

Highlights

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

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- Permutations $\pi \in S_N$: μ_E -optimal gene orderings from each published Boolean model once f is fixed.
- Boolean GRN tables feed a p -adic hierarchy minimizing the expansion score μ_E .
- Scale-resolved $A/E/I$ words distinguish attractors; extension to periodic orbits via f^k cuts μ_E^* by 78%.
- Global optima: branch-and-bound (*A. thaliana*); exhaustive search (*C. elegans*, *Drosophila*).
- Degenerate optima link exchangeable pairs to documented regulatory symmetries.

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

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Abstract

Multiscale discrete dynamics on Boolean state spaces motivate this work: for each application, a published Boolean GRN and its synchronous update rules fix the map f ; the hierarchical p -adic framework searches over permutations $\pi \in S_N$ (gene orderings) and selects π^* minimizing the expansion score μ_E using the transition table of that fixed map, with no prior master-regulator rankings or external hierarchical labels in the optimization. Here μ_E is the expansion term in $\mu = \mu_E + \mu_A + \mu_I$ (Methods). When several orderings attain the same minimum μ_E , π^* denotes the representative used in tables and figures. Branch-and-bound certifies global optima when exhaustive search over all orderings is infeasible. We apply this pipeline to *Arabidopsis thaliana* ($N=13$, floral development), *Caenorhabditis elegans* ($N=8$, cell cycle), and *Drosophila melanogaster* ($N=6$, gap genes), comparing π^* *a posteriori* with experimentally supported master-regulator hierarchies. Leading positions in π^* are consistent with those regulators in each case. Resolution-level $A/E/I$ classification sequences separate fixed-point attractors that flat Boolean attractor analysis does not distinguish: floral organ versus inflorescence fates in *A. thaliana*; distinct cell-cycle regimes in *C. elegans*; gap-gene fixed points in *Drosophila*. For *Drosophila*, limit cycles are analyzed via f^2 and f^4 , reducing μ_E^* by 78%; the sequence table lists fixed points only. Across these models, the μ_E -optimal set ranges from a unique minimum (*C. elegans*) to four $C_2 \times C_2$ -related μ_E -minima in *A. thaliana* and *Drosophila*; larger μ_A -optimizer sets occur in some cases (e.g. eight μ_A -optima in *Drosophila*), and exchangeable pairs align with documented regulatory symmetries where degeneracy appears. None of these pairs satisfies global commutation with f , yet a compensatory cancellation of ball reclassifications preserves the level counts $E(n)$ exactly; a general characterization of this mechanism remains open. Basin analysis and polynomial interpolation on the same transition data provide complementary validation of the inferred hierarchy.

1. Introduction

Gene regulatory networks (GRNs) govern cell differentiation, organ development, and phenotypic stability. Discrete dynamical systems on Boolean state spaces raise a scale-organization question: which components exert the greatest influence on global dynamics, and how does that influence transcend static connectivity? Topology alone (degree, centrality) does not capture the fact that some genes act as master regulators whose state determines whole branches of behavior, while others refine outcomes within an already committed fate.

We use a hierarchical p -adic framework¹ 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

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external regulatory rankings or hierarchical role labels in the search for 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 . A stability measure μ quantifies how much the dynamics *expands* these balls—that is, how often and at what scales the image of a ball escapes its own boundary. The ordering that *minimizes* μ is the hierarchy under which the network is most stable according to this criterion. Comparing the resulting ordering with literature-based master-regulator rankings (Sections 3–5) validates the dynamics-based hierarchy *a posteriori*.

Notation and algorithms are fixed in Section 2: the map f , the decomposition $\mu = \mu_E + \mu_A + \mu_I$ with $\mu_E = \sum_{n=1}^{N-1} E(n) p^{N-n}$, the $A/E/I$ ball classification, and the μ -minimizing ordering π^* . Construction, invariance, stability properties, and connections to p -adic fixed-point theory are proved in the companion paper [21].

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 [2, 10, 18]; (2) *Caenorhabditis elegans* cell cycle ($N = 8$), with CDK–cyclin and checkpoint logic [11, 14]; (3) *Drosophila melanogaster* gap genes ($N = 6$), with morphogen-driven spatial patterning [9, 15]. 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*, and branch-and-bound global certification over the full search space for *A. thaliana* ($N=13$). Where the literature gives clear consensus we cite it; where our analysis suggests a new reading we say so. Applying this framework to epithelial–mesenchymal transition in cancer is planned as future work.

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 (Section 3) and polynomial interpolation (Appendix D). Section 8 summarizes the main findings.

2. Methods

The complete theoretical foundation—including proofs of invariance, approximation bounds for ϕ , and the hierarchical construction—is in the companion paper [21]. *This article* implements the pipeline on three published Boolean models, certifies or exhaustively searches for optimal orderings, and validates π^* against experimental literature; it does not re-prove the analytic results of [21]. The operational definitions and computational procedures are collected here. *Results invoked from [21]*: existence of a rational interpreter; existence of hierarchical approximations F_n ; controlled approximation of ϕ by each F_n , with an explicit bound on $|F_n(z) - \phi(z)|_p$ depending on the image radius of the ball at that resolution level (Proposition 3.11 in [21]); independence of the discrete data (f, ι) for the radii $t_{n,m}$ and hence for the $A/E/I$ classification (see the hierarchical-framework and stability sections of that paper for precise statements and proofs). The analysis pipeline is:

$$\begin{array}{ccccccc} \text{GRN } (f) & \longrightarrow & \text{Encoding } (\iota_\pi) & \longrightarrow & \text{Classification } (A/E/I) & \longrightarrow & \mu\text{-optimization} \\ \text{discrete dynamics} & & p\text{-adic embedding} & & \text{ball-level dynamics} & & \text{optimal ordering } \pi^* \end{array}$$

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, ι_π) 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 $\mathcal{C} = \{0, \dots, p-1\}^N$ 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 throughout, 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 $m_a = \sum_{k=0}^{N-1} x_{\alpha_k} p^k$. 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 [21].

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*).

Maximum common prefix length. For each ball $B_{1/p^n}(m)$, define

$$M_{n,m} := \max\{k \in \{0, \dots, N\} : \iota_\pi(f(c)) \bmod p^k \text{ agrees for all configurations } c \in \mathcal{C} \text{ with } \iota_\pi(c) \in B_{1/p^n}(m)\}. \quad (1)$$

Equivalently, $M_{n,m}$ is the number of leading p -adic digits on which all images agree.

Coarse multiplier and ball classification. The *coarse multiplier* is $\Lambda_{n,m} := p^{n-M_{n,m}}$, and balls are classified as:

- *Expanding* (E): $\Lambda_{n,m} > 1$ (equivalently $M_{n,m} < n$)—image spreads beyond the ball.
- *Contracting* (A ; letter retained from [21]): $\Lambda_{n,m} < 1$ (equivalently $M_{n,m} > n$)—image is contained in a smaller ball.
- *Isometric* (I): $\Lambda_{n,m} = 1$ (equivalently $M_{n,m} = n$)—image has the same scale.

Stability scores. The level counts are $E(n) := \#\{m : M_{n,m} < n\}$, $A(n) := \#\{m : M_{n,m} > n\}$, $I(n) := \#\{m : M_{n,m} = n\}$, satisfying $E(n) + A(n) + I(n) = p^n$. 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. For instance, in *Drosophila* the four non-basal gap-domain fixed points share the word IIEEA (levels 1–5; Section 5), while the quantitative M -profiles still distinguish finer expansion/contraction strengths at intermediate scales. In *C. elegans*, configs 224 and 232 share the full word*

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 $= p^{n-M_{n,m}}$
$E(n), A(n), I(n)$	Counts of expanding, contracting, isometric balls at level n
$\mu = \mu_E$	Stability measure (weighted count of expanding balls)

IAIIAAA but differ in natural encoding and basin size; config 226 is fully expanding (EEEEEEE) and config 0 is quiescent with pattern IAIIIEEA—so the A/E/I word is not determined by a “quiescent vs. cycling” label alone. For *A. thaliana*, Appendix A lists five distinct full-length classification profiles among the fixed points: inflorescence is separated from organ fates, and organ identities are distinguished at fine scales—though some organ types share the same full word when their Boolean configurations coincide at those scales (e.g. sepal, petal, and petal-UFO share IEAAAAIEEEEE). 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.

Terminology. Throughout this paper, the labels A/E/I refer to the *scale-level* ball classification above. We use *expanding/contracting/isometric* for ball-level behavior and reserve *repelling/attracting* 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.

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. 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 .

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 [21]; the bound $|F_n(z) - \phi(z)|_p \leq 1/p^{M_{n,m}}$ on each ball $B_{1/p^n}(m)$ is Proposition 3.11 therein. 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).

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 use branch-and-bound with admissible pruning bounds over the full search space. At each partial ordering of depth k , the algorithm computes a lower bound on μ_E for any completion of that prefix; if the partial μ_E already exceeds the best-known value, the entire subtree is pruned. A symmetric bound (upper bound on μ_A) is used for μ_A

maximization. The pruned search for μ_E -minimization completed in ≈ 29 hours on a single core; 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. [14]: 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 F_n dynamics
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 $V = \bigsqcup_{n=0}^{m_{\max}} V_n$ (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 Section 3.

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.

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 [18], refined by Espinosa-Soto et al. [10], extended to the full flowering transition pathway in [7], and reviewed by Álvarez-Buylla et al. [2]. 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:

Each gene has 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 given in the Supplementary Material.

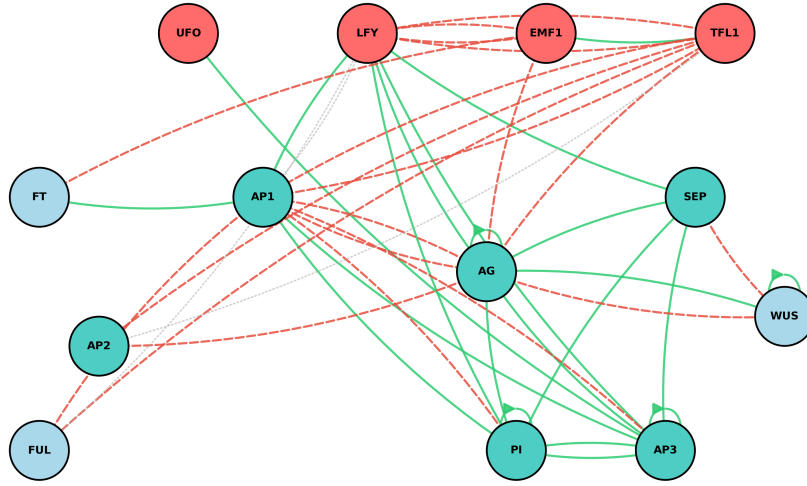


Figure 1: *A. thaliana* floral development network ($N = 13$ genes). Node colors: red = master regulators (UFO, LFY, EMF1, TFL1), dark teal = organ identity genes (AP1, AP2, AG, PI, AP3), light teal = other regulatory genes (FT, FUL, WUS, SEP). Edge styles: solid green arrows = activation, dashed red = repression, solid gray = regulation with context-dependent effect. Network topology and interaction types from [10, 18]; figure generated programmatically from Boolean rules (see Supplementary Material).

Table 2

Genes in the *Arabidopsis thaliana* floral development network.

$x_0 = \text{FUL},$	$x_1 = \text{FT},$	$x_2 = \text{AP1},$	$x_3 = \text{EMF1},$
$x_4 = \text{LFY},$	$x_5 = \text{AP2},$	$x_6 = \text{WUS},$	$x_7 = \text{AG},$
$x_8 = \text{TFL1},$	$x_9 = \text{PI},$	$x_{10} = \text{SEP},$	$x_{11} = \text{AP3},$
$x_{12} = \text{UFO}$			

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); they share the same (μ_E, μ_A, μ_I) and the same level counts, but they assign different genes to the symbols $x_0, \dots, x_{12}$. Throughout this paper, π^* means the *single* ordering displayed in Eq. (3); all polynomials, tables, and figures index variables in that order. The other three minimizers swap $\text{EMF1} \leftrightarrow \text{LFY}$ at positions 1–2 and/or $\text{AP2} \leftrightarrow \text{FT}$ at positions 4–5 only.

$$\begin{aligned}
 & (x_0, x_1, x_2, x_3, x_4, x_5, x_6, x_7, x_8, x_9, x_{10}, x_{11}, x_{12}) \\
 & \parallel \\
 & (\text{UFO}, \text{EMF1}, \text{LFY}, \text{TFL1}, \text{AP2}, \text{FT}, \text{AG}, \text{AP1}, \text{SEP}, \text{AP3}, \text{PI}, \text{WUS}, \text{FUL}).
 \end{aligned} \tag{3}$$

Under π^* , early positions (UFO, EMF1, LFY, TFL1) carry the largest influence on stability, while configurations differing only in late-position genes (FUL, WUS, PI, ...) are p -adically close. The first four positions match the master regulators of floral fate [2, 10, 18]. In particular, 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 in Table 3 in all eight positions (Spearman $\rho = 1.0$, $p = 2.48 \times 10^{-5}$ by exact permutation test; Section 6). This agreement is an external validation of the dynamics-based hierarchy; Remark 3.2 records how the same transition table organizes genes into functional tiers (gene indices unlabeled in the optimization), and Remark 3.3 quantifies local sensitivity around π^* .

Table 3

Ordering π^* (Eq. 3) vs. biological hierarchy for *A. thaliana*. Positions 1–13 in the table correspond to indices 0–12 in the ordering $(x_0, \dots, x_{12})$. Spearman $\rho = 1.0$ on the eight positions; see `compute_official_optima_athaliana.py` in the Supplementary Material.

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

Stability scores. Under π^* the three scores are $(\mu_E, \mu_A, \mu_I) = (26,776, 53,880, 17,648)$, 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 [18] 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: $EMF1 \leftrightarrow LFY$ (interchangeable),
2. Positions 4–5: $AP2 \leftrightarrow FT$ (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 $n = 1, \dots, 12$ (Table 4); 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 3 summarizes the comparison with experimental evidence [4, 23, 25, 29].

Remark 3.2 (Dynamics-based hierarchy aligned with the literature). *The ordering π^* minimizes μ over permutations using the transition table of the fixed Mendoza–Álvarez-Buylla map, with no external regulatory rankings in the optimization (Remark 2.1). The resulting tier structure organizes the 13 genes into seven functional groups that align with the known temporal sequence of floral development [2, 10, 18]:*

Tier	Positions	Gene(s)	Biological function
1	0	UFO	Master switch (F-box cofactor of LFY [22])
2	1–2	EMF1 / LFY	Vegetative $\leftrightarrow$ reproductive transition
3	3	TFL1	Inflorescence meristem maintenance [30]
4	4–5	AP2 / FT	A-class specification / florigen signal
5	6–8	AG, AP1, SEP	C/A/E organ identity cascade
6	9–10	AP3, PI	B-class petal/stamen specification
7	11–12	WUS, FUL	Stem cell niche / downstream effector

Meristem identity genes occupy the coarsest positions (Tier 1–3), organ specification follows (Tier 4–6), and downstream effectors close the hierarchy (Tier 7).

The two symmetry pairs (Observation 3.1) correspond to documented functional antagonisms:

1. $EMF1 \leftrightarrow LFY$. *EMF1* is a Polycomb-group protein that maintains vegetative identity by repressing floral genes [17, 25]; *LFY* is the master transcription factor that activates floral identity [29]. Their mutual exclusion (*EMF1* on $\Leftrightarrow$ *LFY* off, and vice versa) makes them informationally equivalent at the second-coarsest resolution: conditioning on *UFO*, both genes encode the same binary vegetative/reproductive partition of the state space.
2. $AP2 \leftrightarrow FT$. *AP2* represses flowering and specifies sepal/petal identity (A-class); *FT* (florigen) promotes the floral transition. Both are targets of the miR172 regulatory circuit [3]. At the fourth resolution level, the timing signal (*FT*) and the organ identity specification (*AP2*) contribute equivalently to the ball classification.

In a search space of 6.2×10^9 permutations, the only degeneracies correspond to known biological antagonisms. These are also the only positions where permutation freedom exists. This structure is read from the dynamics of f once the Mendoza–Álvarez–Buylla model fixes the network; optimizing μ_E uses no further hierarchical annotations (Remark 2.1).

