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Food allergy affects about 8% of children (1), with milk, eggs, fish, and nuts as causative foods and atopic dermatitis as the most common clinical presentation. However, only about 1% of adults remain allergic to these foods according to European epidemiologic studies (2). By contrast, a recent survey in the USA found that 1.3% of the adult population is allergic to peanuts and tree nuts (3), and this may be explained by the high consumption in that country, as well as by the fact that this kind of allergy is usually not outgrown with age. In adults, vegetable foods become the most frequent source of sensitization, with the typical clinical picture of IgE-mediated food allergy, especially with anaphylaxis and oral allergy syndrome (OAS).

Anaphylaxis results from the action of pharmacologically active mediators targeting cutaneous, gastrointestinal, respiratory, and cardiovascular systems and, according to recent reports, is mainly caused by foods. Peanuts in particular are a major factor in fatal reactions, which can currently be estimated at about 100 cases per year in the USA (3).

OAS is defined by the occurrence of local symptoms at the point of contact between the causative food and the orolabial mucosa, and severe symptoms, including laryngeal oedema and shock, may also follow (4). Even though all the foods may elicit local symptoms, fresh fruits and vegetables are the main causes of OAS. In subjects sensitized to pollens, well-defined clinical associations are observed, such as allergy to birch pollen, apple, and hazelnut; allergy to ragweed and members of the gourd family; and allergy to grasses, tomato, and peanuts. This phenomenon is caused by cross-sensitization to given proteins with similar structures naturally occurring in different, and also botanically unrelated, sources. For example, the major allergens of birch, Bet v 1 – belonging to the family of pathogenesis-

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related proteins – and Bet v 2 – included among the profilins – present with a high degree of homology in foods such as apple, hazelnut, celery, and carrot (4).

However, allergens causing cross-reactions among foods, but not pollens, also exist. This is true of the lipid transfer proteins, which act as major allergens in fruits of the Prunoideae subfamily, such as peach, apricot, plum, and cherry (5), and which are the sensitizing agents in subjects allergic to apple, but not to birch pollen.

The diagnosis of food allergy is hampered by the scant knowledge of the allergenic proteins involved, which thus far has prevented the introduction of standardized extracts and makes the current diagnostic techniques, such as the

skin test and RAST, unreliable. Therefore, clinical reactivity has to be demonstrated by double-blind, placebo-controlled food challenge.

The current treatment is avoidance of the culprit food, but this may prove difficult as some foods are hidden in commercial preparations and expose the allergic subject to unexpected, severe reactions. Therefore, the potential role of specific immunotherapy is currently being evaluated despite the fact that the first attempts with peanuts were discouraging in relation to safety. Developments include vaccines that cannot cause anaphylaxis, an objective achieved either by modifying the allergenic epitopes or by using plasmid DNA encoding the proteins (6).

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