# Effect of Melatonin on inflammation and remyelination in EAE rat as an animal model of mono phasic Multiple sclerosis

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**Introduction:** Melatonin is an endogenous substance that utilized for treatment of diseases model. The administration of hormone of melatonin for the treatment of MS has been investigated in the EAE animal model of this disease. But despite the studies, the effect of melatonin on MS still remains ambiguous. The conflicting results in this regard, motivated our team for investigation the effect of Melatonin in low dose on inflammatory and remyelination process by more details in animal model of acute EAE In order to achieve more accurate results for the clarification of the melatonin role in MS disease.

**Material and method :** Adult Female Lewis rats were immunized subcutaneously by Guinea Pig Spinal Cord homogenate (GPSC) in Complete Freund's Adjuvant (CFA). After onset of EAE, Melatonin (50 mg/kg) was administered for 6 day. Animals were evaluated and scored for clinical signs of the disease. At day 6, rat were sacrificed. Following Histopathological study of lumbar spinal cord, the level of Serum cytokines IL-4 (TH2) and IFN-γ (TH1), was measured.

**Results:** The clinical scores were higher in Melatonin treated rat compared to saline treated EAE. Also the comparison of EAE group with Melatonin group significantly showed more demyelination in the Melatonin group. Inflammatory cells infiltration of the Spinal cord has developed by melatonin administration. IL-4 in EAE group was significantly decreased as compared to control group; however, slightly, this level was increased in Melatonin groups. The level of IFN-γ was increased after EAE induction compared to control group, but it significantly enhanced after treatment by Melatonin.

**Discussion:** According to these results, it seems that Th2 cells are not affected by Melatonin, because their cells are not hormone-dependent. Whereas, Th1 cells are highly influenced by hormones including Melatonin. Eventually, Melatonin exacerbate EAE symptoms by targeting Th1 cell and amplification of IFN-γ secretion .

**Keywords:** experimental autoimmune encephalomyelitis, Multiple sclerosis, Melatonin