

Active mechanics of sea star oocytes

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Cell shape changes, driven by the contractile actomyosin cortex, are essential for multicellular organisms. Yet, how contractility and cell shape changes emerge from molecular-scale interactions between proteins in the actomyosin cortex remains unclear. Oocytes of the bat star *Patiria miniata* exhibit a traveling wave of cellular deformation during meiosis, known as a surface contraction wave. Here, we exploit this highly stereotypical deformation to study how cortical properties are set by microscale processes. By pharmacologically modulating the levels of polymerized actin, we show that the wild-type composition maximizes the cellular deformation rate. To understand these results, we developed an active fluid model for shape changes that demonstrates that deformation rates are set by the ratio between cortical viscosity and active contractile stresses and provides a framework for deriving these two key material properties from coarse-grained molecular-scale interactions. In this model, the ratio of active stress to viscosity peaks at intermediate admixtures of passive cross-linkers. This explains the observations of the drug treatment experiments and makes additional predictions, namely, that overexpression of either myosin or passive cross-linkers would decrease deformation rates, which we verified experimentally. Together, this shows that the interplay between cortical active stress and viscosity relies on balancing the ratio of myosin to passive cross-linkers on each filament, which can be modulated by actin density. Our results argue that changing the relative molecular compositions of cortical cross-links and motors provides a mechanism that biological systems could exploit for robust and predictable control over both viscosity and active stresses.

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I. INTRODUCTION

Actomyosin networks are canonical examples of living matter. They generate nonequilibrium active stresses by converting chemical energy from their environment into mechanical work [1–5]. In cells, actomyosin is the major component of the cellular cortex, which is the prime actuator of cell shape changes. Here, we use a naturally occurring surface contraction wave (SCW) in meiotic *Patiria miniata* oocytes as a platform for studying the mechanisms by which actomyosin-driven cell shape changes are enabled.

The mechanisms underlying the contractility of disordered actomyosin networks such as the cortex remain poorly

understood [6–8]. Contractility in disordered actomyosin networks has been shown to depend not solely on myosin activity, but on the architecture [8,9] and density [10,11] of the actin network. Furthermore, a substantial body of work in *in vitro* systems has demonstrated that in many cases F-actin and myosin alone are insufficient for network contractility and that additional actin cross-linking proteins are required [12–16], though an exception has been found at low pH where myosin itself can function as an effective cross-linker [17].

Understanding how the cellular-scale properties of actomyosin networks emerge from the filament-scale interactions of the network's constituents is an open challenge. To generate contractile stresses, the filament-scale symmetry between contraction and expansion must be broken [18]. A number of microscopic models have been proposed for how this symmetry breaking can occur [8,19]. One class of models relies on polarity sorting—Myosin accumulates at actin barbed ends, clustering barbed ends together, which in turn leads to isotropic contraction [20]. Myosin-2 end accumulation has been demonstrated in a purified system [7], and this mechanism has been argued to give rise to contraction in microtubule networks [21–23]. Alternatively, contractility has been proposed to arise from the nonlinear mechanical properties of F-actin, which can buckle under compression.

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In purified systems, F-actin buckling has been seen to coincide with network contraction [16,24]. Finally, contractility independent of myosin motor activity has been proposed for some structures, such as the contractile F-actin shell that captures chromosomes during sea star oocyte meiosis [25–29]. However, directly assessing the degree of myosin end accumulation or filament buckling *in vivo* presents an experimental challenge due to the high density and small size of myosin and actin filaments, which limits the ability to resolve individual motors and filaments using light microscopy.

Here, we consider the actomyosin-driven surface contraction wave of meiotic *Patiria miniata* oocytes as a model for cellular contractility. Using pharmacological inhibitions targeting actin polymerization dynamics, we find that cellular deformation during the contraction wave is not a monotonic function of cortical actin density, but is instead peaked near the wild-type density. To understand the physical origin of this observation, we first developed a physical theory for deforming active surfaces, which shows that the rate of deformation is proportional to the ratio between the active contractile cortical stress and the cortical viscosity. This led us to conclude that our pharmacological treatments directly affect this key parameter. We note that Rho GTPase signaling, which has been shown to be a key determinant of surface contraction wave speed and spatial patterning [30,31], provides the upstream biochemical input that generates the traveling band of active stress in our system and remains largely unaffected between experiments. Our mechanical model takes this traveling active stress as given and asks how the cortical material properties set the resulting cellular deformation. The two descriptions are thus complementary: Signaling determines where and when the cortex contracts, while mechanics determines how much the oocyte deforms. We next sought to understand how controlling actin density can affect the ratio between the active contractile cortical stress and the cortical viscosity.

To relate the values of this key ratio to the molecular composition of the cortex, we utilized a recently developed theoretical framework for dense cytoskeletal networks [32], which generalizes a model developed for microtubule networks [33] to allow for more elaborate motor and cross-linker properties. Based on this, we developed an active fluid model coarse grained from a microscopic description of actin, cross-linkers, and motors. This model makes quantitative predictions for how the rate of oocyte deformation varies with the concentrations of passive cross-linkers and active motor proteins, namely, that the radial deformation rate slightly increases before decreasing as passive cross-linker concentration increases, and, surprisingly, decreases monotonically with increasing active motor concentration. We compared these predictions with experimental measurements from oocytes overexpressing α -actinin or myosin regulatory light chain (MRLC) and found quantitative agreement. Taken together, these results provide a conceptual bridge from filament-level microscopic interactions, to cellular-scale material properties of the cell cortex, and ultimately to the emergent mechanics that enables whole-cell deformations *in vivo*.

While the result that deformation rates scale with the active stress-to-viscosity ratio is consistent with established active gel theory [3], our contributions are as follows: (1) the first *in vivo* demonstration that this ratio controls a natural

stereotyped deformation, (2) explicit closed-form expressions for viscosity and active stress from a microscopic motor-cross-linker model, enabling quantitative predictions, and (3) experimental verification of the counterintuitive prediction that myosin overexpression decreases the deformation rate.

II. RESULTS

A. Surface contraction wave dynamics

As a model process for actomyosin-driven contraction *in vivo*, we here consider the surface contraction wave preceding the first meiotic division in oocytes of the bat star *Patiria miniata* [31,34,35]. First discovered in developing axolotl [36], surface contraction waves are found in a variety of large eggs, including those of the frog *Xenopus laevis* [37], barnacles [38], and ascidians [39]. In sea star oocytes, these waves are driven by a band of activated Rho that travels across the oocyte from the vegetal to animal pole, guided by a spatial gradient of cdk1-cyclinB [31,35,40]. This traveling band of active Rho locally activates several downstream factors, including myosin via the ROCK pathway and the actin nucleator formin mDial [41]. These in turn lead to local contraction, resulting in a traveling surface contraction wave, whose arrival at the animal pole coincides with polar body extrusion [31,34] [Fig. 1(a); Video S1 of the Supplemental Material [42]]. Due to the ease of meiotic induction, the large, spherical shape of sea star oocytes, and the highly conserved nature of the actomyosin components [18], sea star oocytes are a powerful model system for the study of actomyosin contractility *in vivo*. While a surface contraction wave coincides with each meiotic division, we here consider the first surface contraction wave that takes place during meiosis I. Previous pioneering work [31] has uncovered several key determinants of surface contraction waves in this system. Here, we build on this foundation and develop a quantitative framework for bridging from protein-scale interactions to cellular-scale deformation in this system.

