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Food Allergy in Adults

Food allergy in adults originates from two sources: persistence from childhood and onset after puberty. In infants and young children food allergy is often the first sign of atopy[1]. Allergy to foods usually occurs early (often within the first weeks of life), and allergy to airborne allergens follows later, typically starting after 12 months of age. With adults the opposite is frequently observed. In adult-onset food allergy, primary sensitization is to allergens in the environment, and food allergy follows when food antigens with structures similar to those in the air-borne allergens are ingested as food (1).

Incidence of Adult Food Allergy

The clinical characteristics, the process of diagnosis, and the management of food allergy are not as well documented in adults as they are in children (2), so reliable statistics on the incidence of food allergy in adults are difficult to establish.

The incidence of adult food allergy in total populations is estimated to be around 3.5% worldwide, with figures varying widely between different countries (3,4,5). However, severe anaphylaxis[2] to foods is more common in adults than in children, with a reported incidence of 69% of cases occurring in adults (6).

In adults, food allergies (defined as immunologically-mediated reactions) (7) occur much less frequently than food intolerances (non-immunologic reactions to food components and food additives). The opposite is the case in childhood (2).

Characteristics of Adult Food Allergy

Allergy to specific foods is frequently outgrown by the age of 7 years, but in about 30% of cases of adult food allergy, the condition persists from childhood (8). Peanut allergy is the most frequently reported persistent food allergy, followed by allergy to egg and fish, especially shellfish (3). However, even when the early food allergy is outgrown, the allergic tendency (atopy) persists. Many atopic adults develop aeroallergen-triggered rhinitis and/or asthma, and contact dermatitis in response to a variety of environmental allergens (1). Food allergy may then arise as a sequel to the environmental allergy.

Three important conditions represent a majority of cases of adult-onset food allergy in which the route of primary sensitization is through systems other than the digestive tract:

- Oral allergy syndrome (OAS)
- Latex-food allergy
- Occupational allergy

Oral Allergy Syndrome

The term *oral allergy syndrome* (OAS) (9) describes a complex of clinical symptoms in the mucosa of the mouth and throat, that result from direct contact with food allergens in an individual with respiratory allergy to inhaled allergens, usually tree, weed or grass pollens (pollinosis) (10). Persons with OAS show immediate-type symptoms, which usually begin within 5 minutes, and almost always within 30 minutes after contact with the food (11). Symptoms include itching and irritation of oral tissues, swelling of the lips, tongue, and sometimes papules or blistering of these tissues (12). Swelling and "tightening" in the throat (glottic edema) is the most severe local reaction.

Oral symptoms following ingestion of fruits, vegetables and nuts have been described in patients with co-existing allergy to trees of the birch/alder group (11,12,13,14,15,16,17,18,19); weeds such as mugwort (20,21) and ragweed (22,23); and grasses (24,25). Pollinosis typically precedes the onset of oral allergy syndrome, sometimes for as long as several years.

Table 1 provides a list of the foods most frequently associated with pollen allergy.

.... *Continued on next page*

[1] *Atopy* refers to IgE- dependent allergy characterized by rapid onset of symptoms.

[2] *Anaphylaxis* is an IgE-mediated allergic reaction due to the release of chemical mediators from cells caused to degranulate by complexing of antibody (IgE) with its homologous (matching) allergen. The symptoms include edema (swelling), constriction of smooth muscles in the lungs, stomach, and blood vessels, and a fall in blood pressure that in severe cases can progress to circulatory collapse and heart failure, which can result in death.

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Table 1 Plant Foods Associated with Pollen Allergy
List compiled from References 11-25 and 27.

Plant Pollen Allergens	Food Category	Specific Foods
Birch Alder Mugwort Timothy grass Other grasses	Fruits	apple apricot cherry kiwi melon nectarine orange peach pear plum prune
	Vegetables Herbs Spices	anise carrot celery coriander cumin dill fennel green pepper parsley parsnips potato tomato
	Legumes	beans lentils peanut peas soy
	Nuts	almond hazelnut walnut
	Seeds	caraway sunflower
Ragweed	Fruits Vegetables Edible plants	Banana Cantaloupe Cucumber Honeydew melon Watermelon Zucchini Camomile

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It is thought that oral allergy symptoms are caused by a rapid response of the mast cell-bound IgE to allergens released from raw fruits and vegetables as they enter the mouth and come into contact with saliva. Unusually high concentrations of mast cells[3] in the oral and pharyngeal tissues are postulated to result from continual response to pollen allergens in their native form, resembling the mechanism of interaction between pollens and their homologous (matching) IgE antibodies on mast cells in the mucosa of upper and lower airways (13).

