Special Issue

Molecular Mechanisms of Neuropathic Pain and Nerve Injury

Message from the Guest Editor

Neuropathic pain such as postherpetic neuralgia, post-stroke pain, and trigeminal neuralgia is known to occur as a result of peripheral and/or central neurological disturbances. Neuropathic pain is also clinically typified by intractable non-noxious stimulation-induced pain and thought to be a direct result of a lesion or disease that affects the peripheral somatosensory system. First, a neuroinflammatory reaction is induced, associated with numerous changes in the immune cell microenvironment, surrounding the damaged peripheral nerve. It is reported that immune cells infiltrate the injury site. The injured neurons and Schwann cells release proinflammatory cytokines, chemokines and neuropeptides, which leads to peripheral neuronal hypersensitivity. The injured neurons themselves cause plastic changes associated with decrease of threshold, spontaneous activity, and hyper-responsiveness. The understanding of the involvement of nerve injury in molecular mechanisms of pain hypersensitivity could offer better diagnostic and therapeutic approaches for neuropathic pain. This Issue will be of interest to basic researchers and clinicians interested in neuropathic pain mechanism.

Guest Editor

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