We first quantified a characteristic radial deformation rate and the wave propagation speed during the SCW. To quantify the deformation rate, the distance between the oocyte's center and outer contour at each angle and time point, $R(\theta, t)$, was first normalized by the time-averaged radial distance for that angle, $\langle R(\theta) \rangle_t$, and a time derivative was taken to compute the local deformation rate, $d(\theta, t)$ [Fig. 1(b); see the Supplemental Material [42]]. From kymographs of $d(\theta, t)$, the SCW can be readily visualized as a traveling line of negative values [Fig. 1(c)], and the propagation speed of the wave can be measured from the slope of this line (see the Supplemental Material [42]). A characteristic deformation rate was calculated by first taking the magnitude of the minimum deformation rate for each angle, $|d_{\min}(\theta)|$ [Fig. 1(d), solid line], which was then averaged across angles to determine the characteristic deformation rate, $d_c = \langle |d_{\min}(\theta)| \rangle_\theta$ [Fig. 1(d), dashed line; see the Supplemental Material [42]]. Control oocytes were found to have a mean wave propagation speed of $v = 47 \pm 4 \mu\text{m}/\text{min}$ (mean $\pm$ s.e.m., $n = 25$ oocytes), consistent with previous measurements [31], and a characteristic deformation rate of $d_c = 0.017 \pm 0.002 \text{ min}^{-1}$ (mean $\pm$ s.e.m., $n = 25$ oocytes).

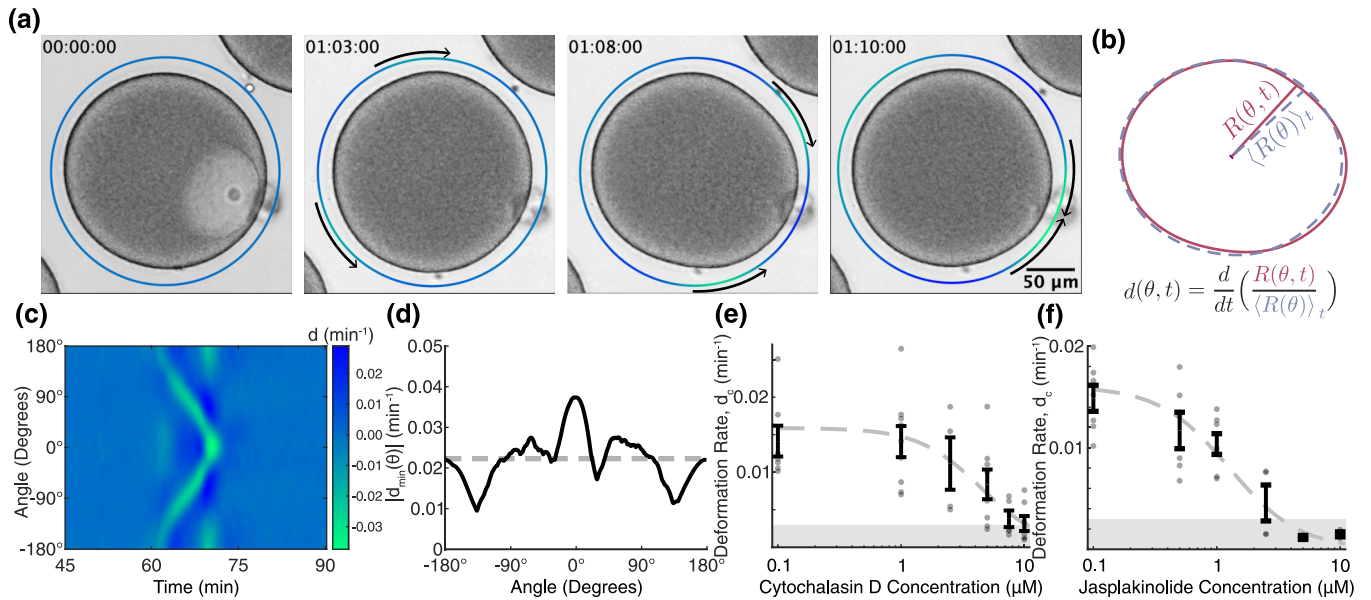


FIG. 1. Surface contraction wave dynamics. (a) Time lapse of oocyte surface contraction wave. The outer circle color denotes $d(\theta, t)$, the radial deformation rate for each angle, colored as in panel (c). Negative (green) values indicate contraction, while positive (dark blue) values indicate expansion. Arrows indicate the direction of wave propagation. (b) Deformation rate calculation. For each angle at each time, the radial distance between the oocyte's centroid and outer contour was normalized by the time-averaged radial distance before a time derivative was taken. (c) Kymograph of $d(\theta, t)$. The surface contraction wave is readily visualized as the converging lines of negative (green) values indicating contraction. (d) Solid line: Magnitude of the minimum deformation rate for each angle, $d_{\min}(\theta)$. Dashed line: the average value of $|d_{\min}(\theta)|$. (e), (f) Average characteristic deformation rate, d_c , as a function of cytochalasin D [panel (e)] and jasplakinolide [panel (f)]. Gray dots: measurements from individual oocytes. Black lines: average deformation rate $\pm$ s.e.m. Gray regions: noise floor, defined as the average deformation rate measured for nonmaturing, germinal vesicle stage oocytes ($n = 9$). Dashed line: IC_{50} fits. Cytochalasin D experiments: $n = 4$ to $n = 9$ oocytes per treatment condition. Data were collected over 9 days using oocytes from six clutches. Jasplakinolide experiments: $n = 2$ to $n = 7$ oocytes per treatment condition. Data were collected over 6 days using oocytes from four clutches.

B. Characteristic deformation rate is maximum at intermediate cortical actin density

We next investigated how modulating actin density through perturbing actin turnover influences deformation dynamics during the SCW. We first considered the actin polymerization inhibitor cytochalasin D, which at high concentrations has been shown to inhibit deformation during the SCW [31]. Measurements of the characteristic deformation rate, d_c , and the wave speed, v , were repeated for oocytes treated with varying concentrations of cytochalasin D. As expected for an actomyosin-driven process, the characteristic deformation rate was found to monotonically decrease with increasing cytochalasin D concentration [Fig. 1(e)]. Fitting a dose-response curve yielded an IC_{50} of $4.4 \pm 2.0 \mu\text{M}$ (fit value $\pm$ 95% confidence interval; see the Supplemental Material [42]). For concentrations of cytochalasin D $\leq 5 \mu\text{M}$, where deformation during the SCW was large enough for the wave speed to be measured, no significant differences in wave speed were found between treatment conditions (Fig. S1 of the Supplemental Material [42]), consistent with previous results arguing that the speed of the SCW is set by the spatiotemporal dynamics of cdk1-cyclinB [31,35].

We next considered the effects of jasplakinolide, which induces actin polymerization and stabilization [43]. We found a dose-dependent decrease in the characteristic deformation rate [Fig. 1(f)], consistent with previous results where actin stabi-

lization with phalloidin suppresses the deformation wave [31]. Fitting a dose-response curve yielded an IC_{50} of $1.3 \pm 0.8 \mu\text{M}$ (fit value $\pm$ 95% confidence interval; see the Supplemental Material [42]). Thus, treatments with drugs that alter actin polymerization reveal that the characteristic deformation rate is maximized at intermediate actin densities.

To quantitatively assess how these perturbations modulate actin density, we next overexpressed and imaged LifeAct-mCherry and used its fluorescence intensity as a proxy for F-actin density (see the Supplemental Material [42]). As expected, LifeAct-mCherry localized to the oocyte's periphery and to the nuclear region shortly after the onset of nuclear envelope breakdown, [Fig. 2(a)], consistent with F-actin's role in nuclear envelope breakdown in sea star oocytes [44,45]. As time progresses, the cortical LifeAct-mCherry signal globally decreases before locally increasing slightly during the surface contraction wave [Fig. 2(a); see Video S2 of the Supplemental Material [42]].

