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# Food Allergies: Type II, III, and IV Hypersensitivities

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*Editor's Note: This is the second of a two-part series on food allergies and the immune system. The first part, "Food Allergies: The Immune Response," appeared in the July 2007 issue of* ***Today's Dietitian*** *and is available as a free download at www.todaysdietitian.com/tcpeexam.shtml.*

The previous article discussed the difference between a food allergy, which is an immunologic response to ingested food, and a food intolerance, which is the result of nonimmunologic mechanisms. The immune mechanisms that cause the most common food allergy, immunoglobulin E (IgE)-mediated hypersensitivity, and its symptoms were explained. This article will pick up where Part 1 left off and discuss other types of food allergies and intolerances.

## Type II and III Hypersensitivities

Type II and III hypersensitivity reactions resemble the type of immune response triggered by an invading pathogen—a virus or bacterium likely to cause disease if not effectively eradicated from the body. This reaction is initiated by Th1 lymphocytes. The foreign antigen is processed by antigen-presenting cells and recognized by T-helper cells, a reaction described as Th1-mediated. This leads to the production of antigen-specific antibodies of the immunoglobulin M (IgM) isotype, followed by an isotype switch to immunoglobulin G (IgG) antibodies.Unlike IgE, IgG does not directly initiate degranulation of mast cells. Instead, the coupling of IgG and its homologous (specific) antigen leads to a complex series of reactions involving triggering the complement cascade. In this, a specific sequence of reactive proteins is activated, designed to lead to the final destruction of the invader by a process of cell lysis.1

As a consequence of the activities of the complement system proteins, several newly formed proteins emerge, two of which (C3a and C5a) are powerful anaphylatoxins that are able to degranulate basophils and possibly mast cells without the mediation of IgE. In addition, complement protein C5a and at least one other complement system protein complex act as chemotaxins and attract granulocytes to the reaction site to augment the local response with their own inflammatory mediators.

The IgG-mediated "allergy" to drugs is a recognized phenomenon.2 Penicillin-induced, Coombs-positive hemolytic anemia is an example of a type II hypersensitivity reaction. Penicillin causes the body to produce IgG antibodies to the drug. The body attacks its own red blood cells containing the IgG antibody to penicillin, causing red blood cell destruction.

The difference between a type II and III hypersensitivity reaction is that a type III reaction can be caused by not only external sources but also antigens to "self." The damage in a type III hypersensitivity reaction is from immune complexes and complement. Immune complexes are formed when antigens bind to antibodies and form a complex, which can cause an immune reaction, damaging organs or tissues. Type III hypersensitivity reactions to antibiotics such as penicillin have been identified, with symptoms such as serum sickness, rash, painful joints, and hives.

Systemic lupus erythematosus is another example of a type III hypersensitivity reaction in which the body attacks "self" cells and immune complexes are formed. As a result of the formation of the antigen/antibody complexes, the complement cascade is active and anaphylatoxins are released, which mediate the degranulation of mast cells. Consequently, inflammatory mediators are released, which results in inflammation and widespread tissue damage.

IgG-mediated reactions are sometimes called anaphylactoid reactions to distinguish them from IgE-mediated, or anaphylactic, responses.3

**Can IgG Hypersensitivity Cause Food Allergy?**
It is relatively easy to measure the level of antigen-specific IgG in blood, and many laboratories now offer "food-allergy blood tests" that measure the IgE and IgG antibodies against specific foods (antifood IgE and IgG). Some practitioners believe that food allergy symptoms that occur from one to several hours (up to 24) after consumption of the allergenic food (delayed food allergy) represent IgG-mediated food allergy, especially when allergen-specific IgE is low or absent.

Theoretically, the release of inflammatory mediators, either by the action of anaphylatoxins or direct bonding of antifood IgG to IgG receptors on basophils, may occur.4 IgG-mediated allergy to egg whites and fish have been reported.5

The delay in the onset of symptoms is thought to be due to the increased time required for activation and progress of the complement cascade prior to release of the inflammatory mediators responsible for the symptoms. However, the mere presence of antifood IgG, even at high levels, does not necessarily indicate that the food is the cause of the reaction.

