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Tricking Turing: How to Build a Geometric Checkerboard on Living Tissue

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Abstract

The snake’s head fritillary (*Fritillaria meleagris*) displays one of the rarest pigmentation motifs in the plant kingdom: a high-contrast checkerboard of alternating purple and white domains on its flowers. Standard reaction–diffusion theory does not generically produce this pattern: while Turing instabilities in isotropic media select a length scale, they impose no preferred orientation or strict alternation between neighbors. We show that a minimal set of three sequential physical mechanisms — a simulated Gray–Scott-type reaction–diffusion prepattern guided by vascular veins, anisotropic diffusion confined to interveinal corridors, and a threshold-switch Hill-type pigmentation response, each already documented separately in living systems — is sufficient to produce a square, alternating motif. We quantify agreement with biological measurements using a dedicated shape metric, the Square Index (SI [%]), capturing how closely a pigment domain approaches a perfect square. The full model raises SI from the classical Turing-spot value ($\approx 76\%$) to $\approx 83\%$, closing roughly a third of the remaining gap to an ideal square lattice (**100%**); we instead account for the pattern’s strict two-dimensional alternation structurally, via vein-imposed staggering. These results show how tissue geometry and transport anisotropy can steer diffusion-driven instabilities into a non-canonical spatial pattern.

Keywords: Turing instability, Reaction–diffusion, Anisotropic diffusion, Pattern formation, *Fritillaria meleagris*, Checkerboard pattern

1 Introduction

In 1952, Turing demonstrated that a spatially uniform mixture of two reacting, diffusing chemicals can spontaneously develop spatial order [1]. The mechanism is elegant: a slowly diffusing activator that promotes its own production, coupled to a faster-diffusing inhibitor that represses it, can turn a featureless equilibrium into a periodic pattern. The instability selects a characteristic wavelength — fixed by the balance between reaction kinetics and diffusion coefficients — and produces spots, stripes, or labyrinths depending on nonlinear parameters [2].

What a standard Turing system does *not* naturally produce is a checkerboard. This observation is sharper than it might first appear. A checkerboard requires two things simultaneously: (i) approximately *square* domains rather than circular spots, and (ii) strict *two-dimensional alternation* — every face-adjacent neighbor of a pigmented domain must be unpigmented. Concretely, these requirements mean that two perpendicular spatial modes of identical amplitude must coexist in a stable equilibrium. This equal-amplitude condition is, as we show below, generically *unstable* in isotropic two-species reaction–diffusion systems: nonlinear mode competition typically breaks the symmetry. Within this two-mode reduction, a single mode comes to dominate, and the result is stripes. A checkerboard is, in a precise mathematical sense, a pattern that Turing mechanics actively resist.

Yet the snake’s head fritillary (*Fritillaria meleagris*) produces exactly this motif on its tepals — the undifferentiated, petal-like organs that make up its perianth (Fig. 1a,b). The tepals bear a high-contrast checkerboard of alternating purple and white domains, remarkably consistent across specimens and across the full surface of each tepal [3]. The pattern is rare in the plant kingdom [3], and that rarity is not accidental: achieving it requires more than a reaction–diffusion instability alone. In a survey of computational flower-pigmentation models spanning vascular patterning, positional information, and reaction–diffusion, Ringham *et al.* identified *Fritillaria*’s vein-correlated blotches as exactly this kind of combined case and designed their modeling framework to accommodate it, but did not implement it as one of their case studies [4]. We take up that open case directly, with a mechanistic model tied to the vascular geometry and a quantitative comparison against measured tepal patches.

This paper asks how the fritillary’s tissue architecture converts a mechanism that resists squares into one that reliably produces them. We construct a minimal three-step biophysical model — the FASHION model (Feedback-driven Anisotropic Self-organized Heterogeneity In Oriented Networks) — that starts from a standard Turing instability and steers it toward a checkerboard through two additional physical ingredients: diffusion barriers imposed by the vascular architecture, and anisotropic transport within interveinal corridors. We also introduce a dedicated shape metric, the Square Index (SI), that quantifies how far any given pattern’s domains deviate from — or converge toward — a square.

The paper is organized as follows. Section 2 reviews the physics of reaction–diffusion instabilities and explains, using a mode-competition argument, why square checkerboards are not the generic outcome. Section 3 introduces the *Fritillaria meleagris* pattern and defines the SI metric. Section 4 presents the FASHION model.

Section 5 provides a quantitative comparison across model phases and biological measurements.

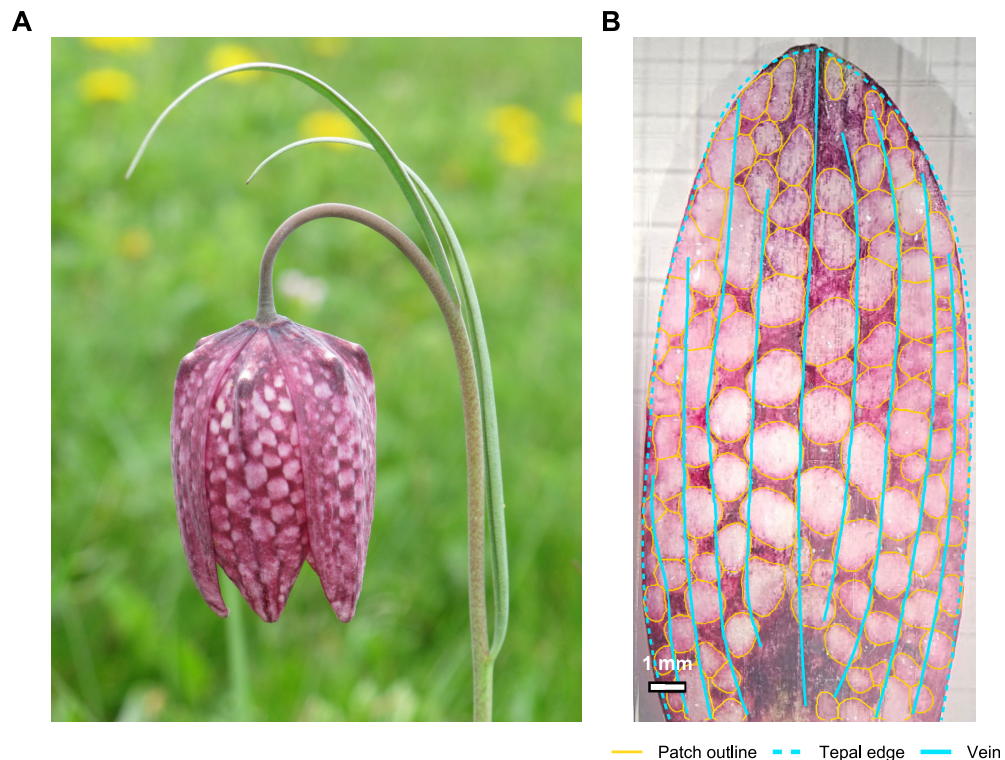


Fig. 1 The snake’s head fritillary (*Fritillaria meleagris*) and its checkerboard pigmentation. (a) In-situ photograph [5] of a whole flower. (b) A tepal from a different specimen, with the segmented pigment patches outlined and longitudinal veins overlaid.