To characterize the cortical actin density, line scans of LifeAct fluorescence were measured midway between the animal and vegetal poles when the SCW passed through this region, and the intensity profiles interior to the cell were fit to a decaying exponential function $I(r) = I_0 e^{-r/\lambda} + I_C$, where r is the distance from the cell's edge. The relative cortical density (RCD) was then calculated from these fitting parameters as $RCD = I_0 / (I_C - I_{BG})$, where I_{BG} is the average fluorescence signal exterior to the cell (see the Supplemental Material

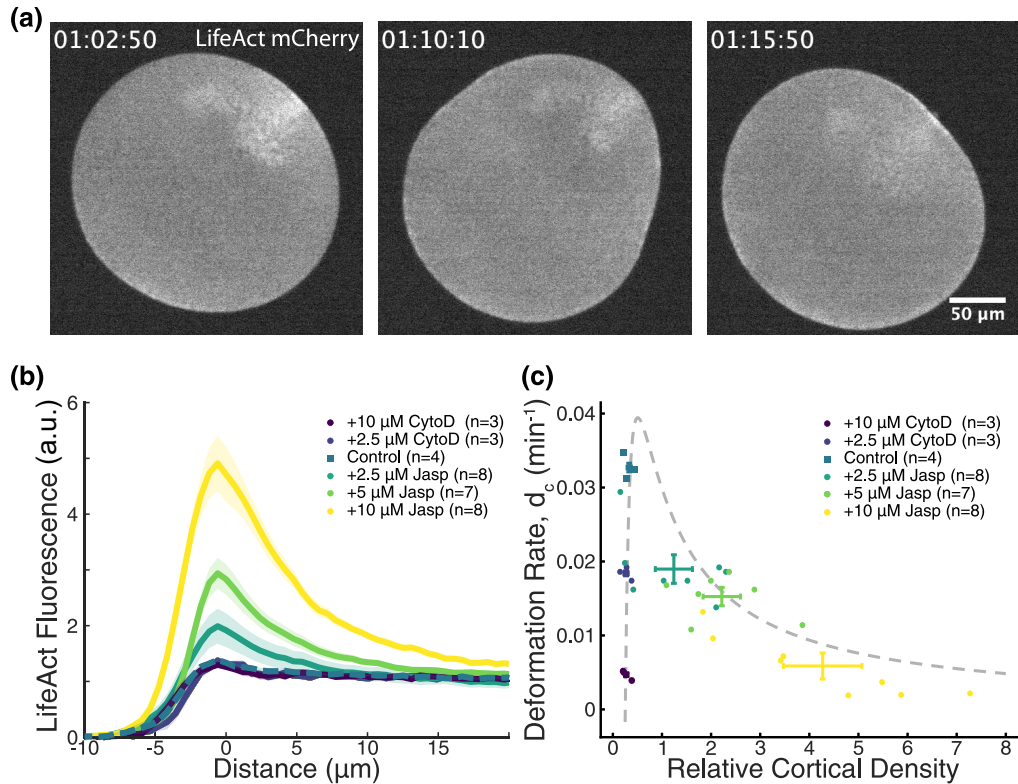


FIG. 2. Characteristic deformation rate is maximized at intermediate actin density. (a) LifeAct-mCherry imaging of F-actin localization during maturation. (b) Average normalized radial line profiles of LifeAct-mCherry fluorescence for varying cytochalasin D and jasplakinolide concentrations (mean $\pm$ s.e.m.). n : number of oocytes per treatment condition. For each oocyte, two fluorescence line scans are taken and included in the average. (c) Characteristic deformation rate, d_c , as a function of relative cortical density. Error bars: mean $\pm$ s.e.m. for each treatment condition. Gray dashed line: model fit (Bayesian $R^2 = 0.67 \pm 0.03$; see the “Bayesian parameter inference and goodness of fit” section of the Supplemental Material [42]). n : number of oocytes per treatment condition. Data were collected over 7 days using oocytes from seven clutches.

[42]). A similar exponential decay was seen when utrophin-mEGFP was used to visualize the cortical actin network (Fig. S2 of the Supplemental Material [42]). Measurements of the relative cortical density and characteristic radial deformation rate were performed for individual oocytes treated with varying concentrations of cytochalasin D or jasplakinolide, allowing a direct comparison between the effects of these two treatments [Fig. 2(b); Fig. S3 of the Supplemental Material [42]]. While the RCD for cytochalasin-treated oocytes is decreased relative to wild-type conditions ($\text{RCD} = 0.27 \pm 0.07$ and 0.27 ± 0.06 for 2.5 and 10 μM cytochalasin D, respectively, compared to $\text{RCD} = 0.33 \pm 0.06$ for wild type (WT), mean $\pm$ s.e.m.), differences in the RCD between the two cytochalasin conditions cannot be resolved, presumably due to the low absolute RCD for these conditions. As anticipated from the measured dose-response curves [Figs. 1(e) and 1(f)], we find that the characteristic deformation rate is not a monotonic function of cortical actin density, but instead sharply increases before slowly decreasing with increasing cortical actin density, with a peak near the wild-type density [Fig. 2(c)]. We note that the characteristic deformation rate for control cells, $0.033 \pm 0.001 \text{ min}^{-1}$ (mean $\pm$ s.e.m., $n = 4$ oocytes), is approximately twofold larger than what we saw previously. We suspect this difference arises from environ-

mental differences between the microscopy facilities used for these experiments.

Previous work has shown that on short timescales, treatment with agents targeting actin polymerization can feed back on the cortical localization of active Rho [30]. As the SCW in this system is driven by Rho activity, if treatment with either cytochalasin D or jasplakinolide decreases the enrichment of active Rho during the wave, then decreased Rho enrichment could explain the decrease in deformation rate seen for these treatments. To test this hypothesis, we overexpressed rGBD-GFP in oocytes as a proxy for active Rho and treated oocytes with either 10 μM cytochalasin D or 10 μM jasplakinolide before maturation. As expected, rGBD-GFP was found to enrich in the cortex. To quantify the enrichment of rGBD-GFP, line scans of fluorescence intensity were collected and analyzed similarly to LifeAct fluorescence, and a Relative cortical enrichment was calculated. rGBD-GFP was found to enrich to a similar degree for control and cytochalasin-D-treated oocytes and to a significantly greater degree for jasplakinolide-treated oocytes (Fig. S4 of the Supplemental Material [42]). Thus, the decrease in contraction rate seen for these treatments, and hence the peak in contraction rate as a function of relative cortical density, cannot be attributed to a decrease in the enrichment of active Rho.

C. Modeling the cell cortex as a thin active viscous shell links deformation rates to material properties

To understand the origin of the observed dependence of the radial deformation rate on actin density, we first asked how the physical properties of the cell cortex changed throughout the perturbation experiments. The force balance of the cortical material in bulk reads

$$\nabla_j(\eta_{ijkl} \nabla_k \mathbf{v}_l + \Sigma^A \delta_{ij} + \mathcal{T}_{ij}) = 0, \quad (1)$$

where η_{ijkl} is the viscosity tensor where the nonzero elements take the form $\eta_{ijkl} = \eta \xi_{ijkl}$, where η is the magnitude of the dominant component of the viscosity and ξ_{ijkl} encodes geometric information and scalings, Σ^A is the active stress generated by interactions between actin filaments, molecular motors, and passive cross-linkers, and $\mathcal{T}$ is a passive stress arising from surface tension. Einstein's convention of summation over repeated indices is implied.

To identify how the material properties of the cell cortex change, we developed a theory for a deformable cell by mapping Eq. (1) onto a thin shell of material (see the Supplemental Material [42]), similarly to Refs. [46–49], and used the equations of motion that we thus obtained to infer the ratio between active stresses and viscosities from experimental data. We treat the cortex as a purely viscous, rather than viscoelastic, material: Cortical actin turns over on a timescale of tens of seconds [50,51], which is short compared to the timescales over which the SCW deforms the cortex (the active-stress band sweeps past a given point over a few minutes, and the full normalized deformation accumulates over $\sim 1/d_c$, i.e., tens of minutes). Cortical elastic stresses thus relax faster than the cortex deforms, justifying the viscous limit (see the Supplemental Material [42]). Our procedure starts by first extracting the shape $R(\theta, t)$ and its normal (V^z) and tangential (V^s) surface velocities from each movie [Fig. 3(a); see Secs. 1 and 2 of the Supplemental Material for details [42]]. These quantities are then inserted into the thin-shell equations of motion to determine the amplitude $||\Sigma^A/\eta||$. The results are shown in Fig. 3(b) (top panels) and demonstrate that the maximum value of the stress-viscosity ratio is highest for wild-type oocytes and decreases for both cytochalasin and jasplakinolide treatments. This mirrors the behavior of the characteristic deformation rate d_c itself [Fig. 2(c)].

