety and costs for patients and caregivers. Common food allergies include peanuts, cow's milk, shellfish, tree nuts, egg, fish, soy, and wheat. Peanut allergy, the most common, is the leading cause of life-threatening anaphylaxis. Children with asthma, allergic rhinitis, atopic dermatitis, or an allergy to insect venom, medications, or latex are at an increased risk of developing food allergies. Diagnosis of food allergy starts with a detailed, allergy-focused history. Serum immunoglobulin E and skin prick testing provide reliable information regarding food allergy diagnoses. Primary treatment involves elimination of the offending food from the diet. Prevention strategies proven to decrease the risk of developing a food allergy include restricting exposure to cow's milk in the first three days of life and early sequential exposure to allergenic foods starting between four and six months of age. Exclusive breastfeeding for three to four months reduces the likelihood of developing eczema and asthma but does not reduce development of food allergies. Most children eventually outgrow allergies to cow's milk, egg, soy, and wheat. However, allergies to tree nuts, peanuts, and shellfish are more likely to be lifelong. (*Am Fam Physician*. 2023;108(2):159-165. Copyright © 2023 American Academy of Family Physicians.)

In the United States, approximately 2% to 3% of adults and 8% of children have a food allergy, and 40% of those children have multiple food allergies.[1,2] About 40% of food allergies in children are reported as severe, which can lead to significant costs and anxiety for parents and caregivers.[2] Common foods that produce allergies are peanuts, cow's milk, shellfish, tree nuts, egg, fish, soy, and wheat.[2,3] Peanut allergy, the most common (2%), is the leading cause of life-threatening anaphylaxis.[2,4] Children are likely to outgrow allergies to egg, cow's milk, wheat, and soy, whereas peanut, tree nut, fish, and shellfish allergies tend to persist throughout life.[3] Peanut allergy resolves in approximately 1 in 5 children in the first four years of life.[5]

CME This clinical content conforms to AAFP criteria for CME. See CME Quiz on page 126.

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Risk Factors

Children with asthma, allergic rhinitis, atopic dermatitis, or an allergy to insect venom, medications, or latex are at an increased risk of developing food allergies[2,6] *(Table 1[2])*. Children with vitamin D insufficiency, a history of antibiotic use

TABLE 1

Risk Factors for Food Allergies

Risk factor	Odds ratio
Latex allergy	7.9
Asthma	3.2
Urticaria	2.9
Insect venom allergy	2.5
Allergic rhinitis	2.3
Atopic dermatitis	1.9
Medication allergy	1.9

Information from reference 2.

SORT: KEY RECOMMENDATIONS FOR PRACTICE

Clinical recommendation	Evidence rating	Comments
Serum immunoglobulin E or skin prick testing should be performed only when a detailed allergy-focused history indicates a high pretest probability of a food allergy.[3,9,12]	C	Expert opinion and consensus guideline in the absence of high-quality clinical trials
Early introduction of peanuts, cow's milk, wheat, and cooked eggs between four and six months of age decreases the risk of developing food allergies.[16-18]	A	Consistent results from randomized controlled trials and observational studies showing decreased development of food allergies
Early introduction of peanuts and cooked eggs at four to six months of age is safe and effective for reducing the risk of food allergy development in high-risk infants.[12,15,19,20]	B	Randomized controlled trials that show decreased likelihood of developing food allergies at 36 months

A = consistent, good-quality patient-oriented evidence; **B** = inconsistent or limited-quality patient-oriented evidence; **C** = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to https://www.aafp.org/afpsort.

in the first two years of life, or a family history of atopy also have an increased risk of developing food allergies.[3,7,8] Other factors, including exercise, emotional stress, menses, alcohol consumption, and having a viral infection, can lower the reaction threshold and increase the risk of having an allergic reaction to food.[4,6]

Symptoms

Food allergies are classified as immunoglobulin E (IgE)- and non–IgE-mediated. IgE-mediated allergies typically have a rapid onset, within seconds to minutes (e.g., pruritus, anaphylaxis). Non–IgE-mediated food allergies are characterized by delayed reactions, within hours to several days (e.g., food protein allergy–induced colitis).[6,9,10] The severity of the reaction is influenced by the amount of food ingested, form of the food (i.e., how it was prepared), and presence of other ingested foods.[6] Symptoms of food allergies are listed in *Table 2*.[3,6,9,11]

