

Regulatory p -Adic Orderings from Stability in Gene Networks: Applications and Validation in Three Developmental Systems

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Abstract

Which genes in a Boolean gene regulatory network control the largest-scale features of the dynamics, and can their identity be read off from the transition table alone? We introduce a non-Archimedean ordering criterion for finite discrete dynamical systems: a coordinate ordering embeds the configuration space into the p -adic integers, so that p -adic balls encode nested regulatory scales. For each ordering we compute a weighted expansion score μ_E and a scale-by-scale $A/E/I$ classification of balls as contracting, expanding, or isometric; the orderings minimising μ_E define inferred regulatory hierarchies using no information beyond the transition table. Applied to three developmental Boolean networks, *A. thaliana* floral development ($N=13$), *C. elegans* cell cycle ($N=8$), and *D. melanogaster* gap-gene dynamics ($N=6$), the method aligns with the literature-supported master-regulator structure and yields certified optima in the systems where exhaustive or branch-and-bound verification is feasible. For *A. thaliana*, branch-and-bound certifies the global μ_E -minimum over all $13! \approx 6.2 \times 10^9$ orderings, and the resulting $A/E/I$ profiles separate canalised organ fates from transient inflorescence states, a distinction invisible to basin-size counts alone. The same construction extends to periodic attractors through iterates f^k . These examples support a testable principle, the chaos-controlled basin hypothesis: stable developmental architectures sit at or near minimisers of their own multiscale expansion

score. We read the resulting object as a p -adic analogue of the Waddington landscape rather than a recovery of it, with valleys realised as coarse-scale contracting balls and the stability profile of each fixed point carried by its $\mathbf{A/E/I}$ word. All definitions, algorithms, computations, biological comparisons, and reproducibility data used here are included in the present article and its supplementary material.

Keywords: p -adic dynamics, discrete dynamical systems, gene regulatory networks, multiscale analysis, basin analysis, master regulators, Boolean networks

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1 Introduction

Which components of a discrete dynamical system exert the greatest influence on its global behavior, and how can that influence be read off from the dynamics alone? In gene regulatory networks (GRNs), which govern cell differentiation, organ development, and phenotypic stability, this question has a sharp form: some genes act as master regulators whose state determines whole branches of cellular behavior, while others refine outcomes within an already committed fate. Topology alone (degree, centrality) does not separate the two roles, and classical descriptors that ignore the order of coordinates cannot rank them.

This article addresses the question *from the dynamics alone*. Building on the hierarchical p -adic framework developed in the companion paper [27], where the ball-level classification is defined through rational dynamics over $\mathbb{C}_p$ and a computability theorem shows that all stability invariants reduce to formulae evaluable from the transition table, we work entirely within $\mathbb{Z}_p$. Ultrametric and p -adic methods have a growing record in mathematical biology, where hierarchical organisation is intrinsic, going back to non-Archimedean models of dynamical and biological systems [23] and including a recent p -adic reaction–diffusion model of coral morphogenesis [13]. In each application, a published Boolean GRN and its synchronous update rules fix a discrete transition map $f: \mathcal{C} \rightarrow \mathcal{C}$. All subsequent steps (p -adic encoding, ball decomposition, and optimization of the stability score) operate on that map (equivalently, its full transition table) and use no external regulatory rankings or hierarchical role labels in the search for the minimizing ordering π^* (Remark 2.1). The configuration space is embedded into the p -adic integers $\mathbb{Z}_p$ under a gene ordering π . At each resolution level n , the space partitions into p -adic balls according to the first n genes in π . At every resolution level, f sends the configurations of each source ball into an image ball whose radius can be strictly smaller (*contracting*), equal (*isometric*), or strictly larger (*expanding*) than that of the source. The stability measure μ counts the expanding balls, weighted so that coarser scales contribute more. The ordering that *minimizes* μ defines the *inferred regulatory hierarchy* of the network, validated *a posteriori* against literature-based master-regulator rankings (Sections 3–5).

Why non-Archimedean.

The non-Archimedean choice is itself part of the applied contribution of this paper. The hierarchical p -adic framework constructed in the companion paper [27] is brought here directly to bear on biological networks. The hierarchy of scales lives in the p -adic metric on $\mathbb{Z}_p$ itself: once an ordering π is fixed, the digit decomposition of a configuration partitions the state space at every resolution, and the standard ultrametric multiplier of f on each ball labels it as contracting, expanding, or isometric. The score μ_E and the $A/E/I$ word are read off directly from these multipliers, intrinsic to the transition table. Two consequences run together throughout the paper: the analysis *reproduces* the literature-supported ordering of the master-regulator subsets in the three developmental networks studied here, and at the same time *reveals* a scale-resolved $A/E/I$ stratification that separates canalised cell fates from transient developmental states at every resolution, a finer invariant than basin-size statistics. We read the resulting object as a p -adic analogue of the Waddington landscape rather than a recovery of it. The basins are p -adic balls, the valleys are coarse-scale contractions, and the stability profile of a fixed point is a word in $\{A, E, I\}^{N-1}$.

Main results.

*The inferred regulatory hierarchy π^* recovers the established master-regulator rankings of three developmental Boolean GRNs from the transition map alone (Spearman $\rho = 1$ in the two larger systems; Sections 3–5). For *A. thaliana* ($N = 13$), the global μ_E -minimum is certified by branch-and-bound over all $13! \approx 6.2 \times 10^9$ orderings, with no enumeration (Section 2). The scale-resolved partition concentrates a large fraction of the state space in a few coarse basins containing all stable cell fates (Section 3), and extension to periodic attractors via iterated maps f^k cuts the optimal expansion score for the *Drosophila* gap-gene network (Section 5). Residual degeneracies in π^* are governed by a compensatory cancellation mechanism that preserves expansion counts at every scale without global commutation; we pose its general characterization as an open problem (Question 6.6).*

Contributions of this paper.

The present article is self-contained for all computational and applied aspects. The mathematical proofs (construction of interpreters, intrinsic invariance theorem) are in [27]. The specific contributions are:

1. *Three developmental GRN applications.* μ_E -minimizing orderings are computed and certified for *A. thaliana* ($N = 13$), *C. elegans* ($N = 8$), and *D. melanogaster* ($N = 6$), and validated against literature-based regulatory hierarchies.
2. *Scale-resolved $A/E/I$ classification.* Every fixed point of each network is assigned a word in $\{A, E, I\}^{N-1}$ recording its multiscale stability profile. This separates states that standard attractor analysis treats as equivalent. In *A. thaliana* it distinguishes the six floral-organ fates from the four inflorescence waypoints, a separation invisible to basin-size counts.

3. *Extension to periodic attractors.* The $A/E/I$ framework applies to limit cycles via iterated maps f^k . For the *Drosophila* gap-gene network, the optimal expansion score drops by 78% under f^2 .
4. *Two-criteria comparison and criterion divergence.* We characterise when μ_E -minimisation and μ_A -maximisation select the same ordering (*A. thaliana*, *Drosophila*) vs. incompatible orderings (*C. elegans*), and identify the compensatory mechanism responsible for degeneracies.
5. *Chaos-controlled basin conjecture.* The empirical coincidence of the certified minimisers with canonical master regulators is formulated as a testable conjecture (Conjecture 7.1): stable developmental architectures sit at or near $\arg \min_{\pi} \mu_E(\pi)$ of their own discrete dynamics.

Notation and algorithms are fixed in Section 2: the map f , the score decomposition (μ_E, μ_A, μ_I) with $\mu_E = \sum_{n=1}^{N-1} E(n) p^{N-n}$, the $A/E/I$ ball classification, and the μ_E -minimizing ordering π^* . Construction, invariance, stability properties, and connections to p -adic fixed-point theory are proved in the companion paper [27]. The present article remains self-contained for all computational and applied aspects.

This article applies the framework to three developmental networks chosen for the diversity of their underlying processes and the availability of validated Boolean models with supporting experimental literature: (1) *Arabidopsis thaliana* floral development ($N = 13$), with fate determination and organ identity [3, 12, 26]; (2) *Caenorhabditis elegans* cell cycle ($N = 8$), with CDK–cyclin and checkpoint logic [14, 19]; (3) *Drosophila melanogaster* gap genes ($N = 6$), with morphogen-driven spatial patterning [11, 20]. For each network we compute the μ_E -minimizing ordering π^* (our primary hierarchy), compare π^* with literature-based regulatory rankings, and interpret the scale-dependent classification of fixed points and basins. In parallel we analyze the contraction score μ_A and document where minimizing μ_E and maximizing μ_A select the same or different orderings, including degeneracies and trade-offs across the three systems. The gap-gene model for *Drosophila* admits both fixed-point and periodic attractors; for periodic regimes we apply the same ball decomposition to iterates f^k ($k = 2, 4$) so that $A/E/I$ classification extends beyond fixed points of f (Section 5). The optimization is explicit exhaustive search for *C. elegans* and *Drosophila*, branch-and-bound certification of the global optimum for *A. thaliana*, and a genetic-algorithm heuristic (validated here against the certified optimum and currently the most practical option for $N \gtrsim 13$) for larger systems.

How the framework is intended to be used.

The optimisation $\pi^* = \arg \min_{\pi} \mu_E(\pi)$ is one of several useful modes, and not always the principal one. In a typical biological application the master regulators of a network are already known or strongly suspected; the framework is then most informative as a *hypothesis-testing* and *structural-analysis* tool. Three modes coexist in this paper. (i) Given an ordering π chosen by the user (e.g. placing the proposed master regulators in the leading positions, or known terminal-effector genes at the end), the framework produces the scale-resolved $A/E/I$ word of every fixed point and the level decomposition of μ , identifying which fixed points lie in contracting, expanding, or scale-isometric balls, and at which resolutions. (ii) Given a *partial* constraint (e.g. “fix

UFO in position 0 and AP1 in position 7, leave the rest free”), the same branch-and-bound or genetic algorithm restricted to the consistent sub-permutations returns the conditionally optimal completion and its stability profile. (iii) Only when no biological prior is available, or when one wishes to test the hypothesis “the dynamics alone determines the hierarchy”, is the full unconstrained minimisation over S_N the right object. The three developmental networks studied here exercise mode (iii). Modes (i)–(ii) extend the practical reach of the method to networks where global certification over S_N is not affordable, without surrendering the scale-resolved $A/E/I$ output.

Contents.

Section 2 gives the operational definitions and algorithms. Sections 3–5 present the three developmental applications. Section 6 compares results across all three networks, including the two-criteria analysis μ_E vs. μ_A , Spearman validation, classification sequences, and the symmetry structure of the optimal set, a comparative analysis of the degeneracies in π^* across the three networks. Section 7 discusses validation and limitations, together with basin analysis and related perturbation checks (Supplementary Appendix S5) and polynomial interpolation (Supplementary Appendix S3). Section 8 summarizes the main findings.

2 Methods

All computations in this article depend only on the transition table f and the gene ordering π . The analytic foundation is developed in [27]: existence of a rational interpreter ϕ , hierarchical approximations F_n with controlled p -adic error ($|F_n(z) - \phi(z)|_p \leq 1/p^{M_{n,m}}$ on each ball, proved therein), and independence of the $A/E/I$ classification from the choice of interpolation. The operational definitions and computational procedures are collected here; proofs of invariance and approximation bounds are not reproduced. The analysis pipeline is:

$$\begin{array}{ccccccc} \text{GRN } (f) & \rightarrow & \text{Encoding } (\iota\pi) & \rightarrow & \text{Classification } (A/E/I) & \rightarrow & \mu\text{-optimization} \\ \text{discrete dyn.} & & p\text{-adic embed.} & & \text{ball-level dyn.} & & \text{optimal } \pi^* \end{array}$$

Each step depends only on the transition table f and the gene ordering π ; biological identities are used only for external validation.

Remark 2.1 (Scope of the biological input). *Biological information enters only through the choice of the GRN and its update rules; the transition table f is a biologically grounded object. The claim is not that the ranking arises without biological information altogether, but that, once f is fixed, the optimization of μ uses no external annotations, no prior hierarchical labels, and no known regulatory roles. The ordering is extracted from the mathematical structure of the pair $(f, \iota\pi)$ and only then compared with biological knowledge.*

Notation.

Let N denote the number of genes, p the number of states per gene ($p = 2$ for Boolean networks), and denote by $\mathcal{C} = \{0, \dots, p-1\}^N$ the configuration space ($|\mathcal{C}| = p^N$). A *gene ordering* is a permutation $\pi = (\alpha_0, \alpha_1, \dots, \alpha_{N-1})$ of $\{0, \dots, N-1\}$; we use 0-based indexing here, so that position 0 in π corresponds to the coarsest p -adic digit and position $N-1$ to the finest. The *p -adic encoding* $\iota_\pi : \mathcal{C} \rightarrow \{0, \dots, p^N-1\} \subset \mathbb{Z}_p$ assigns to each configuration $a = (x_0, \dots, x_{N-1})$ the integer $m_a = \sum_{k=0}^{N-1} x_{\alpha_k} p^k$, where the entry $x_i \in \{0, \dots, p-1\}$ is the state of gene i in the natural indexing of $\mathcal{C}$; the ordering π couples the two indexings by placing gene α_k at digit position k of the encoded integer. Through this encoding, each configuration is identified with the ball $B_{1/p^N}(m_a) \subset \mathbb{Z}_p$, whose elements share their first N p -adic digits with m_a : precisely the gene states of a read in the order π . The space $\mathbb{Z}_p$ partitions as $\mathbb{Z}_p = \bigsqcup_{a \in \mathcal{C}} B_{1/p^N}(m_a)$; biologically, each ball represents a single discrete gene configuration. The full geometric setting (affinoid domain in $\mathbb{C}_p$ and rational interpreter ϕ) is developed in [27].

p -Adic balls and resolution levels.

