

Hysteresis: Why Removing the Cause May Not Reverse the Disease

ABSURD HEALTH INSTITUTE | 15 OCTOBER 2026 | I. Biology of disease states

Abstract

This concept paper examines hysteresis: why removing the cause may not reverse the disease. It develops an argument drawn from Genes, Processes, and Constraints, identifies the relevant biological or clinical mechanisms, and considers where that argument may fail. This is a narrative perspective, not a systematic review or an original clinical study.

The central problem

In systems with hysteresis, the current state depends partly on the path by which it was reached. A disease can remain clinically active after a precipitating event ends because the event produced durable tissue injury, learned regulatory patterns, altered ecological relationships or self-sustaining feedback. These are different mechanisms and should not be conflated. Hysteresis is a rigorous way to ask why the pathway into a disorder may differ from the pathway out.

Mechanisms and causal architecture

Examples include persistent airway remodeling after repeated inflammation, conditioned fear after a threat has passed, or glucose-control changes after prolonged metabolic stress. In each case, temporary exposures can leave enduring substrates. Network feedback may reinforce the substrate: behavioral avoidance prevents corrective learning; chronic inactivity sustains deconditioning; inflammation may remodel tissue and alter local responsiveness. The point is not that every symptom is a protective memory but that treatment may have to address the newly established maintenance conditions, not just remove the original trigger.

A discriminating example

After a viral infection has resolved, fatigue can persist because of tissue injury, dysautonomia, inflammatory sequelae, sleep disturbance or ongoing disease. The disappearance of detectable virus cannot distinguish these possibilities. Calling the persistence hysteresis is only informative if the paper identifies what was left behind and shows how that mechanism sustains symptoms. This distinction matters because different persistence mechanisms require different responses.

Competing explanations and limitations

Persistent disease is also explained by undetected ongoing pathology. A clinician must not treat worsening asthma, infection, malignancy or neurological decline as harmless “system memory.” Furthermore, many phenomena are better described by irreversible tissue damage than by bistability or genuine hysteresis. Calling both an attractor can obscure whether a proposed intervention could plausibly restore function.

What evidence would change the interpretation?

A stringent test compares matched states reached from different histories and measures whether future trajectories diverge despite similar present inputs. Longitudinal response curves, controlled stepwise exposure changes, and mechanistic readouts are more informative than an evocative before-and-after narrative. If current pathology and ongoing exposures account fully for outcomes, invoking hysteresis adds little.

Implications for medical interpretation

This framework offers a patient-respecting explanation for incomplete recovery without suggesting they have failed. It encourages treatment plans to distinguish ongoing cause, residual injury and reversible maintenance loops, and it gives remission and relapse more precise meanings.

Selected scientific reading

Barabási, Gulbahce & Loscalzo (2011), Network medicine: a network-based approach to human disease, Nature Reviews Genetics.

<https://doi.org/10.1038/nrg2918>

McEwen (1998), Protective and damaging effects of stress mediators, New England Journal of Medicine.

<https://doi.org/10.1056/NEJM199801153380307>

Original source: Genes, Processes, and Constraints. All source books supplied by the author.

Conceptual research commentary. Not a peer-reviewed clinical study, original trial, systematic review, validated diagnostic instrument or treatment protocol. Consult qualified care for medical decisions.