To further test this behavior, we next solved for the shape changes expected in a cell with a given stress-viscosity ratio as a function of time. While the surface contraction wave has been shown to be driven by an active band of Rho [31], we here focus on the mechanics of deformation and implicitly include the signaling dynamics of Rho itself by choosing the width of the active Rho band and its traveling speed to take values consistent with experimental measurements (see Table S1 of the Supplemental Material [42]). In Fig. 3(c) (bottom panels), we report the deformation rates from these numerical experiments. We conclude that generically the deformation rate varies almost linearly with the stress-viscosity ratio, as expected [46] [Fig. 3(e)], and thus that our pharmacological treatments altered the stress-viscosity ratio of the cell cortex. We next sought to understand the filament-level origins of this cortical scale effect.

D. An active fluid model coarse grained from microscopic interactions

To obtain predictions for the dependence of η and Σ^A on the actin density, we adapted a recently developed theoretical framework that allows a microscopic description of the system to be coarse grained into an emergent mechanical model [32]. An application of this general framework to the actomyosin system considered here begins with a simplified microscopic description of the system and considers three elements: actin filaments, passive cross-linkers, and molecular motors.

Molecular motors and passive cross-linkers exert forces between the filaments that they connect. For passive cross-linkers, these forces are taken to be proportional to the velocity difference between the points on the actin filament connected by the cross-linker. Thus, for a single cross-linker bound between position s_i on the i th actin filament and s_j on the j th filament, the force exerted between filaments is given by

$$\mathbf{f}_{ij}^X = -\gamma^X \hat{\mathbf{w}} \hat{\mathbf{w}} \cdot (\mathbf{v}_i + s_i \dot{\mathbf{p}}_i - \mathbf{v}_j - s_j \dot{\mathbf{p}}_j), \quad (2)$$

where $\mathbf{v}_i$ and $\mathbf{p}_i$ are, respectively, the velocity and direction of filament i , and γ^X describes the coupling strength [see Fig. 4(a)]. Here, s_i is the arc-length coordinate along filament i , running over $s_i \in [-L/2, L/2]$ for a filament of length L , with $s_i = L/2$ denoting the plus (barbed) end. The unit vector $\hat{\mathbf{w}}$ points from the binding point of the cross-link on filament j to that on filament i and is defined as $\hat{\mathbf{w}} = \mathbf{w}/||\mathbf{w}||$, with $\mathbf{w} = \mathbf{x}_i + s_i \mathbf{p}_i - \mathbf{x}_j - s_j \mathbf{p}_j$. When multiple passive cross-linkers are bound between filament pairs, the net force exerted between filaments becomes

$$\mathbf{f}_{ij}^X = -\gamma^X c^X(s_i, s_j) \hat{\mathbf{w}} \hat{\mathbf{w}} \cdot (\mathbf{v}_i + s_i \dot{\mathbf{p}}_i - \mathbf{v}_j - s_j \dot{\mathbf{p}}_j), \quad (3)$$

where $c^X(s_i, s_j)$ is the number of passive cross-linkers that are bound between positions s_i and s_j , which can spatially vary along filaments. Motor molecules likewise contribute to the frictional coupling between filaments, but, due to their stepping motion along filaments, exert additional active forces. The net force exerted by motors between filaments $\mathbf{f}_{ij}^M$ can be written as

$$\mathbf{f}_{ij}^M = -\gamma^M c^M(s_i, s_j) \hat{\mathbf{w}} \hat{\mathbf{w}} \cdot (\mathbf{v}_i + s_i \dot{\mathbf{p}}_i - \mathbf{v}_j - s_j \dot{\mathbf{p}}_j + V_{||}(\mathbf{p}_i - \mathbf{p}_j)), \quad (4)$$

where the coefficient $V_{||}$ is the unloaded speed of the motor, γ^M is the motor friction, and $c^M(s_i, s_j)$ is the density of motor molecules connecting two specific filament positions. We further postulate functional forms for the motor and cross-linker densities,

$$\begin{aligned} c^M(s_i, s_j) &= C_0^M + (s_i + s_j) C_1^M, \\ c^X(s_i, s_j) &= C_0^X + (s_i + s_j) C_1^X, \end{aligned} \quad (5)$$

where C_0^M , C_0^X represent the number of uniformly bound motors and cross-linkers, and C_1^M and C_1^X capture nonuniformity of binding along filaments. Given Eqs. (3)–(5), predictions for η and Σ^A can be derived by integrating over all possible configurations of motors and cross-linkers [32] [Fig. 4(b); see the Supplemental Material [42]]. The viscosity of the system

