

Pain After the Injury: When Neural Signaling Becomes Self-Sustaining

Paper 29 · Editorial concept paper · Created 15 October 2026

Abstract. Central and peripheral sensitization can amplify nociceptive signaling and persist after an initial injury improves. This paper distinguishes altered pain processing from claims that pain is imaginary and explores the importance of nervous-system plasticity alongside ongoing tissue pathology.

Pain and injury are not identical variables

Nociceptive activity commonly signals tissue threat, but pain is a nervous-system experience shaped by peripheral input and central processing. After intense or repeated nociception, neurons in pain pathways can become more responsive. Central sensitization can produce amplified responses, pain from normally non-painful stimuli and spread of sensitivity. These responses are physiological, not evidence that the patient invented or chose the pain.

How a protective alarm remains activated

Early pain may protect an injured site from further damage. Persistent amplification can outlast much of the initial peripheral lesion. The nervous system can change synaptic efficacy, inhibitory balance and the significance assigned to sensory input. Peripheral inflammatory and immune signals may contribute in some conditions. Distinct syndromes involve different combinations of peripheral generators and central amplification, so a single 'brain pain' label is inadequate.

The causal ambiguity

A patient with persistent pain may have active tissue pathology that deserves treatment, altered neural gain, or both. Simply telling patients their pain is sensitization can miss fractures, tumors, neuropathy, infection or inflammatory disease. Conversely, repeated structural imaging may fail to explain amplified symptoms, and unnecessary procedures can bring their own harms. Mechanistic humility is essential.

Testing the proposition

Sensory testing, characteristic clinical findings and carefully interpreted research measures can support a sensitization hypothesis. No single common symptom conclusively establishes central sensitization in an individual. The critical prediction is that pain intensity and functional limitation can sometimes remain disproportionate to an identifiable current tissue lesion because processing has changed. Improved central or peripheral modulation should be judged by meaningful patient outcomes.

Treatment is not a proof of etiology

A patient's improvement with exercise adaptation, psychological therapy, medications or nervous-system-focused care does not establish that the original illness was psychological. Several approaches can affect common final pathways regardless of the initial cause. Similarly, failure of one intervention does not prove irreversibility.

A broader lesson

Pain is a particularly clear example of biological history altering present function. Neural pathways can retain a changed gain setting after the initiating injury has diminished, but that insight should expand compassionate clinical assessment rather than become an excuse to dismiss ongoing pathology.

Sensitization is measurable, but not a universal explanation

Allodynia, hyperalgesia and altered temporal summation can arise from changes in nociceptive processing. Such observations provide real physiological content to the sensitization concept. But many chronic pain disorders also have ongoing peripheral generators, and in some patients those remain dominant. A responsible account does not substitute neural amplification for a needed investigation of structural, inflammatory or malignant causes.

Memory without psychological blame

The nervous system changes with experience through plasticity. Persistent changes in gain or inhibitory control can outlast the original noxious event. Calling this a form of biological memory does not mean the patient chose to remember pain or failed to forgive an injury. Emotional context and attention can modulate pain while peripheral pathology remains genuine. The model should therefore improve compassion, not encourage diagnostic dismissal.

The clinical target is function as well as intensity

Pain treatment can aim to reduce symptom burden, improve mobility, restore sleep and increase participation in ordinary life. These outcomes may change at different rates. A patient who regains function while still experiencing some pain has made real progress; a patient whose pain score decreases while medication harms cognition has experienced a tradeoff. Such multidimensional evaluation fits the broader recovery framework of this volume.

Selected scientific reading

1. [Woolf CJ. Central sensitization: implications for the diagnosis and treatment of pain. Pain. 2011;152\(Suppl\):S2–S15.](#)
2. [Netea MG et al. Defining trained immunity and its role in health and disease. Nat Rev Immunol. 2020;20:375–388.](#)

Editorial status: Conceptual scientific essay, not peer reviewed. Not original clinical data, a treatment protocol or medical advice. Selected readings are not a systematic review. Evidence and interpretation should be assessed independently.