2 Turing instabilities: a wavelength without a shape

2.1 Diffusion-driven instability and wavelength selection

Turing showed that coupling diffusion to nonlinear reactions between two chemical species can spontaneously destabilize a spatially uniform steady state [1]. In the canonical activator–inhibitor formulation [6], a slowly diffusing self-enhancing activator coupled to a faster-diffusing inhibitor undergoes diffusion-driven instability: local perturbations in activator concentration are amplified while the inhibitor spreads and damps growth in surrounding regions (Fig. 2a). The two-species dynamics with concentrations u , v , isotropic diffusion coefficients D_u , D_v , and local kinetics $R(u, v)$,

$S(u, v)$ read

$$\begin{cases} \frac{\partial u}{\partial t} = D_u \nabla^2 u + R(u, v), \\ \frac{\partial v}{\partial t} = D_v \nabla^2 v + S(u, v). \end{cases} \quad (1)$$

Linear stability analysis of the homogeneous fixed point yields a dispersion relation $\sigma(k)$. In a Turing instability, σ is negative at $k = 0$ (stability to uniform perturbations) and at large k (diffusion damps sharp variations), but positive over an intermediate band around a critical wavenumber k_c (Fig. 2b), selecting the characteristic wavelength $\lambda_c = 2\pi/k_c$ from the balance of reaction kinetics and differential diffusion [7] — not from any external template.

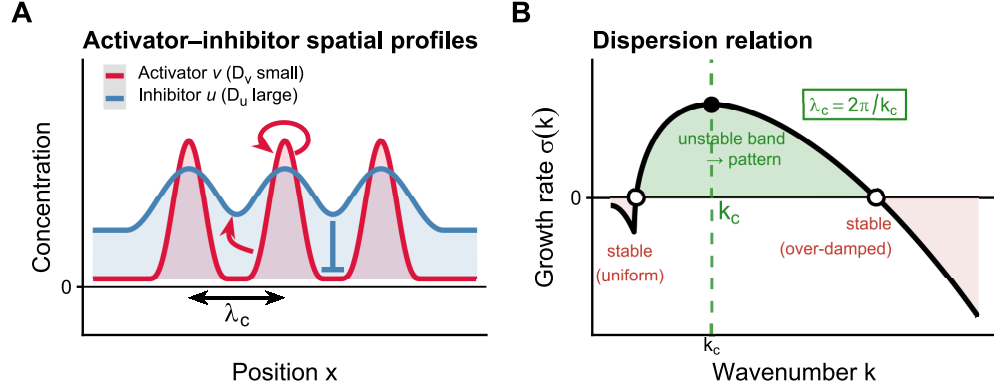


Fig. 2 Turing instability mechanism. (a) Schematic activator–inhibitor profiles: the activator (small D_v) self-enhances locally while the faster-diffusing inhibitor (large D_u) spreads and suppresses surrounding growth, setting λ_c . (b) Dispersion relation $\sigma(k)$: the growth rate is positive only over a finite band around k_c , selecting $\lambda_c = 2\pi/k_c$.

2.2 The standard Turing repertoire: spots, stripes, labyrinths

Reaction–diffusion models generate spots, stripes, and labyrinths [2], each with biological counterparts (Fig. 3): leopard spots [8, 9], zebra stripes [10], and the branching network of *Kohleria amabilis* [4]. The zebrafish is the paradigmatic case: Nakamasu *et al.* identified the short-range activation / long-range inhibition mechanism between melanophores and xanthophores via laser-ablation experiments and reproduced the wild-type stripe pattern in simulation [11] (Fig. 3d,d'), closing the gap between abstract Turing theory and cell-level biology. We follow the same strategy for *Fritillaria*: identify the concrete physical mechanism that a generic reaction–diffusion model leaves unspecified.

What these systems do not generically produce is a checkerboard. Squared domains with strict two-dimensional alternation between neighboring patches remain uncommon [3], for reasons rooted in nonlinear mode competition, which we now quantify.

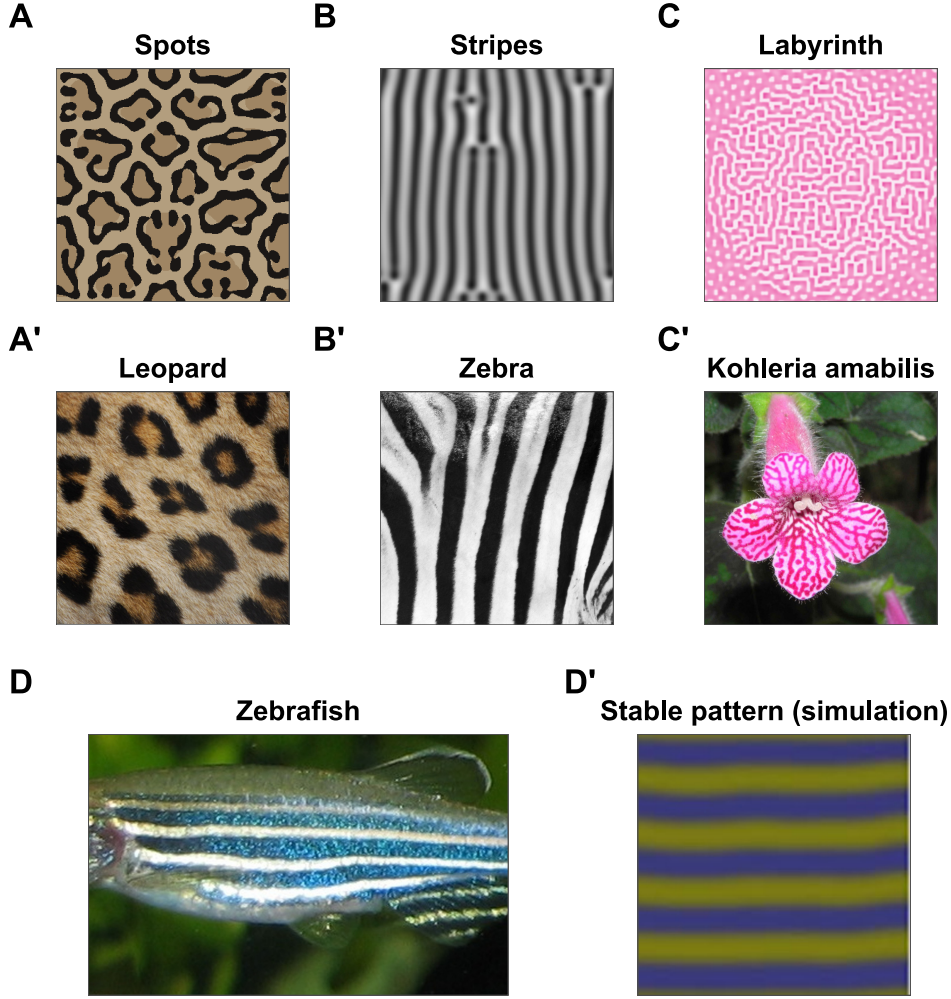


Fig. 3 The standard Turing repertoire, simulated and real. Top row, Gray–Scott simulations: (a) spots, (b) stripes, (c) labyrinth. Middle row, biological counterparts: (a') leopard spots [12], (b') zebra stripes [13], (c') *Kohleria amabilis* [14]. Bottom row: (d) zebrafish photograph [15] and (d') stable stripe pattern from Nakamasu *et al.*'s simulation [11] (reproduced from their Fig. 4b).

