

The clinical spectrum of food allergy in adults

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INTRODUCTION

Adverse reactions to food may occur in all age groups. These true reactions can be subdivided into two general groups based upon the mechanism of reaction¹. Food allergies are those adverse reactions that are caused by or modified by an immune mechanism. Most of these reactions can be classified as Gel and Coombs type I, immune mechanism which involves: 1) an atopic familial tendency to develop IgE reactions to common allergens; 2) prior exposure to the allergen; 3) IgE antibody formation and 4) "sensitization;" mast or basophil "effector" cell fixation - followed by 5) allergen re-exposure; 6) antibody and allergen interaction; 7) release or formation of chemical mediators which 8) result in the signs and symptoms of allergy as well as some degree of general "hyper-reactivity."

Some food allergies involve other immune mechanisms such as Gel and Coombs type III (e.g. Heiner's syndrome) or type IV (the gluten-sensitive enteropathy/dermatitis herpetiformis syndrome).

Food intolerances are caused by non-immune events and involve a wide variety of mechanisms. Some of the clinical manifestations of these reactions, however, are allergic-like, and may be confused with true food allergies. Most reactions to food are food intolerances rather than food allergy.

Recently, Pearson called attention to a group of adults who fall into a third group of adverse food reactions - that of Pseudo-Food Allergy². This refers to the false conviction (on the part of the patient) that s/he suffers from food allergy, which leads to a wide range of mental, behavioral and non-specific (and subjective) somatic symptoms. The idea of the existence of a food allergy offers the patient a physical explanation for some sign or symptoms. It is often re-enforced by misleading public information or cultural beliefs as to the importance of diet or a specific food upon general health. Often these patients have significant psychological problems which are easily somatized and orthodox/traditional medicinal intervention has failed to satisfy the patient's concern about the original perceived sign or symptom.

True food allergies are more frequent and a consideration when dealing with infants and young children. True food allergies do occur in adults, however, especially anaphylaxis. Also in adults, food intolerances which have allergic-like manifestations are important considerations. Finally, pseudo-food allergic problems in adults are an emerging problem which demands attention.

FOOD ALLERGY PREVALENCE AND INCIDENCE IN ADULTS

The true prevalence and incidence of food allergy (or adverse reaction to foods as a whole) are not known in either children or adults. When comparing the information (and estimates) that have been published, the following conclusions can be made. Eriksson has reported that adults claim that food allergy may be responsible for 24% of their common complaints³. This percentage corresponds to the perception of 20-28% of either Swedish or USA mothers who believe that their young infants suffered from food allergy^{4,5}. (Even though only 3% could be proved by double-blind, placebo controlled food challenge [DBPCFC] to be true adverse food reactions⁵) In 1989, Lan, Strimas and Bahna reported on the perception of the prevalence of food allergy among 722 USA physicians who were involved in a large survey, 55% of whom were Board certified allergists⁶ (non-responders to this survey, when re-surveyed recorded similar results). The geographic distribution of the responding physicians was uniform throughout the United States. The overall prevalence of food allergy was reported by this group to be 10% of the general population (13% for children and 7% for adults). It is interesting that otolaryngology specialists (who practiced allergy) reported higher prevalence figures (19% overall; 21% in children and 13% in adults). Heiner has estimated that 10% of children have cow's milk sensitivity during the first few years of life, but the prevalence of this condition is less than 1% thereafter⁷. Buckley has estimated that the prevalence of food allergy in children ranges from 0.3 - 7.5% of the general population and that adults have less allergy⁸.

Less is written about the specific prevalence of food allergy in adults, but Metcalfe has estimated that one in ten atopic adults have some food allergy and this figures out to be about 2% of the general population⁹. In 1988, Yunginger et al noted that the reports of fatal anaphylaxis due to food in the United States up until that time has been rare¹⁰. However, he and his colleagues were able to identify 7 fatal cases in a 16 month period through contacts with pathologists in the USA.

