Nonergodic Development:

How High-Dimensional Systems with Low-Dimensional Anchors Generate Phenotypes

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Abstract

Biological development is a high-dimensional dynamical process that cannot explore its state space in finite time—it is *nonergodic*. We argue that this nonergodicity, combined with low-dimensional genetic anchors, is the fundamental reason why genotype does not algorithmically determine phenotype. The genome constrains which regions of developmental state space are reachable, but environmental history determines which attractor basin the system occupies. Using a minimal developmental network model, we demonstrate that (1) identical genotypes produce substantially different phenotypes depending on which trajectory the system follows, (2) these trapped states constitute "developmental memory" that is invisible to genetic analysis, and (3) the "dimensional gap" Δ_D between genetic parameters and developmental degrees of freedom quantifies this non-identifiability. We apply this framework to recent findings on cooperative lifestyles and cancer prevalence, showing that the pattern "cooperative species have less cancer" admits both allele-based and trajectory-based interpretations. Using Price

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equation decomposition, we demonstrate that "fractal coherence" is a self-stabilizing attractor: cooperative groups construct the low-volatility niches required to maintain high-coherence developmental trajectories, creating a scale-free feedback loop from cells to societies. The models diverge only under environmental intervention. Allele stories are not wrong but are projections that discard the nonergodic structure of development.

1   Introduction

Biological development unfolds in a high-dimensional state space. Gene regulatory networks, signaling cascades, and metabolic pathways create a system with vastly more degrees of freedom than the genome that parameterizes it. This dimensionality has a fundamental consequence: the system is *nonergodic*—it cannot explore its state space in biological time. A developing organism follows one trajectory through developmental state space and ends up in one attractor basin, leaving the vast majority of possible states forever unvisited.

This nonergodicity is not a limitation to be overcome but a feature that enables stable phenotypes. The genome acts as a low-dimensional *anchor* that constrains which regions of state space are reachable, while environmental history determines which specific attractor the system occupies within those constraints. The phenotype is not algorithmically determined by the genotype; it is the trapped state of a nonergodic system whose trajectory was shaped by both genetic and environmental inputs.

This framework offers one perspective on a persistent puzzle in genetics: why do genome-wide association studies fail to account for the majority of heritable variation in complex traits [8, 9]? Many explanations have been proposed—rare variants, epistasis, gene-environment interactions. We add another: trajectory information. From a nonergodic developmental perspective, "missing heritability" includes the environmental history that

determined which attractor basin each individual occupies. Additive genetic models project a high-dimensional, trajectory-dependent reality onto a low-dimensional genotype space, discarding precisely the information that distinguishes individuals with identical genotypes.

We formalize this via the **Dimensional Gap** (Δ_D), which quantifies the mismatch between genetic parameters and developmental degrees of freedom. When $\Delta_D \gg 0$, the mapping from genotype to phenotype is many-to-one: the same genotype can produce different phenotypes depending on which trajectory the system followed. This creates a fundamental *non-identifiability* between allele-based and trajectory-based explanations for phenotypic patterns.

Application: Cooperative Lifestyles and Cancer

We apply this framework to a recent finding in cancer evolution. Sierra et al. (2025) demonstrated that cooperative mammalian species exhibit lower cancer prevalence than competitive species, and modeled this as selection on "oncogenic variants" that function as a mechanism of programmed obsolescence—the "Hydra effect"—in competitive populations [1]. Their allele-based interpretation is compelling—but our framework shows it is not uniquely supported by the data.

The same pattern admits a trajectory-based interpretation:

- **Allele interpretation:** Cooperative lineages have accumulated cancer-suppressing alleles through selection.

- **Trajectory interpretation:** Cooperative environmental cues enable slower, more coordinated development with fewer attractor bifurcations, yielding lower cancer mortality as an emergent property.

These interpretations are *non-identifiable* from cross-species comparative data. They diverge only in predictions for intervention: the allele model predicts that changing an organism's environment will not change its cancer risk; the trajectory model predicts substantial

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phenotypic shifts.

This connects to Lissek's [20] proposal of "cancer memory"—epigenetic mechanisms that maintain malignant phenotypes once initiated. Our framework situates epigenetics within a larger dynamical picture: the high-dimensional developmental system determines *which* epigenetic marks get laid down; epigenetic mechanisms then stabilize the resulting attractor. Epigenetics is not a competing explanation but the molecular implementation of nonergodic trapping. The trajectory through developmental state space creates the epigenetic pattern; the pattern then helps maintain the trapped state.

In this paper, we:

1. Develop the theoretical framework of nonergodic development with low-dimensional anchors (Section 2).

2. Formalize a minimal developmental network model that demonstrates these principles (Section 3).

3. Prove a non-identifiability proposition showing that allele-based patterns can always be matched by trajectory-based mechanisms (Section 4).

4. Demonstrate the framework with simulations, including a "Twin Worlds" experiment (Section 5).

5. Discuss implications for evolutionary biology, GWAS, and intervention design (Section 6).

2 Theoretical Framework

2.1 Nonergodicity in High-Dimensional Systems

A dynamical system is *ergodic* if, given sufficient time, its trajectory will visit every accessible region of state space with frequency proportional to the equilibrium measure. Ergodicity

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underlies much of statistical mechanics: it allows us to equate time averages with ensemble averages, and justifies treating a single long trajectory as representative of the whole system [32].

A subtlety: high dimensionality alone does not guarantee nonergodicity. A liter of air at room temperature is extraordinarily high-dimensional ($\sim 10^{23}$ molecules, each with position and momentum), yet statistical mechanics treats it as ergodic and succeeds. The difference is *structure*. Air molecules are interchangeable; their specific configuration carries no information; the system equilibrates rapidly and forgets its history. Biological systems are different: cells are not interchangeable, specific configurations matter, and developmental history is retained in the system's structure.

The relevant criterion is: **high dimensionality + structured coupling + information retention = nonergodicity**. When the system's state carries information that must be preserved—when configurations are not equivalent—then exploration becomes constrained. State space volume still grows exponentially with dimension (b^D for some base b), trajectory length still grows linearly with time, but now the system cannot simply diffuse through phase space. It must maintain coherence. It becomes trapped.

Biological systems satisfy all three conditions. A gene regulatory network with N genes has k^N possible expression states, but these states are not equivalent—they correspond to different cell types, different developmental stages, different functional outcomes. The system is strongly coupled through regulatory feedback; it retains information in its epigenetic state; it cannot equilibrate without losing its identity. Development, behavior, physiology, and cognition all unfold in state spaces too vast to explore and too structured to forget. High-dimensional biological systems are nonergodic by necessity, not by accident.

Definition 1 (Developmental Nonergodicity)**.** A developmental system is **nonergodic** if its trajectory through state space cannot visit a representative sample of accessible states within the organism's lifetime. Formally, let $\mathcal{M}$ be the accessible manifold of developmental

states. The system is nonergodic if:

$$\frac{\text{Vol(trajectory)}}{\text{Vol}(\mathcal{M})} \to 0 \qquad (1)$$

as the dimensionality of $\mathcal{M}$ increases.

This nonergodicity has a crucial consequence: the developmental trajectory is *trapped*. Once the system enters an attractor basin, it cannot spontaneously explore alternative basins. The final phenotype reflects not the equilibrium distribution over possible states, but the particular basin the trajectory happened to enter.

2.2 Beyond the Computational Metaphor

This continuous dynamical systems perspective differs fundamentally from the dominant metaphor in molecular biology. Since the discovery of the genetic code, biological systems have been described using computational language: genes "encode" proteins, regulatory networks "process" signals, development follows a "program." This framing—treating organisms as discrete-state machines executing algorithms—has been so successful and so pervasive that it is rarely questioned. Network diagrams with nodes and edges are the standard visual vocabulary; information flow through discrete processing steps is the default conceptual framework.

