Elasticity, stability, and quasi-oscillations of cell-cell junctions in solid confluent epithelia

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Macroscopic properties and shapes of biological tissues depend on remodelling of cell-cell junctions at the microscopic scale. We propose a theoretical framework that couples a vertex model of solid confluent tissues with the dynamics describing generation of local force dipoles in the junctional actomyosin. Depending on the myosin-turnover rate, junctions preserve stable length or collapse to initiate cell rearrangements. We find that while the elasticity of solid tissues does not meet the conditions for a stable limit cycle of junctional movements, junctional noise can amplify and sustain transient oscillations to the fixed point, yielding quasi-periodic junctional dynamics. We also discover that junctional stability is affected by cell arrangements and junctional rest tensions, which may explain junctional collapse during convergence and extension in embryos.

INTRODUCTION

In confluent tissues, the adjacent cells adhere to one another through narrow joints known as the adherens junctions. These regions are rich with protein complexes, which govern cell-cell adhesion and couple cells’ cytoskeletons [1, 2]. Forces transmitted along the junctions play a major role in morphogenesis. In particular, contractile tensions generated in the actomyosin [3–5] drive various types of cell deformations and tissue-scale movements [6–8]. For instance, medial actomyosin collapse during cell ingression and oscillates during dorsal closure in Drosophila [9–13], directed junctional collapse drives convergence and extension in the early Drosophila embryo [14–16], whereas junctional fluctuations establish the arrangement of cells in Drosophila pupal notum [17] and fluidize the tissue during vertebrate body axis elongation [18].

Recent measurements of junctional dynamics during tissue remodelling revealed that the rate of junctional collapse often increases with contraction [16, 17]. This suggests a positive feedback loop between junctional contraction and generation of junctional tension. Apart from causing collapse of the actomyosin, such a feedback loop could establish oscillatory dynamics as previously proposed within a generic theoretical framework of active contractile elements [19].

To explore these dynamics in the context of confluent tissues, we develop a vertex model, which assumes a feedback loop between cell-scale junctional contractions and generation of force dipoles at the level of the junctional actomyosin. We find that the nonlinear elastic response of solid tissues to local force dipoles does not satisfy conditions for a limit cycle of junctional movements. Nevertheless, the variety of dynamical regimes remains rich and includes junctions that can either sustain stable length, undergo damped transient oscillations towards the system’s fixed point, or collapse. One of our main findings shows that junctional noise establishes a quasi-periodic junctional dynamics corresponding to permanently sustained transient oscillations. We also discover that junctional stability depends on cell arrangements as well as on non-homogeneities in the distribution of junctional rest tensions, which could be relevant for the active junctional remodelling during tissue morphogenesis [14, 15].

MATERIALS AND METHODS

The model

The tissue is represented by a planar polygonal network of cell-cell junctions parametrized by the positions of cell vertices \( \mathbf{r}_i = (x_i, y_i) \) [20–22]. Forces on vertices \( \mathbf{F}_i \) are assumed conservative such that \( \mathbf{F}_i = -\nabla_i W \), where \( W = \kappa \sum_k (A_k - A_0)^2 + \sum_{ij} \gamma_{ij} l_{ij} \) is the total potential energy of the system and \( \nabla_i = (\partial/\partial x_i, \partial/\partial y_i) \). The first sum in the energy goes over all cells and describes cell-area elasticity (\( A_k \) and \( A_0 \) being the actual and the preferred cell areas, respectively), whereas the second sum goes over all junctions and describes the line energy due to adhesion and cortical tension (\( l_{ij} \) being the junctional length); indices \( i \) and \( j \) denote head and tail vertices of the junctions. Due to strong friction with the environment, the motion of vertices is overdamped:

\[
\eta \ddot{\mathbf{r}}_i = \mathbf{F}_i = -2\kappa \sum_k (A_k - A_0) \nabla_i A_k - \sum_{ij} \gamma_{ij} \nabla_i l_{ij} , \quad (1)
\]

where \( \eta \) is the friction coefficient. The tension \( \gamma_{ij}(t) = \gamma_0 + \Delta \gamma_{ij}(t) \), where \( \gamma_0 \) and \( \Delta \gamma_{ij}(t) \) are the average (rest) tension and the time-depending part, respectively. From now on we use dimensionless quantities by choosing \( \sqrt{A_0} \), \( \tau_0 = (\eta \gamma_0)^{-1} \), and \( \gamma_0 \) as the units of length, time, and tension, respectively, and rescaling the modulus as \( \kappa A_0^{3/2} / \gamma_0 \rightarrow \kappa \).