Local anaphylaxis, or the oral allergy syndrome, may be a fairly common reaction among ragweed allergic individuals in the USA. Anderson in 1970, and Enberg in 1987 estimated that 6-13%, respectively, of patients with ragweed sensitivity may be symptomatic when eating melons, zucchini, cucumbers or bananas¹¹. This equates to approximately 0.1-0.2% of the general population.

A significant number of adult related food allergy problems can be attributed to the work place. Metcalfe has estimated that approximately 10% of adults with asthma may have some occupational food allergy problem. This equates to approximately another 0.2-0.6% of the general population¹².

Finally, Pearson, in England, has called attention to the increasing problem of adults who have significant disability just because they are convinced that they have a food allergy, the so-called pseudo-food allergy syndrome². His personal practice incidence of this problem is 18%. This problem has been recently reported in Canada¹³. The prevalence of this problem in the USA in the general population is unknown, but clinical ecologists in the United States do not seem to lack for patients, some of which undoubtedly fit into this syndrome¹⁴.

On the basis of this information, I estimate that food allergy problems in adults probably are in a prevalence range of 1-2% of the general population.

NATURAL HISTORY OF FOOD ALLERGY IN ADULTS

Dannaeus (Sweden) in a follow up of 82 clinically allergic young children, 2-5 years later, found that clinical tolerance to some degree to the food that they were originally allergic to, had occurred in most children. He noted, however, that there was more likelihood of clinical tolerance with egg sensitivity than to sensitivity to either fish or tree nut¹⁵. Bock (USA), in his prospective study of 501 infants, of which 15 were proven to be food allergic by DBPCFC, within the first year of life, showed that 80% by one or two years and 100% by two to three years later, became clinically tolerant to the food¹⁶. His reports, however, of nine older children, who had had life-threatening systemic reactions to food showed that clinical recovery of food tolerance, which was much slower¹⁷.

In 1989 Sampson and Scanlon (USA) reported on food re-challenge studies in 75 children, ages 3-18, who had been proven to be allergic by DBPCFC and then avoided that specific food¹⁸. Re-challenge, 1-2 years later showed that clinical tolerance had been achieved in some patients. The percent of recovery differed by different foods (soy 50%, wheat 33%, peanut 20%, cow's milk 19%).

Studies in 45 adults, using the DBPCFC technique, have been reported by Adkins and his colleagues (USA) in 1985 (age range 25-51 years). These studies demonstrated that the average food reaction had persisted for 14 years¹⁹. Yunginger (USA) in his reports of food anaphylactic deaths commented that the five adults (18-43 years) had had many years of repeated clinical reactions, before the reaction that resulted in death¹⁰. Peanut was the most common food associated with death in Yunginger series (4/7 cases). Bock reported in 1989 about restudies in 32 proven cases of peanut allergic children 2-14 years later. All of the 32 patients had either recently had a clinical anaphylactic reaction or had positive repeat DBPCFC with peanut²⁰.

It is safe to conclude from the foregoing information that the natural history of food allergy varies by the age of the patient,

by the type of food, and by the severity of the initial reaction. Young infants, sensitive to foods such as soy, wheat and eggs and probably cow's milk, who have a relatively mild reaction, may quickly develop a clinical tolerance to these specific foods. Those children, however, who are sensitized to peanuts, fish and nuts, are less likely to develop a clinical tolerance - if at all. Those patients who have severe reactions to any foods develop a clinical tolerance at a much slower rate. Adults, with a severe anaphylactic history, are at the other end of the spectrum. Potential or actual life-threatening sensitivity may be life long, especially to peanuts but also to crustacean seafood (shrimp, lobster, and crab), tree nuts, fish, and possibly eggs.

