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Title: Neuroprotective Effect of Black Cumin Oil against cerebrovascular hypoperfusion induced Pyramidal Cell Loss in Rats.

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Abstract: Critically attained threshold cerebrovascular hypoperfusion (CATCH) hypothesis is believed to cause age related neurodegeneration. Such threshold was successfully achieved in rats by permanent bilateral common carotid artery ligation (PBCCAL) and the earliest neurons to undergo neurodegeneration were found to be the pyramidal cells of hippocampal CA1 region. Nigella sativa oil extract (NSO) was found to preserve viability of cell in cerebellar neurons culture in vitro putting forward its potential neuroprotective effect. Therefore, the current study was conducted to perform a quantitative histopathological assessment of neuroprotective effect of NSO on CA1 hippocampal pyramidal cells on rats with CATCH induced by PBCCAL. 30 rats were equally divided into three groups: sham control (operated without PBCCAL and no treatment received), untreated PBCCAL (operated with PBCCAL only) and NSO treated group (operated with PBCCAL and received daily oral NSO treatment). After the 10th postoperative week coronal sections of the dorsal hippocampus were stained with cresyl violet stain. The number of viable pyramidal neurons in 1mm horizontal distance of CA1 area on high power field (HPF) magnification power was calculated in all sections. Viable pyramidal neurons on HPF Light Microscopy appeared normal with well demarcated cell membrane and a distinct nucleus, while non-viable cells appeared shrunken with irregular outline and dark pyknotic nucleus. The average number of viable pyramidal cells within CA1 hippocampal region was significantly higher in sham control and NSO treated groups as compared to the untreated PBCCAL group ($p<0.01$). Furthermore, the difference was not significant when comparing the number of viable pyramidal cells for sham control and NSO treated groups ($p>0.05$). It can be concluded that NSO has the potential to protect hippocampal pyramidal cells from neurodegeneration induced by PBCCAL. This fact represent the doorway to use NSO to prevent age related neurodegeneration namely Alzheimer's disease and its subsequent cognitive impairment.