

Emergent cell migration from cell shape deformations and T1 transitions

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T1 transitions—cell neighbor exchanges—are key drivers of tissue remodeling and fluidization in epithelial monolayers. Using a multiphase field (MPF) model that accounts for dynamic cell shapes, alongside an active elastic solid model lacking cell shape degrees of freedom, we study the spatiotemporal organization of T1 transitions. While individual T1 transitions occur as a Poisson process in time, their spatial distribution is strongly influenced by local cell shape deformations. We identify a dual mechanical response: cells losing neighbors tend to relax their shape isotropically, whereas cells gaining neighbors undergo anisotropic elongation. By analyzing successive T1 events in the MPF model, we find asymmetric spatial patterns that depend on whether a cell gains or loses neighbors. These asymmetries lead to persistent, localized directional motion, even in the absence of external cues. Our results suggest an intrinsic mechanism for directed cell migration driven by local topological rearrangements mediated by cell shape dynamics—a process potentially relevant for epithelial flows during development, morphogenesis, and wound healing.

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I. INTRODUCTION

Confluent epithelial monolayers, being composed of tightly packed cells with stable cell-cell junctions, exhibit a complex interplay between tissue rigidity, collective migration, and localized fluidization [1–3]. These mechanical processes are also important in assisting biological phenomena such as morphogenesis, cancer invasion, and wound healing [4–6]. In a confluent tissue monolayer, cells can migrate collectively as a coordinated supracellular unit, or they can rearrange relative to one another through neighbor exchanges—known as T1 transitions—with adjacent cells [1]. Even when cells move as coherent clusters, the cells at the outer edges of these clusters undergo T1 transitions when they move with different velocities relative to their neighbors.

T1 transitions, observed in active cell monolayers [7] and in foams under external forces [8], are fundamental topological transitions. During one T1 transition, a junction between two neighboring cells shrinks and disappears, while two adjacent cells form a new junction typically oriented perpendicular to the original one. T1 transitions result in changes in neighborhood connectivity without any change in cell number. Although the geometric features of T1 transitions are well understood, the underlying mechanisms triggering these events vary significantly. In passive systems such as soap films, T1 transitions are driven by external forces [9]. However, for biological tissues, these transitions are influenced by intrinsic cellular properties, such as active cell migration [10,11], or

mechano-chemical feedback involving actomyosin and junctional protein complexes such as adherens junctions [6,12,13]. While T1 transitions are important for the dissipation of junctional stresses and facilitating solid-fluid transitions in tissues [14–16], the mechanisms driving these events remain incompletely understood.

T1 transitions sustained by cell activity induce large deformations in cell shape, associated with an energy barrier [10,17]. Previously, we have shown that T1 transitions triggered by cell activity induce transient quadrupolar flows, leading to the relative dispersion of cells [11]. Interestingly, the rate of relative dispersion is determined by the mean occurrence rate of T1 transitions, regardless of the underlying mechanisms controlling how T1 transitions may occur. However, the spatiotemporal patterns of T1 transitions, hypothesized as chains or cascades of T1 transitions [10,18] propagating across the tissue to induce tissue fluidization and directional migration on larger scales, remain elusive.

To address this, we use two complementary modeling approaches that allow us to disentangle the role of cell shape dynamics from purely topological rearrangements during T1 transitions. While certain features of T1 transitions—such as their Poissonian temporal occurrence or cell shape elongation—might be anticipated from general considerations of active matter or vertex-based models, a systematic comparison across models with and without explicit cell shape dynamics allows us to assess the robustness of these features to different underlying mechanisms. In particular, we aim to test whether temporal randomness in T1 transitions entails spatial randomness in junction remodeling, and to determine how cell shape-dependent junction remodeling contributes to nontrivial spatial correlations and directed migration that are not captured by topological rearrangements alone.

The multiphase field (MPF) model is particularly useful for capturing complex, nonpolygonal cell shapes and their dynamics, thus allowing us to track shape changes and junctional

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remodeling explicitly. MPF models have been used to study several emergent properties of epithelial monolayers, such as topological features including T1 transitions [10,11,19], p -atic order [20–22], and cell colony growth [23,24]. We use this model to explore how the feedback between cell shape and topological transitions contributes to directional cell migration. In contrast, the active elastic solid (AES) model is also a useful minimal model for elastic tissues, where cells are represented as pointwise self-propelled agents with self-aligning torques determined by the elastic springlike forces between cells [25,26]. This model has been used to study polar ordering in directional migration of activated epithelial monolayers [27]. We enhance this model to enable neighbor exchanges based on a geometric condition for the minimal neighbor distance [28]. The AES model lacks internal shape degrees of freedom, and serves to study neighbor exchanges as topological transitions in the cell-cell connectivity graph without the complexity of shape dynamics.

By contrasting these models, we find that individual cells undergo T1 transitions as a Poisson process in time, with a rate that depends on the self-propulsion speed. This implies that the temporal occurrence of T1 transitions is a generic feature of topological rearrangements in the cell neighbor graph. In contrast, the spatial distribution of T1 transitions is strongly influenced by cell shape dynamics. Within the MPF model, where cell shape deformations are explicitly tracked through phase fields, we observe that successive T1 transitions are anisotropically distributed across a cell's junctions. This anisotropy arises from distinct cell shape responses: cells losing neighbors tend to relax isotropically, while those gaining neighbors elongate along specific axes. These spatially biased patterns of T1 activity can give rise to localized, directional cell motion, which may serve as a fundamental mechanism for emergent tissue-scale flows in confluent epithelial layers.

In Sec. II we introduce T1 transitions in the MPF and AES models. We demonstrate that, in both models, T1 transitions per cell occur as temporal events governed by cell activity, with their occurrence rate following a Poisson process, as discussed in Sec. III. Using radial distribution functions, we observe that T1 transitions exhibit spatial correlations that decay over time, supporting the hypothesis of T1 transition chaining, as elaborated in Sec. IV. Section V shows that T1 transitions induce distinct shape deformations, with cells losing neighbors relaxing their shape, while those gaining neighbors elongate. In Sec. VI we analyze successive T1 transitions undergone by a cell and demonstrate that their spatial distribution is anisotropic and can lead to the emergence of directional migration. Finally, in Sec. VII we summarize the findings and provide an outlook on future research directions.

II. T1 TRANSITIONS

We consider a 2D confluent monolayer of N cells, with their index labels in the set $\mathcal{M} = \{1, 2, \dots, N\}$. The neighbor connectivity is expressed in terms of a symmetric neighbor adjacency matrix $\mathcal{N}_{N \times N}$ at time t as

$$\mathcal{N}_{ij}(t) = \begin{cases} 1: & i, j \in \mathcal{M} \text{ are neighbors at } t \text{ and } i \neq j \\ 0: & \text{otherwise} \end{cases}.$$

TABLE I. Default values of the model parameters. dt denotes the time step size.

N	L	T	ϵ	v_0	Ca	In	a_a	a_r	D_r	α	dt
100	100	150	0.15	0.5	0.2	0.1	1	1	0.01	0.1	0.005

A. Multiphase field approach

In a multiphase field (MPF) model, each cell i is represented by a phase field ϕ_i defined such that $\phi_i \approx 1$ inside the cell and $\phi_i \approx -1$ outside it. The cell boundary is characterized by a thin diffuse region where the phase field transitions smoothly between these two bulk values, with cells defined as neighboring when there is a sufficient overlap of their diffuse boundaries [10]. Such a representation allows us to model cells of arbitrary shapes. The evolution of the phase field is governed by diffusive relaxation (conservative dynamics) aimed at minimizing a free energy functional, with an advection term due to cell self-propulsion. The mathematical model formulation is described in Appendix A and has been previously used in Ref. [11]. The MPF model parameters are as per Table I unless specified otherwise. The conservative dynamics preserves the cell area.

