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Commentary

# Cybernetic Attractor Decay: A Regulatory-Fidelity Framework for Aging

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**Biological aging follows predictable molecular trajectories across individuals and species. This predictability arises despite stochastic biological processes. The same trajectories can be reversed by developmental reprogramming. It is paradoxical that these properties coexist. Pure stochastic-damage models do not accommodate the predictability. Programmed-senescence models do not accommodate the reversibility.**

**To resolve this paradox, I propose a framework called cybernetic attractor decay (CAD). Under CAD, aging clocks measure the predictable drift of developmentally established regulatory architectures. At the core, they do not measure damage load or read out a death program.**

**The attractor is defined as the joint stable configuration of CpG methylation, chromatin state, transcription-factor occupancy, and feedback topology that maintains a cell-type-specific regulatory state. Gerostasis is the maintained, low-drift state of that attractor, and the regulatory condition that interventions aim to preserve or restore. Gerotype is the measurable aging phenotype of a cell, tissue, or regulatory network at a given biological age. The gerotype is the operational expression of attractor decay. It plays the role that catalogs of cellular features (such as "hallmarks") play in other accounts but is tied directly to regulatory state rather than to a list. Computational drift is operationalized as age-associated loss of regulatory precision, measurable as transcriptional, methylation, and chromatin-accessibility dispersion in single-cell data. The empirical face of the gerotype is the rise of Non-Requisite Variety at the expense of Requisite Variety.**

**The framework integrates several existing accounts: quasi-programmed senescence and hyperfunction[1]; the infrastructure-versus-specialized-gene partition[2]; loss of dynamical complexity[3]; and the complex-systems approach[4]. Each is treated as one axis of a broader landscape of regulatory-fidelity loss. CAD makes testable predictions that distinguish it from pure stochasticity accounts. These include tissue-specific signatures of drift, preserved attractor topology in negligibly senescent species, and coordinated system-wide attractor collapse in semelparous organisms such as Pacific salmon, which die within days to weeks of spawning.**

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# 1. Introduction: A Generative Paradox

Aging follows reproducible molecular trajectories across individuals and species. Yet the underlying biology is full of stochastic events. The same trajectories can be partially reset by reactivating developmental programs, including Yamanaka-factor reprogramming, Dauer exit, and fasting-refeeding[5]. Any theory of aging must account for all three facts at once: predictability, stochasticity, and reversibility. Neither pure damage-accumulation[6][7][8][9] nor strict programming accommodates the full set without strain.

Meyer, Maklakov, and Schumacher[10] interpret epigenetic and transcriptomic clocks as passive readouts of molecular errors accumulating under declining post-reproductive selection. This strictly Darwinian framing is valuable but incomplete. It absorbs predictability and reversibility into the same stochastic frame that produced the damage in the first place. In doing so, it reinstates a version of the historical dichotomy.

Weismann[11] proposed aging as an evolved program to clear worn individuals and free resources for the young. In the same period, he also articulated a wear-and-tear account based on gradual deterioration through use and accumulated damage[12]. The fact that the same author held both positions reflects the difficulty of the problem rather than a personal inconsistency. Contemporary scholarship suggests he treated the two mechanisms as coexisting rather than abandoning the first for the second. The field has inherited an unresolved tension between the two positions, and dissolving that tension is the aim of the present paper.

I propose a different framework for interpreting the existing data (the site-specific methylation equilibria of Meyer et al., the developmental-gene enrichment in clock sites, the reversibility under reprogramming) as coherent facets of a single phenomenon: the predictable drift of developmentally established regulatory attractors. I call this cybernetic attractor decay (CAD).

# 2. Operational Definitions

I give each term operational content here. Sections 2.1 to 2.4 operationalize the cybernetic attractor, computational drift, regulatory entropy, and the Requisite versus Non-Requisite Variety metric; Section 2.5 then defines gerostasis and the gerotype, the maintained state and its measurable aging phenotype, in those same terms.