At resolution level $n \in \{1, \dots, N-1\}$, the space $\mathbb{Z}_p$ partitions into p^n balls $B_{1/p^n}(m)$ for $m = 0, \dots, p^n-1$. Configurations in the same ball share the first n gene states in the ordering π (see the Illustration below for a worked example with *Drosophila*). The boundary cases $n = 0$ and $n = N$ are excluded as uninformative: $n = 0$ gives the trivial partition $\mathbb{Z}_p$ as a single ball with no scale comparison available, while $n = N$ partitions $\mathbb{Z}_p$ into singletons on which every map is vacuously isometric.

Maximum common prefix length.

For each ball $B_{1/p^n}(m)$, define $M_{n,m}$ as the largest $k \in \{0, \dots, N\}$ such that $\iota_\pi(f(c)) \bmod p^k$ takes the same value for every c with $\iota_\pi(c) \in B_{1/p^n}(m)$:

$$M_{n,m} := \max\{k : \iota_\pi(f(c)) \bmod p^k \text{ is constant for all } c \in \iota_\pi^{-1}(B_{1/p^n}(m))\}. \quad (1)$$

Equivalently, $M_{n,m}$ is the number of leading p -adic digits on which all images agree. Denoting this common value by $m' := \iota_\pi(f(c)) \bmod p^{M_{n,m}}$ (for any c in the ball), the images therefore lie in a single ball $B_{1/p^{M_{n,m}}}(m')$, the *image ball* of $B_{1/p^n}(m)$.

Coarse multiplier and ball classification.

The *coarse multiplier* is $\Lambda_{n,m} := p^{n-M_{n,m}}$. Comparing the image ball $B_{1/p^{M_{n,m}}}(m')$ with the source ball $B_{1/p^n}(m)$:

- *Contracting* (A ; letter retained from [27]): $M_{n,m} > n$ ($\Lambda_{n,m} < 1$). The image ball has strictly smaller radius than the source; the dynamics compresses at this scale.
- *Expanding* (E): $M_{n,m} < n$ ($\Lambda_{n,m} > 1$). The image ball $B_{1/p^{M_{n,m}}}(m')$ has strictly larger radius than the source; the dynamics magnifies at this scale.
- *Isometric* (I): $M_{n,m} = n$ ($\Lambda_{n,m} = 1$). The image ball has the same radius.

Stability scores.

The level counts are $E(n) := \#\{m : M_{n,m} < n\}$, $A(n) := \#\{m : M_{n,m} > n\}$, $I(n) := \#\{m : M_{n,m} = n\}$. We have $E(n) + A(n) + I(n) = p^n$. The three stability scores are

$$\mu_E := \sum_{n=1}^{N-1} E(n) p^{N-n}, \quad \mu_A := \sum_{n=1}^{N-1} A(n) p^{N-n}, \quad \mu_I := \sum_{n=1}^{N-1} I(n) p^{N-n}, \quad (2)$$

with the control identity $\mu_E + \mu_A + \mu_I = (N-1)p^N$. The *stability measure* is $\mu := \mu_E$; smaller μ means fewer expanding balls (weighted by scale), so the dynamics preserves ball structure more faithfully across resolution levels. The ordering that minimizes μ is denoted π^* and is interpreted as the hierarchy under which the network is most stable in this sense.

Classification sequence.

For a fixed point a with $f(a) = a$, its *classification sequence* is the word in $\{A, E, I\}^{N-1}$ recording the ball classification at each level $n = 1, \dots, N-1$. For instance, IEAA means isometric at $n=1$, expanding at $n=2$, contracting at $n=3, 4$.

Remark 2.2 (Classification sequence vs. M -profile). *The classification word retains only the sign of $n - M_{n,m_a}$; the full M -profile $(M_{1,m_a}, \dots, M_{N-1,m_a})$ records the quantitative expansion or contraction at each scale ($\Lambda_{n,m_a} = p^{n-M_{n,m_a}}$). Two fixed points sharing the same $A/E/I$ word can therefore differ in how strongly they expand or contract at each level. The stability measure μ_E aggregates M -profile information across all balls (not only those containing fixed points); the classification word is a coarsening that suffices for the biological questions addressed here but can be refined when finer distinctions are needed. Per-network illustrations appear in Sections 3–5 and in the comparative analysis (Section 6).*

Terminology.

In this paper, the labels $A/E/I$ refer to the *scale-level* ball classification above. We use *contracting/expanding/isometric* for ball-level behavior and reserve *attracting/repelling* for classical pointwise dynamics. A “potential basin at scale n ” is a contracting n -ball containing at least one fixed point; this is a local notion at scale n , not a global basin of attraction. In biological interpretation sections, we may refer to “expanding basins” and “contracting basins” as shorthand for expanding and contracting balls containing fixed points.

Illustration.

Consider the *Drosophila* gap gene network ($N = 6$, $p = 2$, 64 configurations). At level $n = 1$, the two balls group configurations by the state of Bicoid (the first gene in the optimal ordering). Under the optimal ordering, $M_{1,0} = 1$ (isometric) and $M_{1,1} = 1$ (isometric). Thus $E(1) = 0$, $A(1) = 0$, $I(1) = 2$, contributing $0 \cdot 2^5 = 0$ to μ_E at this level. The full exhaustive search gives $\mu_E^* = 64$ with 4 optimal orderings.

Table 1: Summary of key notation.

Symbol	Meaning
N, p	Number of genes, states per gene ($p = 2$ for Boolean)
$\mathcal{C}$	Configuration space, $ \mathcal{C} = p^N$
π, π^*	Gene ordering (permutation), μ -minimizing ordering (certified)
ι_π	p -adic encoding under ordering π
$M_{n,m}$	Maximum common prefix length for ball $B_{1/p^n}(m)$
$\Lambda_{n,m}$	Coarse multiplier $= p^{n-M_{n,m}}$
$A(n), E(n), I(n)$	Counts of contracting, expanding, isometric balls at level n
$\mu = \mu_E$	Stability measure (weighted count of expanding balls)
π_E^*, π_A^*	μ_E -minimizer; μ_A -maximizer (defined in Section 6)

Hierarchical approximations.

The n -approximation $F_n : \mathbb{Z}_p \rightarrow \mathbb{Z}_p$ is a rational map that agrees with the embedded dynamics $\iota_\pi \circ f \circ \iota_\pi^{-1}$ on n -digit p -adic prefixes: it maps each ball $B_{1/p^n}(m)$ into a single ball, and the relationship between source and image radii yields $\Lambda_{n,m}$. The construction is by successive gluing: $F_0(z) = z$ (trivial), and each F_{n+1} extends F_n by resolving the dynamics at the next digit level while preserving agreement on previous digits. Existence of the maps F_n and non-uniqueness of the construction are proved in [27]; the bound $|F_n(z) - \phi(z)|_p \leq 1/p^{M_{n,m}}$ on each ball $B_{1/p^n}(m)$ is established there. Explicit formulas for *A. thaliana* are in Supplementary Appendix A (PDF); for *C. elegans* and *Drosophila*, explicit F_n are omitted here (see Supplementary scripts).

Stability measure computation.

The stability measure μ is computed directly from the discrete transition data using Eq. (2). For each resolution level $n = 1, \dots, N-1$, we compute $M_{n,m}$ for every ball $m = 0, \dots, p^n - 1$ by checking digit-by-digit agreement among images of all configurations in the ball. The following proposition makes the computational autonomy of the present analysis explicit.

Proposition 2.3 (Self-contained computability). *For every finite map $f : \mathbb{F}_p^N \rightarrow \mathbb{F}_p^N$ and every ordering π , the values $M_{n,m}$, $\Lambda_{n,m} = p^{n-M_{n,m}}$, the level counts $A(n), E(n), I(n)$, and the scores (μ_E, μ_A, μ_I) are determined by finitely many congruences of the form $\iota_\pi(f(c)) \bmod p^k$ evaluated on the p^N configurations. In particular, they do not depend on any interpolation of f to a rational map over $\mathbb{C}_p$.*

Proof Equation (1) defines $M_{n,m}$ as a maximum of integers k characterised by the congruence $\iota_\pi(f(c)) \bmod p^k$ taking a single value over $c \in \iota_\pi^{-1}(B_{1/p^n}(m))$. The congruence depends only on the finite values $f(c) \in \mathbb{F}_p^N$ and the ordering π . The remaining quantities $\Lambda_{n,m}$, $A(n)$, $E(n)$, $I(n)$, and the sums in Eq. (2) are arithmetic combinations of the $M_{n,m}$. Any rational interpreter ϕ compatible with the embedded finite dynamics on the p^N terminal configurations induces the same images of those configurations; therefore, for every coarser ball $B_{1/p^n}(m)$, the common-prefix length $M_{n,m}$ computed from those images is the same, and hence the same triple (μ_E, μ_A, μ_I) is obtained. $\square$

Thus all $A/E/I$ classifications and the measure μ used in this article are determined solely by the values $M_{n,m}$ (and hence $\Lambda_{n,m}$), which are computed from the transition table without constructing F_n .

Optimization of gene orderings.

Minimizing μ over $N!$ orderings is computationally hard. For small N (≤ 8), exhaustive search is feasible; for *A. thaliana* ($N=13$, $13! \approx 6.2 \times 10^9$), we can use branch-and-bound with admissible pruning bounds over the full search space.

Branch-and-bound lower bound (admissibility). Fix a prefix $\pi[0:k] = (i_0, \dots, i_{k-1})$ specifying the first k gene positions of a candidate ordering. The level counts $E(n)$ for $n = 1, \dots, k$ depend only on this prefix because the partition of $\mathbb{F}_p^N$ into balls at radius $1/p^n$ is determined by the first n coordinates, and $E(n)$ counts the balls at that level on which the dynamics expands; this follows from Proposition 2.3. The companion paper [27] gives the corresponding interpolation-theoretic formulation. Define

$$\text{LB}(\pi[0:k]) := \sum_{n=1}^k E(n; \pi[0:k]) p^{N-n}. \quad (3)$$

We have, for every completion π of the prefix, that the missing levels $n = k+1, \dots, N-1$ contribute non-negative terms $E(n) p^{N-n} \geq 0$, so

$$\text{LB}(\pi[0:k]) \leq \mu_E(\pi).$$

The bound is therefore *admissible*: it never overestimates the true μ_E of any completion. Whenever $\text{LB}(\pi[0:k])$ exceeds the current incumbent, the entire subtree of completions of $\pi[0:k]$ is pruned without losing the global minimum. For the μ_A -maximization variant, the corresponding upper bound is computed only at levels whose contraction status is already determined by the available prefix: since $A(n; \pi[0:k])$ counts balls with $M_{n,m} > n$, it requires knowing the $(n+1)$ -st digit, so the admissible upper bound for μ_A sums the contributions $A(n; \pi[0:k]) p^{N-n}$ over $n = 1, \dots, k-1$ (a prefix of length k fixes contractions up to level $k-1$); for $n \geq k$ a worst-case bound $p^n p^{N-n} = p^N$ replaces the unknown count. Traversal is depth-first with best-first ordering on LB; ties are broken lexicographically on the prefix to make the run deterministic.

The pruned search for μ_E -minimization completed with an aggregate cost of ≈ 29 CPU-hours (distributed across multiple cores of a single server; the figure quotes total CPU time, not wall-clock); it certifies the global minimum and identifies all optimal orderings without exhaustive enumeration. Initial candidate orderings were obtained by genetic algorithm heuristic and used to initialize the branch-and-bound incumbent; the same GA runs are used as an additional robustness check (see Supplementary Material for parameters and run outputs). Scripts and certification logs are in the Supplementary Material.

C. elegans model specification (Huang et al.).

For *C. elegans*, the synchronous Boolean map follows Huang et al. [19]: synaptic weights $w_{ij} \in \{-1, 0, 1\}$ from their Table 1, gene order for node indexing from Table 2, thresholds $\theta_i = 0$, tie-breaking at threshold so that $\sum_j w_{ij} S_j = \theta_i$ implies $S_i(t+1) = S_i(t)$, and self-inhibition edges $w_{ii} = -1$ on *cul-1*, *fzr-1*, and *cki-1* as in their Figure 1. The transition table used here is generated by the same rule; alternative tie-breaking or asynchronous updates would change f and hence π^* (not explored). Supplementary script `build_celegans_transitions_huang.py` and `data/README_celegans.md` reproduce the build.

Basin Analysis.

We interpret each p -adic ball at level n as a *potential basin at scale n* (in the sense of the Terminology above). For each ball at resolution level n , we:

1. Identify fixed points within the ball
2. Classify as contracting, expanding, or isometric at that scale based on the multipliers $\Lambda_{n,m}$ (equivalently, the F_n behaviour).
3. Compute basin size: the number of configurations that eventually map into the fixed points in that ball (graph-theoretic basin cardinality in the discrete Boolean state space), used as an external validation measure. This basin cardinality is distinct from the $\Lambda_{n,m}$ -based classification, which concerns the ultrametric geometry of n -balls in the embedded space.

Larger contracting basins correspond to more hierarchically stable states; expanding balls indicate sensitivity to perturbations.

Hierarchical graph.

For visualization, we construct a multilevel directed graph $G = (V, E)$ with $V = \bigsqcup_{n=0}^{n_{\max}} V_n$ (one vertex per ball at each resolution level), intra-level edges encoding ball-to-ball transitions induced by f , and inter-level edges encoding the refinement $B_{1/p^{n+1}}(m') \subset B_{1/p^n}(m)$. Vertices are colored by $A/E/I$ classification. The full construction and figures appear in Supplementary Appendix S4.

