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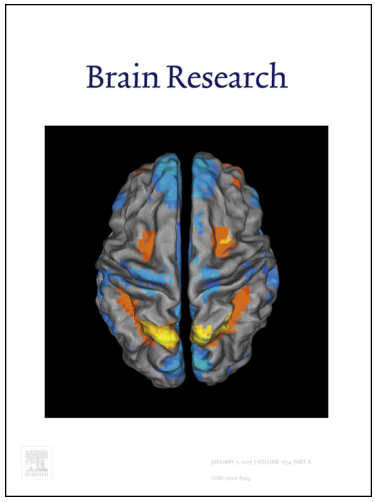

Research report

Antiepileptic effect of uridine may be caused by regulating dopamine release and receptor expression in corpus striatum

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Antiepileptic effect of uridine may be caused by regulating dopamine release and receptor expression in corpus striatum

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Abstract

Uridine is a potential endogenous neuromodulator studied for several decades for its antiepileptic effect, but the results were controversial. One remarkable feature of uridine is its regulatory action on the dopaminergic pathways. In this study, the changes in uridine and dopamine (DA) release were examined in the mouse corpus striatum after pilocarpine (PC) intraperitoneal injection. Then, the effect of uridine pre-treatment on DA release and expression of dopamine receptor (DR) was determined. The results revealed an increased uridine release initially, followed by a downward trend after an injection of 400-mg/kg PC. However, the DA release continuous increased significantly. The expression of dopamine receptor-1 (D1R) increased in a dose-dependent manner while that of dopamine receptor-2 (D2R) decreased significantly. Prophylactic administration of uridine significantly relieved the high-frequency and high-amplitude expression induced by PC as well as dose-dependently reversed the PC-induced changes in DA and DRs levels. These findings suggested that uridine produced an antiepileptic effect, which might have been mediated in part by interfering with the dopaminergic system.

Keywords: Uridine; Dopamine; Epilepsy; Corpus striatum; Microdialysis;

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