Yet the graph model carries a hidden puzzle: how do discrete signals get routed precisely through the molecular chaos of a living cell? The network diagram shows an edge from node A to node B, but physically there is no edge—only molecules diffusing in crowded cytoplasm.

We propose a complementary perspective grounded in continuous physics. Instead of discrete nodes and edges, we have continuous state spaces and smooth trajectories. Instead of algorithms that map inputs to outputs, we have dynamical systems that *flow* through high-dimensional landscapes. The genome does not "compute" the phenotype—it shapes the landscape through which the developmental trajectory flows. The routing puzzle dissolves:

there are no discrete signals being routed, only continuous trajectories constrained by the physics of the substrate.

This shift matters because computational metaphors implicitly assume something like ergodicity. Here is why: algorithms are *time-abstract*. A Turing machine will eventually halt with its output; we analyze what it computes, not how long it takes. This abstraction implies that given any input, the system can "find" the corresponding output—it can reach the correct state. When we model the genotype-phenotype map as a computation, we inherit this assumption: the genome specifies an algorithm; development executes it; the phenotype is the output. The system is assumed capable of reaching whatever state the algorithm specifies.

But physical systems embedded in time cannot do this. Continuous high-dimensional dynamics reveals why: the state space is too vast; trajectories are trapped. A developing organism does not "find" its phenotype the way an algorithm finds its output. It falls into an attractor basin determined by its trajectory. The phenotype is not the output of a computation but the basin into which a physical system settled.

The distinction is not merely philosophical. Algorithms are abstractions that can, in principle, explore any computable state space given sufficient steps. Physical dynamical systems cannot. When dimensionality is high, state space volume grows exponentially while trajectory length grows only linearly with time [28]. This is the "curse of dimensionality"—not a computational inconvenience but a fundamental barrier. Bayesian inference becomes intractable as posteriors concentrate on measure-zero manifolds [29]; MCMC samplers fail to mix as dimension increases [30]; even exhaustive search becomes impossible. Levinthal's paradox [31] made this vivid for protein folding: a polypeptide cannot explore all conformations, so folding must follow constrained trajectories through funnel-shaped landscapes. The same logic applies to development, at a vastly larger scale.

2.3 Low-Dimensional Anchors

If developmental state space is vast and trajectories are trapped, what prevents phenotypic chaos? The answer is *anchoring* by low-dimensional constraints.

Definition 2 (Developmental Anchor)**.** An **anchor** is a low-dimensional structure that constrains which regions of developmental state space are reachable. The genome is the primary anchor, specifying regulatory network topology, protein sequences, and signaling pathways. The epigenome provides secondary anchoring, modulating which regions of the genome-defined landscape are accessible at any given time.

Together, genetic and epigenetic anchors define the "landscape" of possible developmental trajectories. They do not specify which attractor basin the system will occupy—that depends on the trajectory, which depends on environmental history. But the anchors constrain *which basins exist* and *which are reachable* from a given starting point.

Crucially, the epigenome is not independent of the trajectory—it is *shaped by* it. The high-dimensional developmental dynamics determine which epigenetic modifications occur; these modifications then feed back to constrain future trajectories. This creates a hierarchy: genome $\rightarrow$ developmental dynamics $\rightarrow$ epigenome $\rightarrow$ stabilized attractor. Epigenetic marks are the molecular record of where the trajectory has been, not an independent source of phenotypic variation.

This is precisely Waddington's [10] epigenetic landscape, reframed in dynamical systems terms: the genome shapes the hills and valleys; the developmental trajectory is a ball rolling down one particular path. Different environmental perturbations can push the ball into different valleys—but not into valleys that the landscape doesn't contain. As Noble [11] argues, there is no privileged level of causation in biological systems; the genome anchors the dynamics, but environmental boundary conditions constrain them just as fundamentally.

2.4 The Dimensional Gap

We quantify the relationship between anchor dimensionality and state space dimensionality:

Definition 3 (Dimensional Gap)**.** Let L be the dimension of the genotype (anchor) space and k the dimension of measured phenotypic traits. Let the developmental system evolve on a manifold $\mathcal{M}$ with **effective dimension** m_{eff}. The **dimensional gap** is:

$$\Delta_D = m_{\text{eff}} - (L + k) \tag{2}$$

When $\Delta_D \gg 0$, the developmental system has far more degrees of freedom than can be specified by the genome or captured by phenotypic measurement. This creates a fundamental ambiguity:

- The same genotype can produce different phenotypes (depending on which trajectory/attractor)

- The same phenotype can arise from different mechanisms (genotype-determined vs. trajectory-determined)

The dimensional gap is the source of non-identifiability. Projecting from the high-dimensional developmental reality to the low-dimensional genotype-phenotype pair destroys the trajectory information that distinguishes otherwise identical outcomes [6, 7].

2.5 Epigenetic Memory as Trapped States

The connection to epigenetic memory is now clear. "Epigenetic memory" refers to heritable cellular states that are not encoded in the DNA sequence—persistent patterns of gene expression, chromatin modification, or cellular identity that are maintained across cell divisions.

In our framework, epigenetic memory is simply a *trapped state* in a nonergodic developmental system. The genome (anchor) constrains which states are stable, while environmental

history determines which stable state the system entered. Once entered, the state persists because the system cannot spontaneously explore alternatives.

This provides the information-theoretic foundation for Lissek's [20] "cancer memory" hypothesis: oncogenic changes may push the developmental system into a new attractor basin, where the nonergodic dynamics maintain the malignant phenotype without requiring continued mutation accumulation.

2.6 Application Context: Cancer as a Failure of Developmental Buffering

Sierra et al. (2025) found that cooperative mammalian species have lower cancer prevalence than competitive species [1]. Their comparative analysis shows that eusocial and cooperative breeders—mole-rats, some primates, and several cetaceans—combine division of reproductive labor with unusually low cancer mortality, suggesting repeated co-evolution of cooperative life histories and cancer resistance. Importantly, Sierra et al. themselves emphasize that cancer evolution should be treated as a *multilevel selection* process and connect their findings to life-history plasticity. Their stage-structured model explains the pattern via an "oncogenic variant" that raises late-life cancer mortality (μ_S): in competitive contexts this variant can increase fitness via the "Hydra effect" (freeing resources for offspring), while in cooperative contexts it is selected against.

Our framework suggests a complementary, trajectory-based mechanism rooted in life history theory [15, 16]. In Sierra et al.'s model, cooperation (α) and competition (ω) change population structure by altering juvenile survival. In ours, the same social conditions are modeled as changes in developmental coherence c: cooperative environments stabilize trajectories (high c, low emergent μ_S), competitive environments destabilize them (low c, high emergent μ_S). The key difference is explanatory level: we are not challenging their ecology, but moving one level down to the developmental substrate of μ_S. Developmental fidelity is metabolically expensive. In competitive, high-volatility environments ("fast" life histories),

organisms must prioritize rapid growth and early reproduction over somatic maintenance, leading to noisier developmental trajectories and more frequent attractor bifurcations [17]. Crucially, this is not the same as "mutation accumulation": the classic experiments of Mintz and Illmensee [18] showed that teratocarcinoma cells injected into mouse blastocysts contributed to normal tissues—coat, liver, even viable sperm—demonstrating that malignancy involved no irreversible genomic damage, only a change in gene expression. Cancer, on this view, is attractor escape: cells finding an alternative stable state, not cells accumulating broken DNA.

