Reversed polarity: a control law for holding a maintained epigenetic state—and why it is not what bulk aging measures

Nathan Hangen[1]

[1] *Independent Researcher*

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A cell that maintains its epigenetic identity against copy noise can, in principle, do so in two regimes. In the *disordered* regime it pays continuously to correct each locus, and information still decays toward a floor (the aging of the companion paper [1]). In an *ordered* regime—where read–write feedback couples neighboring loci strongly enough—the youthful configuration becomes a broken-symmetry free-energy well, held at near-zero maintenance cost, and drifting away must cross a collective barrier. We formalize this "reverse-polarity" regime with the same governing law as the companion paper, $\tau_0 \dot{M} = JM - T\,\mathrm{artanh}(M) + h_{\mathrm{ext}}$, give the economics of when collective protection beats per-locus pumping, and test it interventionally in simulation. Imposing strong local consensus (neighbor agreement, no stored reference) on a two-dimensional lattice—the rule is Toom's north–east–centre majority [6], a monotone self-dual eroder long known to be a reliable classical memory—sustains an ordered phase that holds an aligned identity domain at retention $R \approx 1$ indefinitely, while a baseline lineage and a one-dimensional coupled lineage both relax to $R \approx 0.5$. The intervention is bounded: it requires $\geq$ 2-dimensional connectivity (1D cannot order), it fails above a critical copy noise $p_c \approx 0.22$, and—a non-obvious negative—near criticality *larger* domains melt *more* readily, because a bigger domain offers more sites to nucleate an aged droplet (melting rate $\propto$ area), contradicting the naive coherent-flip intuition that bigger domains are safer. We map three biological levers (lower noise T, raise coupling J, re-nucleate order) onto Polycomb/heterochromatin read–write feedback, note the oncogenesis/rigidity frontier that bounds over-imposed order, and specify a synthetic-domain size-sweep experiment that would validate the physics. The contribution is a control-theoretic characterization of a maintained epigenetic state—its equilibrium, its barrier, its failure modes—not a claim that natural aging is this melting reversed.

I. FRAMING

This paper gives a control law for the maintained-state dynamics of the same epigenetic order parameter M, and under the same governing law, as the companion paper—but in the opposite *regime*. The companion paper concludes human blood sits in the *disordered* regime ($T > J$, $\mathcal{N} = J/T_{\mathrm{eff}} < 1$): the law reduces to relaxation-to-floor, there is no collective barrier, and the measured aging is cell-composition mixing plus per-locus decay with no within-cell melting signature at composition-inert sites. Here we explore the *ordered* regime ($T < J$, $\mathcal{N} > 1$), where a broken-symmetry barrier exists and can be engineered. This is the honest decoupling: not "different variables" (a physicist rightly reads that as evasion) but *one equation, two regimes separated by* $\mathcal{N} = 1$. We do *not* claim the aging the companion paper measures is a melting run backward—in the $\mathcal{N} < 1$ regime there is no ordered phase to have melted. We ask what a control law that *engineers* the $\mathcal{N} > 1$ regime could do; it is not a wet-lab reversal-of-aging claim. Every "arrests" below is bound to the simulation and its critical-noise regime.

II. ORDERED-PHASE PHYSICS: A MAINTAINED STATE AS A BROKEN SYMMETRY

The governing law's ordered limit ($J > T$, dimension ≥ 2) develops two free-energy wells at $\pm M_0$ separated by a barrier Δf. A domain sitting in the $+M_0$ well is a broken-symmetry memory: no stored reference tells it which well is "correct," but once committed it resists noise, because escaping requires the whole coupled domain to cross the barrier together. The Arrhenius escape time scales as $\tau \propto \exp(n\,\Delta f/T)$ in the coherent-flip picture, where n is the domain size—the naive reading in which bigger domains are exponentially safer. Section IV shows why that reading fails near criticality.

Two hard constraints frame the regime:

- *Dimensionality.* There is no ordered phase in 1D at finite temperature ($T_c = 0$): a 1D chain of coupled loci cannot hold a broken-symmetry state, because a single domain wall costs finite energy and entropy proliferates it. Order requires $\geq$ 2-dimensional connectivity—a testable prediction confirmed in Sec. IV (1D consensus fails, 2D holds). The stronger statement is that no *simple local* 1D rule stores a bit against noise: a reliable 1D memory is not strictly impossible (Gács's positive-rates automaton [7] is non-ergodic in 1D) but requires a

baroque hierarchical self-simulating construction no biological read–write loop possesses, whereas the 2D eroder does it with a three-cell majority.

