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## Can modulating inflammatory response be a good strategy to treat neuropathic pain?

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#### Abstract

Neuronal injury not only results in severe alteration in the function of primary sensory neurons and their central projection pathway, but is also associated with a robust immune response at almost every level of the somatosensory system. Evidence from animal studies suggests undoubtedly that bi-directional signalling between the immune system and the nervous system contribute to the development and maintenance of chronic neuropathic pain. **Non-neuronal cells, including peripheral immune cells, CNS/PNS glial cells and endothelial cells play important roles in the neuroimmune interaction and subsequent persistent hypersensitivity. Various cytokines and chemokines have been identified as key signalling molecules in the crosstalk.** However, majority evidence showing inflammation in neuropathic pain was generated from animal models at acute phase. Whether and to what extent inflammation or non-neuronal cells are involved at chronic stage of neuropathic pain needs to be further explored, and evidence of inflammation in chronic pain from human studies is still largely awaited. Therapeutic agents targeting inflammation provide an exciting prospect. Yet, considering the heterogeneous conditions presented in neuropathic pain, no matter the etiologies, or the pathophysiology during different stages of the disease; and the complexity of the immune response to the damage on the nervous system, **it appears that finely tuned strategies of modulating inflammation are essential to warrant an effective treatment for neuropathic pain. We want to reduce pain; we also want to promote tissue repair and functional recovery.**

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