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Effects of erythropoietin preconditioning on rat cerebral ischemia-reperfusion injury and the GLT-1/GLAST pathway

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We aim to explore whether erythropoietin (EPO) preconditioning protects against rat cerebral ischemia-reperfusion injury and affects the expression of glutamate transporter 1 (GLT-1) and glutamate aspartate transporter (GLAST). One hundred and forty Sprague Dawley rats were randomly divided into four groups: DMSO-sham, EPO-sham, DMSO-MCAO and EPO-MCAO. Neurological function scores were obtained 24 h after reperfusion. Cerebral infarction volume and the number of apoptotic neural cells of each group were measured. The mRNA levels of GLT-1 and GLAST were determined by reverse transcription-polymerase chain reaction (RT-PCR), while the protein levels of GLT-1 and GLAST were determined by Western blot assay. The cerebral infarction volume was significantly greater in DMSO-MCAO group compared with DMSO-sham group ($P<0.01$). The infarction volume of EPO-DMSO group, however, was significantly smaller than that of DMSO-MCAO group ($P<0.01$). Compared with DMSO-sham group, there were more apoptotic cells in DMSO-MCAO group ($P<0.01$). But the number of apoptotic cells in EPO-MCAO group was significantly less than that in DMSO-MCAO group ($P<0.01$). The mRNA and protein levels of GLT-1 and GLAST decreased significantly 24 h after the cerebral ischemia-reperfusion ($P<0.01$) compared with the DMSO-sham group, whereas their levels increased significantly in the EPO-MCAO group ($P<0.01$). EPO preconditioning protect against cerebral ischemia-reperfusion injury and up-regulated the expression of GLT-1 and GLAST.

Biography

Daihua Yu has completed his PhD at the age of 35 years from the Fourth Military Medical University. He is the Associate Director of Department of Anesthesiology, Tangdu Hospital. He has published more than 30 papers in reputed journals and has been serving as an editorial board member of repute. His studies mainly focus on using preconditioning and postconditioning for treatment of human brain ischemic disease, via mechanisms of immunomodulation, neural cell repopulation and neuroregeneration.

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