2.3 Why squares lose the mode competition

In an isotropic medium the dispersion relation $\sigma(k)$ depends only on $k = |\mathbf{k}|$, so *every* orientation on the critical circle $|\mathbf{k}| = k_c$ is equally unstable: linear theory fixes a wavelength but cannot choose between, say, two perpendicular modes of that same wavelength, since both grow at exactly the same rate $\sigma(k_c)$. Deciding between them needs a second stability test, one order beyond the linear one of Section 2.1: once a mode has grown large enough, the nonlinear terms in $R(u, v)$ and $S(u, v)$ start to matter and can suppress it.

The instability threshold is the point where $\sigma(k_c)$ first crosses zero. Close to it, only the two perpendicular candidate modes identified above matter, and deciding between them is a question nonlinear terms must answer, not linear ones alone. The activator deviates from its homogeneous value as $u - u_0 \propto \phi(\mathbf{r}, t) = A_1(t) e^{ik_c x} + A_2(t) e^{ik_c y} + \text{c.c.}$ ¹, where A_1, A_2 play the role of order parameters for the square-versus-stripe competition, forming a square lattice precisely when $|A_1| = |A_2|$, and collapsing to stripes when one amplitude dominates. Unlike the fast spatial oscillation $e^{ik_c x}$, the complex amplitudes $A_1(t), A_2(t)$ vary slowly in time, growing or shrinking only at the rate ε (the distance above threshold, $\varepsilon \rightarrow 0$ at onset). Substituting ϕ back into Eq. (1) and collecting terms order by order in ε — the standard weakly-nonlinear procedure [7, 16–19] — shows that A_1, A_2 obey coupled Landau equations:

$$\begin{aligned}\dot{A}_1 &= \varepsilon A_1 - (|A_1|^2 + \nu |A_2|^2) A_1, \\ \dot{A}_2 &= \varepsilon A_2 - (|A_2|^2 + \nu |A_1|^2) A_2.\end{aligned}$$

Each term has a direct physical reading. εA_i is the linear growth rate that made mode i unstable in the first place. Both modes share the *same* linear growth rate ε — not separate $\varepsilon_1, \varepsilon_2$ — because $\sigma(k)$ depends only on $|\mathbf{k}|$ in an isotropic medium and $|\mathbf{k}_1| = |\mathbf{k}_2| = k_c$. The cubic terms are the leading correction once R, S are Taylor-expanded past first order, and split into two pieces: $|A_i|^2 A_i$ is self-saturation (a mode limits its own growth), and $\nu |A_j|^2 A_i$ is cross-saturation, coupling the two modes because they draw on the same finite pool of activator and inhibitor. The coefficient ν is the ratio of cross-coupling to self-coupling.

These two equations have a fixed point at equal amplitude, $|A_1| = |A_2| = \rho_0 = \sqrt{\varepsilon/(1+\nu)}$ — the square pattern. Writing $A_n(t) = \rho_n(t) e^{i\theta_n(t)}$, perturbing $\rho_1 = \rho_0 + \delta_1$, $\rho_2 = \rho_0 + \delta_2$ and linearizing gives

$$\frac{d}{dt} \begin{pmatrix} \delta_1 \\ \delta_2 \end{pmatrix} = -2\rho_0^2 \begin{pmatrix} 1 & \nu \\ \nu & 1 \end{pmatrix} \begin{pmatrix} \delta_1 \\ \delta_2 \end{pmatrix}.$$

This 2×2 matrix has two eigenvectors by symmetry: perturbing both amplitudes together ($\delta_1 = \delta_2$) gives eigenvalue $\sigma_+ = -2\varepsilon < 0$ — always stable. Perturbing them in opposition ($\delta_1 = -\delta_2$), which would turn a square into a stripe, gives the eigenvalue that decides the outcome:

$$\sigma_- = \frac{2\varepsilon(\nu - 1)}{1 + \nu}.$$

If $\nu < 1$, $\sigma_- < 0$ and any imbalance decays — the square is stable. If $\nu > 1$, $\sigma_- > 0$ and one mode wins, collapsing onto a single-mode stripe pattern. The value $\nu = 1$ is therefore a bifurcation point marking where the square fixed point exchanges stability with the single-mode stripe branch.

In two-species isotropic reaction–diffusion kinetics, $\nu > 1$ is the generic case [16, 20]: stripes, not squares, are the default two-dimensional outcome.

This cubic-order competition is not even the main threat to square order: in an isotropic medium the leading nonlinearity is generically quadratic, and a quadratic

¹c.c. stands for complex conjugate, added so that the physically observable field $u - u_0$ is real.

term lets three wavevectors resonate whenever $\mathbf{k}_1 + \mathbf{k}_2 + \mathbf{k}_3 = 0$ — a condition only a hexagonal lattice satisfies, since two perpendicular modes of equal length never sum to zero. Wherever that quadratic term survives, hexagons pre-empt the square/stripe competition altogether near threshold [7, 16], which is why hexagonal order, not square order, is the generic outcome of two-dimensional pattern formation. A square checkerboard therefore needs not only the non-generic condition $\nu < 1$ to beat stripes, but also some mechanism suppressing the hexagonal channel entirely, enforcing $|A_1| = |A_2|$ by construction rather than by chance. This is exactly the role the tissue geometry of *Fritillaria meleagris* plays, as we show in Section 4.

3 A rare geometry: quantifying the fritillary checkerboard

3.1 A shape metric for checkerboard characterization

The tepals of *Fritillaria meleagris* display a high-contrast checkerboard of alternating pigmented (purple) and unpigmented (white) regions, forming a quasi-rectangular lattice of discrete domains with sharp boundaries, extending over the entire length of the tepal with a characteristic spacing conserved within individual flowers and comparable across specimens (Fig. 1).

The checkerboard pattern has two defining geometric properties that any model must reproduce: (i) individual pigment domains are approximately *square* rather than circular, and (ii) neighboring domains *alternate* strictly between pigmented and unpigmented states. We quantify property (i) with a dedicated shape metric, the Square Index (SI).

3.1.1 Square Index

The standard circularity $C = 4\pi A/P^2$ takes its maximum $C = 1$ for a circle and yields $C = \pi/4 \approx 79\%$ for a perfect square, thereby *penalizing* square shapes. This makes it poorly suited to distinguish the near-circular spots of classical Turing systems ($C \approx 74\text{--}78\%$) from the square-like domains of a checkerboard.

