Special Issue

Oxidative Stress in Human Diseases

Message from the Guest Editor

Increasing scientific evidence confirms that the combination of reactive oxygen species (ROS) overproduction, oxidative stress (OS), and hyperinflammation can cause endothelial layer damage, which eventually leads to endothelial dysfunction. Oxidative stress occurs due to the imbalance between the production of ROS and the availability of antioxidants or radical scavengers. The excess ROS produced can either oxidize biomolecules or structurally modify lipids, proteins, and genes to trigger signaling cascades, leading to the onset and progression of inflammatory diseases. Inflammation causes immune cells to secrete various cytokines and chemokines to recruit other immune cells to the site of oxidative stress/infection. Reflexively, enhanced ROS generation by immune cells at the site of inflammation causes oxidative stress and tissue injury. The inflammatory-initiated endothelial dysfunction can promote chronic inflammation, thrombosis, atherosclerosis, and lung injury. The generation of a large amount of mitochondrial reactive oxygen species (mtROS) and their excess causes OS, which can promote inflammation and cause chronic endothelial dysfunction.

Guest Editor

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Deadline for manuscript submissions

20 January 2026

International Journal of Molecular Sciences

an Open Access Journal by MDPI

Impact Factor 4.9
CiteScore 9.0
Indexed in PubMed

mdpi.com/si/227327

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The International Journal of Molecular Sciences (*IJMS*, ISSN 1422-0067) is an open access journal, which was established in 2000. The journal aims to provide a forum for scholarly research on a range of topics, including biochemistry, molecular and cell biology, molecular biophysics, molecular medicine, and all aspects of molecular research in chemistry. *IJMS* publishes both original research and review articles, and regularly publishes special issues to highlight advances at the cutting edge of research. We invite you to read recent articles published in *IJMS* and consider publishing your next paper with us.

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