Remark 3.3 (Sensitivity and landscape of π^*). *Apart from the two symmetry pairs, every adjacent-position swap in π^* increases μ_E :*

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 is steepest at the top of the hierarchy: placing any gene other than UFO at position 0 (with the remaining positions re-optimized) increases μ_E by 15% to 145%, depending on which gene is forced into the leading slot (File S1, Supplementary Material). Swapping across the meristem-identity/organ-specification boundary (TFL1 $\leftrightarrow$ AP2) costs +22.9%.

Among 2,000 uniformly random orderings, the minimum observed μ_E was 39,472 (47% above μ_E^*), with mean $87,361 \pm 9,268$ (z-score of π^* : -6.5). No random ordering came within 47% of the optimum, confirming that π^* occupies an isolated extremum in the ordering landscape.

How the hierarchy influences the results. The hierarchy is not an input: once f is fixed, π^* is chosen from transition data alone, and balls are induced by π . The μ -optimal orderings identify the scales at which the dynamics is most stable under this partition-based criterion, and hence which perturbations count as local versus global at a given resolution (Section 2). Under π^* , at $n=3$ the partition is by UFO, EMF1, and LFY; refinement to $n=4$ adds TFL1.

Construction of Hierarchical Approximations. The n -approximations $F_0, F_1, \dots, F_4$ are constructed following the gluing procedure of the companion paper [21]; explicit formulas are given in Supplementary Appendix A (PDF). At each level n , F_n maps balls $\mathcal{B}_{1/2^n}(m)$ to balls whose radius determines the ball classification ($\Lambda_{n,m}$). The key features are:

- *Level $n=2$:* All four balls are expanding ($\Lambda > 1$), contributing the largest single-level share of μ .
- *Level $n=3$:* Four balls are contracting (including $\mathcal{B}_{1/8}(4)$, $\mathcal{B}_{1/8}(5)$) and four are expanding (including $\mathcal{B}_{1/8}(2)$, $\mathcal{B}_{1/8}(3)$).
- *Level $n=4$:* Four of the 16 balls are expanding ($\mathcal{B}_{1/16}(2)$, $\mathcal{B}_{1/16}(3)$, $\mathcal{B}_{1/16}(10)$, $\mathcal{B}_{1/16}(11)$) and 12 are contracting; in particular $\mathcal{B}_{1/16}(4)$ and $\mathcal{B}_{1/16}(5)$ (floral organ basins) remain contracting. All four expanding balls descend from $\mathcal{B}_{1/8}(2)$ and $\mathcal{B}_{1/8}(3)$ (expanding at level 3); both sub-balls of each parent remain expanding. In contrast, $\mathcal{B}_{1/8}(6)$ and $\mathcal{B}_{1/8}(7)$ (expanding at level 3) have both sub-balls contracting at level 4—an ultrametric filtration effect in which expansion at one scale *disappears* at the next (Remarks 3.5 and 3.6).

Stability classification by resolution level. We compute the classification of all 2^n balls at each resolution level n according to the criteria in Section 2. Table 4 shows the complete classification for all resolution levels.

Remark 3.4 (Interpretation of Level Counts). *Three patterns emerge from Table 4:*

1. Isometric balls (I) are present at levels $n = 1, 6, 7, 9, 11$. Whether $|I(n)| > 0$ at a given level depends on the network and ordering; it is not an invariant feature and must be checked for each case.

Table 4

Classification of p -adic balls by resolution level for *A. thaliana* under π^* . $E(n)$ = expanding balls (quasi-repelling at scale n ; $\Lambda > 1$; contribute to μ), $A(n)$ = contracting balls ($\Lambda < 1$; stable regions), $I(n)$ = scale-isometric balls ($\Lambda = 1$; scale-preserving). The contribution to μ is $E(n) \cdot 2^{13-n}$.

n	Total balls	$E(n)$	$A(n)$	$I(n)$	Contribution to μ
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					$\mu = 26,776$

2. Contraction dominates at fine scales. At $n=12$, all 4,096 balls are contracting; at $n=11$, non-expanding balls outnumber expanding ones by $\approx 17:1$. The expanding contribution is concentrated at coarse levels: $n=2$ alone contributes 8,192 to μ ($\approx 30.6\%$ of the total), where the partition is determined by UFO and EMF1. The weight 2^{N-n} ensures that these coarse-level instabilities carry the largest penalty, in accord with the hierarchical organization of floral development [2, 10, 18].

Remark 3.5 (Ultrametric Filtration). The transition from level $n=3$ to $n=4$ illustrates two complementary filtration effects. First, contraction refines progressively: $\mathcal{B}_{1/16}(4) \subsetneq \mathcal{B}_{1/8}(4)$ and $\mathcal{B}_{1/16}(5) \subsetneq \mathcal{B}_{1/8}(5)$ remain contracting at level 4 (with $M=6$, larger than $M=4$ at level 3), confirming stability of floral organ basins at finer scales. Second, expansion can either persist or disappear: both sub-balls of $\mathcal{B}_{1/8}(2)$ and $\mathcal{B}_{1/8}(3)$ remain expanding at level 4 ($\mathcal{B}_{1/16}(2)$, $\mathcal{B}_{1/16}(10)$, $\mathcal{B}_{1/16}(3)$, $\mathcal{B}_{1/16}(11)$; all with $M=3$), whereas the expansion in $\mathcal{B}_{1/8}(6)$ and $\mathcal{B}_{1/8}(7)$ vanishes entirely at level 4 (Remark 3.6). This selective disappearance of instability through nested scales is a signature of the ultrametric structure that the p -adic framework provides.

Remark 3.6. The dynamics of f is 3-expanding in $\mathcal{B}_{1/8}(6)$; however, f is not 4-expanding in $\mathcal{B}_{1/16}(6)$ or in $\mathcal{B}_{1/16}(14)$. This implies that, although the 3-level analysis detects expansion (and hence potential repelling behavior) in $\mathcal{B}_{1/8}(6)$, the responsible configurations need not lie in $\mathcal{B}_{1/16}(6) \cup \mathcal{B}_{1/16}(14)$.

Consequently, certain dynamical features visible at one resolution level may not be captured by any single sub-ball at the next level, highlighting the importance of analyzing multiple resolution levels to reconstruct the full dynamical structure.

Identification of Attractors and Repellers. The fixed points of this network correspond to the floral organ identities identified by Mendoza and Álvarez-Buylla [18] and validated by Espinosa-Soto et al. [10] and Pérez-Buendía et al. [20]. 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.

Under π^* , f has exactly 10 fixed points distributed across four balls at level $n=4$, corresponding to the floral organ identities predicted by the ABC model [10, 18]. Configuration numbers are under the ordering π^* ; Table 5 lists both m_A (under π^*) and the natural indexing.

The six floral organ fixed points lie in contracting balls $\mathcal{B}_{1/16}(4)$ (prefix (0, 0, 1, 0): LFY=1, EMF1=0, TFL1=0) and $\mathcal{B}_{1/16}(5)$ (prefix (1, 0, 1, 0): UFO=1, LFY=1); the four inflorescence fixed points lie in expanding balls $\mathcal{B}_{1/16}(10)$ and $\mathcal{B}_{1/16}(11)$. (Stamen configurations have AP2=1 in the encoding, a property of the Boolean model [10, 18], 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 ($\mathcal{B}_{1/16}(4)$ and $\mathcal{B}_{1/16}(5)$), jointly attracting 95% of the state

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

Config (m_A)	Config (orig.)	Ball	Class	Type
10	264	$B_{1/16}(10)$	Expanding	Inflorescence
2,058	328	$B_{1/16}(10)$	Expanding	Inflorescence
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	Inflorescence
2,059	4,424	$B_{1/16}(11)$	Expanding	Inflorescence
1,973	7,734	$B_{1/16}(5)$	Contracting	Petal (UFO)
6,005	7,859	$B_{1/16}(5)$	Contracting	Stamen (UFO)

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 [10, 13, 18, 28]; the same distinction is visible from the transition table through the $A/E/I$ stratification without asserting new biology.

Remark 3.7. In the Boolean model of [10, 18], 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, compatible with the interpretation of π^* as a regulatory hierarchy.

Basin analysis, mutant frequency, and noise sensitivity are developed in Section 3.

The polynomial interpolation analysis for *A. thaliana* (per-gene update polynomials, dependency structure, and polynomial influence ranking) is in Appendix D. The key comparison between polynomial influence and π^* positions is discussed in Section 7.

Basins of attraction and scale-resolved summaries. At level $n=4$ under π^* (Section 3), contracting basins $B_{1/16}(4)$ and $B_{1/16}(5)$ contain floral-organ fates and expanding basins $B_{1/16}(10)$ and $B_{1/16}(11)$ contain inflorescence states. The network has no limit cycles: all 8,192 configurations converge to one of the 10 fixed points. Basin sizes are obtained by forward iteration on the full transition graph (Appendix A). The table below summarizes basin sizes and $A/E/I$ class; it supports geometric interpretation of the synchronous dynamics, not calibrated in vivo frequencies of mutation or environmental noise without extra modeling beyond this Boolean map.

Ball	Class	Fixed points	Basin size	Biological identity
$B_{1/16}(4)$	A (contr.)	4	3,888 (47.5%)	Stamen-UFO, Carpel, Petal-UFO, Sepal
$B_{1/16}(5)$	A (contr.)	2	3,888 (47.5%)	Stamen, Petal
$B_{1/16}(10)$	E (exp.)	2	208 (2.5%)	Inflorescence (EMF1+TFL1 on)
$B_{1/16}(11)$	E (exp.)	2	208 (2.5%)	Inflorescence (LFY off)

The two dominant attractors are Stamen-UFO (3,064 states, 37.4%) and Carpel (2,970 states, 36.3%), which together capture 73.6% of the state space. Sepal and Petal-UFO attract 9.9% and 10.1% respectively; the four inflorescence states collectively attract only 5.1% (see Appendix A for full basin data). The contracting balls $B_{1/16}(4)$ and $B_{1/16}(5)$ each attract 3,888 states (47.5%), capturing 95.0% of the state space.

Contracting basins correspond to stable fates (perturbations return to the fixed point; canalization [28]); expanding basins to transient states that flow toward stable fates. The scale-resolved partition is a discrete counterpart to the epigenetic landscape [13].

Biological reading (qualitative). Larger contracting basins are consistent with stronger canalization toward the corresponding fixed points under synchronous dynamics; expanding basins mark states that flow toward committed fates. This qualitative picture aligns with the known strong phenotypes of master-regulator perturbations versus more localized downstream effects [6, 10, 23, 25, 29], but the table entries are basin-level diagnostics of the Boolean model, not population-level mutant-rate or noise estimates.

Hierarchical Graph Visualization and Interpretation. The hierarchical structure is visualized by a directed graph whose vertices are p -adic balls at levels $n = 0, \dots, n_{\max}$ and whose edges combine (i) ball-to-ball transitions induced by f at each scale and (ii) refinement/inclusion between consecutive levels; vertices are colored by the A/E/I classification from Section 2. Figure 2 was generated with the script `generate_hierarchical_graph.py` supplied in the Supplementary Material, using the same synchronous transition table as the main analysis.

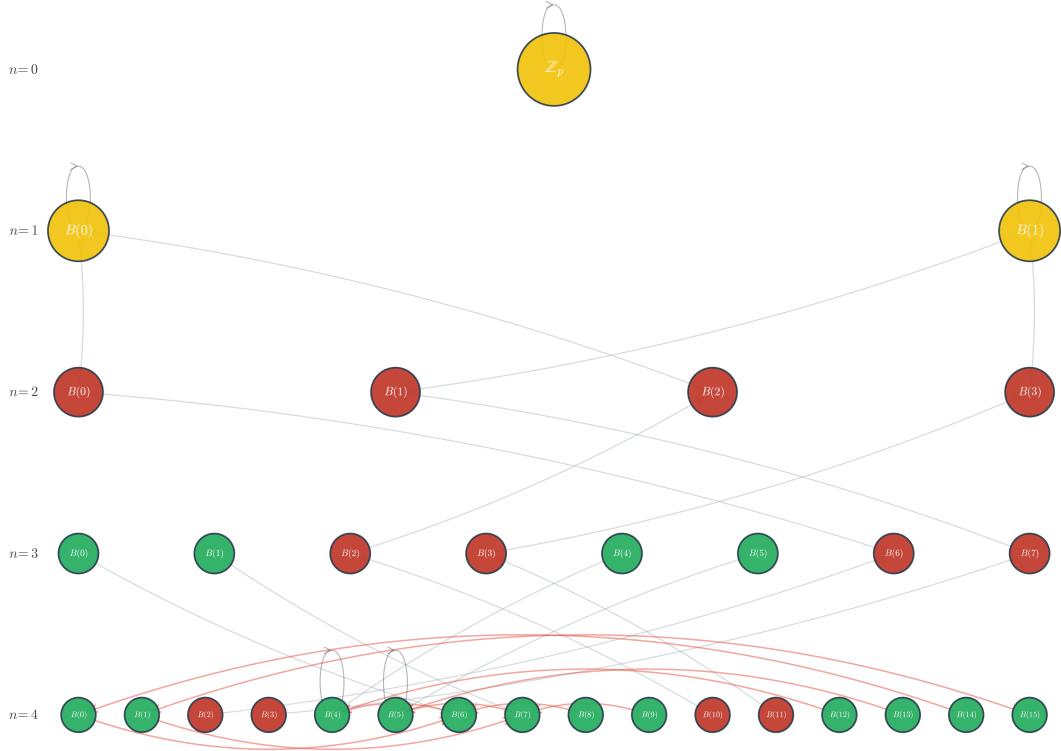


Figure 2: Hierarchical p -adic ball structure for *A. thaliana*. Green: contracting. Red: expanding. Yellow: isometric. Levels $n = 0$ to $n = 4$. Generated with `generate_hierarchical_graph.py` from the synchronous transition table (Supplementary Material).

Interpretation. In the *A. thaliana* graph (Figure 2), green (contracting) vertices $B_{1/16}(4)$ and $B_{1/16}(5)$ attract edges from many other vertices, a signature of canalization [28]. Red (expanding) vertices $B_{1/16}(10)$ and $B_{1/16}(11)$ emit edges toward green vertices, corresponding to the transient nature of inflorescence states. The graph can be constructed at any level n ; for *A. thaliana*, $n=4$ captures the hierarchy determined by the four master regulators in π^* .

The same visualization approach applies to the other networks; the corresponding hierarchical graphs are shown in Section 6.

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 [11]. We use the 8-gene synchronous Boolean model of Huang et al. [14] (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 [5, 14].

We consider the Boolean model of Huang et al. [14] 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 $w_{ii} = -1$ on **cul-1**, **fzr-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.

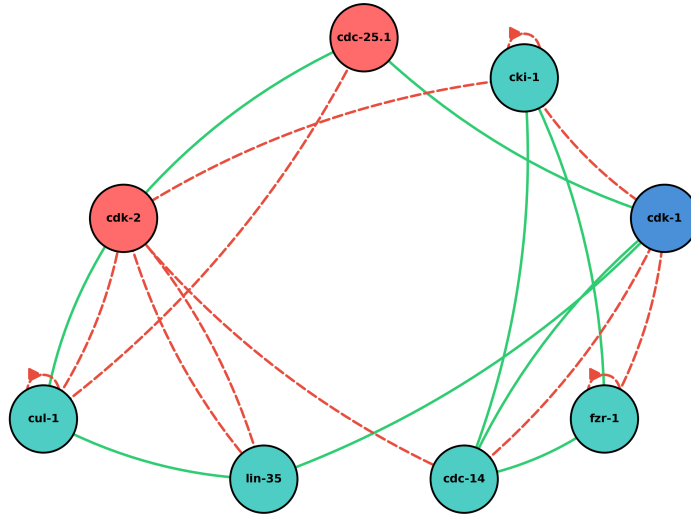


Figure 3: *C. elegans* cell cycle network ($N=8$). Red: G1/S regulators (cdc-25.1, cdk-2). Blue: M-phase regulator (cdk-1). Teal: other cell cycle genes. Green arrows: activation. Red dashed: repression. Network topology and Boolean rules from [14]; figure generated with NetworkX.

The network (Figure 3) 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,

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

Under π^* , $(\mu_E, \mu_A, \mu_I) = (956, 392, 444)$. 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

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

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.

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.

