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Poster Presentation

Effects of Hemin on Ca2+Influx in Neurons of C57BL/6 Mouse Brain

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Abstract

Excitotoxicity results in a significant increase in Ca2+ influx; essentially from open N-Methyl-D-aspartate receptors (NMDARs) channels that cause a secondary rise in the intracellular Ca2+ concentration. It is correlated with neuronal death induced by Ca2+ overload. Dysfunction of NMDARs is associated with excitotoxic neuronal death in neurodegenerative disorders. In this study, the effects of hemin on Ca2+ permeability in neurons of C57BL/6 mouse brain examined. Isolated from 1-dayold C57BL/6 mice, were cultured in serum-free media. Cells were maintained in growth medium at 37◦C in 95% air/5% CO2 for 2 weeks in vitro before treatment. Primary neurons were cultured in serum-free media were treated with hemin (0, 12.5, 25, 50, 75,100 µM) for 18 (h). Intensity of calcium fluorescence was reduced in treated cultures with hemin (100, 86, 78.5, 60, 56, 46%, respective to the concentrations stated previously; $P<0.05$ for all). Hemin increased Ca2+ influx in cultured neurons. NMDAR stimulation by hemin increased the activating of NMDARs and Ca2+ influx in the cultured neurons. Therefore, heminis cytotoxic due to increase of intracellular Ca2+ influx.

Keywords: Mouse, Hemin, Neurons, Treatment

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