Biomarkers and signaling pathways of colorectal cancer stem cells

Danysh Abetov, Zhanar Mustapova, Timur Saliev, Denis Bulanin

Laboratory of translational medicine and Life Sciences Technologies, Department of Biomedical Sciences

Abstract

The progression of colorectal cancer is commonly characterized by accumulation of genetic or epigenetic abnormalities, altering regulation of gene expression as well as normal protein structures and functions. Nonetheless, there are some questions that remain to be elucidated, such as the origin of cancer cells and populations of cells initiating and propagating tumor development. Currently, there are two rival theories describing the process of carcinogenesis. One is the stochastic model, arguing that any cell is capable of initiating and triggering the development of cancer. Meanwhile, the cancer stem cell model hypothesizes that only a small fraction of stem cells possesses cancer-promoting properties. Typically, colorectal cancer stem cells (CSCs) share the same molecular signaling profiles with normal stem cells or embryonic stem cells, such as Wnt, Notch, TGF-β, and Hedgehog. Nevertheless, CSCs differ from normal stem cells and the bulk of tumor cells in their tumorigenic potential and susceptibility to chemotherapeutic drugs. This may be a possible explanation of the high percentage of cancer recurrence in patients who underwent chemotherapeutic treatment and surgery. This review article focuses on the colorectal cancer stem cell biomarkers and the role of upregulated signaling pathways implicated in the initiation and progression of colorectal cancer.

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