Construction of Hierarchical Approximations. The n -approximations $F_0, F_1, \dots, F_7$ are constructed following the gluing procedure of the companion paper [21]. Explicit formulas for the ball maps under the Huang-aligned transition table and π^* are generated by the Supplementary script `compute_Fn_celegans.py`; levels $n = 5, 6, 7$ are omitted in the main text for brevity.

Summary of $E(n)$, $A(n)$, $I(n)$ across all levels. The profiles for levels $n = 1, \dots, 7$ 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^* .

*Fixed Points in the *C. elegans* Cell Cycle Network.* Under synchronous updating with the transition table generated from Huang et al.’s rule (equation (1) with threshold tie-breaking $\sum_j w_{ij} S_j = \theta_i \Rightarrow S_i(t+1) = S_i(t)$), the network has **5** fixed points (Table 7), matching the five attractors reported by Huang et al. [14]. There are no limit cycles under synchronous updating. Configuration 0 (all genes off) is a fixed point by the threshold rule. Configurations 0, 224, and 232 share the four-character prefix IAI (levels 1–4) in the E/A/I sequence under π^* but have distinct full sequences and biological roles (Table 7); the comparative table in Section 6 groups them under one coarse prefix row.

Note: The binary column lists bits in Huang node order and matches **Config (orig.)**; the π^* -reordered integer encoding is m_A .

The classification sequences group into three dynamical patterns:

- *Fully expanding at all levels (EEEEEEE):* Configuration 226 is expanding at every scale. This fixed point lies in a small basin (Appendix B) but is unconstrained by the hierarchical partition at each n .
- *Shared IAI pattern:* Configurations 224 and 232 share the same E/A/I sequence despite different natural encodings; 232 carries most of the state space (basin size 219/256).

Table 7

Fixed points in the *C. elegans* network under $\pi^* = [1, 0, 2, 3, 6, 5, 4, 7]$ (cdc-25.1 $\succ$ cdk-2 $\succ$ $\dots$ $\succ$ cki-1). Config (orig.): Huang Table 2 indexing (bit 0 = cdk-2, ..., bit 7 = cki-1); m_A : encoding under π^* ; Sequence: E/A/I at levels $n=1, \dots, 7$.

Config (orig.)	m_A	Binary (Huang index; LSB=gene 0)	Sequence ($n=1-7$)	Biological type
0	0	00000000	IAIEEA	Quiescent (all off)
224	176	11100000	IAIAAA	Intermediate
226	177	11100010	EEEEEE	Fully expanding profile
232	184	11101000	IAIAAA	Largest-basin attractor (Huang)
234	185	11101010	EEEIAIA	Intermediate

- *Mixed IA/EE and EEEIAIA profiles*: Configuration 0 (quiescent) shows isometry and expansion at coarse scales then contraction and expansion alternating at finer scales (IAIEEA). Configuration 234 (EEEIAIA) mixes expansion-dominated prefixes with alternating coarse classifications.

The polynomial interpolation analysis for *C. elegans* (update polynomials and influence ranking) is in Appendix D. Full fixed-point data, classification sequences, stability profiles, and basin sizes are collected in Appendix B.

Predictions from the ordering. Under π^* , cdc-25.1 is at the coarsest scale and cki-1 at the finest, with cdc-14 and fzf-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 [5, 11].

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) [9, 15]. 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 [9, 15], 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 4 shows the six-gene regulatory graph and the gene labels used below to specify the synchronous Boolean map f .

The network (Figure 4) has $N=6$ genes, $|\mathcal{C}| = 2^6 = 64$ configurations. The Boolean update rules, based on [9, 15], are:

- *Hb* is activated by Bcd (high in anterior) and repressed by Kr and Gt. Boolean rule: $Hb(t+1) = Bcd \wedge \neg Kr \wedge \neg Gt$.
- *Kr* is activated by Bcd and Hb, and repressed by Kni and Gt. Boolean rule: $Kr(t+1) = (Bcd \wedge Hb) \wedge \neg Kni \wedge \neg Gt$.
- *Kni* is activated by Cad (high in posterior) and repressed by Kr and Gt. Boolean rule: $Kni(t+1) = Cad \wedge \neg Kr \wedge \neg Gt$.
- *Gt* is activated by Cad and repressed by Kr and Kni. Boolean rule: $Gt(t+1) = Cad \wedge \neg Kr \wedge \neg Kni$.
- *Bcd* and *Cad* are maternal morphogens. In our Boolean model, we treat them as self-maintaining (their state persists), though in reality they form spatial gradients.

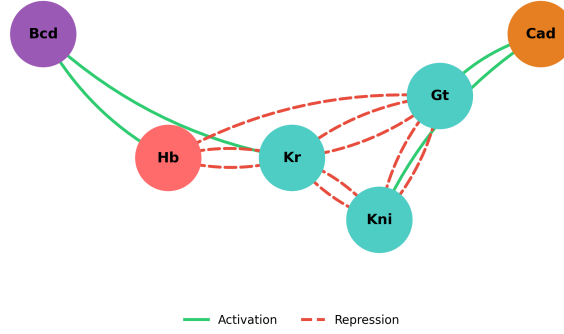


Figure 4: *D. melanogaster* gap gene network (6 genes). Purple/Orange: morphogens. Red: Hunchback. Teal: gap genes. Green arrows: activation. Red dashed: repression. Network topology from [15]; Boolean rules from [9]; discrete Boolean conventions follow [1]; figure generated with NetworkX.

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

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 exchangeable pairs: $\{Bcd, Cad\}$ at positions 0–1 and $\{Kni, Gt\}$ at positions 2–3 (see Supplementary Material and Section 6). *Throughout this paper*, $(x_0, \dots, x_5)$ refers to the *one* ordering written explicitly below; Section D.3 and the polynomial figures use these labels only (the other three minimizers relabel x_0 – x_3 by those swaps, leaving μ unchanged):

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

These four orderings also maximize μ_A (Section 6): minimizing expansion and maximizing contraction select the same hierarchy, a coincidence that sets *Drosophila* apart from the other two networks and implies that a single ordering captures both stability criteria without compromise.

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 [9, 15].

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 .

Construction of Hierarchical Approximations.

Construction of F_0 . From the transition map $f : \mathcal{C} \rightarrow \mathcal{C}$, any 0-approximation F_0 should satisfy $F_0(\mathcal{B}_1(0)) = \mathcal{B}_1(0)$; we choose $F_0(z) = z$.

Construction of F_1 . Any 1-approximation F_1 should satisfy:

$$\begin{aligned} F_1(\mathcal{B}_{1/2}(0)) &= \mathcal{B}_{1/2}(0), \\ F_1(\mathcal{B}_{1/2}(1)) &= \mathcal{B}_{1/2}(1). \end{aligned} \tag{6}$$

Both balls are 1-isometric: the Bicoid partition preserves scale.

Construction of F_2 . Any 2-approximation F_2 should satisfy:

$$\begin{aligned} F_2(\mathcal{B}_{1/4}(0)) &= \mathcal{B}_{1/64}(0), & F_2(\mathcal{B}_{1/4}(1)) &= \mathcal{B}_{1/16}(1), \\ F_2(\mathcal{B}_{1/4}(2)) &= \mathcal{B}_{1/4}(2), & F_2(\mathcal{B}_{1/4}(3)) &= \mathcal{B}_{1/4}(3). \end{aligned} \tag{7}$$

Balls 0 and 1 (both with Cad inactive: $Bcd=0/Cad=0$ and $Bcd=1/Cad=0$, respectively) are contracting (A), since without Cad the posterior gap genes cannot activate and the dynamics collapses to few states; balls 2 and 3 (both with Cad active) are isometric (I), since Kni and Gt compete symmetrically under Cad stimulation, preserving the ball scale.

Construction of F_3 . Any 3-approximation F_3 should satisfy:

$$\begin{aligned} F_3(\mathcal{B}_{1/8}(0)) &= \mathcal{B}_{1/64}(0), & F_3(\mathcal{B}_{1/8}(1)) &= \mathcal{B}_{1/16}(1), \\ F_3(\mathcal{B}_{1/8}(2)) &\text{ splits (image covers more than one } n\text{-ball)}, & F_3(\mathcal{B}_{1/8}(3)) &\text{ splits}, \\ F_3(\mathcal{B}_{1/8}(4)) &= \mathcal{B}_{1/64}(0), & F_3(\mathcal{B}_{1/8}(5)) &= \mathcal{B}_{1/32}(1), \\ F_3(\mathcal{B}_{1/8}(6)) &\text{ splits}, & F_3(\mathcal{B}_{1/8}(7)) &\text{ splits}. \end{aligned} \tag{8}$$

The refinement to level 3 breaks the isometric structure present at level 2: balls 0, 1, 4, 5 (all with Cad inactive, $x_1=0$) remain contracting, since without Cad the posterior genes Kni and Gt cannot activate; balls 2, 3, 6, 7 (all with Cad active, $x_1=1$) become expanding, since Kni and Gt compete under Cad stimulation and the image splits across balls with different x_2 (Kni) values. This yields $E(3) = 4$, $A(3) = 4$, $I(3) = 0$. The F_4 and F_5 approximations are in Appendix C.

Gap gene domains and ball classification. Contracting balls correspond to stable gap gene expression domains along the anterior–posterior axis; expanding balls mark boundary regions where conflicting gap gene signals compete. At coarse scales ($n \leq 2$), morphogen identity suffices to resolve domain membership and all balls are non-expanding; expansion appears at $n=3$ when Kni and Gt compete under Cad stimulation.

Fixed Points in the Drosophila Gap Gene Network. Under π^* , the 6-gene Boolean model has 5 fixed points representing stable gap gene expression domains along the anterior–posterior axis, and 3 limit cycles (lengths 4, 2, 2):

1. *Config 0* (natural indexing; $m_A = 0$): All genes inactive (unpatterned/basal state).
2. *Config 40* ($m_A = 6$): Posterior domain (Kni=1, Cad=1; Bcd=0, Hb=0, Kr=0, Gt=0).
3. *Config 43* ($m_A = 39$): Anterior–posterior overlap (Bcd=1, Hb=1, Kni=1, Cad=1).
4. *Config 48* ($m_A = 10$): Posterior domain with Giant (Gt=1, Cad=1).

5. *Config 49* ($m_A = 11$): Posterior variant (Bcd=1, Gt=1, Cad=1).

The classification sequences (levels $n=1, \dots, 5$) are:

- Config 0: **IAAAA**—isometric at level 1 (the morphogen partition preserves scale), then fully contracting. The empty configuration is maximally stable: all gap genes silenced converge trivially.
- Configs 40, 43, 48, 49: **IIEEA**—isometric at the coarsest two levels (morphogen partition), expanding at the intermediate levels (3–4) where Kni/Gt variation introduces conflicting signals, and contracting at the finest level (5) where Hb–Kr states resolve. The expansion at levels 3–4 stems from the mutual repression among gap genes [15]: at the resolution where the posterior gap genes (Kni, Gt) differ, the dynamics has not yet distinguished between alternative domain states. At level 5, when the full Kr–Hb state is specified, the dynamics contracts to fixed points—as expected once all six genes are specified and the gap gene domains fully determined.

The three limit cycles are:

- *4-cycle* {1, 3, 7, 5} (basin: 16 states): oscillation among Bcd-on anterior states (Bcd $\rightarrow$ Bcd+Hb $\rightarrow$ Bcd+Hb+Kr $\rightarrow$ Bcd+Kr $\rightarrow$ Bcd), capturing the dynamical instability of the Hb–Kr competition in the anterior domain.
- *2-cycles* {32, 56} and {33, 59} (basin: 12 states each): posterior oscillations between bare Cad and full Kni+Gt activation, mirroring the transient mutual repression between Kni and Gt before one dominates.

The transition table is in the Supplementary Material (`transitions_drosophila.csv`).

Basin Analysis for Drosophila. Under π^* , the global stability profile shows contracting dynamics dominating at every scale: $E(n) = 0$ for $n \leq 2$, and at the finest level ($n=5$) 28 of 32 balls are contracting (only 4 expanding). The five fixed points all reside in contracting regions at levels 1–2, in accord with gap gene expression domains being stable once the morphogen partition is established.

The basin of the basal state (config 0) contains 16 states (25% of the state space), all with Cad=0 and no active gap genes. The basin of the 4-cycle oscillation ({1, 3, 7, 5}) also contains 16 states, all with Bcd=1 and Cad=0—the anterior compartment where Hb and Kr compete without resolution. The four non-basal fixed points (40, 43, 48, 49) have small basins (2 states each), since most configurations with Cad active flow first into the 2-cycles before reaching a stable domain.

Mutations in master regulators (Bicoid, Caudal) would be expected to affect the morphogen partition at $n=1$, causing global patterning defects. Perturbations of downstream gap genes (Krüppel, Hunchback at positions 4–5) affect only the finest-scale classification, compatible with their role in refining domain boundaries rather than establishing them [9, 15]. The polynomial interpolation analysis for *D. melanogaster* (update polynomials, isomorphic rules for Kni/Gt, and dependency graph) is in Appendix D. Fixed-point data, classification sequences, stability profiles, and limit cycles are in Appendix C.

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 their update rules are symmetric under swapping Kni and Gt; the optimization therefore treats the pair as equivalent at these positions. In embryos, *kni* and *gt* phenotypes are related but not identical [15]; the symmetry is a property of the Boolean rules used here, not a claim of biological interchangeability.

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 $\leftrightarrow$ 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 $\leftrightarrow$ Cad swap). 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 $\leftrightarrow$ Gt exchangeability, present in $\pi^*(f)$, breaks under iteration—only Bcd $\leftrightarrow$ 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*. Throughout, 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* [10, 18]; cdc-25.1, cdk-2, lin-35 in *C. elegans* [5, 14]; Bicoid, Caudal in *Drosophila* [9, 15]—in line with their disproportionate influence on dynamics.
2. *Stability classification*: differentiated/stable states lie in contracting balls; transitional states lie in expanding balls. This matches the view of stable fates as attractors and developmental intermediates as transient in Boolean and landscape models [10, 13, 18, 26, 28].
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 [14, 15, 18].

Traditional methods identify master regulators via structural metrics (degree centrality, betweenness, K-core, minimum dominating set) or prior knowledge [12, 27]. 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 [16]. Table 10 summarizes the reported orderings and stability measures for the three networks.

Table 9

Position in π^* vs. network centrality for *A. thaliana*. Spearman ρ between π^* and each metric (last row) does not exceed $|\rho| = 0.36$, confirming that μ and topology-based rankings measure different properties.

Gene	Pos(π^*)	Out-deg	In-deg	Total	Betw.	Close.
UFO	0	1	0	1	0.000	0.316
EMF1	1	4	1	5	0.083	0.611
LFY	2	7	4	11	0.310	0.733
TFL1	3	5	4	9	0.138	0.611
AP2	4	2	1	3	0.000	0.458
FT	5	1	1	2	0.002	0.440
AG	6	5	8	13	0.195	0.478
AP1	7	6	6	12	0.441	0.647
SEP	8	4	1	5	0.008	0.393
AP3	9	3	7	10	0.101	0.423
PI	10	3	6	9	0.018	0.423
WUS	11	2	3	5	0.000	0.333
FUL	12	1	2	3	0.000	0.458
$\rho(\pi^*, \cdot)$	—	0.18	-0.36	-0.10	0.12	0.30

Table 10

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 over the full set of $13!$ orderings, without explicit full enumeration. The four optima differ only by the EMF1/LFY and AP2/FT transpositions; see Observation 3.1.

Quantitative comparison with network centrality. To make the distinction between μ and topology-based methods precise, Table 9 compares the position of each gene in π^* with standard centrality metrics² computed on the regulatory digraph of *A. thaliana* (Figure 1; edge $j \rightarrow i$ if gene j appears as an input in the Boolean update function of gene i). The Spearman correlation between π^* and each metric is weak: $\rho = 0.18$ (out-degree), $\rho = 0.12$ (betweenness), $\rho = 0.30$ (closeness), with none exceeding $|\rho| = 0.36$ (Table 9). The case of UFO is illustrative: it has the lowest out-degree (1) and betweenness (0.000) among all 13 genes, yet occupies position 0 in π^* . Its update rule $T_{\text{UFO}} = x_{\text{UFO}}$ makes it a self-maintaining binary switch that partitions the full state space (2^{13} configurations) into two halves with distinct basin structures; the $n=1$ level carries weight $p^{N-1} = 4096$ in μ , so this partition dominates the stability landscape. Conversely, LFY has the highest out-degree (7) and second-highest betweenness (0.310) but sits at position 2: high direct regulatory influence does not imply coarse-scale stability control. These results show that μ captures a dynamics-based hierarchy fundamentally different from network topology (script: Supplementary `compute_centrality_comparison.py`).