In the case of constant line tensions (\( \gamma_{ij} = \text{const.} \)), our model describes an elastic tissue, which can sustain shear stresses [23, 24]. To examine its response to a locally applied force dipole, we assume an excess line tension \( \Delta \gamma \) on a single junction. This deforms the junction away from
its rest length $l_0$, and the elastic deformation propagates into the bulk (Supplemental Material, Fig. S1). Force balance on the junction implies $0 = -2 \Delta \gamma - f(l)$, where $f(l)$ is the elastic restoring force. In our model, $f(l)$ is well described by $f(l) = \sum_{m=1}^{3} \alpha_m (l - l_0)^m$, where $\alpha_2/\alpha_1 = 0.33$ and $\alpha_3/\alpha_1 = -0.10$ for a regular honeycomb cell tiling (Fig. 1A); similar values for the coefficients are found also in disordered tilings (Supplemental Material, Fig. S1). The significant contribution of the second-order term yields softening and stiffening behaviors when the junction is compressed and expanded, respectively (Fig. 1A). Furthermore, due to friction, $f(l)$ displays hysteresis at non-zero rates of change of junctional tension ($\Delta \dot{\gamma} \neq 0$ and thus $\dot{l} \neq 0$) (Fig. 1A).

In tissues, tensions change on time scales associated with the dynamics of the underlying actomyosin (Fig. 1B). We assume a linear relation between junctional tension $\gamma_{ij}(t)$ and myosin concentration [defined by a number of molecular motors per junction length $c_{ij}(t) = N_{ij}/l_{ij}$: $c_{ij}(t) = \alpha \gamma_{ij}(t)$, where $\alpha$ is a constant proportionality factor. The total rate of change of the myosin concentration reads $\dot{c}_{ij} = N_{ij}/l_{ij} - N_{ij} \dot{l}_{ij}/l_{ij}^2$, where the first term describes changes of the number of motors at a fixed junction length, whereas the second term describes changes of the junction length at a fixed number of motors. In particular, the motor-actin binding and unbinding contribute a time scale $\tau_m$ and are described by $\dot{N}_{ij} = -N_{ij} + c_0 l_{ij}/\tau_m$, where $c_0 = \alpha \gamma_0$ is the ambient myosin concentration. In turn, $l_{ij}$ can be explicitly written in terms of the forces acting at the vertices as $\dot{l}_{ij} = r_{ij} \cdot F_{ij}/l_{ij}$, where $r_{ij} = r_j - r_i$ and $F_{ij} = F_j - F_i$. Overall, this yields a deterministic dynamic equation for tension fluctuations:

$$\Delta \dot{\gamma}_{ij} = -\frac{1}{\tau_m} \Delta \gamma_{ij} - \gamma_{ij} r_{ij} \cdot F_{ij}/l_{ij}^2. \quad (2)$$

Here the first term describes tension relaxation due to myosin turnover, whereas the second term describes the feedback loop between tension dynamics and tissue-scale mechanics given by the vertex model (Fig. 1C).

**RESULTS**

**Local model**

First, we apply this model [Eqs. (1) and (2)] on a quartet of hexagonal cells (inset to Fig. 1D). We derive the equation of state, $f(l)$, for the central junction and find it describing a similar elasticity than that of the full tissue (Sec. I of Supplemental Material). Next, we simulate the dynamics, starting in a configuration away from the fixed point ($l, \gamma_0 = (l_0, 0)$), where $l_0 = 3^{-3/4} \sqrt{2} \approx 0.62$ is the side length of a regular hexagon with unit area. We find three types of behavior: The junction either (i) converges directly back to the fixed point (state S), (ii) undergoes damped transient oscillations before reaching the fixed point (state TO), or (iii) collapses its length to zero (state C) (Fig. 1D). A linear stability analysis (Sec. II of Supplemental Material) reveals the analytical condition for the Hopf bifurcation:

$$\kappa^* = \frac{1}{3l_0^3} - \frac{1}{3l_0^2 \tau_m}, \quad (3)$$

meaning that the junction is stable for $\kappa > \kappa^*$ and unstable for $\kappa < \kappa^*$ (Fig. 1E). Transient oscillations appear within the stable regime at $\kappa < \kappa_* < \kappa^*$, where $\kappa_+ = 1/(3l_0^3) + 1/(3l_0^2 \tau_m) \pm (1/3l_0^2) \sqrt{10/10} l_0 \tau_m$ (Fig. 1E). Importantly, when inspecting the first Lyapunov coefficient, we find that the Hopf bifurcation is always subcritical, meaning that no parameter values yield a stable limit cycle (Sec. III of Supplemental Material).

