



## Poster Presentation

### Delayed Imatinib Treatment for Spinal Cord Damage; Role of Serum Biomarkers and Recovery

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#### Abstract

With no routine accessible medicine intervention for spinal cord damage, there is a demand for more remedial candidates. However, for Imatinib to have translational price, it requires to have encouraged obliging effects with delayed start of treatment, as well. Serum levels of 3 chemokines/ cytokines, MIP-3 $\alpha$ , MCP-1, and GRO/ KC (IL-8), to raise over time with Imatinib treatment and to be obviously higher in damaged Imatinib treated animals than in manages pending the early treatment period. Lymphoid organs, first the spleen, were tested to supply information on systemic effects of Imatinib with consider to inflam- matory responses and lymphoid organs as the source of monocyte/ macrophage infiltration into the damage site of the spinal cord. Serum samples at one, three and seven days after damage were tested for INF-1 $\beta$ , MIP-3 $\alpha$ , MCP-1, IL-2, IL-4, IL-5, IL-6, IL-10, IL-13, GRO/ KC and TNF. Three cytokines/chemokines, MIP-3 $\alpha$ , MCP-1 and GRO/ KC demonstrated definite potential as biomarkers in serum. Serum levels of MIP-3 $\alpha$  and MCP-1 were raised one day after damage or sham surgery. GRO/KC concentrations were instead higher in the sham group compared to the contusion-damaged groups without or with Imatinib treatment, making this chemokine a potential serum biomarker for CNS damage. At one day after surgery, there was however no strong effect of Imatinib treatment of animals with spinal cord damage among the tested chemokines. Serum concentrations of MCP-1 and MIP-3 $\alpha$  remained elevated in the damaged and sham damaged group throughout the seven days in comparison to concentrations in uninjured managements.

**Keywords:** Spinal Cord Damage, Serum Biomarkers, Chemokines, Treatment

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