

IgG antibodies in food allergy influence allergen-antibody complex formation and binding to B cells: a role for complement receptors.

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Abstract

Allergen-IgE complexes are more efficiently internalized and presented by B cells than allergens alone. It has been suggested that IgG Abs induced by immunotherapy inhibit these processes. Food-allergic patients have high allergen-specific IgG levels. However, the role of these Abs in complex formation and binding to B cells is unknown. To investigate this, we incubated sera of peanut- or cow's milk-allergic patients with their major allergens to form complexes and added them to EBV-transformed or peripheral blood B cells (PBBCs). Samples of birch pollen-allergic patients were used as control. Complex binding to B cells in presence or absence of blocking Abs to CD23, CD32, complement receptor 1 (CR1, CD35), and/or CR2 (CD21) was determined by flow cytometry. Furthermore, intact and IgG-depleted sera were compared. These experiments showed that allergen-Ab complexes formed in birch pollen, as well as food allergy, contained IgE, IgG1, and IgG4 Abs and bound to B cells. Binding of these complexes to EBV-transformed B cells was completely mediated by CD23, whereas binding to PBBCs was dependent on both CD23 and CR2. This reflected differential receptor expression. Upon IgG depletion, allergen-Ab complexes bound to PBBCs exclusively via CD23. These data indicated that IgG Abs are involved in complex formation. The presence of IgG in allergen-IgE complexes results in binding to B cells via CR2 in addition to CD23. The binding to both CR2 and CD23 may affect Ag processing and presentation, and (may) thereby influence the allergic response.