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Mitochondrial Oxidative and Nitrosative Stress as a Therapeutic Target in Diseases

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Message from the Guest Editors

Although necessary for life, mitochondria are often essential for initiating apoptotic death and deciding cell fate under cellular stress. Mitochondrial oxidative/nitrosative stress has been reported to trigger autophagy by promoting autophagosome formation and induce the release of inflammatory cytokines through the activation of the NLRP3-inflammasome. Thus, it is becoming clear that mitochondria participate in many aspects of cell function, and evidence further suggests that mitochondria impairment is underlying many common human disorders.

Contributions to this Special Issue may cover research findings or review articles related to the role of mitochondrial oxidative stress and mitochondrial dysfunction in different pathological conditions ranging from cancer to neurodegenerative diseases, the mechanistic links between changes in mitochondria functionality and alterations of cellular processes such as autophagy and the inflammatory signaling pathways and its impact on cell viability, and the use of mitochondria-targeted molecules, including antioxidants, as therapeutic strategies for disease intervention.

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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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