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Neuroprotective Effect of Exogenous Melatonin on Apoptosis of Noradrenergic Neurons in Locus Coeruleus of Adult Male Rats After REM Sleep Deprivation

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Abstract:
Sleep as a widespread physiological phenomenon is seen in all vertebrates. In primates as human, the sleep consists of two components Rapid eye movement (REM) & non-REM. One of the major centers involved in the control of REM sleep is Locus Coeruleus (LC). REM sleep deprivation causes neural death in the LC. In the present study, we administered melatonin as an antioxidant factor and neuroprotective agent to prevent neural death.

Material & Methods:
In this study, the Flowerpot approach has been used to induce Reflex Sympathetic Dystrophy (RSD). Melatonin was administered for 7 days, the count & the volume of the LC neurons examined due to stereology methods. The enzymatic test for Glutathione GSH & measurement of Caspase-3 & C-Fos was done to assess the antioxidant property and apoptosis process and neural activity respectively. Immunohistochemistry of Anti-TH factors was done to assess the noradrenergic neurons and the Iba-1 test was also done to show the microglial migration.

Results:
According to the papers the RSD cause neural apoptosis in LC. Melatonin leads to a reduction in the level of apoptotic factor Caspase-3 expression followed by RSD. According to stereology analysis, the count of adrenergic neurons & the volume of the nucleus reduces after RSD & in the administered-melatonin group the apoptotic protein Caspase-3 reduces to prevent neural death. Microglial migration to the LC occurs after neural death and the melatonin increases GSH levels in RSD group finally.

Conclusion:

Melatonin with neuroprotective property can be used to treatment of adrenergic- depended sleep disorders & prevention of neural apoptosis in LC.

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