Abstract:

Pain is a major unmet medical need which has been causally linked to changes in sodium channel expression, modulation, or mutations that alter channel gating properties or current density in nociceptor neurons. Voltage-gated sodium channels activate (open) then rapidly inactivate in response to a depolarization of the plasma membrane of excitable cells allowing the transient flow of sodium ions thus generating an inward current which underlies the generation and conduction of action potentials (AP) in these cells. Activation and inactivation, as well as other gating properties, of sodium channel isoforms have different kinetics and voltage-dependent properties, so that the ensemble of channels that are present determine the electrogenic properties of specific neurons. Biophysical and pharmacological studies have identified the peripheral-specific sodium channels Na(v)1.7, Na(v)1.8 and Na(v)1.9 as particularly important in the pathophysiology of different pain syndromes, and isoform-specific blockers of these channels or targeting their modulators hold the promise of a future effective therapy for treatment of pain.

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