CLINICAL ALLERGIC AND ALLERGIC-LIKE ADVERSE FOOD REACTIONS IN ADULTS

The clinical manifestations of adverse reactions to foods and food additives in adults can be grouped under four headings:

1) allergic reactions, 2) food intolerances (often confused with allergy), 3) other clinical problems possibly affected by diet - but not food allergy, and finally 4) pseudo-allergy. (See table I)

Most true food reaction in adults involves some major sign or symptom or a group of signs or symptoms of anaphylaxis. (See table II) All of these reactions are due to a type I, IgE, immune mechanism.

Additional food reactions, peculiar to adults, include allergic reactions to food protein encountered in the work place. Sensitization (and reactivity) usually occurs through the respiratory route or the dermatologic route. Some patients remain sensitized only by this route (e.g. wheat flour in baker's asthma) and can ingest the cooked food (e.g. bread). In the case of other occupational allergies, sensitization occurs first by the respiratory route (or skin route) but then, once this has occurred, the patient may also react when challenged by mouth¹².

IgE-mediated food reactions involving more elements of chronic inflammation include the uncommon problem of eosinophilic gastroenteritis²¹ and the rare problem of hypersensitive furrowed mouth syndrome²².

In contrast to the other, more usual immunologically mediated food reactions in adults (food allergy) which involve a type I, IgE mechanism is gluten-sensitive enteropathy. Also known as celiac syndrome or sprue - with or without an associated dermatitis herpetiformis, this condition involves a type IV cell mediated pathogenesis²³.

FOOD ANAPHYLAXIS IN ADULTS

Urticaria and systemic anaphylaxis may occur to almost any food in adults²⁴ - even exotic foods²⁵. Serious reactions are more likely to involve the ingestion or exposure to peanuts, crustacean seafood (shrimp, lobster, crab), fish, tree nuts and egg proteins^{9,10,19}. One of the issues that is not clear when dealing with systemic anaphylaxis due to a food, is the relative risk of reaction on re-exposure to that food. Much more information is available on this subject with other forms of systemic anaphylaxis (e.g. penicillin and hymenoptera).

Another form of food anaphylaxis, which usually involves adults in the USA (but is reported in children in other countries such as the Scandinavian countries, is local anaphylaxis of the mouth parts¹¹. This syndrome, involving the concomitant pollen respiratory allergy and the ingestion of raw fruits and vegetables, also is known as the "oral allergy syndrome" and the "fruit and vegetable syndrome".^{24,26}

Anderson et al (USA) in 1970 first recognized that oral pharyngeal symptoms may be provoked in ragweed-sensitive persons in the USA when they ate members of gourd family such as watermelon, cantaloupe, and honey dew melon. They also recognized a similar association when these patients ate banana (a non-gourd). Enberg et al (USA) recently confirmed these cross relationships in ragweed-sensitive patients and extended the list of foods to zucchini squash and cucumbers (other members of the gourd family)¹¹.

Similar associations between pollen sensitivity and oral pharyngeal signs and symptoms to ingestion of hazelnuts, apples, carrots, parsnips, and potato have been recognized in Scandinavia in patients allergic to birch pollen²⁷. Recently, similar reports in patients with grass and tree pollen allergy - among patients in northern Italy with a high degree of association with some of the same foods as noted in Scandinavia including not only apples and nuts, but also peaches, pears, cherries, fennel, tomatoes and carrots²⁶.

In 1989 (Moller, Sweden) reported on the first attempts to treat food sensitivity by either subcutaneous pollen immunotherapy over a three year period with either birch pollen or tree pollen mix in 39 children²⁸. He also reported on a study of 14 children who were treated with capsules containing birch pollen which were orally administered over a 10 month period in a double-blind fashion and paired with a 16 child placebo control group. The pollen immunotherapy, regardless of route, improved the symptoms of rhinoconjunctivitis, but not the local anaphylaxis due to food sensitivity.

Exercise-induced anaphylaxis is a relatively new syndrome described by Sheffer and Austen (USA) in 1980. This syndrome has been

recently reported in many parts of the world, other than the USA and is associated with different exercises - although jogging (69% in USA) is the most frequently reported associated²⁹.