Diagnosis

The rates of food allergies are overestimated because of self-reported food allergies that may be food intolerances.[1,12] Food intolerances are adverse reactions without an immunologic cause (e.g., lactose intolerance) and can be mistaken for food allergies.[6] Differential diagnosis of food allergies is included in *Table 3*.[1,6]

Diagnosis starts with a detailed history from the parent, caregiver, or patient. The amount and type of food that was eaten, form of the food (e.g., raw, extensively baked), time from ingestion to symptoms, presence of symptoms not associated with food, presence of risk factors that increase the likelihood of an allergic reaction (e.g., exercise), and number of reactions are essential components of an allergy-focused history.[6,9,10,12] Non–IgE-mediated food allergies should be considered in children without an adequate response to treatment for atopic dermatitis, gastroesophageal reflux disease, and chronic gastrointestinal symptoms, including chronic constipation.[9]

BEST PRACTICES IN ALLERGY AND IMMUNOLOGY

Recommendations From Choosing Wisely

Recommendation	Sponsoring organization
Do not perform screening panels for food allergies without previous consideration of medical history.	American Academy of Pediatrics
Do not perform food serum IgE testing without a history consistent with potential IgE-mediated food allergy.	American Academy of Allergy, Asthma and Immunology
Do not routinely avoid influenza vaccination in patients allergic to egg.	American Academy of Allergy, Asthma and Immunology

IgE = immunoglobulin E.

Source: For more information on Choosing Wisely, see https://www.choosingwisely.org. For supporting citations and to search Choosing Wisely recommendations relevant to primary care, see https://www.aafp.org/pubs/afp/collections/choosing-wisely.html.

TABLE 2

Symptoms of Food Allergies and Classification of Reactions

Organ system	Minor symptoms	Major symptoms/sequelae
Cardiovascular	Tachycardia	Hypotension, shock, syncope
Gastrointestinal	Blood or mucus in stools, colicky abdominal pain, constipation, diarrhea, fatigue, food aversion/refusal, nausea, oral pruritus, pallor, perianal redness, vomiting	Eosinophilic esophagitis, failure to thrive, growth delay, immune-mediated enterocolitis
Respiratory	Congestion, conjunctivitis, cough, nasal itching, rhinorrhea, sneezing, upper respiratory tract infection symptoms	Chest tightness, Heiner syndrome,* shortness of breath, wheezing
Skin	Acute urticaria, erythema, pruritus, worsening of atopic dermatitis	Angioedema (most commonly lips, face, and periorbital area)
Other	—	Anaphylaxis, systemic allergic reaction

*—Heiner syndrome is a rare disorder in infants caused by ingestion of cow's milk that results in lower respiratory tract symptoms.

Information from references 3, 6, 9, and 11.

Serum IgE or skin prick testing (SPT) should be performed only in patients whose allergy-focused history indicates a high pretest probability of a food allergy. Doing so promptly can prevent unnecessary food avoidance and strain for patients and their families.[3,9,12]

SPT should be performed only in facilities equipped to handle anaphylactic reactions and interpreted by clinicians with an adequate knowledge of history, clinical findings, and allergen pathology.[9] SPT can be performed on patients of any age and has high sensitivity and high negative predictive values.[1,6] Children younger than two years and adults older than 70 years may have smaller positive results.[1] The likelihood of allergy reactivity can be estimated with correlation of wheal size.[3,10]

SPT has greater sensitivity than serum IgE testing.[1] Drawbacks of SPT include the lack of standardization of the extracts used and the interference of antihistamine and certain antidepressant medications.[6,10] Before SPT, patients should abstain from the use of high-potency topical steroids on test sites for three weeks and discontinue any antihistamines and antidepressants with antihistamine properties (e.g., tricyclic antidepressants) for one week.[1] It is not necessary to stop leukotriene receptor antagonists, which are often used in the treatment of asthma.[1]

Serum IgE testing is preferable to SPT in patients at high risk of anaphylaxis.[1] Patients who cannot tolerate the SPT procedure, have uncontrolled medical conditions (e.g., uncontrolled asthma), take essential medications (e.g., beta blockers for coronary artery disease), or have skin conditions that could interfere with SPT should instead undergo serum IgE testing.[1]

