

Food Allergies: Detection and Management

KURT KUROWSKI, MD, *The Chicago Medical School at Rosalind Franklin University of Medicine and Science, North Chicago, Illinois*

ROBERT W. BOXER, MD, *Rush North Shore Medical Center, Skokie, Illinois*

Family physicians play a central role in the suspicion and diagnosis of immunoglobulin E-mediated food allergies, but they are also critical in redirecting the evaluation for symptoms that patients are falsely attributing to allergies. Although any food is a potential allergen, more than 90 percent of acute systemic reactions to food in children are from eggs, milk, soy, wheat, or peanuts, and in adults are from crustaceans, tree nuts, peanuts, or fish. The oral allergy syndrome is more common than anaphylactic reactions to food, but symptoms are transient and limited to the mouth and throat. Skin-prick and radioallergen sorbent tests for particular foods have about an 85 percent sensitivity and 30 to 60 percent specificity. Intradermal testing has a higher false-positive rate and greater risk of adverse reactions; therefore, it should not be used for initial evaluations. The double-blind, placebo-controlled food challenge remains the most specific test for confirming diagnosis. Treatment is through recognition and avoidance of the responsible food. Patients with anaphylactic reactions need emergent epinephrine and instruction in self-administration in the event of inadvertent exposure. Antihistamines can be used for more minor reactions. (*Am Fam Physician*. 2008;77(12):1678-1686, 1687-1688. Copyright © 2008 American Academy of Family Physicians.)



ILLUSTRATION BY MICHAEL KREBS-RUSICK

► **Patient information:** A handout on food allergies, written by the authors of this article, is provided on page 1687.

Food allergies affect 4 to 5 percent of children and 2 to 3 percent of adults, yet false attribution of symptoms to food allergy remains a problem.^{1,2} Population-based studies of children and adolescents have shown that only 10 percent of those who believe they have food allergy can be proven to have one.¹ Disorders associated with food allergy, such as eosinophilic esophagitis, are being increasingly recognized, and some other previously known disorders, such as gastroesophageal reflux disease in infants, are being increasingly attributed to food allergies.³ Food allergy is the leading cause of nondrug-related anaphylaxis.

Pathophysiology

Despite high acidity in the stomach and enzyme activity, 2 percent of ingested food is absorbed through the intestine in a form that is immunologically intact enough to produce a food allergy.⁴ However, most patients have oral tolerance (an active nonresponse to antigens delivered orally) and do not ever develop a reaction. Oral tolerance may occur because

of the way intestinal epithelial cells present the antigen to mucosal lymphatic cells. Low doses of intestinal food antigens preferentially increase regulatory T cell production within the intestinal lymphoid tissue. These regulatory T cells secrete suppressive cytokines that decrease inflammatory reactions. Infants and young children have a more immature mucosal gut barrier and immune response; therefore, a larger percentage of ingested food is absorbed intact. This is believed to account for the increased prevalence of food allergies in this population.⁴

Foods Most Likely to Produce Food Allergies

Although any food is a potential allergen, the foods in *Table 1* account for more than 90 percent of all systemic food allergies.^{2,5} Fruits and vegetables can also produce allergies, but they tend to be milder reactions. Seeds (e.g., sesame, sunflower) have been known to cause severe reactions.^{6,7} Although much less common, allergy to other foods is possible, with manifestations in almost any

SORT: KEY RECOMMENDATIONS FOR PRACTICE

Clinical recommendation	Evidence rating	References	Comments
Immunoglobulin E testing with skin-prick or radioallergosorbent test is appropriate if clinical suspicion for food allergy is high.	C	17	Recommendation from guideline based on nonrandomized studies
Patients (or caregivers of patients) with known or suspected anaphylactic food allergies should carry injectable epinephrine and be instructed on how to use it.	C	17	Recommendation from guideline based on consensus of the Joint Task Force on Practice Parameters
Although there is no evidence to support the use of hydrolyzed formula over breastfeeding, there is some evidence that hydrolyzed formulas reduce infant and childhood allergies compared with cow's milk-based formulas.	B	39, 40	Based on meta-analyses of randomized and nonrandomized studies; however, there was significant inconsistency of results across the trials

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, see <http://www.aafp.org/afpsort.xml>.

organ system. Allergy to food additives is also possible, but rare. Food additive allergy should be suspected when the patient reports allergic symptoms after ingestion of a variety of foods with no shared proteins, and when no reaction occurs with a homemade version of the same foods.⁸ Genetic manipulation of food can also potentially produce proteins that will cross-react with the immunoglobulin E (IgE) of a patient with a food allergy.⁹

Most patients are allergic to between one and three foods. This does not include the cross-reactions to similar proteins that can be common in some food groups. For example, almost all patients who are allergic to cow's milk will also be allergic to sheep's or goat's milk. Most patients who are allergic to shrimp will also react to other crustaceans. Some patients with a latex allergy will react to banana, kiwi, or avocado.

