Oxidative Medicine and Cellular Longevity

Hindawi

Special Issue on

Interplay of Oxidative Stress, Inflammation, and Autophagy: Their Role in Tissue Injury of Heart, Liver, and Kidney

CALL FOR PAPERS

Oxidative Medicine and Cellular Longevity invites manuscripts, whether a research article or review, from both scientists and clinicians to contribute their novel findings for this special issue. The disturbance of the balance of normal redox state of cells can cause toxic effects through the production of unchecked free radicals that target proteins, DNA, lipids, and vitamins, contributing to alterations of their normal functions, leading to the production of diverse biological effects and ultimately the development and/or progression of tissue injury. Oxidative stress may be developed and/or enhanced by various mechanisms. For instance, the development of inflammation and the alteration of autophagy are two major pathways that might play a critical role in the induction and/or progression of deleterious tissue injury via oxidative stress-mediated events.

Conversely, oxidative stress has been reported to mediate the development of inflammation as well as the alteration of autophagy. Thus, there might be either an independent role of each of these factors (i.e., oxidative stress, inflammation, and alteration of autophagy) or interplay among them to cause damaging effects on the tissues. Typically, a series of genes responds to injurious insults, leading to the activation of various pathways, ultimately dictating cell fate in either direction (i.e., whether cell survival or demise), depending on the level of unchecked cellular damage, and thus works as cell fate speedometer. The understanding of these molecular events that lead to these physiologic alterations may also serve as a valuable tool to broaden the perspective of targeted therapeutics which may enhance cellular longevity. These crucial events will be emphasized mainly in this issue for three major organs, namely, heart, liver, and kidney; however, other organs might be considered.

Potential topics include but are not limited to the following:

- Expanding the existing knowledge and providing novel information regarding the role and mechanism(s) in response to endogenous or exogenous factors via which oxidative stress, inflammation, and alteration of autophagy mediate tissue injury, whether alone, combined, or via inducing and/or enhancing the harmful effects of each other in the heart, liver, and kidney
- Providing novel models, methodologies, and treatments that might advance the fields involving these three major injurious factors

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

Lead Guest Editor
Partha Mukhopadhyay, National Institutes of Health, Bethesda, USA
mpartha@mail.nih.gov

Guest Editors
Nabil A. S. Eid, Osaka Medical College, Takatsuki, Japan
nabil@osaka-med.ac.jp

Mohamed A. Abdelmegeed, National Institutes of Health, Bethesda, USA
abdelmegeedm@mail.nih.gov

Aditya Sen, Uniformed Services University, Bethesda, USA
aditya.sen.ctr@usuhs.edu

Manuscript Due
Friday, 25 August 2017

First Round of Reviews
Friday, 17 November 2017

Publication Date
Friday, 12 January 2018