



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

Guest Editors:

**Dr. Amarylis Claudine Bonito
A. Wanschel**

Department of Clinical Sciences
and Pharmacy, High Point
University, High Point, NC, USA

**Dr. Ana Iochabel Soares
Moretti**

State Government Employee
Medical Assistance Institute
(IAMSPE), Health Sciences
Program, Sao Paulo, SP 04029-
000, Brazil

**Dr. Alessandro Gonzalez
Salerno**

Interdisciplinary Stem Cell
Institute (ISCI), Miller School of
Medicine, University of Miami,
Miami, FL 33136, USA

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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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Editors-in-Chief

Prof. Dr. Peter E. Nielsen

Department of Cellular and
Molecular Medicine, Faculty of
Health and Medical Sciences,
University of Copenhagen,
Blegdamsvej 3C, DK-2200
Copenhagen, Denmark

Prof. Dr. Lukasz Kurgan

Department of Computer
Science, Virginia Commonwealth
University, Richmond, VA 23284,
USA

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Biomolecules Editorial Office
MDPI, Grosspeteranlage 5
4052 Basel, Switzerland

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