Analysis of Receptor’s Distribution in Entorhinal Cortex after Induction of Spreading Depression in Juvenile Rats

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Abstract

Spreading depression (SD), discovered by Leao in 1944, is a pathophysiological wave which propagates slowly in the brain (3 mm/min) and cause dramatic ionic and hemodynamic changes. SD appears to act through several mechanisms and receptors which have not completely understood. Here, we studied the effect of inhibitory system in animal model of SD using immunohistochemistry technique. After implanting recording electrodes and cannula over the brain, repetitive SD was induced by KCl injection (2 M) in juvenile rats for four consecutive weeks. Then all rats were decapitated and the brains removed. Mean number of dark neurons in entorhinal cortex were determined using Toluidine blue staining. To identify the prevalence and distribution of γ-aminobutyric acid A (GABA-A) subunit receptors and glutamic acid decarboxylase (GAD), immunohistochemistry technique was performed. The mean number of SD induced by KCl injection was statistically increased during four weeks of experiments ($P=0.036$). The mean number of dark neurons in entorhinal cortex was significantly increased in SD group compared to sham rats ($P\leq0.001$). Also, expression of GAD 65 receptor in the Entorhinal cortex significantly increased in SD group compare to control group ($P<0.05$). GABA-Aα and GABA-Aγ receptors didn’t show significant change in that region. These data suggest that SD is able to damage neural cells and also it could lead to enhancement of GAD, the enzyme which is responsible for synthesizing an important inhibitory neurotransmitter, GABA receptor, in the central nervous system.

Keywords: Cortical Spreading Depression, Entorhinal Cortex, Gamma-Aminobutyric Acid A.

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