Indian Journal of Fundamental and Applied Life Sciences ISSN: 2231-6345 (Online) An Open Access, Online International Journal Available at http://www.cibtech.org/jls.htm 2014 Vol. 4 (2) April-June, pp.133-142/Heshmati and Tiraihi ***Research Article***

THE EFFECTS OF DEPRENYLONSYNAPTIC ZONE AFTER SPINAL CORD INJURY IN RATS

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ABSTRACT

In order to distinguish spinal cord injury (SCI) caused by compression model, we demonstrated ultrastructural and morphometric changes in synaptic lesion after SCI. 72 female Sprague Dawley rats weighining 250-300g, were randomly divided into four groups (N =18). The sham groups were only subjected to laminectomy. All administered 2.5 mg/kg Deprenyl (CIPLA/ India, dissolved in 1cc saline) or equal volume of saline. At the time of operating for sham or SCI surgery groups daily injection were done and continued till they were sacricfied: group A: SCI + Deprenyl, group B: SCI + vehicle, group C: Sham + Deprenyl, and group D: Sham + vehicle. In every group, after 1, 2, 4weeks 6 animals were sacrificed. SCI caused motoneuron decreased in Anterior horn of spinal cord with hole and hemorrhage. Deprenyl decreased this process ($P \leq 0.05$). The most pattern of synaptophysin in group A which received Deprenyl, was cytoplasmic ($P \leq 0.05$). Synaptic and neuronal mitochondria changes were analyzed in a blinded manner for qualitative ultrastructural changes. By SCI, pathological changes including irregularity of the synaptic membrane and synaptic cleft with displacement of synaptic vesicles and irregularity of mitochondria were seen beside synaptophysin reduction. Deprenyl decreased pathological changes and maintained motoneurons after SCI.

Keywords: *Deprenyl, Spinal Cord Injury, Synapse, Rat*

INTRODUCTION

Spinal Cord Injury (SCI) causes a majority of disability and costly human condition with worldwide incidence of 10-40 cases per million. Following SCI tissue damage accurred, neurobiological disability depend on mechanical pressure such as vertebrae dislocation, compression or vertebrae traction. Induce of spinal cord injury (SCI), neurons have been reported to undergo cell death (Nielson *et al.,* 2011) where apoptosis was confirmed by using ultrastructural study (Wong *et al.,* 2012) and TUNEL (Loo, 2011). The molecular changes were characterized by an increase in pro-apoptotic gene expression such as Bax (Nickells *et al.,* 2008) or activation of caspase 3 (De-Bilbao *et al.,* 2000). These studies were used to define the type and structure of the dead cell. Other investigators tried other approaches to characterize the histo-functional feature of apoptotic cells, especially in synaptic zone. Findings were reported about the expression of synaptophysin, as a membrane protein in synaptic vesicle filled with neurotransmitters. When an action potentials depolarizes the presynaptic plasma membrane, Ca^{2+}-channels open, and Ca^{2+} flows into the nerve terminal to trigger the exocytosis of synaptic vesicles, releasing their neurotransmitters into the synaptic cleft. Pathological changes this process lead to neurodegenerative dieases (Shojo *et al.,* 2006). The efficacy of some drugs have been studied in various models of SCI. Results show monoamine oxidases type-B inhibitor (MAOB-Is) Selegiline and (-) Deprenyl, used in Parkinson dieases have a lot of pharmacological activities beside its MAOB inhibitory potency,

can protect neurons from neural degeneration, mitochondrial impairerment, oxidative stress, enhances the synthesis of neurotrophic factors and anti-apoptotic Bcl-2 protein family (Tatton *et al.,* 1996; Maruyama *et al.,* 2013). In present study the effects of Deprenyl on motoneuron survival, pattern of synaptophysin expression and ultrastructural of synapse were evaluated. Morphometric parameters were used to evaluate the trend of changes quantitatively and patterns of synaptophysin were demonstrated by immunohistochemistry.