

# The Tumor as a Wound That Refuses to Close

Absurd Health Institute · Hypothesis and mechanistic perspective

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## Central hypothesis

Tumors often recruit programs also seen in wound healing: angiogenesis, stromal activation, extracellular matrix remodeling and immune-cell recruitment. The “wound that refuses to close” analogy asks whether persistent repair-like signaling becomes part of the ecology sustaining cancer, without claiming that injury alone causes malignancy.

## The biological route

Malignant clones acquire mutations and epigenetic changes that alter proliferation and survival. Transforming growth factor beta, growth factors, immune-cell cytokines and activated fibroblasts can influence the local environment. Hypoxia and mechanical matrix signals may shape invasion and therapy response. Cancer cells can exploit these ordinary tissue programs while remaining fundamentally abnormal clonal populations.

## An illustrative scenario

A tumor with a dense desmoplastic stroma may exhibit changes in drug delivery and immune access. But the first question remains the tumor's cellular pathology and molecular behavior. A wound-healing analogy gains value only when it identifies a particular stroma-dependent mechanism that explains a clinical observation. It should never be used to suggest that malignancy is merely an unhealed wound that can be closed by lifestyle measures.

## Alternative explanations and limits

The analogy must not displace genetic causation, tumor evolution or the fact that many wounds heal without cancer. Persistent wound signaling can be an effect of malignancy, not its original cause. Distinct cancers use different stromal dependencies.

## What would change the conclusion?

One should specify a tumor subtype and ask whether a defined wound-repair pathway predicts tumor progression or therapeutic resistance after controlling for tumor genotype and stage. If manipulating the pathway has no independent effect, it is not a useful sustaining mechanism.

## The distinctive prediction

A repair-program explanation matters only if a defined stromal or inflammatory pathway predicts tumor behavior beyond malignancy-specific genotype and stage. Otherwise it remains an analogy that offers no therapeutic leverage.

## Clinical interpretation

The idea informs oncological research but is not evidence that fasting, anti-inflammatory supplements or a healthier “terrain” can replace effective cancer treatment.

## 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/>

Colom B et al. Mutant clones in normal epithelium outcompete and eliminate emerging tumours. *Nature*. 2021.  
<https://www.nature.com/articles/s41586-021-03965-7>

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