It is common to find antifood IgG antibodies circulating in blood, even in people who have no signs or history of adverse reactions to foods. In fact, an increase in antifood IgG in some cases may be indicative of successful treatment of an IgE-mediated allergy.6

The subject of IgG-mediated food allergy is complicated because of the antibody's nature and the immunological reactions associated with it.

**Food Allergy-associated IgG**
Four distinct subclasses of IgG have been identified: IgG1, IgG2, IgG3, and IgG4. Of these, IgG4 seems to be the subclass with a high affinity for food antigens. IgG4 represents a very small proportion of total IgG in normal sera, but reports of the level of IgG4 differ from laboratory to laboratory (range of 0.7% to 4.9%).7

In a newborn baby, IgG1 and IgG3 levels rise rapidly, and IgG1 may reach concentrations close to the adult level at the age of 8 months. In contrast, IgG4 is still only a fraction of the adult level at the age of 2 and may not reach adult levels until the age of 12.8 IgG4 differs from all other IgG subclasses in that it does not trigger the complement cascade by the classical pathway and therefore is least likely to lead to an inflammatory response.7

There is some evidence that antifood IgG may represent some protection from IgE-mediated food allergy. In a 1978 study, symptom-free children had higher levels of IgG antibodies to milk and egg proteins than those who developed allergic symptoms.9 Results from a later study suggested that a high IgG/IgE ratio in cord blood was a good prognostic sign, suggesting a decreased risk for food allergy.6

In contrast, other studies suggest that increased levels of antifood IgG, especially IgG4, may be associated with allergy, particularly IgG4 antibodies to the milk protein beta-lactoglobulin in atopic dermatitis (eczema) in children.10

IgE-mediated food allergy in infants is frequently associated with an increase in gut permeability, which may allow antigenic food molecules to pass into circulation, triggering production of antifood IgG. Therefore, it is logical to expect to find higher-than-normal levels of antifood IgG in these infants. It is possible that these antifood IgG antibodies would represent a protective mechanism rather than a source of allergic pathology.11 Future research needs to determine the role of IgG, especially IgG4 antifood antibodies, in allergy.

**Type IV Hypersensitivity**
The type IV hypersensitivity reaction is commonly known as a contact allergy. It involves cell-to-cell contact between the antigen (allergen) and T-cell lymphocytes, usually in the skin. Some of the T cells respond to the antigen by producing soluble inflammatory mediators while others develop cytotoxicity, causing damage to cells in the surrounding tissue. The reaction continues as long as the antigen is in contact with body cells; thus, it is often referred to as a cell-mediated response. No antibodies are produced. The reaction appears to be mediated by cytokines such as interleukin 1, interleukin 2, and interferon-gamma released by T cells.12

The allergen involved in a type IV hypersensitivity reaction is usually a simple chemical called a hapten that would normally be unable to elicit an immune system response. By linking with a body protein (usually in the skin), it becomes antigenic. A type IV hypersensitivity reaction is typically a delayed response visible 24 to 72 hours after the skin's exposure to the allergen and is normally diagnosed by means of patch tests. The suspect allergen is applied to the skin and covered by an adhesive bandage, and the site is examined after a delay of up to 72 hours for development of the characteristic reddened wheal.

Although a type IV hypersensitivity reaction can cause damage to intestinal cells in animals, there is no clear

evidence that food can mediate such a reaction in the human digestive tract.13 However, a type of dermatitis on the hands of sensitized individuals in contact with raw foods such as a potato, tomato, apple, watermelon rind, or carrot has been linked to a type IV hypersensitivity response. Speculation has followed that if such a reaction occurs on the hands, a similar response may be expected in the gut epithelium when the food is eaten. At present, there is no good evidence for this, since there are no methods available for assessment of such a reaction within the intestinal epithelial tissue.14

Oral allergy syndrome (OAS) and latex allergy are examples of conditions in which contact with the offending allergen results in local reactivity. Both conditions can lead to severe allergic reactions, and latex allergy is becoming an increasing problem among healthcare workers. Although latex allergy is thought to be initiated by a type IV hypersensitivity reaction, IgE-mediated type I hypersensitivity is the reaction responsible for the acute symptoms in both OAS and latex allergy.

