Commentary

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

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The debate between stochastic deterioration (Meyer et al., 2025) and programmed senescence perpetuates a false dichotomy dating back to Weismann. The precision of molecular aging clocks and aging reversibility demands a different explanation: aging is neither stochastic wear-and-tear nor genetic programming, but rather cybernetic decay, predictable trajectories emerging as developmental regulatory architectures lose information-processing fidelity. I define biological systems as hierarchical information-processing networks where aging represents computational drift from developmental attractors. Three observations support this framework: (1) site-specific equilibrium states inconsistent with pure stochasticity, (2) developmental network dominance in aging signatures, and (3) rejuvenation restoring regulatory configurations rather than repairing molecular damage. This framework reconciles why aging follows predictable population trajectories despite individual variability (conserved computational architectures), why epigenetic clocks work (measuring attractor drift), and why reprogramming reverses aging (restoring computational precision). It predicts that regulatory network entropy outperforms mutation burden in age prediction, and that interventions restoring information coherence reverse aging clocks more effectively than targeted molecular repair. Under this framework, biological entities function as computers, and aging emerges as a fundamentally reversible and controllable process across both short-term development and long-term evolution.

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Introduction

In “Aging by the clock and yet without a program,” Meyer et al.[1] reject adaptive theories of programmed aging, interpreting epigenetic and transcriptomic clocks as passive readouts of molecular errors accumulating under declining post-reproductive selection. This evolutionary framework has been valuable for decades, but it is incomplete. The authors repeated the error of replacing one oversimplification (aging as program) with another (aging as pure stochasticity), missing the integrative explanation their own data suggest.

This binary framing has deep historical roots. August Weismann struggled with this conceptual tension. In his essay “The Duration of Life” (Weismann, 1881, 1889)[2], he proposed that aging was an adaptation of Darwinian evolution, a biological program to remove worn-out individuals and release resources for younger generations. Just one year later, the same Weismann (1882)[3] introduced the wear-and-tear theory, arguing that aging results from gradual deterioration of cells and tissues through accumulated damage and repeated use. The fact that the same scientist had to propose both programmed death and stochastic wear-and-tear as alternative explanations reveals the complexity of the aging problem and the inadequacy of either framework alone in explaining it.

This age-old conceptual schism persists in Meyer’s analysis published in Nature Aging journal in 2025. Three key observations from their paper point toward a more complete model: (1) CG base sites converge toward site-specific equilibrium states determined by local regulatory context in genomes, (2) rejuvenation through developmental programs (Yamanaka factors, Dauer exit, fasting-refeeding) reverses aging clocks, and (3) molecular aging clock sites enrich for developmentally regulated genes. Rather than being anomalies requiring explanation, these patterns reveal aging as the “predictable” degradation of “quasi-programmatic” regulatory architectures under stochastic stress, a synthesis that transcends Weismann’s original dichotomy.

The Equilibrium Attractor Problem: Echoes of Weismann’s Paradox

Meyer et al. provide compelling mechanistic detail showing each epigenetic CG methylation site trends toward a “site-specific equilibrium state” shaped by “local genomic and epigenomic context” (Fig. 2c). This insight inadvertently undermines their stochasticity thesis in three ways, while paradoxically resurrecting the very programmatic elements Weismann (1881, 1889)[2] originally proposed before abandoning them for wear-and-tear explanations.

First, if equilibrium states are site-specific rather than randomly distributed, the predictability of aging derives from these deterministic attractors, not from non-deterministic stochastic accumulation patterns. The stochastic component becomes noise around fundamentally determined trajectories, precisely the "programmed continuation" Meyer argues against.

Second, the authors cannot explain why "imperfect maintenance" produces site-specific equilibria rather than random drift toward uniform intermediate methylation levels. The fact that different sites converge to different equilibrium points (not all 50%) indicates underlying regulatory "programs" are setting these targets. Weismann faced the same problem: wear-and-tear should be random, yet aging follows predictable patterns across individuals and species.

Third, this model is functionally indistinguishable from weak programmatic aging, the very concept they argue against. If regulatory context determines where each site will drift, then aging reflects the systematic erosion of context-dependent regulatory states established during development. Weismann's original insight that early biological development establishes conditions determining later decline resurfaces here despite Meyer's attempt to exclude it.

Recent molecular aging clocks measure the predictable decay of developmental regulatory architectures. These architectures are "informational structures" established during development under host-environment intertwined computational states and "programmatic" in their organization but not actively programmed to fail. Aging emerges from their imperfect maintenance rather than from any "dedicated aging program", resolving an apparent paradox: aging requires neither a "death program" nor "pure stochastic damage", but instead reflects the gradual erosion of developmental control systems that are heavily influenced by para-determined (non-fate) and dynamic (time-dependent) epigenomic and subsequent RNA and protein expression patterns.

The remarkable predictability of epigenomic and transcriptomic aging clocks supports this view. Such precision suggests that aging arises from quasi-programmed processes, developmentally structured computational systems whose decline follows predictable trajectories, rather than from purely random damage accumulation. This interpretation transcends classical frameworks and points toward a broader principle: understanding life history requires focusing on developmental architectures and their "intrinsic constraints and drivers", not merely on trait-by-trait optimization through Darwinian natural selection. Molecular aging clocks do not measure damage, but they measure the "decay of developmental order" itself that has been shaped by the intertwined interaction of the long-term evolution (evo) and short-term development (devo).

