

Independent Study Title Effect of Melatonin on Hippocampal Neurogenesis in the Spinal Cord Injury Model

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ABSTRACT

Stress defined as a condition that seriously perturbs the physiological and psychological balance of an individual. Stress-related psychopathologies such as major depressive disorder (MDD), anxiety, conduct disorders, and posttraumatic stress disorder (PTSD). In humans, dysfunctions of glutamatergic neurotransmission, maladaptive structural and functional changes in hippocampal circuitry, and decreased hippocampal volume have been associated with stress-related conditions such as PTSD. In several studies, the animal models of PTSD resemble the animal models of neurodegenerative disease. To date, one of all neurodegenerative diseases causes permanent paralysis in patients was traumatic spinal cord injury (SCI). One of the neurotrophic factors in our body is melatonin. It has been reported that melatonin has also been shown to ameliorate in adult hippocampal neurogenesis, suggested to occur through its ability to scavenge free radicals.

Currently, we try to investigated the interventions to reduce hippocampal cell loss (dentate and cornu ammonis) after SCI by melatonin treatments immediately following the trauma and continuously administered for 14 days. Female mice were used in this study and were induced SCI via performing laminectomy at T12 vertebrae. The spinal cord was then compressed by using Dumont forceps to made

severe crush injury models. Melatonin was delivered by intra-peritoneal injection after SCI induction. After 14 days mice were sacrificed and brain tissue was removed for the immunohistochemical procedures study for neuronal density in hippocampus.

The previous study was consistent with this finding that melatonin can protect the brain from prolonged oxidative stress and loss of cells resulting from the inflammatory response after traumatic spinal cord injury as seen in neuronal density of hippocampus (dentate gyrus with cornu ammonis) differed significantly between melatonin treatment group and SCI group (16.00 ± 3.16 versus 1.25 ± 0.96) $p < 0.05$. These results can evaluate the neuroprotective effect of melatonin to elevated levels of inflammation on hippocampal in mice with spinal cord injury.

For further study, concerning with the chronically elevated concentration of melatonin within the large experimental group in prolonged duration of melatonin uptake are necessary.

Keywords: Neurogenesis/Hippocampal Cell Density/Spinal Cord Injury/Posttraumatic Stress Disorder (PTSD)/Melatonin