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Poster Presentation

Neuroprotective and Neurogenesis Effects of Curcumin in the Adult Rat Brain Following Transient Global Ischemia

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Abstract

Traumatic brain injury resulting road accidents create damage to the brain. The severe brain injury may cause extensive tissue loss of several parenchyma which results in cavities due to primary destruction and secondary injuries such as ischemia and inflammation. Recent findings suggest that neuronal precursors in the adult mammalian brain can be a therapeutic target in ischemic brain injuries. It has been reported that curcumin reduces oxidative stress and stimulates neurogenesis in the brain. The present study was undertaken to evaluate the neuroprotective and neurogenesis effects of curcumin in a rat model of transient global ischemia (TGI). Fourty-eight adult male Wistar rats were randomly chosen as control, sham (animals only underwent TGI), treatment (animals were treated with 100 or 300 mg/kg curcumin following TGI) and vehicle groups. 5-bromo-2-deoxyuridine was injected intra pritoneally twice daily for three consecutive days. Then, animals were decapitated for 3 and 4 weeks after treatment. Neurogenesis, cell injury and apoptosis in the hippocampus, somatosensory neocortex, subventricular as well as subgranular zone and posterior periventricular region were assessed. We found that the number of dark neurons and apoptotic cells increased after TGI. Treatment with curcumin reduced the number of dark neurons and apoptotic cells in a dose-dependent manner. In addition, application of curcumin increased neurogenesis at low concentration in comparison to control and ischemia groups while higher concentration of curcumin reduced the neurogenesis. The present investigation provides evidences supporting the neuroprotective potential of curcumin in vivo and opens a new horizon for future experiments.

Keywords: Apoptosis**,** Traumatic Brain Injury, Curcumin, Oxidative Stress, Neuroprotection.

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