

Treatment with anti-IL-5 monoclonal antibody can improve allergic disease primarily by blocking IgE class switching in B cells.

True

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IgE-mediated mast cell activation during immediate hypersensitivity requires prior antigen-specific Th2 differentiation rather than continued T-cell involvement during re-exposure.

True

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Goodpasture syndrome represents antibody-mediated tissue injury in which immune complexes deposit in vessel walls activating complements.

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In autoimmune hemolytic anemia, red blood cells predominantly undergo intravascular lysis due to Fc receptor-mediated phagocytosis.

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The direct Coombs test detects complement proteins bound to red blood cells rather than circulating immune complexes.

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ADA deficiency causes lymphocyte toxicity by accumulation of purine metabolites that preferentially damage proliferating mature cells.

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Hyper-IgM syndrome due to CD40L deficiency affects both humoral immunity and macrophage activation.

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During early HIV infection, profound depletion of peripheral blood CD4<sup>+</sup> T-cell counts may occur despite near normal CD4<sup>+</sup> memory T cells in mucosal tissues.

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HIV-specific antibodies frequently fail to control infection because rapid mutation of envelope glycoproteins alters antibody-targeted epitopes without compromising viral fitness.

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X-linked SCID and JAK3 deficiency share a common pathogenic mechanism involving impaired  $\gamma$ c-dependent cytokine signaling.

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