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? The measure μ decomposes into μ_E (expanding balls), μ_A (contracting), and μ_I (isometric); minimizing μ is minimizing μ_E . We answer this by computing (μ_E, μ_A, μ_I) for all orderings—explicit exhaustive search in the small networks and branch-and-bound global certification for *A. thaliana* (Appendix A). Table 11 summarizes the number of μ_E -optima, μ_A -optima, and their intersection. In *Drosophila*, all μ_E -optimal orderings are also μ_A -optimal, whereas the converse fails: there are 8 μ_A -optima and 4 μ_E -optima, with $|\text{Opt}_{\mu_E}(f) \cap \text{Opt}_{\mu_A}(f)| = 4$ (equivalently $\text{Opt}_{\mu_E}(f) \subsetneq \text{Opt}_{\mu_A}(f)$). In *C. elegans*, there is a unique μ_E -minimum and four μ_A -maxima, with empty intersection (trade-off); in *A.*

²On the regulatory digraph G : *out-degree* of node j is the number of genes directly regulated by j ; *in-degree* is the number of its regulators; *betweenness centrality* is the fraction of shortest paths between all pairs that pass through j ; *closeness centrality* measures how close j is to all other nodes (inverse mean shortest-path distance). See [19] for definitions and properties.

Table 11Two-criteria analysis: orderings minimizing μ_E vs. maximizing μ_A . “Intersection” = number of orderings achieving both.

Network	μ_E -optima	μ_A -optima	Intersection	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 (certified)	40 certified (10 orbits $\times C_2 \times C_2$)	0	Mild trade-off (max. $\rho = 0.978$ vs. π_E^*)

thaliana, branch-and-bound certifies 40 μ_A -maximizing orderings—organized in 10 orbits under the same $C_2 \times C_2$ symmetry as the μ_E -optima—and none coincides with the 4 μ_E -optima (mild trade-off: Spearman ρ between π_E^* and a μ_A -optimal representative ranges from 0.72 to 0.978, with 0.978 for the closest orbit; Appendix A).

When every μ_E -optimal ordering is also μ_A -optimal (*Drosophila*), each of the four μ_E -minima lies in the μ_A -optimal set (while four additional μ_A -optima are not μ_E -optimal). In *Drosophila*, Bicoid and Caudal—the primary determinants of anterior-posterior patterning [9, 15]—lead in all four μ_E -optima, each of which is also μ_A -optimal. In *C. elegans*, the unique μ_E -minimizer has $\mu_E^* = 956$ and $(\mu_E, \mu_A, \mu_I) = (956, 392, 444)$, while any μ_A -maximizer achieves $\mu_A^* = 440$ (four orderings) at higher μ_E —for example $[0, 2, 3, 1, 6, 5, 7, 4]$ yields $(1088, 440, 264)$ —so the two criteria are in conflict [5, 14]. In *A. thaliana*, no ordering achieves both optima simultaneously. The μ_E -optimal orderings have $(\mu_E, \mu_A, \mu_I) = (26,776, 53,880, 17,648)$, while the μ_A -optimal orderings achieve $\mu_A^* = 54,560$ with μ_E ranging from 26,976 (best) to 35,168 (worst). The trade-off is concentrated in WUS: its promotion from position 11 to position 9 yields $\Delta\mu_A = +680$ at a cost of $\Delta\mu_E = +200$, together with $\Delta\mu_I = -880$. The closest μ_A -optimal ordering to π_E^* retains UFO in position 0 and agrees with π_E^* on the leading master regulators up to the AP2/FT swap, differing in the tail block (WUS vs. AP3/PI); its Spearman correlation with π_E^* is $\rho = 0.978$ (Supplementary `compute_spearman_rho_muE_muA.py`). Across all 10 orbits, ρ ranges from 0.72 (UFO displaced to position 9) to 0.978, so the two hierarchies are structurally close despite the disjoint intersection [10, 18].

Spearman Rank Correlation: Validation and Robustness. The Spearman rank correlation coefficient ρ measures the monotonic agreement between two rankings of the same set of items [24]. Given N genes, assign to each gene g a *model rank* $r_{\text{mod}}(g)$ (its position $1, \dots, N$ in the μ_E -minimizing ordering) and a *biological rank* $r_{\text{bio}}(g)$ (its position in the literature-based hierarchy, e.g. the order in the validation tables). Then

$$\rho = 1 - \frac{6 \sum_g (r_{\text{bio}}(g) - r_{\text{mod}}(g))^2}{N(N^2 - 1)}. \quad (9)$$

In this work, ρ serves both validation and robustness. For validation, once the published Boolean model fixes f , the optimal ordering is obtained by minimizing μ over permutations with no external regulatory rankings or hierarchical role labels in the optimization (Remark 2.1); comparing that ordering *a posteriori* with the literature-based hierarchy via ρ tests whether the dynamics-based ranking is consistent with experimentally grounded regulatory importance. A high ρ (ideally $\rho = 1$) indicates strong agreement. For robustness, when several orderings share the same minimal μ_E (e.g. 4 for *D. melanogaster*), we compute ρ for each: if all are high, validation is stable across equivalent optima; if only a subset attains $\rho = 1$, the biological table selects one convention among symmetry-equivalent mathematical solutions.

For the three networks analyzed here, we compare the model-induced ranking (position of each gene in the μ_E -minimizing ordering) with the biological hierarchy in Tables 3, 6, and 8. For *A. thaliana*, the reported π^* matches the eight-position biological hierarchy exactly (Spearman $\rho = 1.0$; exact permutation test over all $8! = 40,320$ permutations of the biological ranking gives $p = 2.48 \times 10^{-5}$; see Supplementary `permutation_test_spearman.py`). See also Supplementary `compute_official_optima_athaliana.py`. For *C. elegans*, exhaustive search returns a unique μ_E -optimum; comparing π^* with Table 6 gives $\rho = 1.0$ on all eight genes, with the same exact permutation p -value $1/8! = 2.48 \times 10^{-5}$. For *D. melanogaster*, the four μ_E -optima (Section 5) yield Spearman $\rho \in \{1, 33/35, 31/35\} \approx \{1, 0.943, 0.886\}$ against the fixed ranks in Table 8 (one optimum with $\rho = 1$, two with $\rho = 33/35$, one with $\rho = 31/35$). Under the exact permutation test over $6! = 720$ rank permutations, upper-tail probabilities $P(\rho \geq \rho_{\text{obs}})$ are $1/720$, $6/720$, and $12/720$ for $\rho_{\text{obs}} \in \{1, 33/35, 31/35\}$ respectively.

Table 12

Representative orderings: μ_E -minimizing (π_E^*) and closest/farthest μ_A -maximizing orbits. Differences with π_E^* are **bold**. The full set of 10 μ_A -optimal orbit representatives appears in Appendix A, Table 17.

Criterion	μ_E	μ_A	μ_I	ρ	Ordering
π_E^* (min μ_E)	26,776	53,880	17,648	—	UFO, EMF1, LFY, TFL1, AP2, FT, AG, AP1, SEP, AP3, PI, WUS, FUL
π_A^* orbit 1	26,976	54,560	16,768	0.978	UFO, EMF1, LFY, TFL1, FT , AP2 , AG, AP1, SEP, WUS , AP3 , PI , FUL
π_A^* orbit 10	35,168	54,560	8,576	0.973	EMF1, UFO , LFY, TFL1, FT , AP2 , AG, AP1, SEP, WUS , AP3 , PI , FUL

One can also compute Spearman ρ between a μ_E -optimal ordering and a μ_A -optimal ordering (instead of versus the biological table). This measures how much the hierarchy changes when the criterion is switched from minimizing μ_E to maximizing μ_A : $\rho = 1$ means the two criteria yield the same ordering (or one from the same equivalence class); $\rho < 1$ quantifies the trade-off. For *A. thaliana*, $\rho(\pi_E^*, \pi_A^*)$ ranges from 0.72 to 0.978 across the 10 μ_A -optimal orbits (Supplementary `compute_spearman_rho_muE_muA.py`); the closest orbit retains UFO at position 0, and the farthest displaces it to position 9. The script `report_mu_E_mu_A_mu_I_celegans_drosophila.py` reports “ $\rho(\pi_E^*, \pi_A^*)$ ” for the exhaustive networks (*C. elegans*, *Drosophila*).

Biological significance per network. D. melanogaster. All four μ_E -optima are also μ_A -optima (with four additional μ_A -only optima), and Bicoid and Caudal—the primary morphogens establishing the anterior-posterior axis [9, 15]—occupy the leading positions across those four orderings. That the same four permutations simultaneously minimize μ_E and maximize μ_A may reflect the fact that anterior-posterior patterning is governed by a consistent ordering of regulatory importance on this model.

C. elegans. With the transition table aligned to Huang et al. [14] (Tables 1–2), exhaustive search gives a unique μ_E -minimum ($\mu_E^* = 956$) and four μ_A -maxima ($\mu_A^* = 440$), with empty intersection. The μ_E -optimal hierarchy begins with *cdc-25.1* and *cdk-2* at the coarsest scales [5, 14]; the μ_A -optima favor a different arrangement (Appendix B), illustrating that “most contracting” and “least expanding” hierarchies need not coincide on the same Boolean network.

A. thaliana. Branch-and-bound certifies exactly 4 μ_E -optima ($\mu_E^* = 26,776$) and 40 μ_A -optima ($\mu_A^* = 54,560$), with empty intersection. The 4 μ_E -optima differ only by $\text{EMF1} \leftrightarrow \text{LFY}$ and $\text{AP2} \leftrightarrow \text{FT}$ —pairs corresponding to known Polycomb-mediated [17] and miR172-mediated [3] antagonisms (Remark 3.2). The 40 μ_A -optima organize into 10 orbits under the same $C_2 \times C_2$ symmetry (EMF1/LFY , AP2/FT swaps preserve μ_A). Across these orbits, UFO moves from position 0 to position 9 and the late block (SEP through FUL) is rearranged relative to π_E^* ; the closest μ_A -optimal representative to π_E^* still has UFO at position 0, agrees with π_E^* on the leading master regulators up to the AP2/FT swap, and differs in the tail (WUS vs. AP3/PI) and in μ -scores (Table 12 and Appendix A, Table 17). The structural trade-off discussed in the appendix is localized: for that closest pair, promoting WUS from position 11 (π_E^*) to position 9 (π_A^*) yields $\Delta\mu_A = +680$ at a cost of $\Delta\mu_E = +200$. That the first eight positions of π_E^* match the biological hierarchy of master regulators [10, 18], whereas μ_A -optimal orderings need not, suggests that in this framework the experimentally observed hierarchy aligns with minimizing expansion rather than maximizing contraction.

Basin and Polynomial Comparison.

- *A. thaliana*: contracting balls $B_{1/16}(4)$ and $B_{1/16}(5)$ capture 95.0% of the state space (3,888 states each); the two dominant individual attractors are Stamen-UFO (37.4%) and Carpel (36.3%). Inflorescence states collectively attract only 5.1% [10, 18].
- *C. elegans*: the largest basin belongs to config 232 (219 states, 85.55%), followed by config 234 (16, 6.25%), config 224 (12, 4.69%), config 226 (5, 1.95%), and config 0 (4, 1.56%). No limit cycles; all 256 configurations converge to one of 5 fixed points (percentages: $100 \times \text{basin}/256$, two decimal places; Table 19).
- *Drosophila*: contracting basins for stable gap gene domains [9, 15]; the sequence comparison below is for fixed-point attractors (limit cycles are analyzed via f^2 and f^4 in Section 5; Appendix C).

Table 13

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)
	EEII...	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 E/A/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.

The polynomial interpolation analysis (for the three developmental networks) reveals complementary dependency structures:

- *A. thaliana*: master regulators (UFO, LFY, EMF1, TFL1) often have simpler polynomial forms or act as direct regulators of many downstream genes [10, 18]. Integrator genes (AP1, AG) have complex polynomial forms, corresponding to their role in processing diverse information [2].
- *C. elegans*: under π^* , the algebraic normal forms T_i are computed in $\mathbb{F}_2$ with variables $(x_0, \dots, x_7)$ indexed along π^* (Appendix D). The hub cdk-2 (x_1) and cdc-14 (x_4) attain the largest I_{poly} (4 targets each), while cdc-25.1 at the coarsest scale has a sparse update ($T_0 = x_0 + x_0x_2$) [5, 14].
- *Drosophila*: maternal morphogens (Bicoid, Caudal) appear in other genes' polynomials as activators, while gap genes show mutual repression patterns [9, 15].

The polynomial dependency rankings agree with π^* on top-ranked genes in each network and diverge at intermediate positions, precisely where the multiscale weighting of μ adds information beyond direct syntactic dependence in the update rules [14, 15, 18].

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 13 and Figure 5 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.

Three observations stand out.

A. thaliana. Inflorescence is a developmental waypoint that progresses toward organ formation; mature floral organs are stable endpoints [10, 18]. The scale-dependent classification reflects this distinction: the 6 organ fixed points share the coarse-scale prefix IEAA, while the 4 inflorescence states carry IEEE. The full 12-level sequences (Appendix A) reveal 5 distinct profiles; all six organ types share the prefix IEAAAA (levels 1–6) and diverge at finer scales, encoding organ-specific structure not captured by flat Boolean analysis.

C. elegans. The Huang Boolean model exhibits quiescent, intermediate, and high-activity fixed attractors [11, 14]. Under the Huang-aligned dynamics and π^* , the full sequences ($n=1, \dots, 7$) yield EEEEEEE for config 226 (the high-CDK-activity attractor in Huang's labeling), shared IAIIAAA for configs 224 and 232, IAIIIEEA for config 0, and EEIIAIA for config 234. The dominant attractor (232, 85.55% basin) shares its

coarse-scale prefix with a smaller-basin state (224), while the quiescent state (0) differs at fine scales despite the same four-character prefix IAI (full sequences in Appendix B).

D. melanogaster. Gap gene domains are established by maternal morphogens [9, 15]. Table 13 and Figure 5 list the *four-letter prefixes* (levels $n = 1, \dots, 4$) for fixed-point attractors only; the same gap-gene Boolean model also admits limit cycles, classified via f^2 and f^4 in Section 5 (Appendix C for cycle data). For $N=6$, the full classification words have length $N-1 = 5$: the basal fixed point has IAAAA (isometric at level 1, then contracting), and the four gap-domain fixed points share IIEEA—*isometric at levels 1–2* (the morphogen partition preserves scale), *expanding at levels 3–4* (Kni/Gt variation creates conflicting signals), and contracting at level 5 once Kr and Hb are specified. The expansion at intermediate scales reflects the mutual repression among gap genes [15], which creates instability at the resolution where competing domain assignments coexist.

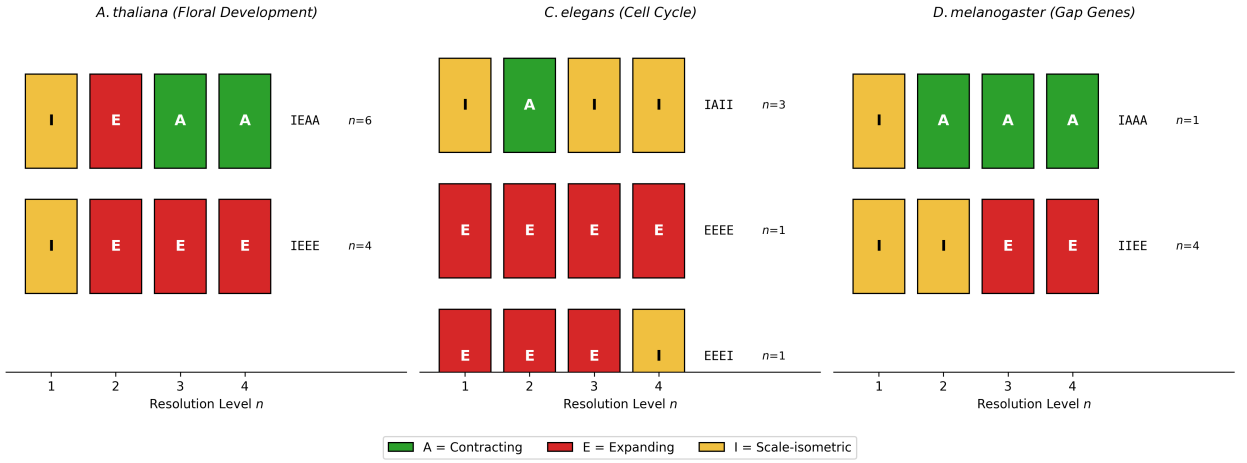


Figure 5: Classification sequences across resolution levels for the three networks.