**Nickel Contact Dermatitis and Food Allergy**

An association between food and contact allergy has emerged from observations of nickel-associated dermatitis. Contact allergy to nickel, often suspected when dermatitis develops at sites where nickel-containing jewelry, watchbands, and other metal objects touch the skin, occurs in approximately 10% of females and roughly 3% of males.15 Since the mid-1970s, a number of reports have indicated that dermatitis, especially on the hands, can be aggravated by oral administration of nickel.16-18

Since nickel occurs in many foods to varying degrees, it was assumed that a diet low in nickel may cure or reduce the severity of dermatitis in people who responded positively to oral nickel challenge.17 However, there are differing opinions about what constitutes a low-nickel diet and the degree to which symptom improvement can be achieved on such a regimen.15

Nevertheless, nickel-associated dermatitis serves as an example of the possible association between food constituents and the immune system that differs from the more familiar protein/glycoprotein antigen in an IgE-mediated reaction. Future research will no doubt elucidate the nature of the reaction and possibly uncover other similar interactions.

**Allergy: An Inflammatory Process**

Regardless of the hypersensitivity reaction involved, allergy symptoms result from the release of inflammatory mediators that act on body tissues and cause the clinical condition. In other words, allergy is an inflammatory process, and each inflammatory mediator released in the hypersensitivity reaction has its own effect.

For example, histamine increases the permeability of small blood vessels (capillaries), so fluid moves from the vessels into tissues and causes swelling in various body sites. Histamine is the only known mediator of itching and also causes a widening of blood vessels (vasodilation) and constriction of smooth muscle (eg, around major organs such as the lungs). These effects result in symptoms such as rhinitis (nasal congestion), earache (sometimes with itching and effusion), urticaria (hives) on the skin, and swelling, often of facial tissues (angioedema).

Other effects from histamine are flushing or reddening, headache, hypotension (decreased blood pressure), tachycardia (increased heart rate), bronchospasm due to contraction of smooth muscle around the lung, and mucosal edema (swelling and irritation in mucus membranes).

Prostaglandins, on the other hand, mediate both vasodilation and vasoconstriction. Leukotrienes cause contraction of smooth muscle and are largely responsible for the bronchospasm of asthma. Bradykinin, in conjunction with prostaglandins, causes pain.

Allergy is the result of the combined effect of all of the inflammatory mediators. Treatment usually involves symptomatic relief with drugs designed to combat the local effects of each type of mediator.

**Nonallergenic Mast Cell Degranulation**

The definition of allergy as an immune system-mediated process becomes a little unwieldy, considering there are mechanisms that lead to the degranulation of mast cells that do not require initial stimulation by an allergen.19

Nevertheless, because the inflammatory mediators are released, the resulting symptoms are the same whether or not their release was allergen mediated. The key event is the degranulation of the mast cell. Since the mast cell is an essential immune system component, these reactions should be included under the umbrella of immune-mediated reactions.

Two important factors that can stimulate mast cell degranulation are substance P and vasoactive intestinal peptide. These are neuropeptides and are frequently detectable in the gastrointestinal tract.19 It is possible that they may stimulate the degranulation of intestinal mast cells, resulting in inflammatory mediators being released into the area and contributing to a chronic inflammation, which may present as irritable bowel syndrome.20

Physical factors can also lead to mast cell degranulation. The abrasion of the skin leading to reddened wheals in dermatographia, inhaled cold air passing over the surface of hypersensitive airways in asthma, and cold-induced urticaria are examples of this type of physical degranulation mechanism.21

Some medications such as codeine and morphine can induce mast cell degranulation. Biologically active chemicals such as interferon, phospholipase, and chymotrypsin and certain serum factors and basic polypeptides involved in other physiological processes can likewise release inflammatory mediators from mast cells.19,22

A great deal of research is required in this field, which is presently poorly understood, but recognizing that factors other than the well-known inhaled, ingested, and injected antigens can lead to symptoms resembling allergy should alert practitioners to the fact that sometimes the search for an external allergenic cause for an adverse reaction may be futile.