Next, to search for the various types of junctional behaviors in the full-tissue setting, we apply our model to the honeycomb cell tiling and numerically preform the linear stability analysis (Sec. IV of Supplemental Material). We identify the same dynamical regimes as in the local model (Fig. 2A). Interestingly, since the collective cell deformations allow preserving cell areas, the Hopf bifurcation in the full model barely depends on the cell-compressibility modulus $\kappa$ (Fig. 2A).
and become sustained indefinitely in the presence of the noise. This gives rise to a quasi-periodic trajectory or the so-called quasi-cycle [27–29]. We refer to these junctional movements as quasi-oscillations.

Next, we examine the observed dynamics in the Fourier space. The quasi-oscillations are seen as clear peaks in power spectral densities (PSD) of tension fluctuations and length fluctuations, PSD$_\gamma$ and PSD$_h$, respectively. These peaks get dominated by the noise when moving away from the bifurcation point (Fig. 2E and Sec. V of Supplemental Material). In fact, in the limit $1/\tau_m \to \infty$ where the relative contribution of the coupling term in the tension dynamics [Eq. (2)] becomes negligible compared to the relaxation term, the PSDs agree with the model in which tensions obey a pure OU process (blue curves in Fig. 2E). Overall, these results highlight the importance of the coupling between junctional contractions and the dynamics of tension in the actomyosin.

In confluent tissues, junctions are interconnected and so need to synchronize their quasi-oscillations. Since three junctions meet at each vertex, junction networks are geometrically frustrated, which can lead to nontrivial spatial patterns of cell deformations (Sec. VI of Supplemental Material).

**Inhomogeneous tissues**

The stability condition of our local model [Eq. (3)] suggests that disorder of cell packing may affect junctional stability. Indeed, it can be recast as $l_0 > l_0^*$, where the $l_0^*$ is the critical rest length for collapse. Since the rest lengths are distributed in disordered tissues, a critical rest length should exist in these tissues, such that junctions shorter than that length would collapse and trigger cell rearrangements. To test this possibility, we examine an ensemble of 100 disordered tissues (inset to Fig. 3A). Starting close to the fixed point (i.e., all junctions at their rest lengths and all tensions equal 1), we simulate the dynamics [Eqs. (1) and (2)] at fixed $\kappa = 15$ and record the rest lengths of the first 10 collapsing junctions. Unlike in the honeycomb lattice where junctions at $\kappa = 15$ collapse only for $1/\tau_m < 3.2$ (Fig. 2A), in disordered tissues short junctions collapse even at higher $1/\tau_m$ values (Fig. 3A), confirming that the distribution of rest lengths in disordered tissues importantly affects local junctional stability.

Finally, recasting the stability condition of the local model in the dimensional form $\kappa^* > \gamma_0/(3l_0^2) - \eta/(3l_0^2\tau_m)$ suggests that the rest junctional tension $\gamma_0$ may affect the critical length for collapse. In particular, tensions with higher $\gamma_0$ are expected to increase the critical rest length $l_0^*$, making longer junctions more susceptible for collapse.

We test this prediction by analyzing a tissue that contains a supracellular cable of enriched junctional myosin, resulting in higher rest tensions on the corresponding....

**FIG. 2.** (A) Phase diagram of the full tissue exhibits stable (S), collapse (C), and transient oscillations to the fixed point (TO). (B,C). Junctional length (B) and tension (C) vs. time for examples with quasi-oscillations (grey and orange curves) and stochastic fluctuations (blue). (D) Amplitude of length fluctuations vs. myosin turnover rate $1/\tau_m$ for $\sigma = 0.01–0.2$ (black-to-red color scheme). (E) Power spectral densities of length and tension fluctuations. Grey, orange, and blue circles (panel A) and curves (panels B, C, and E) correspond to $(1/\tau_m, \kappa) = (3.25, 15)$, (6, 1), and (100, 0.1), respectively, whereas $\sigma = 0.01$.  

