Oxidative Medicine and Cellular Longevity

Special Issue on
Redox Homeostasis and Cancer

Hindawi

CALL FOR PAPERS

Under physiological conditions, a balance between oxidants and antioxidants exists. Reactive oxygen species (ROS) are continuously generated by aerobic cells and eliminated through scavenging systems to maintain redox homeostasis. Disruption of redox homeostasis results in oxidative stress and altered ROS signaling. Higher ROS levels can lead to DNA mutation and genomic instability which can play causal role in cancer development and progression. These mutations, coupled with distorted redox signaling pathways, orchestrate pathologic events inside cancer cells resulting in resistance to stress and death signals, aberrant proliferation, and inefficient repair mechanisms.

Cancer cells are energy hungry, owing to their high rate of proliferation. However, due to impaired TCA cycle and poor blood perfusion, cancer cells switch towards a glycolytic pathway for energy generation termed as 'Warburg effect'. Such pathways lead to higher oxidative environment. The oxidative environment is also enhanced by tumor-infiltrating macrophages and neutrophils. Thus cancer cells are used to a high ROS environment. This redox imbalance allows protumorigenic cell signaling.

In this issue, we explore the relation between ROS and cancer. Although ROS sustain tumorigenesis and cancer progression, these can also be efficient therapeutic tools to fight cancer. Oxidative stress based therapies, like radiotherapy, chemotherapeutic agents, and photodynamic theory, increase ROS levels in the tumor niche, taking advantage of the cytotoxic face of ROS for killing tumor cells through a sudden, localized, and intense oxidative burst. Clinical efficacy of anticancer therapies is often subdued by multidrug resistance (MDR). Redox therapy, using redox-active drugs or inhibitors of inducible antioxidant defense in the tumor microenvironment, has been reported to be effective against MDR tumors.

In light of these developments, we invite authors to contribute original research articles, as well as review articles, exploring the role of redox and its regulation in cancer.

Potential topics include but are not limited to the following:

- Reactive oxygen species (ROS) mediated genomic and epigenomic instability
- Redox signaling as a driving force for epigenetic modifications
- Transcriptional regulation of Warburg effect
- Redox and metabolic circuits in cancer
- Role of tumor infiltrating macrophages and neutrophils in the cancer redox environment
- Redox signaling for overcoming multidrug resistance in cancer chemotherapy
- Dietary antioxidants and cancer prognosis
- Role of redox in cancer caused by ionizing radiation
- Oxidative stress induced by conventional anticancer therapy using ionizing radiation
- Role of redox associated noncoding RNAs in the etiology of cancer
- Oxidative stress and estrogen receptor-associated breast carcinogenesis
- Mitochondrial targeted compounds towards cancer cells

Authors can submit their manuscripts through the Manuscript Tracking System at https://mts.hindawi.com/submit/journals/omcl/reca/.

Papers are published upon acceptance, regardless of the Special Issue publication date.

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Submission Deadline
Friday, 6 September 2019

Publication Date
January 2020