**Prevalence**

Accurate statistics on the prevalence of food allergy in the general population are difficult to obtain. Consumer surveys in North America and Europe indicate that one third of the population believes they have food allergies.23,24 However, the medical literature suggests that true food allergy (defined as an immediate, IgE-mediated type I hypersensitivity reaction) is uncommon.

Based on double-blind, placebo-controlled food challenges, skin tests, and blood tests to detect antifood antibodies, the consensus seems to be that food allergy affects up to 8% of children under the age of 5 and 1% to 2% of the adult population.11,25-27

Food allergy is more common in children than adults, and the majority of children with food allergies experience symptoms of food-related allergy during the first year of life.28 Most children with food allergies outgrow their early ones, especially to cow's milk and egg proteins, by the age of 5, but some IgE-mediated food allergies may persist throughout life.27,29

The most severe allergic response to a food, a life-threatening anaphylactic reaction, mediated by a type I hypersensitivity reaction, is very rare. Estimates indicate that approximately 100 fatal cases of food-related anaphylaxis occur in the United States each year.30 However, because it may result in deadly anaphylactic shock, such a reaction to a food is always treated aggressively. Extreme precautions to avoid the allergen are crucial.31,32

Statistical studies of the incidence of food allergy may not be representative of the incidence of adverse reactions to foods that are mediated by mechanisms other than type I hypersensitivity. In adults, non–immune-mediated intolerance seems to be much more common than food allergy. Due to the lack of definitive, objective tests, it is impossible to provide reliable statistics. Some practitioners estimate that up to 50% of the total population may experience some degree of food-related reaction.

**Allergenic Foods**

Every food contains potentially allergenic proteins, but some are more likely to cause an allergic reaction than others.33,34 In general, the top eight foods that are more frequent causes of allergy are peanuts, tree nuts, shellfish, fish, eggs, milk, wheat, and soy. Children under the age of 5 are more vulnerable to the development of food allergies because of their immature immune and digestive systems. Cow's milk proteins, egg proteins (especially ovalbumin), and peanuts are the most common foods causing type I hypersensitivity in children. Although there are many more food allergens, the top eight account for approximately 90% of food allergies. See Table 1 for a list of the food sources of tree nuts and foods to avoid with a tree nut allergy.

In January 2006, the Food Allergen Labeling and Consumer Protection Act began requiring that foods containing ingredients with the top eight allergens list those ingredients in common terms or "plain English." Thus, the new law will make it easier for consumers to determine whether a food contains an allergen. In the past, a food label might have used the word "albumin," and a consumer would not know that the ingredient came from egg. Now, the manufacturer must state albumin (egg) or casein (milk).

Food allergens that come from spices, spice blends, and food colorings or flavorings are included in this law. If the label states natural flavoring, it must state natural flavoring (egg, soy, nuts) if that is the source of the flavoring. In addition, it must state which fish, shellfish, or tree nut is in the food product.

As mentioned previously, early food allergies are often outgrown by the age of 5, but certain allergies to foods—peanuts, nuts, shellfish, and sometimes fin fish—can last a lifetime. These foods are most frequently implicated in life-threatening anaphylactic reactions.31 Sometimes, inflammatory mediators are released, or their levels are enhanced by food components or food additives acting through mechanisms that are independent of the immune system. This is presently classified as food intolerance rather than allergy.

**The Dietitian's Role**

Understanding the mechanics of allergic reactions and intolerances can help dietitians explain to their clients why identifying allergenic foods is difficult and time-consuming. There are many bogus "allergy tests" being

offered to susceptible clients. Dietitians can play a supporting role in treatment by carefully monitoring clients' participation in valid testing and helping them provide the detailed information required. They can also provide detailed dietary and nutritional advice to avoid the culprit food(s).

