In this Supplementary Theory Note, we detail our data analyses and modelling approaches to understand the mechanisms of non-muscle myosin II (MyoII) accumulation in the neighbouring cells. As shown in Fig. 2a-b and Supplementary Video 3, MyoII accumulation in the neighbouring cells is preceded by a marked decrease in E-Cadherin (E-Cad) signal along the ingressing *adherens* junction (AJ) pulled by the contractile ring. We first study E-Cad dynamics, in order to understand the mechanism of E-Cad depletion in the ingressing AJ during cytokinesis. Secondly, we propose a model, based on the active gel theory, on how a local depletion of E-Cad can drive MyoII accumulation via self-triggered actomyosin flows. We perform numerical integrations in a two-dimensional geometry to reproduce the locality of the MyoII accumulation.

1 E-Cad dynamics

As we saw in the main text (Fig. 2a-b and Supplementary Video 3), MyoII accumulation is preceded by a marked decrease in E-Cad concentration along the ingressing AJ formed between the dividing cell and it's neighbour. The decrease in E-Cad concentration is not impaired when we perturb E-Cad trafficking (Extended Data Fig. 4b-d), and is concomitant with the neighbouring junction elongation (Fig. 3a-c). Moreover, this decrease occurs while the total amount of E-Cad in the junction remains roughly constant (Extended Data Fig. 4e). Therefore, a natural hypothesis is that E-Cad depletion could result from a simple dilution effect due to AJ elongation (Extended Data Fig. 4h). To test this idea quantitatively, we first model the dynamics of E-Cad on a homeostatic junction, i.e. a junction of constant length, and then on an elongating junction, mirroring cytokinesis. We further consider the possibility for the elongation to be local, as we measure experimentally that in wild-type cells, only a small fraction of the AJ ingresses due to the pulling forces from the dividing cell's contractile ring (Extended Data Fig. 4i,j). Based on our experimental data, and consistent with previous reports[71,72], we show that E-Cad consists both of mobile and immobile pools, and its diffusion coefficient is low on the timescale of *wt* cytokinesis (Supplementary Table 1). We thus describe below how we measure the aforementioned parameters underlying E-Cad dynamics, and how we use them to test the proposed dilution model.

1.1 E-Cad dynamics on a junction of constant length

1.1.1 Theoretical considerations

Following[71–74], we write diffusion-reaction equations for both the mobile and immobile E-Cad concentrations (resp. $c_m(x,t)$ and $c_i(x,t)$) at a static junction of constant length l:

$$\partial_t c_i = D_i \partial_{xx} c_i \tag{1}$$

$$\partial_t c_m = D_m \partial_{xx} c_m - \frac{c_m - f_m c^0}{\tau} \tag{2}$$

where τ is the turnover time of the mobile fraction, c^0 the homeostatic total E-Cad concentration, f_m the mobile fraction of E-Cad, assumed constant (so that $f_m c^0$ is the homeostatic concentration of mobile E-Cad), and D_i (resp. D_m) the diffusion coefficient of the immobile (resp. mobile) E-Cad. Here, we used a linear model with the turnover and diffusion constants being independent of respective E-Cad concentrations, an hypothesis we verify later in the text. It should be noted that "immobile" in this context means that there is no exchange between junctional and cytoplasmic E-Cad, although E-Cad molecules at the AJ can still diffuse laterally along the junction.

In order to test the dilution model, we need to measure all the above parameters associated with E-Cad turnover. For that, we performed Fluorescence Recovery After Photobleaching (FRAP) experiments on E-Cad::GFP junctions of interphase cells. To extract each parameter independently in our system, we define $\delta c_m(x,t) = c_m - f_m c^0$ the difference between E-Cad concentration and its homeostatic value, as well as the i-moment of its distribution $M_i^m(t) = \int dx \delta c_m(x,t) x^i$. It can then be shown[75] that

$$M_0^m = M_0^m(0) e^{-t/\tau} \tag{3}$$

and

$$\frac{M_2^m}{M_0^m}(t) = 2D_m t + \frac{M_2^m}{M_0^m}(0) \tag{4}$$

A corresponding relationship can be found for the normalized second moment $M_2^i(t)$ of the immobile E-Cad concentration. Therefore, the evolution of the zeroth moment depends only on the turnover τ, whereas the value of the normalised second-moment depends only on the diffusion D_m, independently of the initial condition (i.e. on how the FRAP experiment was done). From a physical point of view, this means that turnover changes the total mass of E-Cad present at the interface, whereas diffusion only changes the width of the bleached region. Combining the equations of the mobile and immobile fraction predicts that the second moment $M_2(t)$ of the total E-Cad concentration evolves as $\frac{M_2}{M_0}(t) = \frac{M_2}{M_0}(0) + 2t\frac{D_i M_0^i + D_m M_0^m(t)}{M_0^i + M_0^m(t)}$, which after a characteristic time τ also increases linearly as $\frac{M_2}{M_0}(t) \to 2D_i t$. We will therefore see that the mobile fraction turns over sufficiently fast that its diffusion coefficient is irrelevant. Moreover, in the limit that $D_i \approx D_m$, the evolution of the normalized second moment becomes purely affine. We therefore drop the indices in the following sections, and refer to D_i as simply D.

1.1.2 Parameter extraction and fitting

These analytical expressions offer us a way to test *a posteriori* the assumptions of our linear model, by predicting that the two moments should recover as exponential and linear curves respectively. To fit the recovery curves of E-Cad::GFP upon photobleaching using the predictions above, we used a classical least-square fit approach. Firstly, each recovery curve was fitted by a single-exponential, from which we could extract both the turnover time and the mobile fraction. We fitted only the first $100s$ of each FRAP recovery curve, as they were the least sensitive to noise and subsequent bleaching. We found a turnover time $\tau = 64s \pm 26s$ and a mobile fraction of $f_s = 0.44 \pm 0.1$ (mean $\pm$ standard deviation, Supplementary Table 1). To assess the goodness of the fits, we compared in each recovery curve the experimental recovery with the best fit prediction, and calculated the coefficient of determination R^2 of the fit (the closer this value is to 1, the more accurate is the fit). The average of all coefficients of determination was $\langle R^2 \rangle = 0.92$, while the lowest value we found was $R^2 = 0.79$, indicative of consistently good fits.

