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The chondroitin sulfate proteoglycans neurocan, brevican, phosphacan, and versican are differentially regulated following spinal cord injury.

Jones LL, Margolis RU, Tuszynski MH.
Department of Neurosciences-0626, University of California-San Diego, 9500 Gilman Drive, La Jolla, CA 92093, USA.

Abstract
Chondroitin sulfate proteoglycans (CSPGs) are extracellular matrix (ECM) molecules that are widely expressed throughout the developing and adult CNS. In vitro studies demonstrate their potential to restrict neurite outgrowth, and it is believed that CSPGs also inhibit axonal regeneration after CNS injury in vivo. Previous studies demonstrated that CSPGs are generally upregulated after spinal cord injury, and more recent reports have begun to identify individual proteoglycans that may play dominant roles in limiting axonal regeneration. The current study systematically examined the extended deposition patterns after CNS injury of four putatively inhibitory CSPGs that have not been extensively investigated previously in vivo: neurocan, brevican, phosphacan, and versican. After spinal cord injury, neurocan, brevican, and versican immunolabeling increased within days in injured spinal cord parenchyma surrounding the lesion site and peaked at 2 weeks. Neurocan and versican were persistently elevated for 4 weeks postinjury, and brevican expression persisted for at least 2 months. On the other hand, phosphacan immunolabeling decreased in the same region immediately following injury but later recovered and then peaked after 2 months. Combined glial fibrillary acidic protein (GFAP) immunohistochemistry and in situ hybridization demonstrated that GFAP astrocytes constituted a source of neurocan production after spinal cord injury. Thus, the production of several CSPG family members is differentially affected by spinal cord injury, overall establishing a CSPG-rich matrix that persists for up to 2 months following injury. Optimization of strategies to reduce CSPG expression to enhance regeneration may need to target several different family members over an extended period following injury.

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