HYPERSENSITIVITY REACTIONS

Overview

Excessive or inappropriate immune responses sometimes lead to host tissue damage resulting from prolonged or repeated antigen exposure. These reactions, called hypersensitivity reactions, cause tissue injury by the release of chemical substances that attract and activate cells and molecules resulting in inflammation.

Types

These reactions are classified into four hypersensitivity types depending upon the mechanism(s) that underlie the tissue damage (Table 1);

the first three types involve antigen-antibody reactions,

while the fourth is antibody-independent, involving cell-mediated immune responses only:

Type I (also called immediate hypersensitivity) reactions are rapid, occurring within minutes of exposure to an antigen, and always involve IgE-mediated degranulation of basophils or mast cells.

Type II reactions are initiated by the binding of antibody to a cell membrane or to the extracellular matrix.

Type III hypersensitivity reactions involve the interaction of antibodies with soluble molecules to make soluble antigenantibody complexes that become deposited in tissues.

Type IV hypersensitivity reactions are those in which cells of the immune system directly attack host cells in the absence of antibody. These reactions include contact dermatitis (CD, also called contact sensitivity, CS); delayed (-type) hypersensitivity (DTH); and, occasionally, cytotoxic T lymphocyte (CTL) responses

Table 1 .Hypersensitivity Reactions

Type	Synonyms	Disorders	Mediated By	Mechanism(s)

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I	Atopy, anaphylactic hypersensitivity, allergy	Allergic reactions, anaphylaxis, asthma	IgE antibody, complement not involved	Cross-linking of FcRa-bound IgE antibodies on mast cells cause degranulation and release of vasoactive amines (e.g., histamine) resulting in smooth muscle contraction, vasoconstriction, and vasodilation of capillary endothelium.
II	Cytotoxic	Erythroblastosis fetalis, Goodpasture's synprodrome, autoimmune hemolytic anemia	IgM or IgG ± complement	IgM or IgG antibody binds to epitopes on cells or other tissue components promoting phagocytosis, antibodydependent cell-mediated cytotoxicity, antibody-mediated function disruption (receptor blocking), or complementmediated lysis
III	Immune complex disease	Serum sickness, Arthus reaction, systemic lupus erythematosus	IgG ± complement	serum activate complement and attract neutrophils that release lytic molecules
IV	Cell-mediated hypersensitivity	Contact dermatitis, tuberculosis, chronic graft	Cell-mediated, antibodyindependent	Release of mediators by sensitized CD4+ T cells provoke tissue

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		rejection		destruction by mononuclear cells. CD8+ T cells known as cytotoxic T lymphocytes (CTL) may kill chemically modified host cells and cells that display disparate MHC molecules.

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