We instead define the Square Index (SI) for a patch of area A inscribed in its minimum-area bounding box — height H and width W , found by rotating the box to minimize its area, so that SI is independent of the patch’s orientation in space — as a percentage of its 0–100% range:

$$\text{SI} = \underbrace{\frac{A}{H \times W}}_{\text{fill fraction}} \times \underbrace{\frac{\min(H, W)}{\max(H, W)}}_{\text{aspect factor}} \quad [\%].$$

The first factor measures how densely the patch fills its bounding box; the second penalizes elongation (Fig. 4a illustrates both terms on a single patch). Table 1 lists SI for canonical shapes. Unlike circularity, SI is maximized (SI = 100%) by a perfect square and takes a strictly lower value (SI = $\pi/4 \approx 79\%$) for a circle.

Table 1 Square Index reference values for canonical shapes.

Shape	Name	Fill fraction	Aspect factor	SI
	Perfect square	1	1	100%
	Perfect circle	$\pi/4 \approx 0.785$	1	79%
	Ellipse (axis ratio $r=b/a$)	$\pi/4$	r	$(\pi/4) r$
	Ellipse $r = 1/10$ (Phase-B ratio)	$\pi/4$	1/10	$\approx 8\%$
	Same, laterally truncated to $W=H$	$\frac{1}{2} [\sqrt{1-r^2} + \arcsin(r)/r]$	1	$\approx 99.8\%$
	Regular hexagon	3/4	$\sqrt{3}/2 \approx 0.866$	65%
	Rectangle 1:2	1	1/2	50%
	Thin band	1	$\rightarrow 0$	$\rightarrow 0\%$

3.2 Quantitative geometry of pigment patches

We primarily quantified white patches, consistent with our model in which pigmentation is described as the emergence of white domains over a purple background [3].

Patches were segmented from the photographed tepals using Cellpose, a general-purpose deep-learning cell/object segmentation tool [21]. Segmentation of $n = 644$ patches across six tepal images, using the same approach as Fig. 1b, revealed a mean SI = $58.9\% \pm 12.0\%$ (SD) for the biological patches, providing a quantitative target for the model comparison in Section 5.

3.3 Spatial organization and alternation

Patch centroids are distributed quasi-periodically along the longitudinal tepal axis, with relatively regular spacing within interveinal regions. Transverse positioning shows alternation between adjacent corridors, consistent with an inhibitory coupling that offsets patch positions from one corridor to the next (Fig. 4b).

This alternation is robust but not perfect: patches are sometimes truncated at vein boundaries, producing half-sized or irregular domains. Such defects are recurrent features of the pattern rather than measurement noise, suggesting they are intrinsic to the developmental process.

3.4 Vascular architecture as a diffusion template

The physical environment in which patterning occurs is far from a homogeneous slab. Tepals are traversed by longitudinal vascular veins that partition the epidermal surface into parallel interveinal corridors (Fig. 1b). Veins create two types of physical constraints relevant to any diffusing molecule:

Barriers: The dense cellular material of vascular bundles strongly reduces molecular transport across veins, acting effectively as a strong, though not fully impermeable, diffusion barrier on the scale of the patterning wavelength. This matches direct evidence from leaf veins: cell-to-cell permeability (via plasmodesmata) drops sharply between vein and non-vascular tissue as the vein matures, while staying high between cells within the vein itself, so small-molecule movement is blocked laterally even though

the vein’s own longitudinal transport role is preserved [22]. This also mirrors how a rigid, pre-existing tissue geometry has been shown to discretize an otherwise continuous reaction–diffusion field into compartmentalized units elsewhere in nature [23, 24], and is directly consistent with the observation that no pigment motif crosses two *Fritillaria* veins simultaneously [3] (Fig. 4b).

Anisotropy: Within each corridor, transport is not isotropic. We hypothesize that architectural polarity of the tissue generates preferential transport perpendicular to veins (fast) over transport along them (slow). This is biologically plausible: plant cells routinely move one specific molecule anisotropically via dedicated polarized transporters while every other species diffuses isotropically around it, as documented for the plant hormone auxin [25] — though the specific cross-vein direction assumed here is our modeling hypothesis, not a directly measured one. This is conveniently modeled by two separate diffusion coefficients, D_x (across veins) and D_y (along veins), with $D_x \gg D_y$ — the same anisotropic-diffusion-tensor treatment, formalized via a pair of orthogonal diffusivity eigenvectors, that Ringham *et al.* use to orient vein-guided pigmentation patterns across a tepal surface [4].

These two constraints together break the rotational symmetry of the underlying diffusion problem, imposing preferred directions on any pattern that forms within the tissue — precisely the type of symmetry breaking needed to stabilize a square lattice against stripe selection.

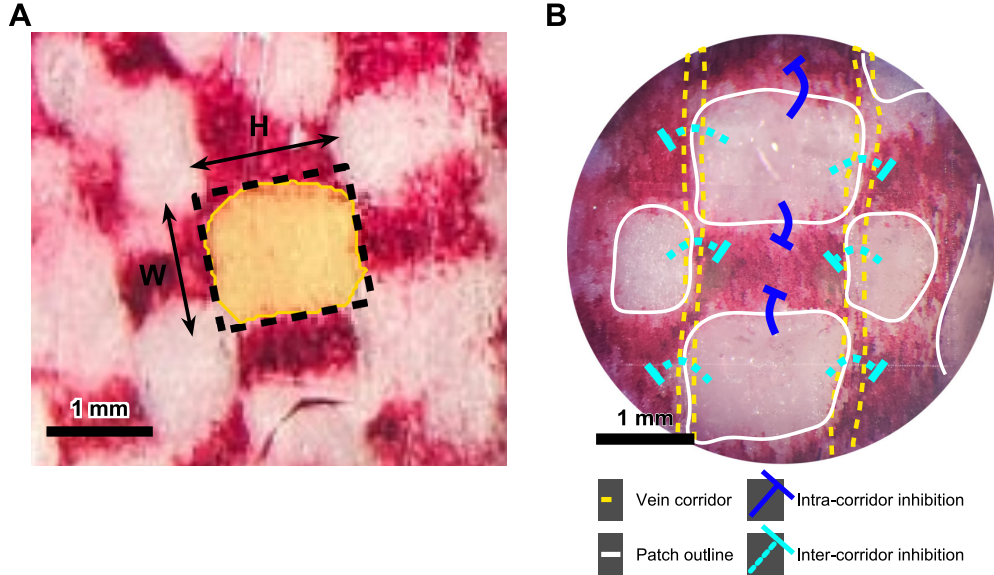


Fig. 4 (a) Construction of the Square Index for a single patch: minimum-area bounding box (height H , width W) with the patch area highlighted, illustrating the fill-fraction and aspect-factor terms of the SI formula (Section 3.1.1). (b) Proposed inhibitory coupling overlaid on a microscope close-up of *Fritillaria meleagris* tepal tissue: intra-corridor inhibition (dark blue) links successive patches along a corridor, while inter-corridor inhibition (cyan) leaks across the semi-permeable vein to couple neighboring corridors — together offsetting patch positions from one corridor to the next.

