

ABSTRACT

TRPV1 mediate Matrix Metalloproteinase expression in spinal cord injury-induced chronic neuropathic pain

Neuropathic pain (NP) is common following spinal cord injury (SCI). Evidence exists for the involvement of many processes in NP development following SCI (Cain et al, 2007). Our past studies identified increased expression of nociceptive genes, including vanilloid receptor-1 (TRPV-1) following SCI in rats. Recent studies have described the matrix metalloproteases (MMPs) role in many disease processes involving the central nervous system. There is evidence that MMPs may be involved in the pathway initiated by TRPV-1 activation. Our current study investigates the role of TRPV-1 receptor activity in the regulation of MMP-2 and following SCI. We hypothesize that 1) SCI will cause TRPV-1 upregulation and a corresponding increase in gene expression of MMP-2. The increased MMP-2 levels promote the manifestation of NP and 2) inhibition of MMP-2 by expression will attenuate NP onset. The results from our studies have potential use in the development of novel treatments for post SCI NP.

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