The Role of Sodium Channels in Nerve Injury Pain

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Management of neuropathic pain is still unsatisfactory mainly because of an insufficient understanding of underlying mechanisms. Sodium channel blockers represent an important group of agents used to treat neuropathic pain, but their mechanism of action and where that effect is exerted is still unsettled. Under normal and pathological conditions neuronal firing is tuned by the complement of sodium channels and their localization along injured fibers in these neurons. Animal and human studies have demonstrated dynamic changes in the expression of channels and their subcellular localization after injury which may contribute to the pain behavior. Sodium channels are expressed both in the peripheral and central nervous system and may therefore also exert their effects peripherally as well as centrally.

While drugs such as opioids, tricyclic antidepressants and calcium channel α2δ subunit ligands seem to have somewhat consistent effects across the spectrum of neuropathic pain conditions, new evidence suggests that the response to sodium channel blocking drugs may depend on the mechanisms that leads to nerve injury and to the characteristics of the pain syndrome in an individual patient. It will be demonstrated how sensory profiling can contribute to predict a better treatment response to a sodium channel blocker. The presentation will review the emerging evidence for sodium channel blocking approaches in peripheral and central neuropathic pain, and discuss the conditions and patient populations in whom pharmacological targeting of sodium channels may be particularly beneficial.