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## Supplemental Information

# Actomyosin Contraction Induces In-Bulk Motility of Cells and Droplets

**Thomas Le Goff, Benno Liebchen, and Davide Marenduzzo**

# Supplementary Material for: Actomyosin contraction induces in-bulk motility of cells and droplets

T. Le Goff, B. Liebchen, D. Marenduzzo

## I. LINEAR STABILITY ANALYSIS

Here, we discuss some details of the linear stability analysis which we use in the main text to predict the transition between a non-motile phase (immobile droplet) and a motile one (moving droplet), both in absence (in bulk) and in the presence of substrate friction, as we will now see in detail:

### 1. *Uniform base state ($\phi = 1$)*

We first identify the uniform solution $(m^*, \mathbf{v}^*) = (m_0, \mathbf{0})$ of Eq. (3) in the main text which represents a nonmoving actomyosin field with density $m_0$. Using the Ansatz $m = m^* + m', \mathbf{v} = \mathbf{v}^* + \mathbf{v}'$ and linearizing the Eqs. (3) in the main text for $\phi = 1$ around the uniform solution to understand the dynamics of small flucations, yields, after Fourier transforming the result:

$$\dot{m}' = m_0\alpha_m \mathrm{i}(\mathbf{q}\cdot\mathbf{v}') - \mathbf{q}^2 m' \quad (1)$$

$$\mathbf{v}' = \frac{-\mathrm{i}\mathbf{q}\chi}{(1+m_0)^2[\Gamma + (2+\eta)\mathbf{q}^2]} \quad (2)$$

Combining these equations for the dynamics of the fluctuations $m', \mathbf{v}'$ and using the Ansatz $\dot{m}'(\mathbf{q}, t) = \exp[\lambda(q)t]m'(\mathbf{q}, 0)$ yields the following dispersion relation for fluctuations around the uniform phase

$$\lambda(\mathbf{q}) = \mathbf{q}^2\left(\frac{\alpha_m\chi m_0}{(1+m_0)^2[\Gamma + (2+\eta)\mathbf{q}^2]} - 1\right) \quad (3)$$

Linear instability of the uniform solution occurs when (the real part of) $\lambda(\mathbf{q})$ is positive for at least some wavevector $\mathbf{q}$, which leads to the instability criterion

$$\frac{\alpha_m\chi m_0}{\Gamma(1+m_0)^2} > 1 \quad (4)$$

This criterion depends only on $\chi\alpha_m, m_0$ and $\Gamma$. If contraction is strong enough and enough myosin is present, with a large enough binding affinity (large $m_0, \chi, \alpha_m$), the positive feedback loop between myosin-induced 'fluid' advection and advection-induced myosin aggregation dominates substrate friction and the uniform phase loses stability in favour of actomyosin-aggregates. For $m_0 = 1, \alpha_m = 1$, as used in most of our simulations, Eq. (4) reduces to $\chi > 4\Gamma$ (see Fig. 2 A, main text) suggesting the onset of cell motion at $\chi > 4$ for $\Gamma = 1$. In absence of substrate friction ($\Gamma = 0$), the myosin feedback loop has no competitor and any positive $\chi$ destabilizes the uniform phase. Note that in all cases, very large $m_0$ values suppress the instability; this represents the scenario where most actin fibres are saturated with myosin so that substantial deviations from a uniform myosin gradient are impossible.
The fastest growing mode (maximum of $\lambda(\mathbf{q})$), which determines the early-time length scale of structures (clusters) growing out of the uniform phase, results from (3) as

$$q_{\max} = \left(\frac{\sqrt{\Gamma\frac{\chi m_0\alpha_m}{(1+m_0)^2}} - \Gamma}{2+\eta}\right)^{1/2} \quad (5)$$

The corresponding length scale $l_{\max} = 2\pi/q_{\max}$ of contraction-induced structures increases with $\eta, \Gamma$ and decreases with $\chi$; that is, we expect large early-time structures close to the onset of instability and smaller ones further away from onset.

