PubMed

Format: Abstract

Full text links

ELSEVIER FULL-TEXT ARTICLE

Eur J Pharmacol. 2007 Apr 10;560(2-3):142-9. Epub 2007 Jan 19.

Minocycline and pentoxifylline attenuate allodynia and hyperalgesia and potentiate the effects of morphine in rat and mouse models of neuropathic pain.

Mika J[1], Osikowicz M, Makuch W, Przewlocka B.

Author information

Abstract

Recent research has shown that microglial cells which are strongly activated in neuropathy can influence development of allodynia and hyperalgesia. Here we demonstrated that preemptive and repeated i.p., administration (16 h and 1 h before injury and then after nerve ligation twice daily for 7 days) of minocycline (15; 30; 50 mg/kg), a potent inhibitor of microglial activation, significantly attenuated the allodynia (von Frey test) and hyperalgesia (cold plate test) measured on day 3, 5, 7 after chronic constriction injury (CCI) in rats. Moreover, the 40% improvement of motor function was observed. In mice, i.p., administration of minocycline (30 mg/kg) or pentoxifylline (20 mg/kg) according to the same schedule also significantly decreased allodynia and hyperalgesia on day 7 after CCI. Antiallodynic and antihyperalgesic effect of morphine (10 mg/kg; i.p.) was significantly potentiated in groups preemptively and repeatedly injected with minocycline (von Frey test, 18 g versus 22 g; cold plate test, 13 s versus 20 s in rats and 1.2 g versus 2.2 g; 7.5 s versus 10 s in mice; respectively) or pentoxifylline (1.3 g versus 3 g; 7.6 s versus 15 s in mice; respectively). Antiallodynic and antihyperalgesic effect of morphine (30 microg; i.t.) given by lumbar puncture in mice was also significantly potentiated in minocycline-treated group (1.2 g versus 2.2 g; 7.5 s versus 11 s; respectively). These findings indicate that preemptive and repeated administration of glial inhibitors suppresses development of allodynia and hyperalgesia and potentiates effects of morphine in rat and mouse models of neuropathic pain.

PMID: 17307159 DOI: 10.1016/j.ejphar.2007.01.013

[Indexed for MEDLINE]

Publication type, MeSH terms, Substances

LinkOut - more resources