Qeios

Peer Review

Review of: "Aging as Cybernetic Attractor Decay: Beyond the Stochastic-Programmed Dichotomy"

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1. Independent researcher

Summary of the Preprint

The author proposes that aging is best understood not as random molecular damage or programmed genetic decline, but as a **decay of cybernetic attractors in regulatory networks**. The model asserts that deterioration of internal system precision leads to characteristic age-related trajectories ("drift"), which can be interpreted as transitions between quasi-stable equilibrium states ("site-specific equilibrium states").

Major Criticisms

1. Lack of Clear Operational Definitions

The concept of *"site-specific equilibrium states"* is central to the author's argument but is not defined in a manner that allows empirical testing or falsification. The manuscript fails to specify: (1) how these states are identified quantitatively, (2) what biological measurements correspond to them, (3) whether they are stable attractors measured in data, (4) or how they differ from stochastic fluctuations within complex systems.

Without formal mathematical definition and operational measurement criteria, this concept remains speculative and non-falsifiable.

Supporting Literature (systems frameworks) Reviews on complex systems approaches to aging emphasize the need for formal, testable models of network dynamics in aging biology. Theoretical

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frameworks integrating emergence, networks, and resilience illustrate how measurable states might be defined but require formal specification. Nat. Aging. 2022 Jul;2(7):580-591. doi: 10.1038/s43587-022-00252-6. Epub 2022 Jul 20.

2. No Strong Empirical Validation or Testable Predictions

The manuscript suggests that the proposed model explains aging better than stochastic or programmed theories, yet it offers no specific **predictions that can be tested experimentally**. A viable theoretical model should state *what observable phenomena would confirm or contradict it*, such as: a) measurable transitions between network states over the lifespan, b) predictions about variance patterns across individuals, c) or identifiable attractor basins in high-dimensional data.

In contrast, existing approaches, such as epigenetic clocks, produce measurable age predictions that correlate with mortality and health outcomes.

3. Fails to Explain Consistent Aging Trajectories

One of the strongest objections is that the model explains *drift* in regulatory precision but not why aging trajectories are **highly reproducible across individuals** (apart from pathological variations). Biological systems show consistent age-related changes in genes, signaling pathways, and physiological measures, and these trajectories are reproducible across populations. Models that treat aging as attractor decay must demonstrate why the attractor landscape is structured in a way that leads to **stereotyped, convergent trajectories** — not arbitrary drift. Empirical measurements of epigenetic clocks and multi-omics aging patterns show reproducible age-related change patterns across individuals.

4. Overlooking Established Mechanistic Theories

The manuscript frames aging as a standalone cybernetic phenomenon but largely ignores or underemphasizes the body of evidence in established aging frameworks:

1. **Evolutionary theories** such as mutation accumulation and antagonistic pleiotropy explain why aging trajectories are conserved — they arise from selective pressures.
2. **Systems biology of aging** illustrates aging as multi-scale network dysregulation that can be measured and modeled.

A model that positions itself as a replacement for existing frameworks should engage directly with these well-validated theories rather than treating them as peripheral.

5. Conceptual Rather Than Mechanistic

By attributing aging to "mathematical drift," the author risks reducing the biological problem to metaphors of control theory without sufficiently explaining **mechanistic causal drivers** (e.g., DNA damage, epigenetic dysregulation, cellular senescence, inflammation). Current literature demonstrates that aging reflects a coordinated breakdown of multiple pathways, not just a loss of system precision, as dysregulation of networks is widely recognized as a hallmark of aging, and epigenomic changes and chromatin state shifts are measurable contributors to aging processes. A robust theory should interface its conceptual attractor framework with **molecular and cellular drivers** measured empirically.

Recommendations for Improvement

To strengthen the manuscript and make it publishable in a peer-review context, the author should:

1. **Formally define key concepts**, especially equilibrium states and attractor structures, in mathematical and biological terms.

2. **Provide explicit, falsifiable predictions** that can be tested with empirical data, such as epigenetic age curves, transcriptomic trajectories, or network instability signatures.

3. **Demonstrate consistency with existing aging biomarkers**, e.g., show that attractor decay corresponds with epigenetic clock acceleration or specific network dysregulation metrics.

4. **Engage directly with evolutionary, physiological, and systems-level theories**, situating the cybernetic perspective within a broader literature context rather than excluding alternative explanations.

Conclusion

Bhak's *cybernetic attractor decay* idea is intriguing and conceptually resonates with complex systems thinking. However, in its current form, it is too **theoretical and under-defined** to serve as a convincing alternative to existing aging frameworks. It lacks **operational clarity**, **empirical anchoring**, **predictive testing**, and **engagement with foundational biological theories**. Incorporating the recommendations above and grounding the theory in well-characterized biological aging phenomena would make the model considerably stronger and more scientifically useful.

Declarations

Potential competing interests: No potential competing interests to declare.