Oxidative Medicine and Cellular Longevity

Hindawi

Special Issue on

New Insights into the Role of Oxidative Stress in Onset of Cardiovascular Disease

CALL FOR PAPERS

Myocardial infarction (MI) and congestive heart failure are still among the most common reasons of mortality and morbidity in the world population. Advances in molecular medicine have enabled us to identify the critical pathways involved in cell survival or death in the myocardium. Furthermore, crucial regulators of these pathways have been identified resulting in the development of novel therapeutic strategies. Nevertheless, further studies are required in order to establish an optimal strategy for maintenance of the cardiomyocyte longevity.

Proteins play a key role in regulation of biological systems. Regulation of their expression and action might be obtained by the interplay of multiple posttranslational modifications (PTMs), including phosphorylation, nitration, and by regulation of their degradation by oxidative stress. Additionally, the signaling pathways leading to these modifications can cross-talk to provide a further level of regulation. Pharmacoproteomic approach gives an in-depth insight into changes in protein expression profile as well as in PTMs in response to pathological process and therapeutic intervention. Therefore, explaining pathophysiological mechanisms allows defining novel pharmacological targets as well as better markers of cardiac damage, which could reflect the changes in homeostasis.

Acute oxidative stress triggers PTMs and regulates degradation of numerous proteins. Peroxynitrite ($ONOO^-$) is a highly reactive oxidant which is generated from the coupling between nitric oxide and superoxide. Its detrimental action on the development of cardiac injury as well as on cardiac systolic function has been well established. Nonetheless, the exact mechanisms by which $ONOO^-$ modulates cardiac injury and promotes systolic dysfunction remain to be explained.

Myosin is the key protein of the cardiac contractile machinery, and its molecule consists of two heavy chains and two types of light chains, two essential light chains (MLC1) and two regulatory light chains (MLC2). Both play important structural and functional roles and therefore any changes in their structure/stability under pathological conditions may result in serious compromising of cardiac performance. There is evidence that MLC1 is modified during oxidative stress. Also, a myosin light chain kinase- (MLCK-) dependent phosphorylation of MLC1 has been documented.

Cardiac hypertrophy (CH) is associated with an increase in cardiomyocyte volume that occurs in response to various pathophysiological stimuli, such as hypertension, valvular disorders, infectious agents, or mutations in sarcomeric genes. CH presents as a complex of cardiac remodelling characterized by foetal gene reactivation, interstitial fibrosis, myocyte apoptosis, and leading to impairment of cardiac systolic and diastolic function. The underlying molecular mechanisms which couple hypertrophic signals initiated at the cell membrane to the reprogramming of cardiomyocyte gene expression remain poorly understood. Therefore, an elucidation of these mechanisms is a central issue and is critical for designing new strategies for prevention and treatment of CH. A number of intracellular signaling pathways have been implicated in transduction of hypertrophic signals. Activation of cell surface receptors for Angiotensin II (Ang II), Phenylephrine, and Endothelin-1 (ET-1) leads to activation of phospholipase C (PLC) pathway. There is also evidence that the mitogen activated protein kinase (MAPK) pathways are transducers of hypertrophic signals; however, the extent to which they are coordinated during cardiac hypertrophy is unknown. Ang II promotes hypertrophy involving multiple signal transduction pathways, such as tyrosine kinases and MAPKs. Furthermore, hypertrophic effect of Ang II is shown to be abolished by use of calcineurin inhibitor, pointing thus at calcineurin-NFAT pathway as a downstream Ang II signaling.

Endothelial cells play a crucial role in maintaining vascular tone and structure, and all the disturbances initiating and promoting progression of atherosclerosis come from these cells. The potential mechanisms underlying these phenomena include decreased nitric oxide bioavailability, hyperaldosteronism, and recurrent changes in the tissue redox potential. Nevertheless, novel diagnostic and therapeutic conceptions related to the known molecular background are still missing.

We invite authors to contribute original research articles as well as review articles that will define novel therapeutic targets and allow providing novel molecular background for diagnostic and therapeutic conceptions supporting significantly the ones currently recommended in clinical practice. The manuscripts published in this issue will provide new insights into the molecular mechanisms of a broad variety of heart pathologies. Additionally, we believe that some of the presented studies will provide new evidence, which could lead to the discovery of potential drug targets for the development of new therapeutic approaches for combating cardiovascular disease in the future.

Potential topics include but are not limited to the following:

- Early risk stratification—new biochemical parameters and imaging diagnostic markers: how to prevent effectively an avalanche of cardiovascular disasters?
- Proteomic approach for studying platelet function and dysfunction: use of proteomics for defining novel oxidative stress-induced functional changes in platelets
- Recurrent changes in oxygen supply and in the tissue redox potential (hypoxia-reoxygenation and ischemia-reperfusion) as a background for cardiovascular negative remodelling
  - PTMs in sarcomeric proteins induced by oxidative stress and their causative role in developing heart failure
  - Endocrine disturbances and oxidative stress induced changes in cardiovascular system (effect on endothelial cells and cardiac myocytes)
  - Oxidative stress and viability of cardiac myocytes
- Novel aspects of subclinical hyperaldosteronism and a role of aldosterone in cardiovascular remodelling
  - MAPK pathways as transducers of hypertrophic and antiapoptotic signals in cardiac myocytes and endothelium
  - Calcineurin-NFAT pathway as therapeutic target preventing cardiac hypertrophy
  - Kinase inhibitors as novel strategies to reduce the impact of cardiovascular disease
- The Janus face of nitric oxide in cardiovascular physiology and pathology
- Nonpharmacological modulation of oxidative stress
  - The use of low level laser therapy in management of endothelium and platelets—molecular background
  - Molecular mechanisms underlying beneficial effects of regular aerobic activity

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