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Format: Abstract[J Neurotrauma](#). 2003 Jun;20(6):593-601.**Neuronal sensitization and its behavioral correlates in a rat model of neuropathy are prevented by a cyclic analog of orphenadrine.**[Biella GE¹](#), [Groppetti A](#), [Novelli A](#), [Fernández-Sánchez MT](#), [Manfredi B](#), [Sotgiu ML](#).**Author information****Abstract**

N-methyl-D-aspartic acid (NMDA) is an agonist at the homonymous receptor implicated in the development of neuronal sensitization and its behavioral correlates. An effective modulation of the NMDA effects, achieved also by uncompetitive antagonists, could contribute to controlling pain symptoms in several neuropathic syndromes. Because nefopam is a known analgesic derivative of orphenadrine and of its congener diphenhydramine, both uncompetitive NMDA receptor antagonists, we tested the effect of nefopam on the developing pain and neuronal anomalies in an animal model of chronic pain with NMDA receptor involvement. A single intraperitoneal injection of nefopam was administered twenty minutes prior to the chronic constriction injury of the sciatic nerve (CCI rats). In the first 10 days, nefopam (30 mg/kg) significantly decreased behavioral signs of neuropathic pain and the stimulus-evoked electrophysiological anomalies in recordings at 14 days, with only slight manifestation afterwards. The dose of 20 mg/kg was ineffective. Nefopam injected after constriction was ineffective. In normal non-operated rats, Nefopam had no effect on the electrophysiological and behavioral parameters. Iontophoretic nefopam (1 mM, 50-80 nA, positive current) in normal rats did not change the spontaneous neuronal activity, but reduced the mean response to noxious stimuli and the concurrent iontophoretic NMDA evoked activity. In CCI rats, iontophoretic nefopam did not significantly modify the spontaneous hyperactivity but reduced significantly both the frequency of the responses to noxious stimuli, and the duration of the afterdischarge. We propose that nefopam exerts a preventive analgesic effect, with a possible role in modulating NMDA receptor-mediated effects in central sensitization.

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