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Ischemia-Reperfusion Injury: A Mechanistic Concept

Rajiv Kumar*

Department of Chemistry, NIET, National Institute of Medical Science, Delhi, India

About the Study

Ischemia-reperfusion injury (heart, liver, pancreas, kidney, intestine) allies with numerous clinical manifestations including acute heart failure, cerebral dysfunction, myocardial hibernation, systemic inflammatory response syndrome, multiple organ dysfunction syndrome and gastrointestinal dysfunction. These injuries are a group of complications and a complex medical ailment that poses a therapeutic ultimatum for researchers. Blood deprivation during ischemia is an undesirable event of hepatic ischemia-reperfusion injury, that is monitored by the recurrence of flow all through reperfusion, comprises a multifaceted series of events, including adenosine-5'-triphosphate depletion, mitochondrial de-energization, alterations of electrolyte homeostasis, upregulation of pro-inflammatory cytokine signaling, oxidative stress changes, and Kupffer cell activation [1].

Involved pathways and numerous mediators participate altogether when Ischemia-reperfusion injury transpired and these elaborated features of the underlying mechanisms of these injuries can be a good choice to do a potential therapeutic intervention and can play a key role in the development of antioxidant therapy too [2]. Although, exposing the pathophysiology of ischemia-reperfusion injury is taught task, and especially to investigate the role of reactive oxygen species and to detect the routes of cell death pathways is a challenging aspect [3]. But, analysis of association of various conversion routes, including NADPH oxidase, xanthine oxidase system, and nitric oxide synthase is can expose the therapeutic targets also labeled (Figure 1).

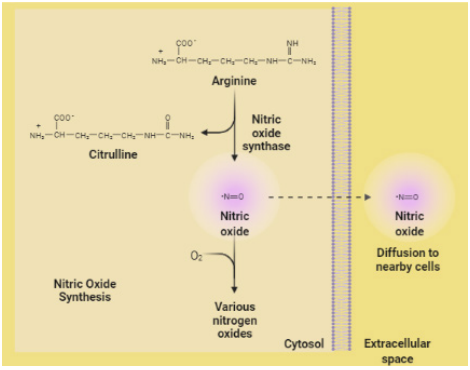

Figure 1: Illustration of association of various conversion routes, including NADPH oxidase and nitric oxide synthase.

Once prolonged ischemia ended, the blood supply must be reestablished, otherwise, an escalation will occurred in the rate of ROS production and local inflammation, and these unnatural disorders initiate secondary injury. These undesirable events triggered by prolonged ischemia-reperfusion injury and that induced cell damage and further initiate apoptosis, necrosis, necroptosis, and autophagy. Authors explain the mechanistic insights into reperfusion-injury by elaborating all the concerned details of the routes of cell death that transpired *via* these aforementioned means. These approaches will expose and elucidate the interlinked signaling pathways of cell death marked routes will be new therapeutic targets, and these types of approaches will enhance the possibilities that definitely cure ischemia-reperfusion injuries [4]. Moreover, a better understanding of the pathophysiology of ischemia-reperfusion injury will enhance the chances of successful innovation of novel treatment interventions. For example, mitochondria produce reactive oxygen species that are a key component of the mechanisms that initiate IR injury [5]. Besides it, stimulation of mitochondrial permeability transition or oxidative damage of intra-mitochondrial structures, inflammatory signaling, and extracellular remodeling, pro-apoptotic signaling, and molecules participated are other components that lead to ischemia-reperfusion injury. The impact of mitochondrial ROS on the post-infarction remodeling is also an important aspect of the mechanism underlying as per the mechanistic concept. Platelets play a key role in hemostasis and initiate the formation of thrombi in pathological surroundings. Scientific evidence suggests that platelets participate in the inflammatory progressions and initiation of acute ischemic stroke. In the end, the author believes that his view on the mechanistic concept of ischemia-reperfusion injury will offer important aspects of reactive oxygen species and will inspire the upcoming generation of researchers to find out the way to detect the routes of cell death pathways that occurred during these injuries [6].

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Availability of Data and Materials

Wherever necessary, relevant citations are included in the reference section.

Competing Interests

The author has declared that no competing interest exists.

***Corresponding author:** Kumar R, Department of Chemistry, NIET, National Institute of Medical Science, Delhi, India, E-mail: chemistry_rajiv@hotmail.com

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