Characteristics of Patients with Food Allergies

Most patients with food allergies have an atopic disorder; however, only 10 percent of patients with atopic disorders have food allergies.¹⁰ A family history of food allergy or other atopic disorders increases the risk of developing a food allergy. Genetic predisposition, including specific haplotypes, has been identified for some common food allergies. The oral allergy syndrome is confined to patients who have allergic rhinitis or asthma. *Table 2* lists historical factors that increase the risk of food allergies.¹¹

Natural History of Patients with Food Allergies

The majority of children will outgrow the most common food allergies; those who do not will have persistent allergies to the same or different foods. Approximately 70 percent of children with egg allergy and 85 percent with milk allergy will outgrow it by five years of age.^{12,13} However, about 40 to 60 percent of these children will develop asthma and 30 to 55 percent will develop allergic rhinitis.^{12,13} Risk of persistent allergy to peanut is much greater, with only 20 percent of children ever developing

Table 1. Most Common Food Allergies in Children and Adults

Children	Adults
Egg	Crustaceans (e.g., shrimp, crab, lobster)
Milk	
Soy	Tree nuts
Wheat	Peanut
Peanut	Fish

NOTE: Food allergies are listed in order of highest to lowest prevalence. Information from references 2 and 5.

Table 2. Historical Factors that Increase the Risk of Food Allergy

History of reaction within minutes to hours of ingestion
Inadvertent ingestions of the same food have produced similar reactions on repeated exposure
Lack of other possible explanations for the reaction besides food allergy
Suspected food is known to be a higher risk for food allergies
Symptom onset in infant or young child
Personal or family history of atopic dermatitis, asthma, allergic rhinitis, or food allergies

NOTE: Although these features would increase the likelihood of a food allergy, the absence of these features does not preclude the possibility of a food allergy.

Information from reference 11.

tolerance.¹⁴ Adolescents with persistent allergies and adults with new onset are particularly prone to fatal food allergies. Increased risk in adolescents may be explained by their tendency to eat foods that could contain allergens and to not carry epinephrine with them (depending on their social situation).¹⁵ Adults with food allergies usually remain allergic.

Differential Diagnosis for Symptoms Suggestive of Food Allergies

Suspicion of food allergy begins with reports of symptoms that appear to be temporally related to food ingestion. Persons with IgE-mediated food allergy develop symptoms within minutes to several hours after exposure; reactions rarely occur later. Even reported reactions within this time are not specific for food allergies. Symptoms from the clinical spectrum reported below, particularly if experienced repeatedly by the patient in response to a food that commonly produces allergies, are more likely to truly represent an allergy. Food-associated symptoms that are not IgE mediated can be further divided into illnesses that are immune mediated, but not completely IgE based (e.g., the principally cell-mediated responses in celiac disease), or the many nonimmune adverse reactions to food.

The nonimmune-mediated reactions include infectious causes, enzymatic food reactions (lactose intolerance), and pharmacologic food reactions (vasoactive amines in scombroid poisoning). Also, symptoms can increase with eating (irrespective of the food ingested) in irritable bowel disease, carcinoid syndrome, and gustatory rhinitis. Distinguishing features of some of these disorders are presented in *Table 3*.^{16,17}

Food diaries can be useful when the patient has symptoms that could potentially be secondary to food allergy, but there is no recognized provoking food. The patient records all foods eaten that day in a diary. The diary is typically continued for weeks.

Family physicians can help determine how likely a patient's symptoms are to be a result of a food allergy and if further testing is indicated. They can redirect the evaluation if symptoms are being falsely attributed to allergies. They can also provide information on food avoidance techniques and can primarily direct the avoidance strategies when the reported reaction is minor (e.g., oral allergy syndrome). Family physicians are often contacted first to assess and treat anaphylactic reactions from food. Allergist referral should be considered when the patient has a history of anaphylactic reactions to food, when there is need for skin-prick or food challenge testing, and when symptoms have not improved with primary care interventions.

Clinical Spectrum of IgE-Mediated Food Allergies ANAPHYLAXIS

Anaphylaxis symptoms occur in multiple organ systems and can include throat swelling, wheezing, rhinorrhea, urticaria, hypotension, and abdominal cramping (*Table 4*).¹⁸ Risk factors for death from anaphylaxis are

adolescent or young adult patient; underlying asthma; allergies to crustaceans, tree nuts, peanuts, or fish; and a delay in or lack of administration of epinephrine.

