

## Tumour propagation models in cancer stem cell

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### Commentary

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### DESCRIPTION

Cancer Stem Cells (CSCs) are cancer cells (found in tumours or haematological cancers) that have characteristics similar to normal stem cells, namely the ability to give rise to all cell types found in a specific cancer sample. CSCs, as opposed to other non-tumorigenic cancer cells, are indeed tumorigenic (tumor-forming). Through the stem cell processes of self-renewal and differentiation into multiple cell types, CSCs can generate tumours. Such cells are thought to persist in tumours as a distinct population, causing relapse and metastasis by generating new tumours. As a result, the development of specific therapies targeting CSCs holds out hope for improved cancer patient survival and quality of life, particularly in patients with metastatic disease.

Cancer treatment procedures are being developed upon approval in clinical trials and the therapies are capable of reducing the tumour effect. Animals, on the other hand, do not provide an accurate representation of human disease. Tumor relapse is particularly difficult to study in mice, whose lifespans do not exceed two years. In the early stages of testing, the efficacy of cancer treatments is frequently measured by the ablation fraction of tumour mass (fractional kill). Because CSCs make up a small proportion of the tumour, drugs that specifically target stem cells may not be chosen. According to the theory, conventional chemotherapies kill differentiated or differentiating cells, which make up the majority of the tumour but do not generate new cells.

A population of CSCs that gave rise to it could remain unaffected, causing relapse. They have been a major focus of cancer research since the early 2000s. Cells within the tumour exhibit functional heterogeneity in different tumour subtypes, and tumours are formed from cells with varying proliferative and differentiation capacities. Because of this functional heterogeneity among cancer cells, multiple propagation models have been developed to account for heterogeneity and differences in tumor-regenerative capacity: The Cancer Stem Cell (CSC) and stochastic models. However, some argue that this distinction is artificial, because both processes act in complementary ways in terms of actual tumor populations. Importantly, the proliferative burden is met by a stochastically dividing basal

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epithelium in healthy human esophageal epithelium. However, when it transitions to the precancerous Barrett's oesophagus epithelium, a small dedicated stem cell compartment appears that supports epithelial proliferation while evidence for a stochastically dividing compartment contributing to tissue maintenance disappears. As a result, dedicated stem cell compartments maintain and expand the size of the transformed compartment in at least some neoplastic tissues.

The cancer stem cell model, also known as the Hierarchical Model, proposes that tumours are organised hierarchically (with CSCs at the top). Cancer Stem Cells (CSC) are tumorigenic cells that are biologically distinct from other subpopulations within the tumour cancer population. They are distinguished by two characteristics: Their long-term ability to self-renew and their ability to differentiate into non-tumorigenic progeny that contributes to tumour growth. According to this model, only certain subpopulations of cancer stem cells have the ability to drive cancer progression, implying that there are specific (intrinsic) characteristics that can be identified and then targeted to destroy a tumour long-term without having to battle the entire tumour.