A T1 transition, denoted by $\mathcal{T}$, is a neighbor-exchange event of finite duration that involves four cells, labeled as A, B, C, D . $\mathcal{T}$ starts at time $t = t_{\mathcal{T}}^-$ when cells C and D lose contact and concludes at $t = t_{\mathcal{T}}^+$ when cells A and B establish contact. This rearrangement process is illustrated in Figs. 1(a)–1(c). Before $t_{\mathcal{T}}^-$, the junction shared by cells C and D shrinks and vanishes at $t_{\mathcal{T}}^-$, and a new junction between A and B , approximately perpendicular to the previous one, is created at $t_{\mathcal{T}}^+$ and subsequently starts to lengthen [11]. Thus, T1 transitions in the MPF model result in the remodeling of cell-cell junctions. This T1 transition can be represented by the set $\mathcal{T} = \{A, B, C, D\} \subset \mathcal{M}$ such that

$$\begin{aligned} \mathcal{N}_{CD}(t_{\mathcal{T}}^-) = 1 &\Rightarrow \mathcal{N}_{CD}(t_{\mathcal{T}}^+) = 0, \\ \mathcal{N}_{AB}(t_{\mathcal{T}}^-) = 0 &\Rightarrow \mathcal{N}_{AB}(t_{\mathcal{T}}^+) = 1, \end{aligned}$$

while all other connections shared among the four cells remain unchanged during the T1 duration $\Delta t_{\mathcal{T}} = t_{\mathcal{T}}^+ - t_{\mathcal{T}}^-$. Cells A and B , which gain neighbors during the T1 transition, are referred to as *gainer* cells, while cells C and D , which lose neighbors, are referred to as *loser* cells. During the T1 transition, an extracellular gap is formed between the four cells in the MPF model, as shown in the Fig. 1(b). While neighbor exchanges involving more than four cells, forming higher-order rosette structures, are possible in the MPF model, they are extremely rare events in the explored parameter regime and thus their influence is ignored.

A three-way vertex denoted by the subset $\mathcal{X} = \{i, j, k\} \subset \mathcal{M}$, is a set of three cells which are mutually adjacent, i.e., $\mathcal{N}_{ij}(t) = \mathcal{N}_{jk}(t) = \mathcal{N}_{ki}(t) = 1$, at a given time t . The set of all vertices at time t is denoted by $\Sigma_{\mathcal{X}}(t)$. For a unique $\mathcal{N}(t)$, there exists a unique $\Sigma_{\mathcal{X}}(t)$, which represents a particular neighborhood topology. T1 transitions are topological changes of $\mathcal{N}$ (or equivalently $\Sigma_{\mathcal{X}}$). From hereon, we will refer to a three-way vertex as just a vertex.

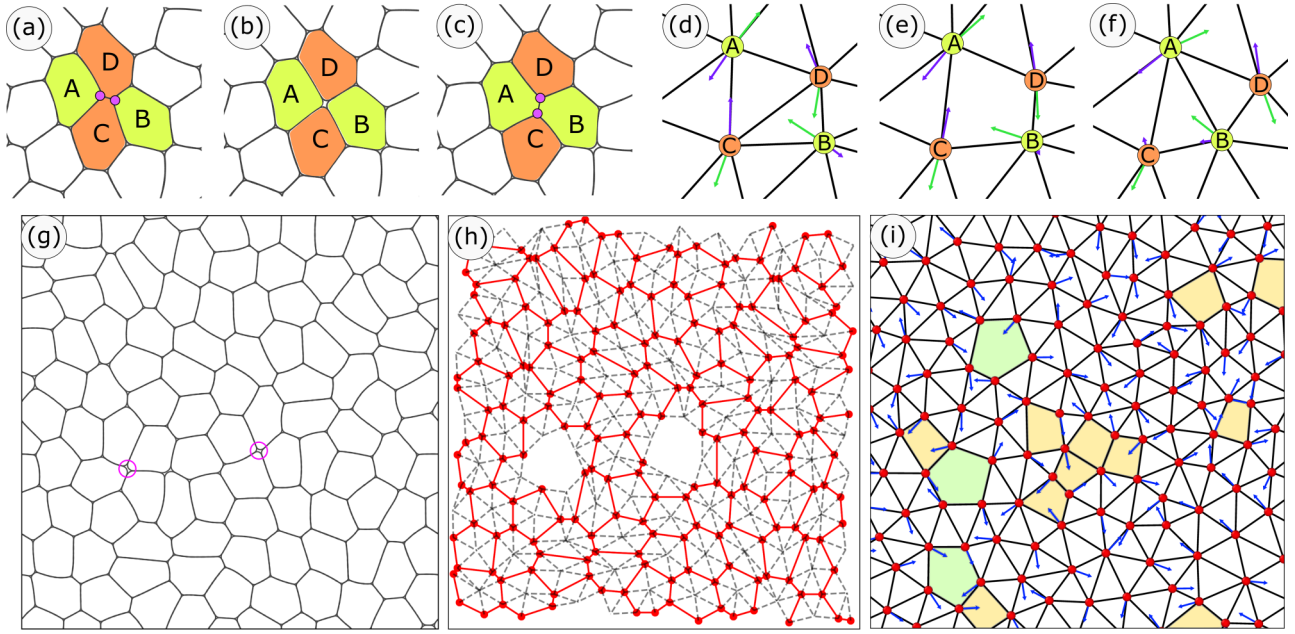


FIG. 1. T1 transition in the MPF model (a)–(c) and in the AES model (d)–(f). (a), (d) before T1; (b), (e) during T1; (c), (f) after T1. A T1 transition $\mathcal{T} = \{A, B, C, D\}$ where cells A and B are loser cells, while cells C and D are gainer cells. (a)–(c) Pink markers corresponds to location of vertices that take part in the T1 transition. (d)–(f) AES: Black line correspond to cell-cell junctions; orange and green circles mark the loser and gainer cells, respectively. The green arrows represents the cell polarity, i.e., direction of self-propulsion, while the purple arrow represents the net elastic force. (g) A snapshot of a tissue in the MPF model with the pink circle marking the location of the T1 transitions. (h) The vertices are marked in red, and vertices that are common to two cells are linked by red lines. The black dashed lines connect the vertices to the center of mass of the cells. The gaps in the plot correspond to T1 transitions. (i) Tissue snapshot of AES model. Cells are marked in red, and a black bar connects neighboring cells. Triangles indicate vertices, yellow trapeziums indicate T1 transitions, and green polygons denote higher order rosettes. See Movie 1 and Movie 2 in the Supplemental Material [29] for simulations in the MPF model and AES model, respectively.

We determine the position of the vertex $\mathcal{X}$ at time t , denoted by $\mathbf{r}_{\mathcal{X}}(t)$, as the location of the minimum value of the sum of its distances to the boundaries of all three cells. The boundary of a cell i is implicitly represented by the contour corresponding to $\phi = 0$, and is represented by the zero-level set $Z_i(t)$ such that

$$Z_i(t) = \{\mathbf{x} \in \Omega \mid \phi_i(\mathbf{x}, t) = 0\}. \quad (1)$$

The position of the vertex $\mathcal{X}$ is then given as

$$\mathbf{r}_{\mathcal{X}}(t) = \arg \min_{\mathbf{x}} \left\{ \sum_{j \in \mathcal{X}} |\mathbf{x} - \mathbf{z}_j| : \forall \mathbf{z}_j \in Z_j(t) \right\}. \quad (2)$$

In Fig. 1(g) we show a typical snapshot of a cell configuration in the MPF model, while in Fig. 1(h) we plot the graph of the corresponding vertices. For each T1 transition $\mathcal{T}$, at time $t_{\mathcal{T},1}^-$, two vertices $\mathcal{X}_{\mathcal{T},1}^- = \{A, C, D\}$ and $\mathcal{X}_{\mathcal{T},2}^- = \{B, C, D\} \subset \mathcal{T}$ move toward each other and annihilate [Fig. 1(a)]. Similarly, at $t_{\mathcal{T},1}^+$, two new vertices $\mathcal{X}_{\mathcal{T},1}^+ = \{A, B, C\}$ and $\mathcal{X}_{\mathcal{T},2}^+ = \{A, B, D\} \subset \mathcal{T}$ are spawned [Fig. 1(c)]. Notice that the small gaps formed between cells during T1 transitions correspond to holes in the *graph of vertices* as shown in Fig. 1(h).

As localized processes of remodeling cell-cell junctions, T1 transitions induce large fluctuations in cell shape and speed, generating transient flows with quadrupolar patterns that result in an increase in the pair-separation distance between cells [10,11].

B. Active elastic solid approach

To better understand the interplay between cell shapes (thus cell junction dynamics) and T1 transitions, we also consider an active elastic solid (AES) model where the tissue is idealized as an elastic sheet formed by pointwise cells which are self-propelled and interact with each other through elastic forces. The deterministic evolution of the polarity for the self-propulsion is determined by the torque induced by misalignments between the net elastic force and the cell polarity [26]. Although this model features tissue elasticity, it is not directly related to the interfacial forces as is the case for the MPF model. The model is detailed in Appendix B and based on the one introduced in Ref. [25]. AES model parameters are as per Table II unless specified otherwise.

The neighboring cells are initially connected by a predefined cell connectivity graph. In previous works, this graph was quenched in time, resulting in a solidlike response when the tissue is out of its elastic equilibria, or a polar collective migration due to alignment of polarities [25]. We augment this model by introducing a geometric relation of nearest neigh-

TABLE II. Default values of the model parameters for AES. dt denotes the time step size.