FOOD-DEPENDENT EXERCISE-INDUCED ANAPHYLAXIS

Food-dependent exercise-induced anaphylaxis is a rare disorder in which patients develop anaphylaxis only if they ingest foods to which they are allergic and then exercise. They are completely asymptomatic if these two elements are not combined. Patients must avoid the provoking foods for as many as six hours before exercise. Wheat is the most common food associated with food-dependent exercise-induced anaphylaxis.^{19,20}

ACUTE URTICARIA

Food allergies account for 30 percent of acute urticaria cases.²¹ Patients become symptomatic minutes to hours after ingestion of the provoking food. Because acute urticaria can be one manifestation of anaphylaxis, care to identify symptoms in other organ systems that would raise the diagnosis to this more urgent level is warranted. Chronic urticaria is much less commonly caused by food allergies (3 to 4 percent of cases).²²

ATOPIC DERMATITIS

About 35 percent of children with atopic dermatitis have a food allergy, based on double-blind, placebo-controlled food challenges.²³ Skin manifestations improve when the suspected foods are removed from the diet; eggs, milk, and peanuts are most commonly implicated. In breast-fed infants, elimination of suspected foods in the mother's diet has produced clinical improvement.

ORAL ALLERGY SYNDROME

The oral allergy syndrome is the most common food allergy; it is clinically recognized in up to 10 percent of patients who have allergic rhinitis or asthma from grass, weed, or tree pollen.²⁴ However, it is believed to have a significantly higher prevalence in patients with birch pollen allergy.²⁵

The manifestations of the oral allergy syndrome are brief in duration, are limited to the mouth and throat, and are sometimes so mild that the patient may not seek evaluation. Proteins similar to the aeroantigens to which the patient is sensitive are present in apples, carrots, and cherries (birch pollen); kiwi and tomato (grass pollen); and melons (ragweed pollen). When these foods come into contact with the oropharynx, a local reaction occurs. *Table 5* lists common food and aeroantigen cross-reactions.¹⁸ Patients may notice lip and tongue swelling and pruritus that can also involve the throat and palate. Progression to

Table 3. Food and Eating-Related Disorders that May Mimic Food Allergies

<i>Disorder</i>	<i>Populations affected/presumed etiology/food sources</i>	<i>Symptoms</i>	<i>Diagnosis/treatment</i>
Carcinoid syndrome	Carcinoid tumors occur throughout adulthood and can develop in late childhood	Watery diarrhea with upper body flushing; symptoms may be provoked by eating (especially cheese) or alcohol intake	Measurement of 5-hydroxyindoleacetic acid from a 24-hour urine sample
Celiac disease	More common in white persons Symptoms can start at any age Sometimes associated with dermatitis herpetiformis Symptoms develop after gluten ingestion (wheat, barley, rye, and, more rarely, oats)	Varied symptoms including diarrhea, malabsorption, weight loss, specific nutrient deficiencies	Immunoglobulin A antigliadin, antiendomysial, and antitissue transglutaminase antibodies are usually present Flattened duodenal villae on biopsy if patient has recently eaten gluten
Giardiasis	Persons who have ingested water or food contaminated with Giardia cysts Fecal-oral spread also occurs in child daycare settings	Chronic symptoms of increased flatus, bloating, and diarrhea are often intermittent and recurring	Detection of Giardia antigen in stool Stool usually negative for occult blood or white blood cells
Gustatory rhinitis	Believed to be nonallergic and mediated through vagus nerve	Nasal congestion and rhinorrhea after eating hot or spicy foods	No specific tests Diagnosed by characteristic history
Irritable bowel disease	Chronic symptoms usually start in young adulthood (before 40 years of age)	No weight loss or fevers Cramping abdominal pain, often with increased flatus Symptoms often increase with eating Diarrhea can alternate with constipation, or one may be predominant	Stool will be negative for occult blood or white blood cells Complete blood count will be normal
Lactase deficiency*	Primary deficiency much more likely to develop in adulthood in nonwhite persons, but lesser degrees of lactase deficiency can be found in 25 percent of white persons	Diarrhea, abdominal pain, and increased flatus after ingestion of dairy products	pH in stool will be decreased Trial elimination of dairy products Breath test for hydrogen
Scombroid poisoning	Bacterial production of excess amines, particularly histamine on food Most cases from tuna, mahi-mahi, and swiss cheese	Patients quickly develop paresthesias, burning sensations, headaches, and pruritus after food ingestion	Portion of the suspected food is tested for histamines Patients improve with antihistamines
Sulfite ingestion†	Sulfites have been banned by the U.S. Food and Drug Administration for preserving raw fruits and vegetables, but they are still found in a variety of cooked and processed foods	Allergic reactions Inhalation produces bronchospasm in about 5 percent of patients with asthma	Treat with beta-agonist inhalers and future avoidance in affected persons with asthma Patients who have sensitivity secondary to sulfite oxydase deficiency can be treated with vitamin B ₁₂

*—Secondary lactase deficiency can occur with small intestinal mucosa brush border abnormalities, such as gastroenteritis and celiac sprue.

†—Can be ingested or inhaled.

Information from references 16 and 17.

Table 4. Symptoms of Anaphylaxis

Abdominal pain, cramping, diarrhea, vomiting	Hypotension, shock
Angioedema, flushing, generalized urticaria, pruritus	Metallic taste in mouth
Chest tightness	Rhinorrhea
Cough, dyspnea, wheezing	Throat swelling
Feeling of impending doom	Uterine contractions

Information from reference 18.

