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Autophagy-Mediated Cellular Oxidative Stress Regulations

Guest Editor:

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closed (15 March 2023)

Message from the Guest Editor

Autophagy can be activated in response to diverse stressors such as nutrient deprivation, hypoxia, drugs and virus-mediated infections. A growing amount of evidence in recent years indicates for autophagy a role as a crucial mediator in the regulation of oxidative stress response. Oxidative stress is the result of the activity of both reactive oxygen species (ROS) and reactive nitrogen species (RNS) mainly produced through oxygen metabolism in mitochondria. ROS/RNS at physiological levels act as signalling molecules that regulate numerous cellular conditions whereas an excessive production can eventually cause cell death and give rise to a variety of diseases. In addition, high levels of oxidative stress can dysregulate autophagy favouring the accumulation of harmful aggregates. Thus, a complex interplay exists between these two processes that still remains unclear.

This Special Issue aims to collect research articles and reviews that deeply dissect and try to unravel the exact mechanisms that regulate the relationship between autophagy and oxidative stress, highlighting the role of mediators of this crosstalk and the correlations with disorders and aging.

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Message from the Editor-in-Chief

It has been recognized in medical sciences that in order to prevent adverse effects of "oxidative stress" a balance exists between prooxidants and antioxidants in living systems. Imbalances are found in a variety of diseases and chronic health situations. Our journal *Antioxidants* serves as an authoritative source of information on current topics of research in the area of oxidative stress and antioxidant defense systems. The future is bright for antioxidant research and since 2012, *Antioxidants* has become a key forum for researchers to bring their findings to the forefront.

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