Protective effect of citicoline against aluminum-induced cognitive impairments in rats.

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Abstract:

The potential protective effect of citicoline on aluminum chloride-induced cognitive deficits was investigated in rats. In a Morris water maze, administration of aluminum chloride to rats for 90 days resulted in increased escape latency to reach the platform and decreased swimming speed in acquisition trials. Similarly, in probe trials, the time required to reach the hidden platform was increased and the time spent in the target quadrant was reduced. Also, administration of aluminum chloride to rats for 90 days increased the reference and working memory errors and time required to end the task in the radial arm maze. In addition, this treatment decreased the step-through latency in the passive avoidance test. Concurrently, treatment of rats with aluminum chloride for 90 days increased hippocampal glutamate, malondialdehyde, and nitrite levels and decreased intracellular reduced glutathione level. In the citicoline-treated group, aluminum chloride-induced learning and memory impairments as assessed by the Morris water maze, radial arm maze, and passive avoidance tests were inhibited. At the same time, treatment of rats with citicoline prevented the biochemical alterations induced by aluminum chloride in the hippocampus. It can be concluded that elevation of hippocampal glutamate level with consequent oxidative stress and nitric oxide (NO) overproduction may play an important role in aluminum-induced cognitive impairments. Also, our results suggest, for the first time, that citicoline can protect against the development of these cognitive deficits through inhibition of aluminum-induced elevation of glutamate level, oxidative stress, and NO overproduction in the hippocampus.

Keywords:

Aluminum chloride, cognitive impairments, citicoline, hippocampus

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