Independent comparative evidence supports this link between life-history tempo and cancer vulnerability. In mammals, Boddy et al. [40] found that litter size—a marker of fast life history—predicts cancer prevalence, while body size and lifespan do not (consistent with Peto's paradox). A parallel analysis in birds found that clutch size, but not body mass or lifespan, predicts neoplasia [41]. These patterns point to a reproduction–somatic maintenance trade-off that operates independently of the allele-frequency story. At the within-species level, the social buffering literature shows that supportive relationships dampen HPA-axis reactivity across the lifespan [42], while adverse early environments are associated with chronic inflammation and elevated cancer risk [43]. Crucially, the same neuroendocrine axis links to social behavior: the "dual-hormone hypothesis" shows that testosterone promotes competitive, status-seeking behavior specifically when cortisol is low [44]—the HPA axis modulates both developmental coordination and social strategy. In our language: buffered inputs reduce stochastic kicks that push cellular trajectories across bifurcation thresholds, and the physiological signature of "not defecting" (low HPA reactivity) is shared across scales.

Cooperative lifestyles fundamentally alter this energetic landscape by **buffering environmental variance**. Food sharing, communal defense, and alloparenting create a low-variance "developmental niche." This stability allows the system to adopt a "slow" life history strategy: allocating more resources to high-fidelity DNA repair and coordinated cell

division rather than rapid mass accumulation.

In our dynamical framework, the cooperative environment acts as a **boundary condition** that restricts the trajectory to the center of the attractor basin, preventing the stochastic excursions (bifurcations) that initiate carcinogenesis. Species that consistently experience cooperative environments effectively "outsource" their stability to the group, allowing their internal developmental dynamics to remain coherent without requiring distinct cancer-suppressing alleles for every possible threat. The lower-cancer phenotype is thus an emergent property of the buffered trajectory, not just the genome.

The efficacy of exercise as a cancer intervention provides further evidence for the attractor framework. Exercise does not repair accumulated mutations, yet post-diagnosis physical activity reduces colorectal cancer recurrence by 45% [47]. Exercise normalizes HPA-axis function, reduces systemic inflammation, and remodels the tumor microenvironment [48, 49]. These are systemic interventions that change the attractor landscape, not targeted therapies that fix broken cells. The mutation-accumulation model cannot explain why whole-organism metabolic state affects cancer outcomes; the attractor model predicts it directly: exercise buffers physiological volatility, stabilizing cellular trajectories within the organismal basin.

More generally, any species experiencing variable environments across generations would be expected to evolve genomes that anchor multiple attractor basins—allowing environment-dependent phenotype determination rather than fixed strategies. Singh and Glowacki [2] documented such environmental variability in human evolutionary history, but the principle applies broadly: when environments vary, selection favors trajectory-responsive developmental systems over rigidly canalized ones. In such lineages the effective developmental dimensionality m_{eff} is large relative to $L + k$, so $\Delta_D \gg 0$ and trajectory non-identifiability is unavoidable.

3   Model

We construct a minimal developmental network that instantiates the theoretical framework: the genotype serves as a low-dimensional anchor, the developmental state evolves in a high-dimensional space, and environmental history shapes the trajectory through that space.

3.1   The Developmental System

Let $g \in \mathbb{R}^L$ be the genotype (the *anchor*, with L small), $e_t \in \mathbb{R}^p$ be the environmental input at time t, and $h_t \in \mathbb{R}^m$ be the developmental state (with $m \gg L$).

The developmental dynamics follow a continuous-time recurrent network:

$$\frac{dh}{dt} = -h + \sigma \left(W_h h + W_e e_t + W_g g \right) \tag{3}$$

where W_h, W_e, W_g are weight matrices and $\sigma = \tanh$ provides nonlinearity. The $-h$ term creates intrinsic decay, ensuring the system relaxes toward attractors rather than exploding. In simulations, we discretize using Euler's method with step size $dt = 0.1$.

The key features of this model:

- **Nonergodicity:** The state space $\mathbb{R}^m$ is high-dimensional; any single trajectory visits a negligible fraction of it.

- **Anchoring:** The genotype g enters at every timestep, constraining which regions of state space are accessible.

- **Trajectory-dependence:** The environmental history $\{e_0, e_1, \ldots, e_T\}$ determines which specific trajectory is followed within the anchor's constraints.

- **Attractor dynamics:** The recurrent structure creates attractor basins; different trajectories can converge to different attractors.

3.2 Phenotype as Trapped State

The phenotype $x \in \mathbb{R}^n$ is a readout of the final developmental state:

$$x = \sigma(W_{out} h_T) \tag{4}$$

where $\sigma = \tanh$ ensures phenotypic values are bounded to $[-1, 1]$.

This final state h_T is a *trapped state*—the endpoint of a nonergodic trajectory. The same genotype g can produce different h_T (and thus different x) depending on which trajectory was followed.

We extract a "developmental coherence" variable $c \in [0, 1]$ from the phenotype, representing how well-coordinated the developmental trajectory was—how consistently all cells followed the same attractor. Cancer, in this framing, is attractor bifurcation: cells that diverge from the organismal trajectory into a different basin. The emergent cancer mortality is:

$$\mu_S(x) = \mu_0(1 - \alpha \cdot c(x)) \tag{5}$$

where μ_0 is baseline mortality and $\alpha \in (0, 1)$ is the efficacy of coherence in preventing bifurcation.

3.3 Environmental Regimes

We define two environmental regimes that guide trajectories into different attractor basins:

- **Cooperative:** Low variance, high social support, low conflict $\rightarrow$ slower, more coordinated development $\rightarrow$ high coherence.

- **Competitive:** High variance, low support, high conflict $\rightarrow$ faster, less coordinated development $\rightarrow$ low coherence.

Empirical grounding: This mapping from environmental stability to developmental coordination has empirical support. In mice, maternal stress reduces blastocyst cell counts

and developmental coordination [36], while genetically identical mice under stress show substantially increased developmental variability—trajectory divergence from the same anchor [37]. Glucocorticoid signaling induces cell cycle arrest via cyclin/CDK repression [38]; GR-null embryos show 1.8-fold increased proliferation rates [39]. Environmental volatility thus directly affects the coordination of cell division.

Parameter values: We use $L = 5$ (anchor dimension), $m = 20$ (developmental state dimension), $n = 10$ (phenotype dimension), and $p = 3$ (environmental input dimension). This gives $\Delta_D = m - (L + 1) = 14$ when measuring a single trait (μ_S). Cancer parameters: $\mu_0 = 0.1$, $\alpha = 0.8$. See code for implementation details.

Note on model scope: The developmental network is intentionally minimal and not fitted to any species. Its role is illustrative: to instantiate the dimensional and identifiability arguments with a concrete dynamical system, not to make quantitative predictions about specific organisms.

3.4 Evolutionary Extension: Coherence as a Self-Stabilizing Attractor

We hypothesize that coherence is not merely a phenotypic outcome—it may be self-reinforcing. If high-coherence organisms create stable group environments, and stable environments enable coordinated development (as the stress literature suggests; Section 3.2), then coordinated development would produce high-coherence offspring. This feedback loop would function as an attractor at the social scale, mirroring the attractor dynamics at the cellular scale. To test whether such a feedback loop, *if it exists*, would be evolutionarily stable, we extended the model to a multilevel simulation. We simulate $N = 30$ groups of organisms evolving over 60 generations:

- **Group selection:** Groups compete based on their aggregate coherence. More cooperative groups create more stable developmental environments for their members.

Groups reproduce via multinomial sampling weighted by group fitness.

- **Individual selection:** Within groups, organisms compete based on a fitness function that includes synergistic benefits from cooperation: $w_i = 1 + \beta \cdot C_g \cdot c_i - k \cdot \mu_i$, where C_g is the group's mean coherence, c_i is the individual's coherence, and we use $\beta = 3.0$, $k = 8.0$ in simulations.

- **Price equation decomposition:** Following the standard multilevel Price decomposition [33, 34, 35], the change in mean coherence $\bar{c}$ can be written as:

$$\Delta\bar{c} = \underbrace{\frac{1}{\bar{W}}\mathrm{Cov}_g(W_g, \bar{c}_g)}_{S_{\text{between}}} + \underbrace{\frac{1}{\bar{W}}\mathbb{E}_g\left[\mathrm{Cov}_i(w_{ig}, c_{ig} \mid g)\right]}_{S_{\text{within}}} \tag{6}$$

We compute S_{between} and S_{within} directly from the realized fitness weights each generation.