Table 5. Potential Cross-Reactions Between Airborne Allergens and Foods

Airborne allergen	Food
Birch pollen	Carrots
	Celery
	Fresh fruit (e.g., apples, cherries, nectarines, peaches, pears)
	Hazelnuts
	Parsnips
Grass pollen	Potatoes
	Kiwi
	Tomatoes
Ragweed pollen	Bananas
	Melons (e.g., cantaloupe, honeydew, watermelon)

Information from reference 18.

systemic manifestations is rare. Denaturing the proteins by cooking, or removing the food from the oropharynx by swallowing or spitting out stops the reaction.

ALLERGIC EOSINOPHILIC GASTROINTESTINAL DISORDERS

Allergic eosinophilic gastrointestinal disorders are particularly prevalent in children and are thought to be

caused by an IgE- and cell-mediated response to specific foods. Patients with these disorders have excess eosinophils in the mucosal and serosal layers of the portion of the gastrointestinal tract that produces their symptoms.²⁶ Only about 50 percent of children with eosinophilic gastrointestinal disorders are positive for specific food allergies on IgE testing,¹⁸ but almost all children improve when switched from milk or soy to an extensively hydrolyzed formula (processed so that peptides are less than 3,000 Da) or to an elemental diet (no proteins; only amino acids) (Table 6^{26,27}).

Diagnostic Testing

All IgE testing for food allergies must be interpreted in the context of the patient's clinical reactions. Many patients will have positive IgE tests to foods despite never having a clinical reaction. Also, IgE will remain positive if they once had food allergies, but have since developed tolerance. The most commonly used method to assess for food-specific IgE is skin-prick testing. In skin-prick testing, a portion of a commercial extract of the food in question is pushed into the epidermis with a needle or probe, and the area is observed for a wheal and flare reaction after 15 to 20 minutes. Some allergists believe that fresh extracts of fruits and vegetables have superior sensitivity and specificity and use them in skin-prick tests. Although generalized reactions rarely occur (about 0.05 percent overall rate), there have been no reported deaths after skin-prick testing.²⁸

Recent reports have suggested similar sensitivity and specificity for the radioallergosorbent test (RAST) compared with skin-prick testing; however, many allergists believe that RAST sensitivity is lower, particularly in older children and adults. RAST involves the detection of preformed antibodies in the patient's serum and thus carries no potential for allergic reactions. In this article,

Table 6. Subclassification of Eosinophilic Gastrointestinal Disorders

Disorder	Population	Signs and symptoms
Allergic eosinophilic esophagitis	Most commonly diagnosed in neonates and infants, but can affect older children and adults	Emesis, dysphagia, or epigastric pain that continues despite antireflux therapy Normal esophageal pH
Allergic eosinophilic gastritis	Children and adolescents	Failure to thrive, diarrhea, emesis, epigastric pain, occult blood in stool, gastric outlet obstruction
Allergic proctocolitis	Usually in young infants; more than 50 percent are exclusively breastfed Sometimes occurs in older children	Can occasionally produce blood-streaked stools

Information from references 26 and 27.

Evaluation of Suspected Food Allergy

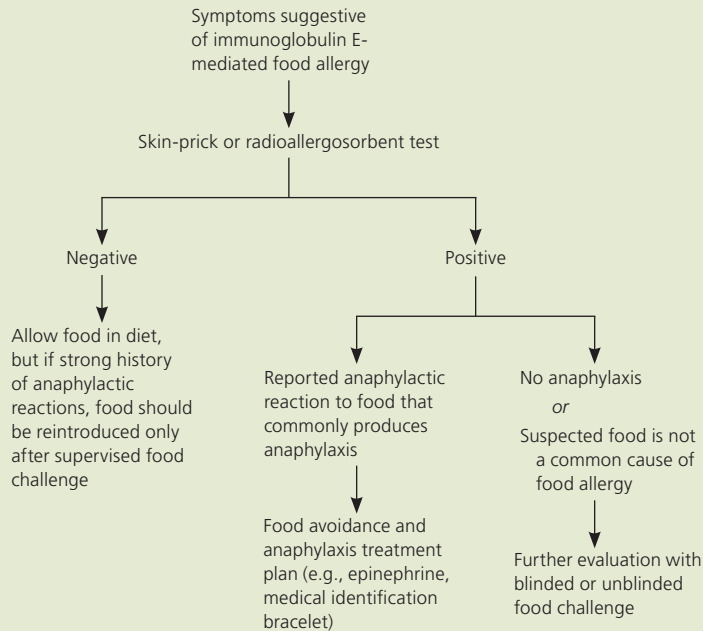


Figure 1. Algorithm for the evaluation of suspected food allergy.

Information from reference 18.