L	T	l_{ij}	$1/a_0^2$	ζ	D_r	D_p	dt	v_0
40	5000	2	0.08	1	0.025	0.15	0.1	0.05

bors which is the same as the one proposed in Refs. [28,30] to allow for neighbor exchanges. Explicitly, we connect all cells i and j and remove the connection if there exists any third cell k which satisfies $r_{ij}^2 > r_{ik}^2 + r_{jk}^2$, with r_{ij} being the distance between cells i and j . Most of the neighbor exchanges correspond to T1 configurations as shown in Figs. 1(d)–1(f) and are marked by the yellow quadrilateral shapes in the connectivity graph [see Fig. 1(i)]. However, neighbor exchanges involving five cells are also present, as illustrated by pentagonal shapes in Fig. 1(i).

For the AES model, note that the T1 transitions are topological changes in the *connectivity graph* and are not directly related to three-way vertices as in the MPF model. In the MPF model, two cells are considered neighbors if their shape boundaries sufficiently overlap, such that topology changes in the connectivity graphs correspond to junction remodeling. In the AES model, cells are considered neighbors if they conform to a specific geometric configuration of cell positions. This geometric dependence of neighborhood topology allows for neighbor exchanges with four or more cells, with neighbor exchanges involving four cells being the most common. By contrast, in MPF models, neighbor exchanges almost always involve four cells within the parameter regime explored. For simplicity, we also use the symbol $\mathcal{T}$ to denote neighbor exchanges in the AES model, even if they involve more than four cells. In the AES model, for our analysis, neighbor exchanges involving more than four cells are counted as one transition, and the algorithm for detecting neighbor exchanges is described in Appendix B.

In the AES model, when the self-propulsion is high, the cells are driven far out of their equilibrium positions imposed by elastic interactions, resulting in a highly nonuniform distribution of distances between particles, analogous to that of a gas. This is an unrealistic limit case for epithelia. Therefore, the AES with neighbor exchanges is valid at sufficiently small activities, where there is still a well-defined reference state for elastic deformations, which is only punctually destroyed by neighbor exchanges. Thus, we restrict ourselves to situations with moderate self-propulsion speeds. Both models have a connectivity graph, encoded within $\mathcal{N}$, where T1 transitions occur as localized neighbor exchange events.

III. WAITING TIME BETWEEN T1 TRANSITIONS

To quantify the spatiotemporal relations between T1 transitions, we introduce the notion of two *successive* T1 transitions $\mathcal{T}_m$ and $\mathcal{T}_n$ involving cell i , i.e., $i \in \mathcal{T}_m$ and $i \in \mathcal{T}_n$. We consider two T1s involving a cell i , *successive* if $t_{\mathcal{T}_m}^+ < t_{\mathcal{T}_n}^-$, and if cell i is not part of any other T1 between $t_{\mathcal{T}_m}^+$ and $t_{\mathcal{T}_n}^-$. The waiting time $t_w(\mathcal{T}_m, \mathcal{T}_n)$ between *successive* T1 transitions $\mathcal{T}_m$ and $\mathcal{T}_n$ is defined as the time interval between the end of $\mathcal{T}_m$ and start of $\mathcal{T}_n$, i.e., $t_w(\mathcal{T}_m, \mathcal{T}_n) := t_{\mathcal{T}_n}^- - t_{\mathcal{T}_m}^+$. The probability distribution of t_w provides insight into the temporal occurrence patterns of T1 transitions.

Figures 2(a) and 2(b) show the distributions of waiting times t_w for varying activity (v_0) in the MPF and AES models, respectively. In both cases, the waiting time is exponentially distributed, as expected for a homogeneous Poisson process. The average waiting time decreases with increasing activity as cells that move faster are likely to undergo their next T1

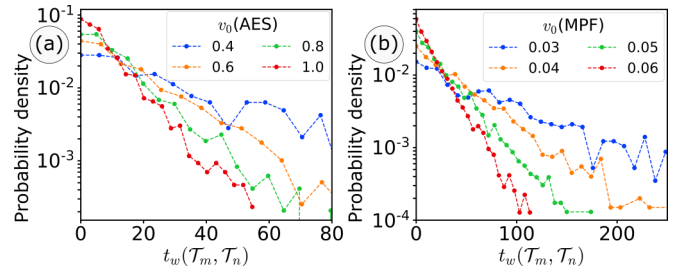


FIG. 2. Waiting time t_w probability density distribution between successive T1s $\mathcal{T}_m$ and $\mathcal{T}_n$ undergone by a cell. (a) MPF model: cell activity v_0 is varied. (b) AES model: cell activity v_0 is varied.

sooner. The mean waiting time does not vary significantly with varying deformability Ca (see Fig. 1 in the Supplemental Material [29]). Cells with lower deformability typically undergo fewer T1 transitions; however, these T1 transitions have a larger duration [10]. In both models, the mean waiting time is much larger than the duration of a T1 transition, indicating that T1 events are well separated in time, i.e., discrete events.

While T1 transitions appear Poissonian in time, we show next that T1 transitions are correlated in space. Later we will use the cell shape of the MPF to describe the spatial correlations in the MPF model.

IV. SPATIAL CORRELATION OF T1 TRANSITIONS

To study the spatial correlations between T1 events, we compute the radial distribution function for pairs of T1 events. We first compute the epicenters of T1 transitions. Within the MPF model, the epicenter $\mathbf{r}_{\mathcal{T}}$ of a T1 transition $\mathcal{T}$ is empirically defined as the location where the sum of distances to the boundaries of all four cells involved in the T1 transition is minimized. This is measured at the time midway through the T1 transition, i.e., at $t_{\mathcal{T}}^m = 0.5(t_{\mathcal{T}}^+ + t_{\mathcal{T}}^-)$ [10,11]. The location of the epicenter is given by

$$\mathbf{r}_{\mathcal{T}}(t) = \arg \min_{\mathbf{x}} \left\{ \sum_{j \in \mathcal{T}} |\mathbf{x} - \mathbf{z}_j| : \forall \mathbf{z}_j \in Z_j(t_{\mathcal{T}}^m) \right\}. \quad (3)$$

The epicenter lies within the extracellular gap created during the T1 transition, as shown in Fig. 1(b). In the AES model, the epicenter is defined as the average of the positions of the four cells involved in $\mathcal{T}$,

$$\mathbf{r}_{\mathcal{T}}(t_m) = \frac{\mathbf{r}_A + \mathbf{r}_B + \mathbf{r}_C + \mathbf{r}_D}{4}, \quad (4)$$

with time taken at the end of the T1 transition, $t_{\mathcal{T}}^m = t_{\mathcal{T}}^+$.

For a specific time window of size ω , we compute the radial distribution function for epicenters of all T1 transitions that occur within that window. Suppose, in a given time window of size ω starting from time t until time $t + \omega$, there are N_{T1} events and let $\rho = \frac{N_{T1}}{A}$ be the number density of T1 events, where A is the area of the computational domain. Then the radial distribution function for this particular time window is calculated as

$$g(r, t, t + \omega) = \frac{1}{\rho N_{T1}} \left\langle \sum_{j=1}^{N_{T1}} \sum_{k \neq j} \delta(r - r_{jk}) \right\rangle, \quad (5)$$

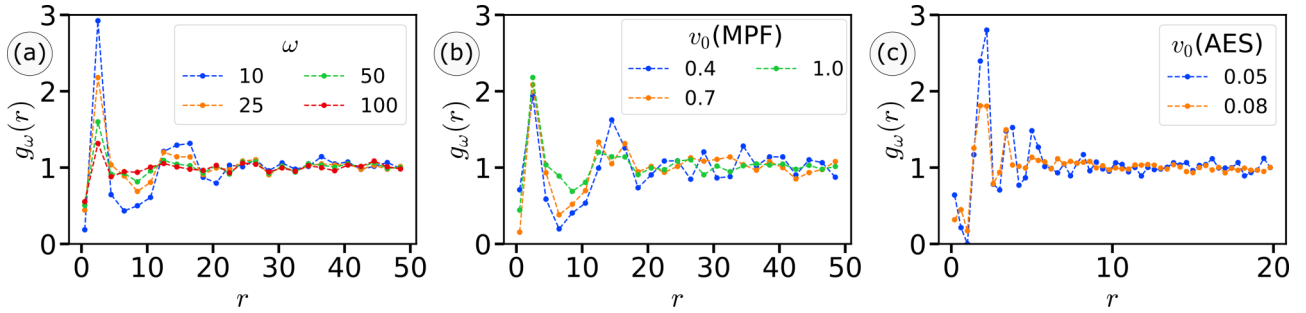


FIG. 3. Radial distribution function $g_\omega(r)$ between T1 transitions for MPF model (a), (b) and AES model (c). (a) $g_\omega(r)$ for different time windows for $v_0 = 1$. (b) $g_\omega(r)$ for varying v_0 with $\omega = 25$. (c) $g_\omega(r)$ for varying v_0 with $\omega = 10$.

