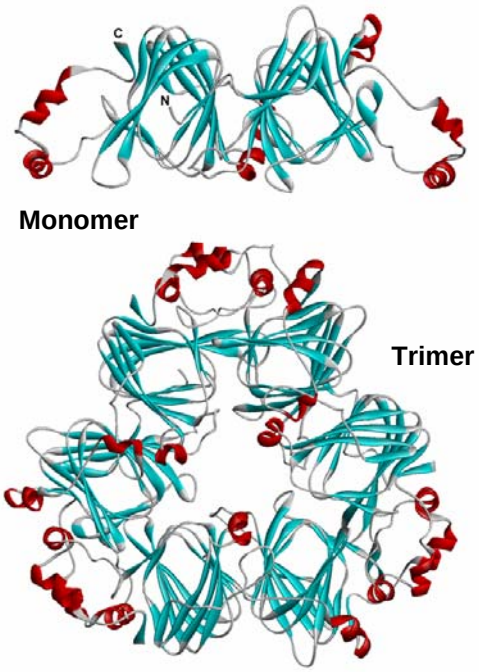
Focus on … Ara h 1 – a Major Allergen (vicilin) from Peanut

Peanuts (*Arachis hypogaea*) are one of the foods most frequently associated with severe allergic reactions, including life-threatening, food-induced anaphylaxis. Peanut allergy often starts early in childhood (median age 24 months), affects 0.5-0.7% of children and in most cases persists through adult life.[1;2] Ara h 1 is a major allergen that causes IgE mediated sensitization in up to 95% of patients with peanut allergy.[3;4] This allergen is a 7S seed storage glycoprotein or vicilin which comprises 12-16% of the total protein content in peanut extracts.[5] The concentration of Ara h 1 in peanuts increases with the size of the kernel (4-16 mg extracted Ara h 1/g peanut), so expression of the protein is associated with peanut maturity.[6]



Ara h 1-homolog[1]

Other vicilin-homologous allergens include Jug n 2 (black walnut), Jug r 2 (English walnut), Len c 1 (lentil), Pis s 1 (pea) and the vicilin-like allergens Cor a 11 (hazel), Ses i 3 (sesame) and Ana o 1 (cashew). Despite the low rate of clinical cross-reactivity between peanut and other legumes, patients with anaphylaxis to pea can have peanut allergy caused by cross-reactive IgE to Ara h 1.[7]

The structure of Ara h 1 has been modeled on the crystal structure of homologous vicilins from kidney bean (phaseolin) and soybean (β-conglycinin) (Figure)[8;9]. Ara h 1 is a homotrimer held together through hydrophobic areas at the distal ends of the monomers, where most of the IgE binding epitopes are located.[10] Each 64.5 kD monomer has a cupin motif which consists of two core β-barrels, each associated to a loop domain of α-helices (Figure, top). Twenty three linear IgE binding epitopes have been mapped in Ara h 1 and substitutions of only one amino acid per epitope led to loss of IgE binding.[11] The interaction between monomers could protect the IgE antibody epitopes from digestion. Stability to thermal denaturation and proteolysis of the trimer may also contribute to the allergenicity of Ara h 1.[8;12]

[1] X-ray crystal structure of the closest crystallized Ara h 1-homolog, β-conglycinin from soy-bean, showing the cupin monomer (top) and the assembled trimer (bottom).

Cooking methods can affect the allergenicity of peanut. Compared to roasted peanuts, the relative amount of Ara h 1 is reduced in fried and boiled peanut preparations.[13] Heat-induced conformational changes do not affect IgE binding to Ara h 1, although heat leads to a secondary more structured conformation with the formation of extended β-structures that alter specific epitopes.[6;14] Roasting increases the efficiency of Ara h 1 extraction and/or the accessibility of the epitopes recognized by the antibodies used to measure the allergen.[6] Ara h 1 was measured in peanut and food extracts using a monoclonal antibody based ELISA (0.1-500 μg Ara h 1/g of food products).[15] Some food matrices, like chocolate, greatly impair extraction and measurement of the allergen.[16] An environmental assessment of Ara h 1, using the same assay, showed that the allergen does not appear to be widely distributed in preschools and schools, and it is easily cleaned from hands and table tops.[17] Another study analyzed the persistence of Ara h 1 in saliva after ingestion of peanut butter, showing levels that are expected to invoke responses in peanut allergic individuals through saliva (kissing, sharing utensils). After evaluating mouth cleansing interventions to reduce salivary peanut allergen, the most effective one was waiting several hours after ingestion and eating a peanut-free meal.[18] You never know what a passionate kiss could lead to!

References

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