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 13 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. Figures 6 and 7 show the hierarchical p -adic ball structure for the *C. elegans* cell cycle and *Drosophila* gap gene networks (the *A. thaliana* graph is in Section 3). 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. IAI..., 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, a sufficient commutation criterion (Remark 6.4), and the compensatory cancellation mechanism (Observation 6.5).

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 $\pi \in \text{Opt}(f)$.*

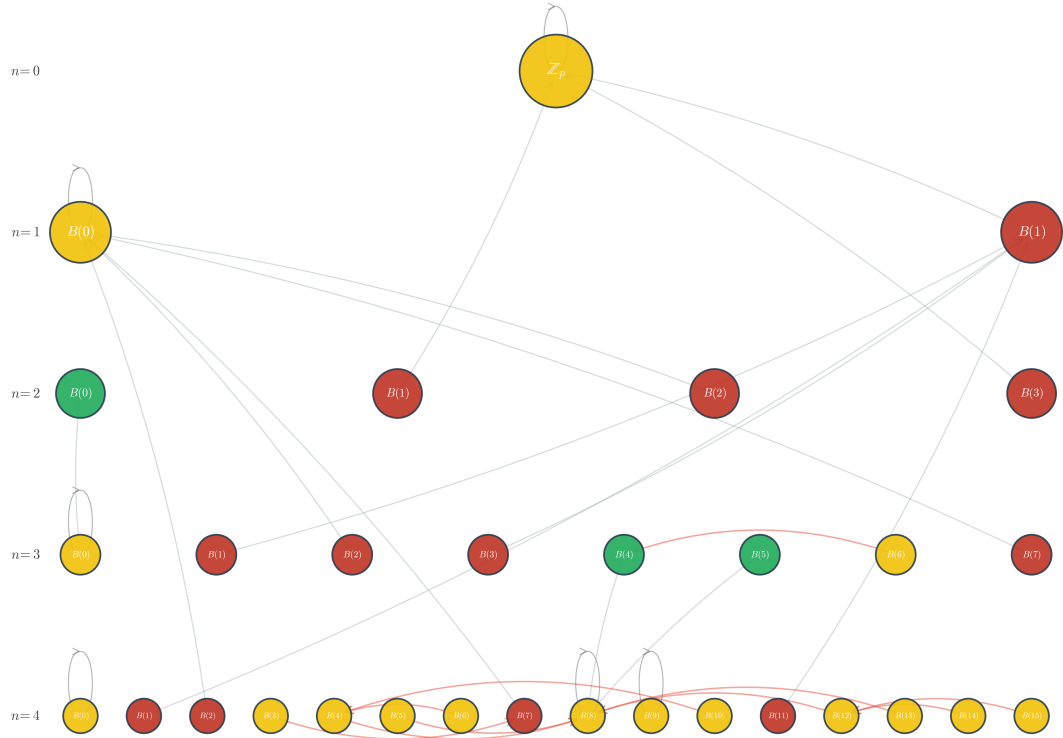


Figure 6: Hierarchical p -adic ball structure for the *C. elegans* cell cycle network (Huang-aligned dynamics). Green: contracting balls. Red: expanding balls. Yellow: isometric balls. Coarse scales follow π^* (cdc-25.1, cdk-2). Regenerate via `figures/regenerate_figures.py` (hierarchical-graph script in Supplementary Material).

Table 14

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^* .

Network	N	$ \text{Opt}(f) $	$G(f)$	r	Exchangeable pairs
<i>A. thaliana</i>	13	4	$C_2 \times C_2$	$11/13 \approx 0.85$	EMF1 / LFY (1–2), AP2 / FT (4–5)
<i>C. elegans</i>	8	1	$\{e\}$	1	—
<i>D. melanogaster</i>	6	4	$C_2 \times C_2$	$2/6 \approx 0.33$	Bcd / Cad (0–1), Kni / Gt (2–3)

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 . 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 14 summarizes the symmetry structure of $\text{Opt}(f)$ for the three networks.*

The rigidity index $r(f) := |\{k : k \text{ is rigid}\}|/N$ measures how sharply μ_E determines the hierarchy. *A. thaliana* has $r \approx 0.85$ (11 of 13 positions rigid); *C. elegans* has $r = 1$ (unique μ_E -minimum, all positions rigid). At the other extreme, *Drosophila* has $r \approx 0.33$: only the last two positions (Krüppel, Hunchback) are rigid, while both morphogen positions and both posterior gap gene positions are exchangeable.

The exchangeable pairs in each network correspond to genes with symmetric functional roles:

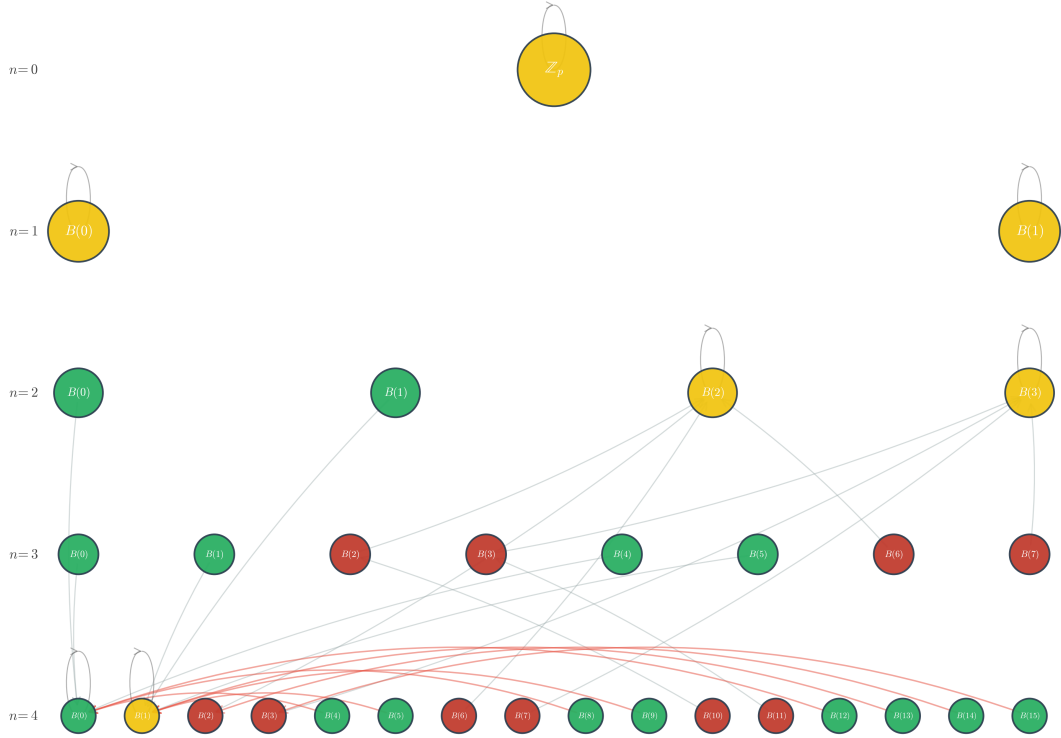


Figure 7: Hierarchical p -adic ball structure for the *Drosophila* gap-gene network.

Green: contracting balls (stable gap-gene expression). Red: expanding balls (transitional patterns). Yellow: isometric balls. Regenerate via `figures/regenerate_figures.py` (calls the `hierarchical-graph` script in Supplementary Material).

1. *Bcd/Cad* (*D. melanogaster*, positions 0–1): both are self-maintaining maternal morphogens ($T_{\text{Bcd}} = x_{\text{Bcd}}$, $T_{\text{Cad}} = x_{\text{Cad}}$) providing the two complementary spatial coordinates (anterior vs. posterior) [9]. At coarse scales the μ_E landscape places the two morphogens symmetrically: they contribute equally to stability in the ordering analysis.
2. *Kni/Gt* (*D. melanogaster*, positions 2–3): both are posterior gap genes activated exclusively by Caudal and subject to mutual repression, with isomorphic update rules $T_{\text{Kni}} = x_1(1+x_3)(1+x_4)$, $T_{\text{Gt}} = x_1(1+x_2)(1+x_4)$ (Section D.3). Their exchangeability is a direct consequence of this functional symmetry.
3. *C. elegans* (Huang-aligned table): $\text{Opt}(f)$ is a singleton, so there are no exchangeable pairs; the unique π^* places *cdc-25.1* and *cdk-2* at the coarsest scales [11, 14].
4. *EMF1/LFY* and *AP2/FT* (*A. thaliana*, positions 1–2 and 4–5): Polycomb-mediated repression vs. floral activation, and the miR172 regulatory circuit linking AP2 and FT, respectively (Observation 3.1).

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.

The rigidity index r is highest in *C. elegans* (no degeneracy in $\text{Opt}(f)$) and still high in *A. thaliana*, and lowest in *Drosophila*, which has two equivalent morphogens and two equivalent posterior gap genes. Each exchangeable pair corresponds to genes that the experimental literature identifies as performing symmetric or antagonistic roles at the same regulatory tier [9, 11, 25, 29]. Once f is fixed from the published Boolean model,

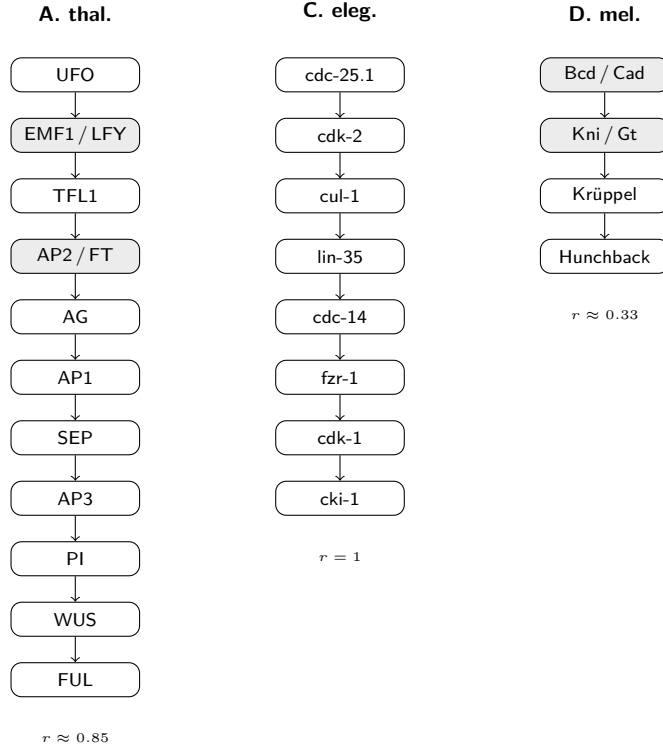


Figure 8: Equivalence-class chains for the three networks. Each node is a rigid gene position (white) or an exchangeable pair (shaded). Arrows indicate the total order inherited from π^* . The rigidity index $r = |\{k : k \text{ rigid}\}|/N$ (fraction of positions fixed across all optima) decreases from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

degeneracies in the μ_E landscape are determined by the transition structure of that map—without additional hierarchical labels in the optimization (Remark 2.1); aligning those pairs with documented regulatory roles is a consistency check with the literature, not an input to the search. These three networks are detailed case studies; a general classification of $\text{Opt}(f)$ for arbitrary Boolean maps remains open (Question 6.6).

Remark 6.4 (Commutation as a sufficient condition). *If $f \circ \tau_{ij} = \tau_{ij} \circ f$ (where τ_{ij} swaps coordinates i and j) and π places i, j at adjacent positions $k, k+1$, then $E^\pi(n) = E^{\pi'}(n)$ for every n : 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. Global commutation is thus a natural sufficient condition for μ -equivalence of adjacent transpositions.*

Observation 6.5 (Compensatory mechanism in the absence of commutation). *No exchangeable pair in the three networks satisfies $f \circ \tau_{ij} = \tau_{ij} \circ f$ (failures range from 8/64 configurations for Kni / Gt in *Drosophila* to 5,120/8,192 for $EMF1 / LFY$ in *A. thaliana*). Nevertheless, $E^\pi(n) = E^{\pi'}(n)$ holds at every level—not because individual M -values are preserved, but because a compensatory cancellation operates: the number of balls reclassified from E to non- E by the swap exactly matches the number reclassified from non- E to E . For instance, swapping Kni and Gt in *Drosophila* at level 3 moves one ball from E to non- E and one from non- E to E , preserving $E(3) = 4$. This compensatory balance holds at every level in every network (verified computationally for all 4 exchangeable pairs across the three networks: Bcd / Cad and Kni / Gt in *Drosophila*; $EMF1 / LFY$ and $AP2 / FT$ in *A. thaliana*).*

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 (Remark 6.4) but not necessary (Observation 6.5). 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

Two features of the three applications call for further discussion: why the μ -based hierarchy and the polynomial influence ranking agree at the top but diverge at intermediate positions, and why minimizing expansion is a more selective criterion than maximizing contraction.

Remark 7.1 (Theorem, computation, correlation, and interpretation). *For clarity: (1) Formal proofs, invariance of the gluing construction, and explicit bounds for the approximations F_n to the interpreter ϕ (Proposition 3.11) are in the companion paper [21] (see also Section 2); this article does not reprove them and only computes μ , orderings, and classifications from the transition table of each fixed published model. (2) Computed here: μ , π^* , classification sequences, level counts. (3) External correlation: comparison of π^* with biologically identified master regulators (Spearman, tables). (4) Interpretation: e.g. the heuristic that the hierarchy favors avoidance of expansion over maximization of contraction.*

Table 9 ($\rho \leq 0.36$ on *A. thaliana*) quantifies how π^* departs from graph-theoretic rankings; Remark 6.1 places this alongside I_{poly} and Boolean sensitivity. Boolean analysis on the digraph treats nodes uniformly, whereas the p -adic embedding distinguishes configurations that share wiring but differ in hierarchical image structure. Epigenetic landscape approaches [8, 20] pursue related goals; here the $A/E/I$ word is computed from the transition table once f is fixed. Gene-level localization of expansion, explicit level weights in μ , and the degeneracy structure of $\text{Opt}(f)$ appear in Sections 3–5 and Section 6 (Remark 3.5 for *A. thaliana*).

Polynomial influence vs. μ -hierarchy. The polynomial influence score I_{poly} measures direct syntactic reach in the update rules; 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, Appendix D). 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 $s_j = |\{c : f(c) \neq f(c \oplus e_j)\}|/|C|$ is a transition-table baseline; Table 15 compares it with π^* .

In *Drosophila*, Caudal is self-maintaining ($T_1 = x_1$), so s is low (0.125) while π^* still places it second: coarse-scale partition weight dominates local sensitivity. The same contrast between single-flip tests and multiscale structure underlies Remark 6.1. Mutual-information orderings and eigenvector centrality remain as further baselines for future work.

The role of UFO across optimization criteria. UFO's update polynomial is $T_0 = x_0$ (self-maintaining, no direct targets). 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 direct regulation. More generally, input-like genes that determine coarse-scale basin membership contribute more to μ than genes with many direct targets, because the $n=1$ partition carries weight p^{N-1} .

The μ_A -optimal set gives a complementary perspective on UFO. Among the 10 certified orbits of μ_A -maximizers, UFO occupies every position from 0 to 9 (Table 17). Despite this variability, μ_A remains constant at 54,560. However, the cost in μ_E is not monotone: UFO at position 0 yields the lowest μ_E (26,976), positions 3–5 produce an intermediate plateau (29,024), and position 1 gives the highest μ_E (35,168). This non-monotone pattern indicates that UFO's contribution to contraction is position-independent (it creates equally contracting partitions wherever it sits), whereas its contribution to expansion is sensitive to its interaction with the master-regulator block. The fact that all orderings in all 10 orbits share the positions of EMF1, LFY, TFL1, and the tail (WUS, AP3, PI, FUL) regardless of UFO's location is consistent with this

Table 15

Spearman correlation between Boolean sensitivity ranking and π^* position. Sensitivity is computed from the full transition table (see Supplementary script `compute_boolean_sensitivity.py`).