where δ is the Dirac delta function and r_{jk} is the distance between the epicenters of a pair of T1 transitions indexed by j and k . If τ is the time resolution, then the radial distribution function is calculated as the ensemble average of time windows, $\{(0, \omega), (\tau, \omega + \tau), (2\tau, \omega + 2\tau), \dots, (T - \omega, T)\}$, such that

$$g_\omega(r) = \frac{\tau}{(T - \omega + \tau)} \sum_{t=0}^{t=T-\omega} g(r, t, t + \omega). \quad (6)$$

Figure 3(a) shows the radial distribution function for the MPF model for varying time window sizes ω . The location of the peaks does not vary with window size, but decrease in magnitude. For small ω , $g_\omega(t)$ appears typical of disordered systems with interacting particles. As epicenters of T1 transitions lie in the vicinity of cell-cell junctions, the peaks in the $g_\omega(t)$ must be relate to where the cell-cell junctions are located. Over time, the junctions remodel due to T1 transitions, and so $g_\omega(t)$ flattens as ω increases. Figure 3(b) shows $g_\omega(t)$ for varying activity v_0 in the MPF model. As activity increases, the correlations reduce due to higher rates of junction remodeling [11]. Similar results are observed for the AES model, as shown in Fig. 3(c), where $g_\omega(t)$ is plotted for varying activity v_0 .

The peaks in the AES model lie at units of the mean separation distance r^* , determined by the number density of cells, which is about $r_{AES}^* \approx 2$, suggesting that consecutive T1 transitions are more likely to occur across different cell neighborhoods. In contrast, for the MPF model, the characteristic cell size r_{MPF}^* —defined as the diameter of the circle with area equal to the cell area—is approximately $r_{MPF}^* \approx 11$. The first peak in $g_\omega(t)$ occurring at $r \approx 3$ and the first valley at $r \approx 8$, both of which being less than r_{MPF}^* , correspond to different junctions of the same cell. This indicates that consecutive T1 events in the MPF model are more likely to arise from junction remodeling processes within the same cell. The spatial distributions hint that the first peak likely corresponds to T1 transitions that involve vertices that are closer to each other with respect to a cell. Whereas, the second peak is likely due to vertices involved in T1s lying on the opposite sides of a cell. We discuss this further in Sec. VI by analyzing spatial distributions of successive T1 transitions undertaken by a cell. We will show that cell elongation due to T1 transitions results in the location of the peak being slightly greater than r^* .

From here on, we make use of the high shape resolution of cells in the MPF model to discuss the interplay between cell shape and T1 transitions. The cell shape will also help us

better understand the profile of the radial distribution $g_\omega(r)$ of MPF model.

V. CELL SHAPE CHANGES FROM T1 EVENTS

The cell shape index p defined as

$$p = \frac{\text{Perimeter}}{\sqrt{\text{Area}}} \quad (7)$$

is a common cell shape descriptor especially for quantifying cross-over regimes in jamming transitions [31]. For instance, in asthmatic airway epithelium, the jamming transition has been associated with a critical shape index of $p \approx 3.81$, below which the cells are jammed [32].

In our MPF model, the cell area remains invariant, meaning that the shape index is determined solely by the cell perimeter. Since the energy is concentrated at the cell-cell junctions, a cell with a higher perimeter (or shape index) possesses a higher associated energy. Therefore, the shape index also serves as an indicator of the energy associated with the shape of a cell.

In Fig. 4(a) we show the time evolution of the shape index of cells that take part in T1 transitions, averaged over all T1 transitions, as a function of the time before and after a T1 transition. The evolution after a T1 transition is shown such that $t_{\mathcal{T}}^+$ is fixed at zero, with positive time representing the period after $t_{\mathcal{T}}^+$. Similarly, the time evolution before a T1 transition is shown such that $t_{\mathcal{T}}^-$ is fixed at zero, with negative time representing the period before $t_{\mathcal{T}}^-$ [10]. The average shape index of cells in T1 transitions increases before the start of T1, reaches its peak at the start of T1, and falls sharply immediately after the end of T1, followed by a slower relaxation.

However, when plotting the evolution separately for *loser* and *gainer* cells, different patterns become evident. Before a T1 event, the shape index of loser cells increases significantly upon elongation, reaching its peak just before the start of the T1 transition. Meanwhile, the shape index of gainer cells remains constant on average. As the four cells are on the verge of undergoing a T1 event, the gainer cells move towards each other, compressing the loser cells such that the junction shared by the loser cells shrinks [10]. Gainer cells elongate during the T1 transition and reach their maximum shape index after the end of the T1 transition, whereas the shape index of loser cells reduces during the T1 transition. At the end of the T1 transition, loser and gainer cells have similar shape indices. After the T1 transition, the shape index of loser cells decays

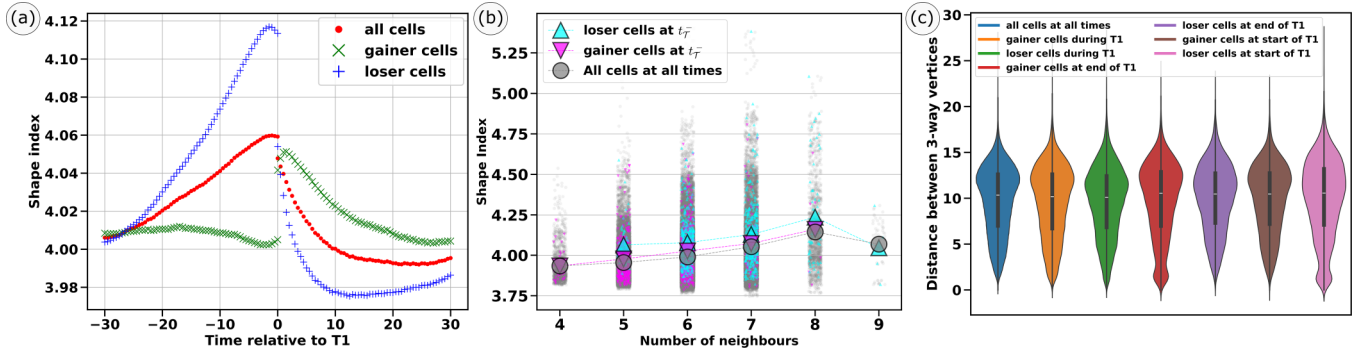


FIG. 4. For the MPF model: (a) Evolution of mean shape index of cells undergoing a T1 transition. Negative time and positive time correspond to relative time before the start of a T1 transition $t_{\bar{T}}$ and relative time after the end of a T1 transition $t_{\bar{T}}^+$. The mean evolution of gainer and loser cells is also highlighted. (b) Cell shape index as a function of number of neighbors. The average shape index for loser and gainer cells at the start of the T1 transition is marked by large symbols, while the small symbols represent the individual shape indices. (c) Violin plots representing the distribution of the distance between all pairs of vertices of a cell in various scenarios. See Movie 3 in the Supplemental Material [29] for a movie of the evolution of the shape of a cell along with its shape index and number of neighbors.

at a faster rate than that of the gainer cells. Thus, the loser and gainer cells experience distinctive shape deformations due to a T1 transition.

To better understand the relationship between cell shape and neighbor topology, we plot the distribution of the shape index p for all cells at all times, grouped by the number of neighbors each cell has [Fig. 4(b)]. The figure also shows the average shape index of *loser* and *gainer* cells at the onset of a T1 transition, compared to the overall means. Cells with fewer neighbors tend to have a lower shape index. During a T1 transition, loser cells typically relax their shape, while gainer cells tend to become more elongated (shape tension). Notably, loser cells still experience larger overall shape changes than gainer cells, as shown by the shape index evolution in Fig. 4(a).

Cells with higher activity v_0 undergo more T1 transitions and have a higher mean shape index [10]. As the mean shape index increases with activity and as cells undergo more T1s, they develop transient elongations, or protrusions.

Figure 4(c) presents violin plots of the distribution of distances between pairs of cell vertices under different scenarios for a T1 transition. Overall the distributions are qualitatively similar, but there are noticeable differences in the detailed profiles, specifically corresponding to pairs of vertices that are about to be annihilated or have recently been created. When two vertices of a cell are close to each other, the shape of the cell is likely very anisotropic (elongated). This explains why the shape index of loser cells peaks before the T1 transition, and the shape index of gainer cells peaks afterward.

