Oxidative Medicine and Cellular Longevity

Special Issue on

Oxidative Stress-Mediated Reperfusion Injury 2014

CALL FOR PAPERS

Oxidative stress plays an important role in the pathogenesis of ischemia reperfusion injury. The mechanisms underlying ischaemia/reperfusion injury have been extensively studied using healthy tissues, yet less attention has been devoted to defining how the different risk factors such as diabetes and aging (situations that are associated with increased oxidative stress) may influence the degree of organ or cellular injury under pathological conditions like ischemia/reperfusion. While rapid production of large amount of reactive oxygen species (ROS), called oxidative burst, causes oxidative damage which contributes significantly to ischemia reperfusion injury, moderate elevation of ROS production induced by therapeutic interventions like ischemic or pharmacologic preconditioning may trigger cellular protective signaling pathways, leading to the attenuation of ischemia reperfusion injury.

We invite investigators to contribute original research articles as well as review articles that will stimulate the continuing efforts to understand the molecular mechanism underlying ischemia/reperfusion injury under normal and diseased conditions to develop strategies to treat these pathological conditions.

Potential topics include, but are not limited to:

- Role of reactive oxygen species (ROS) in organ (heart, lung, brain, liver, kidney, and/or intestine) ischemia reperfusion injury under normal and diseased (e.g., diabetes and hypertension) conditions
- Cellular protective signaling pathways that ROS may trigger that contribute to cellular repairing during ischemia reperfusion injury and the potential interplay between these pathways
- Cardioprotection in aged and/or functionally impaired hearts
- Role of reperfusion-induced oxidative stress and inflammation in mediating remote organ injury
- Recent advances in preventing ischemia-reperfusion injury

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