Secondly, we measured the variance of the size of the bleached region in time to extract the diffusion coefficient (from the first $200s$ of each video). Individual variance extracted from the recovery curves showed a consistent increase in time. As there was substantial noise associated with the measurement, we averaged $n = 18$ cells, and show that the average curve is well-fitted (coefficient of determination $R^2 = 0.84$) by an affine function, as expected from the theory. By fitting the slope of the affine function, we could extract the E-Cad diffusion coefficient $D = 6 \pm 5\ 10^{-4} \mu m^2 s^{-1}$ (mean $\pm$ standard deviation, Supplementary Table 1). In particular, we could not detect a correction to the linear increase in the normalized second moment, which could indicate that $D_m \approx D_i$. As noted earlier, the goodness of both fits validates *a posteriori* the model that we used, showing that linear diffusion and turnover is a good approximation for E-Cad::GFP dynamics in this system.

One should note that the value we extracted for the diffusion coefficient is of the same order of magnitude, albeit 4-fold smaller, than what was reported in MDCK cells[71]. In contrast to MDCK cells however, we show that a large fraction of the E-Cad pool in the *Drosophila* notum is immobile on the timescale of cytokinesis.

To give a better physical intuition on the relative magnitude of diffusion and turnover for junction homeostasis, a useful quantity to define is the characteristic length of diffusion $l_D = \sqrt{D\tau}$. On length scales larger than l_D, turnover is the dominant mode of recovery, whereas for length scales smaller than l_D, diffusion is the dominant mode. With the values we measured, $l_D \approx 0.2\mu m$, validating the idea that turnover is much more important than diffusion at the length scale of the whole junction ($l \approx 7\mu m$ typically), and that we do not expect significant recoveries from diffusion at the scale of a few microns, as in the case of cytokinesis.

We then turned to the E-Cad::GFP recovery dynamics on the junctions of cells undergoing cytokinesis. In this case, long-term data is not easily accessible because of the complex three-dimensional movements associated with cytokinesis, so measuring a diffusion coefficient was not technically possible. Nevertheless, we performed high-frequency imaging (one frame per second) in order to study the short-term FRAP recovery. For AJs of cells undergoing cytokinesis, we found that the E-Cad::GFP mobile fraction and the average turnover time are respectively $\tau = 46s \pm 18s$ and $f_s = 0.41 \pm 0.15$ (mean $\pm$ standard deviation, Supplementary Table 1). Interestingly, the mobile fractions were not statistically different between the interphase and cytokinesis cases ($n = 22$ dividing cells and $n' = 17$ interphase cells, $P > 0.2$, Mann-Whitney test), although the turnover times displayed a significant, albeit slight, difference ($P < 0.05$, Mann-Whitney test). For the sake of consistency, we also performed FRAP on the junction of interphase cells at one frame per second, and saw no statistical differences with the results from videos at a rate of one frame per $5s$ ($n = 26$ interphase cells, $P > 0.1$ for the mobile fraction, $P > 0.05$ for the turnover time, Mann-Whitney tests). When comparing the $1s$ interphase data to the $1s$ cytokinesis data directly, we also found no statistically significant difference ($P > 0.2$ for the mobile fraction, $P > 0.2$ for the turnover time, Mann-Whitney tests).

Altogether, these results show that E-Cad turnover and mobile fraction are largely unaffected during cytokinesis.

1.2 E-Cad dynamics on an elongating junction

Having determined the parameters of E-Cad::GFP dynamics, we analysed how the local elongation of the junction contributes to modulate E-Cad concentration.

1.2.1 Theoretical considerations

In order to get simple analytical insights into E-Cad dynamics on an elongating junction, we start by considering an AJ of variable length $l(t)$ being globally dilated, so that the concentrations of mobile and immobile E-Cad are independent of the position x, and only vary in time. The equation for the mobile and immobile E-Cad concentrations then reads

$$\frac{dc_m}{dt} = -\frac{c_m - f_m c^0}{\tau} - \frac{c_m}{l}\frac{dl}{dt} \tag{5}$$

and

$$\frac{dc_i}{dt} = -\frac{c_i}{l}\frac{dl}{dt} \tag{6}$$

Physically, this means that when the AJ elongates exponentially at a rate $r = \frac{1}{l}\frac{dl}{dt}$, the immobile fraction gets passively diluted as the same rate, whereas the mobile fraction can respond by

turnover, and maintain a steady state concentration c_m^∞, with:

$$c_m^\infty = \frac{c_m(r=0)}{1+r\tau} \tag{7}$$

As expected, as the rate of AJ elongation r becomes large compared to the turnover rate $1/\tau$, steady state E-Cad concentration drops.

Guided by our experimental results (see below), we consider theoretically the alternative possibility for the AJ elongation to be local. In the following section, we therefore note and measure f, the fraction of the AJ being elongated. In this scenario, we cannot consider E-Cad concentration to be independent on position x, and must therefore adapt Eq. (1)-(2) to a locally elongation AJ. We first adimensionalise all lengths x by the length of the junction l, so that our equation is defined on the domain $x \in [0,1]$. The elongating region is defined initially as $x \in [x_i, x_f]$ with $x_i = (1-f)/2$ and $x_f = (1+f)/2$. We then assume that the deformation is affine, and define the stretched coordinates $y = f(x)$, such as:

$$f(x) = \begin{cases} x & \text{for } x < x_i \\ x + \int_0^t r(t)dt(x - x_i) & \text{for } x \in [x_i, x_f] \\ x + \int_0^t r(t)dt(x_f - x_i) & \text{for } x > x_f \end{cases} \tag{8}$$

$r(t)$ being the local stretch ratio at a given time t, and the previous Eq. (1)-(2) can be re-written on the y coordinates, therefore taking into account the locality of the AJ elongation. In the following section, we analyzed whether the local or global model applies, and then performed numerical integration of the corresponding equations using a finite difference method.

