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## Poster Presentation

### Effects of Hemin on Ca<sup>2+</sup>Influx in Neurons of C57BL/6 Mouse Brain

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#### **Abstract**

Excitotoxicity results in a significant increase in Ca<sup>2+</sup> influx; essentially from open N-Methyl-D-aspartate receptors (NMDARs) channels that cause a secondary rise in the intracellular Ca<sup>2+</sup> concentration. It is correlated with neuronal death induced by Ca<sup>2+</sup> overload. Dysfunction of NMDARs is associated with excitotoxic neuronal death in neurodegenerative disorders. In this study, the effects of hemin on Ca<sup>2+</sup> 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% CO<sub>2</sub> 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 Ca<sup>2+</sup> influx in cultured neurons. NMDAR stimulation by hemin increased the activating of NMDARs and Ca<sup>2+</sup> influx in the cultured neurons. Therefore, hemin is cytotoxic due to increase of intracellular Ca<sup>2+</sup> influx.

**Keywords:** Mouse, Hemin, Neurons, Treatment

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