Special Issue

Oxidative Stress, Mitochondrial Dysfunction, and Inflammation in Autism

Message from the Guest Editor

Autism or autism spectrum disorder (ASD) is a neurodevelopmental disorder. Although the exact cause of autism is unknown, several studies suggest that oxidative stress, mitochondrial dysfunction, immune abnormalities, inflammation, and genetic and environmental factors are involved in the pathophysiology and development of autism. Accumulating evidence suggests that oxidative stress and mitochondrial dysfunction may provide a link between susceptibility genes and pre- and post-natal environmental risk factors in autism. Oxidative stress occurs when the levels of free radicals, i.e. reactive oxygen species (ROS) exceed the antioxidant capacity of a cell. ROS are generated endogenously during oxidative metabolism and energy production by mitochondria and are neutralized by antioxidant defense mechanisms. Many studies have also implicated oxidative stress as a major upstream component in the signaling cascade involved in activation of redox-sensitive transcription factors and pro-inflammatory gene expression leading to an inflammatory response. I invite you to submit original research reports and reviews to this Special Issue.

Guest Editor

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Deadline for manuscript submissions

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About the Journal

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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