

## Repair of spinal cord injury (SCI) using ceftriaxone in a rat SCI model

J. Tajkay<sup>1,\*</sup>, B. Habibi Asel<sup>1</sup>, A. Biglari<sup>2</sup>, A. Ramezani<sup>2</sup>

<sup>1</sup>Department of Pharmacology, School of Pharmacy, Tabriz University of Medical Sciences, Tabriz, Iran

<sup>2</sup>Department of Biotechnology, School of Pharmacy, Zanjan University of Medical Sciences, Zanjan, Iran

**Background and Aims:** Subsequent to spinal cord injury many pathological changes may occur that lead to inappropriate environment for repair. Most important of such changes is the death of neurons. Excess glutamate can leading to neuronal damage and eventual cell death, and is called excitotoxicity. Glutamate neurotransmission is highly regulated, largely by glutamate transporters. ceftriaxone, a  $\beta$ -lactam antibiotic upregulates GLT-1 expression. The glutamate transporter GLT-1 is primarily responsible for glutamate clearance. Downregulation of GLT-1 can occur in activated astrocytes, and is associated with increased extracellular glutamate and neuroexcitation. In this study, we examined the effects of ceftriaxone on functional recovery following spinal cord injury (SCI).

**Methods:** Rats were divided into three groups of six randomly. Spinal cord injury was then performed under general anesthesia using the weight dropping method. The ceftriaxone were injected at a dose of 200mg/kg/day in normal saline for seven days before and after SCI. Group one included rats receiving normal saline, group two received ceftriaxone seven days before SCI, group three received ceftriaxone seven days after SCI. A Basso, Beattie and Bresnahan (BBB) score test was performed for six weeks. Two weeks before the end of BBB, biotin dextran amine was injected intracerebrally and at the end of the sixth week tissue staining was performed.

**Results:** There was a significant difference in BBB scores between group one and other groups. There were significant differences in axon counting between group one and other groups.

**Conclusions:** These data suggest that, after SCI, ceftriaxone treatment modulated expression of glutamate transporter GLT-1, attenuated cell death, and improved functional recovery in the injured rat. This approach may provide a therapeutic intervention enabling us to reduce cell death and improve functional recovery after SCI.

**Keywords:** Spinal cord injury; Ceftriaxone; Rat