- *The one-bit bound.* What an ordered domain protects is its coarse *order parameter*—the ∼1 bit of "which well"—not an arbitrary per-locus codeword. Consensus dynamics smooth away per-locus detail; they preserve the collective alignment. This is the von Neumann one-bit reliability bound [2] made physical: a maintained state stores one robust bit per domain, cheaply, and cannot cheaply store fine-grained identity.

III. ECONOMICS: WHEN COLLECTIVE PROTECTION BEATS PER-LOCUS PUMPING

A cell has two ways to resist decay. It can *pump* each locus individually (kinetic reinforcement rate r per site, the disordered-regime maintenance of the companion paper), or it can *couple* loci into a collective ordered domain that holds many sites with one shared apparatus. The trade-off is set by how the apparatus cost scales with the number of protected loci. Costing both strategies per locus held stable over D divisions (in Landauer resets [5], each maintenance write $\geq k_B T \ln 2$): direct per-locus correction costs

$$\mathrm{cost}_{\mathrm{pump}} = D\, p_{\mathrm{eff}}, \tag{1}$$

while an ordered domain in which one collective barrier stabilizes b correlated loci with n_c apparatus units per domain, each turning over with probability γ per division, costs

$$\mathrm{cost}_{\mathrm{ordered}} = D\left(\frac{n_c\,\gamma}{b} + e^{-B/T}\right), \tag{2}$$

where the residual $e^{-B/T}$ is the (negligible, deep-well) spontaneous-flip cost. Equating the two and dropping the residual, the ordered phase wins iff $n_c\gamma/b < p_{\mathrm{eff}}$, i.e. the critical apparatus turnover is

$$\gamma^* = \frac{p_{\mathrm{eff}}\, b}{n_c}. \tag{3}$$

The entire content is two independence assumptions. (i) $n_c \perp b$: the win is the $1/b$ amortization, which requires *sub-linear* apparatus—one collective read–write field, not a reader/writer bolted to every site. If the machinery is per-locus ($n_c \propto b$) the amortization cancels, γ^* pins at p_{eff}, and the ordered phase collapses into per-locus pumping (the "pump the pump" failure mode). (ii) Barrier depth B drops out of Eq. (3) algebraically, but is paid *inside* n_c: depth buys Arrhenius longevity, not economics. Order-of-magnitude biology (Fig. 1, right) puts heterochromatin (collective, durable) below the pump cost, Polycomb on the crossover, and DNA-methylation maintenance ($n_c \sim b$, per-hemisite) at $\approx p_{\mathrm{eff}}$—pumping in a domain's coat. This is a cost claim only: a domain carries ∼1 identity bit, so the win is cheap redundancy across coupled loci, not tighter identity.

IV. THE INTERVENTION TEST: ENGINEERED CONSENSUS ARRESTS DRIFT, BUT IS BOUNDED

We test reverse polarity as an *intervention*: impose strong local consensus and ask whether the youthful state becomes the free equilibrium so that aging is the expensive, uphill move. The protected object is an aligned domain (the ∼1 bit), started all-aligned; each division applies binary-symmetric copy noise (flip probability p) then optional reinforcement. Retention $R(t) = \langle \mathbf{1}[\mathrm{state}_t = \mathrm{state}_0]\rangle$; $R = 1$ fully young, $R = 0.5$ fully melted. Reinforcement is neighbor consensus only—never comparison to a stored codeword—so the dynamics contain no backup; matching to the initial state is a *measurement*, not a correcting force (the load-bearing invariant). This 2D torus neighbor-majority rule is exactly Toom's NEC automaton [6]: the results below are therefore not a new mechanism but an instance of established fault-tolerant cellular-automaton theory, applied to epigenetic maintenance. Toom's stability theorem grounds every qualitative feature we then simulate—the existence of the ordered phase, its robustness below a noise threshold, and the eroder property that removes minority (aged) droplets—so the simulation checks a known rule in this setting rather than asserting an unproven one.

The 2D-coupled domain holds $R \approx 1$ indefinitely (Table I) while baseline collapses to the melted floor within ∼10 divisions (front-loaded, as the core picture predicts) and 1D—which briefly slows decay—still relaxes to ≈ 0.5. This is "aging made expensive," built from local consensus alone, no backup.

