ABSTRACT

Pain is a crucial component of the body's innate defenses, which helps us to respond to the damage that is threatening or imminent. If the pain persists even after the injury has healed, or arises for no apparent reason, it itself becomes harmful. Nociception begins with the detection of a noxious stimulus that irritates free nerve endings on the peripheral projections of spinal ganglion neurons. If the stimulus induces depolarization of the cell and an action potential forms, information of the stimulus is conducted by thinly myelinated A δ fibers, or unmyelinated C fibers to the spinal cord dorsal horn. Here, the first synapses of sensory pathways are located, which allow the transmission of nociception to secondary afferent neurons, and these further direct the information to the higher centers of the CNS. Synapses in the dorsal horn are key to modulating nociceptive signaling, in which the endocannabinoid system, including endogenous cannabinoids and their receptors, plays a significant role. However, under pathological conditions such as the development of neuropathic pain or neuroinflammation, changes in the expression and function of agonists and receptors of the endocannabinoid system occur. These changes are of great importance in the onset and persistence of pathological pain. The study of spinal mechanisms of modulation of nociceptive signaling through CB1 receptors may be a promising way to develop new analgesics.

Key words: nociception, pain, peripheral afferent fibers, dorsal root ganglia, spinal cord, dorsal horn, neuropathy, modulation of synaptic transmission, cannabinoid receptor 1