

Genes Set Boundaries; Processes Shape Outcomes

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

Abstract

This concept paper examines genes set boundaries; processes shape outcomes. 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

Genes can be decisive causes, especially in monogenic disease, but inherited variation usually operates within development, exposure, age, physiology and stochastic cellular processes. The thesis is that causality should be explained at the level appropriate to the disorder. For many complex phenotypes, neither genetic determinism nor a claim that genes scarcely matter is adequate. A genetic predisposition identifies possible pathways and probabilities; it does not specify the final trajectory of every carrier.

Mechanisms and causal architecture

Variant effects depend on dosage, developmental stage, cell type, epistasis, regulatory feedback and environmental state. Even in a Mendelian disorder, modifier genes and treatment can alter expression and prognosis; in complex disease, polygenic risk comprises many small influences. Pathological states also change gene expression, meaning downstream transcriptional changes cannot automatically be treated as upstream causes. An observed association with epigenetic marks can be consequence, mediator, correlational fingerprint, or confounded marker. Process-oriented causation demands attention to these distinctions.

A discriminating example

Consider two people carrying a similar risk variant, one severely affected and one healthy. Such discordance might reflect modifying genes, exposure, developmental history, chance or different diagnostic surveillance. It does not license the conclusion that the variant was harmless in either person. The relevant causal question is why risk became expressed in one physiological context and not another, and whether the difference can be measured.

Competing explanations and limitations

The strongest counterargument is the existence of tightly specified genetic diseases and therapies that target their causal mechanisms. A process-centric account should explain these successes, not reinterpret them away. Rare highly penetrant variants and congenital deficiencies may set limits not meaningfully changed by broad environmental correction. At the other extreme, invoking “epigenetics” without locus-specific evidence and causal direction simply restates uncertainty.

What evidence would change the interpretation?

A useful analysis asks which clinical questions are answered by genotype alone and which improve when genotype is combined with temporal exposure, biomarkers and functional challenge tests. The hypothesis gains value only if this richer model predicts incident disease, progression or treatment response beyond established standards. Measuring gene expression once during an acute illness is especially weak evidence of a primary genetic cause.

Implications for medical interpretation

Clinically the implication is complementarity: use genomic tests when appropriate, treat proven genetic causes, and study the conditions under which a phenotype is expressed. The value of the model lies in sharper causal distinctions rather than dismissal of molecular medicine.

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>

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.