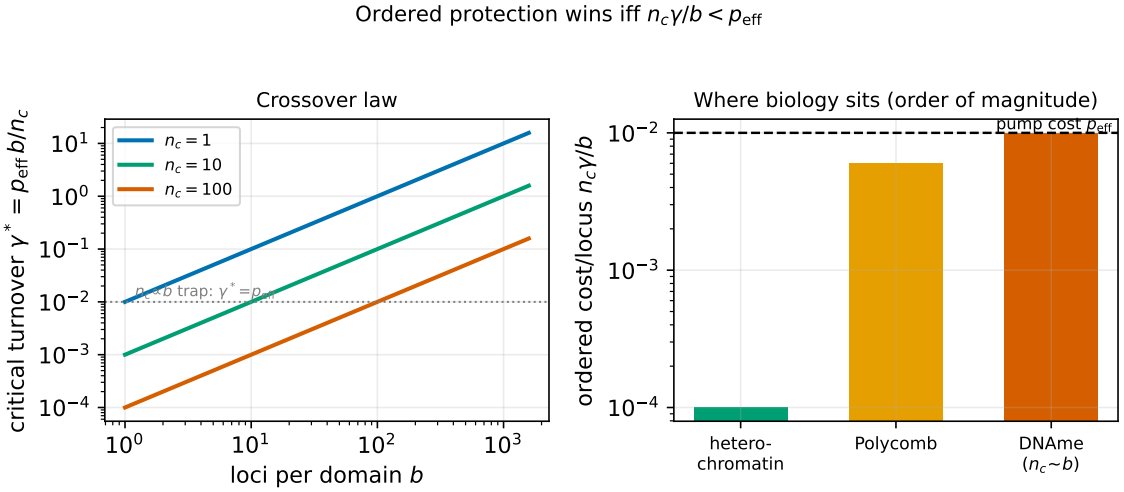

FIG. 1. The maintenance-economics crossover $\gamma^* = p_{\text{eff}}\, b/n_c$ (Eq. (3)). *Left:* critical apparatus turnover vs loci per domain, for several apparatus sizes n_c; collective barriers (large b) and cheap apparatus (small n_c) raise γ^*, while the per-site diagonal $n_c \propto b$ pins it at p_{eff}. *Right:* order-of-magnitude estimates for the three real chromatin systems— heterochromatin wins on maintenance cost, Polycomb sits on the crossover, DNA-methylation maintenance collapses to $\approx p_{\text{eff}}$.

TABLE I. Retention over divisions at sub-critical noise $p = 0.10$ (4096 loci, 150 divisions; seed 20260722).

arm	mechanism	R[10]	R[50]	R[150]
baseline	no reinforcement	0.545	0.496	0.501
1D consensus	ring neighbor majority	0.780	0.533	0.507 (fails, $T_c = 0$)
2D consensus	torus neighbor majority (reverse polarity)	1.000	0.999	1.000 (arrested)

a. Dose-response. Steady R (2D, mean of the last 40 of 150 divisions) stays near 1 for small p ($R = 0.99$ at $p = 0.15$, 0.96 at $p = 0.18$) and collapses through a maintained-to-melted transition at $p_c \approx 0.22$ ($R = 0.57$ at $p = 0.22$, 0.49 at $p = 0.28$). The engineered barrier is finite; above p_c, noise wins regardless of coupling. The threshold is in the family expected for a Toom-class eroder (rigorous bounds are construction-dependent and obtained by the Toom-contour Peierls method [8]); our p_c is a measured value for this specific flip-symmetric rule, not a claim to match any particular published constant.

b. Size dependence near criticality (the non-obvious negative). Retention does *not* improve with domain size—it declines (noisily) as the domain grows: $R = 0.973$ ($L = 8$) $\to$ 0.935 ($L = 16$) $\to$ 0.844 ($L = 32$) $\to$ 0.874 ($L = 64$) at near-critical $p = 0.20$. Larger domains offer more sites to nucleate an aged droplet that then spreads; finite-2D melting is nucleation-dominated with rate $\propto$ area, so the coherent-flip $\tau \propto \exp(n)$ "bigger = safer" intuition is the wrong picture here. *Scaling up a collective domain is not a free lever against aging.* (Deep in the ordered phase all sizes saturate, so this only shows near criticality—the regime an intervention would operate in. The decline is non-monotone at this single seed/lattice; a seed/geometry sweep is required to confirm the area-nucleation trend is not a finite-size artifact.)

