4- hydroxy-3,5-di-tret-butyl cinnamic acid restores the activity of the hippocampal mitochondria in rats under permanent focal cerebral ischemia

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Abstract

Background and Purpose: Ischemic stroke remains one of the leading causes of death in the population. In addition, mitochondrial dysfunction is an essential part of the pathogenesis of cerebral ischemia and is a promising pharmacotherapeutic target. Experimental Approach: the work was performed on male Wistar rats, which were simulated cerebral ischemia by irreversible occlusion of the middle cerebral artery. 4-hydroxy-3,5-di-tret-butyl cinnamic acid (25 mg/kg, 50 mg/kg and 100 mg/kg) was injected intraperitoneally for 3 days after ischemia (daily). On the 4th day of the experiment, the changes of rat's cognitive functions in the Morris water maze test, cellular respiration processes, the activity of the mitochondrial respiratory chain complexes and citrate synthase activity, the intensity of oxidative stress and apoptosis reactions were assessed. Key Results: it was found that the administration of 4-hydroxy-3,5-di-tret-butyl cinnamic acid at doses of 25 mg/kg and 50 mg/kg practically equivalently promotes the restoration of aerobic metabolism reactions and the activity of the mitochondrial respiratory chain complexes, decrease of the intensity of oxidative stress reactions and apoptosis, as well as an increase in the activity of citrate synthase. As a result, the restoration of mitochondrial function in the hippocampal cells contributed to the restoration of the animal's spatial memory. Conclusion and Implications: a study showed that 4-hydroxy-3,5-di-tret-butyl cinnamic acid at doses of 25 mg/kg and 50 mg/kg has a neuroprotective effect on hippocampal neurons under conditions of permanent occlusion of the middle cerebral artery, realized by restoration of mitochondrial function.

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