Special Issue

Oxidative Stress and Alzheimer's Disease

Message from the Guest Editor

Oxidative stress (OS) occurs through the generation of reactive oxygen species (ROS), causing damage to lipids, proteins, and DNA. Antioxidants function as ROS scavengers, limiting the damage produced by excess ROS. Lipid peroxidation and antioxidant depletion were historically discovered in blood from Alzheimer's disease (AD) patients, raising the possibility that OS mechanisms contribute to AD pathogenesis and progression. As antioxidant depletion exacerbates OS, it is likely that the prodromal "silent" period of AD involves antioxidant depletion, shifting the redox balance subtly over years through the oxidation of ion channels, leading to the early stages of AD. There is also an interplay between exogenous and endogenous antioxidants, whereby antioxidant defenses are triggered when the redox balance is disrupted. However, the mobilization of antioxidant defenses and DNA repair mechanisms cannot reverse AD progression, ultimately leading to the homeostatic collapse of antioxidant defenses in AD. Finally, the effectiveness of extent antioxidant supplementation has mixed results in the literature.

Guest Editor

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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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