

The Phantom Injury Hypothesis

Absurd Health Institute · Hypothesis and mechanistic perspective

Central hypothesis

An injury can disappear on imaging while pain, guarding or threat responses continue. This paper asks whether peripheral sensory nerves, local immune cells and tissue mechanics sometimes preserve a response to an injury that is no longer present. Phantom injury is not a new diagnosis: it is a proposed multi-compartment account of selected persistent symptom patterns.

The biological route

Peripheral nerve injury may alter excitability; spinal and supraspinal networks can undergo sensitization; macrophages, mast cells and glial cells influence nociception; extracellular matrix stiffness can change mechanical input. Together, these interactions may produce a self-reinforcing loop in which expectation, afferent traffic and local biology each sustain the others. Central sensitization alone is not an explanation for every symptom, but it offers one mechanism for persistence.

A deeper interrogation

Imagine a patient whose old injury site becomes painful during stress, without new structural damage detected by standard examination. Several explanations remain possible: undetected peripheral injury, altered sensitivity of peripheral nerves, spinal amplification, changes in expectation, or combinations of these. An intellectually honest model cannot choose its preferred story from symptom persistence alone. The phantom-injury framework becomes useful only when it predicts which perturbation should change the symptom and which should not. It also must make sense of spatial specificity. Why would one shoulder or abdominal quadrant remain sensitized while adjacent tissue behaves normally? Resident immune-cell phenotypes, nerve branching, scar mechanics and cortical representation offer different causal routes. A strong paper should compare these possibilities rather than bundle them under “memory.” One possible discriminating prediction is that a subset of patients has persistent local nociceptive activity that can be observed independently of the central experience of pain. In another subset the dominant driver may be central gain or learned threat response. Neither patient is imagining the symptom. They inhabit different physiological explanations that should not be collapsed into one label.

Alternative explanations and limits

Ongoing occult structural disease, neuropathy, inflammatory arthritis, fracture and neuropathic injury must not be dismissed because imaging appears unremarkable. Imaging sensitivity, test selection and physical examination differ by disorder; absence of a visible lesion does not equal absence of pathology.

What would change the conclusion?

A discriminating account should separate tissue-derived nociceptive input from centrally maintained amplification using repeated objective evaluation and carefully interpreted response patterns. The hypothesis weakens if symptoms consistently map to an active peripheral pathology without independent evidence for persistent feedback.

The distinctive prediction

Spatially restricted symptoms create a stronger test than generalized discomfort: can local tissue measurements explain why one region retains the painful response? If not, circuit-level or systemic explanations may be more powerful.

Clinical interpretation

The clinical value is avoiding a false choice between “all tissue” and “all psychology.” Pain is experienced through a nervous system but can have complex and changing biological inputs. No single imaging result settles causation.

Selected scientific reading

Contextual background, not direct validation of the proposed hypothesis.

Astrocyte regulation of synaptic signaling in psychiatric disorders. *Front Mol Neurosci*. 2022.
<https://pmc.ncbi.nlm.nih.gov/articles/PMC9700696/>

Fibroblast Yap/Taz Signaling in Extracellular Matrix Homeostasis and Tissue Fibrosis. *Int J Mol Sci*. 2024.
<https://pubmed.ncbi.nlm.nih.gov/38929890/>

Editorial note: Original theoretical perspective, not an original clinical study, validated diagnosis, or treatment protocol.