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Cancer stem cells niche

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Cancer stem cells (CSCs) are tumourigenic cells that have the ability to initiate clonal tumours through mitotic division of single somatic cells. CSCs are also capable of repopulating themselves indefinitely. They demonstrate flexibility by switching back and forth between stem and non-stem cell stages. CSCs are usually quiescent and remain in the CSC niche, which is a distinct area in the tumour microenvironment. In the niche, the CSCs are protected from any antitumor therapy. The CSC niche is a perfect habitat for the maintenance of various properties of the CSCs. External niche signals control the stem-like status of CSCs and the malignant features of their progeny. A multifunctional cytokine called the transforming growth factor (TGF) is integral in tumour progression and the stem cell niche. TGF is well documented in literature as a tumour suppressor or promoter at various phases of cancer formation. Further, the CSC niches usually include various components, such as blood vessels, fibroblasts and immune cells. The cellular constitution, signalling components and the extracellular matrix (ECM) proteins make the CSC niche molecularly unique. Several studies have supported the assumption that reciprocal interaction between CSCs and their presumed habitats is an integral component of tumour proliferation and growth. In order to better understand the CSC niches, in vitro three-dimensional (3D) culture models have been designed to replicate the cellular heterogeneity, spatial dimension and molecular networks of the tumour microenvironment. Furthermore, the CSC niches send out signals that keep the CSCs alive and well. Invasion and metastasis are frequently promoted by the same microenvironmental cues that induce stemness in the context of therapeutic resistance. A major cause of cancer mortality is metastatic progression which is typically linked to the activation of stem cell programs in tumours. Additionally, the cancer cells must withstand harsh environments at each step. As a consequence, cells with increased invasiveness and the ability to self-renew are selected. CSCs can be activated and attracted into other tissues in response to stress, where they develop and create new cancer cells. Targeting multiple stem cell-related markers and signalling pathways, such as dual-targeting and nanoparticle-enabled medicines may offer a fresh strategy for CSC-targeted therapy. The obstacles related to CSC-focused therapy, such as the risk of harming normal stem/progenitor cells must be studied further. This may help in understanding the complex mechanisms of resistance of these cells to various therapies.

Keywords: Cancer stem cells, Quiescent, Niche, Therapeutic resistance, Metastatic progression

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