The key feature is the *leaky integrator* for group climate: each generation, the group's coherence state is updated as $C_g \leftarrow 0.8 \cdot C_g + 0.2 \cdot \bar{c}$, where $\bar{c}$ is the current mean coherence. This creates a social attractor: groups with high coherence maintain stable environments that enable coordinated development, which produces high-coherence offspring, reinforcing the attractor.

This evolutionary extension is intentionally stylized: its role is to demonstrate that trajectory-based coherence *can* evolve under multilevel selection, not to quantitatively model any particular lineage.

3.5 Contrasting Interpretations

Figure 3 illustrates how the same outcome admits two causal interpretations:

- **Allele model:** $G \rightarrow \mu_S \rightarrow$ Cancer. The genotype algorithmically determines mortality; environment only modulates selection pressure over evolutionary time.

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- **Trajectory model:** $G \to \mathrm{Dev} \to \pi(E) \to r_t \to \mu_S$. The genotype anchors a developmental system; environment shapes the trajectory; the trapped state determines mortality.

Both produce the same aggregate correlation (Environment $\leftrightarrow \mu_S$) when projected to observables—this is the non-identifiability created by $\Delta_D > 0$.

4 Non-Identifiability Result

Proposition 1 (Non-identifiability of allele vs. policy mechanisms). *Let Model A be any allele-based system where late-life mortality takes values $\{\mu, \mu + \delta\}$ across two ecological regimes, with $0 \leq \mu < \mu + \delta \leq \mu_0$. Let Model B be any trajectory-based developmental system **capable of realizing any coherence level $c \in [0, 1]$ via some environmental history**, and where $\mu_S = \mu_0(1 - \alpha c)$ as in Equation 5. Then for any pair $(\mu, \mu+\delta)$ satisfying these constraints, there exists an environment pair (E_{coop}, E_{comp}) such that the induced μ_S distributions in Model B match those in Model A for all species-level summary statistics.*

Proof. Take any $(\mu, \mu + \delta)$ from Model A. We construct environments that induce matching μ_S distributions in Model B.

Choose c_{coop} and c_{comp} such that:

$$\mu_0(1 - \alpha c_{\mathrm{coop}}) = \mu \tag{7}$$

$$\mu_0(1 - \alpha c_{\mathrm{comp}}) = \mu + \delta \tag{8}$$

Solving: $c_{\mathrm{coop}} = (1 - \mu/\mu_0)/\alpha$ and $c_{\mathrm{comp}} = (1 - (\mu + \delta)/\mu_0)/\alpha$.

For sufficiently expressive dynamics (e.g., the recurrent network of Equation 3), there exists a mapping from environmental histories to any desired c; this is empirically illustrated in Figure 1D. Thus there exist environments E_{coop} and E_{comp} that induce these coherence levels.

Therefore, any species-level summary (mean μ_S, variance, etc.) that Model A produces can be exactly matched by Model B with appropriate environment choice. The models are observationally equivalent at this level. $\qquad\square$

Remark 1. This result formalizes the sense in which allele models are "incomplete": they are not wrong, but they are *non-identifiable* from aggregate data. The models become distinguishable only through interventions that change environment while holding genotype fixed.

5   Results

All figures show simulated outputs from the developmental network model; no empirical data are used. The simulations demonstrate the core theoretical claims: trajectory-dependence, attractor dynamics, and non-identifiability.

5.1   Trajectory Divergence: Same Anchor, Different Attractors

Figure 1 demonstrates the fundamental prediction of nonergodic development: identical genotypes (anchors) produce different phenotypes when developmental trajectories follow different paths through state space.

Panel (B) is the key visualization: the two trajectories diverge in state space despite sharing the same anchor constraints. The endpoints represent different attractor basins—trapped states that persist because the system cannot spontaneously explore alternatives. Panel (D) shows the phenotypic consequence: different developmental coherence and thus different cancer mortality from identical genotypes.

5.2   Population-Level Non-Identifiability

Figure 2 shows that aggregate population patterns cannot distinguish between allele-based and trajectory-based mechanisms.
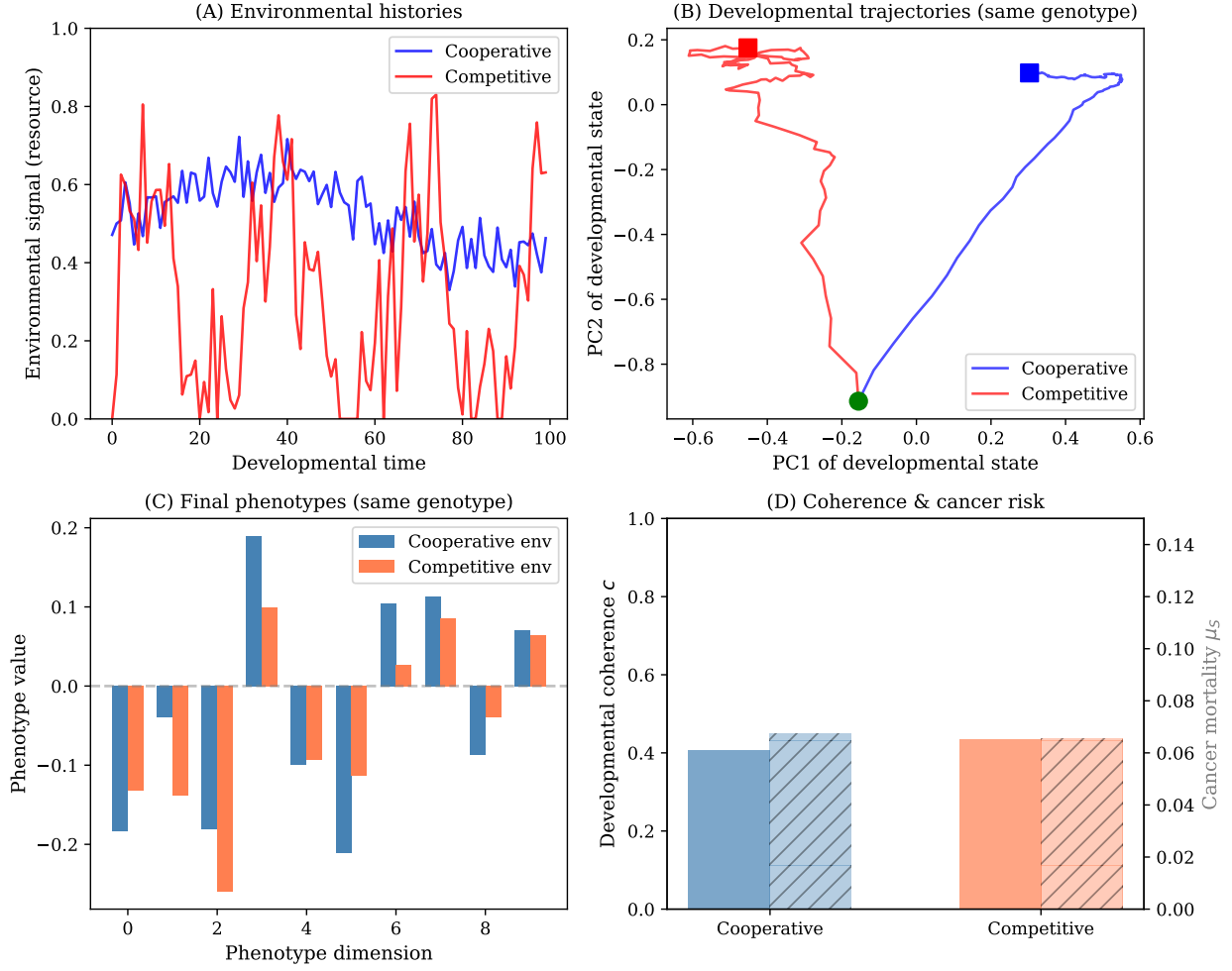
Figure 1: **Trajectory divergence under identical anchoring.** (A) Two environmental histories: cooperative (blue) and competitive (red). (B) Developmental trajectories through state space (PCA projection); same starting point (green circle), different endpoints (squares). (C) Different trapped states produce different phenotypes. (D) Different developmental coherence and cancer mortality emerge from the same genotype—cooperative environments enable more coordinated development with fewer bifurcations.
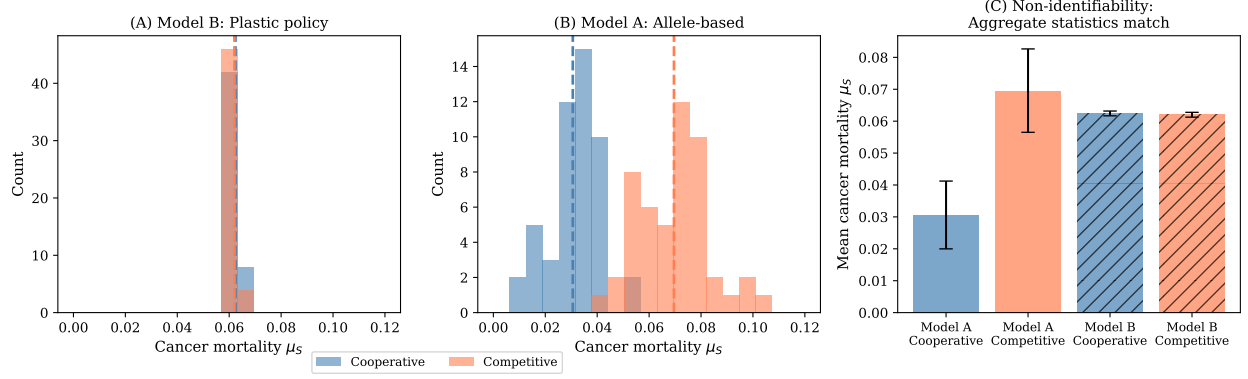
Figure 2: Non-identifiability at the population level. (A) Trajectory model: same genetic distribution in different environments → different μ_S distributions. (B) Allele model: different genetic distributions can produce matching patterns. (C) Aggregate statistics are indistinguishable—this is the Δ_D projection problem.