c. Verdict. Reverse polarity works as an intervention *in the model*: below p_c, high-connectivity consensus sustains an ordered phase holding the youthful domain at $R \approx 1$, using only local consensus and no backup. But it is bounded and conditional—needs $\geq$ 2-dimensional connectivity, fails above p_c, does not get safer by scaling domain size, costs durable collective apparatus, and over-imposing order is the rigidity/oncogenesis failure mode (Sec. VI).

d. Why maintenance must be non-equilibrium. A subtlety the Toom framing makes sharp: the maintained bit is *not* protected by the equilibrium dynamics of the governing law itself. Relaxational (Glauber/model-A) dynamics on the same 2D Ising free energy, run below T_c, fail to preserve a bit against *biased* noise—minority domains are not erased fast enough and the majority phase is slowly consumed [9]. Toom's rule succeeds precisely because it is non-equilibrium: it violates detailed balance and does not relax to the Gibbs state, which is what lets it erode aged droplets regardless of bias. This matters biologically because epigenetic copy error is directional—hemimethylation and 5mC loss on replication are not symmetric spin flips—so the noise a maintenance system faces is biased. The implication is concrete: a maintained epigenetic state cannot be a passively relaxing equilibrium well; it requires an actively

driven, detailed-balance-violating read–write loop. The core aging model (companion paper) is the equilibrium law; the *maintenance* that would hold a state against aging is a separate, non-equilibrium overlay.

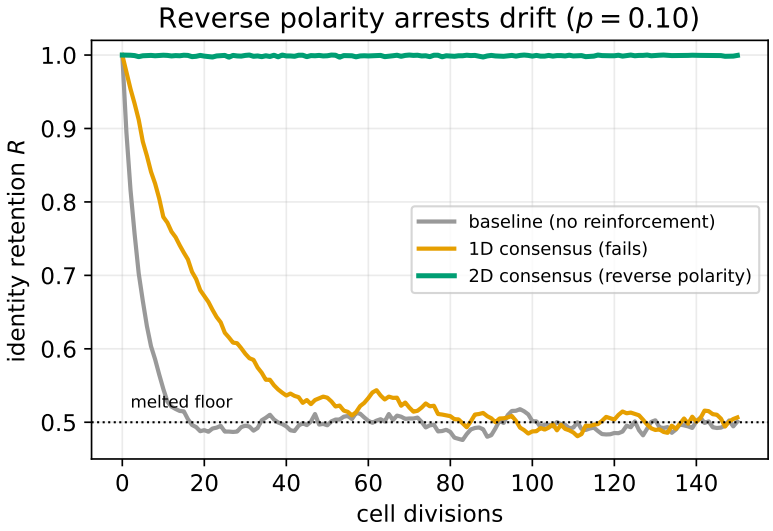

FIG. 2. Identity retention over divisions at $p = 0.10$ (seed 20260722). 2D neighbor consensus (reverse polarity) holds $R \approx 1$ indefinitely; baseline collapses to the melted floor within ∼10 divisions; 1D consensus only briefly slows decay and still relaxes to ≈ 0.5.