Polynomial Interpolation and Validation.

Each gene update function $T_i : \mathbb{F}_2^N \rightarrow \mathbb{F}_2$ admits a unique canonical polynomial over $\mathbb{F}_2$. We compute these by interpolating the transition table; the resulting polynomials reveal direct gene dependencies and regulatory logic complexity, complementing the hierarchical stability analysis.

Validation is external (Remark 2.1): π^* is compared with (i) experimentally identified master regulators (quantified via Spearman ρ when multiple optima exist), (ii) known biological states, (iii) literature-based stability or transience classifications, and (iv) known regulatory logic. Data and code are in the supplementary materials.

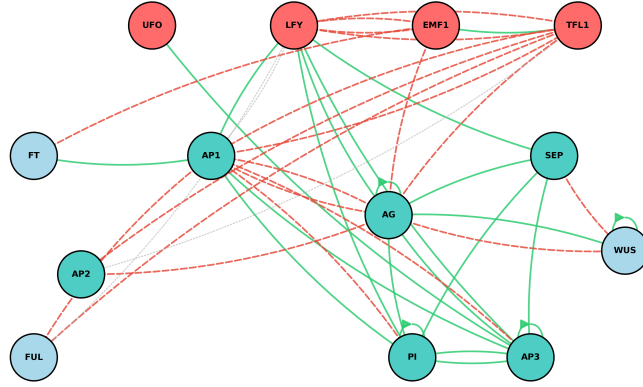


Figure 1: *A. thaliana* floral development network ($N = 13$ genes). Node colors: red = master regulators (UFO, LFY, EMF1, TFL1), dark teal = organ identity genes (AP1, AP2, AG, PI, AP3), light teal = other regulatory genes (FT, FUL, WUS, SEP). Edge styles: solid green arrows = activation, dashed red = repression, solid gray = regulation with context-dependent effect. Network topology and interaction types from [12, 26]; figure generated programmatically from Boolean rules (see Supplementary Material).

3 Application to *Arabidopsis thaliana*

The floral development network of *Arabidopsis thaliana* specifies how cells commit to organ identity (sepals, petals, stamens, carpels) and how inflorescence is maintained until commitment. We ask whether an ordering of genes by “regulatory influence” emerges from the Boolean transition table once the Mendoza–Álvarez-Buylla model is fixed (Remark 2.1), and whether the scale-dependent classification separates stable organ fates from transient inflorescence states.

Biological context.

The ABC model and the underlying Boolean GRN were established by Mendoza and Álvarez-Buylla [26], refined by Espinosa-Soto et al. [12], extended to the full flowering transition pathway in [9], and reviewed by Álvarez-Buylla et al. [3]. We use their model and compare the μ -minimizing ordering to the known master regulators (UFO, EMF1, LFY, TFL1, etc.).

The network (Figure 1) consists of $N = 13$ genes indexed as $x_0 = \text{FUL}$, $x_1 = \text{FT}$, $x_2 = \text{AP1}$, $x_3 = \text{EMF1}$, $x_4 = \text{LFY}$, $x_5 = \text{AP2}$, $x_6 = \text{WUS}$, $x_7 = \text{AG}$, $x_8 = \text{TFL1}$, $x_9 = \text{PI}$, $x_{10} = \text{SEP}$, $x_{11} = \text{AP3}$, $x_{12} = \text{UFO}$, each with two states (0 inactive, 1 active), giving $|\mathcal{C}| = 2^{13} = 8,192$ configurations. The transition rules defining $f : \mathcal{C} \rightarrow \mathcal{C}$ are in the Supplementary Material.

Ordering and hierarchical structure.

For *A. thaliana* ($N = 13$), branch-and-bound over the full set of $13! = 6,227,020,800$ orderings certifies the global minimum of μ_E (Section 2). Exactly four permutations attain it (Observation 3.1). Throughout, π^* denotes the canonical representative $\pi^* = (\text{UFO}, \text{EMF1}, \text{LFY}, \text{TFL1}, \text{AP2}, \text{FT}, \text{AG}, \text{AP1}, \text{SEP}, \text{AP3}, \text{PI}, \text{WUS}, \text{FUL})$; all polynomials, tables, and figures index variables in that order. The other three minimisers swap $\text{EMF1} \leftrightarrow \text{LFY}$ at positions 1–2 and/or $\text{AP2} \leftrightarrow \text{FT}$ at positions 4–5.

Under π^* , early positions ($\text{UFO}, \text{EMF1}, \text{LFY}, \text{TFL1}$) carry the largest influence on stability, while configurations differing only in late-position genes ($\text{FUL}, \text{WUS}, \text{PI}, \dots$) are p -adically close. The first four positions match the master regulators of floral fate [3, 12, 26], and EMF1 (flowering repressor) precedes LFY (master activator), in line with the vegetative-to-floral decision being a coarse-scale step. The ordering matches the biological hierarchy of Table 2 in all eight listed positions, with Spearman $\rho = 1.0$ (Section 6 for details). Remark 3.2 places the remaining five genes ($\text{SEP}, \text{AP3}, \text{PI}, \text{WUS}, \text{FUL}$) in functional tiers, and Remark 3.3 quantifies local sensitivity around π^* .

Stability scores.

We have, at the certified optimum, the three scores $(\mu_E, \mu_A, \mu_I) = (26,776, 53,880, 17,648)$, satisfying the control identity $\mu_E + \mu_A + \mu_I = 12 \cdot 2^{13} = 98,304$. For comparison, the gene ordering of Mendoza and Álvarez-Buylla [26] gives $\mu_E = 80,704$ ($\approx 3.0 \times$ larger), confirming that π^* achieves a substantial reduction.

Observation 3.1 (Certified global optimum and symmetry structure). *Branch-and-bound over all $13! = 6,227,020,800$ orderings certifies that $\mu_E = 26,776$ is the global minimum. Exactly four orderings achieve it; they share 9 fixed gene positions and differ only in two transpositions:*

1. Positions 1–2: $\text{EMF1} \leftrightarrow \text{LFY}$ (interchangeable),
2. Positions 4–5: $\text{AP2} \leftrightarrow \text{FT}$ (interchangeable),

yielding 4 orderings, all with $(\mu_E, \mu_A, \mu_I) = (26,776, 53,880, 17,648)$. The four orderings produce identical level counts $E(n), A(n), I(n)$ at every resolution level $n = 1, \dots, 12$, so the symmetry is exact.

Prior heuristic search (genetic algorithm, 51 independent runs) had recovered one of these four orderings in 60.8% of replicates, with the top four gene positions stable across 92% of runs (Files S1–S4, Supplementary Material).

Table 2 summarizes the comparison with experimental evidence [6, 31, 32, 36].

Remark 3.2 (Dynamics-based tier structure). *The ordering π^* partitions the 13 genes into seven functional tiers that align with the known temporal sequence of floral development [3, 12, 26]: UFO (master switch, F -box cofactor of LFY [30], position 0); $\text{EMF1}/\text{LFY}$ (vegetative $\leftrightarrow$ reproductive transition, positions 1–2); TFL1 (inflorescence meristem maintenance [37], position 3); $\text{AP2}/\text{FT}$ (A -class / florigen, positions 4–5); $\text{AG}, \text{AP1}, \text{SEP}$ ($C/A/E$ organ identity cascade, positions 6–8); $\text{AP3}, \text{PI}$ (B -class petal/stamen, positions 9–10); WUS, FUL (downstream effectors, positions 11–12).*

Table 2: Canonical ordering π^* vs. biological hierarchy for *A. thaliana*. Rows list the eight genes for which the reference hierarchy assigns a consensus rank, with Spearman $\rho = 1.0$ on those paired ranks. The remaining five genes (SEP, AP3, PI, WUS, FUL) are placed in tiers in Remark 3.2.

Pos.	Gene	Biological Role	Evidence
1	UFO	Organ identity modulator	Petal/stamen defects in mutants
2	EMF1	Flowering repressor	<i>emf1</i> mutants flower early
3	LFY	Master regulator, floral identity	<i>leafy</i> mutants fail to flower
4	TFL1	Inflorescence identity	<i>tfl1</i> : terminal flower
5	AP2	Organ identity, represses AG	Homeotic transformations
6	FT	Flowering promoter	<i>ft</i> mutants late flowering
7	AG	Stamen/carpel identity	<i>ag</i> : no reproductive organs
8	AP1	Floral meristem identity	Inflorescence-like structures

The two symmetry pairs (Observation 3.1) align with documented antagonisms: EMF1 (Polycomb repressor [24, 32]) and LFY (master activator [36]) encode the same binary vegetative/reproductive partition under mutual exclusion; AP2 (A-class repressor) and FT (florigen) are joint targets of the miR172 circuit [4]. In a search space of 6.2×10^9 permutations the only degeneracies are these two, and they coincide with documented functional pairings, although as Remark 6.5 of Section 6 shows, this is a property of μ_E over $\text{Opt}(f)$ rather than a graph automorphism of f .

Remark 3.3 (Local sensitivity and isolation of π^*). Apart from the two symmetry pairs (Observation 3.1), every adjacent-position swap in π^* strictly increases μ_E , with steepest gradient at the top of the hierarchy (Table 3). Among 2,000 uniformly random orderings the minimum observed μ_E was 39,472 (47% above μ_E^* , mean $87,361 \pm 9,268$, z -score of π^* : -6.5), so π^* occupies an isolated extremum in the ordering landscape.

Swap	Genes	$\Delta\mu_E$	Increase
pos 0–1	UFO $\leftrightarrow$ EMF1	+8,192	+30.6%
pos 2–3	LFY $\leftrightarrow$ TFL1	+4,096	+15.3%
pos 3–4	TFL1 $\leftrightarrow$ AP2	+6,144	+22.9%
pos 5–6	FT $\leftrightarrow$ AG	+1,024	+3.8%
pos 6–7	AG $\leftrightarrow$ AP1	+4,096	+15.3%
pos 7–8	AP1 $\leftrightarrow$ SEP	+1,472	+5.5%
pos 8–9	SEP $\leftrightarrow$ AP3	+1,584	+5.9%
pos 9–10	AP3 $\leftrightarrow$ PI	+1,256	+4.7%
pos 10–11	PI $\leftrightarrow$ WUS	+1,120	+4.2%
pos 11–12	WUS $\leftrightarrow$ FUL	+448	+1.7%

The gradient falls from +30.6% at the master-regulator boundary to +1.7% at the effector tail, mirroring the tier structure of Remark 3.2: the more coarsely a gene contributes to π^* , the more sensitive the score is to its displacement.

Hierarchical approximations and level counts.

The n -approximations $F_0, F_1, \dots, F_4$ are constructed following the gluing procedure of the companion paper [27]; explicit formulas for the rational maps F_n are in Supplementary Appendix S1. Table 4 gives the complete level-by-level decomposition of μ_E , showing how each resolution contributes to the certified $\mu_E^* = 26,776$ and where the $A/E/I$ structure concentrates.

Table 4: Classification of p -adic balls by resolution level for $A. thaliana$ under π^* , with the per-level contribution to $\mu_E = \sum_n E(n) \cdot 2^{13-n}$. $E(n)$ counts expanding balls ($\Lambda_{n,m} > 1$, contribute to μ_E), $A(n)$ contracting ($\Lambda_{n,m} < 1$), and $I(n)$ scale-isometric ($\Lambda_{n,m} = 1$).

n	Total balls	$E(n)$	$A(n)$	$I(n)$	Contribution to μ_E
1	2	0	0	2	0
2	4	4	0	0	8,192
3	8	4	4	0	4,096
4	16	4	12	0	2,048
5	32	8	24	0	2,048
6	64	16	40	8	2,048
7	128	48	70	10	3,072
8	256	46	210	0	1,472
9	512	88	297	127	1,408
10	1,024	243	781	0	1,944
11	2,048	112	496	1,440	448
12	4,096	0	4,096	0	0
Total					$\mu_E = 26,776$

The expansion contribution concentrates at coarse scales: level $n = 2$ alone contributes 8,192 to μ_E ($\approx 30.6\%$ of the total), as the weight 2^{N-n} places the largest penalty on UFO/EMF1-determined coarse instabilities. Contraction is total at the finest scale ($A(12) = 4,096$). The ball-level dynamics behind these counts is visualised in Figure 2, which renders the first five levels as a dendrogram: the contracting balls $B_{1/16}(4)$ and $B_{1/16}(5)$ (floral organ basins) refine progressively from level 3 to 4, the expansion at $B_{1/8}(6)$ and $B_{1/8}(7)$ vanishes, and the deep contracting subtree (organ basins) separates visually from the expanding path to the inflorescence ridges $B_{1/16}(10), (11)$. The $A/E/I$ labels are independent of the choice of rational interpreter [27].

Classification of fixed points.

Throughout this subsection, ball-prefixes are written LSB-first: digit 0 corresponds to position 0 in π^* (UFO), digit 1 to position 1 (EMF1), and so on (Section 2). The fixed points of this network correspond to the floral organ identities identified by Mendoza and Álvarez-Buylla [26] and validated by Espinosa-Soto et al. [12] and Pérez-Buendía et al. [28]. In our framework, a *fixed point* is a configuration a with $f(a) = a$; its classification as *contracting* or *expanding* depends on the ball classification at each level (Section 2), not merely on being a fixed point.

