Good example of cancer stem cells essay

Law, Evidence

What is the evidence that convinces you Cancer Stem Cells are a subpopulation of tumour cells with distinct tumour-initiating capacity?

What are cancer stem cells (CSCs)? They are cancer cells that have similar characteristics as normal stem cells. CSCs have the potential to give rise to other cells types, and thus are tumorigenic in nature. These cells can generate tumors via stem cell processes of self-renewal, as well as differentiation into different cells (Wang, Chiou & Wu, 2013). CSCs have been found to persist in tumors in the form of a district population and often cause relapse and metastasis giving forth to new tumors. The notion that cancer develops from a subpopulation of tumor cells that possess stem cell properties that have later been found to be cancer stem cells has helped shade more light on CSCs. In other words, it has been found that these unique cells can not only propagate the tumor, but also develop a solid sustainability against therapeutic remedies (Wang, Chiou & Wu, 2013). These cells for that matter explain the metastatic and re-occurrence of malignant tumors, as well as intra-tumoral heterogeneity (Wicha, Liu & Dontu, 2006). The functional characteristics of these cells are used in the definition of those cells. In essence, they have been found to induce tumor in syngeneic or immune-compromised mice. They also have a self-renewal capacity, and this can be assessed by the sphere formation in vitro or tumor formation in secondary mice in vivo (Wicha, Liu & Dontu, 2006).

On top of that, these cells can differentiate into non-self-renewable cancer cells. Research has found the identity, as well as existence of CSCs in hematopoietic tumors. They have also been found in other solid tumors such as head and neck, breast, prostate, colon, lung, and brain among others

(Visvader & Linderman, 2008). CSCs are a unique subpopulation and have been found to have radio or chemo-resistant properties. In addition, they have been found to have a higher potential of tumor initiation, as well as accelerated regrowth after chemotherapy. CSCs were first derived from breast cancer cells. They were found to be a significantly minor population of all cells that expressed a low level of CD24 and a high level of CD44 cell surface markers (Visvader & Linderman, 2008). Besides, they are positive for the cell surface antigen. These evidences substantiate the notion that Cancer Stem Cells are a subpopulation of tumour cells with distinct tumour-initiating capacity (Visvader & Linderman, 2008).

What is the evidence that convinces you Cancer Stem Cells are influenced by their niche components?

The properties, as well as mechanisms of formation of CSCs, have attracted numerous studies particularly because of their heightened ability to initiate and enhance cancer growth and their intrinsic resistance to chemotherapy (Takakura 2012). In order to evaluate the niche supporting stemness, the location of tissue specific stem cells have been researched. Research has shown that neuronal stem cells tend to localize close to the blood vessels (Takakura 2012). This occurs in the sub-ventricular zone and hippocampus. At the time of embryonic brain development, the neural ectoderm is said to synthesize vascular endothelial growth factor (VEGF). This factor is needed for neo-vascularization. Neuronal stem cells and the endothelium localize together before birth in the embryo. This interaction has been found to proceed after birth. Additionally, research has highlighted more useful insights. The characteristics of neuronal stem cells such as self-renewal, and

the maintenance of an immature status tend to be induced by the notch signaling pathway (Fuchs, Tumbar and Guasch 2004).

With respect to glioblastoma tumors, it has been found that there exist CSCs that have heightened DNA repair capacity, as well as tumor-initiating ability (Fuchs, Tumbar and Guasch 2004). To be specific, nestin CSCs are said to localize near the CD34 cells. When these CSCs are inoculated in an immunocompromised mice, tumor formation occurred. It was found to be faster and higher if the endothelial cells derived from the same source as the CSCs were injected in the inoculation (Fuchs, Tumbar and Guasch 2004). Nitric Oxide that is released by the endothelial cells was said to trigger faster CSCs self-renewal through the activation of the notch pathway. Furthermore, it has also been indicated that CSC inhibit the apoptosis of endothelial cells. The resistance nature of CSCs to vascular disruptive is agents attributed to the inhibition of endothelial cells apoptosis (Fuchs, Tumbar and Guasch 2004).

In another example, the niches of skin tumor cells have been elucidated; keratinocyte stem cells are situated in the basal layer. These cells continuously undergo self-renewal in response to keratinocyte turnover. In the skin tumor CSCs model, it has been suggested that CSCs in this niche express neuropilin-1. Neuropilin-1 is activated when VEGF is released by VSCs (Soker et al. 1998). This activation occurs via an autocrine and paracrine loop. Consequently, the CSCs undergo proliferation. These findings highlight that CSCs are influenced by their niche components.

References

Fuchs, E, Tumbar, T, Guasch, G (2004), Socializing with the neighbors: stem cells and their niche, Cell vol. 11, pp. 769–78.

Soker S, Takashima S, Miao HQ, Neufeld G, Klagsbrun M (1998), Neuropilin-1 is expressed by endothelial and tumor cells as an isoform-specific receptor for vascular endothelial growth factor Cell vol. 92, pp. 735–45.

Takakura, N (2012), Formation and regulation of the cancer stem cell niche, Cancer Science Vol. 103, no. 7, pp. 1177–1181.

Visvader JE, Lindeman GJ (2008), Cancer stem cells in solid tumours: accumulating evidence and unresolved questions, Nat Rev Cancer vol. 8 no. 10, pp. 755–768.

Wang, ML, Chiou, SH, and Wu, CW (2013), Targeting cancer stem cells: emerging role of Nanog transcription factor, Onco Targets Ther vol. 6, pp. 1207–1220.

Wicha MS, Liu S, and Dontu G (2006), Cancer stem cells: an old idea – a paradigm shift, Cancer Res vol. 66 no. 4, pp. 1883–1890.