# IgE-Mediated Food Allergy; Complications & Implications

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**Abstract:** IgE-mediated allergic diseases are a major socio-economic problem caused by complex interactions between genes and the environment. In general, the production of IgE and allergic inflammation result from overexpression of T-helper 2[(Th2); IgE-producing T-lymphocyte lineage] type immune responses (type I hypersensitivity reactions) cytokines.

On the average, it is estimated that 30-40% of the world population are now, being affected by one or more allergic condition(s) of which FOOD ALLERGIES, for the most part, account for this augmentation and consequently embrace a major portion of the affections(up to 20%). Of course, it should be noted that, the vast majority of reactions due to food allergy do not require hospitalisation and, thus, the true number of distinguished/reported cases is still, likely to be significantly higher.

Virtually, any food protein can cause clinical syndrome in susceptible individual; however, only a small number of foods are responsible for more than 90 percent of allergic food reactions, and most patients are sensitive to fewer than three foods. In children, the most common foods causing reactions are egg, milk, peanuts, soy, wheat, tree nuts and fish. Adults most often react to peanuts, tree nuts, fish and shellfish.

Despite allergy to food proteins having been known for many years, there is little information on whether food antigens are increased in these conditions and how much antigen is absorbed as well as, the biological activity of the absorbed allergen. One explanation could be the limitations of sensitive analytic methods for analyzing food proteins in human serum.

On the other part, current curative cares under evaluation include strategies to block IgE or IgE synthesis and to interrupt the Th2-dependent allergic cascade.

Altogether, the thing of noteworthy is that, a determined effort using the best appropriate food allergy diagnostic technics would be certainly required in order to, produce the most clinically effective/safe patient results and, develop any effectual/promising therapeutic approaches for IgE-mediated food allergies owing to their epidemic worldwide increase in prevalence and morbidity.

**Keywords:** Allergenic Foods, Diagnostic Procedures, Pathomechanisms & Manifestations, Prevention & Treatment Strategies.

## IGE-MEDIATED FOOD ALLERGY: PATHOMECHANISMS & MANIFESTATIONS

Providing the second exposure to coming to pass, the incriminated allergens penetrate mucosal barriers and subsequently cross-link the IgE antibodies residing primarily (upon/over the sensitizing process) on the effector cells (Mast cells and Basophils). Cross-linking of at least two IgE molecules activates mast cells and basophils *via* high affinity IgE receptors (FcεRIs). This activation can trigger the eruption of a whole host of inflammatory mediators through the effector cells degranulation (volcanic phenomenon; in terms of the author) amongst them histamine is the primary and clinically considered as the most important/potent mediator that can cause all the pathological features of allergic diseases [1].

IgE-dependent food-allergic reactions may engage one or more organs including the skin, the gastrointestinal tract, and the respiratory tract. In the severe cases, the cardiovascular system is also affected, followed by systemic shock expansion.

## DIAGNOSTIC PROCEDURES

An initial evaluation is warranted in patients who have the common clinical manifestations of food allergy. The initial evaluation, beginning with a thorough history and physical examination, must consider a broad differential diagnosis, including metabolic disorders, anatomic abnormalities, malignancy, pancreatic insufficiency, non-immunologic adverse reactions to foods and many other disorders that could lead to similar symptoms. Allergic reactions to substances other than foods (e.g., animal dander, molds, dust) must also be considered. Once food allergy is identified as a likely cause of symptoms, confirmation of the diagnosis and identification of the implicated food(s) can proceed.

## PRICK-PUNCTURE SKIN TESTING (SPT)

In the evaluation of IgE-mediated food allergy, specific tests can help to identify or exclude

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responsible foods. One method of determining the presence of specific IgE antibody is prick-puncture skin testing. While the patient is not taking antihistamines, a device such as a bifurcated needle or a lancet is used to puncture the skin through glycerinated extract of a food and also through appropriate positive (histamine) and negative (saline-glycerin) control substances. A local wheal-and-flare response indicates the presence of food-specific IgE antibody, with a wheal diameter of more than 3 mm indicating a positive response.

Prick skin tests are most valuable when they are negative because the negative predictive value of these tests is very high (over 95 percent) [2, 3]. Unfortunately, the positive predictive value is on the order of only 50 percent [2, 3]. Thus, a positive skin test in isolation cannot be considered proof of clinically relevant hypersensitivity, whereas a negative test virtually rules out IgE-mediated food allergy to the food in question.

Intradermal allergy skin tests with food extracts give an unacceptably high false-positive rate and therefore should not be used [3]. The protein in commercial extracts of some fruits and vegetables is prone to degradation, so fresh extracts of these foods are more reliable [4].

