

Shape Switching and Tunable Oscillations of Adaptive Droplets

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Living materials adapt their shape to signals from the environment, yet the impact of shape changes on signal processing and associated feedback dynamics remains unclear. We find that droplets with signal-responsive interfacial tensions exhibit shape bistability, excitable dynamics, and oscillations. The underlying critical points reveal novel mechanisms for physical signal processing through shape adaptation in soft active materials. We recover signatures of one such critical point in experimental data from zebrafish embryos, where it supports boundary formation.

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Dynamic and nontrivial geometries are hallmarks of soft active matter, because intrinsic stress fields drive autonomous shape changes in deformable materials [1–7]. In turn, boundary geometry influences stresses and material properties, or determines how macroscopic work is extracted from microscopic activity [8–10]. In complex materials that process signals and adaptively respond to their environment [11–14], geometry-dependent feedback arises when signal processing depends on the system’s shape.

In particular living materials possess internal degrees of freedom that adjust their mechanical properties in response to peripheral signals. In cells, external signals trigger biochemical processes including the regulation of genes that control the molecular composition in the bulk and at the surface [15], Chap. 15]. Inhibitory signaling interactions between neighboring cells, for example, lead to the spontaneous symmetry breaking of such internal states, giving rise to distinctly shaped cell types [16,17]. The resulting mechanochemical feedback governs the spatial organization of diverse multicellular systems [18–20], enabling them to solve tasks including locomotion [21–24], self-healing [25,26], and the self-organization of complex structures [27–29]. Yet, how internal cellular states interact with shape dynamics is an open question [29–32], and more generally, how the phase space of adaptive shape-changing materials depends on geometry is unclear.

Uncovering theoretical principles that govern the rich physics of adaptive active matter systems requires minimal, tractable paradigms. Here, we consider adaptive droplets that change their surface tension in response to signals received at contacts with other droplets. To analyze how signal processing interplays with droplet geometry, we introduce a minimal set of equations governing the droplet states, which can be derived from microscopic reaction-diffusion dynamics of signaling and adhesion molecules, as shown in a companion paper, Ref. [33].

We show that feedback between active shape changes and signal-responsive internal states generates nontrivial critical regimes that enable functional collective states. *Positive* mechanochemical feedback drives transitions from loose to compact droplet configurations, while *inhibitory* interactions lead to excitable and oscillatory droplet dynamics.

Applying our theory to zebrafish embryos, we find that positive feedback between cell-cell adhesion and signaling supports the formation of a boundary that eventually separates the interior and exterior parts of the embryo. Our parameter estimates reveal that the embryo operates near a cusp bifurcation point—supporting the emerging view that living matter tunes its proximity to critical points to achieve biological outcomes [34–37].

Adaptive Young-Laplace droplets—We consider configurations of Young-Laplace droplets with interfacial areas governed by the conjugate uniform surface tensions at fixed volumes [Fig. 1(a)]. The total surface energy in a system of N droplets is

$$E = \sum_{i=1}^N \frac{\gamma_c}{2} A_{c,i} + \gamma_f A_{f,i}, \quad (1)$$

in which γ_c and γ_f are the surface tensions of the contact interfaces $A_{c,i}$ and the free surface areas $A_{f,i}$, respectively.

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For configurations in which each droplet has n neighbors and no triple or higher-order junctions are present, the total contact area per droplet that minimizes the surface energy Eq. (1) is [39], Sec. A]

$$\frac{A_c}{A_0} = n \left[1 - \left(\frac{\gamma_c}{2\gamma_f} \right)^2 \right] \left[\frac{2}{\left(2 - \frac{\gamma_c}{2\gamma_f} \right) \left(1 + \frac{\gamma_c}{2\gamma_f} \right)^2 - (n-1) \left(2 + \frac{\gamma_c}{2\gamma_f} \right) \left(1 - \frac{\gamma_c}{2\gamma_f} \right)^2} \right]^{\frac{2}{3}}, \quad (2)$$

in which the reference area $A_0 = (3V/2)^{2/3}\pi^{1/3}$ is defined by the conserved droplet volume V . While Eq. (2) holds for doublets within the full stable-contact regime $0 \leq \gamma_c \leq 2\gamma_f$, square ($n = 4$) and cubic ($n = 6$) lattices form higher order junctions when $\gamma_c/2\gamma_f \leq 1/\sqrt{2}$ [Fig. 1(b)].

