



Oxidative Stress in Age-Related Diseases

Guest Editor:

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Message from the Guest Editor

The understanding of the role of free radicals in aging has undergone significant revisions. Traditionally, the mitochondrial free radical theory of aging (FRTA), proposed by Harman in 1965, implicated reactive oxygen species (ROS) as the primary culprits in cellular aging due to their production exceeding the body's antioxidant defenses. However, studies in various animal models have revealed no consistent correlation between ROS levels and lifespan, prompting a paradigm shift in our comprehension of ROS functions. It is now recognized that ROS play a pivotal signaling role, activating compensatory homeostatic responses within cells. During aging, elevated ROS levels initially trigger these protective mechanisms, but beyond a certain threshold, they contribute to cellular damage and exacerbate age-related pathologies. Experimental data have demonstrated that oxidative damage correlates more closely with frailty than with chronological age, leading to the formulation of the free radical theory of frailty. This Special Issue explores the interplay between redox modulation and age-related diseases, with a particular focus on frailty.





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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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