Oxidative Medicine and Cellular Longevity

Hindawi

Special Issue on

Oxidative Signaling in Cancer Therapy-Induced Cardiovascular Disease

CALL FOR PAPERS

In the last decades, major advances in antitumor therapy have significantly reduced cancer death rates but have concomitantly highlighted that cardiovascular disease is the leading cause of morbidity and mortality among cancer survivors. Common effective anticancer therapies not only destroy transformed cells but significantly damage other cells, especially cardiomyocytes and vascular cells, with possible life-long alterations culminating with heart failure. Despite the cardiotoxic risk of anticancer drugs has been long recognized, cardioprotective strategies for cancer patients are still elusive, mainly because of the limited understanding of the underlying mechanisms. The most accepted view is that common antitumor therapies generate intracellular reactive oxygen species (ROS) that eventually cause cardiac and/or vascular damage, but this phenomenon is just beginning to be unraveled.

We invite authors to contribute original research articles as well as review articles that will illustrate and stimulate the growing efforts to understand the implication of oxidative signaling in cancer therapy-induced cardiovascular disease. We are interested in articles describing new molecular players of oxidative stress as well as new approaches to manipulate ROS-driven processes for the treatment of cardiovascular complications associated with common antitumor therapies.

Potential topics include but are not limited to the following:

- Role of oxidative stress in the cardiotoxicity induced by different, commonly used antitumor regimens, including radiation, anthracyclines, and monoclonal antibodies
- Role of oxidative stress in different types of cardiotoxicities, including not only cardiac dysfunction but also myocardial ischemia, arrhythmias, thromboembolism, arterial and pulmonary hypertension, and peripheral arterial occlusive disease
- Role of oxidative stress in the different cell types targeted by anticancer therapies, including but not limited to cardiomyocytes, cardiac fibroblasts, endothelial cells, progenitor cells, and vascular smooth muscle cells
- Identification of new signaling pathways/players implicated in ROS production in response to anticancer therapies
- Identification of new potential tools to manipulate oxidative signaling for therapeutic purposes in the treatment of cancer therapy-induced cardiovascular disease

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

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