## *IN VITRO* TESTING (RAST)

*In vitro* tests for specific IgE- radioallergosorbent tests (RAST)- are more practical than prick skin tests for food allergy screening in the primary care office setting. As with skin tests, a negative result on RAST testing is very reliable in ruling out an IgE-mediated reaction to a particular food, but a positive result has a low positive predictive value. *In vitro* tests for IgE are generally less sensitive than skin tests; however, when highly sensitive assays are used, the levels of food-specific IgE antibody correlate with clinical reactivity to certain foods (e.g., milk, egg, peanuts, fish) [5]. Because most patients with food allergy are sensitive to only a few foods [6, 7] and a small number of foods are responsible for most reactions [6, 7].

When patients have a history suggestive of food-related illness and tests for IgE antibody to the test food are positive, the first course of action is to eliminate the food from the diet. Further testing is usually not needed in patients with severe/acute reactions.

## ORAL FOOD CHALLENGES (OFC)

Double-blind, placebo-controlled food challenges are considered the gold standard for diagnosing food allergy [2, 6, 8]. The procedure is labor intensive but can be modified for an office setting [8]. Patients avoid the suspected food(s) for at least two weeks, antihistamine therapy is discontinued according to the elimination half-life of the specific medication, and doses of asthma medications are reduced as much as possible. After intravenous access is obtained, graded doses of either a challenge food or a placebo food are administered. The food is hidden either in another food or in opaque capsules.

Medical supervision and immediate access to emergency medications, including epinephrine, antihistamines, steroids and inhaled beta agonists, and equipment for cardiopulmonary resuscitation are required because reactions can be severe. During the challenge, patients are assessed frequently for changes in the skin, gastrointestinal tract and respiratory system. Challenges are terminated when a reaction becomes apparent, and emergency medications are given as needed. Patients are also observed for delayed reactions. If allergy to only a few foods is suspected, single-blind or open challenges may be used to screen for reactivity.

Negative challenges are always confirmed with open feeding of a larger, meal-sized portion of the food. Oral challenges should usually not be performed in patients with a clear history of reactivity or a severe reaction.

## PREVENTION & TREATMENT STRATEGIES

So much is certain that the individuals with food allergy (as the case may be) are in urgent want of proper preventative/prophylactic/remedial protocols for these diseases, which have to be taken into service in the ways otherwise than, relying upon the mere palliative medications prescribed to suppress the pertinent symptoms/signs and/or, going on multiple elimination diets, and in turn, experiencing the supposed potentially detrimental side effects arising from any inconsistent applied medical cares (e.g., as a matter of fact; depletion of a great number of nutrients while exercising dietary restrictions is of frequent occurrence). Taking it all round, some key implications and recommendations which fall into and can be directed as, three stages of a preventative protocol, are;

(1) Primary prevention- the minute investigating/tracing the potential sensitizing-activity of the pre-determined and novel allergenic proteins in any accused food and

correctly identifying the immunogen allergens' characteristics, all for inhibiting IgE and other immunological sensitization of atopic-prone infants and children by avoiding any exposure to allergen in infancy.

(2) Secondary prevention- precisely diagnosing/ screening the sensitized cases and abrogating disease expression subsequent to immunological sensitization and (3) tertiary prevention- rational scrutinizing/delineating the mechanistic aspects of the underlying reactions following to re-exposure events, and eventually, suppressing the manifestations and symptoms after and despite disease expression, as well as, drawing firm conclusions as to more appropriately planning/designing a well-set diet(s) also, following up the respective nutritional deficiencies with regard to the omitted/restricted nutritive materials.

In a word, adopting any expedient contrivances and comprehensive assesses/guidelines by turns, all along the line, whereby the complications of allergic reactions might/could be trammelled, all are of pivotal/vital importance for sufferers, and will promise them well, to hasten tolerance as to allergenic protein(s) and/or outgrow the allergy to the nuisant food(s), in course of time, until more effective and acceptable approaches are developed.

## CONCLUSIONS

Irrespective of just a few interlocutory/polemical findings thanks to incessant efforts of a myriad of researches seeking after the most effective treatment for food allergy, there is, as yet, almost no conclusive/curative/remedial medical care for it. Adversely, the rampant prevalence rate of food allergy is dramatically on the upgrade, day in and day out, which in turn, affecting drastically the quality of life of the sufferers and their families and also, negatively impacting the socio-economic welfare of society. Hence, Food Allergy must be regarded as a major health care problem and as much resolving measures are warranted in this field.

Likely, we have somewhere, gone on wrong lines regarding the precise characterization/categorization of adverse reactions to foods, and so forth! Thus, it is prudent to go, once more, critically through the due pathogenesis to point out exactly the involved mechanism at each point in order to produce any promising approach and/or know the ropes, to deal more appropriately with food allergy one of these fine days.

In addition, it is of great necessity to remind the food allergy sufferers of that, the strict adherence to appropriate diets free of the offending food allergens and, having a clearly-defined plan of action for handling the situations in which a food allergen is accidentally ingested, all can provide valuable assistance regarding their problem in so far as they catch a cure, by and by.

Again, it must be borne in mind that, "The right food is the first and best medicine" and "The longer the initial therapy is delayed, the greater the incidence of complications and fatalities".

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