

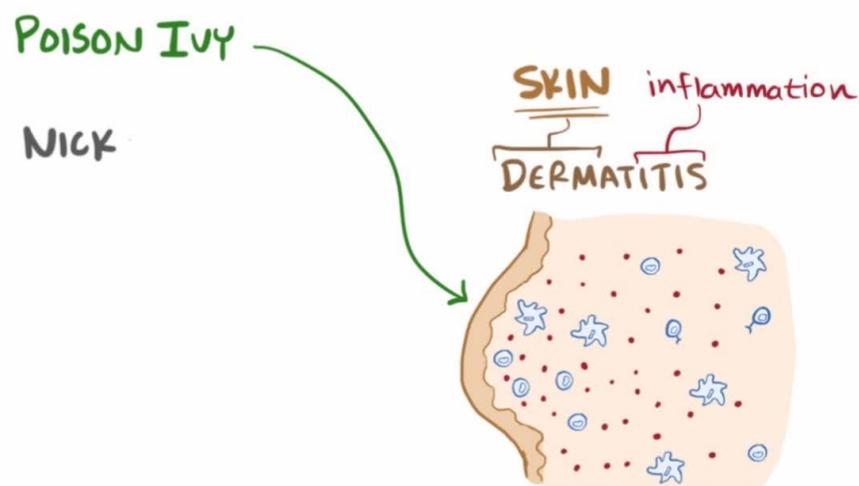
# Immunopathological reaction IV. type

A **late-type immunopathological reaction** (ie, a type IV reaction, late type, cellular) occurs 12-48 hours after exposure to allergen. There are no free Ig in the serum, the reaction is mediated by T-lymphocytes.

## Cellular immunopathological reaction (tuberculin type)

*Delayed type hypersensitivity is also sometimes referred to as DTH (delayed type hypersensitivity).*

The local reaction is caused by inflammation response dependent on TH1-lymphocytes, monocytes and macrophages. **Experimentally**, the animal is immunized intradermally with antigen in a suitable adjuvant. This promotes the increase of TH1-lymphocytes. After several weeks, the antigen is again injected intradermally and within 24 to 72 hours a characteristic local reaction occurs at the injection site. The time delay is caused by the fact that TH1-lymphocytes and macrophages, which stimulate each other, must first migrate to the injection site. A hard swelling (induration) occurs. Under physiological circumstances, this reaction is directed against intracellular parasites. Allergens are bacteria, fungi, viruses and their products. The main changes occur in the corium, most often manifesting as a papule. **During long-term stimulation**, macrophages can change into multinucleated syncytia, so-called *giant cells*. Dermatological manifestations are "id" reactions - mycides, microbides, bacterids, tuberculids. **DTH** is the essence of the tuberculin reaction, which we detect the state of immunity against TB. *Delayed hypersensitivity mechanisms are responsible for tissue damage during TB and leprosy.* Granuloma often occurs, and in extreme cases, **caseous necrosis** (see necrosis). Some autoantigens cause this type of reaction in sarcoidosis or granulomatous vasculitides. Infiltration of TH1-lymphocytes with production of IFN- $\gamma$  is characteristic of **demyelinating autoimmune disease**.



## Cellular cytotoxic reaction (eczema, epidermal, contact type)

Reaction similar to **DT**, but TH1-lymphocytes activate other effector components, especially CD8+ T-lymphocytes. Infected or altered cells are lysed by cytotoxic T-lymphocytes. **It occurs in** viral exanthems and tissue damage caused by some **viruses**, in hepatitis the main liver damage is caused by the immunopathological action of TC- and TH1 - lymphocytes. They **attack and destroy infected hepatocytes**. They are also used in "**acute rejection**" of a transplanted organ, in some forms of "**autoimmune thyroiditis**". The same mechanisms are responsible for contact dermatitis **induced by certain chemicals (nickel, chromium, cosmetic product ingredients and others)**. The dermatological manifestation has the character of eczema with spongiosis in the dermis and lymphocytic infiltrates.

## Foreign body reaction

The reaction is similar to DTH, but occurs to a non-antigenic foreign material. Hydrophilic materials show better biocompatibility:

- contact lenses;
- joint replacements;

- breast implants;
- sewing material;
- dialysis membranes;
- vascular replacements...

**Depends on biocompatibility. Proteins from the blood plasma are adsorbed** on them. Adsorbed proteins are recognized by receptors of monocytes, macrophages, and platelets. Adhered monocytes become activated, express some receptors. Macrophages create **syncytia**, producing IL-1, TNF. These effects create local and systemic reactions.

In **silicosis, asbestosis, beryliosis** inhaled particles are deposited in the lungs, stimulating alveolar macrophages. Granulomas are formed, fibroblasts are stimulated, leading to pulmonary fibrosis. In the case of asbestosis, **carcinogenic effect of asbestos** is added.

## Links

### Related Articles

- Allergy
- Immunopathological reaction type I
- Immunopathological reaction II. type
- Immunopathological reaction III. type

### External links

- Immunopathological reaction IV. type - Youtube video (<https://www.youtube.com/watch?v=C3E5COZ1XC8>)

### References

- HOŘEJŠÍ, Václav – BARTŮŇKOVÁ, Jiřina. *Základy imunologie*. 3. edition. Triton, 2008. 280 pp. ISBN 80-7254-686-4.