In some patients, with this syndrome, anaphylaxis would only occur after the patient ate a specific food such as celery (3 cases^{30,31}), shrimp³², peach³³, wheat (3 cases³⁴), or any meal³⁵, within two hours of exercising. Although the exact reasons foods/meal ingestion influences the onset of anaphylaxis are unclear, mediator release has been shown, and blood flow differences to the gut as well as enhanced food allergen absorption have been suspected.

In the single report from Japan involving three cases of IgE reactions involving exercise and wheat allergens, there was a high correlation between the presence of pepsin-digested wheat gliadin, and glutenin skin test reactivity and the onset of exercise-induced anaphylactic shock³⁴. The authors of this Japanese report have postulated that the new allergens formed in the stomach after ingestion of the native food explains the late association between food and exercise-induced asthma. Spies (USA), in 1970, had previously reported on the generation of "new antigens" after short pepsin digestion of purified milk antigens which were responsible for anaphylaxis in an animal model³⁶.

Another reason for food anaphylaxis in an adult, may be the contamination of that food with an antibiotic.^{37,38} Fortunately, in the USA, this is an uncommon problem, but it may be a significant problem in other parts of the world where antibiotic use in milk cows or in meat bearing animals is not controlled.

Finally, adults may develop isolated gastrointestinal signs and symptoms (nausea, vomiting, diarrhea) or respiratory problems (rhinitis, conjunctivitis or asthma) as the result of food allergy⁹. Although these signs and symptoms are frequently a part of a generalized anaphylactic reaction, they may occur in adults on occasion as isolated reactions.

OCCUPATIONAL FOOD ALLERGY

The first well described occupational food allergy was baker's asthma as described by Bernadino Ramazzini's (Italy) famous 1705 treatise *De Moribus Artificum Diatriba* (Diseases of Workers)³⁹.

The number of workers who develop respiratory or dermatologic sensitization to food allergens is unknown, but in the USA alone, 21 million persons work in the production, transportation, formulation, packing, or presentation of foods¹². Probably the best studied occupational food allergy, world-wide, is baker's asthma.

The mean time before a baker becomes sensitized to flour varies between four and seventeen years, but in one study, 20% of bakers

have asthma/rhinitis after working twenty years, in this industry. Many more bakers had evidence of IgE sensitization. Although it was previously felt that bakers only become sensitive to the water soluble albumin fraction of wheat, Walsh et al (Australia) has found that bakers may become sensitized to many different wheat fractions, water soluble and water insoluble⁴⁰.

Lachance et al (Canada) recently described an interesting 29 year old patient who worked 13 years producing biscuits and who developed an allergy to an alkaline hydrolysate of wheat gluten (AHGD)⁴¹. Inhalation of this material produced rhinitis and asthma within 15 seconds with a drop in pulmonary function (FEV₁ of 40%) in 30 minutes. The patient failed to react on the inhalation of the native wheat flour or lactose (control). IgE specific antibody to AHGD were greater to this antigen than to whole wheat or native gluten.

Insoluble gluten will absorb antibodies responsible for baker's asthma but would not absorb IgE antibodies directed against AHGD demonstrating the existence of highly selective epitopes for modified gluten (AHGD) in this patient but not the existence of reactivity to the native wheat protein.

Although it has been known for some time that enzymatic alteration of native food could result in new allergens (Spies, 1970³⁶), it has been assumed that all food allergic patients would also react to the native food protein. This case demonstrates that it is possible to become sensitized only to a modified allergen. This information may have, at least, diagnostic implications.

Other examples of food industries in which significant food sensitization is possible include spices⁴², seafoods⁴³, coffee⁴⁴, castor bean⁴⁵, bakery (respiratory egg sensitivity)⁴⁶, honey (sunflower)⁴⁷, and restaurant (mustard)⁴⁸.