1.2.2 Predictions and numerical integration of the model

wt dividing cells

We first start by simulating AJ elongation in the case of *wt* dividing cells facing *wt* neighbours. In order to estimate the fraction f of the AJ being elongated, we measured the width t of the ingressing region normalized by total AJ length, and estimated from the time average (Extended Data Fig. 4i,j) that $f = 12\%$ of the junction is pulled by the contractile ring in the wild type condition. We then set the parameters of E-Cad dynamics D, τ and f_s to the values extracted experimentally above ($D = 6.10^{-4}\mu m^2 s^{-1}$, $\tau = 46s$ and $f = 0.41$). Using the results from the measurements of the main text, we consider a junction growing linearly from $l = 7\mu$m to $l = 11\mu$m (i.e. a total 58% junctional growth, see Fig.3a-c and Extended Data Fig. 4k) in $T = 326$s (which corresponds to the average time of cytokinesis), after an initial phase of junctional retraction (see Methods section for additional details). Therefore, experimentally, $r\tau \ll 1$, and from the analytical expression (7) above, we can predict that the mobile fraction is largely unaffected by the AJ elongation, as cytokinesis is slow enough that the mobile fraction is recovered by turnover.

One can also estimate the characteristic time $\tau_{diff} = l_d^2/D$ that diffusion would take to fill a E-Cad depletion of characteristic size $l_d \approx 1\mu m$. With the coefficient of diffusion measured, we obtain $\tau_{diff} \approx 2.10^3$ s. This time is significantly larger that the time T of cytokinesis, meaning that we do not expect diffusion to significantly homogenise the reduction of E-Cad::GFP signal, although some small E-Cad flow will occur towards the ingressing AJ. Indeed, as shown in the kymograph of the full numerical simulation of *wt* cells (Fig. 3d), the simulations predict a persistent E-Cad depletion throughout the process, to levels around 40% of their homeostatic value. As the mobile fraction recovers fast, the bulk of the decrease corresponds to the dilution of the immobile fraction, which therefore plays a crucial role to explain E-Cad depletion upon junction elongation in this system.

To further compare the prediction of the model to the experimental data, we computed E-Cad concentration in the centre of the depletion as a function of AJ elongation (thick line, Extended Data Fig. 4l). In order to estimate a confidence interval for our predictions, we ran the simulations again using the extremal values for the turnover τ, diffusion D and immobile fraction f reported above (i.e. $\pm$ their standard deviation). We then plotted the extremal values for our prediction of the evolution of E-Cad::GFP concentration vs total AJ elongation (thin lines, Extended Data Fig. 4l), which takes into account the uncertainty on the E-Cad::GFP dynamics parameters. We observe that the dilution model can account for a large fraction of the E-Cad::GFP depletion, as well as displaying the correct temporal trend. To quantify the agreement between the experimental data and the model, we calculated the coefficient of determination of our prediction for E-Cad::GFP concentration vs AJ elongation, and found $R^2 = 0.82$, indicative of a good quantitative prediction. As we are dealing with non-linear curves, we also quantified the standard deviation of the residuals, and found $S = 0.12$.

rok neighbouring cells

In order to challenge the model, we first tested the role of *rok* in the E-Cad decrease at the ingressing AJ, as contractility was suggested to regulate E-Cad stability at the AJs[76]. For that, we first measured the normalized width t of the ingressing region to assess the locality of the elongation, and found an average fraction $f \approx 26\%$ being pulled (Extended Data Fig. 4i,j), arguing for a local elongation, although on a larger scale than in the case of *wt* dividing cells facing *wt* neighbours. Moreover, we found that total AJ elongation imposed by a *wt* dividing cell facing a *rok* neighbour is 40%, (Extended Data Fig. 4k), and that the duration of cytokinesis is similar to the *wt* case, suggesting that the neighbour's contractility does not influence these parameters. When we performed our numerical integration under these conditions, simply changing the maximum elongation to 40%, we observed an intermediary phase of milder depletions, similar to the experimental observations (Extended Data Fig. 5c-j). Since Rok is required for MyoII accumulation in the neighbouring cells (Extended Data Fig. 5p-r), these results further show that MyoII accumulation in the neighbours is not required for the decrease of E-Cad concentration along the elongating junction. These findings are also in agreement with the fact that E-Cad decrease precedes MyoII accumulation in the neigbours. To quantify the goodness of our fits, we calculated again the coefficients of determination and standard deviation of the residuals, and found $R^2 = 0.69$ and $S = 0.14$.

pnut dividing cells

Finally, to further validate our model, we examined the effect of *pnut* loss of function on the E-Cad::GFP dynamics during cytokinesis. We find that cells neighbouring *pnut*^{RNAi} cells do not display a marked E-Cad depletion (Fig. 3e-g). We analysed whether the characteristics and kinetics of cytokinesis in *pnut* dividing cells could account for the lack of depletion. Compared to *wt*, as the total time for cytokinesis in *pnut*^{RNAi} is nearly four times longer ($T = 1111s$, Extended Data Fig. 1f and Supplementary Video 2b), and the elongation is markedly reduced (25%, Fig. 3e-g and Extended Data Fig. 4k). Moreover, we measured again the width t of the ingressing region normalized by the length of the AJ to assess the locality of the elongation, and found that, upon *pnut*^{RNAi}, the bulk of the AJ is deformed (Extended Data Fig. 4i,j), thereby often resulting in a triangular-shaped AJ, which contrasts with the characteristic finger-shaped AJ observed in *wt* cells. We thus implemented a global, rather than local, AJ elongation in the numerical integrations for *pnut*. Therefore, the prediction of our model would indeed be that E-Cad is less diluted and has more time to recover, largely suppressing the E-Cad decrease.

Before reaching this conclusion, we needed to exclude a specific function of *pnut* in regulating E-Cad levels or dynamics. Therefore, we measured the parameters of E-Cad dynamics in *pnut* cells by performing FRAP experiments on interphase *pnut*^{RNAi} cells, using the conditions and analysis pipeline previously described. We found an immobile fraction only slightly, although significantly, smaller than in *wt* cells ($f = 0.55 \pm 0.16$, $P < 0.01$, $n = 34$ cells, mean $\pm$ standard deviation, Mann-Whitney test, Supplementary Table 1), and both the turnover time and diffusion coefficient were statistically indistinguishable to the wild type (respectively $\tau = 69s \pm 51$, $P > 0.2$, $n = 34$ cells and $D = 8 \pm 8\ 10^{-4} \mu m^2 s^{-1}$, $P > 0.2$, $n = 20$ cells, mean $\pm$ standard deviation, Mann-Whitney tests, Supplementary Table 1). Therefore, E-Cad kinetics upon *pnut*^{RNAi} are similar to *wt*, and cannot account for the lower E-Cad depletion.