Widely available serum IgE tests provide reliable measures with standardization of accurate laboratory techniques and specified 95% positive predictive values of reactivity within a population.[1,6] Serum IgE testing can indicate sensitization to a specific allergen. Care is necessary when interpreting serum IgE results. A positive test result does not necessarily indicate the existence of a food allergy, and the level of specific IgE does not correlate to symptom severity.[1,10] A negative test result may indicate that a patient has not been sensitized (i.e., exposed) to the allergen, leading to an incorrect dismissal of a food allergy.[1]

Less commonly used allergy testing is typically done by an allergist and includes intradermal tests, atopy patch tests, and basophil activation tests.[1] Intradermal testing can be performed after a negative result on SPT when the clinician still has a strong suspicion of allergy. However, there is a greater risk of systemic allergic reactions during testing without increased sensitivity over SPT.[1,6] Atopy patch testing is indicated for cell-mediated hypersensitivity reactions, such as medication or latex allergies.[1] Use of basophil activation testing is limited as an unproven and nonstandardized test for diagnosis of food allergy.[1,6]

The preferred method of food allergy diagnosis is a double-blind oral food challenge.[11,12] An oral food challenge can reliably disprove a previously diagnosed nonsevere food allergy or confirm an already diagnosed food allergy. It should be done under physician supervision and in a setting capable of managing severe reactions.[10,12] If a child has severe allergic symptoms after a known ingestion and an oral food challenge is not feasible, having no further symptoms after elimination of the food is sufficient for diagnosis.[11]

TABLE 3

Differential Diagnosis of Food Allergy

Allergic reaction to another substance (e.g., medications, insect venom)
Behavioral or mental conditions that lead to food aversion (e.g., anorexia nervosa, Munchausen syndrome by proxy)
Chemical/irritant adverse effects from food additives, preservatives, or coloring
Chronic urticaria
Food intolerance (e.g., lactose-induced gastrointestinal symptoms)
Gastroesophageal reflux disease
Gastrointestinal infections (viral, bacterial, parasitic)
Inflammatory bowel disease
Irritable bowel syndrome
Vasomotor adverse effects from food (e.g., rhinitis from spicy or tart foods)

Information from references 1 and 6.

Treatment and Management

ELIMINATION DIET

The primary treatment for a food allergy is elimination of the offending food from the diet.[11] Nutrition counseling and regular growth monitoring are warranted when multiple foods are eliminated.[11] The National Institute of Allergy and Infectious Diseases recommends that families and caregivers receive training on how to understand ingredient lists on food labels.[6] The U.S. Food Allergen Labeling and Consumer Protection Act of 2004 requires food labels to list when any of the nine major food allergens (i.e., milk, egg, peanuts, tree nuts, soy, wheat, fish, crustacean shellfish, and sesame) are present as ingredients in prepared foods.[6,13] However, including warning labels such as "may contain trace amounts of nuts" or "may be prepared in a facility that also uses nuts" is voluntary.[6]

ALLERGY IMMUNOTHERAPY

No medications are effective for preventing IgE- or non–IgE-mediated allergic reactions to food.[11] Oral and sublingual immunotherapies are methods of desensitization therapy. These treatments induce tolerance to an allergenic food by gradually increasing the dose of the allergenic extract.[3] Patients receiving oral immunotherapy for peanut, egg, and milk allergies are more tolerant of the allergenic food in oral food challenges than patients receiving placebo.[3] Local gastrointestinal and systemic reactions requiring epinephrine have been reported with oral immunotherapy.[3] Patients are more likely to achieve clinical desensitization with oral immunotherapy, but there is a higher risk of adverse effects.[4] Peanut desensitization therapy increases the risk of anaphylactic reactions compared with placebo or avoidance.[14] Immunotherapy (oral and sublingual) with cross-reacting allergens is not recommended to treat food allergies.[11]