5.3 Causal Structure

Figure 3 contrasts the causal graphs. In the allele model, the genotype algorithmically determines the phenotype; environment acts only through evolutionary selection. In the trajectory model, the genotype anchors a developmental system that integrates environmental inputs in real time.
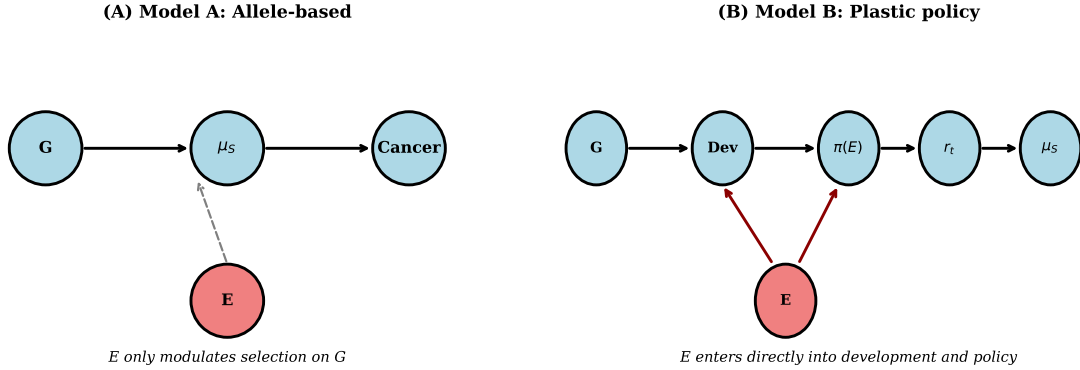

Figure 3: Contrasting causal structures. (A) Allele model: $G \to \mu_S$, environment external. (B) Trajectory model: G anchors development, E shapes trajectory, μ_S emerges from trapped state.

5.4 The Dimensional Gap in Action

Figure 4 visualizes the information loss that occurs when projecting from the high-dimensional (genotype × environment × trajectory) space to the low-dimensional (genotype → phenotype) summary.
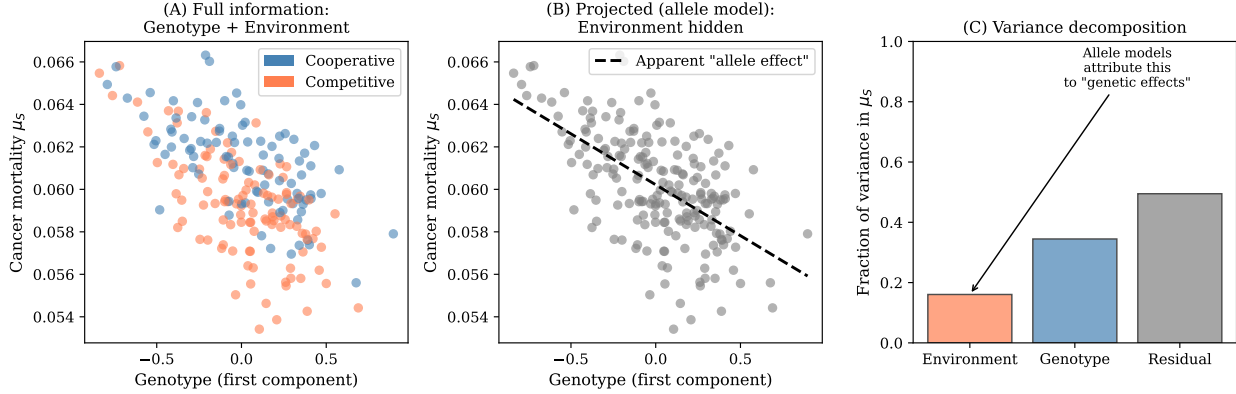

Figure 4: Projection destroys trajectory information. (A) Full space: environments clearly separate. (B) Projected space: trajectory information lost. (C) Variance decomposition: environment explains most μ_S variance, but allele models misattribute this to "genetic effects"—the dimensional gap in action.

5.5 Twin Worlds: The Decisive Experiment

Figure 5 provides the clearest demonstration of nonergodic development. We create two "worlds" with *identical* genotype distributions but different environmental regimes. The developmental trajectories in each world converge to different attractor basins, producing dramatically different phenotype distributions.