VI. EMERGENT DIRECTIONAL MIGRATION

Since cell shape and its tendency to lose or gain neighbors during a T1 transition are interconnected, we now investigate whether the interplay between cell shape and junction remodeling causes coherent flow patterns that span larger length scales than those typical of a T1 transition.

Let $\mathcal{U}_i^-(\mathcal{T})$ and $\mathcal{U}_i^+(\mathcal{T})$ be the set of all vertices that were annihilated and created, respectively, due to the T1 transition $\mathcal{T}$ that involved cell i . For a pair of *successive* T1s $\mathcal{T}_m$ and $\mathcal{T}_n$ involving cell i , i.e., $i \in \mathcal{T}_m$ and $i \in \mathcal{T}_n$, the following

relations hold:

$$\begin{aligned} \mathcal{U}_i^+(\mathcal{T}_m) &= \{\mathcal{X}_{\mathcal{T}_m,c}^+\}: & \text{if } i \text{ is a loser cell in } \mathcal{T}_m, \\ \mathcal{U}_i^+(\mathcal{T}_m) &= \{\mathcal{X}_{\mathcal{T}_m,1}^+, \mathcal{X}_{\mathcal{T}_m,2}^+\}: & \text{if } i \text{ is a gainer cell in } \mathcal{T}_m, \\ \mathcal{U}_i^-(\mathcal{T}_n) &= \{\mathcal{X}_{\mathcal{T}_n,1}^-, \mathcal{X}_{\mathcal{T}_n,2}^-\}: & \text{if } i \text{ is a loser cell in } \mathcal{T}_n, \\ \mathcal{U}_i^-(\mathcal{T}_n) &= \{\mathcal{X}_{\mathcal{T}_n,c}^-\}: & \text{if } i \text{ is a gainer cell in } \mathcal{T}_n, \end{aligned}$$

where $c = 1$ or 2 depending on the vertex which includes cell i .

There are four different scenarios for a cell involved in *successive* T1 transitions, as shown in Figs. 5(a)–5(d), depending on whether it gains or loses a neighbor in the two T1 transitions. These four scenarios are denoted as *loser-loser*, *loser-gainer*, *gainer-loser*, and *gainer-gainer*. The vertices of the highlighted cell involved in $\mathcal{T}_m$ and $\mathcal{T}_n$ are marked in yellow and cyan, respectively. In some situations, the same vertex can be involved in both T1 transitions, such that it is created in $\mathcal{T}_m$ and annihilated in $\mathcal{T}_n$, and is appropriately highlighted using both cyan and yellow. This is possible in all four scenarios of *successive* T1 transitions. A cell undergoes a reversible T1 transition in the scenarios *loser-gainer* and *gainer-loser* only if both the vertices involved in the $\mathcal{T}_m$ are involved in $\mathcal{T}_n$. We do not encounter any reversible T1 transitions in the MPF models in the parameter regime we have explored, but we have encountered several T1 transitions where one vertex was involved in *successive* T1 transitions. The waiting time distributions t_w for each of these four scenarios remain exponential, indicating that successive T1 events occur as a Poisson process regardless of the specific pattern of junction remodeling a cell undergoes. This suggests that the remodeling is primarily shape-dependent, as also reflected in the angular distributions [Figs. 5(f)–5(i)].

For each of the four scenarios of *successive* T1s involving cell i , we first determine the locations of all vertices created after $\mathcal{T}_m$, i.e., $\{\mathbf{r}_{\mathcal{X}}(t_{\mathcal{T}_n}^-) \mid \mathcal{X} \in \mathcal{U}_i^+(\mathcal{T}_m)\}$, and the locations of all vertices that will annihilate in $\mathcal{T}_n$, i.e., $\{\mathbf{r}_{\mathcal{X}}(t_{\mathcal{T}_n}^-) \mid \mathcal{X} \in \mathcal{U}_i^-(\mathcal{T}_n)\}$, both calculated at the start time of $\mathcal{T}_n$ i.e. $t_{\mathcal{T}_n}^-$, which is the latest time where the vertices in both $\mathcal{U}_i^+(\mathcal{T}_m)$ and $\mathcal{U}_i^-(\mathcal{T}_n)$ coexist. Then we determine the location of the center of mass of the cell at $t_{\mathcal{T}_n}^-$, i.e., $\mathbf{r}_i(t_{\mathcal{T}_n}^-)$ [calculated using Eq. (A7)]. We

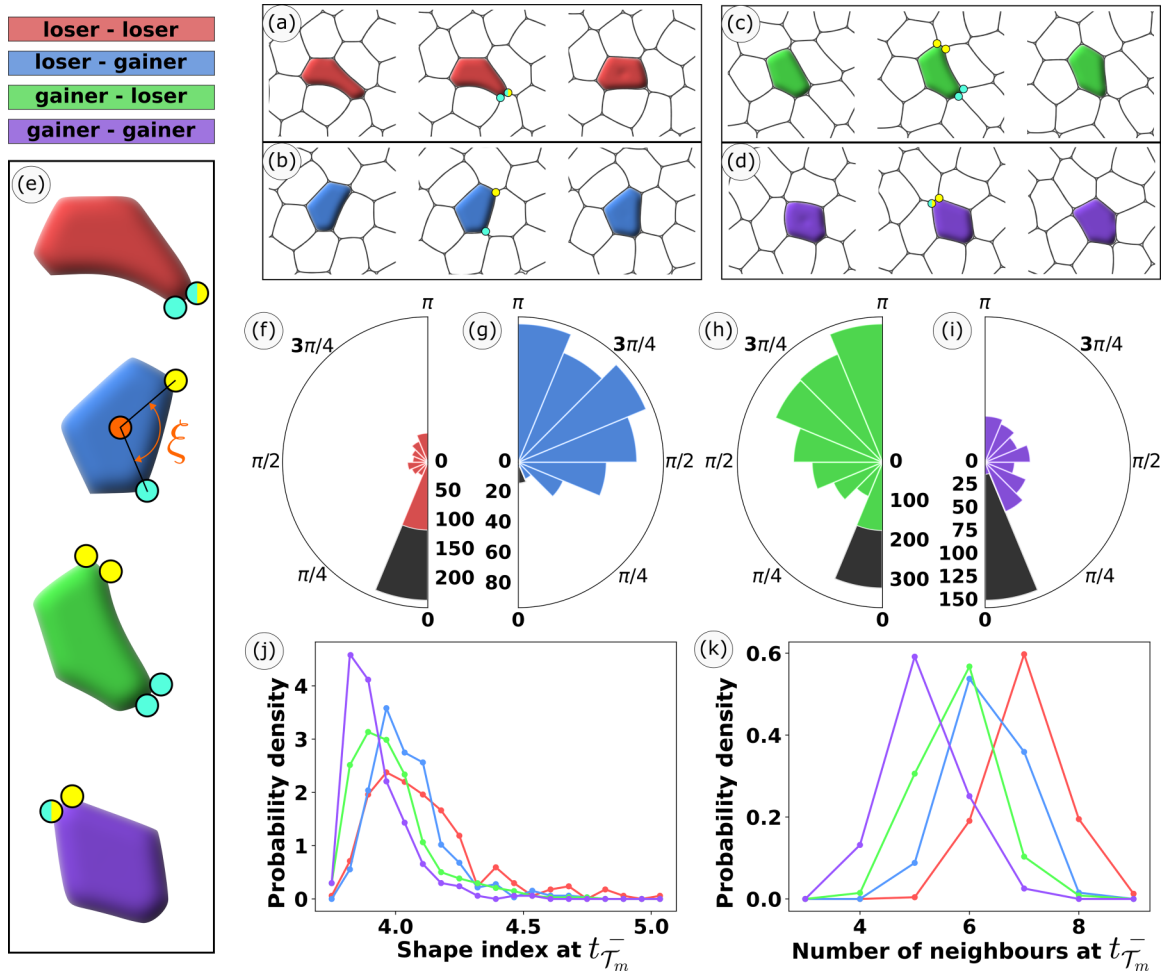


FIG. 5. For the MPF model: (a)–(d) Four different patterns by which highlighted cell i undergoes two *successive* T1 events, i.e., $\mathcal{T}_m$ (first T1) and $\mathcal{T}_n$ (2nd T1). The colors correspond to legend at the top left. $\mathcal{T}_m$ takes place between the left and the middle image, while $\mathcal{T}_n$ takes place between the middle and the right images. The yellow and cyan circles mark the vertices of the highlighted cell that are created in $\mathcal{T}_m$ and annihilated in $\mathcal{T}_n$, respectively. Sometimes vertices take part in both T1s and are marked with both cyan and yellow. (a) *Loser-loser*: Red cell loses neighbors in both T1s. (b) *Loser-gainer*: Blue cell loses neighbors in $\mathcal{T}_m$ and gains neighbors in $\mathcal{T}_n$. (c) *Gainer-loser*: Green cell gains neighbors in $\mathcal{T}_m$ and loses neighbors in $\mathcal{T}_n$. (d) *Gainer-gainer*: Purple cell gains neighbors in both T1s. (e) The zoomed highlighted cells from (a)–(d). (f)–(i) Fixing the origin to the center of mass of cell i , the difference in orientation of each element in the set of vertices created in ($\mathcal{T}_m$) (i.e., $[\mathcal{U}_i^+(\mathcal{T}_m)]$) with each element in the set of vertices annihilated in ($\mathcal{T}_n$) (i.e., $[\mathcal{U}_i^-(\mathcal{T}_n)]$) is histogrammed over all pairs of *successive* T1 transitions for all cells. An example of the difference in orientation is indicated by ξ on the blue cell, as shown in (e). ξ is the difference in orientation of blue and yellow circles measured relative to the orange circle (center of mass). The black color in (f)–(i) represents vertices that are created in $\mathcal{T}_m$ and annihilated in $\mathcal{T}_n$. (j) Shape index and (k) number of neighbors of the cell undergoing *successive* T1s measured at the start of $\mathcal{T}_m$, with the color marking the appropriate scenario. The statistics in (f)–(k) are collected over three simulations.