**Quasi-oscillations**

Noise is ubiquitous in biological systems. In our system, the binding-unbinding dynamics of myosin is in fact a stochastic process that gives rise to fluctuating junctional tensions [17, 25, 26]. To explore the role of noise, we add an extra term to the equation for tension, which now reads $\dot{\gamma}_{ij} = \Delta \gamma_{ij} + \sqrt{2\sigma^2/\tau_m} \xi_{jk}$. Here the first term obeys Eq. (2) like before, whereas the second term describes the white noise with long-time variance $\sigma^2$ and $(\xi_{jk}(t)) = 0$, $(\xi_{jk}(t)\xi_{lm}(t')) = \delta(t-t').$  

Note that in the absence of the coupling term in Eq. (2), our stochastic tension dynamics reduces to the classical Ornstein-Uhlenbeck (OU) process [17, 26].

We simulate the stochastic dynamics in a honeycomb cell tiling and find that while in the stable (S) regime, length (and tension) fluctuations are noisy (blue curves in Fig. 2B and C), the movements become more regular in the regime of transient oscillations (TO) and, in contrast to the deterministic case, manage to sustain a well-defined amplitude (orange and grey curves in Fig. 2B and C). These movements in fact correspond to the transient oscillations, which eventually die out in the purely deterministic case (Fig. 1D), but get amplified (Fig. 2D)....
junctons compared to other junctions (Fig. 3B). Without the coupling term in the dynamic equation for tension [Eq. (2)], this cable would be stable despite being under higher tension, since every vertex within the cable is acted upon by a pair of equal but opposite forces. However, as predicted by the dimensional stability condition of our local model, the coupling indeed affects the stability of the junctions that are under higher rest tension, making them more susceptible for collapse (Fig. 3C).

DISCUSSION

We studied a mechanical model of tissues with a feedback loop between junctional contractions and the dynamics of junctional tensions (Fig. 1). In particular, we used a previously proposed description of force generation at the level of the actomyosin [19] and combined it with the vertex model of solid confluent tissues, which provided a faithful representation of elasticity underlying the response of tissues to local force dipoles at the junctions. We showed that nonlinearities in this system do not meet the conditions for a stable limit cycle of junctional movements (Fig. 1A). Nevertheless, we discovered that junctional noise can amplify and sustain quasi-periodic junctional dynamics (Figs. 2) at biologically relevant myosin-turnover rates [17]. Importantly, this dynamical regime is free of any requirements on the elasticity and may thus be more common than the stable limit cycle. Another important result of our work highlights the role of cell arrangements and the distribution of rest tensions within the tissue for the junctional stability (Fig. 3). Both effects may be present during convergence and extension in Drosophila embryo, where the so-called parasegmental boundaries, enriched with the junctional myosin, frequently collapse their junctions [14, 15].

An interesting future direction would be to explore tissues that are closer to the solid-fluid transition. These tissues are associated with highly nonlinear elasticity [24, 30, 31], which could, in contrast to our model, yield a stable limit cycle [19]. In turn, this could lead to spontaneous organization of junctions into groups of locally synchronized oscillators with possible implications for patterning and the onset of morphogenetic movements in early embryos.

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Elasticity, stability and quasi-oscillations of cell-cell junctions in solid confluent epithelia: Supplemental Material

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FIG. S1. (A) Deformation field due to a local force dipole (red arrows) in a honeycomb cell tiling. (B) Coefficients $\alpha_i$ vs. rest length $l_0$ from force-extension relations $f(l) = \sum_{m=1}^{3} \alpha_m (l - l_0)^m$ for junctions extracted from disordered tissues. (C) The distribution of second- and third-order non-linear coefficients in disordered tissues. The green circle in panel B denotes the pair of nonlinear coefficients corresponding to the state equation of an edge in the honeycomb lattice of cells.

I. EQUATION OF STATE

We treat a quartet of hexagonal cells in which the central pair of vertices with positions $r_1 = (0, y_1)$ and $r_2 = (0, y_2)$ move symmetrically with respect to the x-axis (i.e., $y_1 = -y_2$), whereas all the other vertices are fixed (Fig. S2A). While such vertex displacements preserve the total area of this four-cell neighborhood, the areas of individual cells $A_A$, $A_B$, $A_C$, and $A_D$ do change with vertex displacements. Nevertheless, the system possesses two axes of mirror symmetry, meaning that $A_A = A_B$, $A_C = A_D$, whereas the length of all four edges adjacent to the central edge,

$$\lambda(l) = \sqrt{\left(\frac{\sqrt{3}}{2} l_0\right)^2 + \left(\frac{l}{2} - l_0\right)^2}. \quad (S1)$$

We refer to these edges as peripheral edges and to preserve the symmetry, we assume that they are under the same tension $\gamma_p = \gamma_p(t)$; tension on the central edge is different from that at peripheral edges and is denoted by $\gamma$.

