J Neurosci Res. 2009 Oct;87(13):2937-50.

Traumatic brain injury results in disparate regions of chondroitin sulfate proteoglycan expression that are temporally limited.

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Abstract
Axonal injury is a major hallmark of traumatic brain injury (TBI), and it seems likely that therapies directed toward enhancing axon repair could potentially improve functional outcomes. One potential target is chondroitin sulfate proteoglycans (CSPGs), which are major axon growth inhibitory molecules that are generally, but not always, up-regulated after central nervous system injury. The current study was designed to determine temporal changes in cerebral cortical mRNA or protein expression levels of CSPGs and to determine their regional localization and cellular association by using immunohistochemistry in a controlled cortical impact model of TBI. The results showed significant increases in versican mRNA at 4 and 14 days after TBI but no change in neurocan, aggrecan, or phosphacan. Semiquantitative Western blot (WB) analysis of cortical CSPG protein expression revealed a significant ipsilateral decrease of all CSPGs at 1 day after TBI. Lower CSPG protein levels were sustained until at least 14 days, after which the levels began to normalize. Immunohistochemistry data confirm previous reports of regional increases in CSPG proteins after CNS injury, seen primarily within the developing glial scar after TBI, but also corroborate the WB data by revealing wide areas of pericontusional tissue that are deficient in both extracellular and perineuronal net-associated CSPGs. Given the evidence that CSPGs are largely inhibitory to axonal growth, we interpret these data to indicate a potential for regional spontaneous plasticity after TBI. If this were the case, the gradual normalization of CSPG proteins over time postinjury would suggest that this may be temporally as well as regionally limited. 2009 Wiley-Liss, Inc.

PMID: 19437549 [PubMed - indexed for MEDLINE]PMCID: PMC2821049