



Poster Presentation

Neuroprotective Effect of Noscapine on Cerebral Oxygen-Glucose Deprivation Injury

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Abstract

The present study aims to investigate the effect of noscapine (0.5-2.5 μ M), an alkaloid from the opium poppy, on primary murine fetal cortical neurons exposed to oxygen-glucose deprivation (OGD), an in vitro model of ischemia. Cells were transferred to glucose-free DMEM (Dulbecco's Modified Eagle Medium) and were exposed to hypoxia in a small anaerobic chamber. Cell viability and nitric oxide production were evaluated by MTT assay and the Griess method, respectively. 0.5 μ M noscapine significantly inhibited the neurotoxicities produced by 30 min OGD. The neurotoxicity decreased by noscapine treatment in the concentration-dependent manner. Pretreatment of cells with MK-801 (10 μ M), a non-competitive NMDA (N-Methyl-D-aspartate) antagonist, and nimodipine (10 nM), a L-type Ca^{2+} channel blockers, increased cell viability after 30 min OGD, while the application of NBQX (30 μ M), a selective AMPA (α -amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid)-kainate receptor antagonist partially attenuated cell injury. Noscapine attenuated nitric oxide (NO) production in cortical neurons after 30 min OGD. We concluded that noscapine had a neuroprotective effect, which could be due to its interference with multiple targets in the excitotoxicity process. These effects could be mediated partially by a decrease in NO production.

Keywords: Noscapine, NO, Oxygen-Glucose Deprivation, Cortical Culture.

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