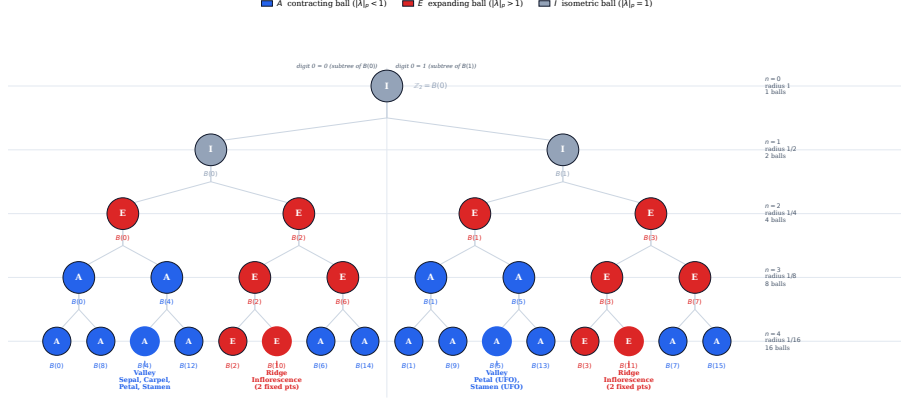


Figure 2: Hierarchical p -adic ball tree of *A. thaliana* under π^* ($p=2$, $N=13$, max level 4). Each node is one ball $B_{1/p^n}(m)$; its colour and inner letter are the $A/E/I$ classification computed from the discrete dynamics. The refinement follows the paper’s LSB-first convention (digit 0 is the coarsest): $B(0)$ at level 1 refines into $B(0)$ and $B(2)$ at level 2, while $B(1)$ refines into $B(1)$ and $B(3)$, and so on. Per-level counts: level 0: 1 I; level 1: 2 I; level 2: 4 E (all balls expand at this scale); level 3: 4 A + 4 E; level 4: 12 A + 4 E. Individual labels under the four occupied leaves identify the canalised organ fates ($B(4)$, $B(5)$) and the inflorescence ridges ($B(10)$, $B(11)$); these sit in opposite subtrees according to the state of the coarsest gene (UFO).

The certified optimal ordering yields exactly 10 fixed points of f , distributed across four balls at level $n=4$ and corresponding to the floral organ identities predicted by the ABC model [12, 26]. Configuration numbers are reported in the π^* -ordered address; Table 5 lists both m_A (under π^*) and the natural indexing.

Table 5: Fixed points in *A. thaliana* network under π^* .

Config (m_A)	Config (orig.)	Ball	Class	Type
10	264	$B_{1/16}(10)$	Expanding	Inflor. ridge
2,058	328	$B_{1/16}(10)$	Expanding	Inflor. ridge
436	1,078	$B_{1/16}(4)$	Contracting	Sepal
5,492	1,715	$B_{1/16}(4)$	Contracting	Carpel
1,972	3,638	$B_{1/16}(4)$	Contracting	Petal
6,004	3,763	$B_{1/16}(4)$	Contracting	Stamen
11	4,360	$B_{1/16}(11)$	Expanding	Inflor. ridge
2,059	4,424	$B_{1/16}(11)$	Expanding	Inflor. ridge
1,973	7,734	$B_{1/16}(5)$	Contracting	Petal (UFO)
6,005	7,859	$B_{1/16}(5)$	Contracting	Stamen (UFO)

Observe that the six floral organ fixed points lie in contracting balls $B_{1/16}(4)$ (prefix (0, 0, 1, 0): LFY=1, EMF1=0, TFL1=0) and $B_{1/16}(5)$ (prefix (1, 0, 1, 0): UFO=1,

LFY=1); the four inflorescence fixed points lie in expanding balls $B_{1/16}(10)$ and $B_{1/16}(11)$. (Stamen configurations have AP2=1 in the encoding, a property of the Boolean model [12, 26], not an assertion about canonical ABC biology.)

Floral organ stability and inflorescence transience.

The ball classification aligns with a known distinction: the six organ fixed points lie in contracting balls ($B_{1/16}(4)$ and $B_{1/16}(5)$, jointly attracting 95% of the state space), while the four inflorescence fixed points occupy expanding balls with small basins (5.1% total). The literature describes organs as stable attractors and inflorescence as a developmental waypoint [12, 18, 26, 35]; the same distinction is visible from the transition table through the A/E/I stratification without asserting new biology.

Remark 3.4 (The p -adic Waddington landscape). *Under π^* , the level-4 partition realises the Waddington epigenetic landscape [18, 35] as a discrete object on a finite state space: the two contracting balls $B_{1/16}(4)$ and $B_{1/16}(5)$ are the valleys (canalised organ fates), the two expanding balls $B_{1/16}(10)$ and $B_{1/16}(11)$ are the ridges (transient inflorescence waypoints), and the A/E/I signature at finer resolutions records how the landscape refines inside each ball (Figure 3). The basins of the synchronous Boolean map literally become a tree, intrinsic to (f, π^*) and requiring no continuous landscape function.*

In the Boolean model of [12, 26], the condition $(0, 1, 1, 1, x_4, \dots, x_{12})$ (UFO=0, EMF1=1, LFY=1, TFL1=1 in the first four positions of π^*) guarantees convergence to the Carpel fixed point regardless of the remaining genes (all 512 configurations in that ball flow to Carpel, verified computationally), so genes in early p -adic positions exert the strongest influence on long-term fate. Polynomial interpolation analysis (I_{poly}), hierarchical ball-diagram visualisations, and basin analysis under mutants and noise are in the Supplementary Material.

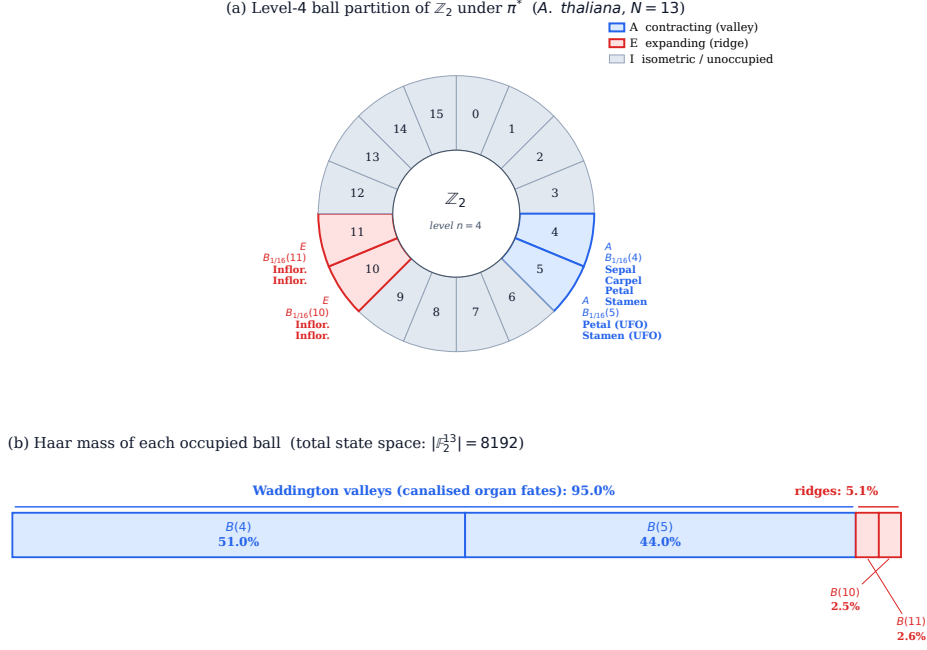


Figure 3: The p -adic Waddington landscape of the *A. thaliana* floral network ($N=13$, $p=2$) under the certified μ_E -minimising ordering π^* . (a) The 16 level-4 balls $B_{1/16}(m)$, $m = 0, \dots, 15$, drawn as nested sectors. Two contracting balls, $B_{1/16}(4)$ and $B_{1/16}(5)$, carry the six floral-organ fixed points (canalised valleys); two expanding balls, $B_{1/16}(10)$ and $B_{1/16}(11)$, carry the four inflorescence fixed points (transient ridges). The remaining twelve balls are unoccupied at level 4. (b) Basin coverage: the two valleys jointly hold 95% of the $2^{13} = 8192$ configurations; the two ridges hold 5.1%. Together panels (a) and (b) realise the p -adic Waddington landscape of Remark 3.4 as a quantitative partition of the state space.

4 Application to *Caenorhabditis elegans*

The cell cycle is driven by CDK–cyclin and CDK-inhibitor axes, with checkpoints (G1/S, G2/M) ensuring ordered transitions [14]. We use the 8-gene synchronous Boolean model of Huang et al. [19] (full specification below). With $N=8$, exhaustive search over all $8!$ orderings certifies the global optimum without heuristics. We compare π^* to the checkpoint organization below and ask a question absent in *A. thaliana*: do the μ_E -minimizing and μ_A -maximizing orderings agree, or do the two criteria impose incompatible hierarchies?

Biological context.

C. elegans is a major model organism for cell cycle and development, with invariant lineage and well-characterized regulators [7, 19].

We consider the Boolean model of Huang et al. [19] for early embryonic cell cycle regulation in *C. elegans*. Weights $w_{ij} \in \{-1, 0, 1\}$ follow Huang’s Table 1 with gene order Table 2 ($\theta_i = 0$ for all i), including self-inhibition edges $w_{ii} =$

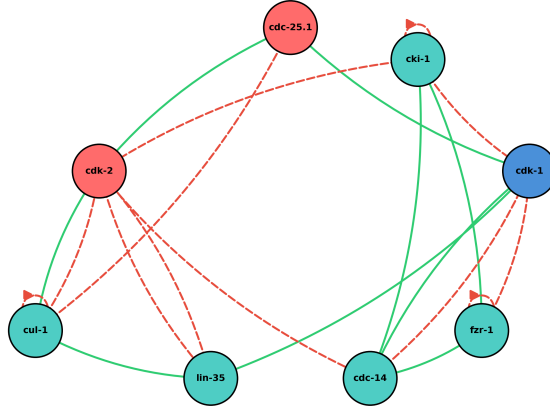


Figure 4: *C. elegans* cell cycle network ($N=8$). Red: G1/S regulators (cdc-25.1, cdk-2). Blue: M-phase regulator (cdk-1). Teal: other cell cycle genes. Green arrows: activation. Red dashed: repression. Network topology and Boolean rules from [19]; figure generated with NetworkX.

−1 on cul-1, fwr-1, and cki-1 as in Huang’s Figure 1. The transition table is generated from equation (1) of that article (synchronous update with threshold tie-breaking $\sum_j w_{ij}S_j = \theta_i \Rightarrow S_i(t+1) = S_i(t)$); Supplementary Material provides the script `build_celegans_transitions_huang.py` and the CSV file `transitions_celegans.csv` listing all 256 images of f . Different tie-breaking at threshold (e.g. flipping the tie rule or using asynchronous updates) can alter f and hence π^* ; we do not explore that sensitivity here.

The network (Figure 4) has $N=8$ genes, $|\mathcal{C}| = 2^8 = 256$ configurations, and 5 fixed points (configurations 0, 224, 226, 232, 234 in the Huang node indexing of Table 2), matching the five attractors reported by Huang et al.

Optimal ordering.

Exhaustive search over all $8! = 40,320$ orderings identifies a *unique* μ_E -minimizing ordering with $\mu_E^* = 956$. In gene-index notation (Huang Table 2: 0=cdk-2, ..., 7=cki-1), $\pi^* = [1, 0, 2, 3, 6, 5, 4, 7]$. Equivalently,

$$\begin{aligned} \pi^* : \quad & \text{cdc-25.1} \succ \text{cdk-2/cyclinE} \succ \text{cul-1/lin-23} \succ \text{lin-35/efl-1/dpl-1} \\ & \succ \text{cdc-14/fzy-1} \succ \text{fwr-1} \succ \text{cdk-1/cyclinB} \succ \text{cki-1}. \end{aligned} \quad (4)$$

We have, at the certified optimum, the stability scores $(\mu_E, \mu_A, \mu_I) = (956, 392, 444)$. The μ_A -maximizing orderings (four, all achieving $\mu_A^* = 440$; Section 6) are entirely disjoint from the unique μ_E -minimizer: for this network, minimizing expansion and maximizing contraction impose incompatible hierarchies. This is the sharpest criterion divergence among the three networks studied here.

Table 6 compares π^* with known regulatory roles. The two coarsest positions are *cdc-25.1* (G2/M phosphatase, CDK-1 activator) and *cdk-2/cyclinE* (G1/S CDK); *lin-35* (Rb homolog) appears at position 4.

Table 6: Comparison of optimal ordering π^* with biological importance for the *C. elegans* cell cycle network. Rows list positions 1–8 along π^* (coarsest to finest).

Pos.	Gene	Function	Evidence
1	<i>cdc-25.1</i>	G2/M checkpoint, CDK-1 activator	Mutants arrest in G2 [16, 19]
2	<i>cdk-2/cyclinE</i>	G1/S transition	Essential for S-phase entry [16]
3	<i>cul-1/lin-23</i>	Protein degradation (SCF)	Cell cycle progression [19]
4	<i>lin-35/efl-1/dpl-1</i>	Rb homolog, G1/S checkpoint	Checkpoint activation [7]
5	<i>cdc-14/fzy-1</i>	Mitotic exit phosphatase	Cytokinesis [16]
6	<i>fzr-1</i>	APC activator	Degrades mitotic cyclins [19]
7	<i>cdk-1/cyclinB</i>	M-phase regulator	Essential for mitosis [16]
8	<i>cki-1</i>	CDK inhibitor (CIP/KIP family)	Enforces cell cycle arrest [7, 17]

Hierarchical interpretation.