## *2.1. Cybernetic Attractor*

A cybernetic attractor is a joint state of a coupled regulatory network. It is self-maintained. In this manuscript, it is defined, operationally, by four layered components: (1) a site-specific CpG methylation pattern; (2) a chromatin state, including PRC2 (Polycomb Repressive Complex 2; modifies chromatin to regulate gene expression) occupancy and broader histone-mark configuration, that reinforces and is reinforced by (1); (3) the transcription-factor occupancy logic anchored by that chromatin state; and (4) the feedback interactions across the resulting regulatory network that return the system to this configuration under perturbation[13][14]. None of the four components is sufficient alone. The attractor is the joint stable basin in the state space defined by these layers. Aging, in this view, is the progressive flattening, broadening, and merging of these basins. It is not a discrete transition between them.

To state this in dynamical-systems terms: let the regulatory state of a cell be a vector s(t) over methylation, chromatin accessibility, transcription-factor occupancy, and expression. A cybernetic attractor is a region A of this state space such that trajectories initialized within its basin B(A) return toward A after perturbation, characterized by three measurable parameters: a recovery rate r (the speed of return to baseline), a basin width b (the magnitude of perturbation tolerated without departure from B(A)), and a state precision p (the tightness of the maintained configuration around A). CAD is the age-dependent decline of p, the narrowing or deformation of b, and the slowing of r, which together raise off-manifold occupancy. This geometric reading is the same quantity formalized information-theoretically in Section 2.4 as the age-dependent rise of X_nr relative to X_req: the present paragraph gives its state-space reading, Section 2.4 its entropic one.

### *2.2. Computational Drift*

Computational drift is operationalized as age-associated loss of regulatory precision. It is measurable as: (1) increased cell-to-cell transcriptional variability in single-cell RNA-seq data[15][16] (dispersion metrics); (2) increased variance in DNA methylation across cells or tissues at co-regulated sites[17]; (3) erosion of cell-type identity, measured as increasing expression of off-lineage gene sets[18]; (4) reduced capacity of regulatory networks to recover from perturbation, including stress response, immune challenge, and metabolic shift[19]. These are not metaphors. They are operations. They constitute the measurable face of the gerotype. The term gerotype is introduced to distinguish the measurable regulatory-aging state of a cell or tissue from chronological age, phenotypic frailty, or a list of molecular hallmarks.

### *2.3. Regulatory Entropy: A Reformulation*

Shannon entropy is a property of an explicit probability distribution. It is not an intrinsic property of a network. An increase in entropy alone cannot distinguish functional variation (which biological systems require for adaptability) from interfering variation (which degrades function). Total state variation X can be decomposed into Requisite Variety (X_req), in the sense of Ashby's requisite variety[20], the variation needed to sustain function under current constraints, and Non-Requisite Variety (X_nr), variation that does not. Computational drift, defined in Section 2.2 as loss of regulatory precision, in operational terms, is the rise of X_nr. This rise is observable as the increasing occupation of off-lineage states, hybrid cell-identity states, and transitions toward trajectories that lie outside the cell's functional domain. The decomposition is empirically testable with single-cell technologies. It also clarifies the relationship to the entropic account of aging. Hayflick[21][22] made thermodynamic instability the universal etiology of aging, equating entropy with the accumulation of molecular disorder in individual biomolecules. This locates entropy in the molecular substrate. Entropy invoked this way, as disorder rather than as a quantity over a defined distribution of states, remains the undefined terminology Hayflick himself[22] identified as crippling the field. The relevant entropy is not a rival quantity. Under the maximum-entropy formulation of statistical mechanics[23], thermodynamic entropy is the Shannon entropy of the probability distribution over microstates, so the substrate-damage account is a special case of the information-theoretic one rather than an alternative to it. The Requisite versus

Non-Requisite Variety decomposition relocates the relevant entropy from the molecular substrate to the regulatory network's control over states and gives it an operational definition: it scores variation against an explicit reference manifold and separates the variation a network requires from the variation that degrades it[24].