### 2. *Presence of a cell ($\phi \neq 1$)*

In the presence of droplet boundaries (cell membrane), $\phi$ builds up a nonuniform steady state profile given by the corresponding solution of Eq. (3) in the main text. Here, we calculate the growth rate of fluctuation around such a nonuniform state in one dimension and use the following approximate representation for the steady state solutions

$$m^*(x) = \phi^*(x) = \frac{1}{2}\left(1 - \tanh\left[\sqrt{\frac{\Gamma}{8D_\phi}}(|x| - R)\right]\right) \quad (6)$$

where $R$ is the radius of the cell, and $v^* = 0$. Now we write $(m, \phi, v) = (m^*, \phi^*, v^*) + (m', \phi', v')$ and linearize the time-dependent equations of motion (Eqs. (3), main text) in the fluctuations $(m', \phi', v')$ around the nonuniform base-state $(m^*, \phi^*, v^*)$. Representing the resulting equations on a grid $-L, -L + dx, ..., L$, algebraically eliminating $v'$ and using the Ansatz $m_i'(t) = \exp(\lambda_i t)m_i'(0), \phi_i'(t) = \exp(\lambda_i t)\phi_i'(0)$ yields a $N = 2L/dx + 1$-dimensional matrix-vector equation which we solve for the eigenvalues $\lambda_1, ..\lambda_N$ by numerical diagonalization. We visualize the result of this procedure in Fig. 2 C,D (main text) for $\chi = 4.5$ (i.e. close to the onset of instability in the corresponding uniform system). Here, panel C shows that a few of the eigenvalues have a positive real part, i.e. the contraction-induced linear instability survives the presence of droplet boundaries and leads to a narrow band of unstable modes close to $\chi = 4$. Panel D visualizes the mode with the largest growth rate in configuration space (red) alongside the base phase field

(blue). Here, deep inside the cell, the wavelength of the shown mode resembles the one of the fastest growing mode of the underlying uniform system (5). However, the figure also shows that instability exists only in the interior of the cell where the actomyosin concentration is highest but is suppressed at the cell-boundaries. (Note that when the cell starts to deform (or move), the maximum of the actomyosin concentration may leave the cell center and the instability might be most effective close to the cell boundaries.)

This finding of suppression of instability close to the cell-boundaries suggests that instability is entirely suppressed if the cell is too small, i.e. the present linear stability analysis suggests that small cells cannot move based on myosin-contraction. We therefore ask: What is the critical cell size to obtain instability and contraction-induced droplet-motility? We first note that instability can only occur if the shortest unstable mode (of the instability band of the underlying uniform system) is smaller than the droplet size. Thus, we predict the critical cell-size as $l = 2\pi/q_c$ where $q_c$ is the short wavelength edge of the instability band of the underlying uniform system (i.e. the point where $\lambda(q)$ crosses the $\lambda = 0$ axis in Fig. 2 A,B, main text). We can readily calculate $q_c$ from the dispersion relation (3) and obtain the critical cell radius $R_{\rm cr}$ from the condition that at least one wavelength of the shortest possible unstable mode fits into the cell, i.e. from $2R_{\rm cr} = 2\pi/q_c$[1], as

$$R_{\rm cr} = \pi\sqrt{\frac{2+\eta}{\frac{\chi m_0 \alpha_m}{(1+m_0)^2} - \Gamma}} \tag{7}$$

We visualize this critical cell size in an instability diagram (or nonequilibrium phase diagram) in Fig. 2E (main text) and find very good agreement with direct x numerical simulations of the equations of motion. Our simulations also confirm that $R_{\rm cr}$ decreases with $\chi$, although we do not have sufficient data to infer a precise exponent for the decay.

## II. SUPPLEMENTARY FIGURE

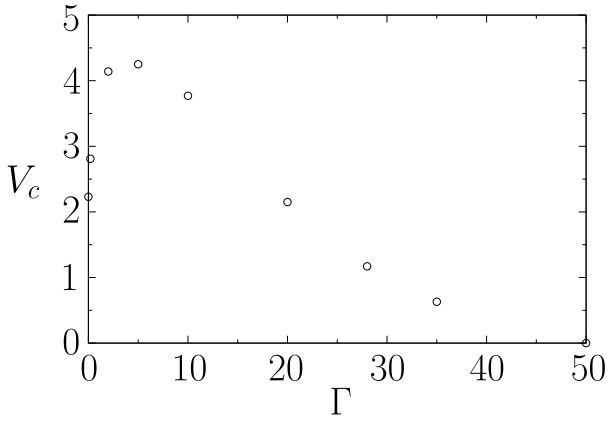


FIG. S1: Evolution of the dimensionless velocity of the cell with respect to friction coefficient Γ. The parameters used in simulations are $D_\phi = 25$, $\chi = 150$, $\alpha_m = 1$ and $V_{tar} = 12.5$.

[1] We note that a complete numerical linear stability in presence of the phase field suggests that in many cases also half a wavelength can build up in the cell, suggesting an somewhat 'earlier' onset of cell-motility than predicted below in terms of $R_{\rm cr}$.