# **Special Issue**

# Exploring New Frontiers in Oxidative Stress and Antioxidant Research: From Model Organisms to Human Disease

## Message from the Guest Editors

Oxidative stress (OS) plays a critical role in the pathogenesis of numerous human diseases, including cancer, neurological disorders, cardiometabolic conditions, and aging. Understanding the intricate mechanisms regulating oxidative stress and the homeostatic antioxidant response is essential for developing innovative therapeutic interventions.

Insights gained from model organisms offer valuable perspectives on the molecular and cellular processes underlying OS, paving the way for novel translational approaches in human disease research. This Special Issue aims to highlight recent advancements in oxidative stress and antioxidant research using model organisms with direct relevance to human health.

We welcome contributions focusing on cellular processes such as cell cycle regulation, intercellular communication, and cell death—key pathways implicated in disease progression. Special emphasis will be placed on studies employing cutting-edge, high-throughput omics technologies, including transcriptomics, single-cell genomics, metabolomics, and proteomics. Additionally, novel strategies for the targeted delivery of antioxidant-based therapies will be of particular interest.

#### **Guest Editors**

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## **About the Journal**

## 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.

### Editor-in-Chief

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