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Targeting the Tumor Microenvironment in Cellular Oncology: Therapeutic Opportunities

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Introduction

The Tumor Microenvironment (TME) is the cellular and non-cellular environment that surrounds and interacts with a tumor. It plays a critical role in tumor development, growth, invasion, and response to therapy. The cancer cells themselves are a major component of the TME. These cells proliferate and evade normal growth control mechanisms, leading to tumor formation. These are non-cancerous cells that provide structural and functional support to the tumor. Examples include Cancer-Associated Fibroblasts (CAFs), immune cells, endothelial cells, and mesenchymal stem cells. Stromal cells can influence tumor behaviour through cell signaling and the secretion of growth factors. The TME contains immune cells, including T cells, B cells, Natural Killer (NK) cells, and macrophages. These cells can either promote or inhibit tumor growth, depending on their type and activation status. Immune checkpoints and immunosuppressive factors in the TME can hinder the immune system's ability to recognize and attack cancer cells [1].

Description

The ECM is a network of proteins and carbohydrates that provides structural support to tissues. In the TME, the ECM can become remodeled and contribute to tumor invasion and metastasis. Tumors need a blood supply to obtain oxygen and nutrients. Angiogenesis, the formation of new blood vessels, is a critical process in the TME that facilitates tumor growth. Lymphatic vessels drain excess fluid and immune cells from tissues. They can also facilitate the spread of cancer cells to nearby lymph nodes and distant sites. Many tumors have areas with low oxygen levels (hypoxia) due to inefficient blood vessel formation. Hypoxia can drive tumor progression and resistance to therapy. Changes in metabolism within the TME can affect tumor growth and response to treatment. Cancer cells often rely on altered metabolic pathways to meet their energy and nutrient needs. These small membrane-bound vesicles, including exosomes, can transport molecules like proteins, RNA, and DNA between cells in the TME, influencing tumor progression and immune responses. The blood vessels in the TME may have abnormalities, such as leakiness, that affect drug delivery to the tumor [2].

Hepatocellular Carcinoma (HCC) is the seventh most common malignancy and the third leading cause of cancer-related death worldwide. Despite the recent advances in diagnosis and treatment of HCC, it remains a highly lethal disease. The main cause of death in HCC patients is tumor progression with metastasis. However, the underlying mechanisms of tumor initiation, progression and metastasis are still not fully understood. Hepatocellular Carcinoma (HCC) is the most common type of liver cancer. The majority of HCC patients have an underlying chronic liver disease, which is a significant risk factor for developing HCC. Liver cirrhosis, characterized by fibrosis and scarring of the liver tissue, is a primary risk factor for the development of HCC. Chronic liver injury, often caused by factors like viral hepatitis, alcohol abuse, or Non-Alcoholic Fatty Liver Disease (NAFLD), can lead to the dysregulated growth of hepatocytes (liver cells). This dysregulated growth results in the formation of regenerative nodules, dysplastic nodules, and eventually, HCC. Cirrhotic Liver-Derived Hepatocytes (CLDHs) are hepatocytes from cirrhotic liver tissue. CLDHs exhibit changes in cellular signaling pathways, specifically transitioning from a MAPK (Mitogen-Activated Protein Kinase)-independent cell survival pathway to a MAPK-dependent cell survival pathway. This transition in signaling pathways may contribute to the survival and proliferation of CLDHs, which can be a precursor to HCC development.

CLDHs show increased expression of venetian and type 1 collagen, which are markers typically associated with mesenchymal cells. EMT is a biological process in which epithelial cells (such as hepatocytes) lose their characteristic features and acquire properties similar to mesenchymal cells. EMT plays essential roles in embryonic development, tissue repair, and cancer progression. In the context of liver disease, EMT may be a critical connection point between inflammation, fibrotic diseases (like cirrhosis), and the development of HCC. During EMT, cells become more motile and invasive, which is often observed in cancer cells as they transition to a more aggressive phenotype. The passage underscores the significance of understanding the molecular and cellular changes that occur in the liver microenvironment during chronic liver disease and cirrhosis, as these changes can contribute to the development of HCC. The transition of hepatocytes to a more mesenchymal-like state through EMT highlights one of the mechanisms by which liver cells become more aggressive and prone to forming cancerous tumors. This knowledge is vital for developing targeted therapies and interventions to prevent or treat HCC in patients with chronic liver disease and cirrhosis [3-5].

Conclusion

The interactions and dynamics within the TME are highly intricate and can vary depending on the type of cancer, its stage, and the genetic mutations present. Understanding the TME is crucial for developing effective cancer therapies. Targeting specific components of the TME, such as immune checkpoint inhibitors or anti-angiogenic therapies, has become an essential strategy in cancer treatment. Additionally, biomarkers within the TME can be used for prognosis and prediction of treatment response. Research in this field continues to uncover new insights into the complex relationship between tumors and their microenvironment.

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Conflict of Interest

None.

References

1. Whiteside, T. L. "The tumor microenvironment and its role in promoting tumor growth." *Oncogene* 27 (2008): 5904-5912.

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2. Bejarano, Leire, Marta JC Jordão and Johanna A. Joyce. "Therapeutic targeting of the tumor microenvironment." *Cancer Discov* 11 (2021): 933-959.

3. Zhou, Jingyi, Weiyu Wang and Qi Li. "Potential therapeutic targets in the tumor microenvironment of hepatocellular carcinoma: Reversing the protumor effect of tumor-associated macrophages." *J Exp Clin Cancer Res* 40 (2021): 1-12.

4. Sheik, Aliya, Marzieh Ramezani Farani, Eunsu Kim and Suheon Kim, et al. "Therapeutic targeting of the tumor microenvironments with cannabinoids and their analogs: Update on clinical trials." *Environ Res* 231 (2023): 115862.

5. Yang, Ju Dong, Ikuo Nakamura and Lewis R. Roberts. "The tumor microenvironment in hepatocellular carcinoma: Current status and therapeutic targets." *Semin Cancer Biol* 21 (2011):35-43.

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