
CHILDHOOD FOOD ALLERGY

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We now know that food allergy occurs in 6% to 8% of children and 1% to 2% of adults.⁴ Significant scientific studies in the past several years have increased the understanding and treatment of these allergic reactions. As much as 15% of the general population believe that they may be allergic to some food. Several well-controlled studies have revealed that the vast majority of food allergic reactions present in the first year of life.^{4,11} These studies also demonstrate that the occurrence of food allergic disease is more prevalent than the medical community previously thought.

DEFINITIONS

A variety of terms have been used to characterize adverse reactions to foods. The American Academy of Allergy and Immunology and the National Institute of Allergy and Infectious Disease (National Institutes of Health [NIH]) have defined several terms in an attempt to standardize the literature.² An *adverse food reaction* is a generic term referring to any untoward reaction after the ingestion of a food. Adverse food reactions may be secondary to *food allergy (hypersensitivity)* or *food intolerance*. A *food allergy* reaction is presumed to be the result of an abnormal immunologic response after the ingestion of a food, whereas a *food intolerance* is the result of nonimmunologic mechanisms.

IgE-mediated hypersensitivity reactions account for the majority of well-documented food allergy reactions, but non-IgE-mediated immune mechanisms do cause a variety of hypersensitivity disorders. Food intolerances probably make up the majority of adverse food reactions. Examples of food intolerance include reactions from toxic contaminants (e.g., histamine in scombroid fish poisoning, toxins secreted by *Salmonella* or *Shigella*), pharmacologic reactions (e.g., to caffeine in coffee, tyramine in aged cheeses), metabolic reactions (e.g., lactase deficiency), and idiosyncratic and psychological reactions.

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FOOD ALLERGENS

Foods are composed of proteins, carbohydrates, and lipids. Primarily, the major food allergens are water-soluble glycoproteins that have molecular weights ranging between 10,000 and 60,000 d.¹⁶ Generally, the allergens are stable to treatment with heat, acid, and proteases. Other properties that may account for their allergenicity are not well understood.

A minority of foods are responsible for the majority of documented food allergic reactions. In children, these foods are milk, eggs, peanuts, fish, soybeans, wheat, and tree nuts (Table 1). In adults, the primary foods causing reactions are peanuts, tree nuts, fish, and shellfish.

PATHOPHYSIOLOGY

IgE

A variety of hypersensitivity responses to an ingested food antigen may result from the genetically predisposed patient's lack of development of oral tolerance or a breakdown of oral tolerance in their gastrointestinal tract. Although IgE-mediated reactions have been studied in great detail, some of these disorders may involve more than one immunologic mechanism. Either a failure to develop or a breakdown in oral tolerance results in excessive production of food-specific IgE antibodies. These food-specific antibodies bind high-affinity FcεI receptors on mast cells and basophils and low-affinity FcεII receptors on macrophages, monocytes, lymphocytes, eosinophils, and platelets. After the food allergen reaches the food-specific antibodies on mast cells or basophils, mediators such as histamine, prostaglandins, and leukotrienes are released. These mediators then promote vasodilation, smooth muscle contraction, and mucus secretion, resulting in the symptoms of immediate hypersensitivity.¹⁵ The activated mast cells also may release various cytokines that play a part in the IgE-mediated late-phase response. With repeated ingestion of a specific food allergen, mononuclear cells are stimulated to secrete histamine-releasing factors (HRF).²³ The spontaneous generation of HRF by the activated mononuclear cells in vitro has been associated with increased cutaneous irritability in children who have atopic dermatitis. A rise in plasma histamine has been associated with IgE-mediated allergic symptoms after blinded food challenges.²⁵ In IgE-mediated gastrointestinal reactions, endoscopic observation has revealed local vasodilation, edema, mucus secretion, and petechial hemorrhaging. Increased stool and serum prostaglandin (PGE2) and PGF2 have been seen after food challenges that cause diarrhea.⁷

Table 1. MAJOR FOOD ALLERGENS IN CHILDREN AND ADULTS

Children		Adults
milk	wheat	peanuts
egg	fish	tree nuts
peanuts	tree nuts	fish
soybeans		shellfish

Non-IgE

Although a variety of reports have discussed other immune mechanisms that cause food allergic reactions, the scientific evidence supporting these mechanisms is limited. Type III (antigen-antibody complex-mediated) hypersensitivity reactions have been examined in several studies. Although IgE-food antigen complexes are seen more commonly in patients who have food hypersensitivity, there is little support for food antigen-immune complex-mediated disease. Type IV (cell-mediated) hypersensitivity has been discussed in several disorders for which the clinical symptoms do not appear until several hours after the ingestion of the suspected food. This type of immune response may contribute to some adverse food reactions (e.g., enterocolitis) but significant, supporting evidence of a specific cell-mediated hypersensitivity disorder is lacking.¹⁷

CLINICAL MANIFESTATIONS

IgE

A variety of gastrointestinal, cutaneous, respiratory, and generalized symptoms and syndromes have been associated with IgE-mediated food allergy.

