



# Hypersensitivity Reactions *ripped shamelessly from a website which can no longer be recalled*

## Type I - immediate hypersensitivity

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 (within seconds to minutes) reaction**.

Some examples:

- Allergic asthma
- Allergic conjunctivitis
- Allergic rhinitis ("hay fever")
- Anaphylaxis
- Angioedema
- Urticaria (hives)

## 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. 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 examples:

- Autoimmune haemolytic anaemia
- Goodpasture's syndrome
- Pemphigus
- Pernicious anemia
- 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

This 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 the 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