We then ran our simulations for these updated parameter values. The smaller immobile fraction measured in *pnut*^{RNAi}, but more importantly the fact that AJ elongation is decreased and occurs on a more global scale, decreased drastically the E-Cad depletion. Altogether, this accounts quantitatively for the absence of large and persistent E-Cad depletion observed in cells neighbouring *pnut*^{RNAi}, validating our hypothesis (Fig. 3h and Extended Data Fig. 5m). To quantify the goodness of our fits, we calculated again the coefficients of determination and standard deviation of the residuals, and found $R^2 = 0.73$ and $S = 0.03$.

One should note that for the sake of simplicity, we have assumed here that the elongation in *pnut*^{RNAi} was fully global, although some cells displayed slightly more local AJ elongation, in particular in the early stages of constriction. Interestingly, we noted that in 50% of cases, we could measure a small E-Cad depletion in *pnut*^{RNAi} at the early stages of constriction, but these depletions were always very short-lived (Extended Data Fig. 5k-l). This type of complex and transient behavior goes beyond the scope of our simple model, but could be explained in a natural manner by modifying dynamically the locality of the AJ elongation for a given condition.

Altogether our model and its validation using *wt* and mutant conditions indicates that a passive dilution mechanism can account for the bulk of E-Cad depletion at the ingressing AJ between dividing and neighbouring cells. One should note that this E-Cad decrease effect is expected to facilitate cortical detachment (Extended Data Fig. 3), by locally lowering the concentration of E-Cad molecules.

2 Actomyosin accumulation in neighbouring cells

So far, we have shown that E-Cad depletion and cortex detachment are likely to be passive consequences of the pulling forces produced during contractile ring constriction in the dividing cell. As we have shown that *pnut* dividing cells, which produce lower pulling forces, can nevertheless induce cortex detachment in the neighbouring cell in 50% of the cases ($n = 20$ cells, Extended Data Fig. 3g and Supplementary Video 4b), and as we found that E-Cad decrease is sufficient to rescue MyoII accumulation under lower pulling forces (Fig. 3i-k, Extended Data Fig. 6 and Supplementary Video 6), we sought to study theoretically how MyoII accumulation could result from an E-Cad depletion, again only using first principles. Theoretically, there could be two classes of origins for the MyoII accumulation:

- either it arises from a local recruitment of MyoII via a classical mechanotransduction mechanism, for instance protein unfolding, as in the case of Vinculin (Vinc) and α-Cat, which leads to Actin binding and/or MyoII recruitment[73,77,78], or from a signalling role of the E-Cad/catenin complex[79],
- either it arises from a convection mechanism, with actomyosin flowing from one region to the next.

As detailed in the main text, we have shown that MyoII accumulation in the neighbours is independent on the classical Vinculin mechano-transduction pathway, as Vinc was not necessary for the accumulation, and did not colocalise with MyoII near the base of the ingressing membrane (Extended Data Fig. 7a-e). Moreover, although E-Cad is known to generate intracellular signals for Rho GTPase signalling and cytoskeletal organization, we found that loss of function of Rho or Diaphanous (Dia) only delayed MyoII accumulation (Extended Data Fig. 7g-j). Finally, we performed an extensive characterisation, discussed in the main text, of all *Drosophila* Formins, as well as Arp2/3 and Enabled function, by single, double and triple mutant analysis (Extended Data Fig. 7l-r and Extended Data Fig. 8a,b). Strikingly, none of these Actin nucleators abrogate MyoII accumulation and only a drastic reduction of F-Actin nucleation in a triple *dia*, *arp3*, *Chic* can abrogate MyoII accumulation (Extended Data Fig. 7l-r and Extended Data Fig. 8a,b).

Therefore, we decided to explore the second hypothesis. In particular, given the robust relationship between E-Cad depletion and MyoII accumulation uncovered in the main text, we wished to examine whether local changes in the mechanical environment of the contractile actomyosin cortex (such as loss of linkage) could theoretically result in actomyosin flows and MyoII accumulation. As briefly described in the main text, by modeling the actomyosin cortex as an active fluid, we show that MyoII motor activity has the tendency to generate flow-driven accumulations. We further show that theoretically, these accumulations can be both triggered and guided by local loss of linkage to the AJ, modelled as an effective friction. Therefore, we first develop analytical arguments to understand qualitatively this behavior, before resorting to one- and two-dimensional numerical simulations to reproduce the experimental geometry and phenomenology.

2.1 Description of the model

We thus start by writing a simple hydrodynamic theory for the actomyosin cortex, using the active gel theory, which has been shown to be adapted to describe actomyosin structures in many instances[75,80–84]. We aim to describe the condition for a stable uniform cortex, and the converse condition for actomyosin flows to occur. Indeed, in the past years, actomyosin flows have been reported in a variety of settings, and proposed to be linked to intrinsic properties of the actomyosin network, such as its contractility[75,84–88]. Thus, we write down a one-dimensional theory for MyoII and Actin concentrations (resp. ρ and a) in the ingressing region. The conservation equation for the bound fraction of MyoII, considered as a solute in the Actin gel and of density ρ reads:

$$\partial_t \rho + \partial_x(\rho v) - D\partial_{xx}\rho = \frac{\rho_0 - \rho}{\tau_m} \tag{9}$$

where τ_m is the turnover time of MyoII and D an effective diffusion coefficient of MyoII relative to the Actin gel. Such a coefficient is needed to prevent infinite accumulation of actomyosin in an infinitely small region. The reference density of MyoII ρ_0 denotes the ratio of polymerization over depolymerization rates, assuming first order kinetics and v refers to the velocity of the Actin gel on which the MyoII motors attach. We thus model actomyosin as a viscous and contractile gel of length l. Force balance and constitutive equation at linear order of the actomyosin gel then read, respectively:

$$\partial_x \sigma = \xi v \tag{10}$$

$$\sigma = \chi \rho/\rho_0 + \eta \partial_x v \tag{11}$$

where σ is the stress in the gel, η its viscosity, χ the contractility arising from bound MyoII motors, ξ the friction coefficient between the actomyosin gel and its neighbours and/or the apical ECM. These equations must be complemented by two mechanical boundary conditions, $v(0,t) = v(l,t) = 0$, since the actomyosin gel is clamped at both ends. We also specify two no-flux boundary conditions $\partial_x \rho(0,t) = \partial_x \rho(l,t) = 0$, i.e. no MyoII flux can enter into the system at the boundaries. The filamentous Actin concentration can be then directly deduced once the velocity v has been determined, through the conservation equation $\partial_t a + \partial_x(av) = R(a)$, R being a reactive term taking into account Actin turnover.