### *2.4. Requisite vs. Non-Requisite Variety as the Central Metric*

In this framework, the central explanatory variable for aging is not entropy in the abstract. It is the age-dependent rise of X_nr at the expense of X_req across hierarchically nested regulatory networks. This follows from the law of requisite variety. Ashby[20] derives the bound $H(E) \geq H(D) - H(R)$, where H(D) is the variety (entropy) of disturbances, H(R) the variety the regulator commands, and H(E) the variety of outcomes; a regulator can force outcome variety down only insofar as it holds matching variety of its own. Under CAD, aging is the age-dependent decline in H(R) as regulatory architecture decays, which raises the lower bound on H(E), and the rise of X_nr at the expense of X_req is the measurable expression of that rising bound. Operationally, X_req is the on-manifold variation that maps onto the reference cell-type expression and methylation manifold, and X_nr is the off-manifold occupancy (off-lineage and hybrid-identity states) scored against that reference; the ratio is therefore computable wherever a reference manifold and single-cell profiles are both available. Whether the total X is conserved or grows is an empirical question and may differ across tissues. The framework predicts that the ratio X_nr/X_req is the cleaner signal. This is what aging clocks measure indirectly. This is what rejuvenation interventions reduce. The gerotype is the measurable expression of this ratio at a given biological age.

Defining the reference manifold in vivo is the principal measurement challenge for this decomposition, since technical dropout in single-cell assays and transient physiological plasticity can both displace a cell from its reference position. CAD addresses this operationally rather than by assumption. The reference manifold is estimated from young or healthy single-cell atlases after denoising and imputation, and the on-manifold region is bounded by its observed dispersion rather than by a hard boundary. Aging drift (X_nr) is then distinguished from reversible plasticity (a transient shift within X_req) not from a single snapshot but by its dynamics: true X_nr is off-manifold occupancy that persists and that fails to revert toward the reference after a standardized perturbation or across longitudinal sampling, whereas functional plasticity reverts. This makes the requisite versus non-requisite distinction a recovery-defined quantity, consistent with the perturbation-recovery criterion used throughout this paper, rather than a static classification vulnerable to noise.

### *2.5. Gerostasis and the Gerotype*

Gerostasis is the maintained, low-drift state of the attractor: the regime in which Requisite Variety dominates and the recovery parameters r, b, and p of Section 2.1 remain near their developmental values. It is the regulatory condition that interventions aim to preserve or restore. The gerotype is the measurable aging phenotype of a cell, tissue, or regulatory network at a given biological age. It is operationalized as the X_nr/X_req ratio of Section 2.4 together with the single-cell dispersion and perturbation-recovery metrics of Section 2.2. The gerotype is the operational expression of attractor decay, and it plays the role that catalogs of cellular features play in other accounts but is tied directly to regulatory state rather than to a list. Gerostasis and the aging gerotype are therefore two ends of

one axis: gerostasis is low and stable X_nr/X_req, and the aging gerotype is its age-dependent rise.

## 3. Three Observations the Framework Reads as One Phenomenon

Meyer et al. report three findings. They report them as data points compatible with stochastic decay. I argue they are jointly more coherent under CAD, where each is a facet of the same underlying gerotype.

### *3.1. Site-Specific Equilibrium States*

CpG sites converge toward site-specific equilibrium methylation levels, not uniformly 50 percent, determined by local regulatory context. Context-free stochasticity should produce drift toward intermediate methylation. It should not produce drift toward distinct site-specific targets. The site-specific equilibria are the empirical signature of underlying regulatory architectures setting those targets. This is what an attractor-based account predicts. Local sequence context and DNMT/TET kinetics can also fix site-specific equilibria without invoking an attractor; the discriminating claim is therefore the stronger one that these equilibria track network-level regulatory state rather than local context alone.

### *3.2. Developmental-Gene Enrichment in Clock Sites*

Aging clocks enrich for PRC2-bound developmental genes[25][26][27]. Meyer et al. attribute this to a clearer signal at developmentally regulated loci. I interpret it differently. Developmentally regulated genes define the boundaries of the regulatory landscape established during ontogeny (the progressive construction and stabilization of biological attractors across the organism's lifetime). The boundaries are where small shifts in regulatory state produce the largest detectable signal. This is consistent with the Salnikov and Baramiya distinction between housekeeping and integrative gene groups, insofar as the boundary genes that aging clocks pick up are concentrated in the developmentally specified, tissue-integrative class. Whether the specific HG-IntG partition validates in independent datasets remains to be established. The framework here is compatible with this partition if it does, and with a more graded picture[15][18] if it does not.