The oral allergy syndrome is considered a form of contact urticaria confined almost exclusively to the oropharynx.¹ Typical symptoms include pruritus and angioedema of the lips, tongue, palate, and throat. The symptoms generally resolve rather rapidly. The symptoms are often associated with the ingestion of various fruits and vegetables. Patients who have allergic rhinitis secondary to ragweed may develop symptoms after ingestion of melons (e.g., watermelon, cantaloupe, honeydew) and bananas. Similarly, patients sensitive to birch pollen may develop oral symptoms after the ingestion of raw potatoes, carrots, celery, apples, and hazelnuts.

Generalized anaphylactic reactions secondary to IgE-mediated sensitivity to foods are not rare. Patients may develop cutaneous, respiratory, gastrointestinal, and cardiovascular symptoms, including hypotension, vascular collapse, and cardiac dysrhythmias. Patients who have fatal or near-fatal food-induced anaphylactic reactions have several clinical features in common, including the following: (1) asthma, (2) accidental ingestion of the food allergen, (3) previous allergic reactions to the incriminated food, and (4) immediate symptoms with approximately one half experiencing a quiescent period before a major respiratory collapse.^{26, 30}

Gastrointestinal anaphylaxis refers to IgE-mediated gastrointestinal symptoms (nausea, abdominal pain, abdominal cramping, vomiting, or diarrhea) that often accompany symptoms in other target organs. Symptoms generally develop within minutes to 2 hours of ingesting the offending food. The frequent ingestion of a food allergen in patients who have atopic dermatitis and food allergy appears to induce a state of partial desensitization of the gastrointestinal mast cells, which will manifest itself in less pronounced clinical symptoms. After the food is removed from the patient's diet, the symptoms may become significantly more evident after an ensuing food challenge.²⁰

Respiratory and ocular symptoms are common manifestations of IgE-mediated reactions to foods. Symptoms may include periorcular erythema, pruritus, and tearing; nasal congestion, pruritus, sneezing, and rhinorrhea; and coughing, voice changes, and wheezing. Isolated nasoocular symptoms are an uncommon manifestation of food hypersensitivity reactions.⁵ Although the ingestion of food

allergens is rarely the main trigger factor of chronic asthma, preliminary evidence suggests that food antigens can provoke bronchial hyperreactivity. Inhalation of a food allergen from cooking or food processing may cause acute bronchospasm.

The skin is a frequent target organ in IgE-mediated food hypersensitivity reactions. The ingestion of food allergens can either lead to immediate cutaneous symptoms or aggravate more chronic symptoms. Acute urticaria and angioedema are probably the most common cutaneous manifestation of food hypersensitivity reactions, generally appearing within minutes of ingestion of the food allergen.²⁹ The foods commonly causing these reactions in children include eggs, milk, peanuts, and tree nuts. In adults, this list includes fish, shellfish, tree nuts, and peanuts. Food allergy causing chronic urticaria is a rare phenomenon occurring in only approximately 1% of patients who have this disorder.⁸

Atopic dermatitis is a chronic skin disorder that generally begins in early infancy and is characterized by typical distribution, extreme pruritus, chronically relapsing course, and association with asthma and allergic rhinitis. In well-controlled studies, approximately one third of children who have atopic dermatitis have food allergic reactions. At the time of food challenge in these patients, the skin symptoms that are provoked include a significantly pruritic, erythematous, morbiliform rash that develops in sites with a predilection for atopic dermatitis. Urticarial lesions on initial challenge in these patients are seen uncommonly. Many of these patients have gastrointestinal or respiratory symptoms during these challenges. Much of the information learned about the pathophysiology of food hypersensitivity reactions has occurred in this group of patients who have atopic dermatitis and food hypersensitivity reactions.²⁷