pair every vertex of $\mathcal{U}_i^+(\mathcal{T}_m)$ with every vertex of $\mathcal{U}_i^-(\mathcal{T}_n)$. By pairing vertices in this way, in the scenarios *loser-loser*, *loser-gainer*, *gainer-loser*, and *gainer-gainer*, we get two, one, four, and two pairs of vertices for each pair of *successive* T1s, respectively.

We fix an origin at the center of mass of the cell, denoted by $\mathbf{r}_i(t_{\mathcal{T}_n}^-)$. For each pair of chosen vertices, we find the difference in orientations of both vertices measured relative to the cell center, which we will call *relative orientation*. An example of such a relative orientation is highlighted by the angle ξ on the blue cell in Fig. 5(e). This process is repeated for all pairs of *successive* T1s and for all cells, and the corresponding histograms are shown in Figs. 5(f)–5(i) for all four scenarios. The relative orientation between each vertex pair varies from

0 to π . If the same vertex belongs to both $\mathcal{U}_i^+(\mathcal{T}_m)$ and $\mathcal{U}_i^-(\mathcal{T}_n)$, then the data are highlighted in black, and the relative orientation between such a pair is 0. If both the vertices are on the opposite side of the cells then the relative orientation between such a pair is π . In essence, the distributions in Figs. 5(f)–5(i) inform us of the spatial distribution of *successive* T1s with respect to the cell center. Figures 5(j) and 5(k) show the corresponding probability density distributions of the shape index and the number of neighbors of the cell in each of the four scenarios measured at the start of $\mathcal{T}_m$.

For the scenario where a cell loses neighbors in both T1 transitions, i.e., *loser-loser*, we see that the distribution peaks at a low value of relative orientation. This suggests that the pairs of vertices that took part in *successive* T1s are close

to each other. A large fraction of these pairs involve one vertex taking part in both T1s. Very few pairs of vertices lie on the opposite sides of the cells, suggesting that most pairs of *successive* T1s happen close to each other on the same side of the cell. Hence, elongated cells are more likely to undergo *successive* T1s of *loser-loser* kind, as shown in Fig. 5(j). An elongated cell can relax its shape by retracting its mass towards the cell center along the direction of elongation through T1 transitions [see red cell in Fig. 5(a)]. Cells in this scenario also have a higher than average number of neighbors as shown in Fig. 5(k).

For the scenario *loser-gainer*, both T1s happen mostly on the opposite sides of a cell as the relative orientation between most pairs is greater than $\pi/2$, as shown in Fig. 5(g). This is akin to a cell retracting mass from one side and moving in the opposite direction. The loser cells, which undergo more drastic shape changes during a T1, as shown in Fig. 4(a), are propelled away from the epicenter [10], and such a cell gains a neighbor on the opposite side.

In the scenario *gainer-loser*, we see two peaks near 0 and π ; see Fig. 5(g). The differences in the distributions for *loser-gainer* and *gainer-loser* suggest that the spatial distribution of *successive* T1s on a cell depends on the order of topological change, even if the net topological change in both scenarios is the same. This might be because gainer and loser cells undergo distinct deformations due to T1 transitions, as shown in Fig. 4(a). In both *loser-gainer* and *gainer-loser* situations, at the start of the first T1, the average number of neighbors is around six, suggesting that these two scenarios involve fluctuations about the mean number of neighbors, which is approximately six.

For the scenario *gainer-gainer*, most pairs of vertices of *successive* T1s lie on the same side of the cell, and most of these also involve the same vertex in both T1s, as shown in Fig. 5(i). If we were to exclude the situations where the same vertex is involved, the remaining pairs are likely to occur at any relative orientation except for very low relative orientation, as indicated by the histogram. Cells that gain two neighbors in *successive* T1s usually have a lower shape index at the start of the first T1 than the other three scenarios, as shown in Fig. 5(j), as well as a lower number of neighbors, as shown in Fig. 5(k).

If the distributions of relative orientation of the vertices involved in *successive* T1 transitions were uniform, then T1 transitions would have no preferred orientation for the quadrupolar flow field that arises due to them [11]. However, the nonuniform distributions in Figs. 5(f)–5(i) suggest an effective directed migration of the cells, depending on the four scenarios, which could lead to emergent flow patterns at larger scales as a result of chaining of T1 transitions.

The anisotropic spatial distribution of paired vertices of *successive* T1 transitions on the cell might be a reason behind the weak polar order within the MPF model, which is typically more than 0.4 [11]. The large number of paired vertices of *successive* T1 transitions occurring at low relative orientation suggest that the *successive* T1 transitions occur close to each other, informing the profile of the radial distribution functions in Fig. 3 for the MPF model, where a peak was observed for very low value of radial distance ($r < 5$).

In the MPF model, the orientation of cell activity tends to align with the direction of its elongation, and this rate is controlled by a parameter α (see Appendix A for details). However, this alignment is not the reason for the specific spatial distribution of *successive* T1 transitions, as similar distributions are observed even when the activity does not align with cell elongation, i.e., when $\alpha = 0$ (see Fig. 2 in the Supplemental Material [29] for corresponding plots). Also, these distributions remain similar even if activity and deformability are varied, suggesting a universal emergent behavior due to the shape response of cells.

Additionally, we note that every cell that gains neighbors in two *successive* T1s where the vertex created in the first T1 is annihilated in the second T1 also has a neighboring cell that loses neighbors in both of these T1s. Consider a T1 $\mathcal{T}_m = \{A, B, C, D\}$ where cell A gains a neighbor B . This process creates two vertices: $\{A, B, C\}$ and $\{A, B, D\}$. If one of these vertices is involved in another T1 $\mathcal{T}_n$ by annihilating such that cell A gains another neighbor, then depending on which vertex took part in the T1, either C or D loses contact with B . So one of C or D loses neighbors in both $\mathcal{T}_m$ and $\mathcal{T}_n$.

VII. CONCLUSIONS

By combining the strengths of two complementary modeling approaches—the MPF model, which captures complex cell shape dynamics and junctional remodeling, and the AES model, which captures neighbor exchanges as topological transitions—we uncover a robust connection between cell shape changes and the spatiotemporal distribution of T1 transitions across cell junctions. While individual T1 transitions occur as temporally uncorrelated, Poisson-distributed events with a mean waiting time governed by cell activity, their spatial distribution across cell junctions is highly anisotropic and capable of driving directional cell migration.

Our statistical analysis reveals a dual role for T1 transitions: cells losing neighbors tend to relax and reduce their shape index, whereas cells gaining neighbors become elongated, increasing shape tension. These deformations are not merely passive consequences of topological change; rather, they feed back into the orientation and likelihood of subsequent T1 transitions, enabling localized, activity-driven fluidization within the tissue.

We further identify four characteristic patterns of *successive* T1 transitions across a cell's junctions. These patterns demonstrate how feedback between cell shape and neighbor topology can bias cell movement directionally, generating relative motion between cells. Although our focus is on local dynamics, such directional biases could, in aggregate, drive coherent flows at a tissue scale in confluent monolayers.

These findings have implications for better understanding epithelial tissue dynamics in biological contexts where collective migration occurs in the absence of strong external cues—for example, during embryonic elongation, wound closure, and morphogenesis. Our results suggest that internal cell mechanics and local topological rearrangements are sufficient to produce emergent directionality and fluidlike behavior in epithelial tissues. The anisotropic spatial correlations of T1 transitions—and the emergence of chaining or cascading events—warrant further exploration in other

modeling frameworks, such as vertex models [33], particularly those that incorporate active stress and complex shape dynamics [15].