RAST refers to any in vitro, food-specific IgE antibody test. IgE testing with skin-prick test or RAST is appropriate if clinical suspicion of food allergy is high.¹⁷ *Figure 1* outlines the evaluation for suspected food allergy.¹⁸

Intradermal testing has poorer specificity for food allergy and greater risk of adverse reaction than a skin-prick test or RAST and therefore, is not appropriate for initial evaluations.¹⁷ Some allergists still argue for its use in subsequent evaluations when clinical suspicion is high and skin-prick test or RAST are negative. Patch testing has shown promise, particularly in children with atopic dermatitis and in the evaluation of delayed reactions, but it requires highly experienced evaluators to properly assess the reactions. *Table 7*²⁹⁻³⁴ presents methods for IgE testing and *Table 8*^{17,30} lists types of food challenges and their uses. Patients describing anaphylactic reactions to a food that is commonly associated with anaphylaxis do not require food challenge if their

Table 7. Immunoglobulin E Determinations for Suspected Food Allergy

Procedures	Observed response	Comments
Skin-prick test		
Portion of commercial extract is pushed into area of epidermis with needle or probe; adjacent area has normal saline as control	Observe for wheal and flare reaction developing after 15 to 20 minutes	85 percent sensitivity and 30 to 60 percent specificity for food allergies based on double-blind, placebo-controlled food challenges ²⁹ Patients must avoid antihistamines for 48 hours before testing because they can blunt reaction
RAST		
Although a serum sample can be assessed for a predetermined food allergy panel, individual food testing based on history is preferred	Levels of immunoglobulin E against predetermined food antigens are measured	Like skin-prick testing, RAST has high sensitivity, but only about 50 percent specificity ^{30,31} ; however, it has a 95 percent specificity in children with atopic dermatitis who are allergic to eggs, milk, peanuts, or fish ³² Preferentially done initially in young children and infants, in adults with significant comorbid conditions, and in patients with such extensive skin involvement that skin-prick testing is prohibited or who cannot discontinue antihistamines for 48 hours before skin-prick testing
Patch testing		
Commercially prepared food extracts applied to skin and occluded with patch	Remove patch and observe for erythema and induration at site at 48 hours	Studied the most in children with atopic dermatitis where positive patch tests have shown to correlate with food challenge-confirmed milk allergies better than skin-prick testing ³³
Overall clinical usefulness unclear	Symptomatic reactions can occur earlier, warranting patch removal after physician notification	Positive predictive value of RAST or skin-prick test combined with patch test is so high that food challenges are often unnecessary ³⁴

RAST = radioallergosorbent test.

Information from references 29 through 34.

Table 8. Food Challenge Testing and Elimination Diet for Suspected Allergies

<i>Procedures</i>	<i>Uses</i>	<i>Comments</i>
Double-blind, placebo-controlled food challenge	Older children and adults with atypical reactions or reported reactions to uncommonly involved foods	Despite being the most specific test for confirming diagnosis, false-positive and false-negative rates are still at least 5 percent; interpretation is difficult because reactions can occur days later and erroneous results can occur if challenge is not designed correctly Time consuming, poorly tolerated by patients, and usually not necessary for diagnosis
Single-blind food challenge	Older children and adults with atypical reactions or reported reactions to uncommonly involved foods, but where there is higher pretest suspicion of true food allergy	Patient bias is reduced because they are blinded Technically easier to perform than double-blind food challenges
Open food challenge	Can be used to test multiple foods with follow-up blinded food challenges for positive reactions	More prone to patient bias; suspected foods are given with masking foods Technically the easiest to perform
Elimination diet	Can be used at any age Multiple foods can be eliminated if there is clinical suspicion for more than one food Usually followed by food challenge if patients improve on elimination	Well tolerated by patients Dietitian consultation usually needed to be certain diet is nutritionally adequate Duration is until symptoms markedly improve without significant medications

NOTE: Procedures are in order of most to least specific.

Information from references 17 and 30.

IgE testing is confirmatory. Food challenges are appropriate when a food is clinically suspected of inducing a food allergy, but IgE testing is negative.

Management

AVOIDING OFFENDING FOODS

Patients with food allergies, and parents of children with food allergies must habitually read labels of any new food to verify the absence of known allergens. When others are cooking, such as at a restaurant or another person's home, ingredients and cooking methods must be known. Desserts, sauces, and fried foods tend to be higher risk. Cooking with butter or a milk-containing margarine could trigger a reaction in persons with a milk allergy.

ANAPHYLACTIC FOOD ALLERGY

Epinephrine should be urgently administered if anaphylaxis is suspected. Intramuscular diphenhydramine (Benadryl), systemic corticosteroids, and histamine H₂ blockers can be added if the patient's symptoms have not completely resolved with epinephrine alone. See Table 9^{18,35} for doses and follow-up. Supplemental oxygen should be administered if the patient has bronchospasm or laryngeal edema.³⁵

Patients who have had even a single anaphylactic reaction to food should have two age-appropriate epinephrine pens with initial instruction in technique and follow-up visits for technique assessment.¹⁷ The second pen is recommended because the first dose can wear off after 20 minutes, possibly before the patient has reached a

medical facility. These patients should also wear a medical identification bracelet that provides information about their allergy. Informing other caretakers and companions of young adults and children about the condition and appropriate use of epinephrine is recommended. Most patients who develop a second phase of anaphylaxis should be admitted to the hospital for observation.³⁵