We take the adhesion between droplets to depend on the signals received at their contact surfaces. Therefore, we assign a dimensionless *internal state* $u_i \in [0, 1]$ to each droplet $i \in 1, 2, \dots, N$ —a variable which increases in response to received signals in a saturating manner [20,54,55]. When the internal states drive active processes that increase the adhesion between droplets, a bilinear relation that links the tension at the interface between droplets i and j to their internal states is obtained when expanding around a constant γ_0 [33]

$$\gamma_c = \gamma_0 - \gamma_A u_i u_j, \quad (3)$$

in which the second term contains the active contributions in response to signaling, and γ_0 all state-independent

contributions. Diverse microscopic processes can modulate the effective tension at the surface of cells, including the generation of stresses by molecular motors [56–58], and biochemical regulation of adhesion ([15], Chap. 19, [59,60]).

Equation (3) can be derived from mass-action reaction kinetics, in which the internal states control the active production of adhesion molecules in the bulk, which bind across the interface and lower the surface energy [33]. The *adaptive adhesion coefficient* γ_A captures the coupling strength between internal states and interfacial tension. Because adhesion reduces the tension, the adaptive term is always negative and the coefficient can be expressed as $\gamma_A = \epsilon/\lambda^2$ in terms of an energy ϵ per adhesion complex and a length scale λ dependent on the rate of the adhesion molecules' turnover at the bulk-surface interface [33]. The adaptive tension vanishes without signaling and it always increases the interfacial area $A_c(\gamma_0) \leq A_c(\gamma_c)$ [Eq. (2)] up to the saturation limit of the internal states.

The state of a droplet u_i is taken to evolve according to a generic signaling interaction [20,55]

$$\tau_u \frac{du_i}{dt} = \sigma(s_i) - u_i, \quad (4)$$

in which $\sigma(s_i)$ is an increasing sigmoidal response function to a signal s_i received by droplet i , which we model with a Hill function $\sigma(s_i) = s_i^h / (s_i^h + 1)$ [61,62] [Fig. 1(c)]. We use in the following a linear-order relation between the received signal s_i and the available external signal ϕ ,

$$s_i = \chi \frac{A_c}{A_0} \phi, \quad (5)$$

which takes into account the dependence of the received signal on the contact area [63,64] with a *susceptibility* χ . Equations (4) and (5) can be derived from reaction-diffusion dynamics of biochemical signaling molecules where u_i is defined as a rescaled and normalized concentration of a regulator molecule, such as a transcription factor [33]. These microscopic equations relate χ to the steady-state concentrations and kinetic rates associated with the turnover, binding, and processing of signaling molecules in a cell.

Provided that the adaptive droplets relax to their equilibrium shapes faster than the signaling time scale τ_u , the collective droplet dynamics are governed by Eqs. (2)–(5). Indeed, for cells and cellular aggregates, the viscoelastic response to mechanical stresses is usually on the order of