MEDICATIONS FOR ANAPHYLAXIS MANAGEMENT

Epinephrine is the initial drug of choice for the management of food-induced anaphylaxis.[4] Epinephrine has a short half-life (minutes) and often requires a second dose for treatment of persistent or recurrent symptoms.[4] Antihistamines, glucocorticoids, and inhaled beta$_2$ agonists can be useful for reducing symptoms but should not be used as first-line treatment for anaphylaxis[4] *(Table 4[6])*. Epinephrine is available as autoinjectors in three doses. Patients and parents should be instructed on the use and administration of epinephrine and need for further evaluation in the emergency department because hospitalization may be required.[4]

Prevention

Although exclusive breastfeeding for three to four months reduces the likelihood of the child developing eczema and asthma, it has not been shown to reduce the development of food allergies.[6,15] Evidence does not support restricting a mother's diet during pregnancy or while breastfeeding to reduce the risk of food allergies.[6,15,16] Restricting cow's milk supplementation for the first three days of life is associated with a significantly lower risk of developing cow's milk, egg, and wheat allergies by 24 months of age in infants who have an increased risk of atopy.[17] Evidence indicates that infants with early sequential exposure to allergenic foods (peanuts, cow's milk, wheat, and cooked eggs) between four and six months of age are less likely (number needed to treat = 63) to develop food allergies at 36 months than unexposed infants.[16-18] The timing of gluten introduction is not associated with the development of celiac disease, type 1 diabetes mellitus, or any other autoimmune disease.[19]

Guidelines for the prevention of peanut allergies recommend that infants with severe eczema, egg allergy, or both be introduced to age-appropriate, peanut-containing food as early as four to six months of age.[12,15,19,20] Other solid foods should be introduced before peanut-containing foods to confirm that the infant is developmentally ready.[15,19] Infants with mild to moderate eczema should start ingesting age-appropriate, peanut-containing food around six months of age[19] *(eTable A)*. In infants without eczema or any food allergy, age-appropriate peanut-containing foods may be

TABLE 4

Medications for Anaphylaxis Management

Outpatient setting

First-line treatment:

Intramuscular epinephrine (autoinjector)

10 to 25 kg (22 to 56 lb): 0.15-mg epinephrine autoinjector (anterolateral thigh)

> 25 kg: 0.3-mg epinephrine autoinjector (anterolateral thigh)

Intramuscular epinephrine (1:1,000 solution)

0.01 mg per kg per dose (anterolateral thigh); maximum dose: 0.5 mg

Epinephrine doses may need to be repeated every five to 15 minutes

Adjunctive treatment:

Bronchodilator (short-acting beta$_2$ agonist): albuterol metered dose inhaler (child: 4 to 8 puffs; adult: 8 puffs) or nebulized solution (child: 1.5 mL; adult: 3 mL) every 20 minutes or continuously as needed

H_1 antihistamine: diphenhydramine, 1 to 2 mg per kg per dose; maximum dose: 50 mg intravenously or orally (oral liquid is more readily absorbed than tablets); a less sedating second-generation antihistamine may be used as an alternative

Supplemental oxygen therapy

Intravenous fluids in large volumes if patient presents with orthostasis, hypotension, or incomplete response to intramuscular epinephrine

Place the patient in recumbent position, if tolerated, with the lower extremities elevated

Hospital-based setting

First-line treatment:

Intramuscular epinephrine (autoinjector)

10 to 25 kg: 0.15-mg epinephrine autoinjector (anterolateral thigh)

> 25 kg: 0.3-mg epinephrine autoinjector (anterolateral thigh)

Intramuscular epinephrine (1:1,000 solution)

0.01 mg per kg per dose (anterolateral thigh); maximum dose: 0.5 mg

Epinephrine doses may need to be repeated every five to 15 minutes

Consider continuous epinephrine infusion for persistent hypotension, ideally with continuous noninvasive monitoring of blood pressure and heart rate; alternatives are endotracheal and intraosseous epinephrine

Adjunctive treatment:

Bronchodilator (short-acting beta$_2$ agonist): albuterol metered dose inhaler (child: 4 to 8 puffs; adult: 8 puffs) or nebulized solution (child: 1.5 mL; adult: 3 mL) every 20 minutes or continuously as needed

continues

introduced with other solid foods in accordance with family preferences and cultural practices.[18-20]

