Journal of Diabetes Research

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Special Issue on

Endothelial Dysfunction in Type 2 Diabetes Mellitus: The Role of the Triad Nitric Oxide/Arginase/Oxidative Stress

CALL FOR PAPERS

Type 2 diabetes mellitus is an essentially metabolic disease characterized by different degrees of impairment in glucose homeostasis. Primarily manifested as an expression of peripheral resistance to insulin actions, the progression of the disease may switch its initial pathophysiology to an absolute lack of endogenous insulin, thus requiring the administration of exogenous insulin as an attempt to restore the metabolic balance, if its carriers live long enough to witness such change. In this scenario, the spectrum of organic damage in such clinical entity ranges from microvascular complications, already identified in the initial stages of the disease, when completely silent because of being predominantly asymptomatic, to macrovascular impairment. Of note, irrespective of the vascular bed affected, all of the compromised vessels seem to share a unifying pathophysiological mechanism, represented by endothelial dysfunction.

Concerning this metabolic web of interconnections, endothelial cells, via its molecular mediators—including nitric oxide, $^{\bullet}$NO—and the related enzymes, such as arginase and nitric oxide synthase, play a critical role in the regulation of vascular structure and function. In addition, the harmonic crosstalk between oxidant agents and antioxidants contributes to the maintenance of vascular health, the so-called redox balance. Nevertheless, the sustained imbalance between these opposite forces, either by increase in prooxidants or by a decrease in antioxidants, results in oxidative stress, a process that may be even cause or consequence of disturbances in glucose metabolism.

Finally, considering the multiple ways through which hyperglycaemia/oxidative stress may imply deleterious effects on vascular function, the in-depth paths responsible for the regulation of redox balance/glucose homeostasis are yet to be identified. In this complex and intriguingly scenario, the study of the metabolic connections that turn the triad $^{\bullet}$NO/arginase/oxidative stress into an almost single entity will undoubtedly allow better understanding of the underlying mechanisms that account for the pathophysiology of the oxidative stress and endothelial dysfunction in type 2 diabetes mellitus, possibly broadening knowledge towards novel potential therapeutic targets.

We invite investigators in basic science and clinical research to contribute with review articles and original papers describing recent advances in endothelial dysfunction in type 2 diabetes, as well as the role of the triad $^{\bullet}$NO/arginase/oxidative stress in the pathogenesis of type 2 diabetes.

Potential topics include but are not limited to the following:

- Role of endothelial dysfunction in type 2 diabetes
- Endothelial dysfunction and the NO/arginase/oxidative stress axis in the mechanistic-based therapeutic approach, translational medicine, and potential novel treatments in type 2 diabetes
- Mitochondrial dysfunction and disruption of redox signalling in type 2 diabetes
- Identification of oxidative and nitrosative products as biomarkers of type 2 diabetes
- New biomarkers of endothelial dysfunction as predictors of cardiovascular diseases risk

Authors can submit their manuscripts through the Manuscript Tracking System at http://mts.hindawi.com/submit/journals/jdr/noar/.

Lead Guest Editor
Luiza A. Rabelo, Delbrück Center for Molecular Medicine, Berlin, Germany
luizaa.rabelo@gmail.com

Guest Editors
Ana P. Davel, University of Campinas, Campinas, Brazil
anadavel@unicamp.br

Luciana V. Rossoni, Universidade de São Paulo, São Paulo, Brazil
lrossoni@icb.usp.br

Natalia Alenina, Max Delbrück Center for Molecular Medicine, Berlin, Germany
alenina@mdc-berlin.de

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