OPEN ACCESS Freely available online

Journal of Cell Science & Therapy

ISSN: 2157-7013

Commentary

Cancer Stem Cell Redox Regulation

Dipak Bodhi*

Department of Genetic Medicine, JSS Medical College, Mysuru, Karnataka, India

DESCRIPTION

Low to moderate levels of ROS within the cell will often encourage cell growth and proliferation while also boosting cell survival. On the other hand, too much ROS can make cells poisonous and start apoptosis. The antioxidant mechanisms in the cell can scavenge ROS and stop cellular oxidative damage before it becomes permanent. As a result, it is crucial for cells to maintain a balance between their ROS generation and antioxidant systems and for growth and development, redox regulation of cellular processes is crucial.

Many cancer cells have greater ROS levels in part because of their faster metabolism. Apoptosis and necrosis in cancer cells can be induced by abnormal ROS levels. High antioxidant capability helps cancer cells scavenge and combat ROS. Induction of ROS-mediated damage in cancer cells by appropriate pharmacological agents that either promote ROS generation beyond the cellular antioxidative capacity or disable the cellular antioxidant system has been considered as a "radical" therapeutic strategy to preferentially kill cancer cells. This is because high antioxidant capacity enhances cell survival and impairs cellular responses to anticancer therapy.

As a fraction of cancer cells with stem cell-like qualities and characteristics have been detected and described in numerous malignancies, including leukaemia, breast cancer, and pancreatic cancer, the concept of Cancer Stem Cells (CSCs) has been gaining support recently. CSCs are believed to be self-renewing and differentiating cells that cause cancer to return after chemotherapy or radiotherapy because they may endure treatment and then swiftly produce new tumours. These capabilities of CSCs give rise to the idea that cancer therapeutic tactics should target CSCs as well as conventional cancer cells.

The majority of cancer cells can be killed by conventional therapies (chemotherapy or radiotherapy) that target redox balance because of the significance of redox balance in cancer cells. The specific redox balance found in CSCs and the underlying mechanisms that shield them from ROS-mediated cell death are still poorly understood. We shall update the effects of ROS/redox regulation on the characteristics and operations of CSCs in this review. We intend to spark significant interest in further examining the function of redox regulation in CSCs and the usefulness of targeting ROS-dependent/redox control of pathways by paying particular attention to the cross talk between CSC-related pathways and redox regulation.

Scavenging ROS in CSCs and regular stem cells is also facilitated by a variety of signalling channels and transcriptional activities (see details in the following sections). To keep low ROS levels in Haematopoietic Stem Cells (HSCs), which are crucial for the stemness of HSCs, Forkhead Homeobox Type O (FOXO) is necessary. Additionally, the Ataxia Telangiectasia Mutation (ATM) can upregulate the antioxidant enzymes and downregulate the genes involved in differentiation and proliferation, hence contributing to the maintenance of stemness and low ROS levels. The preservation of stemness and resistance to 5-fluorouracil and gemcitabine in pancreatic cancer stem cells depends on JNK pathway activation, which inhibits ROS production brought on by these chemotherapy medications.

CONCLUSION

Although little is known about how ROS are regulated in CSCs, there is rapidly growing evidence that ROS may be crucial to the ability of CSCs to differentiate and self-renew. Redox balance and ROS regulation in CSCs are regulated by ROS-dependent signalling pathways and transcriptional processes. Targeting CSCs *via* antioxidant proteins and ROS control has the potential to significantly enhance cancer treatment.

Correspondence to: Dipak Bodhi, Department of Genetic Medicine, JSS Medical College, Mysuru, Karnataka, India, E-mail: jbat23@yahoo.com

Received: 03-Nov-2022; Manuscript No. JCEST-22-21009; **Editor assigned:** 07-Nov-2022; Pre-Qc No. JCEST-22-21009 (PQ); **Reviewed:** 17-Nov-2022; Qc No. JCEST-22-21009; **Revised:** 25-Nov-2022, Manuscript No. JCEST-22-21009 (R); **Published:** 05-Dec-2022, DOI: 10.35248/2157-7013.22.S11.372.

Citation: Bodhi D (2022) Cancer Stem Cell Redox Regulation. J Cell Sci Therapy.S11:372.

Copyright: © 2022 Bodhi D. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.