Oxidative Medicine and Cellular Longevity

Hindawi

Special Issue on

The Interplay of Oxidative Stress and Inflammation: Mechanistic Insights and Therapeutic Potential of Antioxidants

CALL FOR PAPERS

Oxidative stress is caused by the overproduction of Reactive Oxygen Species (ROS) and diminished cellular antioxidant defenses. Under physiological conditions, ROS are involved in processes, including cellular homeostasis, modulation of cellular metabolism, signaling and redox state, and being used by the immune system to inactivate viruses and inhibit bacterial growth. However, excess ROS production can damage lipids, DNA, and proteins which can lead to cell death. ROS-induced damage underpins various oxidative stress-related human diseases and aging. In addition, the cellular antioxidant defenses may decrease during aging which results in age-related increase of ROS and oxidative stress. Excess ROS can also activate the proinflammatory signaling pathways and the release of multiple inflammatory mediators, such as cytokines, chemokines, eicosanoids, and others. Therefore, oxidative stress is implicated in the pathogenesis, development, and progression of a sustained inflammatory state. Furthermore, excess ROS and inflammation work together to trigger and orchestrate necrotic and apoptotic cell death, where mitochondrial dysfunction, caspase activation, and Bcl-2 family proteins are involved in ROS-mediated apoptosis. In addition, several protein kinases and signaling pathways (including mitogen-activated protein kinases, nuclear factor-kappaB, protein kinase C, and others) modulate apoptosis depending on the cellular context.

Oxidative stress and inflammation are increasingly recognized as having key roles in the pathogenesis of various diseases, including diabetes, obesity, cancer, neurodegeneration, metabolic syndrome, cardiovascular disease, liver disease, and others. Therefore, understanding the molecular mechanisms underlying the mutual relationship between oxidative stress and inflammation can lead to the discovery of novel strategies to prevent and/or treat various diseases. Although the cells are equipped with numerous nonenzymatic molecules and enzymatic scavengers of ROS, these defenses are not always adequate to attenuate the excess ROS production. Therefore, agents that can boost antioxidant defense mechanisms and prevent increased ROS generation can represent an effective treatment for oxidative stress/inflammation-related diseases.

We invite investigators to contribute original research and review articles that will help to elucidate the role of oxidative stress/inflammation interplay in the pathophysiology and progression of diseases. We encourage the submission of *in vitro*, *in vivo,* and clinical studies describing the role of oxidative stress and inflammation in different diseases and the modulatory role of antioxidant and anti-inflammatory agents.

Potential topics include but are not limited to the following:

- Role of oxidative stress and inflammation in different human diseases
- Strategies to prevent/treat oxidative stress/inflammation-related diseases
- Role of ROS and inflammatory mediators in cell death
- Molecular mechanisms of oxidative stress in different diseases
- New findings on potential antioxidant/anti-inflammatory agents
- Modulators of the redox-sensitive transcription factors
- Biomarkers of oxidative stress and inflammation in metabolic diseases and drug-induced injury

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

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

Lead Guest Editor
Ayman M. Mahmoud, Beni-Suef University, Beni Suef, Egypt
ayman.mahmoud@science.bsu.edu.eg

Guest Editors
Fiona L. Wilkinson, Manchester Metropolitan University, Manchester, UK
f.wilkinson@mmu.ac.uk

Mansur A. Sandhu, PMAS-Arid Agriculture University, Rawalpindi, Pakistan
mansoorsandhu@uaar.edu.pk

Adam P. Lightfoot, Manchester Metropolitan University, Manchester, UK
a.lightfoot@mmu.ac.uk

Submission Deadline
Friday, 24 January 2020

Publication Date
June 2020