Dietary Management of Oral Allergy Syndrome

Avoidance of the food responsible for OAS is the only treatment. This requires accurate identification of the specific food that triggers the condition by direct challenge with the suspect food, which will elicit symptoms on contact with oral tissues.

It is unnecessary for a person with OAS to avoid all of the foods that cross-react with the pollen; only those to which the individual reacts on contact need to be eliminated from the person's diet. It is possible that the pollen-allergic person will develop OAS to other cross-reacting food allergens in the future, but avoidance will not circumvent this possibility. Furthermore, it is impossible to predict which of the foods will lead to OAS in any individual.

The allergens responsible for OAS in foods are almost exclusively heat-labile, and are destroyed by digestive enzymes and gastric acid when they enter the digestive tract (26). This is in contrast to the powerful allergens in the most highly allergenic foods such as peanuts, tree nuts, egg, milk, fish, etc., which are heat-stable and unaffected by heat and digestive processes. Cooking the food associated with OAS renders it relatively non-allergenic, and people with OAS can usually eat the cooked food with impunity, while the raw form causes the symptoms of OAS. Because the allergens are destroyed in the digestive tract, allergy symptoms rarely occur anywhere other than in oral tissues (27).

Latex-Food Allergy

Allergy to natural rubber latex (NRL) is an IgE-mediated hypersensitivity reaction that is frequently seen in health-care workers, workers in the rubber industry, and individuals who have undergone multiple surgical procedures, especially in infancy, such as children with spina bifida (28). Symptoms of NRL allergy include hand eczema, urticaria, angioedema, rhinoconjunctivitis, asthma, and in extreme cases, anaphylaxis (29). The symptoms are triggered by direct contact with a latex product such as gloves, tubing or other rubber products, or by inhalation of airborne NRL allergens in powder, for example from latex gloves.

The raw material of natural rubber products is obtained from the rubber tree *Hevea brasiliensis.* About 25% of the more than 200 different proteins or polypeptides in NRL are allergenic, and induce the production of IgE in susceptible individuals (30,31).

Natural Rubber Latex Allergens in Unrelated Plants

A significant percentage of people with NRL allergy with IgE antibodies to NRL allergens in their serum develop allergy symptoms after ingestion of related plant foods (32). The so-called latex-food syndrome (33) has been demonstrated to be due to cross-reactivity between antigens in latex and in the associated foods. The allergen cross-reactivity appears to be due to IgE antibodies that recognize structurally similar molecules (epitopes) on different proteins (31). As a consequence of such cross-reactivity, persons may experience hypersensitivity reactions to allergens without previous sensitization, or even exposure, to the allergen via the oral route.

Dietary Management of Latex-Food Syndrome

Unlike OAS, latex-food syndrome can result in anaphylaxis, which in very rare cases can be fatal. Therefore, it is essential that any foods that cross-react with latex and trigger symptoms in a person with NRL should be accurately identified and strictly avoided. It is not necessary for the latex-allergic individual to avoid all of the foods that have the potential to trigger latex-food syndrome; only those to which the person reacts adversely need to be eliminated from their diet (34). However, the potential to develop allergy to the cross-

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3 *Mast cells* are leukocytes found mostly in tissues. Mast cells contain granules (therefore are termed granulocytes) in which inflammatory mediators are stored. The inflammatory mediators are released when allergens couple with mast-cell bound IgE molecules on the cell surface. Inflammatory mediators act on tissues, resulting in symptoms of allergy.

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reacting foods exists, and the latex-allergic person should be warned that allergy to the related food allergens can develop over time (1). Because the first signs of a reaction to these foods usually results in mild symptoms, there is little danger of anaphylaxis when symptoms are first observed on ingestion of the offending food. However, thereafter, any foods eliciting symptoms on ingestion should be avoided because of the risk of a rapid escalation of the reaction to anaphylaxis.

A list of foods most commonly eliciting symptoms in a person with NRL can be found in **Table 2.**

Table 2. Food Allergens Associated with Latex Allergens

Categorization of plant foods based on level of investigation. List compiled from References: 28-34

Type I category has the strongest level of evidence for cross-reactivity
Type II indicates positive, but limited evidence
Type III is based on clinical evidence alone

Category	Food
▪ **Type I** Clinical findings and Identification of cross-reactive allergens	Avocado Banana Cherimoya Passion fruit Kiwi fruit Papaya Mango Chestnut Tomato Bell pepper Celery Potato Custard apple
▪ **Type II** Clinical findings Characterization of cross-reactive components by extract inhibition assays	Peach Fig Melon Pineapple
▪ **Type III** Clinical findings only	Watermelon Apple Cherry Apricot Strawberry Loquat Coconut Carrot Spinach Eggplant (Aubergine)

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Occupational Allergy

Severe allergic reactions to foods often occur in adults who are regularly exposed to food allergens in the workplace (35). Such reactions can have a significant negative impact on the sufferer's livelihood and quality of life.