### *3.3. Rejuvenation reverses clocks without removing molecular lesions*

Yamanaka-factor reprogramming, partial reprogramming, and developmental-program reactivation reverse aging clocks without requiring the complete removal of accumulated molecular lesions. Under CAD, this is direct evidence that clocks measure attractor state, not damage load. Reprogramming returns cells toward developmental attractors in which the feedback architecture maintaining regulatory precision is intact. The damage may persist, but its propagation through the network is suppressed. The gerotype is reset; the underlying lesions are not.

Damage and attractor decay are therefore not competing causes but distinct levels of description. Stochastic molecular lesions can initiate decay by altering transcription-factor binding affinities, feedback gains, and chromatin accessibility; CAD concerns the regulatory level at which such perturbations become clock-like, heritable across cell divisions, and reversible. The claim is not

that damage is absent upstream, but that the proximal determinant read by aging clocks, and the target reset by rejuvenation, is the attractor state rather than the inventory of lesions. A theory pitched at this level is not rendered redundant by the existence of an underlying physical substrate, any more than thermodynamics is rendered redundant by molecular mechanics.

The three observations are individually compatible with stochastic accounts. They are jointly more coherent under CAD.

## 4. Integration with existing frameworks

CAD is positioned as an interpretive umbrella over a family of compatible accounts. The framework does not claim novelty over the mechanisms each of those accounts identifies. It claims that those mechanisms can be read as facets of a common phenomenon: the age-associated decay of developmentally established regulatory architectures. The integrative move is what gives CAD its empirical content, since it allows different operationalizations (hyperfunction, HG-IntG redistribution, dynamical complexity, network resilience) to be compared on a common metric.

This integrative stance does not reduce CAD to a relabeling of existing accounts. Its contribution is methodological and conceptual rather than mechanistic. Methodologically, CAD supplies a single intervention-comparable fidelity metric, the ratio of Non-Requisite to Requisite Variety (X_nr/X_req), together with explicit falsification conditions, which neither epigenetic-landscape models nor information-loss accounts provide. Conceptually, CAD reconceives the developmental attractor: not as Waddington's fixed topography down which the state passively slides, and not as the undirected accumulation of molecular noise, but as an actively self-maintained control structure whose basin is continuously regenerated by requisite variety. Aging is then the closed-loop failure of the regulator to match its variety to disturbance[20], a directed loss of regulatory fidelity rather than either a passive descent or an unstructured rise in entropy. The information-theoretic reading of aging that CAD operationalizes is one the present author has advanced over a long period; CAD's addition is to render that reading measurable and falsifiable.

More broadly, the cybernetic core of CAD can be situated within a wider framework in which the organism is treated as an active biological-information entity that co-constructs, and is co-constructed by, its regulatory environment, so that senescence is the intrinsic decay of this self-maintenance rather than an externally imposed schedule. This distinguishes CAD from a purely homeostatic or control-theoretic reading, in which the set-point is given rather than self-generated. That wider framework is developed separately and is not required here: CAD rests on the operational definitions and falsification conditions given above, and the present paper should be assessed on those alone.

### *4.1. Quasi-programmed senescence and hyperfunction*

Blagosklonny[1] argues that aging is not a dedicated death program but the continuation of developmental programs (mTOR-driven growth signaling) past their useful point. The continuation produces hyperfunctional outputs (hypertension, hyperplasia, insulin resistance) and secondary decline. CAD and quasi-programmed senescence share the rejection of strict programming. Both view developmental architecture, not damage accumulation, as setting aging trajectories. They differ on the primary mechanism. Blagosklonny locates it in

sustained hyperfunction. CAD locates it in the progressive loss of regulatory feedback fidelity within developmentally established attractors. The two views are complementary. Hyperfunction describes one specific route by which gerostatic states destabilize, particularly in tissues where the dominant regulatory loops are growth-promoting. The finding that fat-free-mass-adjusted energy expenditure is stable through midlife before declining in older adults ([28]; see also [29]) suggests that aging at the organismal level is better understood as a redistribution within a roughly stable energy budget than as a continuous gain of function. CAD is compatible with this constraint. Hyperfunction is more challenged by it.

