



Poster Presentation

Neuroprotective Effect of Noscapiene 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  $\mu\text{M}$ ), 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  $\mu\text{M}$  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  $\mu\text{M}$ ), a non-competitive NMDA (N-Methyl-D-aspartate) antagonist, and nimodipine (10 nM), a L-type  $\text{Ca}^{2+}$  channel blockers, increased cell viability after 30 min OGD, while the application of NBQX (30  $\mu\text{M}$ ), a selective AMPA(- $\alpha$ -amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid)-kainate receptor antagonist partially attenuated cell injury. Noscapiene 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:** Noscapiene, NO, Oxygen-Glucose Deprivation, Cortical Culture.

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