

Hypersensitivity

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Hypersensitivity is the name given to a state in which an immune response damages the body's own tissues. There are four or five types of hypersensitivity that are often described, each is a different way the immune system can damage the host. The four group classification was expounded by P H G Gell and R A A Coombs in 1968.

Type I - Anaphylactic Hypersensitivity

Type I, anaphylactic or immediate-type hypersensitivity is an allergic reaction provoked by reexposure to a specific antigen called an allergen. Exposure may be by ingestion, inhalation, injection or direct contact. The reaction is mediated by IgE antibodies and produced by the immediate release of histamine, arachidonate and derivatives by basophils and mast cells. This causes an inflammatory response leading to an immediate (seconds to minutes) reaction (anaphylaxis).

The reaction may be either local or systemic and symptoms vary from mild irritation to sudden death from anaphylactic shock. The treatment usually involves the administration of inhaled steroids. Alternatively, for particularly severe cases of allergic asthma, treatment with omalizumab is possible.

Some clinical examples:

- Allergic asthma
- Allergic rhinitis (hayfever)
- Atopic dermatitis
- Some kinds of food allergy (most notably that to peanuts or nuts)

Type II - Antibody Dependent Hypersensitivity

In type II hypersensitivity, the antibodies produced by the immune response bind to antigens on the patient's own cell surfaces. The antigens recognised in this way may either be intrinsic ("self" antigen, innately part of the patient's cells) or extrinsic (absorbed onto the cells during exposure to some foreign antigen, possibly as part of infection with a pathogen). IgG and IgM antibodies bind to these antigens to form complexes that activate the classical pathway of complement activation for eliminating cells presenting foreign antigens (which are usually, but not in this case, pathogens). That

is, mediators of acute inflammation are generated at the site and membrane attack complexes cause cell lysis and death. The reaction takes hours to a day.

Some clinical examples:

- Autoimmune haemolytic anaemia
- Goodpasture's syndrome
- Immune thrombocytopenia
- Transfusion reactions
- Type III - immune complex hypersensitivity
- In type III hypersensitivity, soluble immune complexes (aggregations of antigens and IgG and IgM antibodies) form in the blood and are deposited in various tissues (typically the skin, kidney and joints) where they may trigger an immune response according to the classical pathway of complement activation (see above). The reaction takes hours to days to develop.

Some clinical examples:

- Immune complex glomerulonephritis
- Rheumatoid arthritis
- Serum sickness
- Subacute bacterial endocarditis
- Symptoms of malaria
- Systemic lupus erythematosus
- Type IV - cell mediated hypersensitivity
- Type IV hypersensitivity is often called delayed type as the reaction takes two to three days to develop. Unlike the other types, it is not antibody mediated but rather is a type of cell mediated response.

CD8 cytotoxic T cells and CD4 helper T cells recognise antigen in a complex with either type I or II major histocompatibility complex. The antigen presenting cells in this case are macrophages and they release interleukin 1, which stimulates the proliferation of further CD4 cells. These cells release interleukin 2 and gamma interferon, which together regulate the immune reaction. Activated CD8 cells destroy target cells on contact while activated macrophages produce hydrolytic enzymes and, on presentation with certain intracellular pathogens, transform into multinucleated giant cells.

Some clinical examples:

- Contact dermatitis (poison ivy rash, for example)
- Hashimoto's thyroiditis
- Insulin dependent (type I) Diabetes mellitus
- Symptoms of leprosy
- Symptoms of tuberculosis
- Transplant rejection

- Type V - stimulatory hypersensitivity

Type V is an additional type that is sometimes (often in Britain) used as a distinction from Type II.

Instead of binding to cell surface components so the cells are destroyed, the antibodies recognise and bind to cell surface receptors, which either prevents the intended ligand binding with the receptor or mimics the effects of the ligand, thus impairing cell signalling.

Some clinical examples:

- Graves' disease
- Myasthenia gravis

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