Central sensitization as a basic mechanism for craniofacial pain

Hyperalgesia and allodynia are basic process involves with pathophysiology of pain. In addition to recent advancement in receptor physiology in TRPV1 (activated by capsaicin) TRPA1 (activated by mustard oil) receptors, inflammatory irritants can activate central nociceptive neurons in subnucleus caudalis. This process is called as central sensitization that includes enlarged receptive fields, reduction of activation threshold and increased responses to noxious stimulation. Excitatory amino acid glutamate) is a prime transmitter at the central synapse. Recently, ATP (acts through purinergic receptor, P2X3) could facilitates glutamate release from presynaptic ends. Furthermore, our recent demonstration astroglia involvement in glutamate production that promotes central sensitization in caudalis nociceptive neurons. The implications for the pain processing associated with central sensitization will be discussed.