



Oxidative Stress in Osteoclasts

Guest Editor:

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Message from the Guest Editor

Oxidative stress develops as a result of an imbalance in redox reactions, leading to the overproduction of reactive oxygen species (ROS) and/or to an impairment of antioxidant systems. Excessive ROS damage the DNA, proteins, and lipids, compromising cellular functions. There is emerging evidence that oxidative stress due to aging or inflammation can affect bone homeostasis as well as other metabolic processes. Osteoclasts originated from bone marrow macrophages play a critical role in excessive bone resorption during bone loss. ROS have been reported to affect the differentiation and function of osteoclasts, but how this occurs is not well known yet. The aim of this Special Issue is to broaden our understanding of the biochemical, cellular, and molecular mechanisms modulated by oxidative stress in bone homeostasis. We highly encourage authors to submit studies that identify target molecules and mechanisms regulated by ROS that could have clinical implications for therapy efficacy.





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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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