As in Section 3, the ordering determines the partition at each level: $n=1$ groups configurations by *cdc-25.1* state (G2/M checkpoint arm); $n=2$ adds *cdk-2/cyclinE* (G1/S CDK activity). The next levels incorporate degradation (*cul-1*) and the Rb pathway (*lin-35*), then mitotic exit and APC (*cdc-14*, *fzr-1*), and finally the M-phase CDK and global inhibitor (*cdk-1*, *cki-1*). Thus the μ_E -optimal hierarchy emphasizes the G2/M and G1/S CDK axes at the coarsest scales, with *lin-35* entering at an intermediate resolution, consistent with a dynamics-based reading in which phosphatase/CDK gating is resolved before the Rb branch.

Level structure.

The n -approximations $F_0, F_1, \dots, F_7$ are constructed following the gluing procedure of [27]. Explicit ball-map formulas under the Huang-aligned transition table and π^* are generated by the Supplementary script `compute_Fn_celegans.py`. The profiles $E(n), A(n), I(n)$ for $n = 1, \dots, 7$ under π^* are:

n	1	2	3	4	5	6	7
$E(n)$	1	3	4	4	20	40	62
$A(n)$	0	1	2	0	12	9	66
$I(n)$	1	0	2	12	0	15	0

At coarse scales ($n \leq 4$), isometric and expanding contributions are substantial; at the finest scale ($n = 7$), contracting balls dominate ($A(7) = 66$ vs $E(7) = 62$). The balance between E , A , and I across levels reflects how the Huang dynamics partitions state space under π^* .

Full fixed-point data, classification sequences, stability profiles, basin sizes, and the polynomial interpolation analysis (update polynomials and influence ranking) for *C. elegans* are in Supplementary Tables S3–S4 and in Supplementary Appendices S2–S3.

Predictions from the ordering.

Under π^* , *cdc-25.1* is at the coarsest scale and *cki-1* at the finest, with *cdc-14* and *fzr-1* at intermediate positions. Within this Boolean model, genes placed earlier in π^* are associated with coarser ball partitions and therefore with stronger effects on basin-scale summaries of the dynamics than genes at fine scales; RNAi or conditional alleles can probe whether phenotypic breadth tracks this tier structure empirically [7, 14].

Here μ_E and μ_A are not optimized by the same ordering, so one should not merge “minimal coarse expansion” and “maximal coarse contraction” into a single narrative without stating which objective is primary; (π_E^*, π_A^*) are complementary views of the same map f .

5 Application to *Drosophila melanogaster*

Spatial patterning along the anterior–posterior axis of *Drosophila* is established by maternal morphogens (Bicoid, Caudal) and refined by gap genes (Hunchback, Krüppel, Knirps, Giant) [11, 20]. The expected biological hierarchy (maternal morphogens before gap genes) provides a stringent benchmark for the μ -minimizing ordering.

Biological context.

The Boolean model we use is derived from the gap-gene regulatory logic in [11, 20], in line with discrete Boolean network analyses of *Drosophila* patterning [1]. With $N = 6$, exhaustive search over $6! = 720$ orderings lists all μ -minima; their agreement with the morphogen–before–gap hierarchy is recorded in Section 5.

Figure 5 shows the six-gene regulatory graph and the gene labels used below to specify the synchronous Boolean map f .

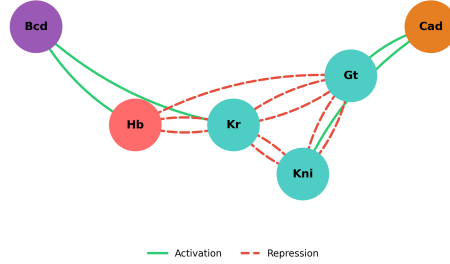


Figure 5: *D. melanogaster* gap gene network (6 genes). Purple/Orange: morphogens. Red: Hunchback. Teal: gap genes. Green arrows: activation. Red dashed: repression. Network topology from [20]; Boolean rules from [11]; discrete Boolean conventions follow [1]; figure generated with NetworkX.

The network (Figure 5) has $N=6$ genes, $|\mathcal{C}| = 2^6 = 64$ configurations. The Boolean update rules, based on [11, 20], are collected in Table 7. Bicoid and Caudal are maternal morphogens treated as self-maintaining ($T_{\text{Bcd}} = x_{\text{Bcd}}$, $T_{\text{Cad}} = x_{\text{Cad}}$); in reality they form spatial gradients.

Table 7: Boolean update rules for the *D. melanogaster* gap gene network.

Gene	Boolean rule	Biological logic
Bcd	$T_{\text{Bcd}} = x_{\text{Bcd}}$	Self-maintaining (maternal, anterior)
Cad	$T_{\text{Cad}} = x_{\text{Cad}}$	Self-maintaining (maternal, posterior)
Hb	$T_{\text{Hb}} = \text{Bcd} \wedge \neg \text{Kr} \wedge \neg \text{Gt}$	Activated by Bcd; repressed by Kr, Gt
Kr	$T_{\text{Kr}} = (\text{Bcd} \wedge \text{Hb}) \wedge \neg \text{Kni} \wedge \neg \text{Gt}$	Requires Bcd $\wedge$ Hb; repressed by Kni, Gt
Kni	$T_{\text{Kni}} = \text{Cad} \wedge \neg \text{Kr} \wedge \neg \text{Gt}$	Activated by Cad; repressed by Kr, Gt
Gt	$T_{\text{Gt}} = \text{Cad} \wedge \neg \text{Kr} \wedge \neg \text{Kni}$	Activated by Cad; repressed by Kr, Kni

The complete transition table is in the Supplementary Material. The dynamics admit both fixed-point attractors and limit cycles; Section 6 restricts the classification-sequence comparison to fixed points.

Optimal ordering.

For the *Drosophila* gap gene network with 6 vertices, exhaustive search over all $6! = 720$ possible orderings identifies the *provably optimal* (minimum $\mu_E = 64$) orderings; there are exactly 4, obtained by independently permuting two pairs that are exchangeable among μ_E -optima (Section 6): $\{\text{Bcd}, \text{Cad}\}$ at positions 0–1 and $\{\text{Kni}, \text{Gt}\}$ at positions 2–3 (see also Supplementary Material). *Throughout this paper*, $(x_0, \dots, x_5)$ refers to the *one* ordering written explicitly below; Supplementary Appendix S3 and the polynomial figures use these labels only (the other three minimizers relabel x_0 – x_3 by those swaps, leaving μ unchanged):

$$\begin{aligned} & (x_0, x_1, x_2, x_3, x_4, x_5) \\ & \parallel \\ & (\text{Bicoid}, \text{Caudal}, \text{Knirps}, \text{Giant}, \text{Krüppel}, \text{Hunchback}). \end{aligned} \tag{5}$$

We have, at the optimal ordering, the scores $(\mu_E, \mu_A, \mu_I) = (64, 156, 100)$, satisfying the control identity $\mu_E + \mu_A + \mu_I = 5 \cdot 2^6 = 320$.

These four orderings also maximize μ_A (Section 6). Four additional orderings achieve μ_A^* without minimizing μ_E : they place Caudal alone at position 0, the Kni/Gt pair at positions 1–2, and Bicoid at position 3, breaking the Bcd/Cad symmetry while preserving contraction. Thus $\text{Opt}_{\mu_E}(f) \subsetneq \text{Opt}_{\mu_A}(f)$: minimizing expansion and maximizing contraction select the same hierarchy plus a strictly larger set, a feature that sets *Drosophila* apart from the other two networks.

Both maternal morphogens occupy the first two positions, as expected from their role in establishing the anterior–posterior axis before gap genes respond. Among the gap genes, the posterior pair (Knirps, Giant), each activated solely by Caudal and repressed by Kr and the other posterior gene, precede the anterior pair (Krüppel,

Hunchback); Krüppel requires *both* Bicoid and Hunchback for activation, placing it among the most downstream. Table 8 compares π^* with known biological roles [11, 20].

Table 8: Comparison of optimal ordering with biological hierarchy for the *Drosophila* gap gene network. Positions 1–6 in the table correspond to indices 0–5 in the ordering $(x_0, \dots, x_5)$.

Pos.	Gene	Role	Evidence
1	Bicoid	Maternal morphogen, anterior	Mutants lack anterior
2	Caudal	Maternal morphogen, posterior	Posterior gradient
3	Knirps	Gap gene, posterior	Activated by Caudal, simple rule
4	Giant	Gap gene, posterior	Activated by Caudal, mutual repression
5	Krüppel	Gap gene, central domain	Requires Bcd $\wedge$ Hb, most downstream
6	Hunchback	Gap gene, anterior	Participates in Hb–Kr oscillation

Hierarchical interpretation.

At $n=1$, configurations are partitioned by Bicoid state; at $n=2$, by the Bicoid–Caudal pair. Placing both morphogens in leading positions minimizes μ , reflecting the developmental hierarchy: spatial coordinates must be established before gap genes can interpret positional information. The posterior gap genes (Kni, Gt) at positions 2–3 have simple activation rules (single morphogen input), whereas the anterior pair (Kr, Hb) at positions 4–5 participate in a 4-cycle oscillation (Bcd $\rightarrow$ Bcd+Hb $\rightarrow$ Bcd+Hb+Kr $\rightarrow$ Bcd+Kr $\rightarrow$ Bcd); confining this dynamically unstable pair to the fine-scale end of the hierarchy minimizes their contribution to μ_E . Section 5 analyzes the same orbit as a periodic trajectory of f (equivalently as fixed points of f^4), reports the $A/E/I$ words for the four-cycle states, and gives the μ_E -optimal reorderings under f^2 and f^4 , including Krüppel advancing ahead of Knirps at coarse scales once the Hb–Kr oscillation is absorbed under f^2 .

Level-by-level structure and gap-gene domains.

The Bicoid-defined balls at $n = 1$ are isometric, the Caudal digit at $n = 2$ produces two contracting (A) and two isometric (I) balls (Cad-inactive balls collapse because the posterior gap genes cannot activate), and the $n = 3$ refinement gives $E(3) = 4$, $A(3) = 4$, $I(3) = 0$ because the Cad-active balls develop expansion driven by Kni–Gt competition. Contracting balls correspond to stable gap-gene expression domains along the anterior–posterior axis, while expanding balls mark boundary regions where conflicting regulatory signals overlap. At coarse scales ($n \leq 2$), morphogen identity alone resolves domain membership and all balls are non-expanding. Expansion emerges at $n=3$ once the Kni–Gt regulatory conflict enters the partition. Fixed-point data, classification sequences, basin coverage, stability profiles, limit cycles, and polynomial interpolation analysis are in Supplementary Tables S5–S6 and Supplementary Appendices S2–S3.

Exchangeability of Knirps and Giant.

Among the four μ_E -optimal orderings, Knirps and Giant occupy positions 2 and 3 as an *exchangeable block*: two orderings place Kni before Gt, and the other two reverse this pair (Section 6). Both are posterior gap genes activated solely by Caudal and repressed by Krüppel and the other posterior gene, so the update rules of Kni and Gt are mutually symmetric. The network as a whole, however, is not equivariant under the swap (Kni, Gt): T_{Hb} depends on Gt but not on Kni, breaking equivariance on 8 of the 64 configurations. The exchangeability is therefore a property of the functional μ_E over $\text{Opt}(f)$, not a graph automorphism of the network. In embryos, *kni* and *gt* phenotypes are related but not identical [20], consistent with this distinction at the regulatory-logic level.

Extension to periodic orbits via f^k .

The $A/E/I$ classification applies naturally to limit cycles by treating k -periodic orbits as fixed points of f^k . The iterated maps f^2 and f^4 are computed from the transition table (Supplementary script `compute_fk_drosophila.py`); the same exhaustive search and ball decomposition apply without modification.

The map f^2 fixes the two 2-cycles (raising the count from 5 to 9 fixed points); f^4 additionally absorbs the 4-cycle (13 fixed points). Classifying the newly fixed configurations under $\pi^*(f)$ reveals two distinct patterns among the 2-cycle states: configs 32 and 33 (only morphogens active, all gap genes off) yield the word **IIIEEA** (the same as the four gap-domain fixed points), while configs 56 and 59 (both posterior gap genes Kni and Gt active) yield **IIIAA**, fully non-expanding. The 4-cycle states ($\{1, 3, 7, 5\}$, the Hb–Kr anterior oscillation) all share **IAAII**: contracting at levels 2–3 where morphogen identity determines basin membership, isometric at levels 4–5 where the Hb–Kr alternation preserves scale. The posterior 2-cycles, driven by Kni↔Gt competition, thus inherit the gap-domain classification or resolve it entirely; the anterior 4-cycle, driven by Hb–Kr mutual activation, contracts at the morphogen scale and stays isometric at the gap-gene scale. The Hb–Kr oscillation is confined to fine-scale dynamics while the morphogen partition remains stable.