Network	$\rho(\text{sens.}, \pi^*)$	p -value	Key divergence
<i>A. thaliana</i>	0.64	0.018	Top 4 genes tied at $s=1.0$; π^* breaks the tie
<i>C. elegans</i>	0.67	0.071	cdc-25.1: π^* pos. 1, sens. rank 5
<i>D. melanogaster</i>	0.14	0.787	Cad: π^* pos. 2, lowest sensitivity ($s=0.125$)

Note. Spearman correlation compares two different constructions: Boolean sensitivity ranks local one-step perturbation effects, whereas π^* ranks genes by multiscale ball expansion under the p -adic embedding. A low ρ therefore does not invalidate π^* ; it signals that coarse-scale partition roles need not align with single-flip sensitivity (see the *D. melanogaster* discussion below).

reading: the regulatory backbone is rigid; UFO's effect on contraction does not depend on its position among them.

Landscape steepness and functional symmetries. For *A. thaliana*, the landscape around π^* is steep (Remark 3.3; numerical sampling summarized in Section 8). Section 6 shows that residual degeneracies in $\text{Opt}(f)$ are governed by exchangeable pairs with documented antagonisms. Global commutation is sufficient for μ -equivalence of adjacent swaps (Remark 6.4); Observation 6.5 records a compensatory cancellation that preserves $E(n)$ without commutation. A general classification of $\text{Opt}(f)$ and a characterization of compensatory cancellation remain open (Question 6.6).

Minimizing expansion vs. maximizing contraction (interpretive reading). In the *A. thaliana* model analyzed here, maximizing contraction (μ_A) admits 40 certified optima, whereas minimizing expansion (μ_E) selects only 4; both criteria use the same ball decomposition and Haar weights, so the tenfold asymmetry in degeneracy is intrinsic to the pair (μ_E, μ_A) on this map. We read this contrast as suggesting that, *within these Boolean dynamics*, avoiding coarse-scale expansion is a more selective constraint than maximizing contraction—a heuristic about the model, not a claim about selection, evolutionary optimality, or fitness [10, 18].

Scope and extensions. All analyses use *synchronous* updates on a single published Boolean model per organism; asynchronous or stochastic update schemes, or alternative tie-breaking at threshold in the *C. elegans* map, would define a different map f and are left for future comparison. Claims are therefore *consistency checks* against literature hierarchies and perturbation biology, not independent experimental proof of those hierarchies (Remark 2.1). Changing the prime p , the gene order used to build the p -adic encoding, or the Boolean map f would generally change π^* and the ball decomposition; we fix $p = 2$ and the published f for each organism as in the companion [21] and the Methods. The p -adic topology on the embedded state space is ultrametric: every triangle is isosceles, and distances take the discrete values p^{-k} . The consistency of π^* with documented master-regulator rankings across three organisms indicates that the tree-like branching of cell-fate decisions carries an ultrametric structure at the coarse scales captured here. Whether this extends to networks with extensive cross-talk between tiers, or to primes $p > 2$, remains to be tested.

Branch-and-bound certifies the global optimum for *A. thaliana* ($N=13$, ≈ 29 hours on a single core). The bound propagation was tightest in the first five positions, where a single misplacement of UFO sufficed to prune over 90% of the search tree; the remaining eight positions required finer distinctions and account for most of the wall time. Tighter bounds, parallel search, or ML-assisted pruning could extend certification to $N \approx 18$ –20; for larger networks, genetic algorithm heuristics (validated here against the certified optimum) provide a practical alternative.

Beyond scalability, the framework extends conceptually. The $A/E/I$ classification applies to limit cycles via the iterated map f^k : applying the same ball decomposition to f^2 and f^4 for *Drosophila* classifies 2-periodic and 4-periodic orbits as fixed points of the iterate (Section 5). Expansion drops by 78% (μ_E^* : $64 \rightarrow 14$), the optimal ordering shifts Krüppel ahead of Knirps—reflecting the stabilization of the Hb–Kr oscillation—and only the Bcd $\leftrightarrow$ Cad exchangeability survives.

The three networks studied here have well-established regulatory hierarchies, which makes them suitable for validating the framework against known biology. A natural next step is a prospective application: computing π^* for a network whose hierarchy has not been established and deriving testable predictions from

the ordering. Integration with experimental data (ChIP-seq, perturbation assays) and additional baselines (mutual-information orderings, eigenvector centrality) are natural next steps.

8. Conclusions

The hierarchical p -adic framework computes a dynamics-based gene ordering from the transition structure of a published Boolean GRN. Once the network and its update rules fix the map f (Remark 2.1), the permutation minimizing μ_E —equivalently minimizing μ in our decomposition $\mu = \mu_E + \mu_A + \mu_I$ —is obtained without external regulatory rankings or hierarchical annotations in the optimization; we then compare that ordering *a posteriori* with experimentally documented regulatory roles. Applied to three developmental models, leading positions are consistent with literature-identified master regulators, and the scale-dependent classification separates fixed points corresponding to distinct biological states. The contribution is a dynamics-based ranking together with a multiscale view of stability that refines flat Boolean attractor analysis.

For *A. thaliana*, branch-and-bound certifies the global μ_E -minimum over $13!$ orderings; the representative π^* matches the eight-position biological table ($\rho = 1.0$), and floral-organ fates occupy contracting basins covering 95% of the state space (Section 3). There are four μ_E -optima and 40 μ_A -optima (no overlap); Spearman comparisons between π_E^* and μ_A -optimal representatives, the WUS trade-off, orbit structure, and the random-ordering landscape are detailed in Section 6 and Section 7 (Observations 3.1, Remark 3.3).

For *C. elegans* and *D. melanogaster*, exhaustive search also certifies optimal orderings: under the Huang-aligned cell-cycle model, *cdc-25.1* and *cdk-2* lead the unique μ_E -minimizer in *C. elegans*, while morphogens lead in *Drosophila*. In both cases, classification sequences distinguish biologically meaningful dynamical regimes (Sections 4, 5). For *Drosophila*, the *A/E/I* machinery extends to periodic orbits via the iterated maps f^2 and f^4 : expansion drops by 78%, the 2-cycle states split into two classification types (one matching the gap-domain fixed points, one fully non-expanding), and the 4-cycle states contract at morphogen scales while remaining isometric at gap-gene scales (Section 5).

The comparative analysis (Section 6) shows that $G(f)$ varies across networks—trivial for *C. elegans* ($r = 1$), $C_2 \times C_2$ for *A. thaliana* ($r \approx 0.85$) and *Drosophila* ($r \approx 0.33$); where exchangeable pairs exist, they align with documented symmetries or antagonisms. Global commutation ($f \circ \tau_{ij} = \tau_{ij} \circ f$) is sufficient for μ -equivalence of adjacent transpositions, but *none* of the observed exchangeable pairs satisfies it; instead, a compensatory cancellation of ball reclassifications preserves $E(n)$ at every level (Remark 6.4, Observation 6.5). A general characterization of this mechanism is left open (Question 6.6).

Basin analysis and polynomial interpolation (Section 3, Appendix D) complement the main ordering analysis: I_{poly} agrees with π^* on top-ranked genes and diverges where the multi-scale μ -hierarchy adds information (Section 7, Remark 6.1). For $N > 13$, heuristic search remains necessary but can be validated by constrained enumeration; extension to contexts such as epithelial–mesenchymal transition is future work. Across the three Boolean models, the μ_E/μ_A trade-off varies from incompatibility (*C. elegans*) to coincidence (*Drosophila*), so the relationship between the two scores encodes model structure beyond either score alone. Low Spearman correlation between Boolean sensitivity and π^* in *D. melanogaster* (Table 15) matches the same distinction between local perturbation tests and multiscale ball expansion (Section 7).

Appendix A Supplementary Data: *A. thaliana* Floral Development Network

Fixed points, classification sequences, and basin sizes. Table 16 lists the 10 fixed points of the floral development network under π^* (Section 3), their full classification sequences at levels $n=1, \dots, 12$, and basin sizes (number of states that converge to each fixed point under iteration of f). The network has no limit cycles: all 8,192 configurations converge to one of the 10 fixed points.

Classification sequence analysis. Five distinct classification profiles emerge from the 12-level sequences:

1. **IEEEEEAAAIA** (4 inflorescence states): expanding at all coarse and intermediate scales ($n=2-7$), contracting only at fine scales ($n=8-10$), with an isometric level at $n=11$. The persistent expansion mirrors the transient nature of inflorescence.

Table 16Fixed points of the *A. thaliana* network under π^* , with full classification sequences and basin sizes.

Config	m_A	Ball $n=4$	Sequence ($n=1-12$)	Basin	Type
264	10	$B_{1/16}(10)$	IEEEEEAAAIA	136 (1.7%)	Inflorescence
328	2,058	$B_{1/16}(10)$	IEEEEEAAAIA	72 (0.9%)	Inflorescence
4,360	11	$B_{1/16}(11)$	IEEEEEAAAIA	136 (1.7%)	Inflorescence
4,424	2,059	$B_{1/16}(11)$	IEEEEEAAAIA	72 (0.9%)	Inflorescence
1,078	436	$B_{1/16}(4)$	IEAAAAIEEEEA	812 (9.9%)	Sepal
3,638	1,972	$B_{1/16}(4)$	IEAAAAIEEEEA	12 (0.1%)	Petal
1,715	5,492	$B_{1/16}(4)$	IEAAAAIEAIAA	2,970 (36.3%)	Carpel
3,763	6,004	$B_{1/16}(4)$	IEAAAAIEAIEA	94 (1.1%)	Stamen
7,734	1,973	$B_{1/16}(5)$	IEAAAAIEEEEA	824 (10.1%)	Petal (UFO)
7,859	6,005	$B_{1/16}(5)$	IEAAAAIEAAAA	3,064 (37.4%)	Stamen (UFO)

2. **IEAAAAIEEEEA** (sepal, petal, petal-UFO): contracting at coarse scales ($n=3-6$), isometric at $n=7$, then expanding at intermediate-to-fine scales ($n=8-11$), with final contraction at $n=12$. These organ types are stable at the level of master regulators but show internal rearrangement at fine scales.
3. **IEAAAAIEAIAA** (carpel): similar coarse-scale contraction, but with earlier return to contraction at fine scales ($n=9$, $n=11-12$). Carpel is the dominant attractor (basin: 36.3%), matching its role as the default organ identity when floral genes are active.
4. **IEAAAAIEAIEA** (stamen): differs from carpel only at level $n=10$ (expanding vs. contracting), capturing the fine-scale distinction between stamen and carpel identity (presence of AP3).
5. **IEAAAAIEAAAA** (stamen-UFO): the most contracting organ profile; fully contracting from $n=8$ onward. This fixed point has the largest basin (37.4%), compatible with UFO stabilizing organ identity.

All six organ fixed points share the coarse-scale prefix IEAAAA (levels 1–6), imposed by the stability of the master regulators (UFO, EMF1, LFY, TFL1, AP2, FT) in the leading positions of π^* . Divergence at levels 7–12 encodes organ-specific fine structure.

Basin structure. The two largest basins are Stamen-UFO (3,064 states, 37.4%) and Carpel (2,970 states, 36.3%), which together account for 73.6% of the state space. Inflorescence states collectively attract only 416 states (5.1%). This asymmetry agrees with the biological observation that floral organ fates act as strong attractors, while inflorescence is a transient developmental waypoint [10, 18].

At the ball level, $B_{1/16}(4)$ and $B_{1/16}(5)$ each attract 3,888 states (47.5%), while $B_{1/16}(10)$ and $B_{1/16}(11)$ each attract 208 states (2.5%). The contracting balls thus capture 95.0% of the state space.

Two-criteria analysis. Branch-and-bound search certifies exactly 4 μ_E -minimizing orderings (symmetry: interchanging EMF1 $\leftrightarrow$ LFY at positions 1–2 and AP2 $\leftrightarrow$ FT at positions 4–5); all yield $(\mu_E, \mu_A, \mu_I) = (26,776, 53,880, 17,648)$. A separate branch-and-bound run certifies exactly 40 μ_A -maximizing orderings ($\mu_A^* = 54,560$), organized into 10 orbits under the same $C_2 \times C_2$ symmetry. None of these 40 orderings coincides with the 4 μ_E -optima (intersection = 0). The Spearman rank correlation between the μ_E -optimal ordering and the closest μ_A -optimal ordering is $\rho = 0.978$; across all 10 orbits, ρ ranges from 0.72 to 0.978 (script: Supplementary `compute_spearman_rho_muE_muA.py`). The trade-off is localized: WUS moves from position 11 (π_E^*) to position 9 (π_A^*), gaining $\Delta\mu_A = +680$ at a cost of $\Delta\mu_E = +200$.

Table 17 lists the 10 μ_A -optimal orbit representatives, sorted by μ_E .

Table 17

All 10 μ_A -maximizing orbit representatives for *A. thaliana* ($\mu_A^* = 54,560$), sorted by ascending μ_E . Each orbit contains 4 orderings related by the EMF1/LFY and AP2/FT transpositions. UFO position and Spearman ρ with π_E^* are shown. The μ_E column reports scores on μ_A -maximizing orderings only; the global minimum of μ_E over all $13!$ orderings is 26 776 (four orderings; disjoint from this set—see “Two-criteria analysis” above). In particular, 26 976 is the smallest μ_E achievable subject to $\mu_A = \mu_A^*$, not the unconstrained global minimum.

Orbit	μ_E	μ_A	μ_I	UFO pos.	$\rho(\pi_E^*)$	Gene ordering
1	26,976	54,560	16,768	0	0.978	UFO, EMF1, LFY, TFL1, FT, AP2, AG, AP1, SEP, WUS, AP3, PI, FUL
2	28,000	54,560	15,744	9	0.720	EMF1, LFY, TFL1, FT, AP2, AG, AP1, SEP, WUS, UFO, AP3, PI, FUL
3	28,384	54,560	15,360	8	0.780	EMF1, LFY, TFL1, FT, AP2, AG, AP1, SEP, UFO, WUS, AP3, PI, FUL
4	28,448	54,560	15,296	7	0.824	EMF1, LFY, TFL1, FT, AP2, AG, AP1, UFO, SEP, WUS, AP3, PI, FUL
5	29,024	54,560	14,720	5	0.896	EMF1, LFY, TFL1, FT, AP2, UFO, AG, AP1, SEP, WUS, AP3, PI, FUL
6	29,024	54,560	14,720	4	0.918	EMF1, LFY, TFL1, FT, UFO, AP2, AG, AP1, SEP, WUS, AP3, PI, FUL
7	29,024	54,560	14,720	3	0.945	EMF1, LFY, TFL1, UFO, FT, AP2, AG, AP1, SEP, WUS, AP3, PI, FUL
8	30,048	54,560	13,696	6	0.863	EMF1, LFY, TFL1, FT, AP2, AG, UFO, AP1, SEP, WUS, AP3, PI, FUL
9	31,072	54,560	12,672	2	0.962	EMF1, LFY, UFO, TFL1, FT, AP2, AG, AP1, SEP, WUS, AP3, PI, FUL
10	35,168	54,560	8,576	1	0.973	EMF1, UFO, LFY, TFL1, FT, AP2, AG, AP1, SEP, WUS, AP3, PI, FUL

Appendix B Supplementary Data: *C. elegans* Cell Cycle Network

Fixed points and classification sequences. Table 18 lists the 5 fixed points of the *C. elegans* cell cycle network, their encoding m_A under the μ_E -optimal ordering $\pi^* = [1, 0, 2, 3, 6, 5, 4, 7]$ (*cdc-25.1* $\succ$ *cdk-2* $\succ$ *cul-1* $\succ$ *lin-35* $\succ$ *cdc-14* $\succ$ *fzr-1* $\succ$ *cdk-1* $\succ$ *cki-1*), and the full classification sequence (E/A/I at levels $n=1, \dots, 7$). The transition table is generated from Huang et al.’s equation (1) [14] with threshold tie-breaking (`build_celegans_transitions_huang.py`).

Note: The binary column lists bits in Huang node order and matches `Config (orig.)`; the π^* -reordered integer encoding is m_A .