Immunizations for Patients With Egg Allergy

Patients with egg allergy, even those with a history of severe reactions, can safely receive vaccines for measles, mumps, rubella, and varicella.[6] However, vaccines for rabies and yellow fever should not be given to patients with a history of urticaria (hives), angioedema, allergic asthma, or systemic anaphylaxis to egg protein unless an allergy evaluation is performed.[6] For influenza vaccines, health care professionals should follow current vaccine recommendations from the Advisory Committee on Immunization Practices.[11] Most available influenza vaccines, with the exceptions of the quadrivalent recombinant influenza vaccine (RIV4; Flublok) and cell culture–based quadrivalent inactivated influenza vaccine (ccIIV4; Flucelvax), are prepared by propagation of the virus in embryonated eggs and might contain trace amounts of egg proteins, such as ovalbumin.[21]

People with egg allergies can safely receive an influenza vaccine if the reaction is limited to urticaria.[21] Individuals who report reactions to egg other than urticaria (e.g., angioedema or swelling, respiratory distress, lightheadedness, recurrent vomiting) or require the use of epinephrine or other emergency medical interventions can also receive any licensed, recommended influenza vaccine.[21] However, if a vaccine other than ccIIV4 or RIV4 is used, administration should occur in an inpatient or outpatient medical setting (e.g., hospitals, clinics, health departments, physician offices) and be supervised by a physician capable of recognizing and managing severe allergic reactions.[21,22]

Prognosis

Most children with food allergies will eventually tolerate cow's milk, egg, soy, and wheat. However, tree nut, shellfish, and peanut allergies are more likely to be lifelong.[6] Most children (75%) with egg and cow's milk allergies can often tolerate baked forms of these foods.[3] The time course of food allergy resolution in children varies by food and may occur as late as their teenage years.[6] An initial high level of allergen-specific IgE against a food is associated with a lower rate of resolution of the allergy over time.[6]

This article updates previous articles on this topic by Yawn and Fenton,[11] Kurowski and Boxer,[23] and Sicherer.[24]

Data Sources: A PubMed search was completed with clinical queries using the key terms food allergies with and without the terms diagnosis, treatment, and prevention. The search included meta-analyses, randomized controlled trials, clinical trials, and reviews. The Cochrane database and Essential Evidence Plus were also searched. Several studies regarding prevalence of food allergies included demographics of race and/or gender as patient categories and were included in our final review. No

TABLE 4 *(continued)*

Medications for Anaphylaxis Management

Hospital-based setting

Adjunctive treatment:

H_1 antihistamine: diphenhydramine, 1 to 2 mg per kg per dose; maximum dose: 50 mg intravenously or orally (oral liquid is more readily absorbed than tablets); a less sedating second-generation antihistamine may be used as an alternative

H_2 antihistamine: famotidine, 1 to 2 mg per kg per dose; maximum dose: 75 to 150 mg intravenously or orally

Corticosteroids: prednisone, 1 mg per kg; maximum dose: 60 to 80 mg orally; or methylprednisolone, 1 mg per kg; maximum dose: 60 to 80 mg intravenously

Vasopressors (other than epinephrine) for refractory hypotension, titrate to effect

Glucagon for refractory hypotension, titrate to effect (child: 20 to 30 mcg per kg; adult: 1 to 5 mg); dose may be repeated or followed by infusion of 5 to 15 mcg per minute

Atropine for bradycardia, titrate to effect

Supplemental oxygen therapy

Intravenous fluids in large volumes if patients present with orthostasis, hypotension, or incomplete response to intramuscular epinephrine

Place the patient in recumbent position, if tolerated, with the lower extremities elevated

At discharge

First-line treatment:

Epinephrine autoinjector prescription (two doses) and instructions

Education on avoidance of allergen

Follow-up with primary care physician

Consider referral to an allergist

Adjunctive treatment:

H_1 antihistamine: diphenhydramine every six hours for two to three days; a less sedating second-generation antihistamine may be used as an alternative

H_2 antihistamine: famotidine, twice daily for two to three days

Corticosteroid: prednisone daily for two to three days

Note: These treatments often occur concomitantly and are not meant to be sequential, except for epinephrine as first-line treatment.