Another gastrointestinal disorder, allergic eosinophilic gastroenteropathy, is characterized by infiltration of the gastric or the intestinal walls with eosinophils, absence of vasculitis, and frequent peripheral eosinophilia. Patients who have this disorder typically present with postprandial nausea and vomiting, abdominal pain, diarrhea, and weight loss in adults or failure to thrive in young infants. IgE-mediated food hypersensitivity reactions have been implicated in a subset of patients who have this disorder. It may take as long as 12 weeks after elimination of the responsible food allergen from the diet for symptoms to resolve.¹³

Infantile colic is a disorder characterized by paroxysmal fussiness, inconsolable crying, flexion of the legs, abdominal distention, and excessive gas that generally occurs in the first 2 to 4 weeks of life. A variety of factors both psychosocial and dietary, have been implicated in the cause of this disorder. Blinded studies suggest that as many as 10% to 15% of patients who have this disorder may have an IgE-mediated food hypersensitivity reaction that causes the symptoms. Periodic rechallenges are necessary after diagnosis because of the short-lived nature of this disorder.¹²

Non-IgE-Mediated Food Hypersensitivity

Food-induced enterocolitis is a disorder generally beginning in young infants between 1 week and 3 months of age.¹⁹ These patients present with vomiting and diarrhea often secondary to the ingestion of cow's milk or soybean protein formulas. At times, the symptoms do not occur until several hours after the ingestion of the protein source. Their stools generally contain occult blood, neutrophils, eosinophils, and are positive for reducing substances. Any examination for specific IgE (prick skin tests, radioallergosorbent test [RAST]) is generally

negative. These patients typically become tolerant of the food by 12 to 24 months of age.

Food-induced colitis, like food-induced enterocolitis, generally presents with similar symptoms in the first several months of life.¹⁰ Appearance of blood in the stool with or without diarrhea is usually observed, but unlike the previous disorder, the patients are not generally as ill. Protein-specific IgE antibodies are typically not found, and these patients usually become tolerant of the food by 12 to 24 months of age.

Malabsorption syndromes (excluding celiac disease) often present in the first several months of life with diarrhea (not infrequently steatorrhea) and poor weight gain. The presenting symptoms include protracted diarrhea, vomiting, failure to thrive, and carbohydrate malabsorption. This syndrome has most commonly been associated with the ingestion of cow's milk proteins but also associated with soybean, eggs, and wheat. Complete resolution of the symptoms may take 6 to 18 months of allergen avoidance.

Celiac disease is a more extensive enteropathy leading to malabsorption.¹⁴ Total villous atrophy and extensive cellular infiltrate are associated with sensitivity to gliadin, the alcohol-soluble portion of gluten found in wheat, oats, rye, and barley. The typical symptoms include diarrhea or frank steatorrhea, abdominal distention and flatulence, weight loss, and occasionally nausea and vomiting. IgA antibodies to gluten are present in more than 80% of adults and children who have untreated celiac disease. The immunopathogenesis of the disorder remains unknown, although studies in animal models suggest that a type IV cell-mediated mechanism is involved. Several antibody determinations have been tried for diagnosis and follow-up, including antigliadin antibodies. Once the diagnosis of celiac disease is established (which requires an intestinal biopsy), life-long elimination of gluten-containing foods is necessary. One strong reason for long-term elimination of gluten in the diet is that this will significantly decrease the possibility of intestinal lymphoid malignancy.

FOOD INTOLERANCE: METABOLIC, IDIOSYNCRATIC, PSYCHOLOGIC

Food intolerance includes toxic, pharmacologic, and psychological reactions, as well as metabolic reactions from enzyme deficiencies.² Toxic reactions may result from naturally occurring toxic substances or from microbial or chemical contamination of foods. Adverse reactions to sulfites, monosodium glutamate, and aspartame have been reported, although they probably occur with very little frequency. Microbial contamination of foods may result in poisoning because of the action of the microbial toxin or the microbial infection. Some foods contain active pharmacologic agents that in susceptible patients may cause clinical symptoms (e.g., caffeine).

The most common metabolic reactions to food are the result of disaccharidase deficiencies. Carbohydrate malabsorption leads to bloating, cramps, flatulence, and diarrhea. For example, lactase deficiency, not uncommonly observed after significant episodes of diarrhea, is secondary to the ingestion of foods containing lactose. The small intestine cannot absorb the lactose, a disaccharide, and bacteria in the large intestine ferment the lactose. This disorder is present in as much as 80% of North American blacks and 5% to 20% of whites. Primary acquired lactase deficiency begins to present in school-aged children.