Stability Measures. Under π^* , the stability decomposition is $(\mu_E, \mu_A, \mu_I) = (956, 392, 444)$. The profiles $E(n)$, $A(n)$, $I(n)$ for levels $n = 1, \dots, 7$ 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

Exhaustive search finds a unique μ_E -minimizer ($\mu_E^* = 956$) and four μ_A -maximizers ($\mu_A^* = 440$); the two sets are disjoint (intersection = 0). One μ_A -optimal ordering in gene-index notation is $[0, 2, 3, 1, 6, 5, 7, 4]$, with $(\mu_E, \mu_A, \mu_I) = (1088, 440, 264)$.

Basin sizes. The network has no limit cycles; all 256 configurations converge to one of the 5 fixed points. Basin sizes (number of states converging to each fixed point under iteration) are in Table 19.

Table 18Fixed points in the *C. elegans* network: natural indexing (Huang Table 2), m_A under π^* , and full classification sequence.

Config (orig.)	m_A	Binary (Huang index; LSB=gene 0)	Sequence ($n=1-7$)	Biological type
0	0	00000000	IAIIIEEA	Quiescent (all off)
224	176	11100000	IAIIAAA	Intermediate
226	177	11100010	EEEEEEEE	Fully expanding
232	184	11101000	IAIIAAA	Largest-basin attractor
234	185	11101010	EEEIAIA	Intermediate

Table 19Basin sizes for the *C. elegans* Boolean network (Huang-aligned dynamics).

Config	Type	Basin	(%)
232	Largest-basin attractor	219	85.55
234	Intermediate	16	6.25
224	Intermediate	12	4.69
226	Fully expanding profile	5	1.95
0	Quiescent (all off)	4	1.56

Percentages: each entry is $(100 \times \text{basin})/256$ rounded to two decimal places (half away from zero), e.g. $16/256 = 6.25\%$.

Appendix C Supplementary Data: *D. melanogaster* Gap Gene Network

Fixed points and classification sequences. Table 20 lists the five fixed points of the gap gene network under the optimal ordering $\pi^* = (\text{Bcd}, \text{Cad}, \text{Kni}, \text{Gt}, \text{Kr}, \text{Hb})$, with their m_A values and full classification sequences (levels $n=1, \dots, 5$).

Stability measures and profile. Under π^* : $\mu_E = 64$, $\mu_A = 156$, $\mu_I = 100$.

Limit cycles. The dynamics has 3 limit cycles:

- 4-cycle $\{1, 3, 7, 5\}$: anterior oscillation ($\text{Bcd} \rightarrow \text{Bcd}+\text{Hb} \rightarrow \text{Bcd}+\text{Hb}+\text{Kr} \rightarrow \text{Bcd}+\text{Kr}$); basin: 16 states.
- 2-cycle $\{32, 56\}$: $\text{Cad} \leftrightarrow \text{Cad}+\text{Kni}+\text{Gt}$; basin: 12 states.
- 2-cycle $\{33, 59\}$: $\text{Bcd}+\text{Cad} \leftrightarrow \text{Bcd}+\text{Hb}+\text{Kni}+\text{Gt}+\text{Cad}$; basin: 12 states.

Hierarchical approximations F_4 and F_5 . F_4 (16 balls): 9 contracting, 1 isometric, 6 expanding.

$$\begin{aligned}
F_4(\mathcal{B}_{1/16}(0)) &= \mathcal{B}_{1/64}(0), \quad F_4(\mathcal{B}_{1/16}(1)) = \mathcal{B}_{1/16}(1), \quad F_4(\mathcal{B}_{1/16}(2)) \text{ splits}, \quad F_4(\mathcal{B}_{1/16}(3)) \text{ splits}, \\
F_4(\mathcal{B}_{1/16}(4)) &= \mathcal{B}_{1/64}(0), \quad F_4(\mathcal{B}_{1/16}(5)) = \mathcal{B}_{1/32}(1), \quad F_4(\mathcal{B}_{1/16}(6)) \text{ splits}, \quad F_4(\mathcal{B}_{1/16}(7)) \text{ splits}, \\
F_4(\mathcal{B}_{1/16}(8)) &= \mathcal{B}_{1/64}(0), \quad F_4(\mathcal{B}_{1/16}(9)) = \mathcal{B}_{1/64}(1), \quad F_4(\mathcal{B}_{1/16}(10)) \text{ splits}, \quad F_4(\mathcal{B}_{1/16}(11)) \text{ splits}, \\
F_4(\mathcal{B}_{1/16}(12)) &= \mathcal{B}_{1/64}(0), \quad F_4(\mathcal{B}_{1/16}(13)) = \mathcal{B}_{1/64}(1), \quad F_4(\mathcal{B}_{1/16}(14)) = \mathcal{B}_{1/64}(2), \quad F_4(\mathcal{B}_{1/16}(15)) = \mathcal{B}_{1/64}(3).
\end{aligned}$$

F_5 (32 balls): 28 contracting, 4 expanding. At the finest resolution, 87.5% of balls are contracting, in accord with the well-defined spatial expression domains of gap genes.

Table 20Fixed points of the *D. melanogaster* gap gene network under π^* .

Config	m_A	Active genes	$n=1$	$n=2$	$n=3$	$n=4$	$n=5$	Interpretation
0	0	(none)	I	A	A	A	A	Basal (unpatterned)
40	6	Kni, Cad	I	I	E	E	A	Knirps posterior domain
43	39	Bcd, Hb, Kni, Cad	I	I	E	E	A	Anterior-posterior overlap
48	10	Gt, Cad	I	I	E	E	A	Giant posterior domain
49	11	Bcd, Gt, Cad	I	I	E	E	A	Posterior variant

Table 21Scale-dependent E/A/I profile for *D. melanogaster* under π^* .

n	$E(n)$	$A(n)$	$I(n)$	Weight p^{N-n}
1	0	0	2	32
2	0	2	2	16
3	4	4	0	8
4	6	9	1	4
5	4	28	0	2

Appendix D Polynomial Interpolation Analysis

Each gene update function $T_i : \mathbb{F}_2^N \rightarrow \mathbb{F}_2$ admits a unique canonical polynomial over $\mathbb{F}_2$. This appendix records a *structural local baseline* (direct dependencies, polynomial influence score I_{poly}) against which to compare the *global dynamical hierarchy* (μ, π^*) from the main text; it complements the four-way comparison in Remark 6.1. Agreement of I_{poly} with π^* on the top-ranked genes is consistent with the framework; disagreements in middle positions show precisely where the multi-scale perspective adds information (Section 7). Full per-gene decompositions for the three networks follow.

D.1 *A. thaliana*

Polynomial representation of update functions. Each gene x_i in the network has an update function $T_i : \mathbb{F}_2^N \rightarrow \mathbb{F}_2$ that determines its state at the next time step. Over $\mathbb{F}_2$, any such function admits a unique canonical polynomial representation:

$$T_i(x_0, \dots, x_{N-1}) = \sum_{e_0, \dots, e_{N-1} \in \{0,1\}} a_{e_0, \dots, e_{N-1}} x_0^{e_0} \cdots x_{N-1}^{e_{N-1}} \pmod{2}, \quad (10)$$

where the exponents $e_j \in \{0,1\}$ (since $x^2 \equiv x \pmod{2}$ in $\mathbb{F}_2$). This representation is unique and can be computed directly from the transition table using interpolation techniques.

For *A. thaliana* under π^* (Section 3, Table 3), the polynomial dependency structure is as follows. Throughout this subsection, the indices $x_0, \dots, x_{12}$ refer to the gene positions under π^* (Table 3), not the original indexing of Table 2:

Genes with simple dependencies (1–2 inputs):

- *UFO* (x_0): $T_0(x_0, \dots, x_{12}) = x_0 \pmod{2}$. UFO maintains its own state (autoregulation), acting as a cofactor for organ identity without being regulated by core floral genes.
- *SEP* (x_8): $T_8(x_0, \dots, x_{12}) = x_2 \pmod{2}$ (where x_2 corresponds to LFY in π^*). SEP is a direct effector of LFY with no additional dependencies.
- *FT* (x_5): $T_5(x_0, \dots, x_{12}) = 1 + x_1 \pmod{2}$ (where x_1 corresponds to EMF1). FT is activated by inactivation of EMF1 ($1 + x_1 = \text{not EMF1}$), in line with EMF1 as repressor of flowering.
- *AP2* (x_4): $T_4(x_0, \dots, x_{12}) = 1 + x_3 \pmod{2}$ (where x_3 corresponds to TFL1)
AP2 is activated by the inactivation of TFL1, mirroring the antagonistic relationship between AP2 and TFL1 in controlling meristem identity.

- *EMF1* (x_1): $T_1(x_0, \dots, x_{12}) = 1 + x_2 \pmod{2}$ (where x_2 corresponds to LFY)
EMF1 is activated by the inactivation of LFY, encoding the mutual antagonism between EMF1 (vegetative state) and LFY (floral identity).
- *LFY* (x_2): $T_2(x_0, \dots, x_{12}) = 1 + x_1 \cdot x_3 \pmod{2}$ (where x_1 and x_3 correspond to EMF1 and TFL1). LFY is repressed only when both EMF1 and TFL1 are active (NAND); it is active when at least one repressor is absent.

Genes with complex dependencies (3+ inputs):

- *FUL* (x_{12}): $T_{12}(x_0, \dots, x_{12}) = 1 + x_7 + x_3 + x_7 \cdot x_3 \pmod{2}$ (where x_7 and x_3 correspond to AP1 and TFL1). FUL depends on AP1 and TFL1 with a non-linear term $x_7 \cdot x_3$, indicating combinatorial regulation.
- *AP1* (x_7): The polynomial for AP1 contains multiple terms involving FT (x_5), LFY (x_2), AG (x_6), TFL1 (x_3), PI (x_{10}), and AP3 (x_9). AP1 integrates signals from timing, identity, and organ-specification genes, matching its role in floral meristem identity.
- *AG* (x_6): The polynomial for AG involves EMF1 (x_1), LFY (x_2), TFL1 (x_3), AP2 (x_4), AG (x_6), AP1 (x_7), SEP (x_8), and WUS (x_{11}). AG is a central integrator (eight inputs, including autoregulation) for carpel/stamen identity and stem-cell signals.
- *TFL1* (x_3): $T_3(x_0, \dots, x_{12}) = x_1 + x_1 \cdot x_2 + x_7 \cdot x_1 + x_7 \cdot x_1 \cdot x_2 \pmod{2}$ (where x_1 , x_2 , and x_7 correspond to EMF1, LFY, and AP1). TFL1 integrates EMF1, LFY, and AP1 with multiple interaction terms, as a master regulator of vegetative, floral, and meristem pathways.
- *WUS* (x_{11}): $T_{11}(x_0, \dots, x_{12}) = x_{11} + x_{11} \cdot x_6 \cdot x_8 \pmod{2}$ (where x_{11} , x_6 , and x_8 correspond to WUS, AG, and SEP). WUS has autoregulation (x_{11}) and depends on AG and SEP (interaction term), for stem-cell maintenance and organ-identity integration.

Gene dependency network. The polynomial representation allows us to construct a *dependency graph* G_{poly} where an edge from gene x_j to gene x_i exists if x_j appears in the polynomial T_i .

Definition D.1 (Polynomial Influence Score). *For each gene x_j , the polynomial influence score $I_{\text{poly}}(x_j)$ is defined as:*

$$I_{\text{poly}}(x_j) := \#\{i \in \{0, \dots, N-1\} : x_j \text{ appears in } T_i\}, \quad (11)$$

i.e., the number of genes whose update function depends explicitly on x_j (including self-dependence when x_j appears in T_j). This equals the out-degree of x_j in G_{poly} (counting self-loops).

Note: The influence score I_{poly} measures *direct* regulatory influence (literal appearance in the canonical polynomial), which differs from other notions such as Boolean sensitivity (average effect of flipping x_j) or structural network centrality (graph-theoretic measures). All three notions capture different aspects of regulatory importance; the polynomial influence score has the advantage of being uniquely determined by the discrete dynamics.

Analyzing the influence scores reveals the hierarchical influence structure:

1. *LFY* (x_2): Appears in 7 polynomials (EMF1, TFL1, AG, AP1, SEP, AP3, PI) — highest influence.
2. *TFL1* (x_3): Appears in 5 polynomials (LFY, AP2, AG, AP1, FUL) — very high influence.
3. *AG* (x_6): Appears in 5 polynomials (AG, AP1, AP3, PI, WUS) — very high influence.
4. *AP1* (x_7): Appears in 5 polynomials (TFL1, AG, AP3, PI, FUL) — very high influence.
5. *EMF1* (x_1): Appears in 4 polynomials (LFY, TFL1, FT, AG) — high influence.

The genes with the highest polynomial influence coincide with those occupying early positions in π^* (Section 3, Table 3). The ranking $\text{LFY} > \text{TFL1} \approx \text{AG} \approx \text{AP1} > \text{EMF1}$ mirrors the regulatory importance of these master regulators: high local influence (many direct targets) correlates with global hierarchical importance (low μ when placed in leading positions).

Non-linear interactions and visualization. Product terms in the polynomials expose non-linear regulatory logic: $T_2 = 1 + x_1 \cdot x_3$ (LFY, a NAND gate), $x_7 \cdot x_3$ in FUL (combinatorial AP1/TFL1 input), and $x_{11} \cdot x_6 \cdot x_8$ in WUS (autoregulation requiring AG and SEP). Figures 9 and 10 show the dependency network and influence ranking; hub genes (LFY, TFL1, EMF1, AG) have many outgoing edges, and the polynomial influence ranking agrees with the μ -based ordering on the top-ranked genes.

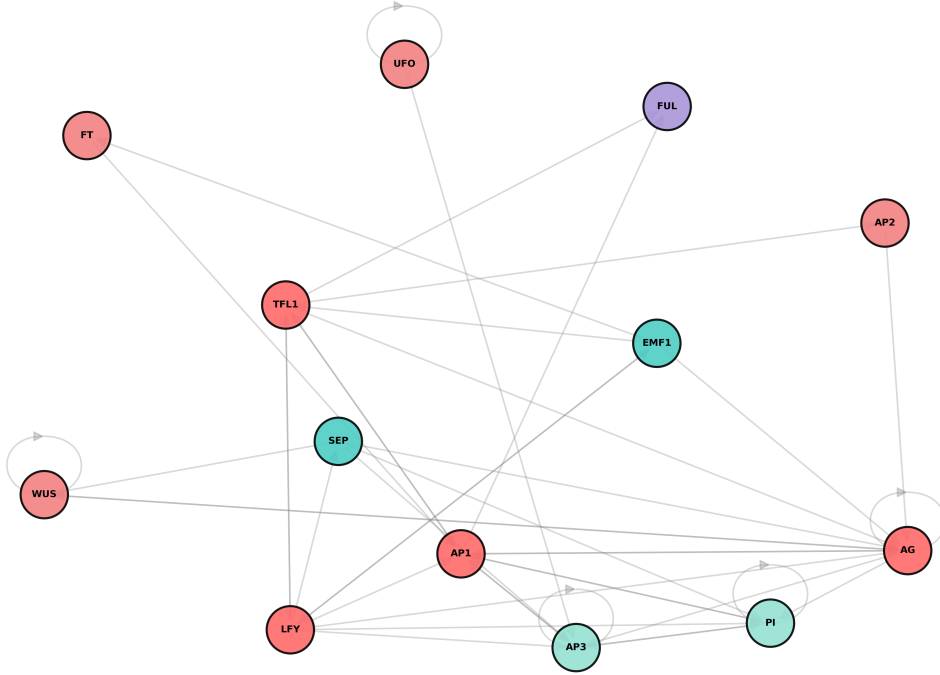


Figure 9: Polynomial dependency network for *A. thaliana* in the coordinates of Eq. 3 (Table 3). Section 3 specifies the certified μ_E -minimum is used for $(x_0, \dots, x_{12})$ (four minima; μ_A -maxima are separate—Appendix A). Edges are direct dependencies in the canonical Boolean polynomials. Generated from transition data in Supplementary Material.

