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Mitochondrial Redox Regulations

Guest Editor:

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Message from the Guest Editor

Mitochondria are recognized as a metabolic hub, but also as the redox hub, controlling cell fate and emanating superoxide/H2O2, which in a regulated form can be regarded as redox signaling. Prominent receivers of the redox signal are extracellular matrix metaloproteinases, redox-sensitive channels and, upon hypoxia, also inhibitable proline hydroxylase domain enzymes (PHD/EglN, plus FIH), leading to HIF-1α accumulation and resulting transcriptome reprogramming. An opposite redox signaling direction is represented by the external redox signaling from the cell towards the mitochondrion, including H2O2 activation of kinases within the intracristal space, likewise leading to, e.g., sulfenylation of critical cysteines in mitochondrial proteins.

Finally, intra-mitochondrial redox signaling exists just within the interior of the outer mitochondrial membrane, exemplified by acetylation of MnSOD making it inactive, or by the H2O2-activated phospholipase iPLA2γ; and by a plethora of situations of elevated superoxide leading to the initiation of apoptosis. Coverage of these topics is welcome for this Special Issue, including both reviews and experimental articles.

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Editor-in-Chief

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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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