Determining the foods responsible for clinical symptoms requires elimination of the suspect foods for a trial period in which the symptoms of concern should disappear. This is followed by careful challenge of each individual food component in a precisely selected and controlled fashion so that its effects on the body can be monitored. This is the only way in which the role of foods in the disease process can be determined accurately. The laborious elimination and challenge process may be used to confirm or refute any prior allergy tests or establish the most appropriate therapeutic diet in managing specific food-related conditions.

To determine which elimination diet is the most appropriate, several pieces of information are required, including the following:

• a careful medical history, ideally supplied by the patient's doctor (It is especially important that any anaphylactic reactions suspected to be due to food be recorded.);

• exclusion of any other cause for the symptoms, determined by diagnostic tests and procedures, carried out by the patient's medical advisors; and

• results of any allergy tests previously carried out.

Dietitians can assist by carefully interviewing the client to compile a detailed diet history (at least seven days) and symptom record. Additional information should include an assessment of the patient's financial situation, living conditions, cultural and religious food practices, and lifestyle so that appropriate adjustments can be made in the directives for substitute meals in the elimination phase of the program.

Additional information required in the choice of the appropriate elimination diet is provided by a seven-day food intake and symptom record, which the patient is instructed to complete before attending the clinic. The patient should record the following:

• all foods, beverages, medications, and supplements ingested;

• approximate quantities of each;

• composition of compound dishes and drinks (ingredient list);

• the time at which each was taken;

• all symptoms experienced, graded on a four-point scale from mild to severe;

• time of onset and duration of the symptoms;

• whether medications were taken to control the symptoms;

• symptom status on awakening in the morning; and

• any sleep disturbance due to specific symptoms.

Beyond these intake procedures, the dietitian can assist in monitoring and advising the client during the ensuing elimination and challenge regime and help plan a nutritionally adequate diet once the test results are determined.

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**Table 1**
**Food Sources of Tree Nuts**

- Almonds
- Artificial nuts
- Brazil nuts
- Caponatanut meat
- Cashews
- Chestnuts
- Filberts/hazelnuts
- Gianduja (a nut mixture in some chocolate)
- Hickory nuts
- Macadamia nuts
- Mandelonas (peanuts altered to look and taste like tree nuts)
- Marzipan/almond paste
- Nan-gai nuts
- Natural nut extract (eg, almond, walnut)
- Nougat
- Nut butters (eg, cashew)
- Nut meal
- Nut oil
- Nut paste (eg, almond)
- Nut pieces
- Pecans (Mashuga Nuts)
- Pesto
- Pine nuts (Indian nuts, pinon nuts, pignoli nuts, pignon nuts, pigñolia nuts)
- Pistachios
- Pralines
- Walnuts

**References**

1. Joneja JMV, Bielory L. ***Understanding Allergy, Sensitivity, and Immunity: A Comprehensive Guide.*** New Brunswick, N.J.: Rutgers University Press; 1990.

2. Holgate ST. The mast cell and its function in allergic disease. ***Clin Exp Allergy.*** 1991;21Suppl 3:11-16.

3. Frankland AW. Anaphylaxis in relation to food allergy. In: Brostoff J, Challacombe SJ (eds). ***Food Allergy and Intolerance.*** London: Bailliére Tindall; 1987.

4. Nakagawa T, Stadler BM, Heiner DC, et al. Flow-cytometric analysis of human basophil degranulation. II. Degranulation induced by anti-IgE, anti-IgG4 and the calcium ionophore A23187. ***Clin Allergy.*** 1981;11(1):21-30.

5. Berrens L, van Dijk AG, Weemaes CM. Complement consumption in eggwhite and fish sensitivity. ***Clin Allergy.*** 1981;11(2):101-109.

6. Dannaeus A, Inganäs M. A follow-up study of children with food allergy. Clinical course in relation to serum IgE- and IgG-antibody levels to milk, egg and fish. ***Clin Allergy.*** 1981;11(6):533-539.

