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Splitting Cognitive and Depressive Brain Circuits in Long Term Peripheral Neuropathy

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Abstract

Neuropathic pain has long-term consequences in terms of affective and cognitive disturbances suggesting the involvement of supraspinal mechanisms.

Objective: In the present study, we use the spared nerve injury (SNI) model to characterize the development of sensory and aversive components of neuropathic pain, and to determine their electrophysiological impact at brain level.

Methods and Results: We show that SNI leads to sensorial hypersensitivity (cold and mechanical stimuli) lasting 12 months after nerve injury. Likewise, a depressive-like profile is reported in both short- and long-term neuropathy. Interestingly, impairments in non-emotional cognitive tasks (novel object recognition and Y maze) which appear in 1-month SNI mice, result normalized after 12 months. In vivo electrophysiology reveales that SNI impaires the Long Term Potentiation (LTP) the prelimbic cortex (PL)- nucleus accumbens core (NAcore) pathway in both 1 and 12 months of neuropathy. On the other hand, a reduced neural activity is recorded in the lateral entorhinal cortex (LEC)-dentate gyrus (DG) pathway in 1 month-, but not in 12 months- SNI mice. Interestingly, we observe the upregulation of specific genes involved in neuroinflammation and apoptosis signaling pathways in the hippocampus of 1 month-, but not 12 months-SNI mice.

Conclusions: In conclusion these data indicate a recovery of cognitive, but not affective disturbances 1 year post peripheral nerve injury. Moreover, they suggest that distinct brain circuits may drive the psychiatric components of the neuropathic pain. Finally, they also provide an immune profile analysis in SNI mice suggesting novel cellular and biomolecular pathways for investigating neuropathic pain mechanisms and related comorbidity.

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Abstract
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