Psychological aversions to foods are not uncommon and can be associated with various symptoms, including nausea, vomiting, diarrhea, and headaches.

In addition, anxiety and depression can present as food allergy. The physician must remain cognizant of these food-aversion reactions and be aware of specific gastrointestinal illnesses such as hiatal hernia or peptic ulcer disease that may mimic these problems.

DIAGNOSIS

The diagnostic approach to adverse food reactions begins with a careful medical history and physical examination. Laboratory studies may then be utilized appropriately in the evaluation. In specific acute reactions to foods, such as systemic anaphylaxis, the medical history is important in diagnosis but in other less acute reactions, the medical history can be verified in less than 50% of reported food allergic reactions.⁶ In the evaluation of possible food allergy, important elements of the history include (1) the food suspected in provoking the reaction, (2) the quantity of the suspected food ingested, (3) the length of time between ingestion of the food and the development of symptoms, (4) a description of the symptoms provoked, (5) if similar symptoms developed on other occasions when the food was eaten, (6) if other factors (e.g., exercise) are necessary, and (7) the time since the last reaction to the food occurred. In chronic disorders the history may not be a reliable indicator of the offending food allergen.

Several methods have been utilized in the evaluation of food allergic reactions, including diet diaries, elimination diets, skin prick testing, radioallergen sorbent tests, basophil histamine release, intestinal mast-cell histamine release, intragastric provocation under endoscopy, intestinal biopsy after allergen feeding, and double-blind placebo-controlled food challenges. A diet diary is frequently used as an adjunct to the medical history by providing a chronological record of all foods ingested over a specified period. This method rarely detects an unrecognized association between a food and a patient's symptoms. Elimination diets are utilized both in diagnosis and in management of adverse food reactions. The success of these diets depends on the identification of the correct allergen(s), the ability of the patient to maintain a diet completely free of all forms of the food allergen, and the assumption that other factors are not necessary or do not provoke similar symptoms. Elimination diets are rarely diagnostic of food allergy, especially in chronic disorders such as atopic dermatitis or asthma.

Allergy skin prick tests are highly reproducible and often utilized to screen patients who have suspected IgE-mediated food allergies.²² The criteria established by May and Alberto¹⁷ and Bock⁴ have proven useful to many investigators and clinicians. The glycerinated food extracts (1:10 or 1:20 w/v) and appropriate positive (histamine) and negative (saline) controls are applied epicutaneously by either the prick or the puncture technique. A food allergen eliciting a wheal at least 3 mm or greater than the negative control is considered positive; anything else is considered negative. A positive skin test to a food indicates only the possibility that the patient has symptomatic reactivity to that specific food (overall the positive predictive accuracy is less than 50%). A negative skin test confirms the absence of an IgE-mediated reaction (overall negative predictive accuracy is greater than 95%). Both of these statements are justified if appropriate and good quality food extracts are utilized.

The epicutaneous (e.g., puncture or prick) skin test should be considered an excellent means of excluding IgE-mediated food allergies but only suggestive of the actual presence of clinical food allergies. There are some minor exceptions,

however, to this general statement, including (1) IgE-mediated sensitivity to several fruits and vegetables (apples, oranges, bananas, pears, melons, potatoes, carrots, celery, and so forth) is frequently undetected with commercially prepared extracts or reagents (this is thought to be the result of the lability of the responsible allergen in the food); (2) children less than 1 year of age may have IgE-mediated food allergy without a positive skin test; (3) children less than 2 years of age may have smaller wheals, possibly caused by the lack of skin reactivity; and conversely (4) a positive skin test to a food ingested in isolation, which provokes a serious systemic anaphylactic reaction and may be considered diagnostic.

An intradermal skin test is a more sensitive method when compared with the skin prick test, but it is much less specific when compared with a blinded food challenge. In one study, no patient who had a negative skin prick test but a positive intradermal skin test to a specific food had a positive double-blind placebo-controlled food challenge (DBPCFC) to that food. In addition, intradermal skin testing has a greater risk of inducing a systemic reaction than does skin prick testing.

