Genome Attractors During Evolution: Structural Parallels with the Attractor Framework

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Abstract

The attractor framework proposes that persistence under perturbation is a key diagnostic criterion for identifying stable configurations in complex systems, with corrective permeability (κ)—a proposed measure of the rate at which a system returns to its basin after perturbation, operationally defined as $\kappa = 1/\tau$, where τ is the time required for the system to return to a specified baseline state following a specified perturbation protocol—serving as one of its central concepts. Kasperski and Kasperska (2021) published a study in *Scientific Reports* using artificial neural networks and semihomologous analysis to identify "genome attractors" in cytochrome b sequences across diverse organisms. Their analysis demonstrates that groups of organisms are trapped in distinct, stable attractors during evolution, separated by large evolutionary distances. They further propose a model of cancer development in which genome instability and reactive oxygen species (ROS) drive transitions between attractor basins, while cells may also evolve within a single basin through cell-fate changes. This paper identifies structural

parallels between the Kasperski and Kasperska model and the attractor framework. Both frameworks use attractors as a formal concept; the parallels are consistency checks, not independent corroboration.

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1. Introduction: Attractors in Evolutionary Biology

The attractor framework (Galida, 2026a, self-published May 2026 at fantasyattractor.com; no DOI) proposes that dissipative attractors—stable configurations toward which systems converge and from which they resist displacement—are proposed units of persistent organization across physical, biological, cognitive, and social domains. Corrective permeability (κ) is a proposed measure of a system's capacity to return to its basin after perturbation, operationally defined as $\kappa = 1/\tau$, where τ is the time required for the system to return to a specified baseline state following a specified perturbation protocol. This operational definition requires a defined baseline and perturbation specification before κ can be measured in any given domain; these prerequisites are not yet established for most applications of the framework.

In 2021, Andrzej Kasperski and Renata Kasperska of the University of Zielona Gora, Poland, published "Study on attractors during organism evolution" in *Scientific Reports*, a peer-reviewed journal in the Nature portfolio. Using a three-layer artificial neural network trained on cytochrome b sequences from 36 organisms spanning the full spectrum of evolution, they demonstrated that organisms are trapped in distinct "genome attractors"—stable configurations of the genome that resist perturbation and are separated from other attractors by large evolutionary gaps. They further proposed a unified model of cancer development in which destabilization

of the current attractor, driven by elevated reactive oxygen species (ROS) and genome chaos, leads to transitions into new attractor basins.

The study did not cite the attractor framework and was conducted within the established traditions of bioinformatics, evolutionary biology, and neural network pattern recognition. This paper identifies structural parallels between the Kasperski and Kasperska model and the attractor framework. Both frameworks use attractors as a formal explanatory concept; the parallels are consistency checks, not independent corroboration.

It should be noted that Kasperski and Kasperska's use of "attractor" derives from neural network classification: a genome attractor is a region of genome space in which the neural network places phylogenetically related organisms. Whether these classification regions constitute attractors in the formal dynamical systems sense—as the attractor framework uses the term—is an assumption that warrants further investigation. The parallels drawn in this paper are contingent on the validity of this assumption.

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2. The Kasperski and Kasperska Model

Kasperski and Kasperska (2021) define an attractor as "a configuration towards which the system evolves over time" and note that "after attaining an attractor a given configuration of a system is sufficiently stable to return to the original state after disappearing an eventual perturbation." They distinguish two classes of attractor dynamics:

2.1 Genome attractors (basins). Using an artificial neural network trained on cytochrome b amino-acid sequences, the authors identified that organisms during evolution are trapped in distinct genome attractors. For human evolution, they

identified six attractors separated by significant evolutionary distances: Tree shrew, Prosimian, New World Monkey, Old World Monkey, Other hominoid, and Old human attractors. Each attractor is a stable region of genome space in which organisms persist over evolutionary timescales. The orbits of these attractors are disturbed by small perturbations (represented as arrows pointing toward other organisms), but the system remains within the basin. The distances between attractor orbits, expressed as distance factors (e.g., the ratio of inner to outer orbit size), quantify the evolutionary gaps between basins. The derivation and units of these distance factors are as given in the original study.

