Special Issue

Nitric Oxide and Endothelial Dysfunction: Is eNOS an Innocent Bystander or a Culprit?

Message from the Guest Editor

NO is a diatomic, amphipathic, free-radical gaseous mediator with a very short half-life whose availability depends on the timed activation of dedicated enzymes (NO synthases). In vessels, the impaired production of NO by the endothelial NO synthase (eNOS) isoform leads to endothelial dysfunction—a condition that precedes and promotes cardiovascular damage. Physiological production of NO by eNOS depends on multiple interrelated steps involving compartmentalization, dimerization, and intracellular Ca2+ and phosphorylation-mediated mechanisms, in the presence of sufficient substrates and specific co-factors. Understanding the dynamic interchange between multiple players that modulate the eNOS activity is fundamental to unravelling novel mechanisms and identifying potential pharmacological targets for the treatment and prevention of several cardiovascular disturbances. This Special Issue is open to all investigations exploring the molecular components of this pathway, or aiming at elucidating alternative targets for this pathway, in order to clarify their impact in experimental models and provide preliminary findings to clinical translation.

Guest Editor

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Deadline for manuscript submissions

closed (31 July 2022)

Antioxidants

an Open Access Journal by MDPI

Impact Factor 6.6
CiteScore 12.4
Indexed in PubMed

mdpi.com/si/87899

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About the Journal

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.

Editor-in-Chief

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