Incidence of Occupational Allergy

The food industry employs probably the largest number of workers who are exposed to numerous allergens that are capable of inducing immunologic reactions leading to allergy - in the most severe cases, life-threatening anaphylactic reactions. It is estimated that between 2% and 15% of all asthma is related to occupation (36).

Allergic reactions also occur due to handling of the food, especially in the raw state. There are numerous case reports of contact dermatitis, atopic dermatitis, urticaria and angioedema in food production workers (35). Workers in the seafood processing industry, laboratory technicians and researchers, jewellery polishers, restaurant chefs, fishmongers and fishermen are among the industries frequently reported to be sensitized to seafood allergens (37,38),but the actual incidence of work-related allergy in any of these professions is unknown. Attempts have been made to define risk levels based on the quantity of allergen in various workplace settings (37), but safe standards for contact with allergens are not yet available in any food-related industry at the present time.

Since the late 1980s the medical and scientific literature has abounded in reports of occupational allergy to foods. For additional information, two reviews are particularly recommended (35, 37). Research in this topic is particularly active, because of its importance in many branches of the food industry. New papers are appearing in the scientific and medical literature with great frequency.

Management of Occupational Allergy

As in all cases of food allergy the only effective management strategy is avoidance of the food responsible. This is clearly a difficult proposition when a person's livelihood depends on being in the situation that puts them most at risk. Several countries have attempted to address the problem by setting limits on the quantity of allergen to which the workers are exposed, for example by air sampling in food production areas when aerosolised allergen is the problem as in grain and flour handlers (39). However, most food industries do not have such standards and limiting exposure becomes the personal responsibility of the worker.

Occupational allergy to food is a fairly recent area of research, but the increasing awareness of the risk to workers and to the general public is prompting a more careful examination of the problem. Reports of fatal and near-fatal anaphylactic reactions to inhaled and contact food allergens and debilitating conditions that can persist for life, such as asthma, make this an extremely important aspect of food allergy in adults.

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References

(1) Moneret-Vautrin DA, Morisette M. Adult food allergy. Curr Allergy Asthma Rep 2005;5:80-85

(2) Crespo JF, Rodriguez J. Food allergy in adulthood. Allergy 2003;58:98-113

(3) Sampson HA. Update on food allergy. J Allergy Clin Immunol 2004;113(5):805-819

(4) Kanny G, Moneret-Vautrin DA, Flabbee J, Beaudouin E, Morisset M, Thevenin F. Population study of food allergy in France. J Allergy Clin Immunol 2001;108:133-140

(5) Zuberbier T, Ebenharter G, Worm M, Ehlers I, Reimann S, Hantke T, Roehr CC, Bergmann KE, Niggemann B. Prevalence of adverse reactions to food in Germany: a population study. Allergy 2004;59:338-345

(6) Moneret-Vautrin DA., Morisset M, Flabbee J, Beaudouin E, Kanny G. Epidemiology of life-threatening and lethal anaphylaxis: a review. Allergy 2005;60(4):443-451

(7) Johansson SGO, Hourihane JOB, Bousquet J, Bruijnzeel-Koomen C, Dreborg S, Haahtela T, Kowalski ML, Mygind N, Ring J, van Cauwenberge P, van Hage-Hamsten M, Wüthrich B . A revised nomenclature for allergy. An EAACI position statement from the EAACI nomenclature task force. Allergy 2001;56:813-824

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(8) Circle of Clinical and Biological Investigations in Food Allergy http://www.cicbaa.org/pages_us/index_us.html

(9) Amlot PL, Kemeny DM, Zachary C, Parkes P, Lessof MH. Oral allergy syndrome (OAS): Symptoms of IgE-mediated hypersensitivity to foods. Clin Allergy 1987; 17: 33-42

(10) Kelso JM. Oral allergy syndrome. J Allergy Clin Immunol 1995; 96(2): 275

(11) Eriksson NE, Formgran H, Svenonius E. Food hypersensitivity in patients with pollen allergy. Allergy 1982; 37: 437-443

(12) Eriksson NE, Wihl JA, Arrendhal H. Birch pollen-related food hypersensitivity: influence of total and specific IgE levels: a multicenter study. Allergy 1983; 38: 353-357

(13) Halmepuro L, Vuontela K, Kalimo K, Bjorksten F. Cross-reactivity of IgE antibodies with allergens in birch pollen, fruits and vegetables. Int Arch Allergy Appl Immunol 1984;74: 235-240

