Neuroprotective effect of diphenyl diselendide in acute experimental stroke: Relationship between mitochondrial damage and the expression of apoptotic genes


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INTRODUCTION: Acute ischemic stroke is a major risk for morbidity and mortality in our aging population. Mitochondria have been the focus of a vast amount of stroke-related research. Here we investigate if treatment or pre-treatment with Diphenyl diselenide (PhSe)2, can avoid mitochondrial damage in the hippocampus and cerebral cortex of rats damage caused by an ischemia and reperfusion (I/R) model. MATERIALS/METHODS: Focal cerebral ischemia was induced in rats by occlusion of the left common carotid arteries using clamp. Adult male rats were assigned into five groups: sham operation, I/R, (PhSe)2 pre-treated + I/R, (PhSe)2 treated + I/R and (PhSe)2 alone group. Measurement of mitochondrial swelling and expression of pro-apoptotic proteins and HSP70 in hippocampus and cerebral cortex was performed. RESULTS/DISCUSSION: Hippocampus and cortex mitochondrial that suffered I/R were swelling. However, (PhSe)2 pre-treated and treated groups can avoid this effect in mitochondria of brain regions. The ischemic damage did not show an increase in expression of pro-apoptotic proteins, suggest that acute model of I/R the neurons appear to be moving toward a more pronounced death process, but this short time of 4h of reperfusion could be not able to detect significant differences. Otherwise the analysis of Hsp70 gene expression in the I/R group were greater than those in sham rats, though this increase cannot be observed in groups treated with (PhSe)2. The HSP induction following ischemia appears to be an attempt to protect neurons against an established damage. (PhSe)2 could probably prevents this overproduction and further the activation of the Hsp70 promoter by decreasing the stress impinged by the ischemic insult on the neurons. CONCLUSION: (PhSe)2 produced a neuroprotective action in the of rats subjected to I/R. It’s properties are very interesting and provide the development of novel therapy against I/R in order to prevent morbidity and deaths.

Palavra chave: selenium compound, Hsp70, ischemia/reperfusion, mitochondria.
Patrocínio: CNPq, CAPES and FAPERGS