

Immune system and cancer illnesses

In antitumor immunity, transplant-type immunity mediated by **T-lymphocytes** and the **cytokines** produced by them may be involved. (positive effect of IL-2, interferons and $\text{TNF}\alpha$ & β). In addition, **NK cells** and **LAK cells** are involved in the anti-tumor reaction (lymphokine-activated killers). 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. Tumor tissue is infiltrated by a functionally modified population of T cells, so-called **tumor infiltrating lymphocytes (TILs)**. These are able, after isolation, in vitro multiplication and after introduction into the organism by infusion, to specifically kill tumor cells by 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 equipment of tumor cells*.

Changes in the antigenic equipment of tumor cells

Qualitative changes

Neoantigens

The formation of **neoantigens** is conditioned by mutations in other, physiological antigens. These are recognized as **strange** and an immune response is initiated against them and the cells that carry them. The new antigens may be specific for each type of tumor. They are then called **tumor-specific transplant antigens (TSTA)**. Transplant-type neo-antigens have the ability to elicit an immune response leading to the destruction of transformed cells at the beginning of the malignant process. **Leakage** of transformed cells by immune control mechanisms may be due to selection in a population of tumor cells or immunosuppression of the individual. Immunological activity depends on the age and condition of the individual. Immunosuppression can also be induced by chemical carcinogens, physical and chemical factors. TSTA expression 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 time (from encounter with the carcinogen to the onset of tumor growth). Tumors of **viral** etiology caused by the same virus carry identical TSTA. Such TSTAs can lead to immunization of the organism. The immune system is then able to **kill the transformed cells**. Cytotoxic T-lymphocytes play a major role in rejection.

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 crucial involvement of T-lymphocytes. On their surface, they recognize **virus-induced TSTA** presented by HLA class I molecules. In the absence of T-cells or in the suppression of their activity, tumor growth develops rapidly.

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 **decreased to zero** in tumors. They may also be antigens that do not occur in healthy, fully differentiated cells. These are often antigens normally present in embryogenesis. Quantitatively altered antigen expression in tumor cells may be an important diagnostic marker. These antigenic markers are either bound to the cell surface (MHC class I and II antigens) or are secreted from tumor cells into the bloodstream. Determination of serum **carcinoembryonic antigen (CEA)** and **alpha-fetoprotein (AFP)** levels is used to refine the diagnosis and control the suitability of cancer treatment.

- **Elevated CEA levels** are characteristic of GIT tumors. However, it is not just a specific marker of tumor growth. Elevated levels have also been reported in other non-cancerous GIT disease processes. In normal cells, its expression is organ-limited and time-limited. In fetuses, 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 glands.
- **Alpha-fetoprotein** is present in the fetal liver, fetal serum, and low levels are found in the serum of healthy adults. In adults, elevated serum levels are often associated with hepatomas or testicular teratomas.
- **MHC antigens** are also used to monitor the course of certain cancers and the effectiveness of treatment. **Decreased expression of class I antigens** is correlated with the aggressiveness and invasiveness of gastric, ovarian, intestinal, renal, breast and pancreatic tumors. The expression of class I antigens in tumor tissue is determined using *β -2-microglobulin*. **Determination of antigen expression MHC II. class** is important for prognosis, especially in leukemias, where their absence in the membrane of leukemic cells means a worse prognosis.

Other antigenic determinants used for diagnosis, prognosis and choice of treatments are **differentiation antigens** (membrane CD markers). They can be used to refine the diagnosis of morphologically indistinguishable cancers, especially hematological malignancies and lymphomas, and subsequently to choose treatment procedures.

Links

References

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References

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