(14) Halmepuro I, Lowenstein H. Immunological investigations of possible structural similarities between pollen antigens and antigens in apple, carrot, and celery tuber. Allergy 1985; 40: 264-272

(15) Calkhoven PG, Aalbers M, Koshte VL, Pos O, Oei HD, Aalberse RC. Cross-reactivity among birch pollen, vegetables and fruits as detected by IgE antibodies is due to at least three distinct cross-reactive structures. Allergy 1987; 42: 382-390

(16) Lahti A, Bjorksten F, Hannuskela M. Allergy to birch pollen and apple and cross- reactivity of the allergens studies with RAST. Allergy 1980; 35: 297-300

(17) Lowenstein H, Eriksson N. Hypersensitivity to foods among birch pollen allergic patients. Allergy 1983; 38: 577-578

(18) Pauli G, Bessot JC, Dietemann-Molard A, Braun PA, Thierry R. Celery sensitivity: clinical and immunological correlations with pollen allergy. Clin Allergy 985;15(3):273-279

(19) Dreborg S, Foucard T. Allergy to apple, carrot and potato in children with birch pollen allergy. Allergy 1983; 38: 167-172

(20) Vallier P, Dechamp C, Vial O, Deviller P. A study of allergens in celery with cross- sensitivity to mugwort and birch pollens. Clin Allergy 1988; 18: 491-500

(21) Wuthrich B, Staeger J, Johansson SGO. Celery allergy associated with birch and mugwort pollinosis. Allergy 1990; 45: 566-571

(22) Anderson LB, Dreyfuss EM, Logan J. Melon and banana sensitivity coincident with ragweed pollinosis. J Allergy 1970; 45: 310

(23) Enberg RN, Leickly FE, McCullough S, Bailey J, Ownby DR. Watermelon and ragweed share allergens. J Allergy Clin Immunol 1987; 79: 867-875

(24) Ekramoddoullah AK, Kisil FT, Sehon AH. Allergenic cross-reactivity of cytochromes c of Kentucky bluegrass and perennial ryegrass pollens. Molec Immunol 1982; 19: 1527-1534

(25) Heiss S, Fischer S, Muller W-D, Weber B, Hirschwehr R, Spizauer S, Kraft D, Valenta R. Identification of a 60 kd cross-reactive allergen in pollen and plant-derived food. J Allergy Clin Immunol 1996; 98(5 part 1): 938-947

(26) Breiteneder H. Ebner C. Molecular and biochemical classification of plant-derived food allergens. J Allergy Clin Immunol. 2000;106(1 Pt 1):27-36.

(27) Joneja JMV. Oral Allergy Syndrome, Cross-reacting Allergens and Co-occurring Allergies. J Nutr Environ Med 1999;9(4):289-303(15)

(28) Wagner S, Breitenender H. The latex-fruit syndrome. Biochem Soc Trans. 2001 Nov;30(Pt 6):935-40

(29) Alenius H, Turjanmaa K, Palosuo T. Natural rubber latex allergy. Occup Environ Med 2002;59:419-424

(30) Kurup VP, Fink JN. The spectrum of immunologic sensitization in latex allergy. Allergy 2001;56(2):2-12

(31) Kurup VP, Sussman GL, Yeang HY, Elms N, Breiteneder H, Arif SAM, Kelly KJ, Bansal NK,and Fink JN. Specific IgE response to purified and recombinant allergens in latex allergy. Clin Mol Allergy 2005, 3:11

(32) Blanco C, Carrillo T, Castillo R, Quiralte J, Cuevas M. Latex allergy: clinical features and cross-reactivity with fruits. Ann Allergy 1994;73:309-314

(33) Blanco C. Latex-fruit syndrome. Current Allergy and Asthma Reports 2003;3:47-53

(34) Brehler R, Theissen U, Mohr C, Luger T. Latex-fruit syndrome: frequency of cross-reacting IgE antibodies. Allergy 1997;52:404-410

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(35) Aresery M, Lehrer SB. Occupational reactions to foods. Curr Allergy Asthma Rep. 2002;2(1):79-86

(36) Lachowsky F, Lopez M. Occupational allergens. Curr Allergy Asthma Rep 2001, 1:587-593

(37) M F Jeebhay, T G Robins, S B Lehrer, A L Lopata Occupational seafood allergy Occup Environ Med 2001;58:553-562

(38) Malo JL, Cartier A. Occupational reactions in the seafood industry. Clin Rev Allergy 1993;11:223-24

(39) Smith TA. Preventing baker's asthma: an alternative strategy. Occup Med 2004;54:21-27