### *4.2. Housekeeping/integrative gene partition*

Salnikov and Baramiya[2] and Salnikov[30] propose that aging is driven by a progressive redistribution of cellular resources from housekeeping genes (HG) to integrative, tissue-specialized genes (IntG). The redistribution starves repair capacity in postmitotic and low-mitotic-index cells. Their methylation evidence[31] shows differential age trajectories between the two gene classes. Independent replication and a direct methylation-to-expression demonstration remain open. CAD is compatible with HG-IntG redistribution as the proximal mechanism if the partition is independently validated. It remains applicable if a more graded loss of regulatory specificity[15][26][27][18] proves to be the more accurate picture. Framing aging as a loss of feedback between infrastructure and specialized gene activity, rather than as "software errors," captures the CAD intent better than an error-propagation account does.

### *4.3. Loss of dynamical complexity*

Kyriazis[3] argues, drawing on chaos theory, that aging reflects a loss of dynamical complexity across hierarchical physiological systems. He proposes that this loss can in principle be slowed by maintaining a multiplicity of stimuli and non-regular dosing of interventions. Kyriazis's framing maps directly onto CAD. Loss of dynamical complexity is loss of the attractor's capacity to support functional variation (Requisite Variety). The practical recommendations (varied physical and mental stimulation, non-monotonic dosing) are precisely the interventions a CAD-informed practice would suggest. Kyriazis also raises a question CAD must address. At what point does information construction (development) cross over into information maintenance failure (aging)? Why does the germline successfully avoid this transition where the soma does not? See Section 5.

### *4.4. Complex systems approaches*

Cohen et al.[4] advocate a complex-systems framework for aging biology, emphasizing emergence, network dynamics, and resilience. CAD is in this lineage. The operational definitions of Section 2 are an attempt to satisfy the call in that literature for formally specified, testable network metrics.

CAD does not replace these frameworks. It re-reads them as views from different angles of a common phenomenon: the age-associated decay of developmentally established regulatory architectures, expressible as the rise of Non-Requisite Variety across hierarchically nested networks, and observable as a measurable gerotype.

## 5. Why does this loss begin? (germline versus soma)

Information content rises during development and falls during aging. What defines the inflection point? Why does the germline avoid the fall? I do not claim to have a complete answer. CAD gives a partial one.

The inflection point is not a calendar age. It is the systems-level transition from net information construction (development organizing matter and energy into regulatory architecture) to net maintenance failure. Once the architecture is no longer being built, it must be preserved by repair, surveillance, and feedback recalibration. The cost of preservation begins to exceed the rate at which preservation succeeds. The crossover is dynamic and tissue-specific. CAD locates the threshold not in a switch but in the rising $X_{nr}/X_{req}$ ratio at the network level. The crossover marks the onset of an aging gerotype.

The germline does not escape entropy in any thermodynamic sense. It is embedded in a reproductive cybernetic loop. The loop includes meiotic bottlenecking, epigenetic reprogramming, repair surveillance, and developmental reconstruction at every generation[32]. The germline loop is a full reset of the attractor architecture. The soma lacks this. The disposable-soma logic[9] and the argument that developed organisms are interlocked feedback systems with their environments converge here. The reset loop is available only along the germline. The remaining question is how the germline sustains the fidelity required to execute this reset generation after generation without incurring the same rising cost of preservation that defeats the soma. CAD does not require the germline to be exempt from that cost; it requires the cost to remain affordable. Three factors keep it so. The germline is a small, largely quiescent lineage, so its per-generation accumulation of regulatory drift is low. It is under strong purifying selection rather than the relaxed post-reproductive selection that permits somatic drift[9], so configurations that would compromise the reset are removed at the population level. And damaged gametes are culled before transmission, so the reset operates on a pre-filtered, low-drift input rather than on the full somatic load. The germline therefore does not maintain perfect fidelity; it maintains a preservation cost low enough that the generational reset can overwrite residual drift, which is precisely the condition the soma fails to meet.