One should note that our two conservation laws for filamentous apical Actin (resp. bound MyoII) assume implicitly that monomeric Actin and unbound MyoII are in excess and diffuse rapidly from the cytoplasm to the cortex on the timescale of turnover. This is a reasonable hypothesis, as verified experimentally by FRAP in cultured cells[89,90], which show that the diffusion coefficients of monomeric Actin and MyoII motors are in the range of $1-20\mu^2 s^{-1}$, a value consistent with the Stokes-Einstein equation. This means that their diffusion within a protrusion of characteristic size 2μ occurs very rapidly, i.e. within fractions of a second to a few seconds. Finally, and as detailed in the main text, we concentrate in the following section on the distribution ρ of MyoII, since we are interested in particular in the MyoII accumulation.

In our model, we describe the coupling between the Actin cytoskeleton and AJ, which is mediated by their interaction via the $\alpha-$Catenin-E-Cad complex, as an effective friction coefficient[91,92]. This friction coefficient ξ has been studied in various contexts, and theoretically is the sum of several different contributions, which can be hard to disentangle[93,94]: coupling of the *adherens* junction to the apical extracellular matrix[95], protein friction linked to the engagement of homophilic adhesion molecules[91] such as E-Cad, or internal friction from cross-linkers in the gel itself[96,97]. Importantly, we expect that lowering the linkage between the cortex and the membrane, in particular by decreasing E-Cad concentration, as described in Section 1, should lower this effective friction. Finally, these equations can be conveniently adimensionalised by rescaling all lengths by the hydrodynamical wavelength $L = \frac{\eta}{\xi}$, all times by $\eta/\xi D$ and all concentrations by ρ_0, as well as using the rescaled junction length $\alpha = l/L$, the rescaled contractility $\chi = \frac{\rho_0 \chi_0}{\xi D}$ and the rescaled turnover $\phi = \frac{\eta}{D\tau_m \xi}$.

2.2 Condition for a stable homogeneous cortex and parameter estimation

In the following paragraphs, we provide successively a qualitative argument and a quantitative criterion as a function of contractility and friction for the appearance of spontaneous MyoII accumulations. Because actomyosin is contractile, an homogeneous state can be spontaneously broken: a small accumulation of MyoII causes contractile flows, which constitute a self-reinforcing loop and can lead to the accumulation of actomyosin locally. Nevertheless, because of turnover, actomyosin is constantly added in the depleted region, and removed in the dense region, causing a steady state flow. It should be noted that this description is very similar to the one proposed for cell motility[85,86]. Starting from an initially uniform MyoII density and an infinitely large cortex, a linear stability analysis of the set of equations (9)-(11) predicts that a uniform cortex is only stable[75] if its contractility χ is smaller than a critical value χ_1^c defined as

$$\chi_1^c = \left(\sqrt{\frac{\eta}{\tau_m}} + \sqrt{D\xi} \right)^2 \tag{12}$$

Therefore, this threshold for the appearance of spontaneous actomyosin flows depends on the value of the friction coefficient ξ, i.e. on the attachment between the cortex and its surrounding through the plasma membrane. In the case of cell motility, authors have explored the idea[86] that a local upregulation of contractility χ allows the system to pass the threshold χ_1^c, therefore triggering ameboid motion. Here, we propose the converse idea that, at

constant contractility, a change in the properties of the gel, such as friction ξ, is sufficient to trigger actomyosin flows. The advantage of such a model would be that it is self-triggered and self-maintained, without the need for an active signal triggering a contractility increase. In our model, the local elongation of the junction depletes E-Cad concentration at the ingressing AJ (Fig. 3a-d and Extended Data Fig. 4l), and this physical cue would be enough to trigger an actomyosin response, via flows, resulting from an intrinsic physical instability of the cortex. This could be a complementary mode of mechano-sensing, relying on generic physical properties of the cytoskeleton rather than on a classical signalling cascade.

One model parameter that can be accessed experimentally in a straightforward manner is the turnover time of MyoII τ_m. Using photobleaching experiments of the MyoII accumulation in the neighbours, performed on MyoII::GFP clones facing MyoII::RFP dividing cells (Fig. 4d,e, Supplementary Video 7a, see also Methods), we found $\tau_m = 22s \pm 2$ (average $\pm$ s.e.m, $n = 14$ cells), consistent with previous experimental values[98]. Moreover, using orders of magnitude from the literature[84,99–101] ($\chi\rho_0 \approx 10^3 Pa, \eta \approx 10^4 Pa.s$ $\xi \approx 10^{16} Pa.m^2.s^{-1}$, $D \approx 10^{-13} m^2 s^{-1}$), we estimate that $\frac{\eta}{\tau} \approx 10^3 Pa$ and $D\xi \approx 10^3 Pa$, confirming that friction plays an important role in setting the contractility threshold described above. Using these orders of magnitude yields for the non-dimensional ratios: $\chi \approx 1$, $\phi \approx 1$ and $\alpha \approx 10$, which are the values we keep in all the numerical integrations below. It is also worth noting that the actomyosin in the medial pool is known to be much more dynamic than the junctional actomyosin[88] (in particular displaying large scale flows), which could be due to their differential attachment or to their differential turnover[102].

Therefore we propose that, at constant contractility and constant cortical properties, a decrease in friction, i.e. cortical attachment, due to E-Cad decrease in the ingressing AJ is sufficient to spontaneously generate actomyosin flows. From Eq. (12), friction has to decrease below a critical threshold $\xi_c = \frac{1}{D}\left(\sqrt{\chi - \frac{\eta}{\tau}}\right)^2$, in order for spontaneous flows to form. The characteristic speed in the friction-less ingressing AJ of length l is then $v_c \propto \frac{\chi\rho_0 l}{\eta}$ which, as expected, increases with contractility, as well as with the length of the ingressing AJ.