3.5 Implications for pattern-forming mechanisms

The combination of (i) regular spacing with strict alternation, (ii) polygonal domain shapes aligned with veins, and (iii) abrupt pigmentation transitions points toward a multistep process involving positional prepatterning, anisotropic transport, and non-linear cellular responses. This characterization directly motivates the model introduced in Section 4.

4 Tricking Turing: the three-step FASHION model

To investigate the developmental origin of the checkerboard pigmentation, we constructed a minimal biophysical framework termed the Feedback-driven Anisotropic Self-organized Heterogeneity In Oriented Networks (FASHION) model. It operates on a two-dimensional domain representing the epidermal surface of the tepal and consists of three sequential processes: (i) Turing-type positional prepatterning, (ii) anisotropic diffusion constrained by vascular architecture, and (iii) a threshold-switch cellular pigmentation response.

4.1 Design constraints

A satisfactory mechanism must reproduce: (1) distinct unpigmented patches on a pigmented background; (2) regular spacing with strict alternation between neighbors; (3) polygonal, approximately square domain shapes oriented along the venation pattern; and (4) sharp pigmentation boundaries.

4.2 Phase A: Turing-type positional prepatterning

Positional information is generated by the Gray–Scott system [2, 26]:

$$\begin{cases} \frac{\partial u}{\partial t} = D_u \nabla^2 u - uv^2 + F(1 - u), \\ \frac{\partial v}{\partial t} = D_v \nabla^2 v + uv^2 - (F + k) v, \end{cases} \quad (2)$$

The reaction terms encode a minimal autocatalytic chemistry: two units of v convert one unit of u into a third unit of v ($u + 2v \rightarrow 3v$, rate uv^2), so v is the self-amplifying activator and u the substrate it depletes — u acts as the inhibitor precisely by running out fastest where v is already abundant. u is continuously resupplied from an external reservoir at rate F (the $F(1 - u)$ feed term), while v is removed both by that same replenishing flow and by an independent decay into an inert product, at the combined rate $F + k$. With $D_u = 0.14$, $D_v = 0.06$, $F = 0.0382$, $k = 0.065$, the inhibitor u diffuses faster than the activator v , the standard short-range-activation/long-range-inhibition requirement for a Turing instability (Section 2.1). Longitudinal veins are modeled as partial, not full, diffusion barriers, reducing local diffusion coefficients by 60% over a width of two lattice sites. This partial permeability is what produces alternation rather than mere spacing: full barriers would isolate each corridor, leaving neighboring rows of spots free to align, whereas the residual inhibitor leaking across veins instead

couples adjacent corridors (Fig. 4b), staggering their activation sites into the shifted lattice already visible at the end of Phase A [3].

Everywhere else the tissue starts from the homogeneous, unpatterned state — substrate abundant, activator absent — and the instability is triggered by a localized initial condition,

$$(u(x, y, t = 0), v(x, y, t = 0)) = \begin{cases} (2.0, 0.75) & \text{at the seed points,} \\ (1, 0) & \text{elsewhere,} \end{cases}$$

one seed point per interveinal compartment. This is a one-time, point-like perturbation, not a continuously maintained source: biologically, it stands in for a brief signal reaching the tepal at its base, after which the Turing instability it triggers grows and spreads on its own, needing no further input. In the large-petal geometry of the extended simulation, seeding from a single edge is not sufficient to sustain the pattern (Section 5): the instability must instead be seeded along both the proximal and distal edges of the domain. This makes the seeding location itself a biologically meaningful parameter rather than a detail — a tepal large enough to need a second, distal trigger implies a second biological signal of its own, one this model does not identify a mechanism for.

4.3 Phase B: Anisotropic transport of a pigment-controlling factor

Each Turing maximum acts as a source of a diffusible pigment-controlling factor C — the same role played by the diffusible activator/inhibitor pair recently identified in a real floral activator-inhibitor pigmentation system [27] — subject to anisotropic transport, modeled here as anisotropic diffusion,

$$\frac{\partial C}{\partial t} = D_x \frac{\partial^2 C}{\partial x^2} + D_y \frac{\partial^2 C}{\partial y^2}, \quad (3)$$

with $D_x = 1.0$ (fast, perpendicular to veins) and $D_y = 0.1$ (slow, parallel to veins). Rather than a one-time deposit left to diffuse freely, each Phase-A activation maximum acts as a continuously maintained reservoir:

$$C(x, y, t) = \begin{cases} 1 & \text{at each Phase-A activation maximum, enforced for all } t \text{ (Dirichlet),} \\ 0 & \text{elsewhere, at } t = 0. \end{cases}$$

Patches are observed to be laterally bounded by veins (Section 3.4), so whatever pigment-controlling determinant is responsible must itself be impermeable to them; veins are accordingly modeled as impermeable to C , confining diffusion to interveinal regions and stretching each circular source into an elongated, ellipse-like domain that is laterally truncated by the veins into a polygon-like shape. Table 1 makes this truncation argument concrete: a 1:10 ellipse (the aspect ratio set by D_x/D_y above) has an SI barely above a thin band, but cutting it laterally down to a square bounding box

— exactly what an impermeable vein does to a Phase-B source that has outgrown its corridor — pushes SI to nearly 100%, since the ellipse is almost flat near its center once $r = b/a$ is this small. Here r is the pre-truncation aspect ratio ($r = 1/10$ above) and the truncation is taken exactly at the point where the remaining width equals the (untouched) height.

4.4 Phase C: Threshold-switch cellular pigmentation response

Phase B’s diffusion equation, Eq. (3), is linear and includes no reaction or decay term: each source, held fixed at $C = 1$, sets up a smooth, anisotropic profile that decreases monotonically away from it, with no mechanism to produce a genuine discontinuity. Reaching the sharp pigmentation boundaries required by design constraint (4) (Section 4.1) therefore needs a further nonlinear step, applied pointwise to this smooth field.

Cells respond to C via a threshold-switch, Hill-type function,

$$f(C) = \frac{C^n}{K^n + C^n}, \quad (4)$$

with $n = 3$ and $K = 0.05$, values well within the range commonly reported for Hill-function switches [28, 29], chosen empirically for the steep, switch-like response they produce, so that a narrow range of C separates fully-white from fully-purple output (Fig. 5). Since C is a non-dimensionalized field in this phase rather than a calibrated physical concentration, no further biologically precise justification of the exact values is meaningful here; the choice is a qualitative one, consistent with the phenomenological character of the rest of the FASHION model. This step is best understood as a biologically plausible transcriptional threshold: it does not alter the domain geometry already fixed by Phase B, only sharpens its boundaries into a near-binary pigmentation mask with crisp cellular edges.

