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Minocycline attenuates mechanical allodynia and central sensitization following peripheral second-degree burn injury.

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Abstract

Burn injury induces severe pain that can be refractory to existing pharmacotherapies. The underlying mechanism of burn pain remains unclear. We previously established an animal model and reported that unilateral burn injury induces chronic and bilateral mechanical allodynia, which is associated with central sensitization and microglial activation in the spinal cord dorsal horn. Modulation of the activity of microglia and p38 mitogen-activated protein kinase (MAPK) has been shown to ameliorate neuropathic pain in several nerve-injury pain models. In the present study, we show in this rat model that daily treatment with the microglial inhibitor minocycline (10 mg/kg), administered at the time of burn injury and for 7 days thereafter, significantly attenuates ipsilateral and contralateral allodynia as assessed up to 1 month following burn injury. These sensory changes are paralleled by significant suppression of evoked hyperexcitability of dorsal-horn neurons and of the expression of phosphorylated p38 (phospho-p38) in OX42+ microglial cells within the dorsal horn. Our results suggest that modulation of inflammation at early times after burn injury may have long-lasting effects, attenuating central neuropathic mechanisms which contribute to pain after burn injury.

PERSPECTIVE: We demonstrate, in a rodent model of burn-associated pain, that the microglial inhibitor minocycline, delivered at the time of burn injury and for 1 week thereafter, has long-lasting effects, attenuating microglial activation and neuronal hyperresponsiveness in the dorsal horns, and ameliorating allodynia for at least 1 month.

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