

Cancer stem cell

Cancer [stem cells](#) (CSC) have been postulated to be responsible for the key features of a malignancy and its maintenances, as well as therapy resistance, while differentiated cells are believed to make up the rapidly growing tumour bulk. It is therefore important to understand the characteristics of those two distinct cell populations in order to devise treatment strategies which effectively target both cohorts, in particular with respect to cancers, such as [glioblastoma](#).

Normal human embryonic stem cells (hESCs) can develop neoplastic cancer stem cell (CSC) properties after coculture with transformed hESCs in vitro.

[Cancer stem cells](#) (CSCs) or cancer initiating cells (CICs) maintain self-renewal and multilineage differentiation properties of various tumors, as well as the cellular heterogeneity consisting of several subpopulations within tumors.

They constitute the diverse hierarchy of cells composing a tumor. When xenografted into an appropriate host, they are capable of tumorigenesis.

CSCs display the malignant phenotype, self-renewal ability, altered genomic stability, specific epigenetic signature, and most of the time can be phenotyped by [cell surface markers](#) (e.g., [CD133](#), [CD24](#), and [CD44](#)).

Numerous studies support the concept that non-stem cancer cells (non-CSCs) are sensitive to cancer therapy while CSCs are relatively resistant to treatment ¹⁾.

In glioblastoma stem cells (GSCs), there is clonal heterogeneity at the genetic level with distinct tumorigenic potential, and defined GSC marker expression resulting from clonal evolution which is likely to influence disease progression and response to treatment.

Given the critical role of cancer stem cells in the pathogenesis of glioblastoma, research into their molecular and phenotypic characteristics is a therapeutic priority ²⁾.

They are functionally defined by their ability to self-renew and propagate tumors similar to the parental tumors from which they are derived. While the field of cancer stem cell biology is relatively young, continued elucidation of the tumor hierarchy holds promise for the development of novel patient therapies ³⁾.

Cancer stem cells contribute to glioma radioresistance by an increase of DNA repair capacity through preferential activation of the DNA damage response checkpoints.

Mesenchymal stem cells and/or neural stem cells were shown to target brain tumors therefore these cells are considered as an effective delivery system to target and disseminate therapeutic agents to brain tumors. [Stem cell therapy](#) for glioblastoma were shown in experiments to be effective way to target brain tumors.

BMP4 reduces effectively proliferation of CD133 positive cells in vitro and the tumor growth in vivo. BMP4 may act as a key inhibitory regulator of cancer initiation and therefore may be used in combined stem cell-based therapy as a non-cytotoxic therapeutic agent ⁴⁾.

see [glioma cancer stem cell](#)

Patients with [glioblastoma multiforme](#) (GBM) that are [cancer stem cell](#) positive (GSC [+]) essentially cannot benefit from [antiangiogenic](#) therapy. In a study, the potential anti-angiogenic and anti-invasive effects of [Olea europaea](#) (olive) leaf extract (OLE) were tested using GSC (+) tumours. OLE (2mg/mL) caused a significant reduction in tumour weight, vascularisation, invasiveness and migration ($p=0.0001$, $p<0.001$, $p=0.004$; respectively) that was associated with reducing the expression of VEGFA, MMP-2 and MMP-9. This effect was synergistically increased in combination with bevacizumab. Therefore, our current findings may contribute to research on drugs that inhibit the invasiveness of GBM ⁵⁾.

1)

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2)

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3)

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