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Redox Regulation in Alcoholic Liver Disease

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Message from the Guest Editors

Oxygen is a double-edged sword for life. It's a key element for optimal energy production but also a toxic compound, which could cause oxidative damage and lead to cell death. As a defense against the toxic effects of oxygen, several enzymatic pathways (e.g, superoxide dismutases) developed. Together with other non-enzymatic compounds, such as urate and reduced glutathione, and with enzymes involved in the maintenance of cell function and DNA repair during oxidative injury, antioxidants are usually in homeostatic balance with oxidative damage. However, chronic exposure to ethanol may alter this delicate equilibrium, leading to accumulation of ROS and to ongoing diseases, such as alcoholic liver disease. This Special Issue's goal is to explore the mechanisms and consequences of altered redox equilibrium in alcoholic liver disease, including but not limited to the acetaldehyde/advanced glycation end-products on oxidative damage, the relation of epigenetic changes induced by alcoholism.

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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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