relation between SCs and TN mainly involves two aspects.Increased Ca2th influx leads to increased release of the excitatory neurotransmitter, Glu, which activates NMDA and a-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) receptors, leading to hyperalgesia and hypersensitivity.70 NGF expression is upregulated in injured SCs.Given the presence of CGRP receptors on SCs, prolonged exposure to CGRP, e.g. after nerve damage, increases release of IL-1b, 39 which induces hypersensitivity to pain and subsequently pathologic neuralgia SP is widely found in the peripheral nervous system and is an important molecule that mediates the production and conduction of pain; moreover, it plays an important role in neuropathic pain.43 Its biological function is mediated by the activation of three different neurokinin (NK) receptors, namely, NK1, NK2, and NK3, and has a preferential affinity for the NK1 receptor.44 Large amounts of SP can directly induce sensory neuron excitability, which leads to hyperalgesia.43 SP, which is the main messenger of harmful information, can be synthesized by TG cells and delivered to the trigeminal sensory nucleus and the peripheral head and face through central and peripheral synapses, respectively.45,46 Studies have reported that after nerve injury, SCs synthesize and release NGF, which can promote SP synthesis.47 Moreover, treatment with SP receptor blockers can alleviate TN caused by infraorbital nerve injury.43 As the upstream SP signal source, activated SCs release a series of neurotrophic factors, cytokines, and extracellular matrix, which can increase the local SP levels and cause TN. In summary, we present a pathway of the involvement of SCs in TN. After peripheral nerve injury, TNF-a expression increases;66 then, TNF-a binds to TNF-a receptors (TNFR) on the SCs and induces SCs to express the P2X4 receptor;67 P2X4 receptor is activated by ATP which is released by neurons and SCs to mediate Ca2th influx and increased BDNF release.68,69 BDNF binds with the trkB receptor that leads to KCC2 downregulation and NMDA receptor upregulation, which lays the foundation for pain production.SP activates the NK1 receptor, while CGRP inhibits SP degradation, leading to TN. Moreover, NGF can promote BDNF expression and thus cause .TN

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