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Overcoming Drug Resistance in Cancer: Strategies and Challenges

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Abstract

Drug resistance is a major challenge in the treatment of cancer. It refers to the ability of cancer cells to survive and continue growing despite exposure to anti-cancer drugs. This uncontrolled growth can result in the formation of a lump or mass called a tumor. These are non-cancerous growths that do not invade nearby tissues or spread to other parts of the body. They tend to grow slowly and are usually not life-threatening. In some cases, cancer cells are inherently resistant to certain drugs. This is known as intrinsic or primary resistance. The genetic makeup of these cells may make them less susceptible to the drug's mechanism of action.

Keywords: Tumor cells • Cancer • EMT

Introduction

The EMT process can contribute to drug resistance. During EMT, cancer cells become more invasive and less responsive to treatment. They can develop characteristics associated with stem cells, making them harder to target. Treatment options for cancer may include surgery, chemotherapy, radiation therapy, immunotherapy, targeted therapy, and hormone therapy, depending on the type and stage of cancer. Early detection and diagnosis of cancer are crucial for effective treatment and improved outcomes. Regular screenings, such as mammograms and colonoscopies, along with lifestyle modifications like a healthy diet and avoiding tobacco and excessive alcohol use, can help reduce the risk of developing cancer and increase the chances of successful treatment if it does occur. In early studies on energy metabolism of tumor cells, it was proposed that the enhanced glycolysis was induced by a decreased oxidative phosphorylation. Since then it has been indiscriminately applied to all types of tumor cells that the ATP supply is mainly or only provided by glycolysis, without an appropriate experimental evaluation. In this review, the different genetic and biochemical mechanisms by which tumor cells achieve an enhanced glycolytic flux are analysed [1-3].

Literature Review

Tumors are often heterogeneous, meaning they consist of a variety of cell types with different genetic profiles. Some subpopulations of cells within a tumor may be resistant to treatment, leading to recurrence. The tumor microenvironment, including factors like hypoxia and the presence of immune cells, can contribute to drug resistance. These factors can create a protective niche for cancer cells. The passage discusses how drugs targeting glycolysis and mitochondrial function can impact tumor cell metabolism and cellular proliferation. This suggests that altering the metabolic pathways of tumor cells could be a potential strategy for cancer therapy. The energy metabolism may serve as an alternative therapeutic target for both glycolytic (hypoxic) and oxidative tumors. In other words, modulating the way tumor cells generate energy could be a viable strategy to modify their growth and response to treatment. Some cancer cells can enhance their DNA repair mechanisms, allowing them to quickly repair the DNA damage caused by chemotherapy and radiation therapy [4].

Tumor microenvironment can inhibit the immune system's ability

The TME is a complex and dynamic environment that surrounds and interacts with cancer cells within a tumor. It includes various cell types, extracellular matrix components, signalling molecules, and physical conditions. The TME can be infiltrated by immunosuppressive cell types, such as regulatory T cells (Tregs), Myeloid-Derived Suppressor Cells (MDSCs), and M2-polarized macrophages. These cells create an immunosuppressive environment by inhibiting the activity of cytotoxic immune cells like T cells and Natural Killer (NK) cells. The TME can produce various signalling molecules, including cytokines and chemokine's that promote an immunosuppressive environment. For example, the release of interleukin-10 (IL-10) and Transforming Growth Factor-beta (TGF-β can inhibit the function of immune cells. Tumor cells and stromal cells within the TME can undergo metabolic reprogramming that leads to nutrient depletion and the accumulation of metabolic by-products that inhibit immune cell function. Tumors often have regions with low oxygen levels (hypoxia), which can promote the expression of immunosuppressive factors and make immune cells less effective [5,6].

Discussion

Tumor cells are characterized by having an elevated capacity for glycolysis, a metabolic pathway that converts glucose into energy even in the presence of abundant oxygen. This metabolic feature is a hallmark of many cancer cells. Accordingly, the dynamic focusing of growth cell contacts in the advancement of new and improvement of existing ways to deal with the treatment of strong cancers is one of the direst areas of current biomedicine. In tumor cells can be significantly higher than in normal, non-cancerous cells. For example, in rat hematomas, the glycolytic flux is reported to be 2 to 17 times higher than that of normal hepatocytes. It is suggested that this increase in glycolytic activity serves as a metabolic strategy for tumor cells. This strategy allows cancer cells to generate energy efficiently in environments with low oxygen concentrations, which are common within solid tumors. Understanding the increased glycolytic capacity in cancer cells, often referred to as the Warburg effect, is important in the context of cancer biology and therapy. This altered metabolism not only supports the high energy demands of rapidly dividing cancer cells but also leads to the accumulation of metabolic by-products, such as lactate, which can create a hostile microenvironment that promotes tumor progression.

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Conclusion

Understanding the growth rate of tumors is important in research because it can provide insights into tumor biology, behaviour, and response to treatment. Fast-growing tumors are often more aggressive and may require different therapeutic approaches compared to slower-growing tumors. Researchers use these classifications to study the characteristics and behaviour of tumors in experimental settings, which can inform our understanding of cancer and the development of potential therapies. The complex relationship between energy metabolism and cancer, suggesting that targeting metabolic pathways in tumor cells may have therapeutic potential, especially in cases where oxidative phosphorylation remains active. This concept is an active area of research in the field of oncology and may lead to new strategies for cancer treatment in the future.

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Conflict of Interest

None.

References

1. Paduch, Roman. "The role of lymphangiogenesis and angiogenesis in tumor metastasis." *Cell Oncol* 39 (2016): 397-410.
2. Kaneno, Ramon, Galina V. Shurin, Felipe M. Kaneno and Hiam Naiditch, et al. "Chemotherapeutic agents in low noncytotoxic concentrations increase immunogenicity of human colon cancer cells." *Cell Oncol* 34 (2011): 97-106.
3. Jotzu, Constantin, Eckhard Alt, Gabriel Welte and Jie Li, et al. "Adipose tissue derived stem cells differentiate into carcinoma-associated fibroblast-like cells under the influence of tumor derived factors." *Cell Oncol* 34 (2011): 55-67.
4. Sharma, Rohit, Rohini Sharma, Tejinder Pal Khaket and Chanchala Dutta, et al. "Breast cancer metastasis: Putative therapeutic role of vascular cell adhesion molecule-1." *Cell Oncol* 40 (2017): 199-208.
5. Di, Jiabo, Tjitske Duiveman-de Boer, Petra LM Zusterzeel and Carl G. Figdor, et al. "The stem cell markers Oct4A, Nanog and c-Myc are expressed in ascites cells and tumor tissue of ovarian cancer patients." *Cell Oncol* 36 (2013): 363-374.
6. Yousefi, Meysam, Rahim Nosrati, Arash Salmaninejad and Sadegh Dehghani, et al. "Organ-specific metastasis of breast cancer: Molecular and cellular mechanisms underlying lung metastasis." *Cell Oncol* 41 (2018): 123-140.

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