Commentary

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Novel Approaches in Neuropathic Pain Treatment Targeting Transient Receptor Potential Channels

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Description

An estimated 1 in 20 people in western countries suffer from neuropathic pain, which is a burden on society and typically affects 7%-10% of the global population. Dose escalation problems, off-target side effects and inadequate conversion of randomized studies into clinical practice are the main constraints of current medicines. In addition to causing maladaptive changes in both neuronal and non-neuronal cells, neuropathic pain is a broad term that includes direct injury or damage to the central and peripheral nervous systems. These changes also contribute to spontaneous pain, sensory and motor deficits and altered sensitivity to both noxious and non-noxious stimuli. Polymodal, non-specific cation channels known as Transient Receptor Potential (TRP) channels function as biosensors to a range of mechanical and chemical stimuli, including as extracellular ATP, shear stress, heat, mechanical stretch, hyperosmolarity and other inflammatory products. At the molecular and cellular level, alteration of these channels results in a variety of physiological and pathological symptoms, including neuropathic pain. Numerous compounds that target TRP channels for neuropathic pain are available on the market, in preclinical research, and in clinical trials. In order to fully utilize the therapeutic potential of TRP channels, this review emphasizes the vital role of several pharmacological modulators for TRP channels that target neuropathic pain and their prospective results.

Mechanisms of neuropathic pain

The international association for the study of pain defines neuropathic pain as "pain induced by a lesion or disease of the somatosensory system," which surround both peripheral and central neurons. Most often, spinal cord and/or brain injuries-including those from multiple sclerosis and post-stroke symptoms-cause central neuropathic pain. On the other hand, peripheral neuropathic pain results from damage to peripheral nerves (trigeminal neurons or sciatic nerves) and is a consequence of several illnesses, such as diabetes and herpes virus infection. It can also be brought on by chemotherapy (taxanes, platinum compounds, vinca alkaloid, *etc.*) and anti-retroviral medications. Since neuropathic pain is linked to a number of complex problems, including spontaneous pain, allodynia and hyperalgesia and poor responsiveness to traditional analgesics, the precise mechanism behind this condition is yet unknown. Peripheral nerve injuries can, in fact, develop into persistent neuropathic pain in a variety of ways. Drugs, cytokines and other proinflammatory and inflammatory mediators can change the density of nerve fibres and the hyperexcitability of neuronal cells after they have been affected by metabolic damage. Neuropathic pain can also develop as a result of malfunctioning peripheral nerve terminals that process pain, such as thinly-myelinated A-fibres and unmyelinated C-fibres. Injuries that can result in fibre degeneration and changes in the expression and composition of channels down the axon include acute nerve injury, compression, hypoxia, inflammation, stimulation overload and chemical damage. These changes can cause ectopic firing and impaired signal transmission.

Pathophysiology of neuropathic pain

The pathobiological events involved in peripheral neuropathic pain as a side effect of chemotherapy treatment, traumatic nerve injury and complications related to diabetes are summarized, as are the events that take place at the peripheral, spinal and supraspinal levels, followed by peripheral or central nerve injury. Furthermore, because the channels are polymodal and widely expressed throughout the somatosensory system, they are strongly linked to neuropathic pain. At the brain's pain processing centers, this enables TRP channels to be triggered down the axon and cell body of neuronal cells involved in signal transduction, transmission and perception of the aberrant stimuli. The international association for the study of neuropathy defines neuropathic pain as "pain induced by a lesion or disease of the somatosensory system," which includes both peripheral and central neurons. Numerous complex diseases, such as spontaneous pain, allodynia and hyperalgesia, and poor responsiveness to traditional analgesics, are associated with pain that has not yet been fully identified. In fact, neuropathic pain can develop in a variety of ways, including through weakly myelinated A-fibres and unmyelinated C-fibres.