In conclusion, we propose a generic, intrinsic mechanism by which local junctional remodeling—through T1 transitions coupled with cell shape feedback—gives rise to emergent, polarized cell migration and localized tissue fluidization in confluent epithelial monolayers. While our model predictions are consistent with experimental observations of feedback between cell shape dynamics and neighbor exchanges, e.g., Ref. [6], a direct quantitative comparison for epithelial tissues warrants a dedicated study of its own. This is due to the inherent complexity of experimental systems and additional factors that may need consideration, such as cell division, junctional Myosin II feedback, and cell area variability. Nonetheless, our numerical results provide a useful baseline for understanding the temporal and spatial patterning of T1 events as topological rearrangements intrinsically coupled to cell shape dynamics via junction remodeling. We hope these findings will inform future experimental investigations into the role of cell shape-driven junction remodeling and directed migration in confluent epithelial tissues.

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DATA AVAILABILITY

The data supporting this study's findings are available within the article.

APPENDIX A: MULTIPHASE FIELD MODEL

The multiphase field model presented here was previously used in Ref. [11] and is based on earlier models in Refs. [10,19]. Consider a set of cells represented by phasefields $\{\phi_i\}$ defined in $\Omega \subset \mathbb{R}^2$, where $i = 1, 2, \dots, N$, and N is the total number of cells. The domain Ω is a square of side length L . The interior and exterior of the cell i is identified as the region in Ω with bulk phase values $\phi_i \approx 1$ and $\phi_i \approx -1$, respectively. The interior is phase separated from the exterior through a diffuse interface of width $O(\epsilon)$. The phase fields ϕ_i evolve according to

$$\partial_t \phi_i + \mathbf{v}_i \cdot \nabla \phi_i = \Delta \frac{\delta \mathcal{F}}{\delta \phi_i}, \quad (\text{A1})$$

while preserving the total cell area. The free energy functional $\mathcal{F}[\{\phi_i\}] = \mathcal{F}_{CH}[\{\phi_i\}] + \mathcal{F}_{INT}[\{\phi_i\}]$ is a sum of energy of the surface and the interaction energy between the cells. The energy of the surface is the Cahn-Hilliard energy given as

$$\mathcal{F}_{CH}[\{\phi_i\}] = \frac{1}{\text{Ca}} \sum_{i=1}^N \int_{\Omega} g(\phi_i)$$

$$\times \left(\frac{\epsilon}{2} \|\nabla \phi_i\|^2 + \frac{1}{4\epsilon} (\phi_i^2 - 1)^2 \right) dx, \quad (\text{A2})$$

where Ca is the capillarity number that controls the deformability of the cells, ϵ controls the thickness of cell interface, and $g(\phi) = \frac{2}{3(\phi+1)(\phi-1)}$ is the diffusion restriction function to prevent bulk diffusion [34]. The first term penalises gradients in the phase field which models cortical tension, while the second term is the demixing part which ensures phase separation between the exterior and interior of the cell. The interaction energy is given as

$$\mathcal{F}_{INT}[\{\phi_i\}] = \frac{1}{\text{In}} \sum_{i=1}^N \int_{\Omega} \sum_{j \neq i} \left(\frac{a_r}{2} (\phi_i + 1)^2 (\phi_j + 1)^2 - \frac{a_a}{2} (\phi_i^2 - 1)^2 (\phi_j^2 - 1)^2 \right) dx, \quad (\text{A3})$$

where, a_r and a_a are used to tune the repulsive and attraction component, respectively. The repulsive part penalises overlap of cell interiors, while the attractive part promotes overlap of cell interfaces.

The cell activity field is given as

$$\mathbf{v}_i(\mathbf{x}, t) = v_0 \mathbf{e}_i(t) \hat{\phi}_i(\mathbf{x}, t), \quad (\text{A4})$$

where v_0 is the magnitude of activity, $\hat{\phi} = 0.5\phi + 0.5$, and $\mathbf{e}_i = [\cos \theta_i(t), \sin \theta_i(t)]$ is the activity orientation vector. The orientation angle θ evolves according to the stochastic differential equation

$$d\theta_i = \sqrt{2D_r} dW_i(t) + \alpha(\beta_i(t) - \theta_i(t))dt, \quad (\text{A5})$$

where D_r is the coefficient of rotational diffusion and W_i is a Wiener process. The angle $\beta_i(t)$ is the angle of shape polarity defined to be the elongation axis of the cell, and is calculated from the eigenvector corresponding to the largest eigenvalue of the shape deformation tensor, η_i^+ , such that

$$\beta_i(t) = \begin{cases} \arg(\eta_i^+(t)): & \mathbf{e}_i(t) \cdot \eta_i^+(t) > 0 \\ \arg(-\eta_i^+(t)): & \mathbf{e}_i(t) \cdot \eta_i^+(t) < 0. \end{cases} \quad (\text{A6})$$

The system of partial differential equations in Eq. (A1) is considered on a square periodic domain. The system is spatially discretized using finite elements with a semi-implicit discretization in time, and is solved using adaptive finite element methods using the C++ FEM tool AMDIS [35,36] with the parallization approach introduced in Ref. [37]. For each simulation, we initialize the system with N square cells arranged in a regular grid with randomly oriented activity, allowing for an initial transient period for cells to mix. We consider the data only after the cell shapes appear sufficiently irregular.

The parameters used are as per Table I unless specified otherwise.

The center of mass of a cell i in the MPF model is calculated as

$$\mathbf{r}_i(t) = \frac{\int_{\Omega} \mathbf{x} \hat{\phi}_i dx}{\int_{\Omega} \hat{\phi}_i dx}, \quad (\text{A7})$$

where $\hat{\phi}_i = 0.5\phi_i + 0.5$ is the rescaled phasefield.

APPENDIX B: ACTIVE ELASTIC SOLID MODEL

The active elastic solid model [25] is a discrete particle model consisting of a collection of N particles at positions $\mathbf{r}_i$ in a flat tissue monolayer. The elasticity of the tissue monolayer is modeled by connecting neighboring cells with springlike forces $\mathbf{f}_i = -\nabla_{\mathbf{r}_i} V$ determined by a generic Gaussian potential for the elastic interactions,

$$V = \sum_i \sum_{j=n.o.f.i} \exp \left[-\frac{(|\mathbf{r}_{ij}| - l_{ij})^2}{a_0^2} \right], \quad (\text{B1})$$

where $\mathbf{r}_{ij} = \mathbf{r}_i - \mathbf{r}_j$ is the separation vector between two cells and l_{ij} is the equilibrium distance between cells. The sum of j is taken over the neighbors on an initially square lattice. The directional self-propulsion follows the cell polarities $\mathbf{p}_i$, which reorients due to a torque induced by elastic interactions. The overdamped dynamics on this system is then described by

$$\dot{\mathbf{r}}_i = v_0 \mathbf{p}_i + \frac{1}{\zeta} (\mathbf{f}_i + \xi_i), \quad (\text{B2})$$

$$\dot{\mathbf{p}}_i = \mathbf{T}_i \times \mathbf{p}_i + \eta_i, \quad (\text{B3})$$

where the polarity torque is

$$\mathbf{T}_i = \frac{1}{\zeta} (\mathbf{p}_i \times \dot{\mathbf{r}}_i).$$

The translational noise has zero mean and

$$\langle \xi_i(t) \xi_j(t') \rangle = D_r \delta_{ij} \delta(t - t'). \quad (\text{B4})$$

Similarly, the rotational noise also has zero mean and

$$\langle \eta_i(t) \eta_j(t') \rangle = D_p \delta_{ij} \delta(t - t'). \quad (\text{B5})$$

We renormalize the magnitude of polarity to unity at every timestep because the noise is not norm conserving. AES simulations are for 576 cells on a periodic domain of length L with time advancement using the Euler method and with the parameters given in Table II unless otherwise given for a figure.

Local cell rearrangements are imposed by a geometric rule for nearest neighbors. Namely, cells i and j are considered neighbors if and only if they are closest in distance to their midpoint than any other third cell. Explicitly, we connect all cells i and j and remove the connection if there exists any third cell k which satisfies $r_{ij}^2 > r_{ik}^2 + r_{jk}^2$, with r_{ij} being the distance between cells i and j . This is the same neighbor criterion as in Ref. [28]. The neighbor criterion is slightly different than using a Voronoi tessellation. While we do gain information about the duration of T1 transitions, since they no longer occur instantaneously, we do not gain any information about the shape of cells. Because of this, we use the multi-phase field model to answer these questions.