4.5 Numerical simulations

Simulations were performed on a 150×150 lattice, in dimensionless grid units throughout; physical units are assigned only *post hoc* (Section 5.3), by calibrating the pixel size of each comparison group so its mean patch area matches the biological mean exactly, rather than assuming one shared, arbitrary global scale. For the final FASHION model (Phase C, with veins), this calibration gives a physical domain of approximately 9.3×9.3 mm at $\delta x \approx 61.9 \mu\text{m}$ per pixel, the value to use whenever a single model resolution needs to be quoted (Fig. 6). This pixel size, obtained purely by matching patch *area* with no cell-scale input, is independently consistent with a single epidermal cell: reported epidermal cell dimensions for *Tulipa biebersteiniana* (Liliaceae), a related tepal-bearing species, span $\sim 20 \mu\text{m}$ to $40 \mu\text{m}$ in width and $\sim 140 \mu\text{m}$ to $270 \mu\text{m}$ in length [30].

Phases A and B are each integrated with explicit (forward) Euler time-stepping on a standard 5-point finite-difference Laplacian, comfortably satisfying the explicit-scheme stability bound $\Delta t \leq \delta x^2 / (4D_{\max})$ throughout. The two phases differ in

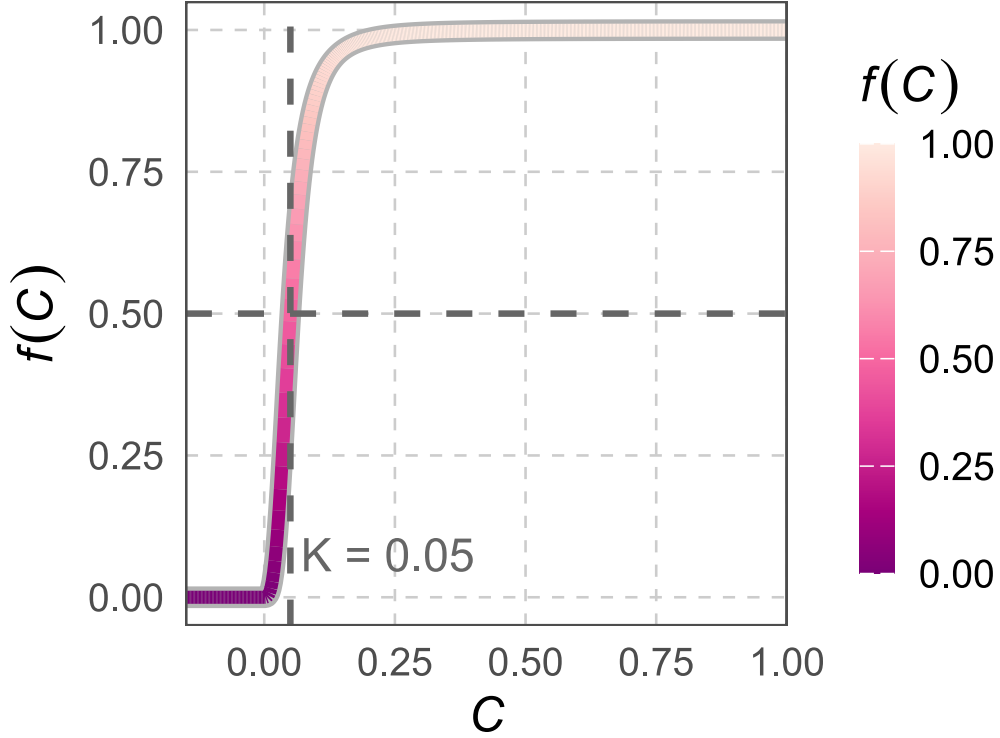


Fig. 5 The threshold-switch Hill-response function, showing an abrupt pigmentation response once the pigment-controlling factor concentration C exceeds the threshold K , as used in the simulated model.

boundary conditions, reflecting their different roles: Phase A (Gray–Scott) uses Neumann (zero-flux) boundaries, while Phase B’s anisotropic transport uses periodic boundaries instead. Neither phase is run to a literal steady state: Phase A integrates a fixed 6×10^4 Gray–Scott steps regardless of parameters, and Phase B then integrates 1.05×10^3 anisotropic-diffusion steps on the resulting prepattern. This is deliberate, not a shortcut around a “true” steady state: unbounded pure diffusion (Eq. (3) has no reaction or decay term) has no nontrivial steady state on a periodic domain — at $t \rightarrow \infty$ it flattens to a spatially uniform value, which would leave Phase C’s threshold with nothing but noise to sharpen. Phase C involves no time integration at all: it is a pointwise algebraic transform (the Hill function of Section 4.4) applied directly to the colorant field produced by Phase B. Each phase is implemented as a short, self-contained script (Section 6), including a standalone vein-free Turing simulator that reproduces the simulated triplet of Fig. 3a–c on its own. Simulated patches are detected by thresholding the relevant field and labeling connected components (threshold 0.15 for the vein-free Turing baseline, 0.5 for the FASHION model and the ideal geometric checkerboard), the same connected-component approach used, after Cellpose segmentation, on the photographed patches (Section 3.2).

In Phase A, the Gray–Scott system generates staggered activation sites across inter-veinal corridors. In Phase B, anisotropic transport expands these sites into bounded

