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Endothelial Inflammation and Cardiovascular Dysfunction: Oxidative, Nitrosative and Reticulum Stress

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Message from the Guest Editors

Endothelial dysfunction is a hallmark of, and the link to, several cardiovascular diseases. At the outset, in considering the mechanisms of endothelial dysfunction and activation, excessive generation of reactive oxygen (ROS) and nitrogen species (RNS) is a key protagonist. ROS and RNS overproduction by a set of enzymes from nitric oxide (NO) synthase and NADPH oxidase families, among others, are commonly involved in cellular damage and CVD development.

This Research Topic postulates a primary role for endothelial cell dysfunction in inciting CVD and the underlying stress in cardiomyocytes and leukocytes to recapitulate the range of features ultimately found in cardiovascular pathologies. Even our understanding of the initiation factors that preceded CVD, drivers, such as hyperglycemia, hypertension, dyslipidemia, and SARS-CoV-2 infection, as well as their association during this process, is still limited. The aim of this Topical Collection of *Biomolecules* is to unravel and highlight fundamental pathophysiological mechanisms and evidence-based therapies in CVD.

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Message from the Editorial Board

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