



Myocardial Ischaemia–Reperfusion Injury

Guest Editor



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Message from the Guest Editor

Dear Colleagues,

Despite percutaneous coronary intervention is mandatory in the therapeutic approach to myocardial infarction (MI), revascularization inflicts heart injury (IRI) due to irreversible microvascular damage and systolic dysfunction. The final consequence of this event is an increased morbidity and mortality. Going further, myocardial no - reflow can still occur and is associated with a worse in - hospital and long - term prognosis, since it refers to a state of myocardium hypoperfusion and microcirculatory dysfunction. Hence, one of the challenges nowadays is to better detect, prevent and treat extended IRI.

A significant factor contributing to IRI is an increased oxidative stress and excessive production of reactive oxygen (ROS), nitrogen (RNS) and sulfur (RSS) species. It leads to increased activation of proteolytic enzymes, contractile proteins degradation, damage of mitochondrial DNA and cell apoptosis. However, the detailed molecular mechanism for myocardial IRI is not fully clear.

Although interventional cardiologists can effectively treat acute epicardial artery occlusion, myocardial damage, as a result of microcirculation impairment, remains a high problem. Endothelial and smooth muscle cells dysfunction, luminal obstruction, and inflammation have been reported in this setting. Numerous efforts have been made to optimize microcirculation impairment during acute coronary syndrome, and some of them reported reduction of infarct size, increased ejection fraction and reduced need for implantable cardioverter-defibrillator implantation, however most of them revealed limited of clinical improvement. Thereby this dictate the need to test new drugs in this area.

The main aim of this Special Issue is to explore aspects of the modulation of myocardial ischemia-reperfusion injury using an interdisciplinary approach involving chemical, biological, physiological, pharmaceutical, pharmacological, and physicochemical perspectives. We are particularly interested in studies addressing, but are not limited to, the following:



- myocardial ischemia reperfusion injury- from bench to bedside,
- new redox-based molecular mechanisms contributing to cardiac ischemia-reperfusion injury,
- exploring the naturally occurring compounds, standard medications, and nutraceuticals in modulating redox-signaling pathways and limiting and/or preventing IRI,
- mechanisms of redox regulation in myocardial ischemia-reperfusion injury,
- novel cellular and molecular mechanisms contributing to IRI,
- molecular mechanisms and clinical significance of post-translational modifications with impact on cellular redox homeostasis disturbances,
- antioxidants in oxidative stress,
- mitochondrial malfunction in IRI,
- new molecular mechanism of IRI,
- novel compounds/therapeutic approaches aiming to prevent and/or reduce cardiac damage during ischemia and/or reperfusion,
- promising therapeutic strategies aiming to enhance endogenous antioxidant capacity to combat oxidative stress,
- new relevant biological markers for assessing/monitoring in vivo myocardial injury with clinical efficacy.

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