The PRC2 Circularity: Weismann's Unresolved Tension

Meyer et al. explain why aging clocks enrich for PRC2-bound developmental genes by arguing these sites were "tightly regulated during development before becoming repressed," making errors more visible. However, this explanation contains circular logic that mirrors Weismann's struggle to reconcile developmental programs with aging mechanisms:

- Clocks enrich for developmentally regulated sites because development created regulatory precision
- That regulatory precision determines which sites show age-dependent patterns
- Therefore, the apparent "program" reflects the developmental program that previously operated for billions of years

The authors essentially concede that "developmental programming" determines clock behavior while insisting this is not programmatic. Weismann made the same conceptual move when arguing that Darwinian natural selection optimizes organisms for reproduction but does not actively program death, yet his own logic implied that developmental optimization determines the trajectory and timing of decline. The more accurate statement is that aging clocks track the loss of developmental regulatory precision, making them "indirect readouts of programmatic architecture decay".

This interpretation explains both why rejuvenation works (reactivating developmental programs restores precision) and why clocks are predictable (they measure loss of conserved architecture). Pure stochasticity cannot explain either phenomenon without invoking deterministic regulatory contexts, which are themselves programmatic elements.

Aging as Computational Phenomenon: The Cybernetic Computation Framework

The binary framing obscures a more complete framework rooted in understanding life as computation. At its core, living systems are not merely doing computation, they are computation as "recursive information processing" that maintains far-from-equilibrium states by continuously transforming energy and matter into organized structure.

Every biological process involves proactive and purposeful (intrinsic teleonomy) information storage, transmission, error detection and adaptive updating, literal computational operations with measurable information-theoretic properties[4]. I propose that a cybernetic computation evolution framework is

necessary to describe the simultaneous evolution of computational architectures across molecular, cellular, tissue, organismal and ecological scales. In this computational perspective of life propagation, aging emerges when computational fidelity degrades across this recursive and fractal stack due to thermodynamic costs, error propagation, optimization trade-offs favoring early-life performance[5][6][7], and attractor drift without sufficient error correction.

Molecular (omic) aging clocks measure this drift. They work because computational decay follows predictable trajectories determined by "network topology and energy constraints", not because aging is "programmed."

This cybernetic view integrates Meyer et al.'s insights through a computational lens:

- Developmental architectures establish regulatory capacities and network topologies (explains cross-individual similarity and developmental gene enrichment)
- Declining selective pressure permits these capacities to be suboptimal for late life (explains directionality and species differences)
- Stochastic perturbations probe architecture robustness at different rates (explains individual variation)
- Equilibrium attractors emerge from regulatory context (explains site-specific trajectories)

The apparent contradiction between predictability and stochasticity dissolves: predictability reflects evolutionarily converged "conserved universal computational architecture"; stochasticity reflects variation in how damage probes and interacts with that architecture.

Stochasticity Requires Disaggregation

Meyer treats stochastic errors as uniformly degradative, but aging involves not just increased noise but loss of capacity to structure noise productively. Exogenous stochasticity (environmental shocks) and endogenous micro-stochasticity (replication errors) drive decline, while control-induced variability in immune repertoires and neural circuits enhances resilience.

The Rejuvenation Paradox Resolved

Meyer's paradox, that developmental programs can reverse aging despite aging being characterized as fundamentally stochastic, resolves through the cybernetic attractor framework. Aging represents drift from optimal attractor states; developmental programs reset regulatory networks to suppress damage

expression rather than overcome physical damage itself. Embryos establish regulatory configurations where damage cannot propagate through the system. Thus, rejuvenation restores “attractor landscapes” rather than repairing every molecular lesion. Reproduction represents the most extreme form of rejuvenation, completely restoring attractor landscapes by resetting both the epigenetic and cellular environment and developmental programs of germline cells.

Testable Predictions

The computational framework suggested here to explain aging can generate falsifiable predictions: (1) aging trajectories will correlate more strongly with Shannon entropy in regulatory networks than DNA mutation burden alone[8]; (2) network redundancy and robustness metrics will predict longevity better than repair enzyme abundance[9][10]; (3) interventions restoring computational precision will reverse aging clocks more effectively than targeted molecular repair; (4) organisms maintaining “beneficial stochasticity” will show enhanced resilience. These predictions distinguish computational frameworks of aging, lifespan, and life propagation from pure stochasticity theories and are testable with current technologies.

Conclusion

Meyer et al. reaffirmed the stochasticity-program dichotomy in aging by overcorrecting against programmatic explanations, framing the ultimate cause as evolutionary neglect coupled with random molecular damage. A more accurate understanding requires integrating both views within a “computational framework”, where life and aging are expressions of recursive information processing that sustain far-from-equilibrium states[11][12]. Aging then emerges as predictable, structured computational degradation: a systematic decline in information-processing fidelity across co-evolving intertwined hierarchical layers, with each layer’s decay amplifying errors through recursive feedback. This “computational evolution” perspective explains why diverse aging clocks, such as Horvath’s epigenetic clock of very limited not-directly aging associated loci[13], capture computational drift, why rejuvenation interventions succeed by restoring computational configurations, and why aging follows trajectories that are both predictable and variable. Meyer’s site-specific equilibrium states can thus be interpreted as computational attractors shaped by regulatory programs.

Statements and Declarations

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Conflicts of Interest

The author is the CSO (Chief Scientific Officer) of AgingLab Inc. and declares no other competing interests.

AI Disclosure

AI tools (Claude Opus 4.5 and ChatGPT 5.2) were used to assist in formatting the reference list and polishing the manuscript language.

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