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## Oxidative Stress in Human Health and Disease

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### Message from the Collection Editors

Dear Colleagues,

Upon oxidative stress, free radicals of oxygen can cause damage to cells, cell organelles, and components, such as lipids, proteins, and DNA. This damage, if not repaired, can lead to cell death. Oxidative changes play a role in the pathogenesis of many diseases, such as diabetes, cardiovascular and neurodegenerative diseases, but also autoimmune processes and cancer, to name a few. To counteract this potential threat of oxidative stress, eukaryotic cells have developed protective systems. These oxidation protection systems have in common that they can prevent or delay oxidative damage and thus help to maintain the balance between the malignant or healthy stage in various diseases. Thus, the oxidative system plays a major role in both the physiology and pathology of various diseases.

This Special Issue entitled “Oxidative Stress in Human Health and Disease” welcomes manuscripts (original papers as well as comprehensive reviews) providing insights into mechanisms of oxidative stress development leading to pathophysiology as well as physiological mechanisms to overcome these harmful events.



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*Cells* has become a solid international scientific journal that is now indexed on SCIE and in other databases. We have successfully introduced a special issues format so that these issues serve as mini-forums in specific areas of cell science. *Cells* encourages researchers to suggest new special issues, serve as special issues editors, and volunteer to be reviewers. Our main focus will remain on cell anatomy and physiology, the structure and function of organelles, cell adhesion and motility, and the regulation of intracellular signaling, growth, differentiation, and aging. We are open to both original research papers and reviews.

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