

# Immune system and cancer

This article has been translated from WikiSkripta; ready for the **editor's review**.

**Transplant-type immunity mediated by T-lymphocytes and cytokines** produced by them (positive influence of IL-2, interferons and TNF $\alpha$  and  $\beta$ ) can be used in anti-tumor immunity . **Furthermore, NK cells** and **LAK cells** (lymphokine activated killers) are involved in the antitumor reaction . NK cells have a toxic effect on tumor cells without prior sensitization. They may be the first natural defense system in the early stages of tumor growth. The tumor tissue is infiltrated by a functionally modified T-ly population, the so-called **tumor infiltrating lymphocytes (TIL)** . These are able, after isolation, in vitro multiplication and after introduction into the organism by infusion, to specifically eliminate tumor cells with a cytotoxic effect.

Differences in the phenotype of normal and tumor cells in the expression of not only membrane antigens are called *changes in the antigenic makeup of tumor cells* .

## Changes in the antigenic makeup of tumor cells

### Qualitative changes

#### Neoantigens

The formation of **neoantigens** is conditioned by the mutation of other, physiological antigens. These are recognized as **foreign** and an immune response is initiated against them and the cells that carry them. New antigens may be specific for individual tumor types. They are then called **tumor-specific transplantation antigens (TSTA)** . Transplant-type neoantigens have the ability to induce an immunological response leading to the elimination of transformed cells at the beginning of the malignant process. **Escape** of transformed cells from immune control mechanisms can be caused by selection in the tumor cell population or by immunosuppression of the individual. Immunological activity depends on the age and condition of the individual. Chemical carcinogens can also induce immunosuppression, physical and chemical factors. The expression of TSTA depends on the **etiology of cell transformation** , it is variable. The expression of TSTA in tumors induced by both **chemical** carcinogens and **radiation** depends on the latency period (from the encounter with the carcinogen to the onset of tumor growth). Tumors of **viral** etiology caused by the same virus carry the same TSTA. Such TSTAs can lead to the immunization of the organism. The immune system is then able to eliminate the transformed cells . Cytotoxic T-lymphocytes play a major role in healing.

An example of the regulation of tumor growth by the immune system is Burkitt's lymphoma - tumor-transformed B-lymphocytes are removed by immunological mechanisms with the decisive participation of T-ly. These recognize on their surface **virus-induced TSTA** presented by HLA class I molecules . In the absence of T-cells or when their activity is suppressed, rapid development of tumor growth will occur.

The immune response directed against TSTA is important in the spontaneous destruction of tumor cells at the beginning of the malignant process.

- **Prevention** : vaccination in endemic areas,
- **therapy** : immunization against neoantigens.

### Quantitative changes

The expression of antigens that normally occur on healthy cells can be **increased** or **reduced to zero** in tumor cells . It can also be antigens that are not present in healthy, fully differentiated cells. These are often antigens normally present in embryogenesis . Quantitatively altered expression of antigens in tumor cells can be an important diagnostic marker. These antigenic markers are either bound on the cell surface (MHC class I and II antigens) or are secreted from tumor cells into the bloodstream. **Determination of the level of carcinoembryonic antigen (CEA) and alpha-fetoprotein (AFP)** in the serum is used to refine the diagnosis and check the appropriateness of cancer treatment .

- **Elevated CEA values** are characteristic of GIT tumors. However, it is not only a specific marker of tumor growth. An increased level was also noted in other non-neoplastic GIT disease processes. In normal cells, its expression is limited by organ and time. In cats, it is found in the tissue of the intestine, pancreas and liver. In adults, it is found in low amounts in the intestinal mucosa, lungs and lactating mammary gland.
- **Alpha-fetoprotein** is present in fetal liver, fetal serum, and low amounts are also found in the serum of healthy adults. In adults, elevated serum levels are often associated with hepatomas or testicular teratoma.
- **MHC antigens** are also used in monitoring the course of certain cancer diseases and the effectiveness of treatment. **Reduced expression of class I antigens** is correlated with aggressiveness and invasiveness of stomach, ovary, intestine, kidney, breast, and pancreatic tumors. Expression of class I antigens in tumor tissue is determined using  *$\beta$ -2-microglobulin* . **Determination of antigen expression II.** MHC class II is important for the prognosis, especially in leukemias , when their absence in the membrane of leukemic cells means a worse prognosis.

Other antigenic determinants used for diagnosis, prognosis and choice of treatment procedures are **differentiation antigens** ( membrane CD signs ). With their help, the diagnosis of morphologically indistinguishable cancer diseases, especially hematological malignancies and lymphomas, can be made more precise, and treatment procedures can then be chosen.

## Links

## References

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## References

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