The total energy of the system is given by

$$W = \kappa \sum_{\alpha} (A_{\alpha} - A_0)^2 + \sum_{<i,j>} \gamma_{ij} l_{ij}. \quad (S2)$$

We are interested in the dynamics of the central edge length $l = y_2 - y_1$. Therefore, we need to first derive the dynamics equations for $y_1$ and $y_2$, which obey $\eta dy_1/dt = -\partial W/\partial y_1$ and $\eta dy_2/dt = -\partial W/\partial y_2$, respectively. In
The equation of state of our local model agrees well with the one describing junctional elasticity in the full tissue. This is somewhat surprising, since any junction deformation in the local model necessarily changes the areas of all 4 cells involved (due to limited number of degrees of freedom), whereas multiple-cells deformations in the full tissue model allow preserving cell areas. Nevertheless, provided that the local model allows significant area deformations ($\kappa = 1$ in Fig. S2B), the equations of state are qualitatively the same in both systems.

The complete dynamics of the local model is given by the following set of differential equations:

\[
\eta \frac{dl}{dt} = -3\kappa l_0^3 \left( \frac{l}{l_0} - 1 \right) - 2\gamma - 2\gamma p \frac{l - 2l_0}{\lambda(l)},
\]

\[
\frac{d\gamma}{dt} = -\frac{1}{\tau_m} (\gamma - \gamma_0) - \frac{\gamma dl}{l dt},
\]

\[
\frac{d\gamma p}{dt} = -\frac{1}{\tau_m} (\gamma_p - \gamma_0) - \frac{\gamma p d\lambda dl}{\lambda(l) dt}.
\]

\[\text{S16}\]

\[\text{S14}\]

\[\text{S15}\]

\[\text{S10}\]

\[\text{S7}\]

\[\text{S6}\]

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\[\text{S4}\]

\[\text{S3}\]

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\[\text{S4}\]

\[\text{S3}\]

\[\text{S2}\]

\[\text{S1}\]

\[\text{S0}\]


\[\text{II. LINEAR STABILITY ANALYSIS}\]

\[\text{A. Hopf bifurcation}\]

We assume a small perturbation from the fixed point by setting $l = l_0 + \delta l$, $\gamma = \gamma_0 + \delta \gamma$, and $\gamma_p = \gamma_0 + \delta \gamma_p$, where $\delta l$, $\delta \gamma$ and $\delta \gamma_p \ll 1$. The linearized system can be written in a matrix form as $d\rho/dt = J\rho$, where $\rho = (\delta l, \delta \gamma, \delta \gamma_p)$ and the Jacobian matrix

\[
J = \begin{bmatrix}
-\frac{3(2\kappa l_0^3 + \gamma)}{2l_0^3} & -\frac{2}{\eta} & \frac{2}{\eta} \\
\frac{3\gamma(2\kappa l_0^3 + \gamma)}{2l_0^3} & \frac{2\gamma}{\eta l_0} - \frac{1}{\tau_m} & -\frac{2\gamma}{\eta l_0} \\
-\frac{3\gamma_0(2\kappa l_0^3 + \gamma)}{8l_0^3} & -\gamma_0/2l_0 & \gamma_0/2l_0 - \frac{1}{\tau_m}
\end{bmatrix}.
\]

The Jacobian has three eigenvalues: The first one, $\Lambda_1 = -1/\tau_m$, is always negative, whereas the remaining two read

\[
\Lambda_{\pm} = \frac{b}{2a} \left( -1 \pm \sqrt{1 - \frac{4ac}{b^2}} \right),
\]

where $a = 2\eta l_0 \tau_m > 0$, $b = \frac{2}{\eta l_0}$, and $c = 3(2\kappa l_0^3 + \gamma_0) > 0$. The eigenvalues are plotted in Fig. S2C.