Adapted with permission from Boyce JA, Assa'ad A, Burks AW, et al.; NIAID-Sponsored Expert Panel. Guidelines for the diagnosis and management of food allergy in the United States: report of the NIAID-Sponsored Expert Panel. J Allergy Clin Immunol. *2010; 126(6 suppl):S39.*

studies indicated race and/or gender as variables for delineation of diagnosis, treatment, or prevention. Search dates: August 15, 2022, and May 8, 2023.

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References

1. Ansotegui IJ, Melioli G, Canonica GW, et al. IgE allergy diagnostics and other relevant tests in allergy, a World Allergy Organization position paper [published correction appears in *World Allergy Organ J*. 2021; 14(7):100557]. *World Allergy Organ J*. 2020;13(2):100080.
2. Gupta RS, Warren CM, Smith BM, et al. The public health impact of parent-reported childhood food allergies in the United States [published correction appears in *Pediatrics*. 2019;143(3):e20183835]. *Pediatrics*. 2018;142(6):e20181235.
3. Patel BY, Volcheck GW. Food allergy: common causes, diagnosis, and treatment. *Mayo Clin Proc*. 2015;90(10):1411-1419.
4. Jones SM, Burks AW. Food allergy. *N Engl J Med*. 2017;377(12):1168-1176.
5. Peters RL, Allen KJ, Dharmage SC, et al.; HealthNuts Study. Natural history of peanut allergy and predictors of resolution in the first 4 years of life: a population-based assessment. *J Allergy Clin Immunol*. 2015; 135(5):1257-1266.e1-2.
6. Boyce JA, Assa'ad A, Burks AW, et al.; NIAID-Sponsored Expert Panel. Guidelines for the diagnosis and management of food allergy in the United States: report of the NIAID-Sponsored Expert Panel. *J Allergy Clin Immunol*. 2010;126(6 suppl):S1-S58.
7. Psaroulaki E, Katsaras GN, Samartzi P, et al. Association of food allergy in children with vitamin D insufficiency: a systematic review and meta-analysis. *Eur J Pediatr*. 2023;182(4):1533-1554.
8. Duong QA, Pittet LF, Curtis N, et al. Antibiotic exposure and adverse long-term health outcomes in children: a systematic review and meta-analysis [published correction appears in *J Infect*. 2023;86(1):118]. *J Infect*. 2022;85(3):213-300.
9. National Institute for Health and Care Excellence. Food allergy in under 19s: assessment and diagnosis. Clinical guideline [CG116]. February 23, 2011. Accessed August 8, 2022. https://www.nice.org.uk/guidance/cg116
10. Steele C, Conlon N, Edgar JD. Diagnosis of immediate food allergy. *BMJ*. 2014;349:g3695.
11. Yawn BP, Fenton MJ. Summary of the NIAID-sponsored food allergy guidelines. *Am Fam Physician*. 2012;86(1):43-50.
12. Sicherer SH, Allen K, Lack G, et al. Critical issues in food allergy: a National Academies Consensus Report. *Pediatrics*. 2017;140(2): e20170194.

