Blocking EphA4 upregulation after spinal cord injury results in enhanced chronic pain.

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Enviado por Annabell Segarra [2] el 11 julio 2014 - 7:16pm


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Título	Blocking EphA4 upregulation after spinal cord injury results in enhanced chronic pain.
Publication Type	Journal Article
Year of Publication	2006
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Journal	Exp Neurol
Volume	202
Issue	2
Pagination	421-33
Date Published	2006 Dec
ISSN	0014-4886
Palabras clave	Animals [13], Chronic Disease [14], Female [15], Fluorescent Antibody Technique [16], Glial Fibrillary Acidic Protein [17], Hyperalgesia [18], Motor Activity [19], Oligonucleotides, Antisense [20], Pain [21], Pain Measurement [22], Phosphopyruvate Hydratase [23], Psychomotor Performance [24], Rats [25], Rats, Sprague-Dawley [26], Receptor, EphA4 [27], Reverse Transcriptase Polymerase Chain Reaction [28], RNA, Messenger [29], Spinal Cord Injuries [30], Time Factors [31], Up-Regulation [32]

Abstract	Spinal cord injury (SCI) is characterized by a total or partial loss of motor and sensory functions due to the inability of neurons to regenerate. This lack of axonal regenerative response has been associated with the induction of inhibitory proteins for regeneration, such as the Eph receptor tyrosine kinases. One member of this family, the EphA4 receptor, coordinates appropriate corticospinal fibers projections during early development and is expressed in spinal commissural interneurons. Its mechanism of action is mediated by repulsive activity after ligand binding, but its role after trauma is unknown. We examined the temporal expression profile of this receptor after spinal cord contusion in adult rats by RT-PCR and immunohistochemistry. SCI induced a biphasic gene expression profile with an initial downregulation at 2 and 4 days post-injury (DPI) followed by a subsequent upregulation. Double labeling studies localized EphA4 immunoreactivity in neurons from the gray matter and astrocytes of the white matter. To test the role of this receptor, we reduced gene upregulation by intrathecal/subdural infusion of EphA4-antisense oligodeoxynucleotide (ODN) and subsequently assessed behavioral outcomes. No locomotor recovery was observed in the rats treated with the EphA4-antisense ODN. Interestingly, reducing EphA4 expression increased mechanical allodynia, as observed by the Von Frey test and decreased exploratory locomotor activity. These results indicate that upregulation of EphA4 receptor after trauma may prevent the development of abnormal pain syndromes and could potentially be exploited as a preventive analgesic mediator to chronic neuropathic pain.
DOI	10.1016/j.expneurol.2006.07.005 [33]
Alternate Journal	Exp. Neurol.
PubMed ID	16959251 [34]
Grant List	GM-68138 / GM / NIGMS NIH HHS / United States NS39404 / NS / NINDS NIH HHS / United States S06-GM008224 / GM / NIGMS NIH HHS / United States

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http://dx.doi.org/10.1016/j.expneurol.2006.07.005 [34]
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