In the simulations used in this paper, since we are concerned with the behavior of T1s specifically, we update the neighbor list at every time step. We then define a T1 as occurring in the following way: when cells i and j cease to be neighbors this is the start $t_{\mathcal{T}}^-$ of a potential T1; a T1 occurs when cells m and n become neighbors at $t_{\mathcal{T}}^+$, where both m and n are neighbors of both i and j ; it is canceled if i and j become neighbors again. According to these rules rosettes are counted as one T1 transition. The neighbor criterion induces changes in the neighbor lists with time. Thus, the T1 transitions are also of finite duration in the AES. For the values in Table II, the mean waiting time is about 25.1 and much larger than the typical T1 duration, which is about 4.8.

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- [1] B. Ladoux and R. M. Mège, Mechanobiology of collective cell behaviours, *Nat. Rev. Mol. Cell Biol.* **18**, 743 (2017).
 - [2] V. Hakim and P. Silberzan, Collective cell migration: A physics perspective, *Rep. Prog. Phys.* **80**, 076601 (2017).
 - [3] A. Das, S. Sastry, and D. Bi, Controlled neighbor exchanges drive glassy behavior, intermittency, and cell streaming in epithelial tissues, *Phys. Rev. X* **11**, 041037 (2021).
 - [4] P. Friedl and D. Gilmour, Collective cell migration in morphogenesis, regeneration and cancer, *Nat. Rev. Mol. Cell Biol.* **10**, 445 (2009).
 - [5] A. Brugués, E. Anon, V. Conte, J. H. Veldhuis, M. Gupta, J. Colombelli, J. J. Muñoz, G. W. Brodland, B. Ladoux, and X. Trepas, Forces driving epithelial wound healing, *Nat. Phys.* **10**, 683 (2014).
 - [6] S. Curran, C. Strandkvist, J. Bathmann, M. De Gennes, A. Kabla, G. Salbreux, and B. Baum, Myosin II controls junction fluctuations to guide epithelial tissue ordering, *Dev. Cell* **43**, 480 (2017).
 - [7] K. D. Irvine and E. Wieschaus, Cell intercalation during *Drosophila* germband extension and its regulation by pair-rule segmentation genes, *Development* **120**, 827 (1994).
 - [8] D. Weaire and N. Rivier, Soap, cells and statistics—Random patterns in two dimensions, *Contemp. Phys.* **25**, 59 (1984).
 - [9] J. Stavans, The evolution of cellular structures, *Rep. Prog. Phys.* **56**, 733 (1993).
 - [10] H. P. Jain, A. Voigt, and L. Angheluta, Robust statistical properties of T1 transitions in a multi-phase field model of cell monolayers, *Sci. Rep.* **13**, 10096 (2023).
 - [11] H. P. Jain, A. Voigt, and L. Angheluta, From cell intercalation to flow, the importance of T1 transitions, *Phys. Rev. Res.* **6**, 033176 (2024).
 - [12] C. Bertet, L. Sulak, and T. Lecuit, Myosin-dependent junction remodelling controls planar cell intercalation and axis elongation, *Nature (London)* **429**, 667 (2004).
 - [13] J. T. Blankenship, S. T. Backovic, J. S. Sanny, O. Weitz, and J. A. Zallen, Multicellular rosette formation links planar cell polarity to tissue morphogenesis, *Dev. Cell* **11**, 459 (2006).
 - [14] A. Mongera, P. Rowghanian, H. J. Gustafson, E. Shelton, D. A. Kealhofer, E. K. Carn, F. Serwane, A. A. Lucio, J. Giammona, and O. Campàs, A fluid-to-solid jamming transition underlies vertebrate body axis elongation, *Nature (London)* **561**, 401 (2018).
 - [15] S. Kim, M. Pochitaloff, G. A. Stooke-Vaughan, and O. Campàs, Embryonic tissues as active foams, *Nat. Phys.* **17**, 859 (2021).
 - [16] A. Mongera, M. Pochitaloff, H. J. Gustafson, G. A. Stooke-Vaughan, P. Rowghanian, S. Kim, and O. Campàs, Mechanics

- of the cellular microenvironment as probed by cells *in vivo* during zebrafish presomitic mesoderm differentiation, *Nat. Mater.* **22**, 135 (2023).
- [17] D. Bi, J. H. Lopez, J. M. Schwarz, and M. L. Manning, Energy barriers and cell migration in densely packed tissues, *Soft Matter* **10**, 1885 (2014).
- [18] M. E. Rosa and M. A. Fortes, Nucleation and glide of dislocations in a monodisperse two-dimensional foam under uniaxial deformation, *Philos. Mag. A* **77**, 1423 (1998).
- [19] D. Wenzel, S. Praetorius, and A. Voigt, Topological and geometrical quantities in active cellular structures, *J. Chem. Phys.* **150**, 164108 (2019).
- [20] R. Mueller, J. M. Yeomans, and A. Doostmohammadi, Emergence of active nematic behavior in monolayers of isotropic cells, *Phys. Rev. Lett.* **122**, 048004 (2019).
- [21] T. B. Saw, A. Doostmohammadi, V. Nier, L. Kocgozlu, S. Thampi, Y. Toyama, P. Marcq, C. T. Lim, J. M. Yeomans, and B. Ladoux, Topological defects in epithelia govern cell death and extrusion, *Nature (London)* **544**, 212 (2017).
- [22] J.-M. Armengol-Collado, L. N. Carenza, J. Eckert, D. Krommydas, and L. Giomi, Epithelia are multiscale active liquid crystals, *Nat. Phys.* **19**, 1773 (2023).
- [23] M. Nonomura, Study on multicellular systems using a phase field model, *PLoS ONE* **7**, e33501 (2012).
- [24] H. P. Jain, D. Wenzel, and A. Voigt, Impact of contact inhibition on collective cell migration and proliferation, *Phys. Rev. E* **105**, 034402 (2022).
- [25] E. Ferrante, A. E. Turgut, M. Dorigo, and C. Huepe, Elasticity-based mechanism for the collective motion of self-propelled particles with springlike interactions: A model system for natural and artificial swarms, *Phys. Rev. Lett.* **111**, 268302 (2013).
- [26] L. Angheluta, A. Lång, E. Lång, and S. O. Bøe, Topological defects in polar active matter, [arXiv:2504.03284](https://arxiv.org/abs/2504.03284).
- [27] E. Lång, A. Lång, P. Blicher, T. Rognes, P. G. Dommersnes, and S. O. Bøe, Topology-guided polar ordering of collective cell migration, *Sci. Adv.* **10**, eadk4825 (2024).
- [28] R. D. Ho, S. O. Bøe, D. K. Dysthe, and L. Angheluta, Role of tissue fluidization and topological defects in epithelial tubulogenesis, *Phys. Rev. Res.* **6**, 023315 (2024).
- [29] See Supplemental Material at <http://link.aps.org/supplemental/10.1103/d6qj-775d> for details.
- [30] S. B. Nissen, S. Rønhild, A. Trusina, and K. Sneppen, Theoretical tool bridging cell polarities with development of robust morphologies, *eLife* **7**, e38407 (2018).
- [31] D. Bi, X. Yang, M. C. Marchetti, and M. L. Manning, Motility-driven glass and jamming transitions in biological tissues, *Phys. Rev. X* **6**, 021011 (2016).
- [32] J. A. Park, J. H. Kim, D. Bi, J. A. Mitchel, N. T. Qazvini, K. Tantisira, C. Y. Park, M. McGill, S. H. Kim, B. Gweon *et al.*, Unjamming and cell shape in the asthmatic airway epithelium, *Nat. Mater.* **14**, 1040 (2015).
- [33] S. Alt, P. Ganguly, and G. Salbreux, Vertex models: From cell mechanics to tissue morphogenesis, *Philos. Trans. R. Soc. B* **372**, 20150520 (2017).
- [34] M. Salvalaglio, M. Selch, A. Voigt, and S. M. Wise, Doubly degenerate diffuse interface models of anisotropic surface diffusion, *Math. Methods Appl. Sci.* **44**, 5406 (2020).
- [35] S. Vey and A. Voigt, AMDiS: Adaptive multidimensional simulations, *Comput. Visual Sci.* **10**, 57 (2007).
- [36] T. Witkowski, S. Ling, S. Praetorius, and A. Voigt, Software concepts and numerical algorithms for a scalable adaptive parallel finite element method, *Adv. Comput. Math.* **41**, 1145 (2015).
- [37] S. Praetorius and A. Voigt, *Collective Cell Behaviour—A Cell-Based Parallelisation Approach for a Phase Field Active Polar Gel Model*, NIC Series No. 49 (Forschungszentrum Jülich GmbH, Zentralbibliothek, Jülich, 2018).