In the absence of friction, as studied by Turlier *et al.*[81] spontaneous flows occur if contractility is larger than a second, lower critical value χ_2^c defined as:

$$\chi > \chi_2^c = \frac{\eta}{\tau_m} \tag{13}$$

Interestingly, another parameter that can be extracted from experiments is the ratio of viscosity η over the active contractility χ, which dictates the characteristic time $\tau_r = \frac{\eta}{\chi}$ of junctional recovery upon laser ablation of a junction. Therefore, the criterion for flows to occurs from Eq. (13) can be rewritten as

$$\frac{\chi}{\chi_2^c} = \frac{\tau_m}{\tau_p} > 1 \tag{14}$$

Using laser ablation in pupae from 14 to 18 hAPF, we have previously shown[103] that $\tau_p \approx 10s$. Therefore, we find a ratio $\tau_m/\tau_p \approx 2$, and the theory predicts that one should see actomyosin flows upon loss of friction, as we confirmed experimentally both from live-imaging (Fig. 4d,e,g,h, Extended Data Fig. 9a,b and Supplementary Videos 7a,7c,7d) and MyoII::Dendra2 photoactivation (Fig. 4f and Supplementary Video 7b). The characteristic flow velocity can also be estimated as

$$v_c \propto \frac{l}{\tau_p} \approx 0.2 \mu m s^{-1}$$

which has the same order of magnitude as the experimentally measured value of $v_{exp} = 0.1 \pm 0.04 \mu m s^{-1}$ (mean $\pm$ standard deviation), both for F-Actin and MyoII (Extended Data Fig.

9c). Importantly, we also measured very similar F-Actin flow velocities for *moe* and *E-Cad* neighbors (Extended Data Fig. 10g-j,l,m), where friction is also expected to be low, while we observed statistically lower velocities and intermittent flows in cells neighboring a *pnut* dividing cell (Fig. 4i-l and Supplementary Video 8a), which produce lower elongation and E-Cad depletion (Fig. 3e-g), and where friction is thus expected to be higher.

Interestingly, as noted in the main text, although we observed sustained flows in the ingressing region, where E-Cad is low, the contribution of flows seems to be minimal in the lateral direction, i.e. coming from the remaining attached cortical actomyosin, in a region where E-Cad is high (Fig. 4d,e,g,h, Extended Data Fig. 9a,b and Supplementary Videos 7a,7c,7d). If the accumulation was mainly due to a local up-regulation of the contractility χ at the base of the ingressing region, we would predict equal flows from both regions, on the lengthscale of the hydrodynamic length $l_h = \sqrt{\frac{\eta}{\xi}}$. The experimentally observed prevalence of flows along the ingressing region therefore reinforces the idea that E-Cad depletion is necessary to induce large-scale flows, and that friction along the AJ in the E-Cad high region is sufficiently large to strongly reduce flows along the lateral direction. Indeed, using the parameters described above yields a hydrodynamic length of $l_h \approx 1\mu m$, consistent with a large screening role for friction.

In conclusion of this section, our theoretical model suggests that MyoII accumulation arises from retrograde actomyosin flows in the ingressing region (Fig. 4c and Extended Data Fig. 8j,k). This would be distinct from the medial pulsatile flows associated with apical constriction and cell intercalation[104–108]. The distinction is at least three-fold: geometric, mechanistic and functional. From a geometric point of view, apical pulses flow centripetally to generate large accumulations in the centre of the cell, and are therefore not associated with cell-cell junctions. Conversely, flows associated with intercalation emerge from the medial pool and flow towards the cell cortex. In contrast, flows in our system arise in the ingressing AJ, and occur along this junction towards the E-Cad rich boundary. Secondly, from a mechanistic point of view, apical flows during apical constriction and intercalation have been shown to be associated with G-coupled or Toll receptor signaling, that locally control cell contractility[109,110]. Similarly, the basal actomyosin flows that have been proposed to drive cell migration[86], are thought to be triggered by modifications in active contractility. In sharp contrast, we have uncovered that flows can be triggered by a reduction of E-Cad at the cell cortex, which locally reduces friction. Finally, from a functional perspective, the flows described in apical constriction, cell intercalation and cell migration, have been shown to be 'force production' entities, necessary to trigger cell shape change. Here we demonstrate that the flows are used as 'force transmission' entities, i.e. to transmit a force between two neighbouring cells in response to division.

2.3 Localisation of the accumulation

Next, we explore how a local decrease in friction can dictate the position of an actomyosin accumulation, successively in one-dimensional and in two-dimensional models.

2.3.1 One-dimensional model

For an actomyosin gel of length l with spatially homogeneous properties, studied in the context of cell migration[85,86], the accumulation arises with equal probability at both sides of the gel ($x = 0$ or $x = l$). To go further, and in particular explore the directionality of the flows in the case of non-uniform friction, we performed a full numerical integration of our one-dimensional model (Mathematica code provided as a Supplementary File).

For the sake of simplicity, we start with the simplified case of an actomyosin gel of length l with uniformly high friction $\xi_h > \xi_c$ (for a normal cortex, we know that $\xi > \xi_c$ since we do not observe spontaneous flows), except in the centre of the gel, where friction is much lower than the threshold ($\xi_l \ll \xi_c$) on a length l_{hole}, on a domain $x \in [l/2 - l_{hole}, l/2 + l_{hole}]$. We used the same boundary conditions as described above. We performed a parameter sweep, varying the central lower friction ($\xi_l = \xi_h/2, \xi_h/5, \xi_h/10, 0$), as well as the contractility χ and the size of the low-friction domain. For each value of the parameter set, we performed 100 parallel numerical integrations, starting from different random initial conditions, and performed statistics on the resulting actomyosin profile. This is important, as previous work has shown that this system of equations has a large amount of metastable, long-lived states[75]. In all the conditions examined, provided that $l < \lambda_c = 2\pi(\eta D\tau/\xi_l)^{1/4}$, actomyosin accumulations formed at the boundaries between low and high friction (Extended Data Fig. 8c for $\xi_l = 0, \chi = 1.3\chi_c$). This analysis, in a simplified geometry, suggests that an actomyosin accumulation can be guided and localised by spatial modulation of friction, with the accumulation occurring at the interfaces between low and high frictions.

