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Unravelling the Intricacies: The Role of Stem Cells in Brain Tumour Initiation and Progression

Peter Wallstrom*

Department of Pre-Clinical Oncology, University of Nottingham, United Kingdom

DESCRIPTION

Brain tumors, with their heterogeneous nature and complex microenvironment, pose formidable challenges in both understanding their pathogenesis and developing effective treatment strategies. Among the various cell populations implicated in brain tumor development, stem cells have emerged as key players, contributing to tumor initiation, progression, and therapeutic resistance. Understanding the role of stem cells in brain tumor biology is essential for elucidating underlying mechanisms and identifying novel therapeutic targets to combat these devastating diseases.

Stem cells possess unique properties, including self-renewal and multipotency that enable them to give rise to different cell types within the central nervous system. Within the context of brain tumors, several sources of stem cells have been implicated in tumor initiation and maintenance, including neural stem cells (NSCs), cancer stem cells (CSCs), and mesenchymal stem cells (MSCs). These stem cell populations contribute to tumor heterogeneity and plasticity, fueling tumor growth, invasion, and recurrence. One of the primary sources of stem cells implicated in brain tumor initiation is neural stem cells (NSCs), which reside in specialized niches within the adult brain, such as the subventricular zone (SVZ) and the dentate gyrus of the hippocampus. NSCs have the capacity to self-renew and differentiate into neurons, astrocytes, and oligodendrocytes under physiological conditions. However, under pathological conditions such as brain tumor formation, NSCs can undergo dysregulated proliferation and differentiation, contributing to tumor growth and invasion.

Moreover, mesenchymal stem cells (MSCs), which normally reside in the bone marrow and other tissues, have been shown to migrate to sites of brain injury and inflammation, including brain tumors. Once within the tumor microenvironment, MSCs can undergo differentiation into stromal cells, such as cancer-associated fibroblasts (CAFs), and contribute to tumor growth, angiogenesis, and immunosuppression. Additionally, MSCs have been implicated in promoting therapeutic resistance and tumor recurrence through paracrine signaling and modulation of the tumor microenvironment. The interaction between stem cells and the tumor microenvironment plays a critical role in brain tumor initiation and progression. The tumor microenvironment, characterized by hypoxia, inflammation, and dysregulated signaling pathways, provides a niche conducive to the survival and expansion of stem cell populations. Crosstalk between tumor cells, immune cells, endothelial cells, and stromal cells within the tumor microenvironment orchestrates complex signaling networks that promote tumor growth, invasion, and immune evasion.

Understanding the molecular mechanisms underlying the aberrant behavior of stem cells in brain tumors is essential for developing targeted therapies that disrupt tumor-promoting pathways while sparing normal stem cell function. Several signaling pathways and molecular markers implicated in stem cell dysregulation in brain tumors have been identified as potential therapeutic targets. For example, targeting Notch signaling, which plays a critical role in stem cell maintenance and differentiation, has shown promise in inhibiting CSC self-renewal and tumor growth in preclinical models of brain tumors. Stem cells play a multifaceted role in brain tumor initiation and progression, contributing to tumor heterogeneity, therapeutic resistance, and tumor recurrence. Understanding the molecular mechanisms underlying stem cell dysregulation in brain tumors is essential for developing targeted therapies that disrupt tumor-promoting pathways while sparing normal

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Corresponding author Peter Wallstrom, Department of Pre-Clinical Oncology, University of Nottingham, United Kingdom, E-mail: wallstrom_p7845@hotmail.com

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stem cell function. By elucidating the intricate interplay between stem cells and the tumor microenvironment, we can uncover novel therapeutic targets and strategies to combat these devastating diseases and improve outcomes for patients with brain tumors.

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CONFLICT OF INTEREST

The author's declared that they have no conflict of interest.