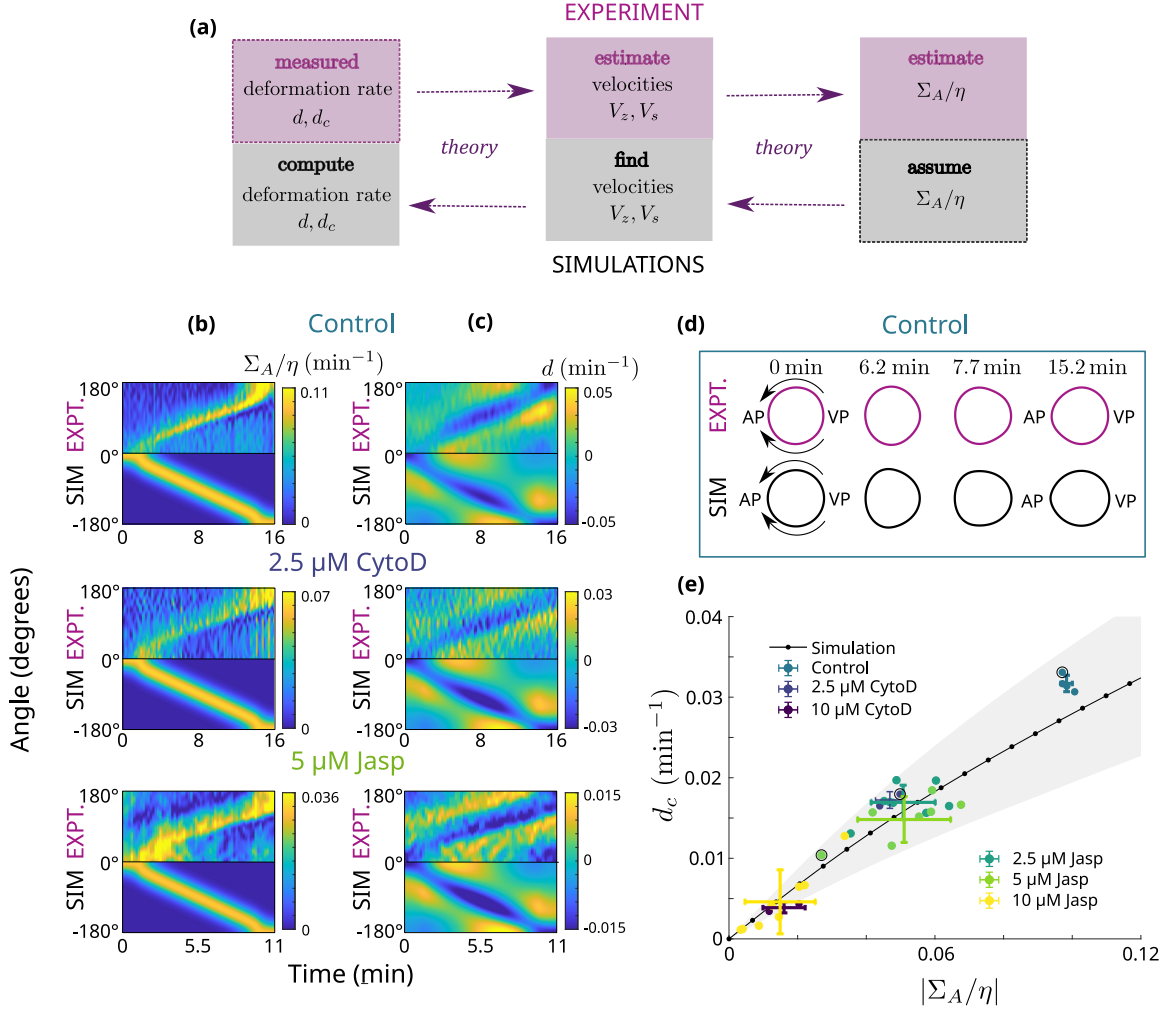


FIG. 3. Using the active surface model to quantitatively understand experiments. (a) Description of steps followed to extract the active stress/viscosity ratio Σ^A/η and the deformation rate d from experiments and simulations, respectively, within our active surface model theory, discussed in detail in the Supplemental Material [42]. (b) Comparison of the estimated experimental and assumed simulation active stress Σ^A/η kymograph. (c) Comparison of the measured experimental and the computed simulation deformation rate d kymograph for different cases. The corresponding active stresses are shown in panel (b). (d) Exemplary experimental and simulation oocyte shapes for different points in time corresponding to the control case. (e) Plot of the characteristic deformation rate d_c against the characteristic amplitude of active stress $|\Sigma^A/\eta|$ obtained in a similar way as d_c is derived from d . Shown are both the simulation values and data points of representative experiments. The vertical errors for simulations (gray area) stand for the standard deviation of the minimum values of d in time. The vertical and horizontal errors for the experimental data points stand for the standard deviation in d_c and $|\Sigma^A/\eta|$ for different datasets for each color-coded drug treatment. The experimental points surrounded by the black circle are the ones used for comparison of simulations and experiment in panels (b) and (c). The parameter values used for the simulation are shown in Table 1 of the Supplemental Material [42].

is predicted to be

$$\eta \propto \rho^2 (\gamma^X C_0^X + \gamma^M C_0^M). \quad (6)$$

Furthermore, the active stress is predicted to be

$$\Sigma^A \propto \rho^2 \gamma^M V_{||} \left(C_0^M \frac{\gamma^M C_1^M + \gamma^X C_1^X}{\gamma^M C_0^M + \gamma^X C_0^X} - C_1^M \right). \quad (7)$$

With this, the contraction rate is expected to be

$$d = \frac{\Sigma^A}{\eta} = \gamma^M V_{||} \frac{C_0^M \frac{\gamma^M C_1^M + \gamma^X C_1^X}{\gamma^M C_0^M + \gamma^X C_0^X} - C_1^M}{\gamma^M C_0^M + \gamma^X C_0^X}. \quad (8)$$

To relate these results to our experimental findings, we next need to specify how C^M and C^X change as a function of actin

density ρ . The average number of passive cross-links and motor molecules bound between two filaments can depend on the overall actin density. We illustrate this by discussing two cross-links A and B with different behaviors. Assume that the total cortical concentration of A is independent of actin filament numbers and low enough such that the binding rate of A is limited by the total concentration of A itself. As the number of filaments in the cortex is increased, we expect the number of A per filament pair to drop roughly inversely proportional to the number of actin filaments. In contrast, if cross-link B is so abundant that its binding is limited by the number of available binding sites, we expect a different behavior, where the number of B bound depends on the total number of binding sites available per filament, which is

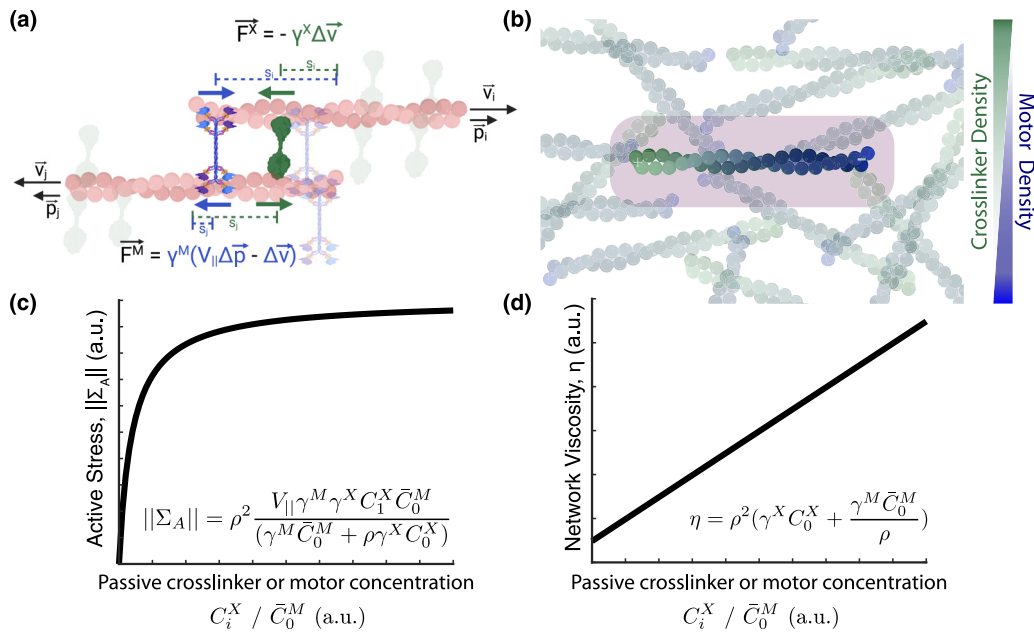


FIG. 4. Active fluid model. (a) Schematic of the filament-scale forces acting between a pair of antiparallel actin filaments, generated by individual motors (blue) and passive cross-linkers (green). (b) Microscopic model used in the main text. Motors (blue) are distributed uniformly along the filaments ($C_1^M = 0$), whereas passive cross-linkers (green) are enriched toward the filament end toward which the motor walks. This asymmetric cross-linker localization breaks the filament-scale symmetry between contraction and expansion and is what produces a net contractile active stress. Predicted functional forms for how the active-stress magnitude $\|\Sigma^A\|$ [panel (c)] and the viscosity η [panel (d)] scale with the concentration of either passive cross-linkers (C_i^X) or motors ($\bar{C}_0^M$).

independent of the total number of filaments. While the binding of myosin and passive cross-links in the cortex is likely much more complex, the key point that the average number of each type of cross-link per filament pair depends on actin density differently holds irrespectively. Importantly, since the concentrations of motors and passive cross-links per filament pair are functions of the total actin amount, d is a function of ρ as well. To capture this, we take $C_0^M = c_0^M (\rho - \rho_0)^{\mu^M}$, $C_1^M = c_1^M (\rho - \rho_0)^{\mu^M}$ and $C_0^X = c_0^X (\rho - \rho_0)^{\mu^X}$, $C_1^X = c_1^X (\rho - \rho_0)^{\mu^X}$. For the case adopted below, $\mu^M = -1$ and $\mu^X = 0$, it is convenient to write the motor amplitude as $\bar{C}_0^M = \rho C_0^M$, as we do in Fig. 4, where ρ_0 is suppressed for clarity. With this, Eq. (8) becomes

$$d = V_{||} \gamma^M \gamma^X \frac{(\rho - \rho_0)^{\mu^M + \mu^X} (c_0^M c_1^X - c_0^X c_1^M)}{(c_0^M \gamma^M (\rho - \rho_0)^{\mu^M} + c_0^X \gamma^X (\rho - \rho_0)^{\mu^X})^2}. \quad (9)$$