The system is at the critical point when $\Lambda_+ = 0$; i.e., $\Lambda_+ < 0$ yields stable fixed point, whereas $\Lambda_+ > 0$ yields unstable fixed point. Since the expression in parenthesis in Eq. (S18) is always strictly negative, the Hopf bifurcation needs to satisfy the condition $b = 0$. This gives the critical area-compressibility modulus, which reads

\[
\kappa^* = \frac{\gamma_0}{3l_0^3} - \frac{\eta}{3l_0^3 \tau_m}.
\]

\[\text{S19}\]
B. Transient oscillations

By considering the stable system, i.e., \( \kappa > \kappa^* \), we derive the condition for the existence of transient oscillations, which imply that the eigenvalues have a non-zero imaginary part. This is true when \( 1 - 4ac/b^2 < 0 \) or

\[
\kappa < \gamma_0 \frac{3\beta}{3\beta_0 + \frac{\eta}{3\beta_0}} + \frac{1}{3\beta_0} \sqrt{10\gamma_0} \frac{\tau_0\gamma_0}{\tau_0\gamma_0}. \tag{S20}
\]

The frequency of transient oscillations is given by \( \omega = (1/2) \sqrt{4ac/b^2 - 1} \) and reads

\[
\omega = \sqrt{\frac{6\gamma_0\tau_0(2\gamma_0 + \gamma_0)}{(\eta_0 + 3\beta_0\tau_0 - \gamma_0\tau_0)^2} - 1}. \tag{S21}
\]

III. FIRST LYAPUNOV COEFFICIENT

To examine the stability of the limit cycle, we calculate the first Lyapunov exponent \( \lambda_1 \) at the critical point, i.e., for \( \kappa = \gamma_0/(3\beta_0) - \eta/(3\beta_0\tau_0) \). First, we compute eigenvectors and eigenvalues of the Jacobian \( J \) at the critical point and we denote by \( q \) the eigenvector associated to the eigenvalue \( i\omega_0 \) where \( \omega_0 = \sqrt{(b^2 - 4ac)} \). We do the same for the Jacobian transpose \( J^T \) and denote by \( p \) its eigenvector associated with eigenvalue \( -i\omega_0 \). Then we Taylor-expand our system around its fixed point to third order:

\[
\frac{d\rho}{dt} = F(\rho) = J\rho + \frac{1}{2}b(\rho, \rho) + \frac{1}{6}c(\rho, \rho, \rho), \tag{S22}
\]

where the components of \( b \) and \( c \) read

\[
B_j(u, v) = \sum_{k,l=1}^{3} \frac{\partial^2 F_j(\rho)}{\partial \rho_k \partial \rho_l} \bigg|_{\rho = 0} u_k v_l, \tag{S23}
\]

and

\[
C_j(u, v, w) = \sum_{k,l,m=1}^{3} \frac{\partial^3 F_j(\rho)}{\partial \rho_k \partial \rho_l \partial \rho_m} \bigg|_{\rho = 0} u_k v_l w_m. \tag{S24}
\]

The first Lyapunov coefficient \( \lambda_1 \) is then given by

\[
\lambda_1 = \frac{1}{2\omega_0} \Re \{ \langle \delta F_j, c(q, q, q) \rangle - 2 \langle \delta F_j, b(q, L^{-1}b(q, q)) \rangle + \langle \delta F_j, b(q, (2i\omega_0 I_3 - L)^{-1}b(q, q)) \rangle \} \tag{S25}
\]

and it turns out strictly positive for \( 1/\tau_m \in (0, 1/\lambda_0) \) and therefore the system never displays a stable limit cycle (Fig. S2D).

IV. LINEAR STABILITY ANALYSIS OF FULL TISSUE

To study the stability of the full tissue, we need to write down the system of equations in its full form. In particular, given \( N_v \) vertices and \( N_e \) edges, we have \( 2N_v \) differential equations for positions of the vertices and \( N_e \) differential equations for tensions. In particular, the force exerted on vertex \( i \) reads

\[
F_i = \eta \frac{dr_i}{dt} = -\sum_j \gamma_0 \nabla_{ij} - 2k \sum_k (A_k - A_0) \nabla_i A_k, \tag{S26}
\]

whereas the rate of change of tension at junction connecting vertices \( i \) and \( j \) reads

\[
\frac{d\gamma_{ij}}{dt} = -\frac{1}{\tau_m} (\gamma_{ij} - \gamma_0) - \frac{\gamma_k(F_j - F_i) \cdot (r_j - r_i)}{l_{ij}^2 \eta}. \tag{S27}
\]

We perturb the system around the fixed point, where the vertices assume their positions in a regular hexagonal lattice, whereas tensions on all junctions equal 1. We linearize the system, which then reads

\[
\delta F_i = \eta \frac{d\delta r_i}{dt} = \sum_j \delta A_{ij} + \sum_k \delta A_{ik}, \tag{S28}
\]

and

\[
\frac{d\delta \gamma_{ij}}{dt} = -\frac{1}{\tau_m} \delta \gamma_{ij} - \delta \Gamma_{ij}. \tag{S29}
\]

