Diphenyl Diselenide Prevents the Damage Caused by Quinolinic Acid in Hippocampus and Striatum of Adult Rats

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Neurotoxic action have been attributed to quinolinic acid (QA; 2,3-pyridine dicarboxylic acid), a major component of the kynurenine pathway of tryptophan metabolism. QA is shown to be an endogenous glutamate agonist with a relative selectivity to the N-methyl-D-aspartate (NMDA) receptor, causing neuronal death in vitro and in vivo. One putative mechanism may involve free radicals. In this way, an antioxidant supplement may prevent from the QA toxicity. In vitro and ex vivo studies have demonstrated that diphenyl diselenide ($(PhSe)_2$) is a potential antioxidant compound. Additionally, $(PhSe)_2$ has a protective role in a variety of experimental models associated with the overproduction of free radicals in brain and liver and has been reported a hepato-protective effect in diabetic rats. Thus, the purpose of the present study was to investigate whether $(PhSe)_2$ may protect from QA-induced damage in cortex, hippocampus and striatum slices of adult rats. Cellular viability, demonstrated by decrease in mitochondrial viability (assessed by MTT method) was reduced by QA (1mM) in all brain structures. QA-induced damage was significantly decreased by $(PhSe)_2$ (1 and 10 µM) in hippocampus and striatum (0,05 µM) slices, but not in cortex. In conclusion, the results presented in this study revealed that $(PhSe)_2$ showed a good protection, and it is believed to be due its antioxidant ability against free radical production, in agreement with other studies.

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