2.2 Cancer as attractor destabilization. The authors propose a two-mode model of cancer development. **Vertical development** occurs within a single genome attractor: the cell changes its cell-fate attractor (gene expression program) without leaving the genome basin. This is an adaptation to environmental or internal perturbations that does not require genome re-organization. **Horizontal development** occurs when elevated ROS levels cause genome instability and genome chaos, leading to a change of genome attractor—a transition into a new basin with a re-organized genome. Horizontal development is always followed by vertical development, as the cell must establish a new cell-fate program to survive in the new genome basin. The authors note that cancer cells, driven by ROS, can undergo repeated horizontal transitions, creating an "impression that cancer cells want to escape from the internal ROS flame through permanent changes of genome attractors."

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3. Structural Parallels with the

Attractor Framework

The claims in this section are subject to the limitations discussed in Section 4, particularly regarding the qualitative nature of κ, the model-dependence of the neural network attractors, and the provisional status of the $\kappa = 1/\tau$ definition. The parallels identified are structural analogies, not formal derivations.

3.1 Genome Attractors as Basins. The genome attractors identified by Kasperski and Kasperska are stable configurations in genome space that resist perturbation and persist over evolutionary timescales. This is structurally analogous to the attractor framework's concept of a basin. The evolutionary distances between attractors correspond to the framework's distinction between distinct basins, and the small perturbations (arrows) that disturb but do not displace the attractor correspond to the framework's concept of perturbation within a basin.

3.2 Cancer as Basin Transition. Horizontal cancer development—the destabilization of the current genome attractor, genome chaos, and stabilization in a new genome attractor—is structurally analogous to the framework's concept of a phase transition between basins. The chaotic intermediate state (genome chaos) is the transition phase; the re-stabilization in a new attractor is the system finding a new basin. Vertical cancer development—cell-fate changes within a genome attractor without leaving the basin—corresponds to the framework's concept of perturbation absorption without basin transition. This distinction between within-basin adaptation and between-basin transition is a core feature of both models.

3.3 ROS as the Perturbation Mechanism. [Note: The claims in this section are subject to the limitations described in Section 4, particularly the lack of formal κ measurement and the neural network/attractor assumption.] In the Kasperski and

Kasperska model, elevated ROS acts as the destabilizing force that pushes the cell out of its current genome attractor. This maps onto the framework's concept of a perturbation that exceeds the system's corrective permeability, forcing a basin transition. The repeated horizontal transitions observed in cancer cells—successive escapes from one genome attractor to another under persistent ROS pressure—are structurally analogous to the framework's description of a system undergoing repeated basin transitions when corrective mechanisms are saturated by sustained perturbation.

3.4 Attractor Depth and Persistence. [Note: The claims in this section are subject to the limitations described in Section 4, particularly the qualitative nature of the distance-factor-to-basin-depth mapping.] The large evolutionary distances between genome attractors, quantified by distance factors, reflect the depth of the basins in the Kasperski and Kasperska model. A larger distance factor indicates a wider evolutionary gap between attractors, consistent with the framework's concept that deeper basins require more energy (or more sustained perturbation) to exit. However, the mapping between distance factors and basin depth is intuitive rather than derived. Basin depth in formal dynamical systems is a property of the energy landscape; distance factors from neural network classification are a related but distinct quantity. The parallel is offered as a qualitative structural analogy, not a formal equivalence.

3.5 The Atavistic Theory and the Permian Parallel. [Note: This section introduces a third domain (climate) to reinforce an analogy between two already-analogized domains. Accumulating analogies without formal constraints is a known risk for unfalsifiable frameworks; the present parallel is speculative and is retained here as an illustration of heuristic reach only.] The atavistic theory of cancer, which Kasperski and Kasperska reference, proposes that cancer cells revert to ancient, unicellular survival programs under extreme stress.

