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Short Commentary

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Central Sensitization: A Generator of Pain Hypersensitivity by Central Neural Plasticity

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Acute nociceptive pain is that physiological sensation of hurt that results from the activation of nociceptive pathways by peripheral stimuli of sufficient intensity to lead to or to threaten tissue damage (noxious stimuli). Nociception, the detection of noxious stimuli is a protective process that helps prevent injury by generating both a reflex withdrawal from the stimulus and as a sensation so unpleasant that it results in complex behavioral strategies to avoid further contact with such stimuli. An additional important phenomenon that further enhances this protective function is the sensitization of the nociceptive system that occurs after repeated or particularly intense noxious stimuli, so that the threshold for its activation falls and responses to subsequent inputs are amplified. In the absence of ongoing tissue injury, this state of heightened sensitivity returns over time to the normal baseline, where high-intensity stimuli are again required to initiate nociceptive pain; the phenomenon is long lasting but not permanent.

The nociceptor-induced sensitization of the somatosensory system is adaptive in that it makes the system hyperalert in conditions in which a risk of further damage is high, for example, immediately after exposure to an intense or damaging stimulus. This sensitization is the expression of use-dependent synaptic plasticity triggered in the central nervous system (CNS) by the nociceptor input and was the first example of central sensitization, discovered 26 years ago. Since then, we have learned that a number of different forms of functional, chemical, and structural plasticity can sensitize the central nociceptive system to produce pain hypersensitivity under both normal and pathological circumstances, some of which are persistent.

In many clinical syndromes, pain is no longer protective. The pain in these situations arises spontaneously, can be elicited by normally innocuous stimuli (allodynia), is exaggerated and prolonged in response to noxious stimuli (hyperalgesia), and spreads beyond the site of injury (secondary hyperalgesia). Central sensitization has provided a mechanistic explanation for many of the temporal, spatial, and threshold changes in pain sensibility in acute and chronic clinical pain settings and has highlighted the fundamental contribution of changes in the CNS to the generation of abnormal pain sensitivity.

Although phenomenologically central sensitization may appear to be comparable to peripheral sensitization, it differs substantially, both in terms of the molecular mechanisms responsible and its manifestation. Peripheral sensitization represents a reduction in threshold and an amplification in the responsiveness of nociceptors that occurs when the peripheral terminals of these high-threshold primary sensory neurons are exposed to inflammatory mediators and damaged tissue and, in consequence, is restricted to the site of tissue injury.

Although peripheral sensitization certainly contributes to the sensitization of the nociceptive system and thereby to inflammatory pain hypersensitivity at inflamed sites (primary hyperalgesia), it nevertheless represents a form of pain elicited by activation of nociceptors, albeit one with a lower threshold due to the increased peripheral transduction sensitivity, and generally requires ongoing peripheral pathology for its maintenance. Peripheral sensitization appears to play a major role in altered heat but not mechanical sensitivity, which is a major feature of central sensitization.

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