The genetic distance between worlds is $F_{ST} \approx 0$ (by construction), yet phenotypic distance is $P_{ST} \gg 0$. A GWAS would find no significant variants and conclude "missing heritability"—but the heritability is not missing, it is *trajectory-based*. The anchor constrains which attractors exist; the environment determines which attractor is entered.
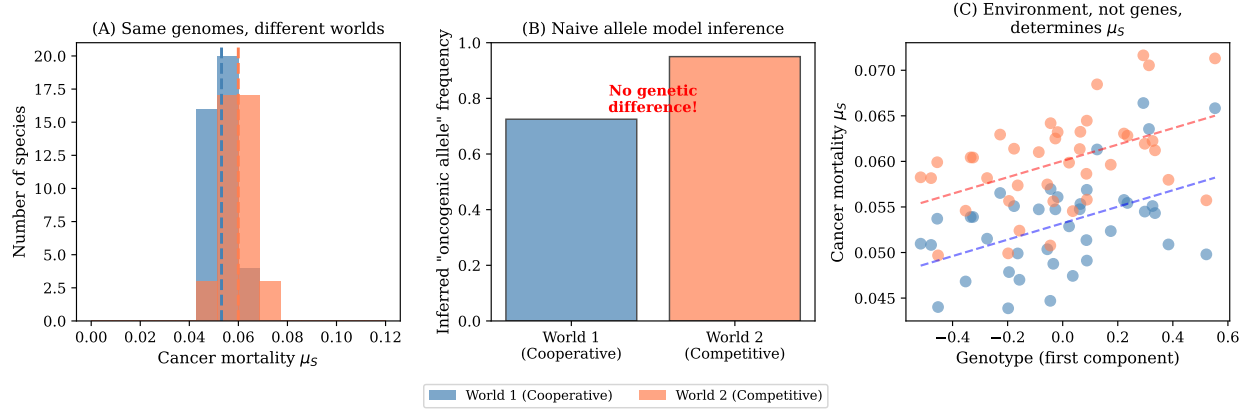
Figure 5: Twin worlds experiment. (A) Identical genomes, different worlds → different μ_S distributions. (B) A naive allele analysis would infer "oncogenic allele" frequency differences where none exist. (C) Genotype (anchor) shows minimal correlation with μ_S; trajectory determines outcome.

5.6   Divergent Predictions: The Intervention Test

Figure 6 shows where the models diverge. The allele model predicts that phenotype is locked by genotype—environmental change cannot alter the trapped state. The trajectory model predicts that environmental change can guide the system toward a new attractor.
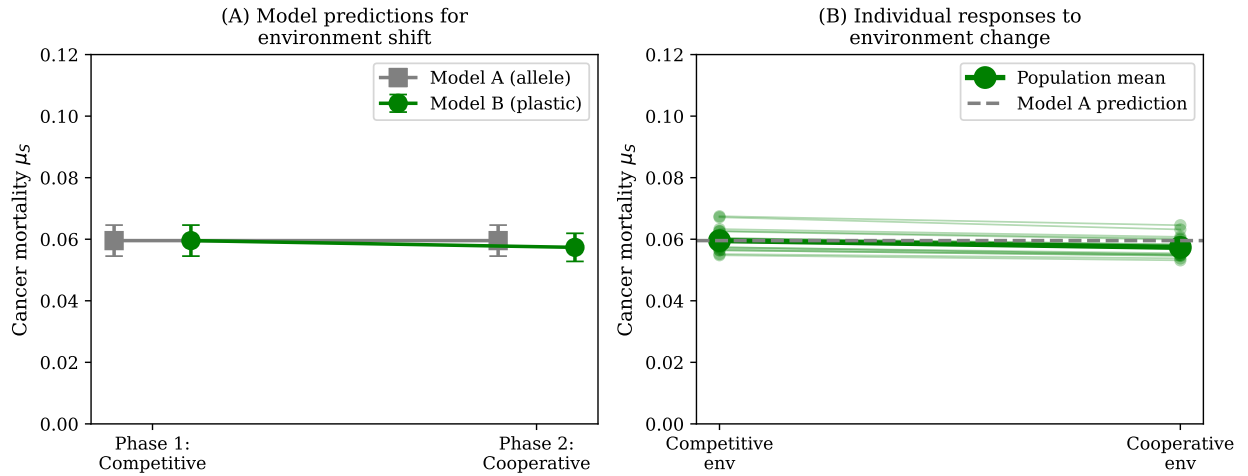

Figure 6: The intervention test. (A) Allele model: no change when environment shifts (genotype determines phenotype). Trajectory model: substantial shift (new environment → new attractor). (B) Individual trajectories converge toward the new attractor—phenotypic change without genetic change.

This is the critical empirical test. If moving organisms from competitive to cooperative

environments produces phenotypic shifts toward higher developmental coherence (fewer bifurcations), the trajectory model is supported. If phenotypes remain stable, the allele model is supported.

We acknowledge a confound: organisms placed in mismatched environments may show phenotypic changes due to stress rather than trajectory guidance *per se*. Teasing apart "cooperative cues guide development" from "environmental mismatch causes stress" is difficult. But this difficulty is itself the point: the system is high-dimensional, and clean causal decomposition may be impossible. What cooperative environments provide is *stability*—slower, more predictable conditions that allow cellular trajectories to remain coordinated. Whether we call this "reduced stress" or "trajectory guidance," the mechanism is the same: stability enables coherence; instability enables bifurcation. The phenotype emerges from trajectory dynamics that resist simple causal attribution.

Crucially, empirical analogs of this intervention already exist. Cross-fostering studies in rodents demonstrate that pups born to low-care (high-licking/grooming) mothers but raised by high-care foster mothers develop the low-stress phenotype, complete with altered methylation of the glucocorticoid receptor gene [45]. The phenotype tracks the developmental trajectory, not the genetic anchor. Our model provides the dynamical explanation: the high-care environment buffers the trajectory, allowing it to settle into the high-coherence attractor despite the "low-coherence" genetic background. This is Figure 6 realized in actual rodents.

5.7   Nonergodicity at the Social Scale: Fractal Cooperation

The preceding results demonstrate nonergodicity at the cellular-to-organismal scale: developmental trajectories get trapped in attractor basins, and coherence determines which basin. But coherence is not just a trait—it is *self-stabilizing*. For trapped states to persist over evolutionary time, they must construct the environmental conditions that ensure their own re-creation. This is niche construction at the dynamical systems level.

The result is a scale-free feedback loop: high cellular coherence (cancer suppression)

$\rightarrow$ viable organisms $\rightarrow$ cooperative groups $\rightarrow$ stabilized environmental volatility $\rightarrow$ high-coherence development in the next generation. This is the "fractal" structure: **organisms that maintain high coherence maintain high coherence**. The property that prevents cellular bifurcation (cancer) is the same property that stabilizes group dynamics (cooperation). Nonergodicity doesn't just trap individual trajectories—it creates nested attractors at every scale where the same coherence parameter operates.

To demonstrate this explicitly, we simulated the co-evolution of social cooperation and cancer suppression over 60 generations. Figure 7 shows the result.
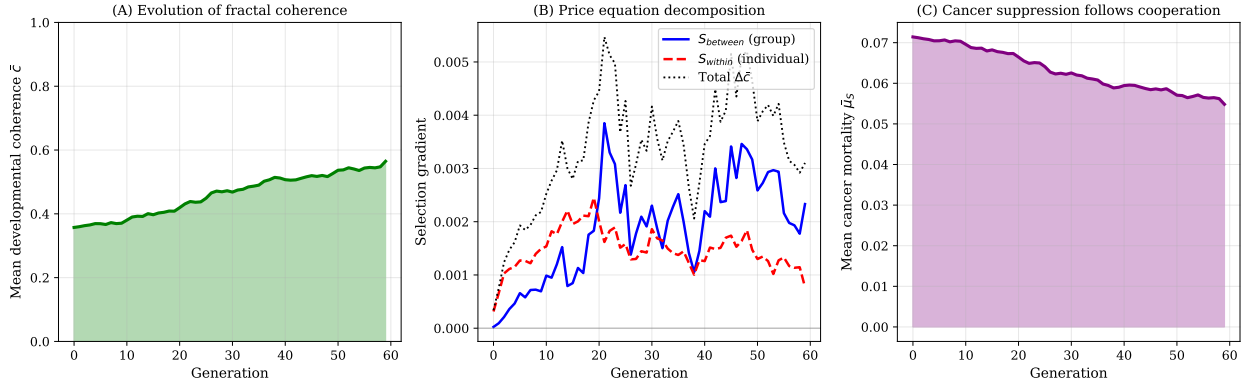

Figure 7: **Fractal Cooperation and the Price Equation.** (A) Over 60 generations, the population converges on the high-coherence attractor (coherence evolves from ~ 0.35 to ~ 0.55). (B) Price Equation decomposition reveals the mechanism: both between-group selection ($S_{\text{between}} > 0$) and within-group selection ($S_{\text{within}} > 0$) favor coherence due to the synergistic fitness structure. (C) Cancer mortality decreases as coherence increases—the same developmental parameter solves problems at both cellular and social scales.