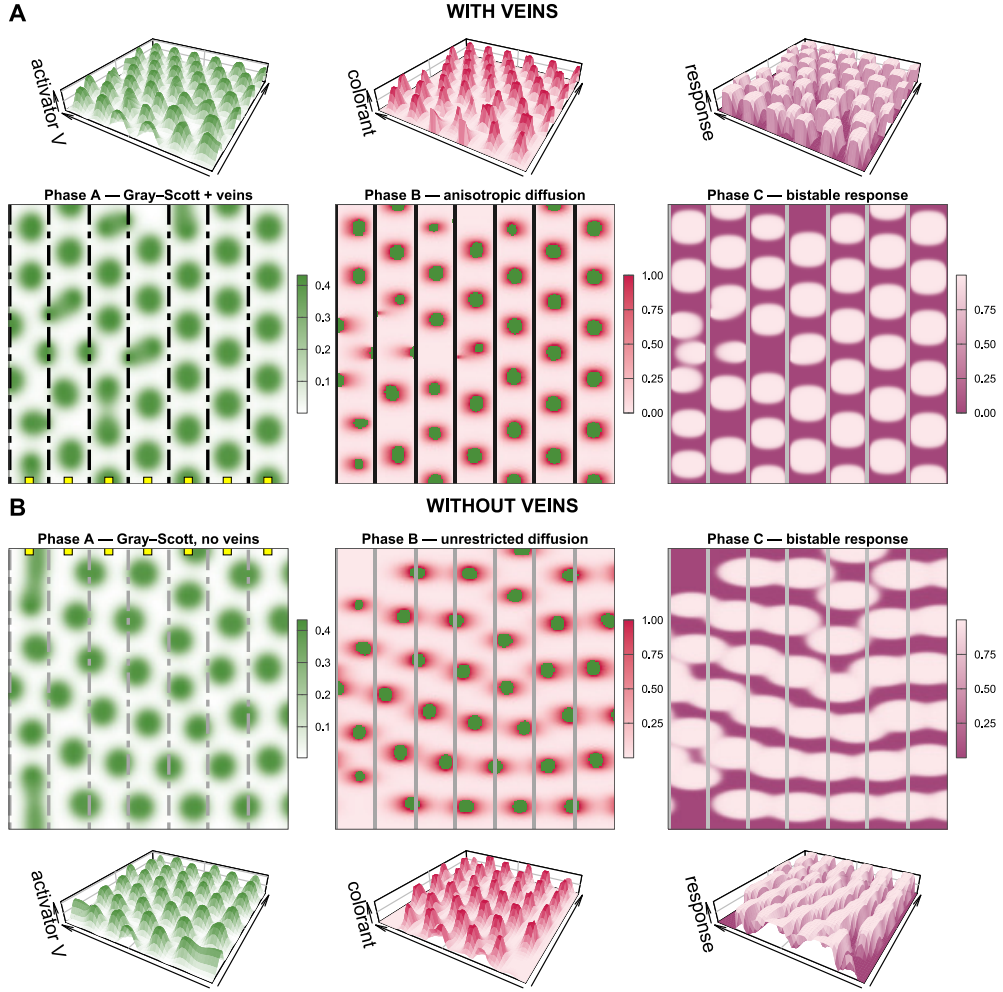


Fig. 6 Simulation pipeline of the FASHION model, (a) with and (b) without vascular veins. Each panel shows the same three sequential phases, left to right — Phase A (Gray-Scott prepattern), Phase B (diffusion of the pigment-controlling factor), Phase C (threshold-switch response) — as two-dimensional field maps, alongside three-dimensional renderings of the activator, colorant, and response fields. With veins (a), activation sites are confined to and staggered between interveinal corridors, and sharpen into the square, alternating checkerboard. Without veins (b), the same sequence instead produces unconstrained, elongated stripes. Yellow squares on the Phase-A panels mark the localized seed points of the initial condition (Section 4.2).

polygonal domains. In Phase C, the threshold switch sharpens boundaries and produces high-contrast pigmentation (Fig. 6a). Together the three steps reproduce a discontinuous, spatially constrained checkerboard pattern closely resembling that of *Fritillaria meleagris*.

5 From Turing spots to checkerboard: a quantitative progression

5.1 Realistic imperfections and defects

The simulated pattern is not perfectly regular: truncated domains, half-patches adjacent to veins, and positional shifts arise naturally, closely resembling the defects observed in real flowers (cf. Fig. 1b).

5.2 Restricted parameter space and rarity of the motif

Checkerboard formation occurs only within a narrow parameter window, bounded by distinct failure modes rather than simple degradation. Vein permeability must be high enough to keep nucleation off the vein itself, but not so high as to starve a corridor of the substrate exchange its Gray–Scott autocatalysis needs to replicate patches along its length: neither limiting case (0% or 100% permeability) supports alternation. In the underlying vein-free Gray–Scott system, Eq. (2), the diffusion coefficients D_u, D_v are markedly more sensitive still: a joint increase of only about ten percent — which, for an isotropic Turing instability, should merely rescale the wavelength — is already enough to collapse the pattern entirely. Seeding the instability from a single domain edge is similarly fragile: the replication front stalls in a persistent fraction of compartments regardless of F , run time, or grid resolution — finer grids make the stall worse, not better, ruling out a simple discretization artifact — and is resolved only by seeding from both the proximal and distal edges (Section 4.2). Seeding with random noise in u, v across the whole domain, rather than at these two localized edges, is worse still: activation sites tend to agglomerate against the veins and the pattern collapses.

The vein-free control (Fig. 6b) reinforces the anisotropy argument of Section 3.4 directly: across the full range of F, k explored, it never settles on a square lattice, instead selecting a hexagonal, staggered arrangement — the generic quadratic-resonance channel of Section 2.3, now showing up in the simulation itself. Breaking the square/stripe degeneracy alone is therefore not sufficient, confirming that the vein geometry is doing real work suppressing hexagons, not just tipping an already-symmetric square/stripe competition. Together, these failure modes explain both the pattern’s intraspecific stability and its rarity in nature.

5.3 SI comparison across groups

We compared SI across four groups (Fig. 7a): the classical Turing baseline with no vein confinement, the full FASHION model (all three phases, with veins), an idealized geometric checkerboard, and biological measurements from *Fritillaria meleagris* tepals.

The geometric checkerboard sits close to the ceiling ($SI = 98.3\% \pm 2.4\%$ SD, $n = 162$) — not exactly 100% even for this idealized reference, since it too is built on a pixel-rasterized grid. The vein-free Turing baseline lands close to the circular reference ($SI = \pi/4 \approx 79\%$), as expected in an isotropic medium with no square-selecting mechanism. Adding the full FASHION pipeline moves SI clearly above that baseline, closing roughly a third of the remaining gap to the ideal square, and all four groups differ significantly from one another (Fig. 7a).

The biological *Fritillaria* patches (Section 3.2; $n = 644$ patches, $SI = 58.9\% \pm 12.0\%$ SD) sit *below* every other group, including the circular reference — around a fifth lower than the vein-free baseline, and well below the full model. Real patches are not simply noisy squares: they are measurably rougher than *either* idealized reference shape. In addition to imperfect segmentation (photographed patch boundaries are pixel-jagged, unlike clean simulated masks), vein-adjacent truncation, fusion between neighboring patches, and biological variability in pigment deposition genuinely give real domains more irregular outlines than either simulated shape reproduces. The biological SI is therefore better read as evidence that real patches occupy a rougher shape category than the smooth geometric references used for the other three groups, not as a failure of the square-selecting mechanism itself.

Patch *area* makes the same point more cleanly once the physical scale of each group is calibrated so that its mean patch area matches the biological mean exactly (Section 4.5): with the means pinned to the same value, any remaining difference in spread is purely a difference in shape regularity, not an artifact of comparing two different absolute scales. The FASHION model’s patches come out far more uniform in area (SD 0.22 mm^2 around the shared mean of 1.46 mm^2 — a coefficient of variation of about 15%) than the real ones (SD 0.87 mm^2 , a coefficient of variation of about 60%, roughly fourfold higher). This follows directly from the modeling simplification: patch area in the model is set by the spacing between straight, exactly evenly-spaced veins, whereas real veins are neither perfectly straight nor evenly spaced, so the interveinal corridor width — and with it, patch area — fluctuates along and across the tepal in a way the idealized vein geometry used here cannot capture.

5.4 Progressive transformation across model phases

Figure 7b tracks SI through the three phases of the FASHION model, run in parallel with and without the vascular veins.

