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Upregulation of calcium channel α -2-delta 1 subunit in dorsal horn contributes to
spinal cord injury-induced tactile allodynia

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Abstract

BACKGROUND CONTEX: Spinal cord injury (SCI) commonly results not only in
motor paralysis but also in the emergence of neuropathic pain, both of which can
impair the quality of life for SCI patients. In the clinical field, it is well known that
pregabalin, which binds to the voltage-gated calcium channel $\alpha_2\delta$ -1 subunit has
therapeutic effects on neuropathic pain after SCI. A Previous study has demonstrated
that SCI increased $\alpha_2\delta$ -1 in L4–6 dorsal spinal cord of SCI rats by Western blot
analysis and that the increase of $\alpha_2\delta$ -1 was correlated with tactile allodynia of the hind
paw. However, the detailed feature of an increase in $\alpha_2\delta$ -1 protein in the spinal dorsal
horn and the mechanism of pregabalin effect on SCI induced neuropathic pain has
not been fully examined.

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