History of Food Allergies

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Adverse reactions to foods, especially large dietary proteins in foods resulting in an allergenic response are referred to as a food allergy. Although food allergies are not a new development and occur in only a small percent of the population, diagnosis and treatment remain a topic of interest with practitioners.¹

In the United States it is estimated that approximately 6-8% of children and 3-4% of adults display true food allergies². In using data gleaned only from hospital discharge records, Chafin et al. (2010) identified that the number of diagnosed food allergies has increased over time. According to their research in 1997; 3.3% of US children had a diagnosed food allergy and 10 years later this number increased to 3.9%, which was considered a statistically significant difference.³ The cause of this increase was unclear; it could be attributed to an actual increase in an immune triggered reaction to food or better reporting mechanisms. Nonetheless, food allergies remain a public health problem, accounting for over 30,000 severe allergic food reactions each year, including hundreds of deaths.²

Causation and Symptoms

Food allergies result from abnormal immunologic reactions to the protein in foods. Traditionally, these reactions have been categorized by those that are immunologically mediated; (IFA) by immunoglobulin (Ig)E, and all others which are nonimmunologically; non-IgE mediated (NFA).
In IgE-mediated reactions, symptoms are rapid, and onset can begin in seconds or as long as two hours after the ingestion of the food substance. In this type of hypersensitivity, the onset is rapid and the duration of the event is short but not without the possibility of severe consequences.

IgE mediated reactions are usually accompanied by symptoms relating to histamine production including flushing, swelling of the lips, face or throat, urticaria and pruritus. In some cases gastrointestinal (GI) involvement occurs and is demonstrated by cramping, nausea and diarrhea.

Non-IgE mediated food allergies were first considered to be a benign condition mediated by cellular immune responses, primarily affecting the GI mucosa. More recent research has focused on “subtle changes in interactions between environmental factors (microbiota, dietary components, etc.) and the gut immune responses… which can result in undesired adverse reactions to food proteins (FPs).”  

The onset of symptoms in NFA occurs more slowly than in IFA making the food offender difficult to assess. The lack of easily accessible diagnostic measures also contributes to the problem. Clients suffering from NFA may report symptoms as widely ranging as joint pain, chronic headaches and irritable bowel disease. Non–IgE-mediated gastrointestinal food allergies include food protein–induced enterocolitis syndrome (FPIES), food protein–induced proctocolitis and food protein–induced enteropathy. While not life threatening, these symptoms affect quality of life and may cause morbidity in rapidly growing infants and toddlers when the NFA is caused by infant formula proteins.

The cause of the NFA appears to be related to the ability of the gut to recognize and tolerate large dietary proteins (DPs). Immune tolerance to (DPs) is partially maintained by active suppressive mechanisms involving antigen (Ag)-specific regulatory T cells.
Healthy subjects without a food allergy frequently have low concentrations of food-specific IgG, IgM, and IgA antibodies in their serum. Food protein–specific IgG antibodies tend to rise in the first months after the introduction of a food and then begin a gradual decline, even though the DP continues to be ingested.\(^6\) If homeostasis is not reached and antibodies remain high, an inflammation of the gastric mucosa will occur.

To further complicate the diagnosis and treatment of NFA and IFA, both IgE and non-IgE can occur in some eosinophilic GI disorders which involve the gastric mucosa.\(^4\)

**The role of food proteins**

Allergic reactions to egg, milk, peanut, tree nuts, fish, shellfish, wheat and soy account for most significant food allergies in the United States, although any food can trigger an allergic response (see Table 1).\(^7\)

<table>
<thead>
<tr>
<th>Prevalence</th>
<th>Infant/child</th>
<th>Adult</th>
</tr>
</thead>
<tbody>
<tr>
<td>Milk</td>
<td>2.50%</td>
<td>0.30%</td>
</tr>
<tr>
<td>Egg</td>
<td>1.50%</td>
<td>0.20%</td>
</tr>
<tr>
<td>Peanut</td>
<td>1.00%</td>
<td>0.60%</td>
</tr>
<tr>
<td>Tree nuts</td>
<td>0.50%</td>
<td>0.60%</td>
</tr>
<tr>
<td>Fish</td>
<td>0.10%</td>
<td>0.40%</td>
</tr>
<tr>
<td>Shellfish</td>
<td>0.10%</td>
<td>2.00%</td>
</tr>
<tr>
<td>Wheat/soy</td>
<td>0.40%</td>
<td>0.30%</td>
</tr>
<tr>
<td>Sesame</td>
<td>0.10%</td>
<td>0.10%</td>
</tr>
<tr>
<td>Overall</td>
<td>5%</td>
<td>3-4%</td>
</tr>
</tbody>
</table>

Jenkins et al. (2007) compared animal food allergens and their human homologs by evaluating protein families, sequence analysis and evolutionary relationships. They noted that “sequence identities to human homologs of greater than 62% typically excluded the protein from being
allergenic in human subjects.”\textsuperscript{8}. This evolutionary look at protein relationships may offer more diagnostic clues when evaluating adverse food reactions.

Interestingly, food preparation appears to affect allergenicity. It is thought that preparation methods may explain the higher rates of peanut allergy in the US and other westernized countries, where peanuts are consumed after roasting. China has lower prevalence rates of peanut allergies which may be attributed to the consumption of peanuts after they have been boiled or fried. The high temperature of roasting (180 °C) peanuts leads to a Maillard reaction that appears to increase protein stability and allergenicity.\textsuperscript{9 10}

**Diagnosis**

Food allergies are frequently misdiagnosed by consumers, resulting in a higher number of self-reported cases than are found when clinical testing occurs. In one study, self-reported cow’s milk allergy was claimed to occur in 3.5\% of the research population while testing of the same population found that the rate of cow’s milk allergy was actually 0.6\%-0.9\%.\textsuperscript{3}

Expert interviewing skills are needed to assess symptomology, time of onset and obtain a list of foods consumed around the time of the adverse reaction. Many times the food is unusual in the diet and may be identified with careful questioning. Questions regarding new ingredients added to previously well-tolerated dishes may prove to be the allergen. In other instances the allergen may be introduced through possible cross-contamination. This may occur in food buffets, salad bars, food manufacturing and processing, and the reuse of oil in a deep fat fryer.

In addition to questions regarding food consumption, it has been documented that exercise, close to the time of the adverse reaction, may have served as a trigger instead of an actual food.
Medications and environmental exposures may also serve as triggers in a small number of people.  

Clinical laboratory testing such as Mediator Release Testing for NFA and for IFA antibody titers, elimination diets, skin prick tests and oral challenge tests can help narrow the field of client specific food allergens.

**Conclusion**

An adverse reaction to a food substance can be classified as IgE mediated or non IgE mediated. These classifications differentiate the onset, symptomology and severity of the reaction. While IgE mediated allergies have been well research, non IgE have been topics of more recent discovery and involve intricate interactions between large dietary proteins and the gut mucosa. The NFA is a topic of much discussion in the Academy of Nutrition and Dietetics Integrative and Functional Medicine listserve as members share expertise in uncovering dietary triggers in the elimination of adverse food reactions in clients.
References


