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## The role of the neuroimmune interface in neuropathic pain and therapeutic implications in the clinical scenario

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Neuropathic pain poses a significant burden on the individual and society, because of a lack of effective pharmacological therapies. Interventional pain therapies may be more effective in a select cohort of patients. Neuropathic pain results from a lesion or disease of the somatosensory system. Traditionally neuropathic pain was attributed to dysfunctional activity and hypersensitivity of the neuron. However a maladaptive immune response is increasingly recognized as contributing to the maintenance of chronic neuropathic pain. Glial cell activation and altered peptide biosynthesis are implicated in the maintenance of persistent neuropathic pain. Human data suggests that NGF, VEGF and BDNF play a role here. This neuroimmune process is maintained by cellular and neuropeptide biosynthetic responses which become pathological. Intra-thecal opioids and steroids are associated with alterations in cell signaling and the levels of inflammatory mediators at the neuroimmune interface in humans *in vivo.* Markers of glial and immune cell activity, up-regulated in neuropathic pain states, are down-regulated in spinal cord stimulator patients. Pulsed radiofrequency of the dorsal root ganglion in patients with radicular pain alters CSF lymphocyte subsets and peptide concentrations.

### Biography

David Moore is a Post-doctoral research fellow at the Department of Pain Medicine, St James Hospital, Dublin and the Institute of Neurotherapeutics, Dublin City University.

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