



<p><b>Type I</b></p> <p><b>IgE-Mediated Hypersensitivity</b></p>	<p><b>Type II</b></p> <p><b>IgG-Mediated Cytotoxic Hypersensitivity</b></p>	<p><b>Type III</b></p> <p><b>Immune Complex-Mediated Hypersensitivity</b></p>	<p><b>Type IV</b></p> <p><b>Cell-Mediated Hypersensitivity</b></p>
<p>IgE is bound to mast cells via its Fc portion. When an allergen binds to these antibodies, crosslinking of IgE induces degranulation.</p>	<p>Cells are destroyed by bound antibody, either by activation of complement or by a cytotoxic T cell with an Fc receptor for the antibody (ADCC)</p>	<p>Antigen-antibody complexes are deposited in tissues, causing activation of complement, which attracts neutrophils to the site</p>	<p>Th1 cells secrete cytokines, which activate macrophages and cytotoxic T cells and can cause macrophage accumulation at the site</p>
<p>Causes localized and systemic anaphylaxis, seasonal allergies including hay fever, food allergies such as those to shellfish and peanuts, hives, and eczema</p>	<p>Red blood cells destroyed by complement and antibody during a transfusion of mismatched blood type or during erythroblastosis fetalis</p>	<p>Most common forms of immune complex disease are seen in glomerulonephritis, rheumatoid arthritis, and systemic lupus erythematosus</p>	<p>Most common forms are contact dermatitis, tuberculin reaction, autoimmune diseases such as diabetes mellitus type I, multiple sclerosis, and rheumatoid arthritis</p>

# Hypersensitivity Mechanisms An Overview

**RD Boyd**



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