Taking $|\mu^M - \mu^X| = 1$, which we show below to be the relation selected by our data, we obtain

$$d = d_{\max} \frac{4\bar{\rho}}{(1 + \bar{\rho})^2}, \quad \text{where} \quad \bar{\rho} = \frac{\rho - \rho_0}{\rho_{\max} - \rho_0}, \quad (10)$$

with

$$d_{\max} = V_{||} \frac{(c_0^M c_1^X - c_0^X c_1^M)}{4c_0^M c_0^X}, \quad (11)$$

$$\rho_{\max} = \begin{cases} \frac{c_0^X \gamma^X}{c_0^M \gamma^M} + \rho_0, & \text{if } \mu^M - \mu^X = 1, \\ \frac{c_0^M \gamma^M}{c_0^X \gamma^X} + \rho_0, & \text{if } \mu^M - \mu^X = -1. \end{cases} \quad (12)$$

Importantly, the form of Eq. (10) is independent of microscopic assumptions. It is parametrized by the three fit parameters $d_{\max}$, $\rho_{\max}$, and ρ_0 . The first fit parameter $d_{\max}$ has

units of contraction rate (min^{-1}). It determines the height of the peak of the density contraction curve. The second, $\rho_{\max}$, has dimensions of relative density and sets the position of the maximum. Finally, ρ_0 sets the zero intercept of the curve and could be interpreted as the percolation threshold below which the network is too sparse to be fully connected by motors and cross-links, and thus cannot contract. The sign of $d_{\max}$ determines whether the model cortex is contractile ($d_{\max} > 0$) or extensile ($d_{\max} < 0$). In our experiments, $d_c > 0$, and thus only microscopic models with $d_{\max} > 0$ are consistent with the data. Thus, our data could be explained by a microscopic model in which (1) passive cross-linkers accumulate near the end of the filament that motors walk toward, $V_{||} c_1^X > 0$; (2) the motor accumulates near the filament end that it walks away from, $V_{||} c_1^M < 0$; or (3) a combination of (1) and (2). For example, scenario (1) would be consistent with a cross-linker that tracks growing filament ends, for example, a formin, some of which have been shown *in vitro* to be capable of cross-linking actin filaments [52]. Scenario (2) would be consistent with actin filament plus ends growing at a faster speed than motors walk, leading to an effective depletion of motors at the fast growing end. This effect has been previously recognized to promote network contraction in an *in vitro* microtubule/motor system [53]. In any case, we stress that these two scenarios lead to equivalent predictions for the characteristic deformation rate considered here.

We next asked for which parameter combinations Eq. (10) was quantitatively consistent with the observed deformation rate changes. Fitting Eq. (10) to the average characteristic radial deformation rate and relative cortical actin density for each treatment condition reproduces the measured data well [Fig. 2(c), Bayesian $R^2 = 0.67 \pm 0.03$]. The

underlying model parameters that best describe the data are $d_{\max} = 0.04 \pm 0.002 \text{ min}^{-1}$, $\rho_{\max} = 0.51 \pm 0.02$, and $\rho_0 = 0.25 \pm 0.002$ (fit values $\pm$ standard deviation). All active-fluid-model parameters throughout this work are inferred within a Bayesian framework, and we report goodness of fit as a Bayesian coefficient of determination R^2 ; the inference procedure, full posterior distributions, and the definition of the Bayesian R^2 are given in the “Bayesian parameter inference and goodness of fit” section of the Supplemental Material [42]. Note that models with $|\mu^M - \mu^X| \neq 1$ failed to produce convincing fits.

E. Testing and parametrizing the active fluid model through protein inhibition and overexpression experiments

In the active fluid model, forces are generated by the activity of molecular motors. To test this assumption and confirm that myosin activity underlies deformation during the SCW, oocytes were treated with the myosin inhibitor blebbistatin, which has previously been shown to almost completely suppress contraction during the SCW [31]. We find that treatment with 200 μM blebbistatin substantially decreases the characteristic deformation rate, consistent with myosin’s role in driving contraction, while treatment with an inactive blebbistatin enantiomer did not significantly change the deformation rate (Fig. S5 of the Supplemental Material [42]).

To further test the active fluid model, we performed experiments by increasing the concentration of either a passive actin cross-linking protein or an active motor. In the active fluid model, the viscosity η and the active-stress magnitude $||\Sigma^A||$ are predicted to scale differently when the concentrations of either passive cross-linker or active motors are varied [Figs. 4(c) and 4(d); Eqs. (6) and (7)]. The concentrations of passive cross-linkers and motor proteins enter the characteristic deformation rate by modulating $\rho_{\max}$, i.e., the actin density at which contraction rates are maximized [see Eq. (12)].

Upon modulating the amount of passive cross-linker and motor, we expect $\rho_{\max}$ to depend on the fractional changes f^X, f^M of the passive cross-linker and motor concentrations, respectively, as

$$\rho_{\max} = \begin{cases} \rho_{\max}^* \frac{1+f^X}{1+f^M} - \rho_0 \frac{f^X - f^M}{1+f^M}, & \text{if } \mu^M - \mu^X = 1, \\ \rho_{\max}^* \frac{1+f^M}{1+f^X} - \rho_0 \frac{f^M - f^X}{1+f^X}, & \text{if } \mu^M - \mu^X = -1, \end{cases} \quad (13)$$

where $\rho_{\max}^* = 0.51 \pm 0.02$ denotes the value of $\rho_{\max}$ for the case of unperturbed concentrations. Further, the density ρ_0 , which is related to the cross-link density at which the actin network becomes fully percolated [15], is independent of the densities of cross-links and motors. In what follows, we will present our fits assuming that $\mu^M - \mu^X = -1$, i.e., that the number of motors per filament pair decreases as more actin becomes available, while the number of passive cross-linkers per filament pair stays constant. This is consistent, for example, with a scenario in which motor abundance is limited by the cytoplasmic motor concentration, while cross-linker abundance is limited by the number of available binding sites. We do, however, note that the opposite choice, $\mu^M - \mu^X = 1$, provides equally good fits, and our data do not *a priori* allow

distinguishing between the two scenarios (see the Supplemental Material [42]).

In unperturbed oocytes, the cortical density is close to $\rho_{\max}^*$ [see Fig. 2(c)]. Thus, according to our theory, they operate near the maximum contraction rate that the system can achieve. This implies a testable prediction: The contraction rates should go down as passive cross-linkers are overexpressed and, more surprisingly, that contractions should also decrease when the motor myosin is overexpressed [see Eqs. (10) and (13)].

To test these predictions, we first overexpressed an actin cross-linking protein in untreated oocytes. While a variety of passive actin cross-linkers localize to the cortex [54], we here use α -actinin as a model passive cross-linker. Fluorescent mEGFP-labeled *Patiria miniata* α -actinin [44] was overexpressed by injecting oocytes with the corresponding mRNA (see the Supplemental Material [42]), and we make use of the natural variability in protein expression level to assess changes in the characteristic radial deformation rate over a range of α -actinin concentrations.

Characteristic deformation rates and the fluorescence signals of α -actinin-mEGFP, which we use as a proxy for α -actinin concentration, were measured for individual oocytes [Fig. 5(a); Video S3 of the Supplemental Material [42]]. Overall, we find a general trend of decreasing deformation rate with increasing levels of α -actinin overexpression, qualitatively consistent with the prediction of the active fluid model. To quantitatively test the model, we take the actin density to be the previously measured wild-type value, $\rho = \rho_{\text{WT}}$, and relate the measured α -actinin-mEGFP fluorescence signal to the fractional change in passive cross-linker concentration as $f^X = \alpha_X I_X$. Using the values for $\bar{\rho}$, ρ_0 , and $d_{\max}$ determined in Fig. 2(c), we fit to the experimental data and found quantitative agreement between theory and experiment, providing a measure of the sole fit parameter, $\alpha_X = 11.9 \pm 5.1$ (fit value $\pm$ 95% credible interval). Thus, our active fluid model is consistent with the results of the α -actinin overexpression experiments.

