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Cellular-Redox-Related Stress Responses in Renal Disease and Their Related Complications

Guest Editors:

Dr. Raul Rodrigues-Diez

Dr. Beatriz Suárez-Álvarez

Dr. Lucas Matias Opazo-Ríos

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Message from the Guest Editors

Cells continuously experience several classes of stress, which contributes to cellular damage. To resolve this situation, cells trigger specific transcriptional programs and cellular stress responses to restore cellular homeostasis. Some of these are hypoxic stress response, unfolded protein response in mitochondria and endoplasmic reticulum, oxidative stress response, heat shock response, or autophagy, among others. When these stimuli or stressors are extraordinarily strong or persistent on time, cells undergo irreversible damage or cell death, contributing to enhancing susceptibility to disease or aggravating it. Once we know the mechanisms involved in the intracellular responses to stress, it is relevant to elucidate their regulation, the pathophysiological consequences in the renal context, and the signals triggered by these responses to communicate damage to other cells and tissues such as the cardiovascular system. Striking a balance between the molecular mechanisms aimed at restoring homeostasis and those which contribute to maladaptation and disease is key to slowing the progression of disease.

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Editor-in-Chief

Prof. Dr. Alessandra Napolitano
Department of Chemical Sciences, University of Naples "Federico II", Via Cintia 4, I-80126 Naples, Italy

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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Contact Us

Antioxidants Editorial Office
MDPI, Grosspeteranlage 5
4052 Basel, Switzerland

Tel: +41 61 683 77 34
www.mdpi.com

mdpi.com/journal/antioxidants
antioxidants@mdpi.com
𝕏@antioxidants_OA