

## Special Issue on **Cellular and Microbiota-Derived Oxidative Stress Mechanisms in Chronic, Age-Associated Disorders**

# CALL FOR PAPERS

Aging is a multifactorial process associated in humans with chronic oxidative stress-related diseases, most pronouncedly cardiovascular diseases, fibrotic diseases, cancer, and neurodegenerative disorders. The foremost concept of aging is the free radical theory, suggesting that progressively increasing oxidative stress may cause irreversible damage to macromolecules, resulting in a decline of organ function as well as the development of various pathologies. The mechanisms in which individual genes and signaling molecules influence the process of chronic pathogenesis are only partially understood. Importantly, aging is an evolutionary conserved mechanism, hence sharing similar characteristics in different species ranging from primitive eukaryotic organisms, for example, *Saccharomyces cerevisiae* and *Caenorhabditis elegans*, to small rodents and to larger animals, such as sheep, pig, rabbit, and nonhuman primates. As the human physiology is highly complex, more sophisticated combinations of different models are needed to study age-related diseases.

Cellular sources of reactive oxygen species (ROS) that promote pathogenesis vary depending on the status of the disease and related cellular level alterations, consequently causing aberrant signal transduction that regulates the redox balance. Interestingly, recent studies have suggested microbiota as a source of ROS, which then augments the cell stress and promotes the disease progression. Therefore, development of age-associated diseases may depend on the origin of cell stressors, as well as the concentration, location, and persistence of ROS production.

The aim of this special issue is to introduce common oxidative stress factors promoting age-associated disease progression. We invite both original research article and review article submissions describing basic and translational studies.

Potential topics include but are not limited to the following:

- ▶ Common oxidative stress mechanisms involved in the progression of chronic age-associated diseases
- ▶ Oxidative stress-derived changes in cellular signal transduction and tissue function in age-associated diseases
- ▶ Development of humanized model systems for translational studies of age-associated diseases
- ▶ Genetic mutations inducing oxidative stress in age-associated diseases
- ▶ Role of microbiota in age-associated diseases

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

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

### Lead Guest Editor

Mikko O. Laukkanen, IRCCS SDN,  
Naples, Italy  
[mlaukkan@yahoo.com](mailto:mlaukkan@yahoo.com)

### Guest Editors

Jan H. Bräsen, Medizinische  
Hochschule Hannover, Hannover,  
Germany  
[braesen.jan@mh-hannover.de](mailto:braesen.jan@mh-hannover.de)

Olli Leppänen, Enköping Hospital,  
Uppsala, Sweden  
[olli.leppanen@regionuppsala.se](mailto:olli.leppanen@regionuppsala.se)

### Submission Deadline

Friday, 1 February 2019

### Publication Date

June 2019