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What is unique about the IgE response?

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Abstract

IgE antibodies are involved in allergic reactions. High affinity IgE antibodies can cause anaphylaxis when cross-linked by minute amounts of antigen. The issue of how the IgE response is initiated and maintained is addressed in this review. A model has been proposed by which IgE(+) cells expressing antibodies that bind with high affinity to their antigens are generated through an IgG1 intermediate, which goes through affinity maturation in germinal centers (GC) before undergoing sequential switching to IgE. Mice deficient in IgG1 produce IgE at almost normal levels, but the IgE antibodies produced in IgG1-deficient mice lack the antigen-binding strength and the somatic mutations associated with affinity maturation. A GFP reporter strain, which expresses a modified IgE molecule, was recently developed and was utilized to challenge the sequential switching model. Several molecules that are highly expressed in GC can antagonize class switching to IgE in GC antagonize partially class switching to IgE; in addition, GC IgE(+) cells are gradually lost from GC as the immune response progresses, as shown with another recently developed, Venus-expressing IgE reporter mouse strain. In contrast, as a population, IgG1 cells thrive in the GC environment. Membrane IgE-expressing plasmablasts and plasma cells (PC) were recognized as a major component of the IgE response in secondary lymphoid organs. The swift development of IgE cells toward the PC fate, together with the affinity maturation of the IgE response via an IgG intermediate, represent the most salient features of the IgE immune responses, which make them distinct from IgG responses.

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