RASTs and similar *in vitro* assays are used to identify food-specific IgE antibodies in the serum. Although frequently utilized to screen for IgE-mediated food reactions, these assays are generally considered slightly less specific than the skin prick tests. Highly positive levels of food-specific IgE antibodies determined by *in vitro* tests from high-quality laboratories can provide information similar to that of skin prick tests.²² Recent studies with the Pharmacia CAP System FEIA²⁴ have demonstrated that for some of the major food allergens a better positive predictive accuracy can be achieved with the utilization of this test. These studies have shown that there may be better positive and negative predictive values for anticipating the outcome of an oral food challenge. For example, a level of greater than 15 kU_A/L of specific IgE to peanut has greater than 95% positive predictive accuracy of predicting a positive food challenge. For this test there are both positive and negative predictive values that aid in the diagnosis of patients who have possible food-allergic reactions. Now the predictive values of CAP-RAST results are limited to the major food allergens (milk, egg, peanuts, wheat, and soybeans).

Basophil histamine release assays specific for food allergens have been developed for diagnosis of food allergy, but in their present state they are not more predictive of clinical sensitivity than the skin prick test. Intestinal mast-cell histamine release is a research procedure in which intestinal mast cells obtained from biopsy specimens are added to specific food antigens and a percentage histamine release is determined. The procedure correlates closely with symptoms of the gastrointestinal tract after challenge but not with other symptoms provoked by challenge.

The DBPCFC has been labeled the gold standard for the diagnosis of food allergies.²¹ This technique has been used by many investigators and clinicians in the evaluation of both adults and children who have suspected food allergic reactions. If the blinded part of the procedure (i.e., administration of the suspect food in liquid or opaque capsules) is negative, the open part of the challenge must be completed to ensure that the challenge is negative and to confirm the safe ingestion of a normal amount of the food in its usually prepared state.

UNPROVEN DIAGNOSTIC AND THERAPEUTIC TECHNIQUES

Because of the lack of complete understanding about the basic immunopathologic mechanisms and the prevalence of individuals who think they have had

an adverse food reaction, numerous medical techniques have been developed for the diagnosis and treatment of these problems. Disproven or unproven techniques include cytotoxicity testing, provocative and neutralization treatment, autogenous urine immunization, IgG subclass diagnosis of food allergy, and food immune complexes.²

Tests for IgG or IgG₄ to a specific food or specific food immune complexes have been proposed for use in the diagnosis of food allergic reactions, but there is little evidence to support their use. Development of food-specific IgG and IgG₄ is a normal immune response to food ingestion. Similarly, immune complex formation is a normal event in the course of an immune response and allows antigen elimination. Masking and addiction, rotary diets, and fasting are all various techniques of dietary manipulation that have shown to have no role in the diagnosis or the treatment of food allergic diseases.

PRACTICAL APPROACH TO DIAGNOSIS

The diagnosis of food allergy continues to be a clinical exercise that includes a careful history, the search for specific IgE (e.g., skin prick tests) if an IgE-mediated disorder is suspected, an appropriate period of elimination of the suspected food, and challenge with the food in question. For patients who have suspected IgE-mediated food allergy, the initial step is a 1- to 2-week elimination diet that consists of all foods suspected by the history or the skin prick testing. If no improvement is noted, it is not likely that food allergy is involved. Because atopic dermatitis and chronic asthma are multifactorial in nature, the elimination of the food allergen may not appear to change the course of the disease significantly. Open or single-blind challenges are useful in a clinical setting to screen suspected food allergens. Only if challenges for more than one of the major food allergens is positive would a DBPCFC be necessary to prove the existence of reactions to multiple foods. Presumptive diagnosis of food allergy based only on skin prick tests is no longer acceptable. Of those children allergic to foods, approximately 80% are allergic to only 1 or 2 foods.

For patients who have suspected food allergic reactions that are not thought to be IgE mediated, a careful medical history and an elimination diet of the suspected food for a period of 1 to 2 weeks (except in some gastrointestinal disorders where a period as long as 12 weeks may be necessary) should be done before a food challenge is conducted.

For patients who have food-induced enterocolitis or colitis, the symptoms provoked by food challenge (often done openly rather than by DBPCFC) include vomiting and diarrhea but dehydration and hypotension may occur. The symptoms may not occur until 1 to 6 hours after the ingestion of the food. In addition to the clinical symptoms, the patient's absolute polymorphonuclear (PMN) count may increase at 3500 cells/mm³ at 4 to 6 hours after the challenge and the patient's stools will have increased PMNs and eosinophils.

