

## FOOD ALLERGY AND ORAL ALLERGY SYNDROME. Part I. A review

Miglena Balcheva<sup>1</sup>, Angelina Kisselova<sup>2</sup>

1) Faculty of Dental Medicine, Medical University-Varna, Bulgaria

2) Faculty of Dental Medicine, Medical University-Sofia, Bulgaria

### SUMMARY:

Food allergy and intolerance are wide spread nowadays. However, the problem existed and was registered and described still by the ancients. It was fully understood and scientifically depicted in the XXth century after IgE and anaphylaxis were discovered, new diagnostic tests were initiated and the term “allergy” was introduced.

There are some interesting aspects of the problem. Epidemiology is the first one – in the last two decades the number of people suffering from food allergy increased significantly and reached 4% of the population. Food allergy covers all ages, both sexes; atopic people and these with other allergic or digestive diseases are in the risk group also. There is certain influence of the eating habits as well. Etiology is rich and varied. It includes all foodstuffs of plant and animal origin, spices, honey, medicinal products - milk, eggs, meat, fish, nuts, fruits and vegetables, etc. Pathogenesis is complex.

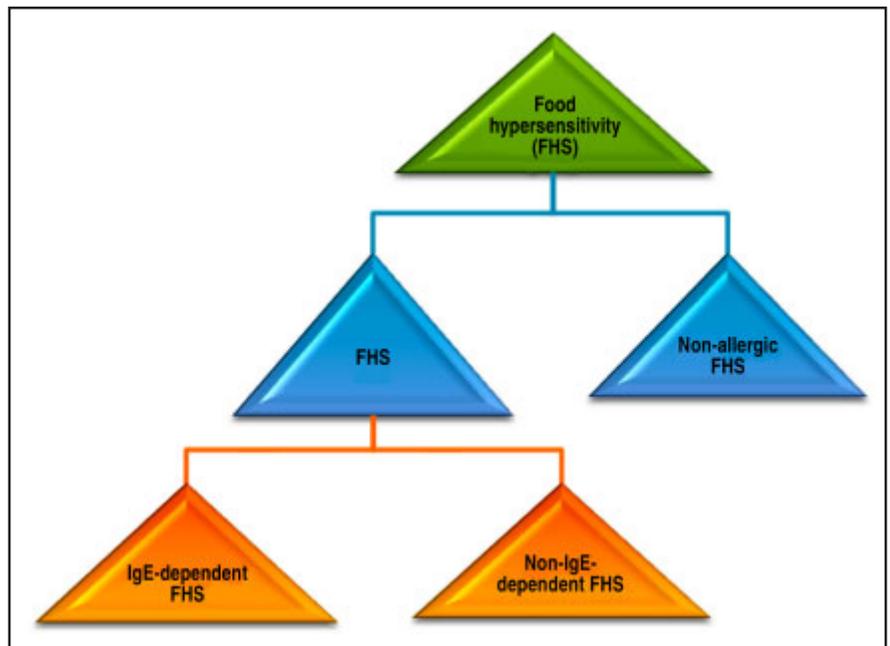
**Key words:** food allergy, oral allergy syndrome, hypersensitivity

### Definition and characteristics:

There is a great variety of symptoms and syndromes, developing after ingestion or contact with certain foodstuffs. It is due to different routes of food allergen penetration (digestive tract, skin and/or respiratory tract), as well as to the mechanisms of origin.

This necessitates creation of exact classification and nomenclature. In 2001, a group of 12 international experts works on allergological nomenclature and offers the use of term “hypersensitivity” as an “umbrella” term.[1] (Figure 1) The umbrella “food hypersensitivity” (FHS) covers the manifestations of “non-allergic food hypersensitivity” (“food intolerance”) and true “food allergy”, as the last could be “IgE-dependent hypersensitivity” or “non-IgE-dependent hypersensitivity”.

Fig.1: Food hypersensitivity “umbrella”



Simpson describes two classes of food allergy (FA), according to route of allergen penetration. In class 1 FA, the food allergens are water-soluble glycoproteins and get into the digestive tract – these are cow's milk, egg white, peanut, soy bean, fish and crustaceans allergens, etc. Class 2 FA is typical for infancy and teenage group. Class 2 FA develops after aeroallergen sensitization, as the allergens are mostly proteins of plant origin (fruits, vegetables, pollens and latex).[2] It occurs in adolescents mainly.