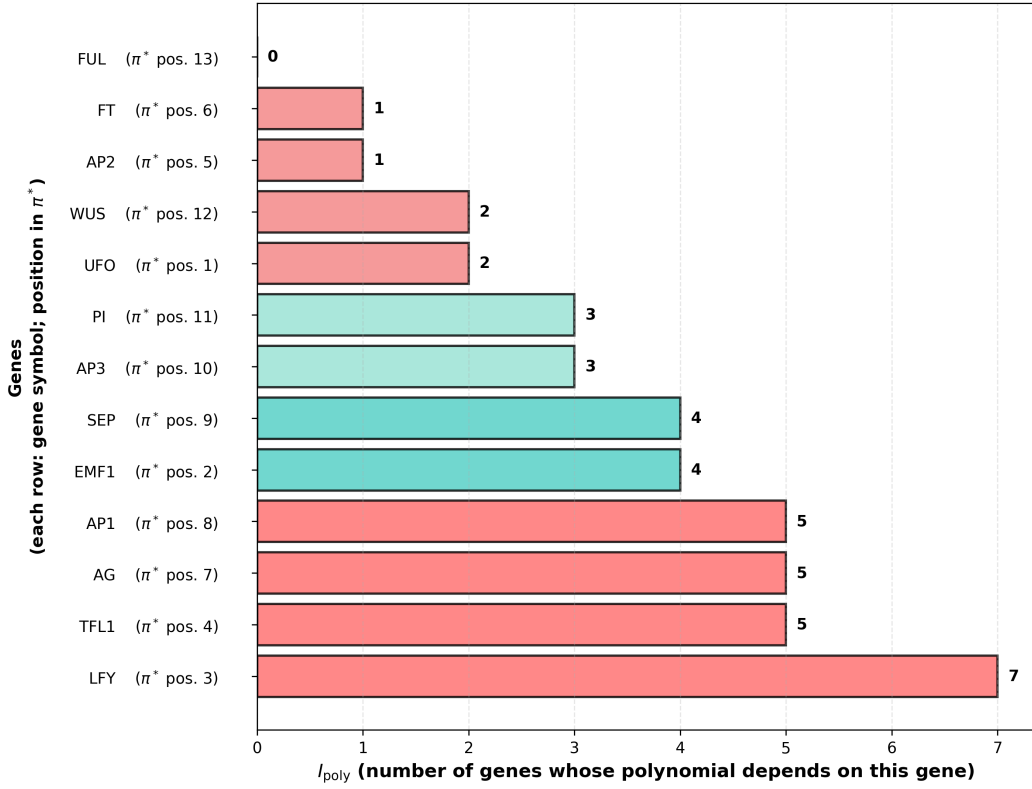


Figure 10: Influence ranking for *A. thaliana*: I_{poly} (bar length) and position $k \in \{1, \dots, 13\}$ along the ordering in Eq. 3 (Table 3). Same labeling convention as Section 3 (one fixed μ_E -minimum among four; Appendix A). Generated from transition data in Supplementary Material.

D.2 *C. elegans*

The canonical $\mathbb{F}_2$ polynomial representation of each update function T_i was computed from the Huang-aligned transition table [14] under the ordering π^* (equation (4)). Variables $(x_0, \dots, x_7)$ denote the *ordered* coordinates along π^* : x_0 is cdc-25.1 (coarsest), $\dots$, x_7 is cki-1 (finest).

- T_0 (cdc-25.1) = $x_0 + x_0x_2$: depends on cdc-25.1 and cul-1, consistent with SCF-mediated inputs to the G2/M phosphatase arm [14].
- T_1 (cdk-2) is a multilinear polynomial of degree 4 in $(x_0, x_1, x_2, x_3, x_7)$ with 20 monomials; it encodes integration of cdc-25.1, cdk-2, cul-1, lin-35, and cki-1 at the second scale of π^* .
- T_2 (cul-1) = x_1 : cul-1 tracks cdk-2 activity.
- T_3 (lin-35) = $x_2 + x_3 + x_1x_2 + x_1x_3 + x_2x_3$: Rb homolog update depends on cdk-2, cul-1, and lin-35.
- T_4 (cdc-14) = $x_4 + x_6 + x_1x_4 + x_1x_6 + x_4x_6$: mitotic exit phosphatase couples to cdk-2 and cdk-1.
- T_5 (fzr-1) = x_4 : APC regulation follows cdc-14.
- T_6 (cdk-1) is a multilinear polynomial of degree 4 supported on $\{x_0, x_3, x_4, x_5, x_6, x_7\}$ with 40 monomials (full ANF in Supplementary `compute_polynomials_celegans.py`).
- T_7 (cki-1) = $x_4 + x_5 + x_4x_5$: inhibitor update combines cdc-14 and fzr-1.

Polynomial Influence Ranking. The polynomial influence score $I_{\text{poly}}(x_j)$ (number of update functions that depend on x_j) is:

j	Gene	I_{poly}	Targets
1	cdk-2	4	cdk-2, cul-1, lin-35, cdc-14
4	cdc-14	4	cdc-14, fzf-1, cdk-1, cki-1
0	cdc-25.1	3	cdc-25.1, cdk-2, cdk-1
2	cul-1	3	cdc-25.1, cdk-2, lin-35
3	lin-35	3	cdk-2, lin-35, cdk-1
5	fzf-1	2	cdk-1, cki-1
6	cdk-1	2	cdc-14, cdk-1
7	cki-1	2	cdk-2, cdk-1

Cdk-2 (x_1) and cdc-14 (x_4) share the highest $I_{\text{poly}} = 4$; π^* nevertheless places cdc-25.1 (x_0) at the coarsest scale, illustrating the distinction between local polynomial fan-out and the multi-scale stability objective (Section 7).

Dependency Structure and Comparison with π^* . The dependency graph G_{poly} has edges $j \rightarrow i$ whenever x_j appears in T_i . Two structural features align with the first half of π^* :

1. *Top-down cascade:* $\text{cdc-25.1} (x_0) \rightarrow \text{cdk-2} (x_1) \rightarrow \text{cul-1} (x_2) \rightarrow \text{lin-35} (x_3)$, matching positions 1–4 of π^* .
2. *Mitotic exit block:* $\text{cdc-14} (x_4) \leftrightarrow \text{fzf-1} (x_5) \leftrightarrow \text{cdk-1} (x_6) \leftrightarrow \text{cki-1} (x_7)$, with feedback from cdk-1 to cdc-14 and cross-talk from cki-1 to cdk-2 and cdk-1.

Figures 11 and 12 display the dependency network and influence comparison (generated from the Huang-aligned polynomials).

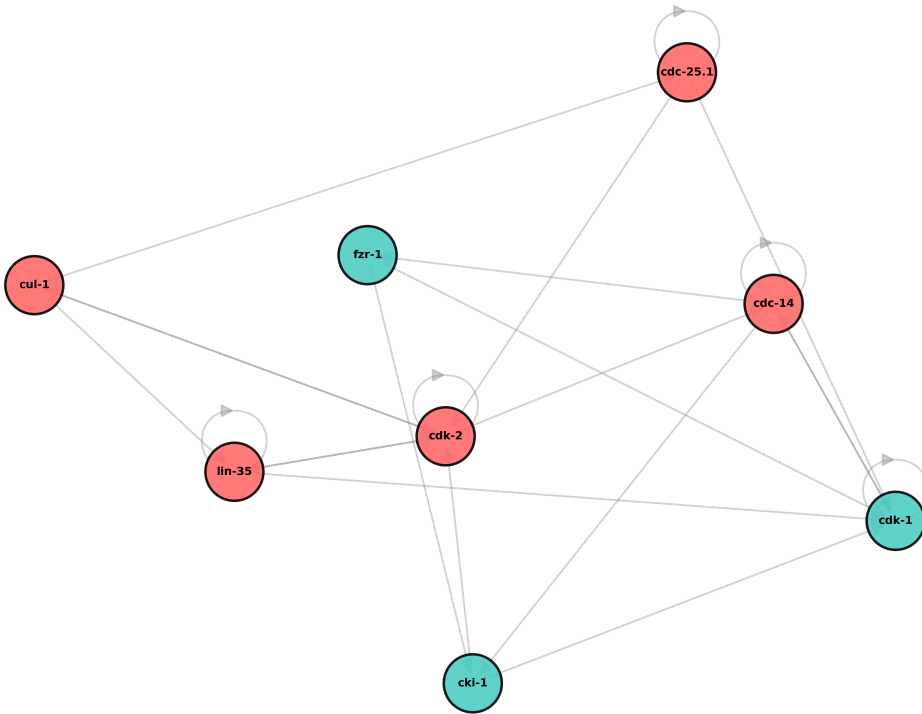


Figure 11: Polynomial dependency network for the *C. elegans* cell cycle (Huang-aligned dynamics). Edges $j \rightarrow i$ indicate that x_j appears in T_i . Node color encodes I_{poly} : red = highest (4), teal = 3, pink = 2.

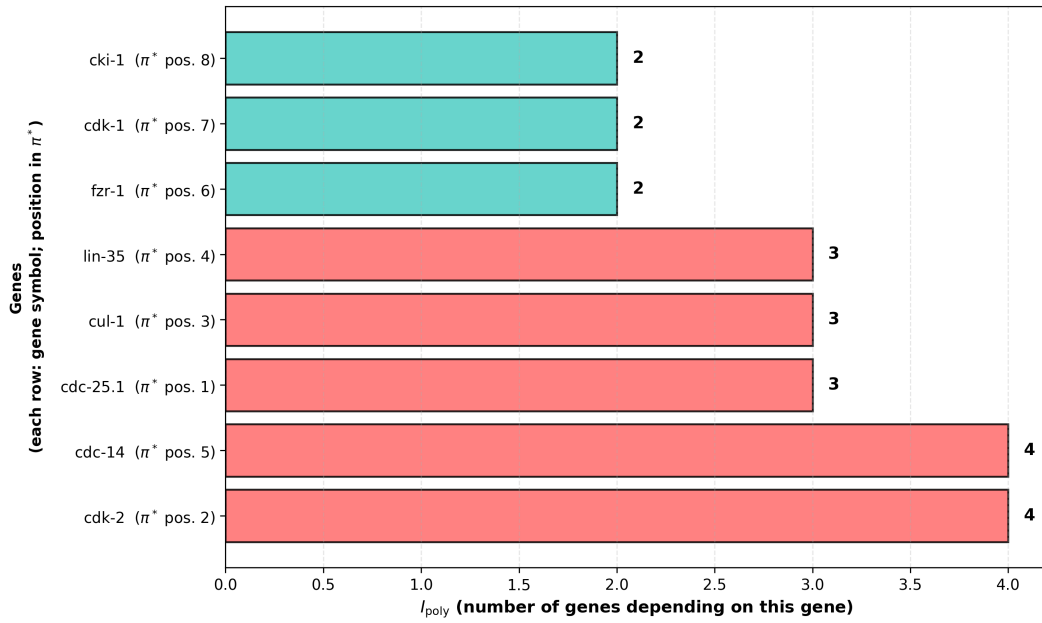


Figure 12: Polynomial influence ranking for *C. elegans*: I_{poly} vs. position in π^* . Cdk-2 and cdc-14 share the highest scores, while cdc-25.1 leads the hierarchy for μ_E .

D.3 *D. melanogaster*

Under $\pi^* = (\text{Bcd}, \text{Cad}, \text{Kni}, \text{Gt}, \text{Kr}, \text{Hb})$, the canonical $\mathbb{F}_2$ polynomials for the six update functions are:

- *Bicoid*, *Caudal* (x_0, x_1): Self-maintaining ($T_0 = x_0, T_1 = x_1$).
- *Knirps* (x_2): $T_2 = x_1(1 + x_3)(1 + x_4) \pmod{2}$: activated by Cad, repressed by Gt and Kr.
- *Giant* (x_3): $T_3 = x_1(1 + x_2)(1 + x_4) \pmod{2}$: activated by Cad, repressed by Kni and Kr.
- *Krüppel* (x_4): $T_4 = x_0 \cdot x_5(1 + x_2)(1 + x_3) \pmod{2}$: requires both Bcd and Hb (x_5 , the last position); repressed by Kni and Gt.
- *Hunchback* (x_5): $T_5 = x_0(1 + x_3)(1 + x_4) \pmod{2}$: activated by Bcd, repressed by Gt and Kr.

The polynomial influence ranking is: Bcd, Cad, Gt, Kr: $I_{\text{poly}} = 3$ each; Kni: $I_{\text{poly}} = 2$; Hb: $I_{\text{poly}} = 1$. The four top-ranked genes are tied at $I_{\text{poly}} = 3$, so I_{poly} alone does not separate the morphogens from the gap genes—the μ -ordering does (Bcd and Cad at positions 0–1 in π^* ; Section 7). Krüppel has the most complex polynomial (four inputs, including Hb).

The dependency structure is: $T_2(\text{Kni}) \leftarrow \text{Cad}, \text{Gt}, \text{Kr}$; $T_3(\text{Gt}) \leftarrow \text{Cad}, \text{Kni}, \text{Kr}$; $T_4(\text{Kr}) \leftarrow \text{Bcd}, \text{Kni}, \text{Gt}, \text{Hb}$; $T_5(\text{Hb}) \leftarrow \text{Bcd}, \text{Gt}, \text{Kr}$. Maternal morphogens (Bcd, Cad) appear only as activators; gap genes appear through repression terms $(1+x_i)$, creating the mutual inhibition that sharpens domain boundaries [1, 15]. Figures 13 and 14 illustrate the dependency network and influence comparison; edges $j \rightarrow i$ denote that x_j appears in T_i (Section 7).

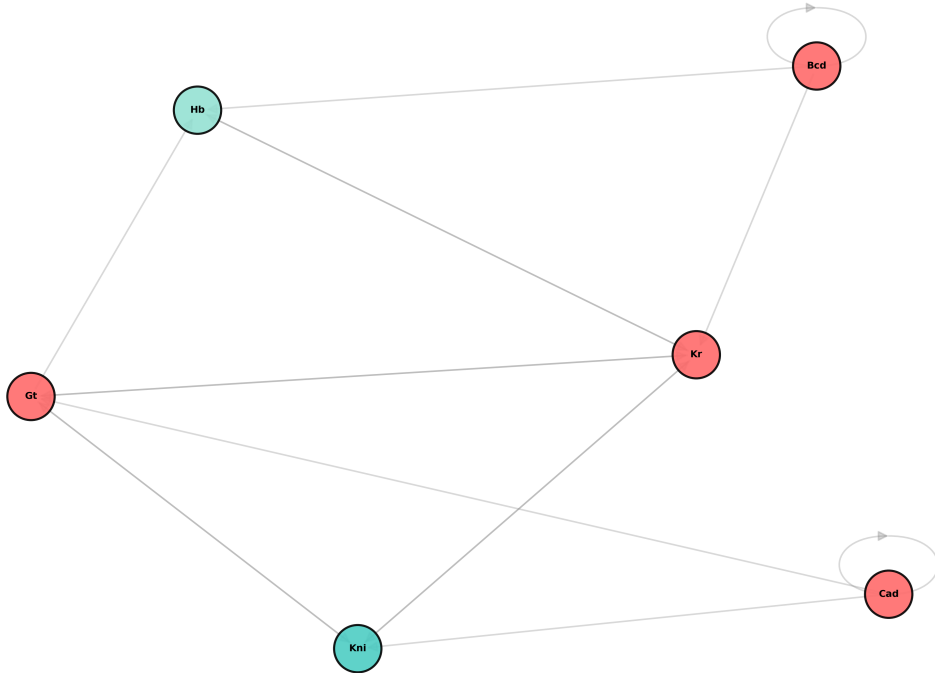


Figure 13: Polynomial dependency network for *Drosophila melanogaster* (gap genes) in the coordinates of Eq. 5 (Table 8). Section 5 specifies which of the four μ_E -minima is used for $(x_0, \dots, x_5)$ (Appendix C). Edges are direct dependencies in the canonical Boolean polynomials. Generated from transition data in Supplementary Material.



Figure 14: Influence ranking for *D. melanogaster*: I_{poly} (bar length) and position $k \in \{1, \dots, 6\}$ along the ordering in Eq. 5 (Table 8). Same $(x_0, \dots, x_5)$ convention as Section 5 (one fixed μ_E -minimum among four; Appendix C). Generated from transition data in Supplementary Material.

Supplementary material

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	—
Files S1–S4	Yes	—
Full transition CSVs; code; extended B&B logs; extra GA outputs	No	Available to editors/referees on request during review; public repository (DOI) at acceptance (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 (companion paper [21]); 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):** upon acceptance, the artifact bundle referenced in Data availability (CSVs, code, logs, reproducibility notes) will receive a public repository DOI; during peer review the same bundle is available to the editor and referees on request, without duplicating the Data availability statement here.

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. During peer review, the complete bundle is available to the editor and referees upon request; upon acceptance, it will be deposited in a public repository (DOI to be added). The deposited archive is accompanied by a readme documenting file organization and numerical verification checks.

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