The active fluid model further predicts how the characteristic deformation rate should change with the concentration of active motors. MRLC overexpression in *Patiria miniata* oocytes has previously been shown to increase the strength of the SCW [28,31] and to increase nonequilibrium activity in the cortex [55]. In the active fluid model presented here, motors contribute both active forces and an effective friction between sliding filaments. As such, changes in the concentration of motor proteins are predicted to change both the emergent active stress and network viscosity. As motor concentration increases, the model predicts that network viscosity will grow faster than active stress, and hence the rate of deformation will decrease with increasing motor concentration [Figs. 4(c) and 4(d)].

To experimentally test this prediction, mEGFP-labeled *Patiria miniata* myosin regulatory light chain was overexpressed in oocytes [Fig. 5(c); Video S4 of the Supplemental Material [42]]. The characteristic deformation rate and MRLC-mEGFP fluorescence were measured for individual oocytes (see the Supplemental Material [42]) and the resulting data were fit to Eqs. (10) and (13), under the assumption that the measured MRLC-mEGFP fluorescence signal is

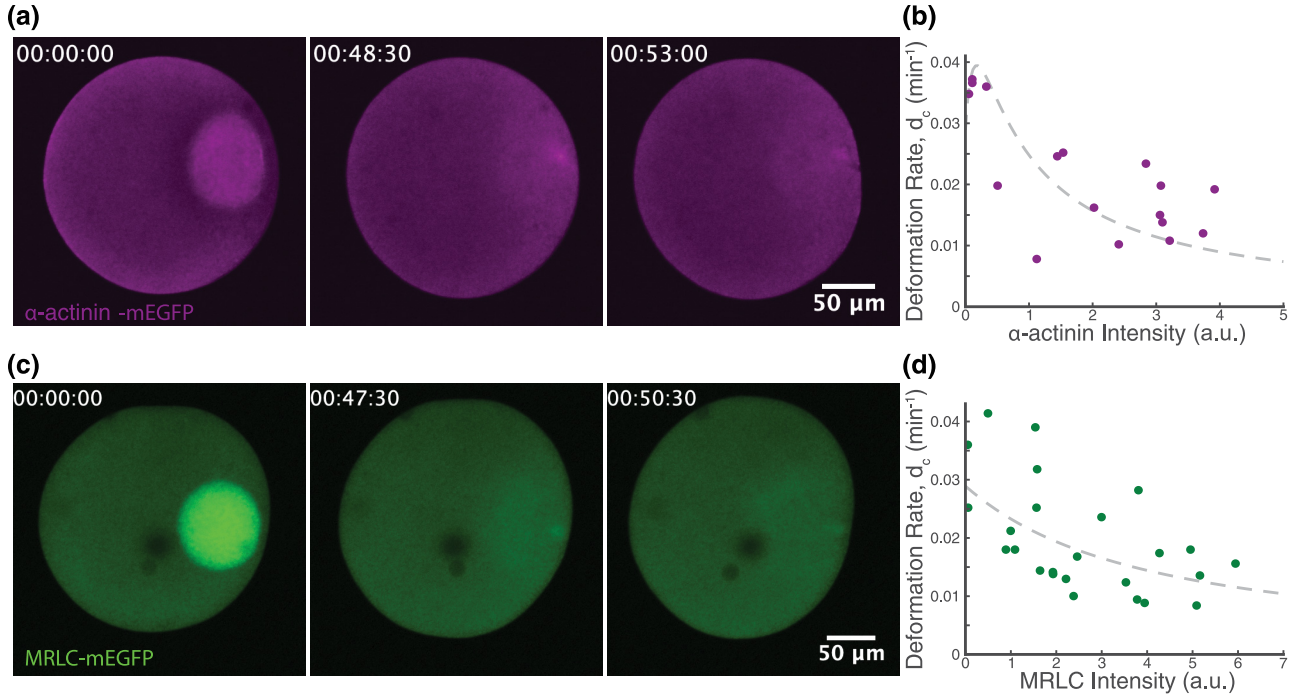


FIG. 5. Cross-linker and motor overexpression to test active fluid model predictions. (a) Time course of SCW for an oocyte overexpressing α -actinin-mEGFP. (b) Characteristic deformation rate as a function of α -actinin-mEGFP intensity ($n = 17$ oocytes). Data were collected over 3 days using oocytes from three clutches. Gray dashed line: model fit (Bayesian $R^2 = 0.70 \pm 0.03$). (c) Time course of SCW for an oocyte overexpressing MRLC-mEGFP. (d) Characteristic deformation rate as a function of MRLC-mEGFP intensity ($n = 25$ oocytes). Data were collected over 4 days using oocytes from four clutches. Gray dashed line: model fit (Bayesian $R^2 = 0.28 \pm 0.05$).

proportional to the fractional change in active motor concentration, $f^M = \alpha_M I_M$. Once again, using only a single free fitting parameter, α_M , we find that the measured data follow the trend predicted by the active fluid model [Fig. 5(d)], providing a measure of $\alpha_M = 0.45 \pm 0.25$ (fit value $\pm 95\%$ credible interval). This dataset is the noisiest of the three, and the fit accounts for a correspondingly smaller fraction of its variance (Bayesian $R^2 = 0.28 \pm 0.05$), while still constraining α_M tightly (see the Supplemental Material [42]). We conclude that our model is consistent with the observed effects of myosin overexpression. To demonstrate this, we plot all of our data in terms of $\bar{\rho}$ and demonstrate that two types of overexpression experiments, as well as our pharmacological perturbations and the unperturbed data, fall onto a single master curve predicted by Eq. (10) (Fig. 6).

III. DISCUSSION

Here, we used surface contraction waves in maturing sea star oocytes as a model to study actomyosin contractility *in vivo*. By controlling cortical actin density, we find that the deformation rate is maximum near the wild-type density and decreases when the cortical actin density is either increased or decreased. To understand this phenomenon, we first analyzed the deformations in terms of a deformable active surface model for the oocyte and found that the observed deformation rates indicate that the ratio of cortical contractile stress to cortical viscosity was actively modulated by actin density. To better understand how this could be happening, we developed an active fluid model coarse grained from a microscopic de-

scription of the system. The model predicts the dependence of the radial deformation rate on the relative abundance of passive cross-linkers and motors per filament pair, and shows that contraction rates are maximized for intermediate amounts of passive cross-links. As the abundances of passive and

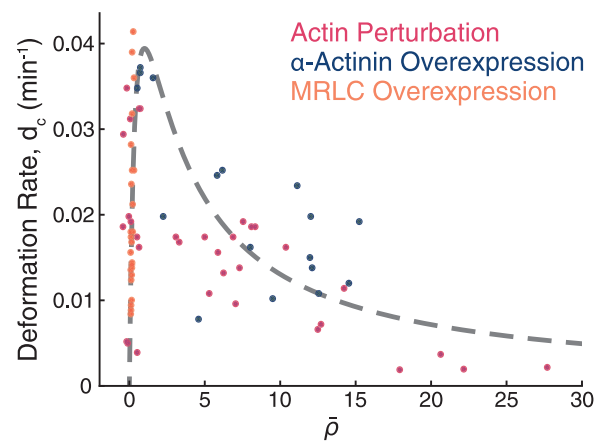


FIG. 6. Using the fit parameters, data from experiments with varying relative cortical density [Fig. 2(c)], α -actinin-mEGFP concentration [Fig. 5(b)], and MRLC-mEGFP concentration [Fig. 5(d)] can be reparametrized and plotted as a function of $\bar{\rho}$. Upon replotting, the data collapse to a curve predicted by the active fluid model (Bayesian $R^2 = 0.49 \pm 0.003$ for the pooled data; see the “Bayesian parameter inference and goodness of fit” section of the Supplemental Material [42]).