Other authors introduce the term “adverse reactions to foodstuffs” and divide these reactions into toxic and non-toxic ones, which respectively are immunomediated (food allergy) and non-immunomediated reactions (food intolerance or pseudo allergy).[3]

Toxic reactions arise to substances that are: natural toxins or others obtained after food preparation, soils (pesticides, etc.) and food additives (sulphites, acetylsalicylic acid, monosodium glutamate). An interesting phenomenon is the so called “histamine shock” or scombroid food poisoning – it is due to foodstuffs, containing histamine: strawberries, tinned fish, rich in histidine. The name is derived from the fish species, initially associated with the syndrome, i.e. Scombridae family – mackerel, tuna, bonito. The symptoms which occur rapidly after ingestion are: hot flushes, dizziness, blurred vision, headache, nausea, vomiting, abdominal pain, diarrhoea, urticaria and generalised pruritus. Monosodium glutamate (MSG), a food additive containing in the seaweed, is blamed for the Chinese restaurant syndrome – headache, flushing, chest pain, numbness or burning in and around the mouth, facial pressure or swelling, and sweating. The syndrome usually begins within two hours after eating and lasts anywhere from a few hours to a couple of days.

Food intolerance is a result of variety of enzyme deficiencies or mediator releasing (tyramine, histamine, etc.). It refers most often to disaccharidase deficiency - milk intolerance due to lactase deficiency in the intestinal mucosa, for example, or alcohol intolerance due to alcohol dehydrogenase deficiency in the liver.

True food allergy (*allergia alimentaris*) is defined as an immune response to allergens of foodstuffs and their additives, mediated most frequently by antigen-antibody humoral reactions (Fries JH, 1981).

### **Epidemiology:**

The frequency of food allergies is increasing significantly in the last two decades and now affects 4% of the general population, irrespective of age. The distribution of food allergy according to age, gender, foodstuffs, eating habits, geographical location, etc. is a subject of various studies, showing that:

Food allergies in children pose almost as many problems for Public Health, as asthma does.[4] According to other researches the adverse immune response to food proteins affects 6% of children and 3%-4% of adults in

countries with Western lifestyle.[5] After studying different sources, Kagan (2003) gives the following data for frequency of food disease - 5% - 6% in children and 1%-2% in adults.[6]

There is higher incidence of food allergy in females than in males.

The frequency of food allergies is much greater in atopic individuals (in cases of pollinosis, asthma and atopic dermatitis particularly), as well in individuals with diseases of the digestive tract.[7]

The major etiological factors are fruits and vegetables (Apiaceae, Fabaceae and Rosaceae families), milk, eggs, fish and seafood, nuts, meat.

The presenting symptoms of food allergy are urticaria, oral allergy syndrome (OAS), asthma, anaphylaxis, gastrointestinal symptoms, rhinitis and rhino-conjunctivitis.

The eating habits of a population exercise certain influence over the food allergy distribution also. Investigations show that in some countries, the common foodstuffs eaten are more often responsible for food allergies. Here are some interesting examples: fish in Scandinavian countries and Spain, raw fish (responsible for allergies to *Anisakis*) in Spain, The Netherlands and Japan, citrus fruits and fruits in general in Israel and Spain, lentils in Spain, snails in Italy and Spain, seafood, certain tropical fruits and birds' nests in South-East Asia and Singapore.[8-12]

### **Etiology and pathogenesis:**

Etiology of food allergy is extremely rich and various - it includes practically all foodstuffs, as well as preserving agents, additives and spices. In addition, the culinary treatment modifies the antigen features of foodstuffs. The risk of sensitization is increasing also in cases of uniform regimen.

Specific food allergens are referred to the different age groups. Class 1 food allergy prevails in the childhood and the allergens are milk, eggs, soya, peanuts, other nuts and fish. This allergy begins in early infancy, but there is also a tendency for acquiring tolerance to the causal allergens. The interesting fact is that the skin tests remain positive.[3] Sensitization to cow's milk for example is caused by caseins and beta-lactoglobulins mainly, but it disappears in 90% of cases and tolerance appears till the age of 2 years. For eggs, whose allergens are contained mostly in the egg white, the sensitization disappears in 50% of cases till the age of 5 years. A problem may arise only when an immunization is necessary and the vaccine is cultured on embryonated hen's egg. Sensitization to soya disappears in 90% of cases till the age of 5 years also.

The mechanism of immunological tolerance acquisition is not completely clear, but the role of the maturation of the digestive immune system (GALT) has been suggested. The specific IgE-response and the nature of the antigenic epitopes are also discussed.[13]

On the other hand, sensitization to peanuts, other

nuts, fish and seafood has a tendency to persist, at times even for life.[14]

Class 2 food allergy prevails in adults and it is most frequently caused by fresh fruits and vegetables - apple, peach, kiwi, banana, strawberry, celery, carrot. It results with specific reaction - the so called oral allergy syndrome (OAS). The risk for "new" allergens' appearance is increasing with the development of the society - eating habits are modifying with the introduction of new foodstuffs and spices; contemporary culinary technologies may lead to structural changes and rise of various food allergens; there is greater probability for cross reactivity. The last one is due to similarity of the allergenic epitopes in the proteins. The cross reactivity between latex, with which we are in daily contact, and the allergens of chestnut, spinach, walnut, kiwi, banana, avocado

and melon, is very important for us as dentists.