For both iterates, exhaustive search over f^2 and f^4 yields $\mu_E^* = 14$ (a 78% reduction from $\mu_E^*(f) = 64$); in each case there are only 2 optimal orderings (the Bcd↔Cad swap), with $(\mu_E, \mu_A, \mu_I) = (14, 182, 124)$ (control: $14 + 182 + 124 = 320$). The optimal ordering for f^2 is (Bcd, Cad, Kr, Gt, Kni, Hb): morphogens still lead, but Krüppel advances from position 4 to 2 while Knirps drops to position 4. Under f^2 , the Hb–Kr oscillation is absorbed and Hb–Kr states stabilize; at that point, Krüppel’s broader regulatory reach ($I_{\text{poly}} = 3$, vs $I_{\text{poly}} = 2$ for Knirps) makes it more informative than Knirps at coarse scales. The Kni↔Gt exchangeability, present in $\pi^*(f)$, breaks under iteration: only Bcd↔Cad persists. Level counts under $\pi^*(f^2)$ show near-complete contraction: at level 5, 29 of 32 balls are contracting, 2 isometric, and only 1 expanding. The μ_E/μ_A coincidence persists under iteration: the 2 orderings that minimize $\mu_E(f^2)$ are exactly the 2 that maximize $\mu_A(f^2) = 182$, so for *Drosophila* a single hierarchy captures both stability criteria at every iterate. The set of optimal orderings for f^4 is identical to that for f^2 (same two permutations, same $\mu_E^* = 14$), confirming that the 2-cycles, not the 4-cycle, drive the reoptimization.

6 Comparative Analysis

We now synthesize patterns across the three models (Sections 3–5). In each case, μ -minimizing orderings and scale-dependent $A/E/I$ classifications align with documented developmental roles while refining flat Boolean attractor analysis. Exhaustive search certifies optima for *C. elegans* and *Drosophila*; branch-and-bound certifies global μ_E - and μ_A -optima for *A. thaliana*. In what follows, we distinguish claims supported by the experimental literature from interpretations internal to the p -adic construction.

Remark 6.1 (Four complementary views of structure). *We compare four non-equivalent readings of the same Boolean model: topology (graph centralities), polynomial influence I_{poly} , Boolean sensitivity, and the multiscale stability score μ built from the counts $M_{n,m}$ (Section 2). In short: topology records wiring, I_{poly} records direct syntactic reach in the update rules, Boolean sensitivity records one-step perturbation effects, whereas μ records how strongly the dynamics preserves or breaks hierarchical partitions of the state space across scales.*

Master regulators occupy leading positions of π^* in every network. The comparison organizes itself around three recurring patterns:

1. *Regulatory hierarchy*: UFO, EMF1, LFY, TFL1 head the ordering in *A. thaliana* [12, 26]; cdc-25.1, cdk-2, lin-35 in *C. elegans* [7, 19]; Bicoid, Caudal in *Drosophila* [11, 20], in line with their disproportionate influence on dynamics.
2. *Stability classification*: differentiated/stable states lie in contracting balls; transitional states lie in expanding balls (Figure 6). This matches the view of stable fates as attractors and developmental intermediates as transient in Boolean and landscape models [12, 18, 26, 33, 35].
3. *Hierarchical robustness*: configurations differing only in downstream genes (late positions) tend to converge to the same fixed point; differences in master regulators (early positions) lead to different outcomes [19, 20, 26].

Traditional methods identify master regulators via structural metrics (degree centrality, betweenness, K-core, minimum dominating set) or prior knowledge [15, 34]. The stability measure μ is distinct: it incorporates discrete dynamics, not just topology, in line with design principles of canalization and redundancy in biological GRNs [21]. Table 9 summarizes the reported orderings and stability measures for the three networks.

Quantitative comparison with network centrality.

Supplementary Table S7 compares the position of each gene in π^* for *A. thaliana* with standard centrality metrics on the regulatory digraph (out-degree, in-degree, betweenness, closeness). The Spearman correlation between π^* and each metric is weak: $\rho = 0.18, 0.12, 0.30$ for out-degree, betweenness, closeness respectively, with $|\rho| \leq 0.36$ in all cases. UFO is illustrative: out-degree 1 and betweenness 0, yet position 0 in π^* . Its self-maintaining rule $T_{\text{UFO}} = x_{\text{UFO}}$ partitions the state space into two dynamically invariant halves, and the level $n = 1$ weight $p^{N-1} = 4096$ makes this partition dominant in μ . LFY has the highest out-degree (7) but sits at position 2: direct regulatory

reach does not imply coarse-scale stability control. The stability measure μ captures a dynamics-based hierarchy distinct from network topology.

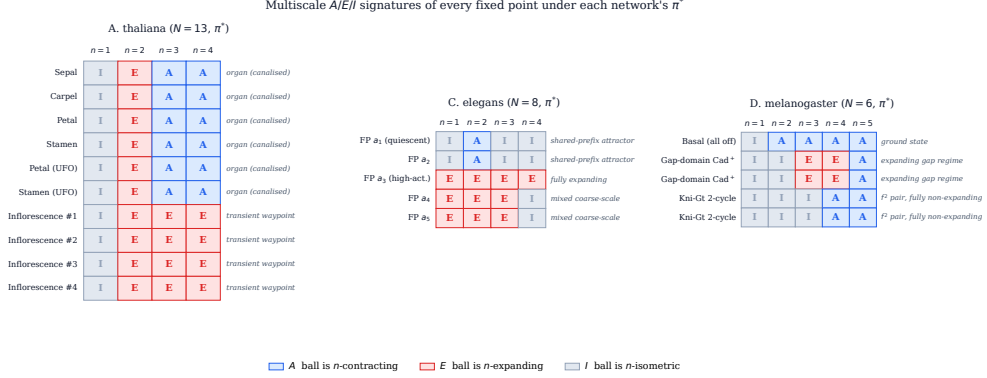


Figure 6: Multiscale $A/E/I$ signatures of every fixed point in the three developmental networks, under each network's certified μ_E -minimising ordering π^* . Rows are fixed points; columns are resolution levels $n = 1, \dots, N - 1$; each cell is the ball-level label (A contracting, blue; E expanding, red; I isometric, grey) computed from the multipliers $\Lambda_{n,m}$ of Section 2. The framework discriminates fixed points that flat Boolean attractor analysis conflates: in *A. thaliana*, organ identities share the canalised tail **IEAA** while inflorescence fixed points share the unstable tail **IEEE**; in *C. elegans* the quiescent attractors carry **IAII** against the fully expanding **EEEE** of the high-activity attractor; in *D. melanogaster*, the gap-domain fixed points (**IIIEEA**) are visibly different from the Kni-Gt f^2 -cycle pair (**IIIAA**) and from the basal ground state (**IAAAA**). Annotations on the right name the biological reading of each signature class.

Table 9: Reported gene orderings and stability measures for the three analyzed networks.

Network	Ordering	μ_E	Verification
<i>A. thaliana</i>	UFO, EMF1, LFY, TFL1, AP2, FT, AG, AP1, SEP, AP3, PI, WUS, FUL	26,776	BnB-certified [†] (4 optimal)
<i>C. elegans</i>	cdc-25.1, cdk-2, cul-1, lin-35, cdc-14, fzf-1, cdk-1, cki-1	956	Exhaustive (unique μ_E -opt.)
<i>D. melanogaster</i>	Bicoid, Caudal, Knirps, Giant, Krüppel, Hunchback	64	Exhaustive (4 optimal)

[†]Global optimality certified by branch-and-bound (Section 2); orbit structure of the four optima in Observation 3.1.

Two-criteria analysis: minimizing μ_E vs. maximizing μ_A .

Do the same orderings minimize μ_E and maximize μ_A , or do the two objectives conflict? Writing π_E^* for a μ_E -minimizer and π_A^* for a μ_A -maximizer (unqualified π^* denotes π_E^* throughout), we compute (μ_E, μ_A, μ_I) for all orderings (exhaustive search in the small

networks, branch-and-bound certification for *A. thaliana*, see Supplementary Tables S1–S2); we have the result summarised in Table 10.

Table 10: Two-criteria analysis: orderings minimizing μ_E vs. maximizing μ_A . “Intersection” = number of orderings achieving both.

Network	μ_E -opt.	μ_A -opt.	Intersect.	Interpretation
<i>C. elegans</i>	1	4	0	Trade-off (disjoint μ_E - and μ_A -optima)
<i>D. melanogaster</i>	4	8	4	$\text{Opt}_{\mu_E} \subseteq \text{Opt}_{\mu_A}$ (proper: 4 of 8)
<i>A. thaliana</i>	4 (cert.)	40 (10 orbits $\times C_2 \times C_2$)	0	Mild trade-off (max. $\rho = 0.978$ vs. π_E^*)

The three regimes are qualitatively distinct. *D. melanogaster* exhibits $\text{Opt}_{\mu_E}(f) \subsetneq \text{Opt}_{\mu_A}(f)$ (the four μ_E -optima are a proper subset of the eight μ_A -optima). *C. elegans* shows outright conflict: the unique μ_E -minimum is disjoint from the four μ_A -maxima. In *A. thaliana*, branch-and-bound certifies 40 μ_A -maximizing orderings organised in 10 orbits under the same $C_2 \times C_2$ symmetry as the four μ_E -optima (Supplementary Table S2), none of which coincides with a μ_E -optimum. The closest μ_A -orbit reaches $\rho(\pi_E^*, \pi_A^*) = 0.978$, so the trade-off is mild.

Spearman rank correlation: validation and robustness.

The Spearman correlation ρ between the μ_E -induced ordering and the literature-based hierarchy serves both validation (consistency of the dynamics-based ranking with experimentally grounded regulatory importance) and robustness (stability of validation across μ_E -equivalent optima). For *A. thaliana*, π^* matches the eight-position biological hierarchy exactly ($\rho = 1.0$, exact permutation p -value 2.48×10^{-5} on $8!$ permutations). For *C. elegans*, the unique μ_E -optimum agrees on all eight genes with the literature table ($\rho = 1.0$, same p). For *D. melanogaster*, the four μ_E -optima give $\rho \in \{1, 33/35, 31/35\} \approx \{1, 0.943, 0.886\}$ with exact-permutation upper-tail probabilities $1/720, 6/720, 12/720$ respectively. Computing ρ between μ_E - and μ_A -optimal orderings (instead of versus biology) for *A. thaliana* yields $\rho(\pi_E^*, \pi_A^*)$ from 0.72 to 0.978 across the 10 μ_A -orbits; the closest retains UFO at position 0, the farthest displaces it to position 9.

Basin and polynomial comparison.

Across the three networks, dominant fixed-point attractors reside in contracting balls and capture the majority of the state space: 95.0% in two contracting balls for *A. thaliana* (Supplementary Appendix S5), 85.55% in a single basin for *C. elegans*, and contracting basins for all stable gap-gene domains in *Drosophila*. The polynomial influence ranking I_{poly} (Supplementary Appendix S3) agrees with π^* on top-ranked genes and diverges at intermediate positions, where the multiscale weighting of μ adds information beyond syntactic dependence in the update rules [19, 20, 26].

Scale-dependent classification.

The p -adic framework exposes qualitative differences between fixed points through their *classification sequences* across resolution levels. For each fixed point, we compute whether it lies in a contracting (A), expanding (E), or scale-isometric (I) ball at each level $n = 1, \dots, N-1$. In Table 11 and Figure 7 we show the first four characters of the classification sequence (levels $n = 1, \dots, 4$) for the three developmental networks; the full sequence has $N-1$ characters, but the first four levels capture the coarse-scale structure essential for cross-network comparison. For *D. melanogaster* ($N=6$, $N-1=5$ levels), the table’s Pattern column lists the four-level prefix for uniform comparison; the full five-character word appears in the interpretation column and in the text below. This analysis distinguishes fixed points that Boolean models treat as equivalent.

Table 11: Classification sequences of fixed points across the three networks.

Network	Pattern	Count	Biological interpretation
<i>A. thaliana</i>	IEAA	6	Floral organs (LFY=1): <i>stabilize</i> at fine scales
	IEEE	4	Inflorescence (TFL1=1, LFY=0): <i>remain unstable</i>
<i>C. elegans</i>	IAII...	3	Shared four-character prefix under π^* (configs 0, 224, 232): quiescent, intermediate, and dominant-basin attractors [†]
	EEEE...	1	Fully expanding at coarse scales (config 226)
	EEEL...	1	Mixed coarse-scale expansion/isometry (config 234)
<i>D. melanogaster</i>	IAAA	1	Four-letter prefix (levels 1–4); full $(N-1)$ -word IAAAA ($N=6$): isometric at level 1, then contracting (stable) at finer scales
	IIEE	4	Four-letter prefix; full word IIEEA: isometric at levels 1–2, expanding at 3–4, contracting at level 5 (Kr/Hb resolved)

[†] The three configurations share the same first four A/E/I symbols under π^* but are distinct fixed attractors in Huang’s model (quiescent 0, intermediate 224, largest-basin 232); the table row is a *prefix* summary, not a single biological phenotype.

Three patterns are common across the networks. In *A. thaliana*, the six organ fixed points share the coarse-scale prefix IEAA while the four inflorescence states carry IEEE, separating stable organ endpoints from developmental waypoints [12, 26]. The full 12-level sequences (Supplementary Table S1) refine this into 5 distinct profiles, where all six organ types share the prefix IEAAAI up to level 6 and diverge at finer scales. In *C. elegans* [14, 19], the full sequences ($n=1, \dots, 7$) yield EEEEEEE for the high-activity attractor (config 226), shared IAIIAAA for configs 224 and 232 (the dominant attractor, 85.55% basin), IAIIIEEA for the quiescent state (config 0), and EEEIAIA for config 234. States sharing the four-character prefix can differ at fine scales (Supplementary Table S3). In *D. melanogaster* ($N=6$, full word length 5), the basal fixed point has IAAAA and the four gap-domain fixed points share IIEEA: isometric at levels 1–2 (morphogen partition), expanding at 3–4 where Kni/Gt variation conflicts [20], contracting at level 5 once Kr and Hb resolve. The gap-gene limit cycles are classified via f^2 and f^4 in Section 5 (Supplementary Tables S5–S6 for cycle data).