13. U.S. Food and Drug Administration. Allergic to sesame? Food labels now must list sesame as an allergen. January 10, 2023. Accessed May 8, 2023. https://www.fda.gov/consumers/consumer-updates/allergic-sesame-food-labels-now-must-list-sesame-allergen
14. Chu DK, Wood RA, French S, et al. Oral immunotherapy for peanut allergy (PACE): a systematic review and meta-analysis of efficacy and safety [published correction appears in *Lancet*. 2019;393(10184):1936]. *Lancet*. 2019;393(10187):2222-2232.
15. Greer FR, Sicherer SH, Burks AW; Committee on Nutrition; Section on Allergy and Immunology. The effects of early nutritional interventions on the development of atopic disease in infants and children: the role of maternal dietary restriction, breastfeeding, hydrolyzed formulas, and timing of introduction of allergenic complementary foods. *Pediatrics*. 2019;143(4):e20190281.
16. Halken S, Muraro A, de Silva D, et al.; European Academy of Allergy and Clinical Immunology Food Allergy and Anaphylaxis Guidelines Group. EAACI guideline: preventing the development of food allergy in infants and young children (2020 update). *Pediatr Allergy Immunol*. 2021;32(5):843-858.
17. Urashima M, Mezawa H, Okuyama M, et al. Primary prevention of cow's milk sensitization and food allergy by avoiding supplementation with cow's milk formula at birth: a randomized clinical trial. *JAMA Pediatr*. 2019;173(12):1137-1145.
18. Skjerven HO, Lie A, Vettukattil R, et al. Early food intervention and skin emollients to prevent food allergy in young children (PreventADALL): a factorial, multicentre, cluster-randomised trial. *Lancet*. 2022;399(10344):2398-2411.
19. Slawson DC. Early egg and peanut introduction reduces allergy risk; timing of gluten intro does not reduce risk of celiac disease. *Essential Evidence Plus*. November 24, 2016. Accessed September 8, 2022. https://www.essentialevidenceplus.com/content/poem/181125
20. Johnson JL, Gupta RS, Bilaver LA, et al. Implementation of the Addendum Guidelines for Peanut Allergy Prevention by US Allergists, a survey conducted by the NIAID, in collaboration with the AAAAI. *J Allergy Clin Immunol*. 2020;146(4):875-883.
21. Grohskopf LA, Blanton LH, Ferdinands JM, et al. Prevention and control of seasonal influenza with vaccines: recommendations of the Advisory Committee on Immunization Practices - United States, 2022-23 influenza season. *MMWR Recomm Rep*. 2022;71(1):1-28.
22. Fleisher DM, Sicherer S, Greenhawt M, et al.; American Academy of Allergy, Asthma & Immunology; American Academy of Pediatrics, American College of Allergy; Asthma & Immunology, Australasian Society of Clinical Immunology and Allergy; Canadian Society of Allergy and Clinical Immunology; European Academy of Allergy and Clinical Immunology; Israel Association of Allergy and Clinical Immunology; Japanese Society for Allergology; Society for Pediatric Dermatology; World Allergy Organization. Consensus communication on early peanut introduction and the prevention of peanut allergy in high-risk infants [published correction appears in *J Allergy Clin Immunol*. 2015;136(6):1710]. *J Allergy Clin Immunol*. 2015;136(2):258-261.
23. Kurowski K, Boxer RW. Food allergies: detection and management. *Am Fam Physician*. 2008;77(12):1678-1686.
24. Sicherer SH. Manifestations of food allergy: evaluation and management. *Am Fam Physician*. 1999;59(2):415-424.

eTABLE A

Evaluation of Children With Severe Eczema, Egg Allergy, or Both Before Peanut Introduction

Peanut serum IgE level*		Peanut skin prick test		
< 0.35	≥ 0.35	0.2-mm wheal	3- to 7-mm wheal	≥ 8-mm wheal
Risk of reaction low (more than 90% will have a negative result on peanut skin prick testing) Options: Introduce peanuts at home Supervised feeding in the office (based on physician/parent preference)	Refer to a specialist for consultation and peanut skin prick testing protocol	Risk of reaction low (95% will not have a peanut allergy) Options: Introduce peanuts at home Supervised feeding in the office (based on physician/parent preference)	Risk of reaction varies from moderate to high Options: Supervised feeding in the office Graded oral food challenge in a specialized facility	Infant probably allergic to peanut Continue evaluation and management by a specialist

IgE = immunoglobulin E.

*—To minimize a delay in peanut introduction for children who may test negative, testing for peanut serum IgE levels may be the preferred initial approach in certain health care settings. Food allergen panel testing or the addition of serum IgE testing for foods other than peanuts is not recommended due to poor positive predictive value.[A1]

Information from:

A1. Johnson JL, Gupta RS, Bilaver LA, et al. Implementation of the Addendum Guidelines for Peanut Allergy Prevention by US Allergists, a survey conducted by the NIAID, in collaboration with the AAAAI. J Allergy Clin Immunol. *2020;146(4):875-883.*

A2. Skjerven HO, Lie A, Vettukattil R, et al. Early food intervention and skin emollients to prevent food allergy in young children (PreventADALL): a factorial, multicentre, cluster-randomised trial. Lancet. *2022;399(10344):2398-2411.*

A3. Slawson DC. Early egg and peanut introduction reduces allergy risk; timing of gluten intro does not reduce risk of celiac disease. Essential Evidence Plus. *November 24, 2016. Accessed September 8, 2022. https://www.essentialevidenceplus.com/content/poem/181125*