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Peer Review

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

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The article “Aging as Cybernetic Attractor Decay: Beyond the Stochastic-Programmed Dichotomy” by Jong Bhak offers a fresh take on a debate that is more than a hundred years old. It presents a cybernetic computational framework for thinking about aging. It differs mainly in what it treats as the primary “thing that ages.” Traditional theories focus on damage or lack of evolutionary intent or neglect; the cybernetic view focuses on loss of regulatory information-processing fidelity in hierarchical control networks.

In stochastic damage theories, aging is driven by cumulative, largely random molecular lesions (DNA damage, misfolded proteins, oxidative damage), and functional decline results from accumulated failures. Programmed or quasi-programmed theories suggest that aging reflects the continuation or misfiring of developmental programs or genetically encoded timing mechanisms.

In the cybernetic computation framework, aging is computational drift where the organism’s multi-level regulatory networks (epigenetic, transcriptional, cellular signaling, tissue homeostasis) lose the ability to maintain and error-correct youthful states, so the system drifts away from developmental “attractors.”

The main argument is that aging is predictable, or, more specifically, that molecular clocks work at all. For a damage-focused view, predictability must stem from similar rates of damage accumulation and shared constraints, while randomness should be substantial. In the cybernetic view, predictability arises from organisms sharing conserved network architectures and attractor landscapes. Random perturbations occur, but they mainly create noise around a structured trajectory determined by the control system.

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So, the argument must come down to how we interpret aging clocks. The traditional view holds that epigenetic/transcriptomic clocks are readouts of accumulated errors or downstream correlates of damage. The author argues that clocks work well because they measure the state drift of regulatory networks (movement away from youthful attractors), not just damage load. Which side of this argument you favor probably depends on whether you think clocks extract beautiful nuggets from a sea of noise or whether their functionality points to an underlying commonality to all aging.

The most interesting argument is reframing the mechanism of rejuvenation. How can Yamanaka factors reverse aging if aging is random? To reverse aging, you must repair or remove the underlying molecular lesions (senescent cells, mutations, crosslinks, etc.). In the author's cybernetic framing, rejuvenation can occur by resetting control architectures (partial reprogramming) and restoring coherent regulation, so the organism returns toward youthful attractors even if not every lesion is individually repaired.

Overall, an intriguing take, even offering testable hypotheses for distinguishing among the models. Perhaps even shifting what we should target, moving away from individual "hallmarks of aging." The new framework, or "network-level metrics" (regulatory coherence, entropy, robustness, redundancy, feedback control quality), predicts that these may outperform damage metrics in forecasting age and in guiding interventions. Essentially, damage theories emphasize "what breaks," while the cybernetic computation framework emphasizes "what loses control," treating aging as a systems-level degradation of information processing that can, in principle, be reset.

Declarations

Potential competing interests: No potential competing interests to declare.