Next, we wished to examine the more complex, but more biologically realistic case, of the localisation of actomyosin due to the juxtaposition of a low friction region ($x \in [0, l/2]$) and high friction region ($x \in [l/2, l]$). Although this case might superficially look similar to the previous one, it actually bears crucial differences, as this configuration allows for an alternative actomyosin localisation at $x = 0$, i.e. at the boundary of the gel in the low friction region. To come back to the *in vivo* situation, this corresponds to the possibility of actomyosin accumulating at the tip of the ingressing region versus accumulating at the base of the ingressing region, at the high/low E-Cad boundary. Indeed, in that case, performing the same parameter sweep as before on ξ and χ, we find (Extended Data Fig. 8d,e for $\xi_l = 0, \chi = 1.3\chi_c$) that the accumulation can take place with nearly equal probability at the boundary between high and low friction ($x = l/2$), or at the boundary of the gel ($x = 0$). Nevertheless, as these two solutions have similar stabilities, even a very small imperfection or tilt would be enough to robustly localise actomyosin at the base of the ingressing region, as observed experimentally.

Therefore, in the next paragraphs, we explore four possibilities, based on our experimental observations, which can robustly guide the actomyosin accumulation at the base of the ingressing region: the role of the detached cortex, the role of the temporal increase in the length of the ingressing region, possible biases in F-actin nucleation and the two-dimensional geometry of the ingressing region.

Firstly, the presence of a cortex at the base of the junction can theoretically serve as a guiding cue for actomyosin accumulation, although we have seen in the main text (Fig. 2d, Extended Data Fig. 3g-j and Supplementary Videos 4a-d) that this cannot be the exclusive determinant. This can be seen qualitatively by considering the fact that actomyosin flows towards denser (i.e. more contractile) regions, so that a dense cortex can serve as a strong cue. To demonstrate this, we performed the same numerical integration as above (Extended Data Fig. 8f), assuming that the steady state value ρ_0 of MyoII (and Actin) from turnover is 10% larger in the cortex (high friction region) than in the ingressing domain (low friction region), and saw that this was enough to robustly localise the accumulation at the high/low friction boundary (100 $\%$ of the time in $n = 100$ numerical integrations, starting from different random initial conditions).

Secondly, taking into account the slow advancement of the ingressing region localises the actomyosin accumulation at the base of the ingressing AJ (at the high/low friction boundary). To take this into account in our system of equations, we now make the length of the domain $l(t)$ explicitly dependent on time. We take $l(t) = l_0(1 + r't)$ linearly increasing in time, as the data from the main text suggest, with

$$r' \approx 2.10^{-3} s^{-1}$$

We must modify the clamped boundary condition to take into account that we now have an expanding domain ($x \in [l_0 - l(t), l_0]$), with $v(l_0 - l(t)) = -l'(t)$ replacing $v(0) = 0$ (see[85] for details on moving boundary conditions in this system). Qualitatively, this depletes actomyosin at the front of expansion, biasing accumulation at the base of the ingressing region. If friction was low everywhere, the accumulation would travel to the trailing front, but the high friction zone blocks such flows, causing the accumulation to localise at the low/high friction interface. Quantitatively, we integrated our system of equations with these new boundary conditions, and again could see a robust localisation of actomyosin at the high/low friction interface in all cases considered ($\xi_l = 0.01\xi_h, \chi = 2\chi_c$, 100 % of the time in $n = 100$ numerical integrations, starting from different random initial conditions, see Extended Data Fig. 8g for the steady-state MyoII concentration profile).

Thirdly, an assumption of our model is that the velocity of the actomyosin flows tends to zero at the boundary (i.e. at the tip of the ingressing AJ). A theoretical prediction is then that the velocity of the actomyosin flows should increase away from the tip, reach a maximal value and then decrease in the region close to the MyoII accumulation (Extended Data Fig. 8h). Due to the reduced size of this region and the lower Lifeact::GFP signal at the tip of the protrusion (compare Fig.4g,h, Extended Data Fig. 9a,b and Extended Data Fig. 10a,b), it is difficult to determine, in a quantitative manner in all kymographs, whether such profile is observed experimentally even with our current best imaging resolution conditions. A qualitative analysis nevertheless suggests that in some kymographs, the velocity of F-Actin speckles increases rapidly from the tip of the ingressing AJ, reaching near constant velocities (i.e. straight lines in the kymographs, Fig. 4h). Such a behavior could be consistent with a more complex description of the actomyosin cortex as a visco-elastic material[94], instead of a simple viscous material as we assumed here. Alternatively, this could be reproduced by changing our boundary conditions, i.e. allowing for a non-zero speed v at the tip ($x = 0$), directed towards the high friction zone, mimicking the assembly of new filaments at the tip at a rate sufficient to feed the flow. Similarly to the hypotheses above, this would break the symmetry of the problem by biasing the flow towards the base of the accumulation. Quantitatively, we verify this by performing the same simulations as above, but simply allowing a non-zero velocity at the tip $v(x = 0) = 0.1$ or $v(x = 0) = 0.2$ (Extended Data Fig. 8h), which biases the accumulation at the low-high friction boundary. Moreover, this only marginally modifies the density profile compared to the $v(x = 0) = 0$ case (dashed lines, reproduced from Extended Data Fig. 8d for comparison), while the velocity v now starts at a high value at the tip of the protrusion, and adopts a flatter profile for increasing boundary velocity.

2.3.2 Two-dimensional numerical integrations for the ingressing region

The simple one-dimensional model has helped us establish that a local decrease in friction can both trigger and guide actomyosin flows. Nevertheless, the geometry of the ingressing region *in vivo* is two dimensional in nature, and could also participate in guiding the accumulation (Extended Data Fig. 1a-d and Supplementary Video 1).

Therefore, as a fourth possibility, we sought to perform a more realistic two dimensional integration of our set of active gel equations taking into account the geometric parameters of the ingressing region, in order to make more refined predictions on the localisation of the actomyosin accumulation. In particular, we wished to examine whether simulations can robustly reproduce the presence of the accumulation at the base of the ingressing AJ. This would resonate with *in vitro* experiments, which have suggested that the geometrical architecture of the cortex can determine MyoII activity and resulting steady-state configuration[111]. As the flows of F-Actin are restricted to the plane of the AJ (Extended Data Fig. 9e-g and Supplementary Video 7e), we do not consider three-dimensional movements in the model.

