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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 μM), 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 μM noscapine were 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 μM), 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 μM), 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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