**Immunity in Neuropathic Pain**

Neuropathic pain (NP) is a devastating chronic pain state not treatable by current analgesics. NP may arise spontaneously (e.g. shooting pain) or from light (e.g. tough) stimuli in people suffering from PNS/CNS damage caused by physical trauma, neurodegenerative and autoimmune disease, metabolic disease (e.g. diabetes), chemotherapy, viral pathogens, etc. We have established many mechanisms of neuro-inflammation involved in the development of NP. Thus, we identified an inflammatory cytokine TNF-α as a spinal disc component and a therapeutic target in radiculopathy (1). By establishing the ability of TNF-α to undergo axonal transport (2), we proposed a new mechanism of CNS plasticity and remote neuroinflammation after peripheral lesions. Our work was the first to implicate metalloproteinases (reviewed in 3, 4) and myelin autoantigens (5-7) as pain mediators. Our current research suggests that the release of the algesic fragments of myelin basic protein (MBP) from the intact myelin sheath protein contributes to autoimmune pathogenesis of NP, especially in females (micrograph). Our long-term aim is to develop novel, non-addictive therapeutic strategies in treating chronic neuropathic pain.

**Representative papers**


