**The hematopoietic factor granulocyte-colony stimulating factor improves outcome in experimental spinal cord injury.**

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[**Author information**](http://www.ncbi.nlm.nih.gov/pubmed/20202082)

**Abstract**

Granulocyte-colony stimulating factor (G-CSF) is a potent hematopoietic factor that drives differentiation of neutrophilic granulocytes. We have recently shown that G-CSF also acts as a neuronal growth factor, protects neurons in vitro and in vivo, and has regenerative potential in various neurological disease models. Spinal cord injury (SCI) following trauma or secondary to skeletal instability is a terrible condition with no effective therapies available at present. In this study, we show that the G-CSF receptor is up-regulated upon experimental SCI and that G-CSF improves functional outcome in a partial dissection model of SCI. G-CSF significantly decreases apoptosis in an experimental partial spinal transsection model in the mouse and increases expression of the anti-apoptotic G-CSF target gene Bcl-X(L). In vitro, G-CSF enhances neurite outgrowth and branching capacity of hippocampal neurons. In vivo, G-CSF treatment results in improved functional connectivity of the injured spinal cord as measured by Mn(2+)-enhanced MRI. G-CSF also increased length of the dorsal corticospinal tract and density of serotonergic fibers cranial to the lesion center. Mice treated systemically with G-CSF as well as transgenic mice over-expressing G-CSF in the CNS exhibit a strong improvement in functional outcome as measured by the BBB score and gridwalk analysis. We show that G-CSF improves outcome after experimental SCI by counteracting apoptosis, and enhancing connectivity in the injured spinal cord. We conclude that G-CSF constitutes a promising and feasible new therapy option for SCI.