With veins present, SI rises from Phase A to Phase B and then holds exactly flat into Phase C, for a net increase of roughly a fifth overall. That Phase C leaves SI completely unchanged is itself informative: since the Hill threshold (Eq. (4)) is pointwise and monotonic, it cannot alter geometry already fixed, and the measured SI confirms this directly rather than merely by construction. Anisotropic transport (Phase B, Eq. (3)) stretches sources into elongated, ellipse-like domains that are laterally truncated by the veins, and it is specifically the threshold-switch response (Phase C, Eq. (4)) that sharpens these truncated boundaries into a visibly square outline. Without veins, the trend is downhill throughout instead, ending at roughly a fifth of its starting value as unconstrained maxima elongate into stripes; by the final phase the two conditions are nearly sixfold apart — the clearest evidence that the square shape is vein-driven, not an incidental byproduct of the Gray–Scott or Hill-response kinetics alone.

Two-dimensional alternation, by contrast, is already fixed structurally in Phase A, once veins partition the domain into staggered corridors (Section 4.2), independently of the later shape-sharpening: *vein geometry sets the alternation, the threshold switch sharpens boundaries into a visibly square shape*, and the two trajectories only agree once Phase B and C act on the vein-partitioned geometry — at Phase A alone, vein

confinement briefly *lowers* SI relative to the unconstrained case, since it forces still-round spots into elongated corridor shapes.

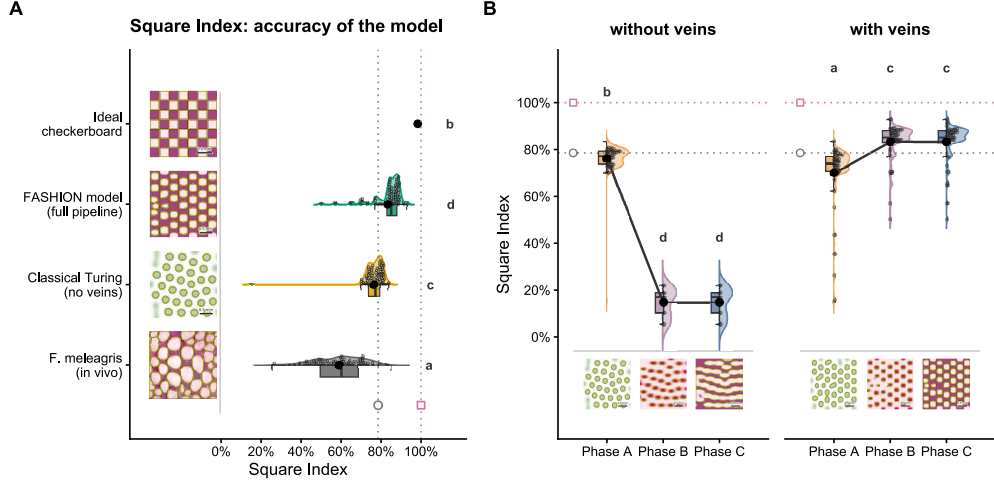


Fig. 7 Square Index, quantitative comparison. (a) SI across the classical vein-free Turing baseline, the full FASHION model, an ideal geometric checkerboard, and biological *Fritillaria meleagris* patches; dashed lines mark the circle ($SI = \pi/4 \approx 79\%$) and square ($SI = 100\%$) references. Compact-letter-display annotations (Kruskal-Wallis test with Dunn post-hoc) mark pairwise significance. (b) SI at each of the three FASHION model phases, run with and without vascular veins, showing the two trajectories diverge from Phase A onward.

6 Conclusion

Our results demonstrate that a checkerboard pigmentation pattern can emerge from classical reaction–diffusion mechanisms when embedded in anisotropic and geometrically constrained media. By coupling a Turing instability to vascular architecture and threshold-switch cellular responses, the FASHION model generates a noncanonical motif that lies outside the standard repertoire of Turing patterns.

The Square Index provides a physically motivated shape metric for such patterns, capturing what distinguishes a square domain from a Turing spot, and may be useful beyond this specific system wherever the distinction between isotropic Turing-like structures and geometrically ordered lattices needs to be made quantitative. The complementary property, strict spatial alternation, is accounted for here structurally — by the vein-imposed compartmentalization of Section 3.4 — rather than through a dedicated statistic.

This is an explanatory, not a validated, mechanism: each of the three steps is a physically plausible candidate rather than an experimentally confirmed one. No activator-inhibitor pair has been molecularly identified for the Gray–Scott prepattern in *Fritillaria*. The Hill-type response (Eq. (4)) is a phenomenological fit chosen for its switch-like shape rather than a measured dose-response curve. Finally, the anisotropic polarized transport (Section 3.4) is inferred by analogy to auxin transport rather than

directly measured in tepal tissue. The model demonstrates that this combination of mechanisms is *sufficient* to reproduce the checkerboard’s geometric signature; it is not evidence that this is the pathway the plant actually uses. A further limitation is timing: Phase C reads out the colorant field at a fixed simulated time rather than at a true dynamical steady state (Section 4.5), implicitly assuming some biological clock that determines when this readout happens. We have not identified a plausible mechanism for such timing, though we have not found one that rules it out either.

More broadly, this work illustrates how tissue geometry and transport anisotropy can reshape the outcomes of diffusion-driven instabilities, offering both a physical explanation for a rare biological pattern and a case study in how geometric constraints can steer reaction–diffusion instabilities into non-canonical regimes. The tepal itself grows during development, and coupling the FASHION model to a growing domain — known to reshape Turing patterns through peak splitting and insertion [31, 32] — is a natural next step left for future work.

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Declarations

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Competing interests

The authors have no relevant financial or non-financial interests to disclose.

Authors’ contributions

J.P.: Conceptualization, Writing – review & editing. P.L.: Conceptualization, Writing – review & editing. A.M.: Conceptualization, Methodology, Software, Formal analysis, Visualization, Writing – original draft, Writing – review & editing.

Use of artificial intelligence tools

Large language model assistance (Claude, Anthropic) was used at two stages of this work. First, the manuscript was drafted and structured with AI assistance, including journal-format adaptation and L^AT_EX typesetting. Second, AI assistance was used in the development of the numerical simulation code (Python) and the data-visualization and figure-preparation scripts (R and Python). All scientific content, methodology, results, and conclusions are the authors’ own. The authors reviewed all AI-assisted material and take full responsibility for the accuracy and integrity of the published work.

Data availability

Simulation code is archived on Zenodo at <https://doi.org/10.5281/zenodo.21944387> and actively maintained at <https://github.com/adamambaye-ens/Fritillaria>. The repository includes three self-contained, runnable scripts: (1) a classical Gray–Scott Turing-pattern simulator (vein-free, cf. Section 2.2), (2) the full three-phase FASH-ION model (Section 4), and (3) an animation script that generates the phase-by-phase pattern-formation movie summarized in Fig. 6. All three depend only on NumPy and Matplotlib (Numba is an optional accelerant for the animation script; ffmpeg is required to encode the movie) and run in under two minutes each on an ordinary laptop, except the animation script, which takes about six minutes. The repository also includes two large-domain (15-vein) variants of scripts (1) and (2), used to generate the quantitative statistics reported in Section 5.

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