DISSECTING THE MECHANISMS OF CHRONIC POSTSURGICAL PAIN
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Chronic postsurgical pain is a well-recognized complication affecting at least 10% of patients undergoing common operations, such as hernia repair, mastectomy and joint replacement surgery.\(^1,2\)

Emerging data suggest that neuronal plasticity after surgical trauma is the key to the development of chronic postsurgical pain.\(^2,3\) Following incision, there is an increase in neuronal excitability and remodeling of peripheral nociceptors, resulting in paresthesia and allodynia.\(^11,12\) There is also crosstalk between the immune system and neural circuitry, where pain signaling molecules from nearby inflammatory cells are released to perpetuate hyperalgesia (primary hyperalgesia). A large number of pro-nociceptive mediators are involved in this process, such as pro-inflammatory cytokines, IL1ß, IL6, TNFα, reactive oxygen species, nitric oxide synthase, monocyte chemotactic protein 1 and cyclooxygenase. These mediators increase excitatory postsynaptic current directly or by an indirect mechanism, through their interactions with other receptors, such as the NMDA receptor.

With an intense barrage of pain signals, there is induction of central sensitization with synaptic plasticity and amplification of pain trafficking. This is followed by a spread of amplified response beyond the primary site of injury leading to wind-up and secondary hyperalgesia. Although many of these events involve post-translational modification of preexisting proteins in the dorsal horn, altered gene transcription may produce structural and functional changes in the pain pathway and contribute to persistent maladaptive pain behavior.\(^4\) In this talk, I will discuss the various ways that transcriptional regulation may lead to chronic postsurgical pain.

REFERENCES