The routes of sensibilization with food allergens are digestive tract mainly and skin and respiratory tract to a lower degree. The allergens, passed through the physiological and immunological barriers of digestive tract, provoke immune response. The majority of food allergies is characterized by a rapid onset following exposure to the allergen and belongs to Type I, IgE-mediated hypersensitivity. The mediators of these reactions are histamine, leukotrienes. The symptoms are anaphylactic shock, urticaria, angioedema, laryngeal swelling, oral allergy syndrome, vomiting and diarrhoea, respiratory symptoms, ophthalmic pruritus and oozing, etc. There is no convincing data about the other humoral mechanisms' involvement. Symptoms like contact dermatitis and dermatitis herpetiformis are assigned to cell-mediated hypersensitivity.

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## REFERENCES:

1. Johansson SG, Hourihane JO, Bousquet J, Brujnzeel-Koomen C, Dreborg S, Haahtela T. et al. A revised nomenclature for allergy. An EAACI position statement for the EAACI nomenclature task force. *Allergy*. 2001 Sep;56(9):813-24. [[PubMed](#)] [[CrossRef](#)]
2. Simpson HA. Adverse reactions to food. In: *Allergy – principles and practice*. Ed. by Middleton E, Mosby, St. Louis, 2003, 1619-1643
3. Petrunov B, Dimitrov V, Kisselova-Yaneva A. – Clinical immunology. Clinical allergology. Dental clinical allergology. *ARSO Medical Publisher*, 2009 [in Bulgarian]
4. Rance F, Dutau G. Food allergy. *Expansion Formation et Editions, Paris, France*, 2008.
5. Sicherer SH, Sampson HA. Food allergy: recent advances in pathophysiology and treatment. *Annu Rev Med*. 2009 Feb;60:261-77. [[PubMed](#)] [[CrossRef](#)]
6. Kagan RS. Food allergy: an overview. *Environ Health Perspect*. 2003 Feb;111(2):223-225. [[PubMed](#)]
7. Mileva Zh. Frequency of allergic diseases in Bulgaria. *Allergy&Asthma*. 2000; 5(Supl. 1):3-32 [in Bulgarian]
8. Hill DJ, Hosking CS, Heine RG. Clinical spectrum of food allergy in children in Australia and South-East Asia: identification and targets for treatment. *Ann. Med.*, 1999 Aug; 31(4): 272-81. [[PubMed](#)]
9. Crespo JF, Pascual C, Burks AW, Helm RM, Esteban MM. - Frequency of food allergy in a pediatric population from Spain. *Pediatr Allergy Immunol.*, 1995 Feb;6(1):39-43. [[PubMed](#)]
10. Kivity S, Dunner K, Marian Y. The pattern of food hypersensitivity in patients with onset after 10 years of age. *Clin Exp Allergy*. 1994 Jan;24(1):19-22. [[PubMed](#)]
11. Pascual CY, Fernandez-Crespo J, Sanchez Pastor S, Ayuso R, Garcia Sanchez G, Martin-Esteban M. Allergy to lentils in Spain. *Pediatr Pulmonol*. 2001, Suppl 23, 41-3. [[PubMed](#)]
12. Goh DL, Lau YN, Chew FT, Shek LP, Lee BW. Pattern of food-induced anaphylaxis in children of an Asian community. *Allergy*. 1999 Jan; 54(1):84-6. [[PubMed](#)] [[CrossRef](#)]
13. Chatchatee P, Jarvinen KM, Bardina L, Vila L, Beyer K, Sampson HA. Identification of IgE- and IgG-binding epitopes on b- and k-casein in cow's milk allergic patients. *Clin Exp Allergy*. 2001 Aug;31(8):1256-62. [[PubMed](#)] [[CrossRef](#)]
14. Sampson HA. Update on food allergy. *J Allergy Clin Immunol*. 2004 May;113(5):805-19. [[PubMed](#)] [[CrossRef](#)]

### Address for correspondence:

Dr Miglena Balcheva  
FDM, MU-Varna  
84, Tzar Osvoboditel Boul., office 623; 9002, Varna, Bulgaria  
Tel.: +359 888 571 862  
E-mail: dr\_balcheva@abv.bg