Specific immunity

The phylogenetically newer part of the immune system (present only in vertebrates) is represented by **specific immunity**. Only the basics are found in an individual's genome. During development and differentiation there are changes in the individual cells' genome which are then reflected in their phenotype. Specific immunity physiologically develops only after birth. It doesn't function on its own, but always cooperates with natural immunity.

Most specific immunity disorders have very serious consequences (e.g. AIDS).

The basic characteristics of specific immunity are:

- it consists of two parts: **cellular** and **humoral**;
- **antigen specificity**;
- activation after meeting a particular antigen;
- **slower onset** than non-specific mechanisms;
- different course during a repeated encounter;
- the ability to **remember**.

Components of specific immunity

Cellular component

The **cellular component** of specific immunity is made up of T-lymphocytes, B-lymphocytes, and plasmatic cells. These originate in the bone marrow from a **lymphoid progenitor**.

T-lymphocytes

They are transported to the thymus (thymocytes) where they divide and where their specificity is designated. Cells reacting to the body's own antigens or with non-functioning recognition mechanisms are destroyed. Only around 5% survive and are transported via blood to secondary lymphatic organs. Here they encounter "their own" antigen and are activated. After the immune response fades, **memory T-lymphocytes** remain.

B-lymphocytes

Their specificity is determined in the bone marrow. From there they are released into the bloodstream and populate the secondary lymphatic organs. They are activated predominantly by helper T_H-lymphocytes. After activation they multiply and a part of them transforms into **memory B-lymphocytes**. Most of them mature into **plasmatic cells** which produce antibodies and relocate back to the bone marrow.

🔎 *For more information see T-lymphocytes, B-lymphocytes.*

Humoral component

The humoral component consists mostly of antibodies and cytokines.

Antibodies

Glycoproteins that are in the serum. They play a very important role in the induction of certain events such as opsonisation and phagocytosis. They cooperate with natural immunity cells. They help them **find and determine** the target of destruction.

🔎 *For more information see Antibodies.*

Cytokines

A broad range of signal peptides some of which have hormonal effects. They serve not only in the communication of leukocytes but bone marrow cells, endothelial cells, and other cells.

🔎 *For more information see Cytokines.*

Antigen specificity

On the basis of random regrouping of the genome and deletion of its parts, an enormous diversity of the **variable parts** of binding places for the antigen epitope arises. The changes in genetic information are irreversible and permanent, therefore cells created by the proliferation of the same lymphocyte will have the same specificity (they inherit it). These changes are fundamental on the variable domains **TcR**, **BcR**, and **antibodies**.

🔎 *For more information see Genetics of Ig, B, and T receptors.*

Ideally, for each amino acid sequence of a variable domain, only one epitope should exist. **In reality**, one epitope can fit multiple sequences. However, those can bind to it with different **affinity**. With antibodies one can also mention **avidity**, or, the overall capability of one particular antibody to bind and hold an antigen.

With B-lymphocytes there can additionally be small changes in the structure of BcR and antibodies, and thus the maturation of antibodies when during the reaction the affinity of antibodies to the antigen **increases**.

Activation of specific immunity

Cells of specific immunity have achieved their specificity in the **primary lymphatic organs**. In the secondary lymphatic organs they are, in connection with MHC, presented with antigens. If a cell is specific to this antigen and has enough **co-stimulating signals** (ligands, cytokines…), it is activated and starts producing the humoral components. They then lead to increased proliferation, antibody production, or increase in cytotoxicity.

In the case of recognising an antigen but insufficient co-stimulation, the following three situations can happen:

- non-reactivity - the cell recognises, but doesn't react;
- tolerization - the cell recognises, and then **never activates** in response to the stimulus;
- disposal - the cell recognises, and initiates apoptosis.

All four types of activation are very important in autoimmune disorders and allergic reactions.

Immunological memory and dynamics of response

The difference between the first and next encounter is used in active immunisation, and conversely can be the cause of some hypersensitivities.

File:Dynamika protilatek.png
Dynamics of antibodies

Immunological memory

Immunological memory may be much simpler than the mechanisms of neuronal memory, it is, however, much more long-lasting (even lifelong). It is based on the existence of so-called **memory cells**. These are "veterans" who have been proven to be able to destroy an infectious agent. They are daughter cells created by the proliferation of T- and B-lymphocytes which had reacted to a pathogen and proliferation signals (IL-2).

Primary response

The first encounter with an antigen (e.g. bacteria) is followed by a **slowly-developing** response. Specific T- and B-lymphocytes are activated and antibodies, firstly IgM and later IgG, start being produced. After the first encounter ends, the antibodies remain within plasma and memory cells in the secondary lymphatic organs.

Secondary response

The second and each successive encounter happens much faster. The organism **remembers** the last encounter. Memory cells specific for a given antigen are diffused all over the body and there are many more of them. This is why the answer to an antigenic stimulus is **faster** and much stronger. This stops pathogens from multiplying and infection from developing. The secondary response can happen **without any clinical signs** of an illness that had caused the activation (the disease doesn't get the chance to develop and is gotten rid of).

Links

Related articles

- Antibody
- B-lymphocytes
- T-lymphocytes
- Non-specific immunity
- Immune system
- Active immunisation
- Passive immunisation

Bibliography

- HOŘEJŠÍ, Václav – BARTŮŇKOVÁ, Jiřina. *Základy imunologie.* 3. edition. Triton, 2008. ISBN 80-7254-686-4.
- ŠTERZL, Ivan. *Základy imunologie pro zubní a všeobecné lékaře.* 1. edition. Karolinum, 2005. ISBN 978-80-246-0972-0.