This is a real-world biological instance of a system reverting to a much older, simpler attractor when pushed beyond its current basin's capacity. The attractor framework has described a structurally analogous dynamic in other domains—specifically, the hypothesis that when the climate system is pushed too far from the Holocene basin, it may not merely shift to a neighboring attractor but can revert to a much older, lethal state, analogous to the Permian extinction's anoxic conditions. This cross-domain parallel is speculative and is offered as an illustration of the framework's heuristic reach, not as a confirmed prediction.

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4. Limitations

This mapping is post-hoc. The parallels identified here are structural analogies, not independent evidence for the framework. Kasperski and Kasperska developed their model within the established traditions of bioinformatics and evolutionary biology; they did not set out to test the attractor framework.

The framework's κ remains qualitatively defined. While the distance factors separating genome attractors provide a quantitative measure of basin depth in the Kasperski and Kasperska model, no formal mapping between these factors and κ has been derived. The provisional definition $\kappa = 1/\tau$ is not yet linked to any specific measure in the Kasperski and Kasperska data, and the prerequisites for measuring τ (a specified baseline state and a specified perturbation protocol) have not been established for the genomic or cellular domains discussed here.

The neural network approach used by Kasperski and Kasperska is one of several methods for analyzing evolutionary distances, and the specific attractor configurations identified depend on

the choice of training organisms, the neural network architecture, and the amino-acid coding scheme. The attractor interpretation of evolutionary data is therefore model-dependent. Furthermore, whether the stable classification regions identified by a neural network constitute attractors in the formal dynamical systems sense—the sense in which the attractor framework uses the term—is a substantive assumption. The parallels drawn in Section 3 are contingent on the validity of this assumption.

The attractor framework is self-published and has not undergone independent peer review. The foundational paper (Galida, 2026a) was published on fantasyattractor.com in May 2026 and is not archived with a DOI.

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5. Falsifiability Conditions

The following observations would weaken or invalidate the parallels drawn here:

- **Disconfirming observation 1:** If genome attractors were shown to be *artifacts of the neural network architecture* rather than genuine properties of genome space, the basin analogy would fail.
- **Disconfirming observation 2:** If the distance factors separating genome attractors were shown to be *continuous* rather than discontinuous, the basin-transition model would be weakened.
- **Disconfirming observation 3:** If alternative models of cancer progression (e.g., purely stochastic mutation accumulation without attractor dynamics) were shown to explain the data with equal or greater parsimony, the attractor interpretation would not be uniquely supported.

Affirmative prediction: If genome attractors function as basins in the attractor framework's sense, then experimental manipulations that increase ROS levels should increase the probability of attractor transitions (horizontal development) in a dose-dependent manner, while manipulations that reduce ROS should stabilize the current attractor and favor vertical development. This prediction is testable in cell culture models with controlled oxidative stress. It should be noted that measuring "attractor transition probability" in such an experiment requires specifying how the neural network's classification scheme maps onto the experimental observables—e.g., whether a transition is identified by a shift in the cytochrome b sequence profile as classified by the trained ANN, or by a proxy measure such as karyotype or gene expression signature.

Framework falsifiability: The attractor framework itself requires independent falsifiability conditions. Specifically: (a) if κ, as operationally defined, cannot be correlated with any independently validated measure of system resilience across multiple domains (physical, biological, or cognitive), the framework's central construct lacks empirical grounding; (b) if attractor-like dynamics in cancer progression are shown to be explained with equal or better parsimony by clonal evolution models (e.g., standard somatic mutation accumulation theory as reviewed in Greaves & Maley, 2012) when fitted to the same genomic data, the attractor framework's claim to offer a unified explanatory vocabulary would be weakened.

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6. Conclusion

The genome attractor model of Kasperski and Kasperska (2021) exhibits structural parallels with the attractor framework's description of basins, basin transitions, and perturbation-driven attractor shifts. Their distinction

between vertical and horizontal cancer development maps onto the framework's distinction between within-basin adaptation and between-basin transition. The ROS-driven mechanism of attractor destabilization is a molecular analogue of the framework's perturbation concept. These parallels are structural analogies, not independent validation. The framework remains a self-published, preliminary research program. This mapping is a contribution to its ongoing development.

References

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