The simulation confirms that selection for group-level stability effectively pulls the developmental system toward high-coherence attractors. Notably, both S_{between} and S_{within} are positive (Figure 7B). This is not a generic result—in many cooperation scenarios, within-group selection favors defection ($S_{\text{within}} < 0$). Here, both terms favor coherence because individual fitness includes the synergy term $\beta \cdot C_g \cdot c_i$: high coherence pays off *more* in cooperative groups. This constructive example shows that when the developmental architecture creates individual-level benefits from coherence in cooperative contexts, selection at all levels can align.

The "fractal" nature of the architecture means that solving the social dilemma (stabilizing the group) automatically solves the cellular dilemma (suppressing cancer). This is not coincidence but consequence: the same dynamical structure—high-dimensional development with low-dimensional anchoring—operates at both scales.

Figure 8 shows how different selection regimes affect the outcome. Strong group selection produces the lowest cancer mortality; weaker group selection produces intermediate results.
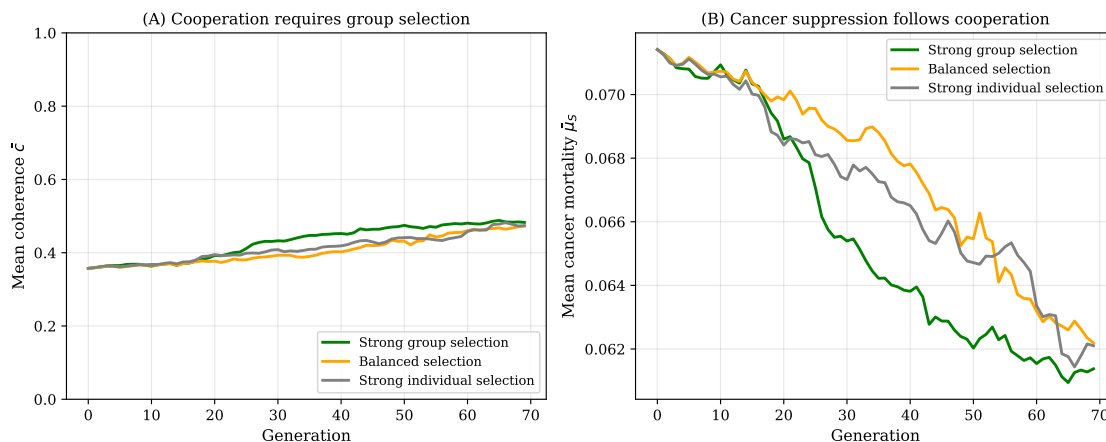

Figure 8: **Selection Regime Comparison.** (A) Cooperation evolves under all regimes, but strong group selection (green) maintains the highest coherence. (B) Cancer mortality is lowest under strong group selection—confirming that group-level stability recruits the developmental coherence that suppresses cellular bifurcation. Parameters: $N = 25$ groups, 70 generations.

This provides a concrete instance of the multilevel selection perspective that Sierra et al. themselves advocate: the same high-coherence developmental architecture that stabilizes groups also suppresses within-organism cheater dynamics (cancer). Cooperative species have lower cancer not (only) because selection modified their genomes, but because cooperative environments *are* the selection pressure that maintains high-coherence developmental attractors. The allele story and the trajectory story are two views of the same underlying process—but the trajectory story is more parsimonious, requiring no assumption that cancer mortality was ever positively selected.

6 Discussion

6.1 Nonergodicity as the Core Insight

The central contribution of this paper is recognizing that developmental systems are *nonergodic*. The state space is too vast to explore; trajectories are trapped; phenotypes reflect which attractor was entered, not an algorithmic genotype-to-phenotype mapping.

Crucially, the property that creates trapping—coherence—is itself what gets trapped. High-coherence systems maintain high coherence; low-coherence systems maintain low coherence. This self-stabilization operates at every scale: cells within organisms, organisms within groups. What we call "cooperation" at the social level and "developmental coordination" at the cellular level are the same dynamical phenomenon: attractor stability in a nonergodic system. The "fractal" in fractal cooperation is not metaphor but mechanism.

The defection framing makes this explicit [17]: cancer is cellular defection from organismal cooperation; competition is organismal defection from group cooperation. Both are bifurcations away from coordinated attractors. The claim is not that cooperative organisms happen to also suppress cancer—it is that **not defecting and not defecting are the same thing at different scales**. An organism that maintains cellular coherence is, by the same developmental dynamics, an organism that maintains social coherence. This is why "fractal coherence" evolves as a package: selection cannot pull apart what the dynamical architecture has unified.

This reframes several longstanding puzzles:

- **Missing heritability:** May include missing trajectory information, not only missing variants. GWAS project out the environmental history that determines which attractor each individual occupies.

- **Epigenetic memory:** Not a separate mechanism, but trapped states in nonergodic dynamics. The genome anchors; the trajectory traps.

- **Genetic assimilation:** How plastic responses become canalized. If an attractor basin is consistently entered across generations, selection can modify the anchor to make that basin deeper or more accessible—converting trajectory-dependence into anchor-dependence.

- **Developmental Origins of Health and Disease (DOHaD):** Our framework formalizes the DOHaD hypothesis [46], which posits that early environmental exposures establish long-term phenotypic trajectories. In our terms, early perturbations push the system into specific attractor basins (e.g., metabolic thrift, high inflammation) that persist throughout life. The "missing heritability" of these traits is the missing record of initial trajectory divergence.

6.2   Steelmanning the Allele View

Allele-based models are not wrong. Sierra et al.'s finding that cooperative species have lower cancer prevalence is real and important [1]. Over evolutionary time, selection does modify anchors.

Our point is that the aggregate pattern "cooperative = less cancer" is equally consistent with:

1. **Anchor modification:** Selection has reshaped the attractor landscape to favor high-coherence developmental modes.

2. **Trajectory guidance:** Cooperative environments enable more coordinated development, reducing bifurcation probability.

3. **Genetic assimilation:** An initially trajectory-dependent response becomes anchor-encoded through sustained selection.

These are non-identifiable from comparative data. Distinguishing them requires intervention experiments. However, our evolutionary simulation (Figures 7–8; see Section 3.4 for

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model details) demonstrates that the trajectory-based mechanism is evolutionarily robust: selection for group stability naturally recruits developmental coherence, creating the very correlation between cooperation and cancer suppression that allele models seek to explain. Our result should therefore be read as a *completion* of Sierra et al.'s model rather than a refutation: allele-level and trajectory-level accounts are two projections of a single underlying high-dimensional developmental system.

The key difference lies in causal framing. Sierra et al. propose that high cancer rates in competitive species are an *adaptation*—a "phenoptotic" program to accelerate generational turnover (the Hydra effect), where early death of adults frees resources for offspring, yielding net fitness gains under competition. Our framework offers a more parsimonious explanation: high cancer rates are simply the *entropic consequence* of developing in a high-volatility environment without social buffering. Both frameworks predict the same comparative pattern. But the Hydra effect requires cancer to have been positively selected as a population-level strategy; our account requires only that cooperative environments enable slower, more coordinated development—cancer suppression emerges as a byproduct, not an adaptation. The distinction matters for intervention: if Sierra et al. are right, competitive species have been "designed" for high cancer; if we are right, reducing environmental stress should reduce cancer even in lineages with no evolutionary history of cooperation.