7. Shakib F. The role of IgG4 in food allergy. In: Brostoff J, Challacombe SJ (eds). ***Food Allergy and Intolerance.*** London: Bailliére Tindall; 1987.

8. Van der Giessen M, Rossouw E, van Veen TA, et al. Quantification of IgG subclasses in sera of normal adults and healthy children between 4 and 12 years of age. ***Clin Exp Immunol.*** 1975;21(3):501-509.

9. Dannaeus A, Johansson SG, Foucard T. Clinical and immunological aspects of food allergy in childhood. II. Development of allergic symptoms and humoral immune response to foods in infants of atopic mothers during the first 24 months of life. ***Acta Paediatr Scand.*** 1978;67(4):497-504.

10. Jenmalm MC, Björkstén B. Exposure to cow's milk during the first 3 months of life is associated with increased levels of IgG subclass antibodies to beta-lactoglobulin to 8 years. ***J Allergy Clin Immunol.*** 1998;102 (4 Pt 1):671-678.

11. Sampson HA. Food allergy. Part 1: Immunopathogenesis and clinical disorders. ***J Allergy Clin Immunol.*** 1999;103(5 Pt 1):717-728.

12. Holgate ST, Church MK, Lichtenstein LM (eds). Allergy, 2nd ed. London: Mosby; 2001

13. Stokes CR, Miller BG, Bourne FJ. Animal models of food sensitivity. In: Brostoff J, Challacombe SJ (eds). ***Food Allergy and Intolerance.*** London: Bailliére Tindall, 1987.

14. Wright R, Robertson D. Non-immune damage to the gut. In: Brostoff J, Challacombe SJ (eds). ***Food Allergy and Intolerance.*** London: Bailliére Tindall, 1987.

15. Booth J. Nickel in the diet and its role in allergic dermatitis. ***J Human Nutr Dietetics.*** 1990;3:233-243.

16. Kaaber K, Veien NK, Tjell JC. Low nickel diet in the treatment of patients with chronic nickel dermatitis. ***Br J Dermatol.*** 1978;98(2):197-201.

17. Veien NK, Hattel T, Laurberg G. Low nickel diet: An open, prospective trial. ***J Am Acad Dermatol.*** 1993;29 (6):1002-1007.

18. Stoltz A, Sauvage C, Lamblin C, et al. Chronic urticaria due to nickel food allergy (Urticaire chronique par allergie alimentaire au nickel) ***Rev Fr Allerg Immunol.*** 2003;43(8):492-496.

19. Brostoff J, Scadding GK, Male D, et al. ***Clinical Immunology.*** New York: Gower Medical Publishing; 1991.

20. Gui XY. Mast cells: A possible link between psychological stress, enteric infection, food allergy and gut hypersensitivity in the irritable bowel syndrome. ***J Gastroenterol Hepatol.*** 1998;13(10):980-989.

21. Champion RH. Urticaria. In: Champion RH, Burton JL, Ebling FJG (eds). ***Textbook of Dermatology, Volume 1.*** 5th ed. Oxford: Blackwell Scientific, 1992.

22. Brostoff J. Mechanisms: An introduction. In: Brostoff J, Challacombe SJ (eds). ***Food Allergy and Intolerance.*** London: Bailliére Tindall; 1987.

23. Sloan AE, Powers ME. A perspective on popular perceptions of adverse reactions to foods. ***J Allergy Clin Immunol.*** 1986;78(1 Pt 2):127-133.

24. Young E, Stoneham MD, Petruckevitch A, et al. A population study of food intolerance. ***Lancet.*** 1994;343 (8906):1127-1130.

25. Botey J, Roger A, Eseverri JL, et al. Immunoallergic techniques for the diagnosis of food allergy. In: ***Food Allergy in Infancy: Proceedings of the International Symposium.*** Palma de Mallorca, Spain. 1991.

