

The Geometry of Chronic Pain

Absurd Health Institute · Hypothesis and mechanistic perspective

Central hypothesis

Chronic pain may sometimes be sustained by the geometry of local tissue: stiffness, pressure, fascial or scar organization, and altered nerve exposure. The provocative idea is that spatial arrangements can maintain peripheral input and interact with central sensitization even after the initiating injury ends.

The biological route

Peripheral nociceptors respond to mechanical and chemical environments. Fibrotic matrix changes traction and stiffness; scar formation can alter nerve movement; persistent inflammation influences channel expression; repeated nociceptive input can shape spinal and supraspinal gain. Geometry should be treated as one candidate source of input, not a universal explanation of pain.

An illustrative scenario

An old scar near a peripheral nerve may plausibly alter mechanical glide and local nociceptive input. A patient with generalized widespread pain, in contrast, may have no similar focal geometry. The tissue model must remain local and specific where the evidence is local and specific. Physical findings, neurological examination, and interpretation of central sensitization should coexist without implying that every chronic pain symptom has a discoverable fascial lesion.

Alternative explanations and limits

Many chronic pain syndromes occur without measurable tissue architectural abnormalities, and visible changes on imaging may be incidental. Psychiatric distress, sleep disruption and central pain processing also matter. Claims that fascia stores emotional trauma literally are not demonstrated by matrix biology.

What would change the conclusion?

A serious investigation would measure regional tissue mechanics and local nerve function, then compare them with pain mapping, temporal fluctuation and response to validated treatments. If structural variation fails to predict pain after controlling for sensitization, the proposed mechanical contribution should be downgraded.

The distinctive prediction

The model should identify a particular nerve-matrix interface where mechanical conditions predict pain and respond in a mechanistically coherent way. Generalized pain without local tissue findings would weigh against a purely geometric explanation.

Clinical interpretation

The framing avoids the false binary of tissue versus brain. Pain can be real, multidetermined and amenable to different treatments without requiring speculation about hidden toxins or a single universal tissue lesion.

Selected scientific reading

Contextual background, not direct validation of the proposed hypothesis.

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

Seth AK, Critchley HD. Extending predictive processing to the body: emotion as interoceptive inference. Behav Brain Sci. 2013.
<https://www.cambridge.org/core/journals/behavioral-and-brain-sciences/article/abs/extending-predictive-processing-to-the-body-emotion-as-interoceptive-inference/A53E081B5EEBD7CF7658F3D484714AFE>

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