



Biological Sciences Seminar

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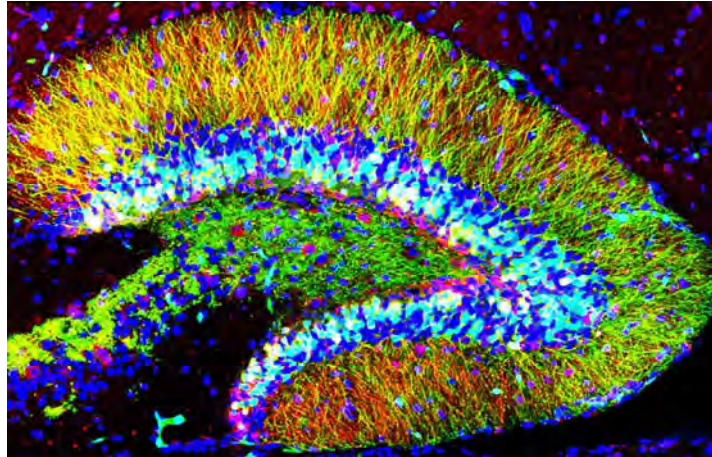
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“Neuroprotection and Neuroplasticity following Traumatic Brain Injury”

Abstract: In the United States (US), there are 1.5 million civilian incidents of traumatic brain injury (TBI) annually graded from mild to severe. Furthermore, it is estimated that more than 300,000 US veterans of the wars in Iraq and Afghanistan have sustained TBIs from blast waves of wartime improvised explosive devices (20% of 1.6 million). TBI represents one of the most serious consequences of accidents that human beings can suffer, often leading to dramatic motor impairment and neurological disorders. Unfortunately, little can be done to reverse the initial brain damage caused by trauma. Due to the serious damage and limited therapeutic approaches available, TBI is the leading cause of death in children and young adults. Here we show that a small peptide blocking NMDA 2B phosphorylation reduces calcium over-influx, protects neurons from excitotoxicity in vitro, and ameliorates cortical damage in vivo following TBI. This peptide is engineered to be able to cross the blood-brain barrier (BBB), and it can be administered systemically by intravenous injection. Intravenous injection of this peptide after TBI reduces cell death, both in the cortex and in the hippocampus, and improves behavioral performance on sensory, motor, and memory tasks. These data suggest that this small molecule is not only potent in neuroprotection following TBI in the mouse model, but it is also practical and convenient in future therapeutic developments for emergency care.

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