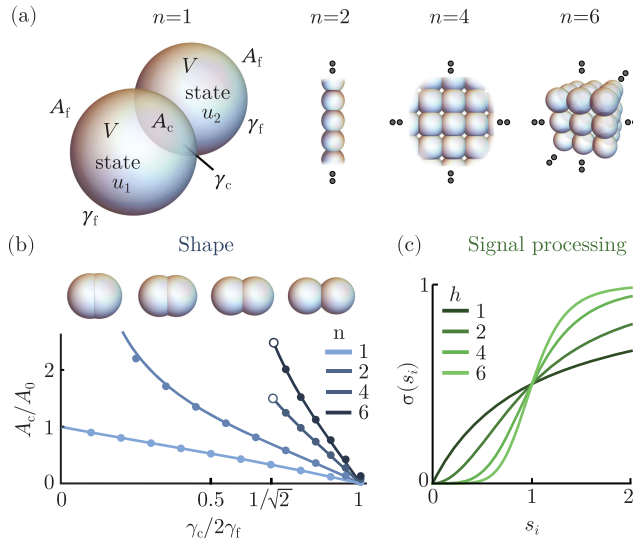


FIG. 1. Droplets, characterized by equal conserved volumes and uniform surface tensions γ_f and γ_c , form n contacts without triple or higher-order junctions. (a) Equilibrium shapes in various conformations. (b) Total contact area per droplet depends on n and the ratio $\gamma_c/2\gamma_f$. Solid line: Eq. (2). Filled circles: numerical simulations [38], [39], Sec. B]. Empty circles: appearance of higher-order junctions. Top images: contacts with $\gamma_c/2\gamma_f = \{0.2, 0.4, 0.6, 0.8\}$. (c) Sigmoidal response of the internal states $u_{i=1,2,\dots}$ [Eq. (4)], which control γ_c , to A_c -dependent signals s_i .

seconds to minutes [65,66], while protein production and degradation take place on timescales of tens of minutes to several hours [67,68]. However, surface-mechanical changes due to local biochemical processes are faster [68], and could lead to interplay with the shape dynamics depending on the frictional timescale. In the low-friction limit, two coupling coefficients characterize the feedback between the system's geometry and the internal states: the adaptive adhesion coefficient γ_A controls the effect of signaling on the surface mechanics and, thereby, on the shape; the signal susceptibility χ captures how transmitted signals depend on the contact area.

Tension adaptation produces a shape transition—Numerical continuation of Eqs. (2)–(5) for constant external signals ϕ and varying parameters γ_A and χ reveals a bistable regime [39, Sec. C], delineated by two saddle-node (SN) lines originating from a codimension-2 cusp point [Fig. 2(a)]. Within this regime, two stable droplet configurations coexist: a weakly adhesive configuration with small interfaces and uniform low levels of u_i , and a large-contact configuration with high u_i . This bistability is a signature of the positive feedback between the contact areas and the strength of the signals received by the droplets. Since the contact area limits the amplitude of adhesion-inducing signals, small contact areas remain weakly adhesive, whereas the large ones support transmission of strong signals, in turn promoting adhesion.

We expect this feedback-driven shape transition to occur in diverse systems where area-dependent signals affect mechanical changes. As an example of a specific mechanochemically regulated system, we experimentally investigated cell shapes and signaling responses in the zebrafish blastoderm. In this embryonic tissue, it was previously shown that Nodal, an extracellular signaling molecule involved in cell fate specification [70–72], increases intercellular adhesion [36], and that cell-cell contacts can in turn enhance the competence of cells to respond to Nodal [73–75]. In the presence of such adaptive adhesion, we predict that cellular configurations should undergo a switch from strong to weak adhesion as a function of the external signal ϕ in Eq. (5), which thereby acts as the control parameter. Importantly, the external level of Nodal varies spatially in the embryo [Fig. 2(b)], decreasing from the tissue margin to the animal pole [72], allowing us to directly test this prediction. In particular, we expect that a positive mechanochemical feedback between cell-cell adhesion and received signals leads to distinct tissue regions with high- and low-adhesion conformations, respectively.

We measured cell-cell contact angles θ from fluorescence microscopy images of embryos taken at two different time points (five hours post fertilization and 30 min later) at varying positions $y \geq 0$ along the embryo axis [Fig. 2(b)]. In addition, we measured the relative nuclear fluorescence intensity of the Nodal signal-transducer, the phosphorylated Smad complex I_s [69,76–78], [39, Sec. D].

