FOOD ALLERGIES AND INTOLERANCE: ROLE OF DIETARY INTERVENTIONS IN EARLY CHILDHOOD

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Summary

Allergic reactions and food intolerance are widespread phenomena. They affect people of all ages, ethnic backgrounds, and geographic regions. In only a few cases is it life-threatening, but many people suffer chronic disability because of allergy. Food allergy can manifest in any part of the body and can vary in severity from mild to fatal. This depends upon the age of the subject, family history, prior sensitization, other diseases, and on the allergen involved. In young infants, gastrointestinal symptoms are the most common.

The prevalence of atopic diseases is high in most industrialized countries and has risen progressively from the mid-1970s to the present time. The contributing factors for this increase are not clear. The current estimates of the prevalence in most industrialized countries are 10% to 20% for allergic diseases and 1% to 5% for food allergy in children up to 16 years old.

At the very outset, one can state the conclusion that it **is** possible to reduce the incidence of allergic diseases by dietary and other precautions in the first few years of life.

This article is based on original research papers, reviews and monographs and summarizes a series of studies conducted in the late twentieth century on adults and children. Terminology is defined, and the principles of diagnosis, treatment, and prevention are outlined.

1. Food Intolerance

It is important to define the term *food allergy* as an immunologically mediated clinical syndrome that develops after the ingestion of a dietary product. This is to distinguish it from the all-inclusive term *food intolerance*, which also refers to nonimmunologically mediated reactions such as symptoms due to enzyme defects, food toxins, and microbial contamination, and idiosyncratic reactions. The common causes of nonallergic reactions include:

(a) Gastrointestinal symptoms can be caused by reduced activity of lactase or other disacharidases.
(b) Vasoactive amines in foods can cause nonallergic adverse reactions (Table 1). Several vasoactive amines (histamine, tyramine, dopamine, epinephrine, norepinephrine, 5-hydroxytryptamine, and phenylethylamine) are present in a variety of fruit, cheeses, wine, and chocolate. The mediators act on the skin, blood vessels, and bronchioles and produce clinical manifestations such as urticaria, angioedema, difficulty in swallowing, wheezing, and migraine. A list of common foods containing histamine and tyramine is given in Table 2.
(c) Toxins, which are both present naturally (examples are proteases in legumes, hypoglycin in akee plant, cyclopeptides and muscarine in mushrooms, cycads in palm tree nuts, and oxalates in spinach and rhubarb) and added fortuitously during preparation and processing, can also cause adverse reactions (see *Antinutritional Components in Food Legumes and Effects of Processing*). In addition, urticaria can be induced by food dyes (azo dyes), preservatives (benzoate, nitrite, and sorbic acid), flavoring agents (salicylates), antioxidants (hydroxytoluene, sulfite, and gallate), and emulsifiers or stabilizers (polysorbate and gums).

Other foods consumed in large amounts may cause local gastrointestinal irritation, for example prunes and onions.

Compound	Example
Histamine	Wine, fermented cheese, anchovies, spinach
Tyramine	Wine, beer, pickled herring, cheese
Glutamate	Soy sauce
Phenylethylamine	Chocolate, cheese, wine
Nitrite	Sausage

Table 1. Vasoactive compounds in food

Histamine and Tyramine	Content μg/g
Histamine	
Cheese (Cheddar, Gouda, Stilton, Roquefort etc.)	20-2200
Spinach	40
Tomato	24

Sausage	240
Anchovies	40
Sardines	128
Canned foods	20-400
Wine, beer	10-28
Tyramine	
Cheese (Camembert, Cheddar, Stilton, Roquefort, Parmesan)	40-2000
Beer, wine	4-28
Yeast	1600
Marinated herring	3800
Avocado	24
Raspberry	64

Table 2. Approximate content of histamine and tyramine in selected foods

2. Pathogenesis and Immunologic Mechanisms

After ingestion, foods are broken down into small fragments but some undigested macromolecules may be absorbed intact and stimulate the production of antibodies of various immunoglobulin isotypes (IgE [Type I reaction], IgG, IgA, IgM) as well as cell-mediated immune responses (Type IV reaction). On subsequent exposure to the same allergen, immune complexes are formed (Type III reaction). Depending upon their size and the efficiency of the scavenger reticuloendothelial system, these complexes may be promptly cleared from the body or may be lodged in sites such as the perivascular tissue, renal glomerulus, or the skin.

Food allergy occurs more often when the above noted pathways are facilitated. For example, if the intestinal mucosal barrier is not intact, as in low birth-weight infants, or following an episode of viral gastroenteritis, there is enhanced absorption of macromolecules. In those with a strong family history of atopy, there is increased production of IgE antibodies. Genetic factors may determine the affinity of antibody produced and thus the type of immune complex formed.

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Biographical Sketch

Ranjit Kumar Chandra graduated in medicine from Punjab University in India and did residency training in pediatrics in New Delhi and postdoctoral work in Immunology at the University of London. Dr. Chandra is a University Research Professor at Memorial University of Newfoundland and Director of Immunology/Allergy at the Janeway Child Health Center in St. John's, Canada. He is also the first Carnegie Visiting Professor at the John Hopkins University. He is an Adjunct Professor at New York Medical College and Honorary Professor at universities in Beijing, Napoli, Santiago do Chile, and Antigua (Colombia). Currently, he is President of Universite Internationale des Sciences de la Sante.
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