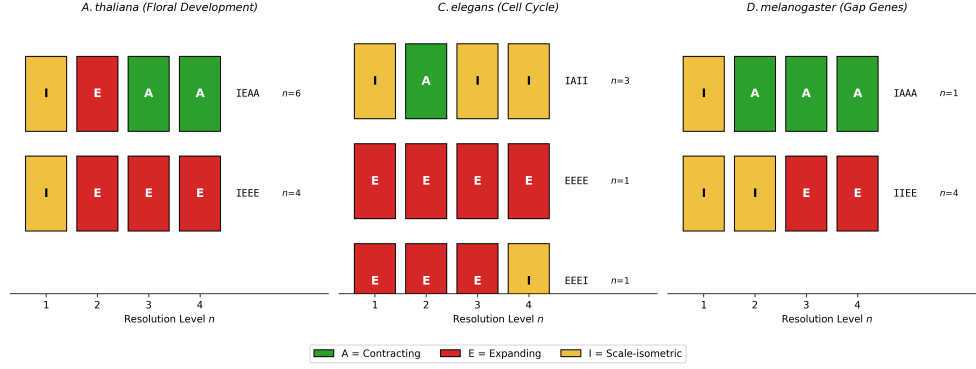


Figure 7: Classification sequences across resolution levels for the three networks. Each cell: contracting (A, green), expanding (E, red), or scale-isometric (I, yellow) at that level. Panels show levels $n = 1, \dots, 4$ only. Full sequences have length $N-1$; for *D. melanogaster* ($N=6$), the fifth symbol appears in Table 11 and in the *D. melanogaster* fixed-point paragraph in this subsection, not in the graphic. Rows use Supplementary transition CSVs and documented μ_E -optima for *A. thaliana*, *C. elegans*, and *Drosophila* (gap-gene rules: Section 5). Labels follow computed prefixes. Regenerate via `figures/regenerate_figures.py`.

Hierarchical graph comparison.

Supplementary Figures S1 and S2 show the hierarchical p -adic ball structure for the *C. elegans* cell cycle and *Drosophila* gap gene networks (the *A. thaliana* graph is in Supplementary Appendix S4). The three graphs exhibit distinct biological structures: *A. thaliana* shows clear separation between contracting (organ) and expanding (inflorescence) basins; *C. elegans* (Huang-aligned table) shows several coarse-scale prefixes (e.g. IAII..., EEEE...) across the five attractors; *Drosophila* exhibits a high proportion of contracting basins, consistent with the well-defined spatial expression domains of gap genes.

Symmetry structure of the optimal set.

We describe the symmetry structure of $\text{Opt}(f)$ for the three maps: exchangeable pairs, rigidity index, and the relation between commutation and compensatory cancellation (Observation 6.4).

The μ_E -minimizing orderings for each network are not always unique: several permutations may achieve the same global minimum. The structure of this degeneracy encodes which gene positions are uniquely determined by the dynamics and which admit permutation freedom.

Definition 6.2 (Symmetry group of the optimal set). *Let $\text{Opt}(f) = \{\pi \in S_N : \mu_E(\pi) = \mu_E^*\}$ be the set of μ_E -minimizing orderings. Two positions $i < j$ in π^* are exchangeable if $\text{Opt}(f)$ contains an ordering σ that agrees with π^* except that $\sigma(i) = \pi^*(j)$ and $\sigma(j) = \pi^*(i)$. The symmetry group $G(f) \leq S_N$ is the group generated*

by all such transpositions $(i\ j)$. A position k is rigid if $\pi(k) = \pi^*(k)$ for every $\pi \in \text{Opt}(f)$.

When $|\text{Opt}(f)| > 1$, the optima in our examples differ only by disjoint transpositions of adjacent positions, so $G(f)$ is a direct product of copies of C_2 . Although $G(f)$ is defined via μ_E -optima, the same transpositions preserve μ_A as well, so $G(f)$ acts on $\text{Opt}_{\mu_A}(f)$ and partitions it into orbits of size $|G(f)|$ (Table 10). For *C. elegans* (Huang-aligned dynamics), $\text{Opt}(f)$ is a singleton, so $G(f)$ is trivial and every position is rigid. Exhaustive or branch-and-bound search certifies the following (Section 2; scripts in Supplementary Material):

Observation 6.3 (Symmetry groups and rigidity across networks). *Table 12 summarizes the symmetry structure of $\text{Opt}(f)$ for the three networks.*

Table 12: Symmetry structure of μ_E -minimizing orderings. $|\text{Opt}(f)|$: number of optimal orderings; $G(f)$: symmetry group; r : rigidity index (fraction of positions fixed across all optima). Exchangeable pairs are listed with their positions in π^* and functional role.

Network	$ \text{Opt}(f) $	$G(f)$	r	Exchangeable pairs (positions, functional role)
<i>A. thaliana</i> ($N=13$)	4	$C_2 \times C_2$	0.85	EMF1 / LFY (1–2, Polycomb vs. floral activation), AP2 / FT (4–5, miR172 circuit) [4, 32, 36]
<i>C. elegans</i> ($N=8$)	1	$\{e\}$	1	none. The unique π^* places <i>cdc-25.1</i> and <i>cdk-2</i> at the coarsest scales [14, 19]
<i>D. melanogaster</i> ($N=6$)	4	$C_2 \times C_2$	0.33	Bcd / Cad (0–1, anterior/posterior maternal morphogens) [11], Kni / Gt (2–3, isomorphic posterior gap rules; Supplementary Appendix S3)

When exchangeable pairs exist, $G(f)$ is generated by disjoint transpositions of adjacent positions, and the quotient of the total order π^* by these transpositions yields a chain: the *equivalence classes* $[g] = \{g\}$ for rigid genes and $[g] = \{g, g'\}$ for exchangeable pairs inherit a total order from π^* . For *C. elegans*, each class is a singleton. Figure 8 displays these chains for the three networks.

Each exchangeable pair corresponds to genes that the experimental literature identifies as performing symmetric or antagonistic roles at the same regulatory tier [11, 14, 32, 36]. Since f is fixed from the published Boolean model, these degeneracies are determined by the transition structure of the map (Remark 2.1). A general classification of $G(f)$ for arbitrary Boolean maps remains open (Question 6.6).

Observation 6.4 (Commutation vs. compensatory cancellation). *Global commutation $f \circ \tau_{ij} = \tau_{ij} \circ f$ (where τ_{ij} swaps coordinates i and j) is sufficient for $E^\pi(n) = E^{\pi'}(n)$ at every n when π places i, j at adjacent positions $k, k+1$: for $n \leq k$ the n -balls coincide under both orderings, and for $n > k$ the transposition maps each π -ball bijectively onto the corresponding π' -ball while preserving image digit-sequences. However, no exchangeable pair in the three networks satisfies this stronger condition (failures range from 8/64 configurations for Kni / Gt in *Drosophila* to 5,120/8,192 for EMF1 / LFY in*

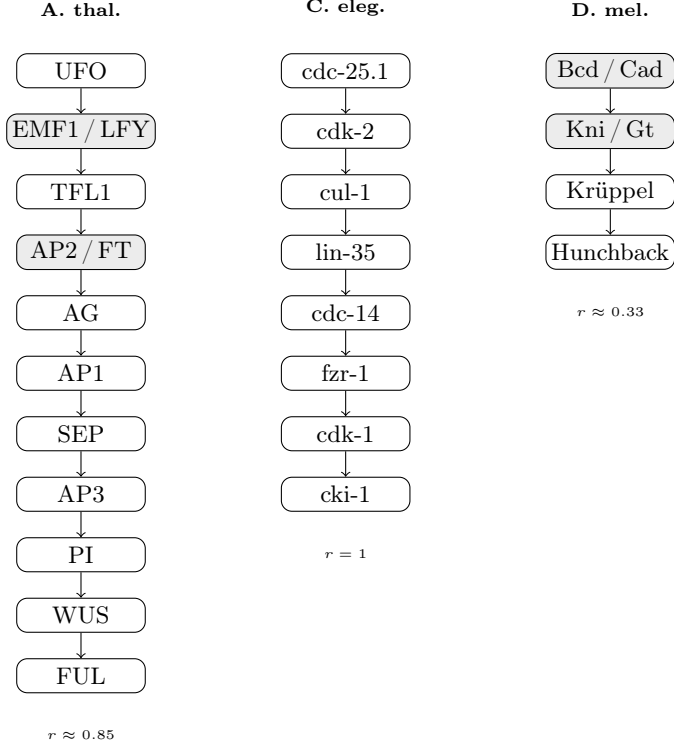


Figure 8: Equivalence-class chains for the three networks. Each node is a rigid gene position (white) or an exchangeable pair (shaded). Arrows indicate the total order inherited from π^* . The rigidity index $r = |\{k : k \text{ rigid}\}|/N$ (fraction of positions fixed across all optima) decreases from left to right; $r = 1$ means the optimal ordering is unique, while lower values indicate greater permutation freedom among the optima.

A. thaliana). Nevertheless, $E^\pi(n) = E^{\pi'}(n)$ holds at every level through a compensatory cancellation: the number of balls reclassified from E to non- E by the swap exactly matches the number reclassified from non- E to E . Swapping *Kni* and *Gt* in *Drosophila* at level 3 moves one ball each way, preserving $E(3) = 4$. The balance holds at every level for all 4 exchangeable pairs: *Bcd / Cad* and *Kni / Gt* in *Drosophila*, plus *EMF1 / LFY* and *AP2 / FT* in *A. thaliana*.

Remark 6.5 (μ_E -equivalence over $\text{Opt}(f)$ vs graph automorphism). *Observation 6.4* above is best read against a stricter notion. A transposition τ_{ij} that satisfies $f \circ \tau_{ij} = \tau_{ij} \circ f$ on the whole configuration space is a graph automorphism of the network: the dynamics is genuinely invariant under the swap at every state. We denote the group generated by such automorphisms by $\text{Aut}(f) \leq S_N$. The exchangeable pairs documented in this paper generate μ_E -equivalence over $\text{Opt}(f)$ without satisfying this stronger condition. The symmetry group $G(f)$ of $\text{Opt}(f)$ is therefore in general strictly larger than $\text{Aut}(f)$, and captures functional equivalence at the level of the variational ordering

rather than structural equivariance of the map. A direct transposition-automorphism detector applied to the three networks studied here yields the trivial automorphism group in each case, so the entire exchangeable-pair structure reported above is purely μ_E -degenerate. The coincidence of those pairs with documented antagonistic gene pairs (EMF1/LFY Polycomb antagonism, AP2/FT miR172 circuit, Kni/Gt posterior gap homology) is therefore a property of μ_E over $\text{Opt}(f)$, not a consequence of network symmetry.

Question 6.6 (Structure of $G(f)$). *In the three networks analyzed here, $G(f) \cong C_2^k$ for some $k \geq 0$, each generating transposition exchanges adjacent positions, and the quotient order is a chain; whether this always holds for Boolean networks is left open. Two structural questions arise:*

1. Sufficiency gap: global commutation is sufficient for μ -equivalence but not necessary (Observation 6.4). What is the minimal condition on f that guarantees the compensatory cancellation? The failures concentrate on genes at late π^* -positions and propagate through downstream targets, suggesting a condition involving the restriction of f to the first $k+2$ coordinates of π^* .
2. Polynomial symmetry: isomorphic update polynomials (e.g., Kni and Gt in *Drosophila*) imply exchangeability, but exchangeability can occur without polynomial isomorphism (EMF1/LFY in *A. thaliana* have non-isomorphic update rules). How far does the gap between polynomial symmetry and μ -equivalence extend?

7 Discussion

With $\text{Opt}(f)$ and its symmetry structure characterised in Section 6, we now turn to the broader interpretation of π^* and its comparison with alternative rankings. The μ -based hierarchy and the polynomial influence ranking agree at the top of each network but diverge at intermediate positions, and minimising expansion is a more selective criterion than maximising contraction. These contrasts, together with the validation against literature hierarchies, structure the discussion that follows.

Two distinct contributions.

This work delivers two distinct contributions. The first is the μ_E -minimising ordering π^* , which assigns a coarse-to-fine hierarchy to the coordinates and recovers the canonical master regulators of the three developmental networks from the discrete dynamics alone. The second, conceptually independent, is the *scale-resolved A/E/I classification of every fixed point* of the synchronous map: at each resolution n the ball containing a fixed point is labelled contracting (A, $\Lambda_{n,m} < 1$), expanding (E, $\Lambda_{n,m} > 1$), or isometric (I, $\Lambda_{n,m} = 1$), producing a word in $\{A, E, I\}^{N-1}$ that summarises the multiscale stability profile of the orbit. The biological reading is direct: A-rich profiles signal robust developmental endpoints (canalised fates), E-rich profiles signal transient or decision states, and mixed profiles signal bifurcation- or threshold-like regimes. In *A. thaliana* this separates the six floral organ fixed points (prefix **IEAA**) from the four inflorescence fixed points (**IEEE**...), a distinction invisible to ordinary basin-size analysis, in line with the experimentally documented role of inflorescence as a developmental waypoint.

Why p -adic, and why the Haar weighting?

Three candidate alternatives address the same question without invoking ultrametric geometry: basin-membership ordering, conditional entropy over nested partitions, and eigenvector centrality of the transition digraph. The p -adic framework differs in three respects. The weighting p^{N-n} is the Haar volume of a level- n cylinder, fixed by the metric structure rather than chosen externally. The $A/E/I$ classification carries a dynamical-systems interpretation (point-level attracting/repelling/indifferent of ϕ at the corresponding scale, via the companion paper [27]) absent in graph- or entropy-based methods. And the variational principle is global on S_N rather than greedy. A systematic empirical comparison against these alternatives is outside the present scope.

