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Perspective

Glycan-Protein Interactions in Cancer Metastasis: A Glycobiology Overview

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DESCRIPTION

Cancer metastasis, the process by which cancer cells spread from their primary site to distant organs, is a leading cause of cancer-related morbidity and mortality. Despite significant advances in cancer research, metastasis remains one of the most challenging aspects of cancer treatment. Recent insights into the role of glycans the sugar molecules attached to proteins and lipids in cancer progression have opened new avenues for understanding and targeting metastasis. Glycans play critical roles in a variety of cellular processes, including cell adhesion, migration, and immune evasion, all of which are integral to the metastatic process. In this article, we will explore the role of glycan-protein interactions in cancer metastasis and how targeting these interactions could provide new therapeutic strategies.

Glycans and cancer metastasis

Metastasis is a multi-step process that involves the detachment of tumor cells from the primary tumor, invasion into surrounding tissues, entry into the bloodstream or lymphatic system, extravasation into distant organs, and colonization of these new sites. Each of these steps is influenced by glycosylation, the covalent attachment of sugar chains to proteins and lipids on the cell surface. Alterations in glycosylation can significantly impact the ability of cancer cells to interact with their environment, facilitating the metastatic cascade. Glycosylation changes in cancer cells often result in the production of abnormal glycan structures that are not typically present in normal cells. These changes can enhance the aggressiveness of the cancer by affecting critical cellular functions such as adhesion, signaling, and motility. Aberrant glycosylation is now recognized as a hallmark of cancer, with altered glycan profiles contributing to tumor progression, immune escape, and metastatic potential.

Glycan-protein interactions in metastasis

At the core of glycosylation's involvement in metastasis are the glycan-protein interactions that mediate important cellular behaviors. The key processes affected by these interactions include:

Cell adhesion and migration: One of the first steps in metastasis is the detachment of cancer cells from the primary tumor. The glycosylation of cell surface proteins, particularly adhesion molecules such as integrins, cadherins, and selectins, regulates the ability of cancer cells to adhere to Extracellular Matrix (ECM) components and neighboring cells. For example, fucosylation (the addition of fucose, a sugar molecule) of integrins has been shown to enhance their ability to interact with the ECM, promoting cell migration and invasion essential steps in metastasis.

Immune evasion: Glycans also play a key role in enabling cancer cells to evade detection by the immune system. Many cancers exhibit immune checkpoint modulation, which allows them to suppress immune responses. Alterations in glycosylation affect the ability of immune cells to recognize and attack cancer cells. For example, sialic acid, a sugar often found on the surface of tumor cells, can inhibit immune cell activation by interacting with Sialic acid-binding immunoglobulin-like lectins (Siglecs) on immune cells. This interaction suppresses the immune response, allowing cancer cells to evade immune surveillance and promoting metastasis.

Cancer stem cell behavior: Cancer Stem Cells (CSCs) are a subpopulation of cells within tumors that have the ability to self-renew and drive metastasis. Recent studies have highlighted the role of glycosylation in regulating CSC properties, such as their ability to migrate and invade. Alterations in the glycosylation of glycoproteins on CSCs have been shown to affect their interactions with the tumor microenvironment and the immune system, further contributing to the metastatic potential of the tumor.

Angiogenesis: The development of new blood vessels, or angiogenesis, is essential for the growth and metastasis of tumors. Glycans play a critical role in angiogenesis by modulating the activity of key signaling molecules involved in blood vessel formation. For example, the glycosylation of Vascular Endothelial Growth Factor (VEGF), a potent angiogenic factor, can influence its binding to VEGF receptors on endothelial cells. Altered glycosylation of VEGF can enhance

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its ability to promote angiogenesis, providing cancer cells with a blood supply needed for metastatic growth.

CONCLUSION

Glycan-protein interactions play a fundamental role in the metastatic process, influencing key steps such as cell adhesion, migration, immune evasion, and angiogenesis. Altered glycosylation patterns on cancer cells contribute to their metastatic potential, and targeting these glycan-mediated interactions offers a promising strategy for developing new cancer therapies. By interfering with the enzymes responsible for glycosylation, inhibiting glycan-binding proteins, or designing glycan-based drugs, researchers are uncovering novel therapeutic approaches to block metastasis and improve cancer outcomes. As our understanding of glycobiology deepens, glycan-targeted therapies hold the potential to revolutionize cancer treatment and provide more effective, personalized options for patients with metastatic disease.