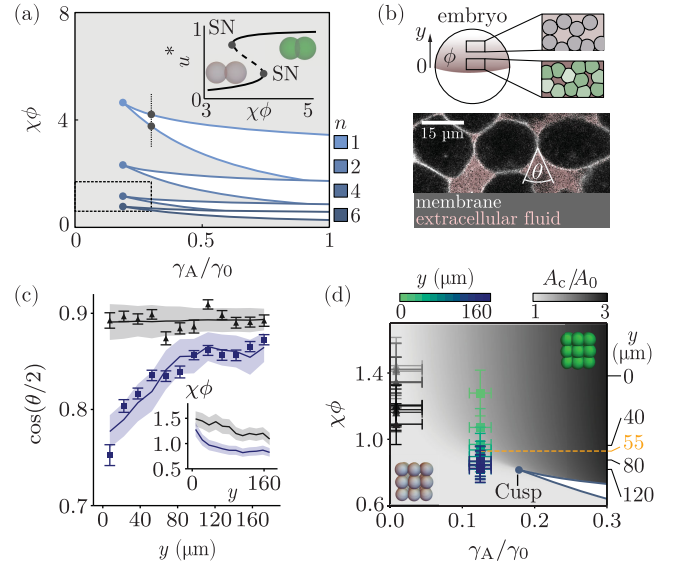


FIG. 2. Adaptive tension produces a shape transition which supports patterning in zebrafish embryos. (a) Positive feedback between contact-dependent signals and adaptive adhesion leads to bistability between small- and large-contact configurations. White regions are bistable; the dashed box corresponds to the regime shown in panel (d). Inset: steady state bifurcation curve along dotted line. Blue points: cusps. (b) The cells of the zebrafish blastoderm respond to a signal gradient (brown shading) along the embryo axis y by increasing cell-cell adhesion. Bottom: we measured contact angles θ between cells from membrane-labeled fluorescence microscopy images. (c) Fitting contact angle profiles from wild-type (blue squares, 2096 cells from 8 embryos) and *silberblick* mutants with disrupted adhesion regulation (gray triangles, 1209 cells from 6 embryos) allows estimating their adaptive adhesion coefficients $\gamma_A/2\gamma_f$, and computing $\chi\phi(y)$ (inset) from fluorescence-intensity measurements of the signal-transducer molecules [69] [39, Fig. S2]. Error bars and shaded areas: standard error of the mean. Solid lines: best fit of Eq. (6) [39, Sec. E2]. (d) The estimated parameters locate the wild-type cells close to the cusp, whereas mutants lose adaptive adhesion (images illustrate $\gamma_c/2\gamma_f = \{0.88, 0.77\}$). Spatial positions (color, right axis), computed from interpolation of $\chi\phi(y)$ (c, inset), show where proximity to the cusp separates tissue regions of weak and strong adhesion with distinct contact areas (gray shading). Orange dashed line: position of the predicted half-maximum contact area for the wild-type adaptive tension estimate. The white region is bistable. (a)–(d) Were computed with $h = 4$ and Eq. (2) linearized around $\gamma_c/2\gamma_f = 1$ [39, Fig. S1(d)].

Averaging these measurements over 15- μm bins along the tissue axis, we approximate the average signal received by cells within a bin centered at y_k as $s(y_k) = s_0 I_s(y_k)$ with a scaling factor s_0 . Evaluating the interfacial tension at steady state as $\gamma_c(y_k) = \gamma_0 - \gamma_A \sigma(s(y_k))^2$ [Eq. (3)], i.e., neglecting differences in the received signal between the cells within the same bin, we performed a least-square fit of the contact angle profile using the force-balance relation

$$\cos\left[\frac{\theta(y_k)}{2}\right] = \frac{\gamma_c(y_k)}{2\gamma_f}, \quad (6)$$

and found an excellent agreement between fitted curves and measurements [Fig. 2(c)], [39, Sec. E]. The obtained parameter estimates $\gamma_0/2\gamma_f = 0.88 \pm 0.01$, and $\gamma_A/2\gamma_f = 0.11 \pm 0.02$ are corroborated by simulation-based inference on the pooled contact-angle measurements, assuming an exponential profile of the external signal [39, Sec. E4].

Genetic perturbation of adhesion regulation in *silberblick* mutants (SLB) [36,79,80] yielded a significant reduction in the best-fit adaptive tension coefficient as expected, with the baseline tension remaining close to the wild-type value ($\gamma_{A,SLB}/2\gamma_f = 0.01 \pm 0.03$, $\gamma_{0,SLB}/2\gamma_f = 0.89 \pm 0.01$). The scaling coefficient converting fluorescence intensity ratios into received signals was assumed identical across time points and conditions ($s_0 = 1.0 \pm 0.1$).