26. Farrell M. Food allergy. In: Lawlor GJ, Fischer TJ. (eds) ***Manual of Allergy and Immunology.*** Boston: Little Brown; 1988.

27. Sampson HA, Burks AW. Mechanisms of food allergy. ***Ann Rev Nutr.*** 1996;16:161-177.

28. Bock SA. Prospective appraisal of complaints of adverse reactions to foods in children during the first 3 years of life. ***Pediatrics.*** 1987;79(5):683-688.

29. Høst A, Halken S. A prospective study of cow milk allergy in Danish infants during the first 3 years of life. Clincal course in relation to clinical and immunological type of hypersensitivity reaction. ***Allergy.*** 1990; 45:587-596.

30. Sampson HA. Fatal food-induced anaphylaxis. ***Allergy.*** 1998;53(46 Suppl):125-130.

31. Sampson HA, Mendelson L, Rosen JP. Fatal and near-fatal anaphylactic reactions to food in children and adolescents. ***N Engl J Med.*** 1992;327(6):380-384.

32. Weiler JM. Anaphylaxis in the general population: A frequent and occasionally fatal disorder that is underrecognized. ***J Allergy Clin Immunol.*** 1999;104(2 Pt 1):271-273.

33. Joneja JMV. ***Managing Food Allergy and Intolerance: A Practical Guide.*** Vancouver: McQuaid Publishers; 1995.

34. Joneja JMV. ***Dietary Management of Food Allergies and Intolerances: A comprehensive guide.*** 2nd ed. Hall Publications, Vancouver. 1998.

**Examination**

1. Which of the following antibodies is produced in a type II hypersensitivity reaction?
a. Immunoglobulin A
b. Immunoglobulin D
c. Immunoglobulin E (IgE)

d. Immunoglobulin G (IgG)

2. The tissue damage from a type III hypersensitivity reaction is caused by which of the following?
a. IgE and mast cells
b. Immune complexes and complement
c. IgG and basophils
d. Degranulation of mast cells

3. The presence of antifood IgG always indicates a food allergy.
a. True
b. False

4. A family member develops a rash 24 to 72 hours after coming in contact with a chemical. The doctor diagnoses it as an allergy. Which type of allergy is it?
a. Type I hypersensitivity
b. Type II hypersensitivity
c. Type III hypersensitivity
d. Type IV hypersensitivity

5. Clinical symptoms of a food allergy such as rhinitis, earaches, hives, and swelling are caused by which of the following?
a. Histamine
b. Cytokines
c. IgE
d. T lymphocytes

6. Based on double-blind, placebo-controlled food challenges, skin tests, and blood tests, what is the prevalence of food allergies in children and adults?
a. Up to 8% of children under the age of 5; 1% to 2% of adults
b. 10% to 12% of children under the age of 5; 3% to 6% of adults
c. Up to 8% of children under the age of 10; 2% to 4% of adults
d. 8% to 12% of children under the age of 10; 3% to 6% of adults

7. What are the most common food allergies in children?
a. Shellfish, peanuts, and cow's milk proteins
b. Egg proteins, wheat, and soy
c. Cow's milk proteins, egg proteins, and peanuts
d. Egg proteins, shellfish, and peanuts

8. If an individual has a tree nut allergy, which of the following foods should be avoided?
a. Peanuts, walnuts, pralines, and soy
b. Pesto, marzipan, walnuts, and pralines
c. Almonds, wheat, cashews, and pesto
d. Cashew butter, soy, marzipan, and peanuts

9. Which of the following statements about the Food Allergen Labeling and Consumer Protection Act is true?
a. All foods that cause a food allergy must appear on the food label in plain English.
b. Allergenic foods must be identified on all food labels, except if they are found in miniscule amounts.
c. The source of flavoring and spices do not need to be listed, even if they are an allergic food.
d. The top eight allergenic foods must be listed on the food label in plain English, including flavorings and spices.

10. The optimal method for determining foods responsible for clinical symptoms related to a food allergy is:
a. a skin prick test.
b. blood testing.
c. elimination and challenge dieting.
d. hair analysis.