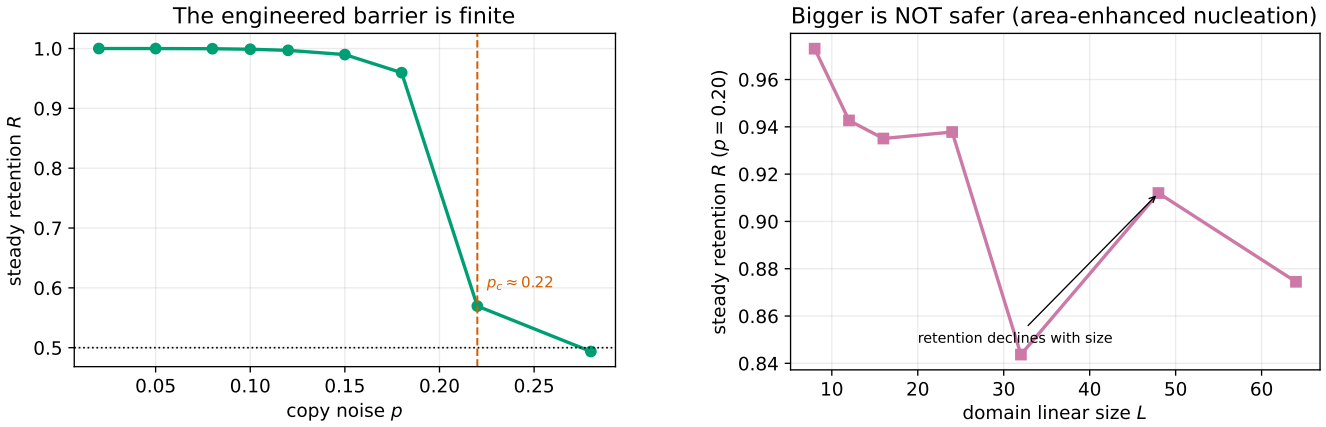

FIG. 3. Bounds on the intervention. *Left:* dose-response—steady retention holds near 1 up to a maintained-to-melted transition at $p_c \approx 0.22$. *Right:* the non-obvious negative—near criticality ($p = 0.20$) retention *declines* (noisily) with domain size, because larger domains offer more sites to nucleate an aged droplet (melting rate ∝ area). Bigger is not safer; single seed/lattice, to be swept.

V. INFORMATION VERSUS POWER: A HARD CEILING AND A KINETIC FLOOR

Ordered protection does not evade the information limits of the companion paper; it relocates them. There is a hard *information* ceiling [4]: no maintenance—collective or per-locus—can raise $I(\text{birth}; \text{state})$ without an external reference (the $\mathbb{Z}_2$ symmetry). Reverse polarity buys *retention* of the ∼1 collective bit, not *restoration* of lost per-locus identity. There is also a *kinetic* floor distinct from Landauer's thermodynamic one: holding the barrier requires a minimum reinforcement rate to outrun copy noise (the Eigen threshold [3] $r > p_{\text{raw}}(1 - p^*)/p^*$), and this floor is set

by rates, not by the $k_B T \ln 2$ erasure cost. The information wall (if $T(\rho) > J$, the ordered phase is unreachable at any metabolic power) is the hard boundary of what any intervention can achieve.

VI. BIOLOGICAL REALIZATION AND ITS FRONTIER

The model's J (collective read–write coupling) and T (effective copy noise) map onto real chromatin feedback. Polycomb (H3K27me3) and constitutive heterochromatin (H3K9me3) are read–write systems: a mark recruits the enzyme that writes the same mark on neighbors— exactly the neighbor-consensus coupling that produces J. In the companion paper's reader/paper framing these are the three places to act—fix the reader, protect the paper, or rewrite it from an outside exemplar—giving three distinct interventional levers, each with a different mechanism and risk:

1. *Lower T*—improve copy fidelity / reduce the effective per-division flip rate. Broad but weak; approaches the information wall asymptotically.

2. *Raise J*—strengthen read–write feedback at identity loci (the reverse-polarity lever). Locus-selective; the intervention of Sec. IV.

3. *Re-nucleate*—re-establish an ordered domain that has already melted, from a boundary or seed. This is the only lever that *restores* rather than *retains*, and—consistently with the companion paper—it requires a reference (a seed/boundary the domain did not itself contain), i.e. an h_{ext}-like input.

a. The frontier. Over-imposed order is pathological. A locus locked into a collective well cannot respond to legitimate developmental or environmental signals (rigidity), and inappropriately maintained domains are a route to the epigenetic dysregulation of oncogenesis. Ordered protection is therefore necessarily locus-selective: biology spends it on identity cores and cannot afford (or survive) genome-wide order. This bounds the therapeutic envelope: the levers must be targeted, reversible, and regulated, not global.

VII. DISCUSSION

a. What this is. A control-theoretic characterization of a maintained epigenetic state: its equilibrium (an ordered well), its stability (a finite collective barrier), its controllability (three levers), and its failure modes (dimensionality, critical noise, nucleation, rigidity). The reverse-polarity regime is real physics with clean, falsifiable structure—and, we now recognize, an application of Toom's fault-tolerant automaton to epigenetic maintenance rather than a new mechanism. The contribution is the mapping (identity maintenance = Toom-type reliable memory; aging = its sub-threshold failure) and the non-equilibrium requirement it exposes, not the automaton itself.

b. What this is not (restating Sec. I). It is not a claim that natural blood aging is this melting run backward. The companion paper shows measured bulk aging is composition mixing plus per-locus relaxation with no within-cell melting signature at composition-inert sites; the ordered phase here is the *same* order parameter M under the *same* law, in the $\mathcal{N} > 1$ regime that natural blood ($\mathcal{N} < 1$) does not occupy. The honest link is negative: *because* natural blood aging is in the disordered regime with no ordered phase to melt, a reverse-polarity intervention would be engineering an $\mathcal{N} > 1$ regime nature does not use here, not restoring one it lost.

