Mediators of Inflammation

Hindawi

Special Issue on

Novel Approaches in Diagnosing the Role of Inflammation in the Onset Cardiovascular Disorders

CALL FOR PAPERS

New mechanisms in the pathology of cardiovascular diseases have shifted the focus to the inflammatory response and its regulation. Advances in molecular medicine have enabled us to identify the crucial regulators of inflammation involved in cell survival or death in the myocardium, endothelial cells, and brain resulting in the development of novel strategies for treatment of coronary artery disease, hypertension, and congestive heart failure. Nevertheless, further studies are required in order to establish an optimal strategy for prevention of the disease progression at its early stages to limit the extent of early inflammatory response leading to the onset and progression of atherosclerosis.

Endothelial dysfunction (ED) occurring with an overproduction of mediators of inflammation plays a key role in the pathogenesis of cardiovascular diseases. Since recovery of appropriate endothelial reactivity follows the cyclooxygenase inhibiting and normalizing the nitric oxide (NO) bioavailability, drugs modifying these pathways should provide an opportunity for a better prevention of major cardiovascular complications.

The childhood malignancies survivors and subjects with connective tissue inflammatory diseases are at higher cardiovascular risk than general population. Persistent ED may be linked to an imbalance between molecules involved in NO synthesis like L-arginine and asymmetric dimethylarginine (ADMA). On the other hand, there are some reports stating that increased inflammatory response is present in malignancies at the time of diagnosis, which indicates that ED may be present before the treatment begins.

Age is closely associated with the progression of changes in the cardiovascular system, particularly in the vascular wall. Besides alternating the autonomic nervous system activity, these ageing-induced changes may prime in the endothelium, myocytes, or vessel wall intercellular matrix and be closely related to inflammatory response. With age, the acceleration of endothelial cells turnover and apoptosis mechanisms intensification occurs. These phenomena may also be exacerbated by concomitant cardiovascular risk factors. However, the exact molecular mechanisms responsible for the aging-induced development of cardiovascular disorders remain still poorly understood.

We invite authors to contribute original research articles as well as review articles that will illustrate and stimulate the continuing effort to understand the molecular mechanisms that prime the pathogenesis of cardiovascular disorders including coronary artery disease, congestive heart failure, stroke, and arterial and pulmonary hypertension.

Potential topics include but are not limited to the following:

- Multiomic (proteomic, genomic, and metabolomic) approach in defining pathophysiology of cardiovascular disorders with respect to inflammation
- Regulation of inflammation by the renin-angiotensin-aldosterone system
- Imaging diagnostics in cardiovascular disease, early diagnosis of consequences of inflammation
- Regulation of endothelial and platelet function with respect to inflammatory response
- Nonpharmacological modulation of inflammation:
  - The use of low level laser therapy in management inflammatory reactions in endothelium and platelets—molecular background
  - Modulation in inflammation using the low- and high-temperature exposure—molecular background

Authors can submit their manuscripts through the Manuscript Tracking System at https://mts.hindawi.com/submit/journals/mi/cddd/.

Papers are published upon acceptance, regardless of the Special Issue publication date.

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