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Cancer Stem Cells Concept of Cancer Therapy and Strategies

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Editorial

Intratumor heterogeneity is caused by a variety of mechanisms, including genetic mutations, the microenvironment, and the presence of cancer stem cells, which have increased renewal capacity and the ability to replicate the heterogeneity found in primary tumours (CSCs). We discuss how the concept of CSCs was defined, what assays are currently used to define CSC functional properties, what intrinsic and extrinsic mechanisms regulate CSC functions, how plastic CSCs are, and the importance of epithelial-to-mesenchymal transition in conferring CSC properties in this review. Finally, we discuss how CSCs can resist medical therapy and contribute to tumour relapse. Cancer patient survival has increased significantly, owing primarily to multidisciplinary care, improved chemotherapeutic agents in both adjuvant and metastatic settings, the introduction of targeted biologic agents, and the incorporation of palliative care services into the management scheme. Despite these advancements, a significant proportion of patients continue to have recurrence after adjuvant treatment, and survival associated with stage IV solid tumours remains low. Many of the agents used to treat patients with cancer fail due to primary or acquired resistance to chemotherapeutic and biologic agents. The presence of intratumoral heterogeneity and the molecular complexity of many cancers can explain this. Genetic mutations, interactions with the microenvironment, and the presence of cancer stem cells are all factors that contribute to intratumoral heterogeneity. Breast cancer, brain tumours, lung cancer, colon cancer, and melanoma have all been found to contain cancer stem cells. Cancer stem cells have the ability to self-renew, produce progeny that are distinct from them, and use common signalling pathways. Cancer stem cells could be the source of all tumour cells in a malignant tumour, the cause of resistance to the chemotherapeutic agent used to treat the malignant tumour, and the source of cells that cause distant metastases. This will focus on cancer stem cell properties; compare and contrast the cancer stem cell model with the clonal evolution model of tumorigenesis; discuss the role of cancer stem cells in the development of chemotherapy resistance; and review the therapeutic implications and challenges of targeting cancer stem cells, with an assessment of the potential such an approach holds for improving outcomes for cancer patients. Chemoresistance is a major issue in cancer therapy because cancer cells develop mechanisms to counteract the effect of chemotherapeutic compounds, resulting in relapse and the development of more aggressive cancers, which contribute to a poor prognosis and survival rate in treated patients. Cancer stem cells (CSCs) play an important role in this occurrence. Aside from their slow proliferation, CSCs have evolved a variety of cellular processes involving drug efflux, drug enzymatic inactivation, and other mechanisms. Furthermore, the microenvironment in which CSCs evolve (CSC niche) plays an important role in cancer initiation, progression, and chemoresistance. Immune cells, mesenchymal stem cells (MSCs), endothelial cells, and cancer associated fibroblasts (CAFs) all contribute to the maintenance of CSC malignancy in the CSC niche by secreting factors that promote cancer progression and chemotherapy resistance. Because of these impediments to successful cancer therapies, CSCs are the focus of intense research aimed at better understanding CSC behaviour and developing efficient targeting therapies. We provide an overview of cancer stem cells, their role in cancer initiation, progression, and chemoresistance, and discuss the progress made in the development of CSC targeted therapies [1-5].

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