Oral or parenteral antihistamines can be administered for more minor reactions, such as isolated pruritus or urticaria. Children can take liquid diphenhydramine.³⁵

PREVENTION OF FOOD ALLERGIES

The American College of Allergy, Asthma and Immunology recommends exclusive breastfeeding for the first six months in infants with a family history of two primary relatives with an atopic disease, and continued breastfeeding through at least the first year, with solid food not being introduced until after six months of age.³⁶ Because approximately one half of all women are secretors (what they ingest will appear in their breast milk), the breastfeeding mother should avoid eggs, milk, tree nuts, peanuts, and seafood. In the child's diet, nuts, shellfish, and fish are delayed until three to four years of age. Although there is some evidence of a decrease in food allergies and atopic dermatitis for the first two years of life with this approach, some recent studies have shown no persistent decrease in atopic parameters past the first few years.^{37,38} Using a soy formula instead of a cow's milk-based formula does not appear to reduce allergies.³⁹ However, there is some evidence that infants on hydrolyzed formulas

Table 9. Treatment and Follow-Up for Food Allergy Anaphylaxis

Medication	Adult dose	Children's dose	Follow-up/comments
Epinephrine*	0.3 to 0.5 mg IM of a 1:1,000 solution of aqueous epinephrine	0.01 mg per kg IM up to a maximal dose of 0.3 to 0.5 mg	About 20 percent of patients will have recurrence within several hours; therefore, a four-hour monitoring period is recommended
Diphenhydramine (Benadryl)	50 mg IM or orally	1 mg per kg IM or orally up to a maximal dose of 50 mg	Dose can be repeated every four to six hours 12.5 mg per 5 mL liquid preparation available for children
Ranitidine (Zantac)	50 mg IM or IV or 150 mg orally twice daily	2 to 4 mg per kg IV every eight hours up to maximal dose of 50 mg or 2 to 4 mg per kg orally daily (divided into two doses) up to maximal dose of 300 mg per day	Dose can be repeated every 12 hours
Systemic corticosteroids†	Dexamethasone 6 to 10 mg IV, IM, or orally	Methylprednisolone (Depo-Medrol) 1 to 2 mg per kg IV	Usually not repeated

IM = intramuscularly; IV = intravenously.

*—There is increased risk of myocardial infarction and stroke in patients with risk factors. Effectiveness is diminished in patients on beta blockers.

†—Although not effective for acute treatment, they can reduce recurrence of some symptoms.

Information from references 18 and 35.

have fewer allergies, including food allergies, compared with regular cow's milk formulas.⁴⁰ There is no evidence to support that extensively hydrolyzed formulas reduce allergy development in infants relative to breastfeeding,⁴⁰ but hydrolyzed formula feeding is an option for high-risk infants whose mothers cannot comply with avoiding likely allergen-containing foods during breastfeeding.

Immunotherapy has not been proven to be effective in the prevention of food allergies.⁴¹

PREVENTION OF INADVERTENT EXPOSURES FOR SENSITIZED PATIENTS

Food labels must state if the food contains any of the most common ingredients known to produce systemic reactions. The Food Allergy and Anaphylaxis network (<http://www.foodallergy.org>) has information for patients and families on home food preparation, restaurant dining, responses to allergic reactions, and adjustments for specific social situations.⁴²

NOVEL TREATMENTS AND POTENTIAL NEW DIRECTIONS

The commercial food extracts used in IgE testing contain other nonallergic components, resulting in a test that is difficult to standardize. Use of recombinant antigens that are selected based on their association with food allergy instead of commercial food extracts may allow for improved specificity.^{43,44}

Specific oral tolerance induction, which is when patients ingest daily small quantities of the offending food and then increase the amount until reaching what would be in the diet, has shown some promise, but only in small nonplacebo-controlled trials.⁴⁵

Injection of monoclonal IgG that binds to IgE and masks regions responsible for receptor binding to mast cells and basophiles partially protects patients with peanut allergies and shows promise for use in other food allergies.⁴⁶

A specific Chinese herbal tea formula has been shown to be highly effective in preventing peanut allergies in animals. Trials in humans will be conducted soon.⁴⁷

The Authors

KURT KUROWSKI, MD, is an associate professor of family and preventive medicine at The Chicago Medical School at Rosalind Franklin University of Medicine and Science in North Chicago, Ill., and associate director of the Swedish Covenant Family Practice Residency in Chicago. He received his medical degree from the University of Wisconsin Medical School (now called the University of Wisconsin School of Medicine and Public Health) in Madison, and completed a family practice residency at Resurrection Hospital in Chicago.

ROBERT W. BOXER, MD, is a practicing allergist at Rush North Shore Medical Center in Skokie, Ill. He received his medical degree from Northwestern University in Chicago, and completed a rotating internship and internal medicine residency at Cook County Hospital and an allergy and immunology fellowship at the University of Illinois College of Medicine, both in Chicago.