Here

\[
\delta A_{ij}^l = \frac{\gamma_0}{l_{ij}^3} \left[ (y_i - y_j) \right] \left[ (y_i - y_j) \right] \left( -\delta x_i + \delta x_j \right) + \left( x_i - x_j \right) \left( \delta y_i - \delta y_j \right) \left( 1 \right), \tag{S30}
\]

\[
\delta A_{ik}^A = \frac{K}{2} \left[ (y_{ik+1} - y_{ik-1}) \left[ -\sum_{\nu \in k} (y_{\nu+1} - y_{\nu-1}) \delta x_\nu + \sum_{\nu \in k} (x_{\nu+1} - x_{\nu-1}) \delta y_\nu \right] \right] \left( 1 \right), \tag{S31}
\]

and

\[
\delta \Gamma_{ij} = \frac{\gamma_0 (\delta F_j - \delta F_i) \cdot (r_j - r_i)}{l_{ij}^2 \eta} = \frac{\gamma_0}{l_{ij}^2 \eta} \left[ \delta F_j^2 (x_j - x_i) + \delta F_j^y (y_j - y_i) - \delta F_i^x (x_j - x_i) - \delta F_i^y (y_j - y_i) \right]. \tag{S32}
\]
Here \( r_i = (x_i, y_i) \) and \( l_{ij} = \sqrt{(x_i - x_j)^2 + (y_i - y_j)^2} \) are positions of vertices and edge lengths, respectively, at the fixed point. Figure S3 shows the dependence of the maximal eigenvalue of the Jacobian as a function of \( 1/\tau_m \) at fixed \( \kappa = 0.1 \).

![Figure S3. Maximal real and imaginary parts of the eigenvalues of the Jacobian versus \( 1/\tau_m \) at fixed \( \kappa = 0.1 \).](image)

V. NOISE

We rewrite the system of differential equations, adding the noise to the tension dynamics:

\[
\eta \frac{dl}{dt} = -3K_0^3 \left( \frac{l}{l_0} - 1 \right) - 2\gamma - 2\gamma_p \frac{l - 2l_0}{\lambda(l)} , \quad \text{(S33)}
\]

\[
\frac{d\gamma}{dt} = -\frac{1}{\tau_m} (\gamma - \gamma_0) - \frac{\gamma}{l} \frac{dl}{dt} + \frac{2\sigma^2}{\tau_m} \xi(t) , \quad \text{(S34)}
\]

\[
\frac{d\gamma_p}{dt} = -\frac{1}{\tau_m} (\gamma_p - \gamma_0) - \frac{\gamma_p}{\lambda(l)} \frac{dl}{dt} + \frac{2\sigma^2}{\tau_m} \xi_p(t) , \quad \text{(S35)}
\]

where \( \xi(t) \) is a Gaussian random variable with zero average and variance equal to 1. Its correlator reads

\[
\langle \eta(t) \eta(t') \rangle = \langle \eta_p(t) \eta_p(t') \rangle = \delta(t - t') \quad \text{(S36)}
\]

and

\[
\langle \eta(t) \eta_p(t') \rangle = 0 . \quad \text{(S37)}
\]

The noise increases the probability for the system to escape the basin of attraction and thus expands the collapse regime in the \((1/\tau_m, \kappa)\) parameter space. Close to the boundary between the collapse and stable regimes, the system exhibits quasi-oscillations. These are instances where the noise amplifies the transient oscillations and sustains them for an indefinite time period.

To compute the power spectrum of the quasi-oscillations, we again linearize the system around the fixed point: \( l = l_0 + \delta l \), \( \gamma = \gamma_0 + \delta \gamma \), and \( \gamma_p = \gamma_0 + \delta \gamma_p \).

The linearized system of stochastic differential equations can then be written in a matrix form as

\[
\frac{d}{dt} \mathbf{v} = \mathbf{J} \mathbf{v} + \mathbf{\eta} . \quad \text{(S38)}
\]

Next, we write \( \mathbf{v} \) in the Fourier space as \( \tilde{\mathbf{v}}(\omega) = \int \mathbf{v}(t) \exp(i\omega t) \) to obtain \( \langle \omega^2 \rangle d\omega = (\mathbf{\Phi}^{-1}(\omega) \mathbf{D} \mathbf{\Phi}(\omega) \rangle \tilde{\mathbf{v}}(\omega) \).