motorized cross-links per filament pair are functions of the actin density itself, we further predict that this implies that the maximal contraction rate is reached at intermediate cortical densities following the functional form given by Eq. (10), which is in good quantitative agreement with our data. The theory that we obtain has only three free parameters, which are the maximum contraction rate $d_{\max}$, the actin density at which this rate is reached $\rho_{\max}$, and the minimal density ρ_0 below which the cortex cannot contract—presumably because it is no longer fully percolated. Strikingly, $d_{\max}$ is fully set by the asymmetry of the motor and cross-link binding, and motor speed, but is independent of absolute motor concentrations (a consequence of the equal density-scaling exponents we adopt for C_0 and C_1 , rather than a model-independent feature). In contrast, $\rho_{\max}$ depends crucially on concentrations, but is independent of detailed motor properties. This highlights the role that actin density itself plays for controlling cortical contractility. Because actin density sets both the active stress and the viscosity, but with different density dependence [Eqs. (6) and (7)], the deformation rate $d = \Sigma^A/\eta$ is a nonmonotonic function of density that peaks at $\rho_{\max}$, so that increasing the density beyond $\rho_{\max}$ lowers the deformation rate. This is a self-limiting, mechanically homeostatic mechanism rather than a biochemical feedback loop: The regulated quantity is the stress-to-viscosity ratio, and hence the deformation rate d , and the regulating process is the density-dependent comodulation of active stress and viscosity, which requires no additional biochemical feedback. By using model parameters measured from fitting the active fluid model to the actin density, α -actinin overexpression, and MRLC overexpression data, data from all three sets of experiments could be reparametrized and plotted as a function of the same parameter, leading to data collapse to a curve predicted by the active fluid model.

Having established quantitative agreement between the active fluid model and the data, we now discuss the microscopic picture that underlies this model. The key microscopic ingredient is a passive cross-linker that clusters near the filament end toward which the motors walk, together with a uniformly distributed motor whose per-filament-pair concentration scales inversely with actin density, in line with the exponent relation $|\mu^M - \mu^X| = 1$ selected by our fits. We stress that other models are also consistent with the data presented here. These include models where passive cross-linkers are uniformly distributed on filaments, while myosin accumulates toward the end it walks away from, and a combination of these models where myosin and passive cross-linkers accumulate at opposite ends. Our key advance is to identify the unifying features of all allowed microscopic models, thus identifying the control knobs available to cells during development. The key features are (1) microscopically broken symmetry on the filament-scale, which requires an asymmetry either in the driving force (due to the spatial localization of motors) or in the frictional coupling (due to the spatial localization of passive cross-linkers), and (2) the balance between per filament motoring and friction. Importantly, these two key aspects of the microscale physics can be independently regulated, as is demonstrated by their very different dependence on actin density. Beyond actin density, cross-linker turnover has also been shown to be a mechanism for regulating active stresses [56]. We speculate that it acts via the same two control

knobs. The filament-scale asymmetry we invoke is not *ad hoc*. A recent thermodynamic theory of cytoskeletal cross-linking [57] derives the active and frictional force coefficients, together with the along-filament patterning of cross-linkers, from measurable cross-linker properties and their chemical potentials, showing that such broken symmetry can in principle arise from binding thermodynamics rather than being imposed by hand. Crucially, our earlier coarse graining of actively cross-linked filament networks [32,33] shows that only the lowest moments of the motor and cross-linker distributions along filaments, their mean and first moment, enter the large-scale active stress and viscosity, while finer-scale details of these distributions contribute only higher-order terms to the stress tensor. This is why we need not invoke more elaborate spatial distributions of motors and cross-linkers, and why the minimal closure we adopt for the filament-scale forces is complete, within this family of linear, pairwise force laws, at the level of the large-scale mechanics. That framework describes the forces between individual filament pairs. It could be extended to yield how the per-filament-pair motor and cross-linker numbers depend on the cortical actin density, $C(\rho)$; closing this loop for a cortex would require a binding model with many more parameters than cell-scale measurements can constrain. We therefore deliberately adopt a phenomenological closure formulated at the scale set by our measurements, so that every parameter is experimentally constrained; consistent with this, the relation $|\mu^M - \mu^X| = 1$ is selected by the data rather than assumed. Along the same lines, myosin motors are known to unbind in a force-dependent manner [58]. In our coarse-grained description, this is one further microscopic process shaping how the number of engaged motors per filament pair depends on actin density: It modifies the origin of $C^M(\rho)$ but, like the other microscopic scenarios above, leaves the functional form of Eq. (10) unchanged (see the Supplemental Material [42]). Finally, our description treats the cortex as mechanically isotropic. Were such anisotropy present, it would introduce an orientational order parameter and render the viscosity and active stress anisotropic (see Ref. [32]).

Similar to previous *in vitro* observations of contractile actomyosin [12,13,59], both the *in vivo* measurements and the active fluid model presented here show a decrease in network contractility with increasing cross-linker concentration. In contrast to previous work, the results here show that at high motor concentration, network contractility decreases, a qualitatively different behavior from both previous *in vitro* measurements [12,60] and theoretical predictions from a filament buckling model [61], where the network contraction rate instead saturates with increasing motor concentration rather than decreasing. Network connectivity has been used to explain previous *in vitro* observations: At low connectivity, motor forces cannot propagate to larger scales [15], while at high connectivity network contractility decreases due to either a substantial increase in network rigidity [1,62] or a decrease in filament buckling [13,61]. The active fluid model that we developed elucidates an alternative mechanism that can lead to this phenomenon. At low cross-linker concentrations, active stress falls off faster than viscosity, and thus the network deformation rate decreases. Experimentally measuring the network connectivity of *in vivo* actin cortices is a challenge, and in the future electron microscopy could potentially

be used to measure whether our experimental results at low actin density are above the percolation threshold. Such studies could also uncover how actin perturbations in this system modulate cortical actin network architecture, which can influence contractility separately from actin density alone [8,9,13]. At high cross-linker concentration, the model predicts that viscosity increases faster than active stress, and hence the deformation rate decreases. This idea shares similarities with models where increased network connectivity leads to high stiffness: Both would emerge from a high degree of filament cross-linking. An intriguing possibility is that both classes of models are limiting cases of a universal mechanical framework. Exploring this possibility will be an exciting avenue for future research.

Finally, we note that in the system considered here, the deformation rate is maximal near the wild-type composition, and perturbing the system through changing the cortical actin density, α -actinin concentration, or MRLC concentration largely only decreases the deformation rate. For the model considered in the main text, in which the motor is uniformly distributed along the filaments ($C_1^M = 0$), the maximal deformation rate is given by $d_{\max} = \frac{V_{||}C_0^x}{4C_0^x}$, implying that increasing the deformation rate further would only be possible through either increasing the relative asymmetry of cross-linker localization or increasing the motor walking speed. Increasing the motor walking speed or changing the system composition would have energetic consequences and energetic considerations can impose additional constraints in living nonequilibrium systems [63]. Understanding the energetic constraints of the emergent dynamics could further constrain possible

microscopic models and would require going beyond network architecture toward a thermodynamic description of such living active systems [5].

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P.J.F. and N.F. initiated the project and designed the experiments. P.J.F. and J.L. performed the experiments and analyzed the experimental data. S.F. and A.Z. designed the active fluid model. All authors participated in interpreting the experimental and theoretical results and in writing the manuscript.

The authors declare no competing interests.

DATA AVAILABILITY

All data that support the plots within this paper and other findings of this study are available from the corresponding authors upon reasonable request. Images were analyzed using custom written MATLAB code available in Ref. [71].

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