Address correspondence to Kurt Kurowski, MD, 3333 Green Bay Rd., North Chicago, IL 60064 (e-mail: Kurt.Kurowski@rosalindfranklin.edu). Reprints are not available from the authors.

Author disclosure: Nothing to disclose.

REFERENCES

- Roehr CC, Edenharter G, Reimann S, et al. Food allergy and non-allergic food hypersensitivity in children and adolescents. *Clin Exp Allergy*. 2004;34(10):1534-1541.
- Sicherer SH, Muñoz-Furlong A, Sampson HA. Prevalence of seafood allergy in the United States determined by random telephone survey. *J Allergy Clin Immunol*. 2004;114(1):159-165.

3. Nielsen RG, Bindslev-Jensen C, Kruse-Andersen S, Husby S. Severe gastroesophageal reflux disease and cow milk hypersensitivity in infants and children: disease association and evaluation of a new challenge procedure. *J Pediatr Gastroenterol Nutr.* 2004;39(4):383-391.
4. Sampson HA. Food allergy. Part 1: immunopathogenesis and clinical disorders. *J Allergy Clin Immunol.* 1999;103(5 part 1):717-728.
5. Osterballe M, Hansen TK, Mortz CG, Host A, Bindslev-Jensen C. The prevalence of food hypersensitivity in an unselected population of children and adults. *Pediatr Allergy Immunol.* 2005;16(7):567-573.
6. Gangur V, Kelly C, Navuluri L. Sesame allergy: a growing food allergy of global proportions? *Ann Allergy Asthma Immunol.* 2005;95(1):4-11.
7. Axelsson IG, Ihre E, Zetterström O. Anaphylactic reactions to sunflower seed. *Allergy.* 1994;49(7):517-520.
8. Wilson BG, Bahna SL. Adverse reactions to food additives. *Ann Allergy Asthma Immunol.* 2005;95(6):499-507.
9. Cantani A. Benefits and concerns associated with biotechnology-derived foods: can additional research reduce children health risks? *Eur Rev Med Pharmacol Sci.* 2006;10(4):197-206.
10. Dreskin SC. Genetics of food allergy. *Curr Allergy Asthma Rep.* 2006;6(1):58-64.
11. American Gastroenterological Association medical position statement: guidelines for the evaluation of food allergies. *Gastroenterology.* 2001;120(4):1023-1025.
12. Host A, Halken S, Jacobson HP, Christensen AE, Herskind AM, Plesner K. Clinical course of cow's milk protein allergy/intolerance and atopic diseases in childhood. *Pediatr Allergy Immunol.* 2002;13 suppl 15):23-28.
13. Ricci G, Patrizi A, Baldi E, Menna G, Tabanelli M, Masi M. Long-term follow-up of atopic dermatitis: retrospective analysis of related risk factors and association with concomitant allergic diseases. *J Am Acad Dermatol.* 2006;55(5):765-771.
14. Moneret-Bautrin DA, Morisset M. Adult food allergy. *Curr Allergy Asthma Rep.* 2005;5(1):80-85.
15. Sampson MA, Muñoz-Furlong A, Sicherer SH. Risk-taking and coping strategies of adolescents and young adults with food allergy. *J Allergy Clin Immunol.* 2006;117(6):1440-1445.
16. U.S. Food and Drug Administration. Center for Food Safety and Applied Nutrition. Foodborne Pathogenic Microorganisms and Natural Toxins Handbook. The "Bad Bug Book." <http://www.cfsan.fda.gov/~mow/intro.html>. Accessed February 7, 2008.
17. American College of Allergy, Asthma, & Immunology. Food allergy: a practice parameter. *Ann Allergy Asthma Immunol.* 2006;96(3 suppl 2):S1-S68.
18. Nowak-Węgrzyn A, Sampson HA. Adverse reactions to foods. *Med Clin North Am.* 2006;90(1):97-127.
19. Beaudouin E, Renaudin JM, Morisset M, Codreanu F, Kanny G, Moneret-Bautrin DA. Food-dependent exercise-induced anaphylaxis—update and current data. *Allergy Immunol (Paris).* 2006;38(2):45-51.
20. Oyefara BI, Bahna SL. Delayed food-dependent, exercise-induced anaphylaxis. *Allergy Asthma Proc.* 2007;28(1):64-66.
21. Legrain V, Taieb A, Sage T, Maleville J. Urticaria in infants: a study of forty patients. *Pediatr Dermatol.* 1990;7(2):101-107.
22. Kulthanan K, Jiamton S, Thumpimukvatana N, Pinkaew S. Chronic idiopathic urticaria: prevalence and clinical course. *J Dermatol.* 2007;34(5):294-301.
23. Eigenmann PA, Sicherer SH, Borkowski TA, Cohen BA, Sampson HA. Prevalence of IgE-mediated food allergy among children with atopic dermatitis. *Pediatrics.* 1998;101(3):E8.
24. Ma S, Sicherer SH, Nowak-Węgrzyn A. A survey on the management of pollen-food allergy syndrome in allergy practice. *J Allergy Clin Immunol.* 2003;112(4):781-788.