## 6. Testable predictions

CAD makes specific predictions that distinguish it from pure stochasticity accounts. They are testable with current technologies. Each prediction concerns a measurable feature of the gerotype.

### *6.1. Tissue-specific signatures of attractor drift*

Different regulatory architectures should show distinguishable signatures of drift. A stress-response gene regulatory network, organized around NF-κB, AP-1, and HSF1, relies on transient inducible activation and rapid feed-forward motifs. Under CAD, it should show attractor drift as kinetic decoherence: broader and more variable activation kinetics with age, a slower return to baseline, and incomplete induction of target sets. A metabolic homeostasis network, organized around stable set-points with tight negative feedback (insulin-glucose, AMPK-mTOR), should show attractor drift as set-point drift and reduced feedback gain: progressive baseline shifts, oscillatory instability, and widened tolerance bands.

The same underlying drift produces distinct, measurable tissue-level signatures. Both are testable in existing single-cell time-series and time-resolved multi-omics datasets. The distinction defines two contrasting gerotypes within the same organism.

### *6.2. Variance metrics outperform mean metrics*

If CAD is correct, age-associated changes in transcriptional, proteomic, and methylation variance, within and between cells, should track biological age and predict mortality better than mean-level changes or mutation burden alone. Tong et al.[33] report that approximately 63 to 90 percent, depending on the clock, of epigenetic clock predictive accuracy is captured by a stochastic process. This is consistent with CAD. Clocks measure stochastic trajectories through a structured landscape, and the variance is the dominant signal. The residual non-stochastic 10 to 37 percent should localize preferentially to PRC2-bound, co-regulated, and developmentally constrained sites, the boundary regions of the regulatory landscape.

### *6.3. Co-regulated and stochastic CpG decomposition*

Tarkhov et al.[17] distinguish co-regulated from stochastic CpG sites in single-cell methylation data during aging. This provides empirical resolution of the two components CAD requires: (1) a structured scaffold (co-regulated sites) and (2) a noise field (stochastic sites). CAD predicts that the co-regulated component will dominate in the boundary regions of the attractor landscape (developmental, PRC2-bound). The stochastic component will dominate in the bulk.

### *6.4. Comparative biology among species*

CAD makes predictions across species with unusual aging trajectories. Negligibly senescent organisms (Hydra, planarians, naked mole rats, quahogs) should show preserved attractor topology under perturbation. This is measurable as maintained feedback gain and resistance to set-point drift. Their gerotype should remain stable across decades. Organisms whose senescence is triggered abruptly after reproduction (semelparous Pacific salmon) should show coordinated, system-wide attractor collapse rather than the gradual broadening predicted for typical mammalian aging. Under CAD, this collapse is not a dedicated death program but the action of a single post-spawning regulatory trigger (a hormonal cascade) that withdraws the feedback sustaining many attractors at once, so the gerotype should pivot abruptly rather than drift. The distinguishing claim is that the abrupt phenotype reduces to loss of the same recovery parameters (r, b, p) as gradual aging, applied synchronously, rather than to a separate aging program. Short-lived but iteroparous models such as the killifish Nothobranchius, by contrast, should show compressed yet still gradual drift rather than abrupt collapse. Eusocial insect castes provide a particularly clean test. Queens and workers share identical genomes but show order-of-magnitude lifespan differences. Under CAD, the difference should localize to feedback architecture and chromatin state, not to genome-encoded program differences.

### *6.5. Intervention strategy for aging*

Interventions that restore network topology, including feedback gain, regulatory circuit fidelity, and chromatin architecture, should outperform interventions

targeting individual nodes or lesions. Partial reprogramming, senolytic intervention, circadian restoration, and rapamycin-class agents acting on mTOR are consistent with this prediction. That pulsed and non-monotonic dosing regimens outperform constant dosing follows from the same principle. A system whose attractor topology is preserved by feedback can be perturbed more usefully by stimuli that exercise the feedback than by stimuli that override it. CAD provides a framework for evaluating these against a common metric (Requisite vs. Non-Requisite variety, expressed in the gerotype) rather than against disparate molecular endpoints.

