2018

4th CONGRESS OF PHYSIOLOGICAL SCIENCES OF SERBIA WITH INTERNATIONAL PARTICIPATION

Organized by the Serbian Physiological Society

Under the auspices of

Federation of European Physiological Societies (FEPS)
International Union of Physiological Sciences (IUPS)
International Society for Pathophysiology (ISP)
International Academy of Cardiovascular Sciences (IACS)

CURRENT TRENDS IN PHYSIOLOGICAL SCIENCES: FROM CELL SIGNALS TO THE BIOLOGY OF AGING

ABSTRACT BOOK

September 19-23, 2018
Faculty of Medicine, University of Nis,
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PRETREATMENT OF DIABETIC RATS WITH ETHYL PYRUVATE UPREGULATES HMGB1-DRIVEN PROTECTIVE AUTOPHAGY IN THE LIVER

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Oxidative stress, cell damage and cell death underlies the etiology of liver damage/dysfunction in diabetes. High Mobility Group Box 1 (HMGB1) is a nuclear protein that is released from damaged/stressed liver cells during diabetes and contributes to oxidative stress–mediated autophagy and cell death/survival. Since ethyl pyruvate (EP), an HMGB1 release inhibitor, possesses anti-oxidative properties, we have examined whether the pretreatment of diabetic rats with EP, has an effect on HMGB1 release/expression and autophagy in the liver of diabetic rats. Diabetes was induced by streptozotocin (65mg/kg/ip). Pretreated group of diabetic rats (EP+D) started treatment with EP (80mg/kg/daily) three days before diabetes induction, while in the second group (D+EP) treatment started ten days after diabetes induction. In comparison with D+EP group, EP+D group had better glycemic status and higher activity of antioxidative enzymes SOD and CAT in diabetic liver. According to Western immunoblot analyses, EP+D group showed higher expression of extracellular HMGB1 in comparison with D+EP. Consequently, detected increase in HMGB1/RAGE interactions in EP+D group were followed by higher expression of LC3-II, HMGB1/Beclin 1 interaction and activation of autophagy. Expression of LC3-II and HMGB1/Beclin 1 interaction were at the control level in D+EP. Preserved liver morphology in both EP treated groups, observed by electron microscopy, implicated existence of adaptive mechanisms in EP+D group. Further analyses showed that protective autophagy (mitophagy) was enhanced in EP+D group compared with diabetic and D+EP groups. Thus, treatment of diabetic patients with EP may constitute a new strategy for the treatment of diabetes-related tissue injury.

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