Denoting \( \Phi(\omega) = i\omega \mathbb{I} - \mathbf{J} \) and solving for \( \tilde{\mathbf{v}}(\omega) \) gives

\[
\tilde{\mathbf{v}}(\omega) = \Phi^{-1}(\omega) \mathbf{\eta}(\omega) . \quad \text{(S39)}
\]

Now we can calculate the power spectral density matrix (PSDM) with the elements given by

\[
P_{ij}(\omega) = \langle \tilde{v}_i(\omega) \tilde{v}_j^*(\omega) \rangle = \sum_{l=1}^{3} \sum_{m=1}^{3} \Phi_{ij}^{-1}(\omega) D_{lm} \left[ \Phi_{lm}^{-1}\right]^*(\omega) , \quad \text{(S40)}
\]

where

\[
D = \frac{2\sigma^2}{\tau_m} \begin{pmatrix} 0 & 0 & 0 \\ 0 & 1 & 0 \\ 0 & 0 & 1 \end{pmatrix} . \quad \text{(S41)}
\]

The diagonal entries of the PSDM are real and coincide with the power spectra for the fluctuations, associated with the three species \( l \), \( \gamma \), and \( \gamma_p \). In particular, the power spectrum for the junctional length fluctuations reads

\[
P_l(\omega) = \frac{16\sigma^2}{\tau_m g(\omega)} \left( \frac{1}{\tau_m^2} + \omega^2 \right) , \quad \text{(S42)}
\]

where

\[
g(\omega) = -i\omega^3 + \left( \frac{\gamma_0}{l_0} - 3\kappa l_0^2 - \frac{2}{\tau_m} \right) \omega^2 + \left( \frac{1}{\tau_m^2} + \frac{\gamma_0}{2l_0\tau} + \frac{6\kappa l_0^2}{\tau_m^2} \right) \omega + \left( \frac{3\gamma_0}{2l_0\tau} + \frac{3\kappa l_0^2}{\tau_m^2} \right)^2 . \quad \text{(S43)}
\]

VI. CORRELATION OF QUASI-OSCILLATIONS

In tissues, cell-cell junctions are physically coupled and are therefore forced to synchronize their movements. For
example, junctions can lengthen in expense of the shortening of their immediate neighbors. In polygonal networks, three or more junctions meet at a vertex, which prevents this pairwise relation from being simultaneously satisfied for all pairs of adjacent junctions. To explore what patterns of junctional movements this geometric frustration establishes, we simulate collective quasi-oscillations within a regular honeycomb cell tiling. We measure the Pearson correlation coefficient, $p$, between junctional lengths $l_{ij}(t)$ and the length of the reference junction $l_{\text{ref}}(t)$. Not surprisingly, we find that overall, the correlation strength $|p|$ decreases with the topological distance from the reference junction $d$ (Fig. S4A); the topological distance $d$ is defined as the integer shortest path between a junction and the reference junction; $d = 1$ for the nearest neighbors, $d = 2$ for the next-nearest neighbors, etc. However, $|p|(d)$ is non-monotonic, which suggests a nontrivial structure of the correlation map within the cell-junctions network. Indeed, the correlation pattern exhibits two clear features: (i) correlation is stronger between junctions that are mutually parallel (Fig. S4B) and (ii) chains of cells running perpendicularly to the reference junction alternate in the strength of correlation (black arrows in Fig. S4B).

It is also interesting to observe how junctions synchronize their quasi-oscillations within individual cells. In particular, we focus on the sign of the correlation coefficient $p/|p|$, which tells whether the pair of junctions is correlated ($p/|p| = +1$) or anti-correlated ($p/|p| = -1$) (Fig. S4C). We define cumulative correlation of adjacent junctions in a given cell by $P = \sum_{\mu=1}^{6} \left( p_{\mu}/|p_{\mu}| \right) \cdot \left( p_{\mu+1}/|p_{\mu+1}| \right)$, where the index $\mu = 1, ..., 6$. As a result of geometric frustration at the vertices, not all cells can have $P = -6$ (the case in which all pairs of adjacent junctions within a cell are anti-correlated). Interestingly, this gives rise to the formation of a local stripe-like pattern of cells, where $P$ alternates between $-6$ and $+2$, highlighting the role of mechanics in synchronization of cell deformations (Fig. S4D).