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Oxidative Stress, Calcium Dysregulation, and Mitochondrial Dysfunction in Human Diseases

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Deadline for manuscript submissions:
closed (15 March 2022)

Message from the Guest Editors

The interplay between oxidative stress, mitochondrial dysfunction and elevated calcium (Ca^{2+}) intracellular levels seems to be a central element in several pathological conditions, ranging from diabetes and cardiovascular diseases to neurological disorders and cancer. However, the contribution of each of these three pivotal events to the development and progression of diseases or as molecular triggers of cell death has not yet been fully elucidated. In this Special Issue, we aim to bring together manuscripts, in the form of either Original Research or Reviews, that highlight the relevance of disease models (human, cellular, murine and invertebrate) in our understanding of the reciprocal interactions between Ca^{2+} dysregulation, elevated ROS levels and mitochondrial dysfunction in the onset and progression of human disorders. Additionally, manuscripts that report emerging pharmacological approaches targeting such cellular alterations in human diseases will be also welcome. Our aim is for this collection to stimulate further research that will go beyond the *state of the art* in order to open new avenues for the design of novel therapies to treat these diseases.

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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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