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Prostaglandins Control Tumour Microenvironment

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Introduction

Prostaglandins, bioactive lipids produced by the cyclooxygenase metabolism of arachidonic acid, have potent effects on many constituents of tumour microenvironments. We will describe the formation and activities of prostaglandins in the context of the tumour microenvironment in this review. We will discuss how prostaglandins regulate cancer-associated fibroblasts and immune constituents, as well as their roles in immune escape during tumour progression. The review concludes with future prospects for improving immunotherapy efficacy by repurposing nonsteroidal anti-inflammatory drugs and other prostaglandin modulators [1].

Description

Prostaglandins are bioactive eicosanoids that play a variety of homeostatic biological functions as well as inflammation. They are formed during the metabolism of arachidonic acid by Cyclooxygenase (COX), which is followed by various isomerases. This -6-unsaturated 20-carbon fatty acid is covalently linked to the position of glycerophospholipids as a component of cellular membranes under normal conditions. Its release is tightly controlled by metabolic and physiologic processes. Arachidonic acid can be released from the plasma membrane during cellular responses to a variety of cytokines, growth factors, or other hormones via secretory, cytoplasmic, or both types of phospholipase, and then converted to various bioactive lipids known as eicosanoids. These eicosanoids can act as a second messenger or via their cognate receptors, eliciting a variety of cellular responses.

The fibroblast is an important component of the TME. A fibroblast is a mesenchymal cell that is primarily responsible for the maintenance and remodelling of the ECM, the stimulation and regulation of inflammation, the regulation of epithelial differentiation and proliferation, and wound repair. Fibroblasts hold noncancerous tissues together and control their functions to maintain tissue homeostasis, especially after tissue damage. Normally, tissue damage causes an inflammatory response. Many molecules involved in this response, such as growth factors and cellular adhesion molecules activate fibroblasts. These activated fibroblasts or my fibroblasts, produce ECM matrix components and matrix-modifying proteins, such as type I collagen and matrix metalloproteinases, to remodel and repair the damaged tissue.

However, in the presence of accumulated cellular stresses, such as chronic inflammation, the insult to the tissue is never fully resolved, resulting in pathological, sustained activation of fibroblasts. In the context of cancer, the same fibroblasts that normally protect against tumorigenesis and invasion can be reprogrammed to promote tumorigenesis. When a fibroblast undergoes such reprogramming, it is referred to as a cancer-associated fibroblast. CAFs function similarly to my fibroblasts in wound healing, secreting molecules to control and change the tissue's composition. However, the CAF's actions create a TME favourable for tumour growth, endowing a tumour with many of the major characteristics of cancer [2].

Carcinogenesis is a multi-step, complex process that results in the formation of a mass of malignant cells, known as a tumour. A tumour that undergoes carcinogenesis acquires most, if not all, of the hallmarks of cancer: sustained proliferative signalling, evasion of growth suppressors, replicative immortality, invasive ability and metastasis, induced angiogenesis, resistance to cell death, deregulation of cellular energetics, genomic instability and mutation, avoidance of immune destruction, and tumour-promoting inflammation. Numerous studies over the last several decades have established the role of genetic alterations in a neoplasm's acquisition of these major cancer characteristics. The identification of oncogenes and tumour suppressor genes, as well as their associated signalling pathways and mechanisms of pro-oncogenic genetic alterations, has provided direct explanations for the uncontrolled growth of tumours.

The tumour microenvironment is a highly dynamic, complex environment that evolves in tandem with the multi-step tumorigenesis process. It is a fusion of cancer cells, non-cancerous cells such as fibroblasts, immune cells, vascular endothelial cells, and non-cellular elements such as extracellular matrix. Non-neoplastic TME components have multiple functions that may not be as clearly pro-oncogenic as an oncogene. In fact, many of these functions have anti-oncogenic properties. During the multi-step tumorigenesis process, however, these TME components provide functions that can collaborate with oncogenic genetic changes. Signals originating in native and/or modified micro environmental factors may train a tumour into one of several possible molecular evolution pathways [3].

Through the use of these collaborative pathways, the neoplasm acquires the major characteristics of cancer. Simply put, tumorigenesis involves both changes in gene expression and the development of TME, as well as complex interactions between the two via complex and overlapping signalling pathways. In this section, we will discuss the roles of prostaglandins, a type of bioactive lipid, in the formation and modulation of TMEs, with a focus on their immune components, during tumorigenesis, and the implications for cancer prevention and treatment. There are several excellent reviews on prostaglandins in other aspects of cancer biology [4,5].

Conclusion

Immune system cells are dynamic components of the TME. Immune cells are primarily responsible for defending against foreign organisms and removing damaged tissue. These functions are normally tightly controlled by both feedforward and feedback control mechanisms in order to maintain tissue homeostasis. Immune cells are important constituents of the tumour stroma in TMEs and play an active role in the formation and evolution of TMEs during the multi-step tumorigenesis process. TMEs contain both innate and adaptive immune cells. Macrophages, neutrophils, DCs, innate lymphoid cells, Myeloid-Derived Suppressor Cells (MDSCs), and natural killer cells are among the innate immune cells found in TMEs, with the majority of them implicated in tumour progression. Typically, the presence of specific innate immune cells, such as M1-polarized macrophages and Batf3-dependent CD103+ subtype DCs, is linked to better clinical outcomes. Monocytes and M2-polarized macrophages within tumours, on the other hand, promote the formation of an immunosuppressive environment and contribute to tumour growth, progression, and metastasis, all of which lead to poor clinical outcomes.

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Conflict of Interest

None.

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