Mini Review
Volume 8:3, 2023

Journal of Oncology Medicine & Practice

ISSN: 2576-3857 Open Access

Angiogenesis, the Tumour Microenvironment and Proteoglycans in Cancer Biology

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Abstract

Angiogenesis, the tumour microenvironment, and proteoglycans play pivotal roles in cancer biology. Angiogenesis, the formation of new blood vessels, is a critical process for tumour growth and metastasis. The tumor microenvironment, consisting of various cellular and non-cellular components, provides a supportive niche for cancer progression. Proteoglycans, a class of complex molecules, are involved in modulating both angiogenesis and the tumour microenvironment. Angiogenesis in cancer is driven by a delicate balance of pro-angiogenic and anti-angiogenic factors. Tumour cells release pro-antigenic factors, such as Vascular Endothelial Growth Factors (VEGFs), to stimulate the sprouting of new blood vessels from existing vasculature. The tumour microenvironment, including immune cells, fibroblasts, and extracellular matrix components, also contributes to angiogenesis through the secretion of angiogenic factors and remodelling of the surrounding vasculature. Disrupting angiogenesis has emerged as a promising therapeutic strategy in cancer treatment, with the development of anti-antigenic drugs targeting VEGF signalling.

Keywords: Microenvironment • Cancer • Angiogenesis

Introduction

The tumour microenvironment encompasses a complex network of interactions between cancer cells and their surroundings. Stromal cells, immune cells, and extracellular matrix components create a dynamic milieu that promotes tumour growth, invasion, and evasion of immune surveillance. The tumour microenvironment can influence angiogenesis, immune responses, and drug sensitivity, impacting cancer progression and treatment outcomes. Understanding the intricate crosstalk between cancer cells and their microenvironment is essential for developing effective therapeutic interventions. Proteoglycans, abundant components of the extracellular matrix, have multifaceted roles in cancer biology. They modulate cell signalling pathways, interact with growth factors, and regulate the physical properties of the tumour microenvironment. Proteoglycans, such as heparin sulphate proteoglycans, influence angiogenesis by sequestering or presenting angiogenic factors to their receptors. Alterations in proteoglycan expression and structure can promote tumour growth, invasion, and metastasis. Targeting proteoglycans and their associated signalling pathways has emerged as a promising strategy for cancer therapy.

Literature Review

Due to their polyhedric nature and their capacity to interact with both ligands and receptors that regulate neoplastic growth and neovascularization, proteoglycans, which are key molecular effectors of the cell surface and pericellular microenvironments, perform multiple functions in cancer and angiogenesis. Some proteoglycans, for example, perlecan, have favorable to and hostile to angiogenic exercises, though other proteoglycans, for example, syndecans and glypicans, can likewise straightforwardly influence disease development by regulating key flagging pathways. The bioactivity of these proteoglycans is additionally adjusted by a few classes of chemicals inside the growth microenvironment: sheddases, which cleave syndecans and glypicans that are cell-associated or transmembrane, various proteinases, which cleave the protein core of pericellular proteoglycans, and heparanases and endosulfatases, which alter the structure and bioactivity of various heparan sulphate proteoglycans and the growth factors that are bound to them. Conversely, a portion of the little leucine-rich proteoglycans, for example, decorin and lumican, go about as cancer repressors by truly irritating receptor tyrosine kinases including the epidermal development factor and the Met receptors or integrin receptors in this manner bringing out enemy of endurance and favorable to apoptotic pathways. New proteoglycan functions modulating cancer progression [1,2].