THERAPY

Once the diagnosis of food allergy is established, the only proven therapy is the strict elimination of the food from the patient's diet. Elimination diets may lead to malnutrition or eating disorders, especially if they exclude several foods or are utilized for extended periods.⁹ Studies have shown that except for sensitivity to peanuts, tree nuts, and seafood, symptomatic food sensitivity

Table 2. FOOD ALLERGEN CROSS REACTIVITY

	Specific IgE to Multiple Members of the Family	Clinical Reactivity
Milk	common	common
Legume	common	uncommon
Wheat	common	uncommon
Fish	common	uncommon
Crustacea: mollusks	common	??
Tree nuts	common	uncommon
Egg (chicken)	occasional	rare
Milk or beef	occasional	uncommon

generally is lost over time. Symptomatic food sensitivity is usually very specific, so patients rarely react to more than one member of a botanical family or animal species (Table 2).³ Several medications have been used in an attempt to protect patients who have food hypersensitivity; these medications include oral cromolyn, H₁ and H₂ antihistamines, ketotifen, corticosteroids, and prostaglandin synthetase inhibitors.²⁸ Some of these medications may modify food allergy symptoms, but overall they have minimal efficacy or unacceptable side effects. The use of epinephrine is vitally important in acute anaphylaxis. Recent blinded, placebo-controlled studies of rush immunotherapy for the treatment of peanut hypersensitivity demonstrated efficacy in a small number of patients.¹⁸ However, the adverse reaction rates were significant and preclude general clinical application for now. Newer types of immunotherapy vaccines that are being developed include B- and T-cell epitope peptides and cDNA vaccines.

NATURAL HISTORY

Food hypersensitivity is most common in the first several years of life. In one study, 80% of children developed their reactions in the first year of life.⁴ The average length of time until the food could be tolerated was 1 year. Of the studied group of patients who had atopic dermatitis and food hypersensitivity, those placed on an appropriate elimination diet experienced a significantly greater improvement in their rash when compared with a similar group of patients who did not have food allergy or who did not adhere to the strict elimination diet.²⁷ After 1 to 2 years of complete avoidance of the offending food allergen, approximately one third of symptomatic food reactions were lost. The probability of developing tolerance with specific food avoidance varies with different allergens; patients commonly became tolerant of soybeans, whereas development of tolerance to peanuts was rare. Overall, patients who have peanut, tree nut, fish, and shellfish allergies rarely lose their clinical sensitivity. Skin prick tests or RAST results do not predict which individuals will lose their clinical sensitivity. It is generally thought that non-IgE-mediated food hypersensitivities in infants and young children (other than celiac disease) are outgrown more quickly, but there are few studies to document this conclusion.

PROPHYLAXIS

The possible prophylaxis of food allergy has been debated for many years. Recent studies suggest that in a group of infants at high risk for the development

of allergic disease, maternal (peanuts during the last trimester) and infant avoidance of major allergenic foods (cow's milk, eggs, and peanuts in the first year of life) results in at least a decreased prevalence of atopic dermatitis.³¹ Nursing mothers who do not restrict their diet from such foods allow these food proteins to be ingested by the infants. For such diets to be used for both mothers and infants, these families should be in this high risk group and not necessarily used for the general population.

SUMMARY

After the diagnosis of food hypersensitivity is established, the only proven therapy is strict elimination of the offending allergen. Note that prescribing an elimination diet is like prescribing a medication; both can have positive effects and unwarranted side effects. Elimination diets may lead to malnutrition or eating disorders, especially if they exclude several foods or are utilized for extended periods. Patients and parents should be taught and given educational material to help them detect potential sources of hidden food allergens by appropriately reading food labels. Education of the patient and family is vital to the success of the elimination diet. Families should be given instructional material to help them remember what foods contain the allergen they are to avoid. It is often difficult to determine what foods will contain an allergen without careful reading of the label. Studies in children and adults indicate that symptomatic reactivity to food allergens is often lost over time, except possibly for peanuts, nuts, and seafood.

Symptomatic reactivity to food allergens is generally very specific. Patients rarely react to more than one member of a botanical family or animal species. Note that initiation of an elimination diet totally excluding only foods identified to provoke food allergic reactions will result in symptomatic improvement. This treatment generally leads to resolution of the food allergy within a few years and is unlikely to induce malnutrition or other eating disorders.

Clinical investigations in the past have characterized relatively well the food hypersensitivity disorders, but our understanding of the basic immunopathologic mechanism remains incomplete. Current progress in allergen characterization and the rigorous scientific methods now being applied to this field by many investigators provide hope that new information regarding the pathogenesis of these disorders and new forms of therapy will soon become available. For now, practicing physicians must carefully diagnose specific food sensitivities and educate patients and their families in the elimination of the responsible food allergen.

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