Neuroprotective Effects of Melatonin following TBI

Kamali, Andrew

Traumatic brain injury (TBI) is a multi-faceted injury and is considered one of the leading causes of death and disability in soldiers and civilians. During injury, a surge in reactive oxygen species (ROS) facilitates a vicious cycle that accelerates mitochondrial damage, excitotoxicity, lipid peroxidation, and inflammation, thus leading to possible increased cranial pressure, lack of natural plasticity, and neurochemical imbalance. Further, mitochondrial targeting strategies in TBI have been increasingly studied as their maintenance will potentially preserve brain function. Melatonin is synthesized naturally with in the body. Melatonin is the hormone that sets the circadian rhythm. Melatonin also functions as an antioxidant, which is beneficial to the health of the mitochondria and promoting cell survival. TBI was induced over the S1 region of the cortex in male Sprague Dawley rats to mimic a mild injury. Subsequently, MRI scans and behavioral assessments were performed on the day of injury and on 1, 2, 7 and 14 days post injury to determine lesion volume in TBI versus TBI + Melatonin treated animals. On day 14, the animals were sacrificed by a laboratory technician and immunohistochemistry was performed using Nissl and Fluro Jade B to further characterize the lesion. Based on the results, it was concluded that Melatonin significantly reduced lesion volume following a TBI. Additionally, behavioral improvements were seen with Melatonin treatment using the asymmetry test and the foot fault test.

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