IMMUNOLOGY AT THE CONFLUENCE OF MULTIDISCIPLINARY APPROACHES

ABSTRACT BOOK

Institute for Biological Research "Siniša Stanković" National Institute of Republic of Serbia

University of Belgrade

Immunological Society of Serbia

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MELDONIUM PREVENTS ACUTE ISCHEMIA/REPERFUSION INDUCED-RENAL CELLS DEATH IN RATS

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Acute renal ischemia/reperfusion (I/R) is a temporary restriction of kidney blood supply, followed by blood flow restoration and re-oxygenation. During I/R, decreased oxygen supply disturbs ion transport, intracellular ATP, calcium and pH levels, and numerous signalling pathways. Upon reperfusion, a restoration of oxygen level rises a reactive oxygen species generation, cytokines and chemokines release from activated tissue-resident macrophages, and infiltration of pro-inflammatory neutrophils into ischemic tissues. All these changes result in cell swelling and rupturing, and consequent necrotic or apoptotic cell death. Meldonium is an anti-ischemic drug clinically used to treat myocardial and cerebral ischemia, which acts by shifting energy production from fatty acid oxidation to glycolysis. We investigated the effects of a 4-week meldonium pre-treatment with 300 mg/kg b.m./day of rats subjected to a well-established experimental model of renal I/R, with ischemia lasting for 45 minutes, followed by 4 hours of reperfusion. The degree of apoptosis and necrosis was evaluated by measuring renal pro-apoptotic Bax and anti-apoptotic Bcl-2 ratio, serum and kidney levels of necrotic marker - high mobility group box 1 protein (HMGB1), together with the kidney histology analysis. Our results showed that apoptotic and necrotic cell death occur simultaneously under I/R conditions, judging by the renal Bax/Bcl2 ratio rise (2.7-fold), increase in serum (22%) and renal (30%) levels of HMGB1, as well as severe tubular necrosis with dilatation of the tubular structure, cast formation, tubular lumina dilatation, brush border reduction, and loss in some renal areas cells. Meldonium pretreatment reduced the elevated Bax/Bcl2 ratio by 35%, as well as the serum and renal HMGB1 levels by 20% and notably diminished histological evidence of renal I/R necrotic injury, especially regarding tubular structures. These findings proved that meldonium protects renal cells against I/R-induced necrosis and apoptosis.