The clinical manifestations of food protein allergy, acquired in the work place include the isolated respiratory symptoms of rhinitis, conjunctivitis, and bronchial asthma as well as hives (in contact urticaria) and systemic anaphylaxis. Sensitization of the patient is usually through the respiratory or dermatologic route. Some patients only react on re-challenge through inhalation or skin contact. (e.g. baker's asthma from flour exposure)⁴⁰. These patients can usually ingest the food products (e.g. bread) without problems. Other patients, however, after being sensitized through inhalation or skin contact, are later reactive when the food is ingested (e.g. crab in seafood workers⁴³, egg in bakery workers⁴⁶, ingestion of spice in foods in spice processors⁴², ingestion of honey in patients sensitized to sunflower⁴⁷).

OTHER GASTROINTESTINAL PROBLEMS WHICH MAY BE DUE TO FOOD-INDUCED IgE CHRONIC INFLAMMATION

Eosinophilic gastroenteritis is a relatively uncommon condition which is characterized by an inflammation of the stomach and intestine with either localized or diffuse eosinophilic infiltrates and associated peripheral blood eosinophilia²⁴. The peak incidence of this condition is in the sixth decade of life. Out of 50-60 reported cases, approximately 1/2 have severe food allergies and associated other allergic problems²¹.

Hypersensitive furrowed mouth: In 1970, Huntley (USA) described a child with a protein losing gastroenteropathy who had an unusual swelling, fissuring, and furrowing of the tongue, lips and buccal mucosa. In 1980, Anderson (USA) described two severely food allergic patients, who are now adults, who had a similar appearing mouths²². Total IgE values in the 40,000 unit range were found in the index patient. Biopsies of the buccal mucosa demonstrated numerous IgE forming cells. The index patient ultimately developed eosinophilic gastroenteritis with ulcerations and inflammatory polyps. This patient was sensitive to most protein containing foods, to some degree as demonstrated by both antigen specific antibody levels and the direct stimulation of eosinophilia in food stimulated skin window studies.

GLUTEN-SENSITIVE ENTEROPATHY - DERMATITIS HERPETIFORMIS

Celiac disease (or sprue) - with or without dermatitis herpetiformis, is a non-IgE immune reaction of the GI tract (probably a type IV cell mediated reaction) to storage proteins of wheat and other cereals²³.

PRACTICAL APPROACH TO THE DIAGNOSIS AND MANAGEMENT OF ADULTS SUSPECTED OF ALLERGIC OR ALLERGIC-LIKE REACTIONS TO FOOD

An adequate history can usually establish a reasonable likelihood that the presenting complaint is either due to a food allergy, food intolerance, or is a problem that might be affected by diet (but not allergy) and finally may be a pseudo-food allergy.^{2,9,12,14,20,24,26} Most true food allergy reactions appear like allergic reactions to other allergens (aero-allergies, drugs, insects).

If true food allergy is suspected, IgE-allergen specific antibody tests are advisable since most of these reactions are Type I IgE mediated immunologic events. Adkins et al (USA) showed that there is a good correlation between positive IgE allergen specific antibody skin test reaction and positive DBPCFC results in adults as has been shown previously in children^{19,49}. Except for systemic anaphylaxis, it is often difficult to find a correlation between the patient's history (perception) of reacting to a specific food and the results of DBPCFC. This is as true for adults as it is for children^{19,49}. A specific food history has also proved unreliable in

some food intolerance reactions such as in adults who are suspected of reacting sulfite preservatives in specific food who, then, are challenged either by single blind or by double-blind techniques⁵⁰.

If the patient is suspected of anaphylaxis to a specific food protein, in the recent past (two weeks to six months) usually food allergen specific antibody levels will be high enough that a properly performed IgE food RAST/ELISA test will be positive. If this test is positive, then a presumptive diagnosis of food allergy can be made and the patient can be told to avoid that food. That patient should also be given an epinephrine syringe or an epinephrine auto-injector such as an Epi-Pen (0.3cc of aqueous epinephrine 1:1000) to use in times of an emergency with an inadvertent specific food exposure.