Comparison with BioBalm and the Kadelka meta-analysis. BIOBALM [33] decomposes a Boolean network into a directed acyclic graph of trap spaces (subspaces that no trajectory can leave). It reads the Boolean update rules symbolically and produces a partition determined by the algebraic structure of the rules, whereas our partition reads only the transition table f and produces a partition determined by the metric structure of $\mathbb{Z}_p$ under a coordinate ordering. BioBalm is invariant under coordinate permutation, whereas π^* is precisely the coordinate ordering that the dynamics makes special. The two views are complementary. The meta-analysis of [21] surveys ~ 100 published Boolean GRNs and quantifies design principles (canalisation, redundancy, effective connectivity, output bias). The μ_E -score is a finer, scale-resolved companion: where [21] reports network-level scalars, our framework reports a distribution of $A/E/I$ labels indexed by the ball at each resolution. A systematic application of μ_E to that corpus is the natural empirical extension.

Comparison with the coherence-gap analysis of Bavisetty, Wheeler, and Kadelka (2025). A recent contribution from the Kadelka group [5] reports a systematic *coherence gap* across 122 expert-curated biological Boolean models: attractor states are consistently less stable to single perturbations than the basin states approaching them, with magnitude predicted by network bias ($\rho = -0.997$). Their analysis and ours address the same underlying phenomenon, canalisation as the source of robustness in cell-fate decisions, through complementary formalisations. The coherence gap is a single cross-corpus statistic comparing attractor-state to basin-state stability under one-bit perturbation. The descriptor introduced here is per-network and resolves stability across scales through the $A/E/I$ word and the variational ordering π^* on S_N . Where the coherence gap collapses the stability profile of an orbit to one scalar, the $A/E/I$ word records contraction, isometry, and expansion at each of the $N - 1$ resolution levels, so an A-rich profile with an isolated E suffix recovers the qualitative pattern they document. The two viewpoints are orthogonal rather than redundant. We view a systematic application of μ_E and the $A/E/I$ classification to the 122-model corpus as the natural empirical extension of the present work.

Polynomial influence vs. μ -hierarchy.

The polynomial influence score I_{poly} measures direct syntactic reach in the update rules. The ordering π^* ranks genes by multiscale ball expansion. The two rankings agree on top genes in each network and diverge at intermediate positions, where the

weighting of μ adds information beyond I_{poly} (Section 6, Supplementary Appendix S3). Polynomial degree captures topological centrality of the wiring while μ_E -ordering captures dynamical centrality on the state space. Both notions of “master” largely coincide. Self-maintaining switches like UFO (indeg = 0, outdeg = 1, yet position 0 in π^*) are visible only to the latter. In *Drosophila*, Giant and Krüppel have the largest I_{poly} yet follow the morphogens in π^* because Bcd and Cad set the coarsest partition ($n=1$, weight p^{N-1}). Boolean sensitivity gives a complementary baseline: Supplementary Table S8 reports $\rho(s, \pi^*) \in \{0.64, 0.67, 0.75\}$ across the three networks, with agreement at extremes (top and bottom) and divergence at intermediate ranks.

The role of UFO across optimization criteria.

UFO’s update polynomial is $T_0 = x_0$ (self-maintaining), and its only direct target is AP3. Its leading position in π_E^* stems from its role as a binary switch that partitions the configuration space into two halves with distinct basin structure, not from that single outgoing edge. The μ_A -optimal set gives a complementary perspective: among the 10 certified orbits of μ_A -maximisers, UFO occupies every position from 0 to 9 while $\mu_A = 54,560$ stays constant, but μ_E is non-monotone (lowest 26,976 at position 0, highest 35,168 at position 1; Supplementary Table S2). UFO’s contribution to contraction is position-independent, while its contribution to expansion is sensitive to its interaction with the master-regulator block. The blocks EMF1–LFY–TFL1 and the tail WUS, AP3, PI, FUL keep the same relative order across all μ_A -orbits.

Robustness checks and scope.

We have used *synchronous* updates on a single published Boolean model per organism. Two robustness checks confirm that π^* is not an artefact of these choices.

Asynchronous-update robustness (*C. elegans*). Two standard asynchronous schemes were tested [25]: a random-order asynchronous scheme (ROA, projected to a deterministic map by majority over $R = 256$ schedules) and a general-asynchronous scheme. The map f_{ROA} differs from f_{sync} on 57% of source configurations, yet both schemes preserve Huang’s five fixed points and the basin ranking. Exhaustive search of 8! orderings on f_{ROA} yields π_{ROA}^* matching π_{sync}^* on the first five positions exactly, with only a within-tier swap of *cdc-14* $\leftrightarrow$ *fzr-1* at positions 6–7 (both APC/Cdh1 mitotic-exit components), Spearman $\rho(\pi_{\text{sync}}^*, \pi_{\text{ROA}}^*) = 0.976$. The master block is not an artefact of synchronous timing.

Rule-set robustness (*A. thaliana*). The *A. thaliana* variant of Chaos et al. [8] differs from Espinosa-Soto et al. [12] on 31.7% of configurations. A 2-opt hill-climber returns the *same* ordering and the same $C_2 \times C_2$ orbit structure under the Chaos rule set, with $\rho = 1.0$ between the optimal orderings of both variants. The master-regulator backbone identified by μ_E -minimisation is stable under rule-level perturbations of order one third of the state space.

Scalability beyond the certified range.

Branch-and-bound certifies the global optimum for *A. thaliana* ($N=13$). For larger networks, genetic algorithms provide a practical alternative (the GA recovered one of the four *A. thaliana* optima in 60.8% of runs). On two synthetic 3-tier Boolean networks

with planted master-regulator hierarchies and genuine feedback, branch-and-bound at $N = 15$ improves on the planted-hierarchy warm-start while keeping every planted master and intermediate in its tier. At $N = 20$ (2^{20} configurations) a GA reaches $\mu_E = 0$ in 8/10 runs and places at least two of the four planted masters in the leading positions every time, whereas 10,000 random orderings stay four orders of magnitude above the optimum. The procedure recovers a genuinely present hierarchy well beyond the exhaustively certifiable range. The framework supports three modes (Section 1): scale-resolved analysis at a chosen ordering (mode i, cost $O(N p^N)$), constrained optimisation under partial biological priors (mode ii), and unconstrained minimisation (mode iii), the last exercised here. For $N \sim 30\text{--}50$ where master regulators are already known by experiment, modes (i)–(ii) on the literature-grounded ordering and its neighbourhood yield diagnostically useful $A/E/I$ words without ever solving the global optimisation.

What the agreement actually means: a chaos-minimisation reading.

The framework recovers the most influential coordinates from scratch: unconstrained mode (iii) computations on the three networks produce the master regulators in the leading positions of π^* with no biological input (Spearman $\rho = 1$ on the master subsets of the two larger systems).

Mathematical reading. By construction the certified minimiser π_E^* identifies the coordinates whose placement at coarse scales contributes most to reducing μ_E and, by the control identity $\mu_E + \mu_A + \mu_I = (N-1)p^N$, equivalently maximises $\mu_A + \mu_I$ at the same scales. This is a tautology of the optimisation. Whenever certified by branch-and-bound or exhaustive enumeration, rather than reported as a GA output that may be a local minimum, the mathematical claim is unconditional.

Biological reading. What is genuinely surprising is that the mathematical minimisers of μ_E coincide with the regulators that decades of experimental genetics have identified as canonical in each of the three networks. The framework was not engineered to recover those genes. This coincidence admits two interpretations. First, that natural selection has explored regulatory architectures and, in the stable regimes that support robust development, those architectures sit close to the μ_E -minimiser: a chaos-controlled basin of dynamical organisation, in the spirit of the “edge of chaos” picture [2, 22] and the recent finding that biological regulatory ensembles cluster near the order-chaos boundary across 67 Boolean models [10]. Second, more weakly, that whatever else evolution optimises, the resulting architectures are at least *compatible* with low coarse-scale expansion, a necessary condition for canalisation in the sense of [35]. The three networks studied here do not, by themselves, settle the question; they make the following conjecture quantitative and testable.

Conjecture 7.1 (Chaos-controlled basin hypothesis). *Stable developmental regulatory architectures sit at or near $\arg \min_{\pi} \mu_E(\pi)$ of their own discrete dynamics.*

Empirical tests of the conjecture, including a deployment on networks outside developmental biology, are discussed in Section 8.

8 Conclusions

Operating within the hierarchical p -adic framework of the companion paper [27], we have computed a dynamics-based gene ordering from the transition structure of a published Boolean GRN, without external regulatory rankings. Applied to three developmental models, leading positions match literature-identified master regulators, and the scale-dependent $A/E/I$ classification separates fixed points corresponding to distinct biological states beyond what flat Boolean attractor analysis reveals. Branch-and-bound certifies the global μ_E -minimum for *A. thaliana* ($\rho = 1.0$ vs the eight-gene biological table, floral-organ basins covering 95% of the state space). Exhaustive search certifies the unique minimiser for *C. elegans* and the four-element optimum for *Drosophila*, where the $A/E/I$ machinery further extends to periodic orbits via f^2 and f^4 (expansion drops by 78%). The symmetry group $G(f)$ of $\text{Opt}(f)$ varies systematically across networks, and is in general strictly larger than the network’s graph automorphism group: the observed exchangeable pairs are μ_E -degenerate over $\text{Opt}(f)$ rather than network automorphisms, a distinction whose biological reading is discussed in Remark 6.5.

A testable biological conjecture.

The coincidence of the certified μ_E -minimisers with documented master regulators across three independent developmental networks, with no biological input to the optimisation, is the empirical basis for the chaos-controlled basin hypothesis (Conjecture 7.1, Section 7). The hypothesis is quantitative and testable: it predicts the same coincidence in other networks with documented master-regulator hierarchies, and predicts that networks where it fails should either optimise an alternative criterion (e.g. μ_A instead of μ_E , Section 6) or display genuine evolutionary indifference to coarse-scale dynamical organisation.

Directions for further work.

Three extensions complete the present picture. First, the framework deploys naturally to networks where master regulators are already identified by experiment but multiscale stability is not, via the modes (i)–(ii) of Section 1. Applying it to epithelial–mesenchymal transition in cancer is a planned next step. Second, a systematic sweep over primes p and over alternative Boolean discretisations of the same biology will sharpen the rule-set robustness already verified for *A. thaliana* (the Chaos et al. variant of 31.7% rule-level perturbation preserves the master backbone) into a quantitative invariance statement. Third, the chaos-controlled basin hypothesis (Conjecture 7.1) is testable on networks outside developmental biology, with empirical validation across a broader corpus of certified Boolean GRNs a natural extension of the present work.

Software and reproducibility.

All algorithms (computation of (μ_E, μ_A, μ_I) , branch-and-bound search, genetic-algorithm heuristic, $A/E/I$ classification) are bundled in the open implementation **p-GNR-Suite** [29], with a browser-based applet pre-loaded with the three reference networks (see the Zenodo bundle for the deployment link). The reproducibility bundle

(transition tables, branch-and-bound logs, supplementary figures, scripts) is archived at Zenodo, [doi:10.5281/zenodo.19533579](https://doi.org/10.5281/zenodo.19533579).

Supplementary material

Supplementary PDF.

A self-contained companion document (`supplementary_appendix.tex` / `.pdf`) collects: (i) detailed fixed-point and $E/A/I$ data per network (Appendix S1); (ii) extended fixed-point tables and basin coverage (Appendix S2); (iii) the full polynomial interpolation analysis with per-gene update polynomials and the influence ranking I_{poly} (Appendix S3); (iv) hierarchical ball-diagram visualisations for the three networks (Appendix S4); (v) basin analysis under mutants and noise (Appendix S5); and (vi) extended centrality and sensitivity comparison tables (Appendix S6). Tables and figures are numbered with the prefix S.

Reproducibility bundle and supplementary formulas.

Supplementary materials are organized as follows. A short reproducibility guide (commands and script file names, independent of any local folder layout) is included with the supplementary file package.

Item	Initial submission	Extended release
Supplementary Appendix A (PDF)	Yes	n/a
Files S1–S4	Yes	n/a
Full transition CSVs; code; extended B&B logs; extra GA outputs	No	Zenodo DOI 10.5281/zenodo.19533579 (see Data availability)

- **Submitted with the article (and cited in the text where indicated):** Supplementary Appendix A (PDF) with explicit formulas for $F_1, \dots, F_4$ (*A. thaliana*), rational maps at resolution levels $n = 1, 2, 3, 4$ under π^* , generated from the transition table by the gluing procedure ([27]); and Files S1–S4 (genetic-algorithm robustness CSVs, reproducibility script, and branch-and-bound certification summary for optimal orderings).
- **Extended materials (review and public release):** the full artifact bundle (CSVs, code, logs, reproducibility notes) is deposited at Zenodo [29]; during peer review the same bundle is also available to the editor and referees on request.

Declarations

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Competing interests

The authors declare that they have no competing interests.

Authors' contributions

All authors contributed equally to this work. All authors read and approved the final manuscript.

Data availability

The transition tables (CSV), Python analysis scripts, branch-and-bound and robustness logs, and supplementary PDF Appendix A cited in the text are the reproducibility bundle for this manuscript. Directory layout and numerical verification checks are described in `supplementary/README-SUPPLEMENTARY-MATERIAL.md` and accompanying README files under `supplementary/`. The complete bundle is publicly available at Zenodo [29], <https://doi.org/10.5281/zenodo.19533579>.

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