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The Transition of Acute Postoperative Pain to Chronic Pain: An Integrative Overview of Research on Mechanisms

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Abstract

The nature of the transition from acute to chronic pain still eludes explanation, but chronic pain resulting from surgery provides a natural experiment that invites clinical epidemiological investigation and basic scientific inquiry into the mechanisms of this transition. The primary purpose of this article is to review current knowledge and hypotheses on the transition from acute to persistent postsurgical pain, summarizing literature on clinical epidemiological studies of persistent postsurgical pain development, as well as basic neurophysiological studies targeting mechanisms in the periphery, spinal cord, and brain. The second purpose of this article is to integrate theory, information, and causal reasoning in these areas. Conceptual mapping reveals 5 classes of hypotheses pertaining to pain. These propose that chronic pain results from: 1) persistent noxious signaling in the periphery; 2) enduring maladaptive neuroplastic changes at the spinal dorsal horn and/or higher central nervous system structures reflecting a multiplicity of factors, including peripherally released neurotrophic factors and interactions between neurons and microglia; 3) compromised inhibitory modulation of noxious signaling in medullary-spinal

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