Equation (5), which relates the contact area and received signal, allows estimating the local value of $\chi\phi$ according to $\chi\phi(y_k) = s_0 I_s(y_k) A_0/A_c(y_k)$ [Fig. 2(c), inset]. To approximate the average cell-cell contact area, we use the *ordered* lattice expression Eq. (2) with $n = 6$ [39, Fig. S5], corresponding to the average number of cell-cell contacts, in the small-angle limit consistent with the data [39, Figs. S1(d)–S1(e)]. Using these estimates, we find that wild-type blastoderm cells are close to the critical cusp point of the bistable regime, which locates the transition between low- and high-contact regions at approximately 60 μm above the margin of the tissue [Fig. 2(d), orange line]. We thereby recover a length that corresponds to the size of the subsequently developing rigid tissue region, which later forms the internal parts of the organism [69,81–83].

Adaptive tension promotes symmetry breaking—When droplets or cells exchange *state-dependent* signals $\phi = \phi(u_1, \dots, u_N)$, the additional coupling between their internal states gives rise to new regimes. For example, strong *mutually inhibitory* interactions generically lead to spontaneous symmetry breaking, whereby initially small differences between interacting units diverge to low- and high-value steady states [16], a mechanism relevant for the patterning of different cell types [17]. To investigate how signal-induced adhesion affects symmetry breaking, we performed numerical continuation of Eqs. (2)–(5) [39, Sec. C] for droplet pairs with $\phi = \phi_0(1 - u_j)$, an expression we derived from the microscopic dynamics of Delta-Notch signaling [19,33,63]. Droplet pairs undergo symmetry breaking of internal states above a line of supercritical pitchfork bifurcations (PF) in the state diagram spanned by the signal susceptibility χ and the adaptive adhesion coefficient γ_A [Figs. 3(a) and 3(b) triangle]. Adaptive adhesion promotes symmetry breaking by transiently expanding the contact area, which lowers the threshold susceptibility (Movie 1(f) [39]). Adaptive contact dynamics indeed occur in developing zebrafish sensory cell pairs, which exchange mutually inhibitory signals across a

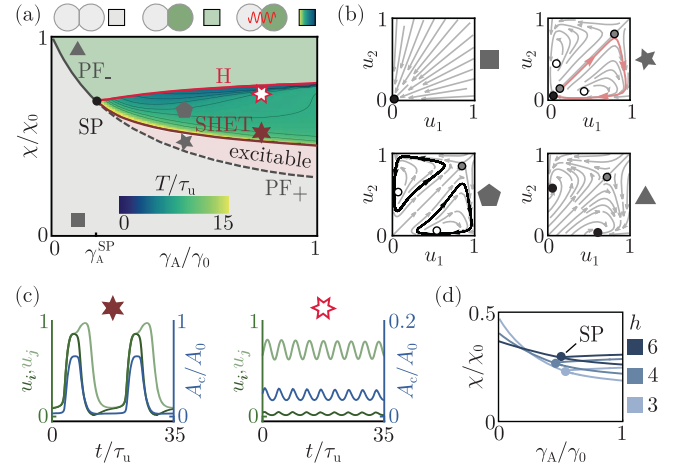


FIG. 3. Excitability and oscillations of adaptive droplet pairs. (a) Tension adaptation increases the parameter regime (green) of symmetry breaking due to mutual inhibition (PF₋: supercritical pitchfork, PF₊: subcritical pitchfork), and leads to self-sustained oscillations of states and droplet shapes. Color gradient and contour lines: oscillation period T . Saddle-heteroclinic (SHET) and Hopf (H) bifurcation lines bound the oscillatory regime, originating from a saddle-node pitchfork codimension-2 point (SP). Reference susceptibility is set by $\chi_0 = \lim_{\gamma_A \rightarrow 0} \chi_{PF}$ in the limit of vanishing adaptive tension. (b) Phase portraits for parameter values marked in (a). Filled black circles: stable steady states, filled gray circles: saddles, open circles: unstable steady states, pink line: trajectory in the excitable regime, black lines: limit cycles. (c) Oscillation amplitudes decrease with waveforms changing from relaxationlike (near SHET) to sinusoidal (near Hopf) for increasing χ . (d) Strong adaptive adhesion lowers symmetry-breaking and oscillation thresholds for small Hill coefficients [39], Parameter values given in Table S1], [cf. Ref. [33], Fig. 5].

transient contact surface to acquire distinct fates before detaching from each other [20,84].

