Spinal long-term potentiation is associated with reduced opioid neurotransmission in the rat brain.

Neuronal events leading to development of long-term potentiation (LTP) in the nociceptive pathways may be a cellular mechanism underlying hyperalgesia. In the present study, we examine if induction of spinal LTP may be associated with functional changes in the supraspinal opioidergic system. The opioid receptors (ORs) play a key role in nociceptive processing and controlling the descending modulatory system to the spinal cord. Spinal LTP was induced by electrical high-frequency stimulation (HFS) conditioning applied to the sciatic nerve, and the excitability at spinal level was verified by spinal field potential recordings. To study supraspinal changes in opioid neurotransmission following the same HFS conditioning, we used small animal positron emission tomography (PET) and [(11)C]Phenethyl-Orvinol ([(11)C]PEO). All rats included in the PET study were scanned at baseline and 150 min after HFS, and specific binding was calculated with a reference tissue model. A clear C-fibre LTP, i.e. increased C-fibre response and reduced C-fibre threshold, was observed 150 min after HFS conditioning (t-test, Pbaseline, P<0.05, n = 8). HFS conditioning of the sciatic nerve resulted in both spinal LTP and functional changes in supraspinal opioidergic signalling. Our findings suggest that induction of spinal LTP may be associated with reduced opioid neurotransmission in brain regions involved in pain modulation.
and affective-emotional responses.

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