25. Ghunaim N, Grönlund H, Kronqvist M, et al. Antibody profiles and self-reported symptoms to pollen-related food allergens in grass pollen-allergic patients from northern Europe. *Allergy.* 2005;60(2):185-191.
26. Rothenberg ME. Eosinophilic gastrointestinal disorders (EGID). *J Allergy Clin Immunol.* 2004;113(1):11-28.
27. Burks AW. Food Allergies: diagnosis. ACP Medicine Online [login required]. <http://www.medscape.com/viewarticle/535012>. Accessed February 7, 2008
28. Devenney I, Fäth-Magnusson K. Skin prick tests may give generalized allergic reactions in infants. *Ann Allergy Asthma Immunol.* 2000;85(6 pt 1):457-460.
29. Eigenmann PA, Sampson HA. Interpreting skin prick tests in the evaluation of food allergy in children. *Pediatr Allergy Immunol.* 1998;9(4):186-191.
30. Sicherer SH. Food allergy: when and how to perform oral food challenges. *Pediatr Allergy Immunol.* 1999;10(4):226-234.
31. Sampson HA, Albergo R. Comparison of results of skin tests, RAST, and double-blind, placebo-controlled food challenges in children with atopic dermatitis. *J Allergy Clin Immunol.* 1984;74(1):26-33.
32. Sampson HA, Ho DG. Relationship between food-specific IgE concentrations and the risk of positive food challenges in children and adolescents. *J Allergy Clin Immunol.* 1997;100(4):444-451.
33. Isolauri E, Turnjanmaa K. Combined skin prick and patch testing enhances identification of food allergy in infants with atopic dermatitis. *J Allergy Clin Immunol.* 1996;97(1 pt 1):9-15.
34. Roehr CC, Reibel S, Ziegert M, Sommerfeld C, Wahn U, Niggemann B. Atopy path tests, together with determination of specific IgE levels, reduce the need for oral food challenges in children with atopic dermatitis. *J Allergy Clin Immunol.* 2001;107(3):548-553.
35. Sampson HA. Anaphylaxis and emergency treatment. *Pediatrics.* 2003;111(6 pt 3):1601-1608.
36. Fiocchi A, Assa'ad A, Bahna S. Food allergy and the introduction of solid foods to infants: a consensus document. Adverse Reactions to Foods Committee, American College of Allergy, Asthma and Immunology. *Ann Allergy Asthma Immunol.* 2006;97(1):10-20.
37. Zutavern A, Brockow I, Schaaf B, et al., for the LISA Study Group. Timing of solid food introduction in relation to atopic dermatitis and atopic sensitization: results from a perspective birth cohort study. *Pediatrics.* 2006;117(2):401-411.
38. Poole JA, Barriga K, Leung DY, et al. Timing of initial exposure to cereal grains and the risk of wheat allergy. *Pediatrics.* 2006;117(6):2175-2182.
39. Osborn DA, Sinn J. Soy formula for prevention of allergy and food intolerance in infants. *Cochrane Database Syst Rev.* 2006;(4):CD003741.
40. Osborn DA, Sinn J. Formulas containing hydrolysed protein for prevention of allergy and food intolerance in infants. *Cochrane Database Syst Rev.* 2006;(4):CD003664.
41. United States National Institute of Allergy and Infectious Diseases Division. Food allergy: an overview. NIH publication no. 04-5518. Bethesda, Md.: United States Department of Health and Human Services, National Institutes of Health, National Institute of Allergy and Infectious Diseases; 2004.
42. The Food Allergy & Anaphylaxis Network. <http://www.foodallergy.org>. Accessed January 28, 2008.
43. Reuter A, Lidholm J, Andersson K, et al. A critical assessment of allergen component-based in vitro diagnosis in cherry allergy across Europe. *Clin Exp Allergy.* 2006;36(6):815-823.
44. Nowak-Węgrzyn A. Future approaches to food allergy. *Pediatrics.* 2003;111(6 pt 3):1672-1680.
45. Rolinck-Werninghaus C, Staden U, Mehl A, Hamelmann E, Beyer K, Niggemann B. Specific oral tolerance induction with food in children: transient or persistent effect on food allergy? *Allergy.* 2005;60(10):1320-1322.
46. Leung DY, Sampson HA, Yunginger JW, et al., for the Avon Longitudinal Study of Parents and Children Study Team. Effect of anti-IgE therapy in patients with peanut allergy. *N Engl J Med.* 2003;348(11):986-993.
47. Srivastava KD, Kattan JD, Zou ZM, et al. The Chinese herbal medicine formula FAHF-2 completely blocks anaphylactic reactions in a murine model of peanut allergy. *J Allergy Clin Immunol.* 2005;115(1):171-178.