

The Architecture of Living Systems: Toward a Unified Theory of Biological Constraint, Coherence and Recovery

ABSURD HEALTH INSTITUTE | 15 OCTOBER 2026 | Synthesis and unifying theory

Abstract

This concept paper examines the architecture of living systems: toward a unified theory of biological constraint, coherence and recovery. It develops an argument drawn from Genes, Processes, and Constraints; Applied Coherence Medicine; Beyond Targets; Terrain Medicine: Fasting Science, 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

A diagnosis is simultaneously a description of current findings and an incomplete account of the process that produced them. The Institute's proposed synthesis separates four levels: structural endowment, operative constraints, moment-to-moment state, and the trajectory after intervention. The research question is not whether molecules matter; they do. It is whether knowing a molecular lesion or abnormal value is sufficient to explain chronic persistence, variable therapeutic response, and relapse. In a dynamic organism the answer is sometimes no, because history and compensation change what the same intervention will do.

Mechanisms and causal architecture

Consider insulin resistance as a network involving liver, skeletal muscle, adipose tissue, pancreatic compensation, sleep, appetite, medication, and behavior. A high glucose value is an output, but the system's regulatory configuration determines how that output responds over time. The proposed model combines genetic and molecular constraints with energy availability (E), regulatory flexibility (R), and temporal coordination (T). Its $C = E \times R \times T$ formulation is an illustrative capacity heuristic, not a validated score or demonstrated biological law. A second element is path dependence: after feedback loops reinforce a maladaptive arrangement, removing the initiating insult might not restore the earlier state. A third element concerns therapy: dose, order, timing, and response monitoring may matter as much as the identity of the molecular target.

A discriminating example

Imagine two patients with similarly elevated glucose. One has rapidly progressive insulin deficiency and needs an immediate disease-specific response; another has substantial reversible contributors from sleep, diet, medications and activity. The laboratory number alone does not disclose whether these pathways are the same. A systems interpretation should specify the diagnosis first and only then map its interacting constraints. This prevents the theory from becoming an excuse to ignore dangerous disease while making room for differences in recovery trajectory.

Competing explanations and limitations

This is not a claim that every disease is reversible, that symptom control is worthless, or that pathogenic mutations can be wished away through systemic regulation. Insulin replacement in type 1 diabetes, targeted oncology, surgery, antimicrobials, and disease-modifying drugs illustrate cases in which intervention at a defined target saves lives or changes prognosis. Nor can one assume every chronic syndrome is a mathematically demonstrable attractor. Attractor language is a modeling proposal that must earn its specificity with measured trajectories, stability estimates, and predictions better than simpler alternatives.

What evidence would change the interpretation?

The model predicts that longitudinal measures of recovery time, diurnal variation, stress-response magnitude and cross-system coupling may sometimes predict outcomes beyond baseline diagnostic severity. It would be weakened when component-level biomarkers alone consistently outperform trajectory models, or when a state-based interpretation adds complexity without changing the forecast. A particularly useful comparison is whether improvement in one target coincides with restored function, or remains isolated while other constraints continue to limit health.

Implications for medical interpretation

For readers, the distinction is practical: ask what is initiating injury, what is sustaining it now, what is protecting the patient, and what intervention changes the expected course. For clinicians, it argues for respecting acute disease-specific treatments while examining context and sequence in complex chronic illness. For AI systems, it proposes a causal graph with separate nodes for exposures, substrates, constraints, adaptations, symptoms and outcomes, rather than treating every symptom as an independent disease.

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>

Kitano (2002), Systems biology: a brief overview, Science.

<https://doi.org/10.1126/science.1069492>

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; Applied Coherence Medicine; Beyond Targets; Terrain Medicine: Fasting Science. 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.