Hippocampal response to ischemic preconditioning: gene expression of Ca^{2+} ATPase (SPCA) and ERK pathways after cerebral ischemia/reperfusion in rats

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Stroke is the third leading cause of death in humans. Ischemic preconditioning (IPC) is phenomenon of CNS adaptation which results in increased tolerance. The Golgi apparatus, as a part of secretory pathways (SP), is Ca^{2+} store with secretory Ca^{2+}ATPases (SPCA1). SP are involved in stress sensing and transduction of apoptotic signals. ERK protein is part of the mechanisms leading to survival of neurons. We have determined the effect of ICP on alterations of mRNA and protein levels of SPCA1 and level of ERK and pERK in hippocampus. RT-PCR and Western blot analysis detected expression of SPCA1 gene in injured area after ischemia/reperfusion. In injured area, an increase of mRNA was maximal in the reperfusion period. IPC did not change significantly the expression profile, however the tissue response was elevated. Similar pattern was observed on the translational level by Western blot analysis. Protein level of SPCA1 and pERK was highest in the reperfusion time and IPC initiated elevation of tissue response. In addition, both the *in vitro* oxidative stress and IRI induced lipo- and protein oxidation as well as depression of SPCA activity which were partially reversed by IPC [1].

[1] Pavlíková et al. (2009) Cell. Mol. Neurobiol. 29, 909-916

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