6.3   When Nonergodicity Dominates

Taxonomy: When Trajectory Matters

Anchor-dominated (allele stories work):

- Low-dimensional development (Δ_D small)

- Few attractor basins

- Stable environments across generations

- Strong canalization

Trajectory-dominated (nonergodicity matters):

- High-dimensional development ($\Delta_D \gg 0$)

- Many attractor basins accessible from same anchor

- Variable environments across generations

- Weak canalization, high plasticity

Cancer risk, facial morphology, and human life-history traits sit squarely in the trajectory-dominated regime.

6.4   Specific Empirical Predictions

While the models often produce indistinguishable aggregate statistics, they diverge structurally. Table 1 contrasts the mechanistic assumptions and specific predictions of each framework.

6.5   The Projection Problem

Genotype-phenotype inference involves a cascade of projections:

Table 1: **Anchor-based vs. Trajectory-based Models.** Indistinguishable from static data; diverge under intervention.

Feature	Anchor Model (Allele-Based)	Trajectory Model (Nonergodic)
Core Mechanism	Genotype algorithmically determines phenotype.	Genotype anchors high-D system; trajectory determines which attractor.
Role of Environment	*Selection pressure:* Modulates which anchors are favored over generations.	*Trajectory guidance:* Shapes which attractor basin is entered in real-time.
"Cooperative Safety"	Selection modified anchors to favor low-cancer attractors.	Cooperative cues guide trajectories into existing low-cancer attractors.
Divergent Predictions (Testable)		
Environmental Shift	**No change.** Trapped state is anchor-determined.	**Shift occurs.** New trajectory $\rightarrow$ new attractor.
Twin Worlds	Identical anchors $\rightarrow$ identical phenotypes.	Identical anchors $\rightarrow$ divergent phenotypes ($F_{ST} \approx 0$, $P_{ST} \gg 0$).
GWAS	"Missing heritability" = many small-effect variants.	"Missing heritability" = trajectory information projected out.

$$(\text{anchor, trajectory, trapped state}) \quad \xrightarrow{\text{projection}} \quad (\text{genotype, phenotype})$$

$$(g, E_{0:T}, h_T) \in \mathbb{R}^{L+pT+m} \qquad\qquad (g, x) \in \mathbb{R}^{L+n}$$

Allele-based models further marginalize over trajectories:

$$P(x \mid g) = \int P(x \mid g, E) P(E) \, dE \tag{9}$$

This marginalization discards the trajectory information that determines which attractor basin was entered [6, 7]. The "genetic effect" β is not a property of the anchor alone but of the anchor-trajectory interaction averaged over some implicit distribution of environmental histories.

In our minimal model ($L = 5$, $m = 20$, $n = 10$), $\Delta_D = 14$. In real organisms with $m \sim 10^4$ regulatory degrees of freedom, the dimensional gap is enormous—making trajectory-dependence correspondingly important.

6.6 Beyond Nature versus Nurture

One might object that we are simply rebranding the "nurture" side of the nature-nurture debate. We are not. The traditional framing assumes that genetic and environmental contributions are separable and additive: $P = G + E + G \times E$. Variance can be partitioned; heritability can be estimated; the question is merely "how much of each?"

Our framework rejects this separability. In a nonergodic high-dimensional system, the phenotype is not a sum of genetic and environmental contributions—it is the attractor basin that a trajectory happened to enter. The trajectory is shaped by both anchor and environment simultaneously; there is no meaningful sense in which their contributions can be decomposed. Asking "how much is genes versus environment?" is like asking how much of a river's path is due to gravity versus the shape of the landscape. The question is malformed.

This is why Δ_D matters: it quantifies when the additive decomposition breaks down. When Δ_D is small, the anchor dominates and variance partitioning works. When Δ_D is large, the trajectory carries information that neither anchor nor simple environmental measures capture. The "missing heritability" is not missing nature or missing nurture—it is the signature of a high-dimensional system whose behavior cannot be projected onto low-dimensional summaries without information loss.

6.7 Connections to Related Work

Our framework provides formal foundations for several recent proposals:

Cancer memory [20]: Lissek argued that malignancy can progress through epigenetic mechanisms once initiated. In our terms: oncogenic changes perturb the developmental trajectory, pushing the system into a new attractor basin. The nonergodic dynamics *are* the memory—the system cannot spontaneously return to its previous basin.

Cancer attractors [13]: Huang proposed that cancer represents regression to an atavistic attractor state—a reversion to unicellular-like dynamics encoded in the genome's deep history. We extend this by explicitly linking the *stability* of that attractor to the *volatility* of

the external environment via the Dimensional Gap. While Huang focuses on the topography of the attractor landscape, we focus on the trajectory's capacity to navigate it: cooperative buffering flattens the effective landscape, making the healthy attractor deeper and the atavistic basin harder to reach.

Bioelectric goal states [21]: Levin has proposed that cells maintain "goal states"—anatomical target morphologies encoded in bioelectric patterns. When cells "forget" their goal, cancer results. Our framework provides the dynamical systems formalization: the "goal" is the organismal attractor basin; "forgetting" is bifurcation to a divergent attractor. This reframes teleological language in mechanistic terms while preserving the key insight that cancer is fundamentally a failure of coordination, not merely accumulated mutations.

Developmental plasticity [22, 23]: High-dimensional developmental systems with large Δ_D naturally exhibit plasticity—many attractor basins are accessible from a single anchor.

Systems evolution [24]: Evolution is primarily about functional organization, not individual genes. In our terms: selection modifies anchors, but anchors constrain organizational possibilities without fully determining them.

Development-ageing-cancer [25]: Unified developmental models linking these processes are natural in a nonergodic framework, where trajectory history shapes multiple downstream outcomes.

Process structuralism [26]: Goodwin argued that biological forms are generic properties of developmental dynamics—intrinsic to the physics of excitable, structured media—rather than genetic programs executed by molecular machinery. The genome selects which forms are realized; it does not specify them algorithmically. Our Δ_D metric quantifies the gap between genetic parameters and the structural possibilities of the developmental medium. The "book of life" is not the blueprint; it is the index to a library of dynamically accessible forms.

In the tradition of critiques of algorithmic biology [27], we demonstrate that the gene-as-algorithm assumption is not merely an approximation but a projection that actively discards

the trajectory structure of nonergodic development.

7 Conclusion

Biological development is nonergodic. The state space is too vast to explore; trajectories are trapped; phenotypes are attractor states, not algorithmic outputs.

The genome is a low-dimensional anchor that constrains which attractor basins exist, but environmental history determines which basin is entered. This anchor-trajectory duality offers one lens on the genotype-phenotype relationship: the mapping is many-to-one not only because of noise or missing variants, but also because trajectory information that distinguishes outcomes is projected out by genotype-phenotype analysis.

Sierra et al.'s finding that cooperative species have lower cancer prevalence is real. Our contribution is to show that it admits both anchor-based (selection modified the genotype) and trajectory-based (environment guided development) interpretations—non-identifiable from comparative data, distinguishable only through intervention.

Some of the "missing heritability" in GWAS may be missing trajectories, not missing variants. The dimensional gap Δ_D quantifies how much information is lost when we project from the high-dimensional (anchor, trajectory, attractor) space to the low-dimensional (genotype, phenotype) summary.

Genotype does not algorithmically determine phenotype. Genotype anchors a nonergodic developmental system; environmental history traps it in an attractor; the phenotype is the trapped state. Recognizing this is essential for prediction, intervention, and understanding biological change.

Data and Code Availability

Simulation code is available at `https://github.com/todd866/nonergodic-development`. The repository includes `nonergodic_development.py`, which implements the developmental

network and generates Figures 1–6, and `multilevel_simulation.py`, which implements the evolutionary extension with Price equation decomposition (Figures 7–8).

Declaration of competing interest

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During the preparation of this work the author used Claude Code (Claude 4.5 Opus) for primary drafting and model development, with feedback from Gemini 3 Pro and GPT 5.1 Pro. After using these tools, the author reviewed and edited the content as needed and takes full responsibility for the content of the published article.

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