In other suspected cases of non-life threatening episodes of food allergy, the epicutaneous IgE food skin test (prick, scratch, puncture) with proper allergens is usually advised. These skin tests are generally felt to be more sensitive than *in-vitro* tests⁴⁹.

As screening tests, Sampson (in children) and Atkinson (in adults) have shown that if the IgE skin test (or RAST) to a specific food is negative, then there is close to a 100% chance that the patient would be negative if a DBPCF challenge with that food was done^{19,49}.

If the food allergen specific IgE skin tests (or RAST) is positive, it narrows the field as to which foods the patient may be likely to be allergic. In children, however, when the prevalence of food allergy is high, such as in the case with atopic dermatitis, with a positive skin test to food such as eggs, peanuts, milk, wheat or fish there is close to a 50% likelihood that the patient would react if a DBPCFC to those foods were done⁴⁹. In adults, however, where the prevalence of food reaction is much lower, the predictability of a positive IgE food test is probably less than 3%.

Dietary manipulation (food diary, specific food elimination followed by open challenge, restricted diets followed by challenge, and single-blind and DBPCFC) have their place in the evaluation of the suspected patient with allergic or allergic-like food reactions. Although these tests, in themselves, do not determine the mechanism of the reaction, they can determine the existence of a true or false food reaction^{9,24}. The details concerning diets and the method of challenge including DBPCFC for adults are readily available in the literature^{24,26,51}.

DBPCFC is often necessary to assess food intolerant reactions in adults such as those suspected of reacting to food additive (e.g. sulfites, tartrazine and other food preservatives).^{50,52,53} DBPCFC is also helpful in convincing patients that s/he is suffering from pseudo-food allergy².

Specific food/food additive avoidance is good advice in the management of patient's whose dietary problem has been identified. However, there are problems in effectively avoiding specific foods, even if the patient is aware that s/he could have a reaction - even a life-threatening one. Some of the important problems include denial or minimalization on the part of the patient, failure to inquire about the ingredients of food prepared by others, and poor labeling information on prepared foods⁵⁴. Allergists need to be aware of these management problems and to continue to follow and council patients afflicted with an allergic or an allergic-like adverse food reaction.

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Table I

CLINICAL MANIFESTATIONS OF ALLERGIC AND ALLERGIC-LIKE FOOD REACTIONS IN ADULTS

- I. Allergic Reactions
 - A. Anaphylaxis
 - B. Occupational food allergies
 - C. Other gastro-intestinal problems
 1. Eosinophilic gastroenteritis
 2. Hypersensitive furrowed mouth
 - D. Gluten-sensitive enteropathy - Dermatitis herpetiformis
- II. Food Intolerance - Often Confused with Allergy
 - A. Lactose intolerance (Primary adult-type late-onset)
 - B. Natural pharmacological agents in food
 - C. Food additives
 1. SO₂ and Sulfites
 2. Benzoates, BHA/BHT, nitrates, nitrites
 3. Tartrazine (FDA #5 yellow)
 4. Monosodium glutamate (MSG)
 5. Aspartane
- III. Other Clinical Problems Possibly Effected By Diet - But Not Allergy
 - A. Migraine headache
 - B. Rheumatoid arthritis
- IV. Pseudo-Food Allergy

Table II

FOOD ANAPHYLAXIS IN ADULTS

- A. Urticaria and generalized systemic reactions
- B. Local anaphylaxis (oral allergy syndrome, fruit and vegetable syndrome)
- C. Food-related Exercise-induced anaphylaxis
- D. Anaphylaxis to antibiotics contaminating food
- E. Isolated gastrointestinal signs and symptoms (nausea, vomiting, diarrhea)
- F. Isolated respiratory signs and symptoms (rhinitis, conjunctivitis, asthma)

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