Discussion

Over 50 years ago, it was discovered that proteoglycans are involved in the biology of cancer. Pathologists first noticed that some carcinomas cause the host stroma and connective tissue surrounding the cancer cells to experience a reaction similar to desmoplastic hyperproliferative. This reaction frequently stains metachromatically with cationic dyes like alcian blue, indicating that the desmoplastic reaction contains a lot of proteoglycans and thus glycosaminoglycans. Given the way that desmoplasia happens in growths with different histogenetic foundations, it appears to be conceivable to consider this unusual articulation of proteoglycans a kind of broad response to intrusion with agonistic and opposing components implanted in a similar stroma. The specific roles that proteoglycans play in cancer biology are becoming clearer as molecular and cellular biology advances. We now know, for instance, that some heparan sulphate proteoglycans act as pro-angiogenic factors by presenting various growth factors to their cognate receptors after binding to multiple growth factors. Other proteoglycan C-terminal fragments, on the other hand, stop the angiogenic process by stopping the activity of several integrins that are necessary for the movement of endothelial cells. Over the past two decades, structural and functional characterization of cell surface heparan sulphate proteoglycans has revealed how these molecules affect cell behavior broadly. Cancer progression has been regulated by members of the syndecan and glypican families, which may also serve as biomarkers for disease severity or early detection. Interestingly, new research suggests that, depending on the type and stage of cancer, cell surface heparan sulphate proteoglycans may play a variety of roles in cancer and act as either inhibitors or promoters of tumor progression [3,4].

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Received: 01 June 2023, Manuscript No. jomp-23-101574; **Editor assigned:** 03 June 2023, PreQC No. P-101574; **Reviewed:** 15 June 2023, QC No. Q-101574; **Revised:** 21 June 2023, Manuscript No. R-101574; **Published**: 28 June 2023, DOI: 10.37421/2576-3857.2023.08.196

What's more, the capability of these proteoglycans can be changed by two classes of chemicals inside the growth microenvironment, those that can deliver proteoglycans from the cell surface (sheddases) and those that can adjust the construction of heparan sulfate chains (heparanases and endosulfatases). As a result, the expression of these enzymes may be able to dynamically control the location and function of proteoglycans, as well as the behavior of tumor cells. The so-called Small Leucine-Rich Proteoglycans (SLRPs), which carry primarily dermatan or chondroitin sulfate, function as tumor repressors by inhibiting the activity of RTKs like Met and the Epidermal Growth Factor Receptor (EGFR). Additionally, some of the SLRPs alter innate immunity and adhesion through interactions with integrins and Toll receptors. In order to critically evaluate the roles that HSPGs and SLRPs play in cancer biology, the tumor microenvironment, and angiogenesis, we will discuss examples of both in this review. Perlecan has the potential to have both pro- and anti-angiogenic effects as a prototype basement membrane and cell-associated HSPG [5].

The idea of enigmatic pieces of huge protein centers with natural action that vary from those of the parent particle is presently being acknowledged, to the extent that other HSPGs, for example, collagen XVIII likewise contain comparable bioactivities inserted at their C-end. Depending on the structure, function, and localization of the proteoglycan, as well as the type and stage of the tumor, syndecans and glypicans have the ability to promote or inhibit the initiation and progression of cancer. They can also mediate a variety of functions within tumours. Because of their diversity, these proteoglycans have the potential to regulate tumor behavior in multiple layers. Cell surface proteoglycans may be viable targets for cancer therapy, despite not being discussed in this review. In view of their useful qualities, remedial targets might incorporate explicit areas of proteoglycan center proteins as well as heparan sulfate chains. Furthermore, the proteoglycan changing chemicals, especially the endosulfatases and heparanase, address alluring restorative targets. Proteoglycans may directly regulate the transcription of genes that control the behavior of tumours, according to reports that heparin sulphate in the nucleus can regulate gene transcriptional activity. There is still time to investigate this exciting possibility [6].

Conclusion

The SLRPs have emerged as potent signaling molecules that control a wide range of processes and have the potential to be targets themselves as well as novel therapeutic modalities against cancer. Understanding the molecular mechanisms by which these proteoglycans derived from stroma affect cell cycle, survival, and cancer growth may provide new insights into how tumour cells control their environments.

Acknowledgement

None.

Conflict of Interest

No potential conflict of interest was reported by the authors.

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How to cite this article: Ramakrishna, Seeram. “Angiogenesis, the Tumour Microenvironment and Proteoglycans in Cancer Biology.” *J Oncol Med & Pract* 8 (2023): 196.