c. The validating experiment. Synthesize an ordered chromatin domain (e.g. a targeted Polycomb read–write loop over a defined locus array), vary its size, and measure the age/division dependence of its retention. The model predicts (i) a 2D-connectivity requirement, (ii) a critical-noise cliff, and (iii) the counter-intuitive size negative—larger engineered domains melting faster near criticality. Point (iii) is the sharpest kill-shot: it is opposite to the naive design intuition, so a size sweep cleanly tests whether the nucleation picture (model) or the coherent-flip picture (naive) governs. Flagged as future wet-lab work; this paper stops at the theory and simulation that motivate it.

VIII. METHODS

a. Dynamics. Binary-symmetric copy channel (flip probability p) plus invariant-safe local consensus reinforcement (1D ring and 2D torus neighbor majority, radius 1, adoption strength 1.0), no stored reference.

b. Order parameter / retention. Aligned-domain start; $R(t) = \langle \mathbf{1}[\text{state}_t = \text{state}_0] \rangle$; steady R = mean of the last 40 of 150 divisions.

c. Free energy / barrier. $f(M) = -\frac{J}{2} M^2 - T\, s(M)$; barrier and order parameter from the mean-field module.

d. Code and reproducibility. All simulation code is in the palimpsest repository; the automated test suite (76 tests) passes. Run values use seed 20260722.

Appendix A: Assessment of claims

TABLE II. Claims and the strength of their support.

claim	evidence	strength
Ordered phase exists (2D), holds identity at near-zero cost	sim + mean-field + Toom eroder theorem	strong
No order for a simple local 1D rule	sim + Toom/Gács CA theory	strong
Engineered consensus arrests drift below p_c (Toom's rule)	intervention sim + Toom stability	strong
Size is not a free lever (nucleation $\propto$ area near criticality)	sim	moderate (single seed)
Maps to Polycomb/heterochromatin read–write feedback	mechanism analogy	speculative
Natural aging is reversible melting	—	not claimed

Appendix B: Open experiments

1. Regenerate all provisional figures and numbers from a frozen run.
2. Replace the order-of-magnitude biological n_c, b, γ estimates in Fig. 1 with measured values (the crossover law itself, Eq. (3), is exact).
3. Stress-test the size negative: vary seed geometry, radius, and strength to confirm the nucleation-$\propto$-area result is not an artifact of one lattice/seed configuration.
4. Frame every claim as theory + simulation; do not present it as a therapy result.

[1] N. Hangen, *Aging as irreversible information decay: a governing law, and the absence of a single-cell melting signature in human blood* (companion manuscript).

[2] J. von Neumann, *Probabilistic logics and the synthesis of reliable organisms from unreliable components*, in *Automata Studies* (1956); the one-bit reliability bound.

[3] M. Eigen, *Selforganization of matter and the evolution of biological macromolecules*, Naturwissenschaften **58**, 465 (1971); the quasispecies error threshold.

[4] C. E. Shannon, *A mathematical theory of communication*, Bell Syst. Tech. J. **27**, 379 (1948); mutual information and the channel framing the information ceiling rests on.

[5] R. Landauer, *Irreversibility and heat generation in the computing process*, IBM J. Res. Dev. **5**, 183 (1961); the $k_B T \ln 2$ cost of a logically irreversible bit operation.

[6] A. L. Toom, *Stable and attractive trajectories in multicomponent systems*, in *Multicomponent Random Systems*, Adv. Probab. Related Topics **6** (1980) 549–575; the NEC (north–east–centre) majority rule, the eroder property, and the stability theorem for reliable classical memory in 2D.

[7] P. Gács, *Reliable cellular automata with self-organization*, J. Stat. Phys. **103** (2001) 45–267; the positive-rates counterexample—a non-ergodic 1D probabilistic cellular automaton via hierarchical self-simulation.

[8] J. M. Swart, R. Szabó, C. Toninelli, *Peierls bounds from Toom contours*, arXiv:2306.13226 (2023); rigorous threshold bounds for Toom-type eroders.

[9] *Exploring the landscape of non-equilibrium memories with neural cellular automata*, arXiv:2508.15726 (2025); the 2D non-equilibrium memory landscape and the failure of below-T_c Glauber dynamics to store a bit against biased noise.