### *6.6. Falsification conditions*

CAD is not confirmed by every outcome, and the framework specifies results that would count against it. First, if age-associated decline in perturbation-recovery capacity (feedback gain and return-to-baseline kinetics) is absent once mean-level molecular change is controlled for, the central claim that clocks read attractor state rather than damage load fails. Second, if negligibly senescent species (Hydra, planarians, naked mole rats, ocean quahogs) show the same age-related loss of feedback gain and set-point stability as typical mammals, the prediction of preserved attractor topology fails. Third, if the non-stochastic residual of epigenetic clocks does not localize preferentially to co-regulated, PRC2-bound, developmentally constrained sites but is instead distributed uniformly across the methylome, the boundary-region claim of Sections 3.2 and 6.2 fails. Fourth, if variance and dispersion metrics do not track biological age or predict mortality beyond mean-level and mutation-burden predictors, the operational core of the gerotype is unsupported. Fifth, if rejuvenation interventions reverse clocks only by physically removing molecular lesions, with no measurable restoration of feedback architecture, the attractor-reset interpretation of Section 3.3 fails. Sixth, if semelparous Pacific salmon reach death through the same gradual, distributed broadening of attractor basins seen in iteroparous mammals, rather than through an abrupt coordinated collapse following a reproductive trigger, the system-wide-collapse prediction of Section 6.4 fails. CAD therefore stands or falls on the measurability of regulatory recovery and variance, not on the mere presence of structure in aging data.

## 7. Scope and limits

I propose that the existing data on epigenetic clocks, partial rejuvenation, developmental-gene enrichment, site-specific methylation equilibria, transcriptional noise, and cell-identity erosion are jointly more coherent under CAD than under pure stochasticity or strict programming. The operationalized quantities of Section 2 definitions give CAD empirical content rather than metaphorical content. The gerotype is the empirical object the framework targets.

I am not claiming that Darwinian natural selection plays no role in aging. I am claiming that selection acts on parameters within a regulatory architecture whose topology is set by developmental, physical, and energetic constraints. The architecture itself is not pre-specified, only tuned, by selection. The “structurally constrained” reading of programmed aging is what I intend. “Selection-adjusted” is compatible with this. “Selection-generated” is not. I leave a fuller defense of this distinction to a separate treatment.

I am not claiming that CAD makes immediate clinical predictions beyond those of the frameworks it integrates. Its principal value is heuristic. It gives a common

metric across damage-based, hyperfunction-based, and HG-IntG-based accounts. It gives a common operational basis (the gerotype) for comparing interventions that act on different molecular layers.

## 8. Conclusion

The data Meyer et al. present, including site-specific methylation equilibria, developmental-gene enrichment in clock sites, and reversibility under reprogramming, are jointly the signature of an underlying process. That process is best described as predictable drift of developmentally established regulatory attractors. Cybernetic attractor decay is a framework that makes this reading explicit. It anchors its terminology in measurable quantities: single-cell dispersion, methylation variance, perturbation recovery, and Requisite vs. Non-Requisite Variety, integrated as the gerotype. It treats existing accounts (quasi-programmed senescence, hyperfunction, HG-IntG redistribution, loss of dynamical complexity, complex-systems aging) as compatible views of the same phenomenon from different angles. The framework is testable through tissue-specific signatures of attractor drift, variance-based aging metrics, comparative aging across species with unusual trajectories, and intervention strategies that target network topology rather than individual lesions. The empirical paradox that opened this paper—predictable yet stochastic yet reversible aging—is not a paradox under CAD.

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### *Potential Competing Interests*

The author is the founder and CSO (Chief Scientific Officer) of AgingLab Co., Ltd. and declares no other competing interests.

### *Data Availability*

No new data were created or analyzed in this study. Data sharing is not applicable to this article as it is a theoretical framework and synthesis of existing literature.

### *Author Contributions*

J.B. was the sole author and is responsible for all aspects of the manuscript.

### *Use of Generative AI*

AI tools (Claude Opus and ChatGPT) were used to read previous literature, follow instructions and directions, generate and clarify sentences, integrate cited references into the framework, and polish English. All scientific claims, structural arguments, and the framework itself are the author's original contribution and responsibility.


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