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# Oxidative Stress, Endoplasmic Reticulum and Mitochondrial Stress and Senescence

Guest Editors:

**Prof. Dr. Han Moshage**
Department of Gastroenterology and Hepatology, University of Groningen, Groningen, The Netherlands

**Prof. Dr. Amalia Dolga**
Department of Molecular Pharmacology, University of Groningen, Groningen, The Netherlands

Deadline for manuscript submissions:
**closed (15 December 2021)**

## Message from the Guest Editors

Aging leads to increased exposure to oxidative stress. Aging mitochondria and ER are more susceptible to oxidative stress but also generate more pro-oxidants. Oxidative DNA damage may induce senescence. Taken together, oxidative stress and senescence are intricately connected. Oxidative stress induces aging/senescence, and aging induces oxidative stress. Since mitochondria and ER are both important sources and targets of intracellular oxidative stress, the key to understanding the interaction between oxidative stress and aging/senescence is in understanding the role of ER and mitochondria in aging/senescence.

In this Special Issue of *Antioxidants*, we invite you to present your latest research or a review on this topic. We welcome submissions on any topic at the intersection of oxidative stress, aging/senescence, and ER/mitochondrial stress. In addition, we also welcome contributions addressing the prevention of and intervention in the detrimental effects of oxidative stress and aging/senescence on organellar function.

We look forward to your contribution and are open to discussing your suggestions.

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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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*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