

The Wound That Never Received the Message to Stop

Absurd Health Institute · Hypothesis and mechanistic perspective

Central hypothesis

Tissue repair requires as much termination as initiation. A wound might close visibly while persistent fibroblast, macrophage or neural signaling sustains abnormal physiology. The hypothesis is that in some disorders the missing step is not initiation of repair, but successful exit from the repair program.

The biological route

In healing tissue, leukocyte recruitment should give way to resolution, myofibroblast activity should decline, and matrix remodeling should reach an appropriate balance. Stiff extracellular matrix can reinforce YAP/TAZ-dependent fibroblast activation; persistent damage-associated signals may recruit innate immune cells. An initial scar can become an input to ongoing fibrogenesis. This is a physically plausible feedback loop rather than merely a metaphor.

An illustrative scenario

A healed skin wound provides a useful thought experiment: the epithelium has closed, but fibroblast activation may remain abnormal beneath it. If tissue stiffness keeps driving matrix synthesis, then manipulating the original inflammatory trigger may not reverse the new mechanical loop. If the tissue is simply a mature, inactive scar, the mechanism is different. The essay must therefore distinguish ongoing pathology from permanent but biologically quiet structural change.

Alternative explanations and limits

Continued inflammation may reflect a real ongoing insult such as infection, mechanical stress or autoimmunity. Fibrosis can also be an irreversible consequence rather than an active self-sustaining process. A visible scar does not prove pathological signaling is still occurring.

What would change the conclusion?

The model predicts persistent molecular or mechanical activation after the initiating lesion is controlled, and responsiveness to interruption of the feedback circuit rather than repeated removal of triggers. If signaling ends promptly yet dysfunction remains due solely to fixed architecture, the model needs revision.

The distinctive prediction

The most distinctive sign would be pathological fibroblast activation that persists after the primary inflammatory stimulus is controlled but decreases when a specific matrix-feedback pathway is interrupted. An inactive mature scar would not meet this definition.

Clinical interpretation

The concept reframes some recovery failures as termination failures while preserving the need to treat continuing infection, ischemia and other causes of tissue damage. Anti-fibrotic strategies must be disease-specific and clinically validated.

Selected scientific reading

Contextual background, not direct validation of the proposed hypothesis.

Liu F et al. Mechanosignaling through YAP and TAZ drives fibroblast activation and fibrosis. *Am J Physiol Lung Cell Mol Physiol*. 2015.
<https://pmc.ncbi.nlm.nih.gov/articles/PMC4329470/>

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.