Tunable self-sustained oscillations—For large adaptive adhesion coefficients, simulations and numerical continuation of Eqs. (2)–(5) [39, Sec. C] reveal an oscillatory regime separating regions of symmetric and symmetry-broken steady states [Fig. 3(a)], that originates from a saddle-node pitchfork bifurcation point [33,85]. These oscillations are driven by the competition between shape dynamics and symmetry breaking: the internal states increase the contact area according to Eq. (3), thereby driving their own inhibition, leading to negative feedback. For susceptibilities χ close to the transition between stable uniform and oscillatory states, droplet pairs become excitable [Fig. 3(a)]. In this regime, small perturbations can trigger a large increase in contact area A_c , followed by transient symmetry breaking (Fig. 3(b) star and Movie 1(b) [39]). Stable oscillatory cycles appear once χ is large enough to transmit signals that break symmetry so as to decrease adhesion and the contact area to the point where signals again drop *below* the symmetry-breaking threshold.

Subsequently, the product of states can increase again, and the cycle repeats.

At the critical susceptibility χ_{SHET} , the droplet pair undergoes a saddle-heteroclinic bifurcation, where the uniform stable fixed point collides with the saddle point that sets the excitation threshold. Above the transition, the ghost of this destroyed saddle point slows down the dynamics of the droplet pair, leading to relaxation oscillations, in which the droplets spend a large fraction of the cycle in the low-contact configuration, followed by spikes in the contact area and rapid, transient symmetry breaking [Fig. 3(c)]. At larger susceptibilities, oscillations progressively approach harmonic waveforms, and the limit cycles eventually contract into symmetry-broken fixed points via a Hopf bifurcation [Fig. 3(b) triangle]. Consequently, the supported oscillatory dynamics span a wide range of waveforms and periods, tunable by the droplets' susceptibility.

The size of these dynamical regimes depends on the baseline tension ratio $\gamma_0/2\gamma_f$, and the Hill coefficient h [33]. We found shape bistabilities and symmetry breaking for Hill coefficients $h \geq 2$, and oscillations for $h \geq 3$ [Fig. 3(d)]. Strongly nonlinear response functions are common in cellular regulatory feedbacks [55,86,87], and experimental evidence [88] including our zebrafish embryo data [39, Figs. S4,S7(a)] suggests large Hill coefficients for Nodal signaling.

Together, these results illustrate how shape adaptation serves as a mechanism of signal processing and generates complex temporal dynamics, such as excitability and self-sustained oscillations.

Conclusions—Our analysis of adaptive, interacting droplets reveals the rich physics arising in signal-processing active materials. Coupling between droplet geometry and contact-dependent signals drives shape bistability, robust symmetry breaking, excitability, and oscillations of different waveforms—enabling a wide range of time-encoded outputs with minimal degrees of freedom. Dynamic signaling levels can indeed program distinct cell fates [89–91], whereby shape-dependent feedback could self-organize multicellular structures [92–94]. Applied to experimental observations from zebrafish embryos, our theory suggests that positive shape-adaptive feedback aids tissue boundary formation in the blastoderm.

Our findings will enable the discovery of new collective phenomena in active signal-processing materials, where mechanical feedback drives spontaneous patterning and wave dynamics [95–97]. Investigating fluctuation-induced effects in the excitable regimes, for example, could reveal how large area deviations trigger topological transitions governing global rheological properties [98,99]. Analyzing these collective dynamics would uncover how geometry-dependent feedback produces novel modes of self-organization in adaptive materials, and may inform new paradigms of physical learning [100,101], where materials encode information through dynamical responses shaped by feedback.

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