Position of the problem

To simplify the analysis, we adopt a quasi-static approach, where the geometry of the ingressing region is fixed in time, and investigate the steady-state configuration for different amounts of ingression. As we saw earlier in the one-dimensional model, the AJ advancement helps to localise the accumulation at the base of the ingressing AJ. However, we wish here to examine whether this feature can arise from simpler geometrical considerations. We parametrise the ingressing region by the height function $h(x) = w_0 + h_0 exp(-x^4/d_0^4)$, h_0 being the maximal length of the ingressing region, d_0 its characteristic width, and w_0 the typical thickness of the cortex.

In order to solve our system of equation, we use a classical finite element method (with the Freefem++ software, code provided as a Supplementary File). We consider a two-dimensional domain, defining as before l the rescaled initial junction length:

$$\Omega = \{(x,y) \in \mathbb{R}^2 : \{y \in [0,h(x)], x \in [l/2, l/2]\}\}$$

on which we solve the non-dimensional conservation equation and force balance

$$\left.\begin{aligned} \partial_t \rho &= -\alpha^{-1}\partial_x(\rho \mathbf{v}) + \alpha^{-2}\Delta\rho - \phi(\rho - 1) \\ 0 &= \chi\alpha^{-1}\nabla\rho + \alpha^{-2}(\Delta\mathbf{v} + \nabla(\nabla.\mathbf{v})) \end{aligned}\right\} \text{in } \Omega$$

as well as the clamped and no normal (along vector **n**) flux boundary conditions.

$$\left.\begin{aligned} \mathbf{v}.\mathbf{n} &= 0 \\ \partial\rho/\partial n &= 0 \end{aligned}\right\} \text{on } \partial\Omega$$

Additionally, friction now enters as a surface, rather than bulk force, therefore setting a boundary condition for the tangential stress:

$$(\sigma.\mathbf{n}).\mathbf{t} = \xi \mathbf{v}.\mathbf{t} \text{ on } \partial\Omega$$

with σ the stress tensor and **t** the vector tangential to Γ. To guaranty the stability of the numerical scheme, we use a very small time step compared to the turnover time $dt \ll 1$ (in practise, we used $dt = 10^{-3}$), and an adaptive meshing, to concentrate more accurately on the sharp variations of ρ (Fig. 4a for typical initial mesh). Finally, we monitor the second moment of the density distribution ρ in time, to make sure the profiles we report are at steady state. One should note that we assume the bulk viscosity η_b and shear viscosity η_s to be equal.

Finally, we need to define an non-homogenous friction force at the boundary at the tissue. We defined three boundaries: Γ_m the interface with the medial pool ($y = 0$), $\Gamma_0 = \{(x,y) \in \mathbb{R}^2 : \{y = h(x), x \in [-2d_0, 2d_0]\}\}$ the interface with the dividing cell with low friction (lining the ingressing AJ) and Γ_d the rest of the interface with the dividing cell, together with the left and right borders (Fig. 4a). Therefore $\Gamma = \partial\Omega = \Gamma_m \cup \Gamma_0 \cup \Gamma_d$ and $\Gamma_m \cap \Gamma_0 \cap \Gamma_d = \emptyset$. We thus consider a very high friction $\xi \to \infty$ on Γ_d (i.e. no slip boundary condition at the E-Cad-rich AJ, whereas the friction Γ_0 is low $\xi = \xi_{low}$ on Γ_d (ingressing region).

Moreover, as our aim is to model only the cortical region close to the membrane, we assume that the turnover kinetics of MyoII (and all other coefficients apart from friction) are uniform in space and time. Clearly, this is an approximation, as the presence of a detached cortex could imply enhanced polymerization at the base of the ingressing region. As discussed in the one-dimensional model (subsection 3.3.1), this would further enhance the localisation of actomyosin at the base of the ingressing AJ. Nevertheless, in order to verify that actomyosin accumulation can form at the correct location without the need for such cues, we neglect it.

This two-dimensional approach leaves open the question of the exact nature and contribution of the medial pool: indeed, taking it into account would require adding parameters for its turnover, viscosity, diffusion etc, which would increase significantly the parameter space of the problem, without giving rise to qualitatively different physics. As this is largely out of the scope of this study, and we have experimentally seen that the medial pool did not contribute significantly to the MyoII accumulation (Fig. 4d,e,g,h, Extended Data Fig. 9a,b and Supplementary Videos 7a,7c,7d), we restrict the integration domain to a small domain of width w_0 below the AJ, in order to consider only the vicinity of the cortical region. We consider that the cortex is in frictional contact with the medial pool, although we expect the friction to be lower than the one at the AJ. We then performed simulations for various values of both ξ_{low} on Γ_0 and ξ_0 on Γ_m, and saw that they only had a very weak effect on the results. In the data shown in Extended Data Fig. 8i-o, we use $\xi_{low} = \xi_0 = 0.1$. Further studies will be necessary to model the medial pool in more detail, in order to understand whether and how this actomyosin network contributes for MyoII accumulation in the neighbours. Although some actomyosin speckles coming from the medial pool do integrate the accumulation, these flows are not as prominent as one might expect. One possibility would be that the contractility of the medial pool is lower than observed for junctional actomyosin. Alternatively, the architecture and orientation of the actomyosin network, two features that influence heavily its contractility[111], could be such that they prevent prominent medial flows. Accordingly, the orientated filaments in the accumulation could act as an anchor against medial flows. In order to discriminate between these two possibilities, higher resolution analysis of the dense microscopic structure of the actomyosin network within the accumulation is required, via correlative electron microscopy for example.

Finally, in order to analyse whether our findings are general, we use two different boundary conditions at the interface with the medial pool ($y = 0$): either the no-flux (Neumann) boundary condition described above $\partial_y \rho(x,0,t) = 0$, or a Dirichlet boundary condition $\rho(x,0,t) = \rho_0$, which could arise from an intrinsic regulation of actomyosin density at the interface with the medial pool. We also use again the non-dimensional ratios $\phi = 1$ and $\alpha = 10$, and the non-dimensional geometric factors $d_0 = 2w_0 = 1$, and $h_0 \in [0, 2.5]$.

Simulation results and experimental tests

First, we verify, for the sake of consistency, that if contractility χ is lower than the threshold $\chi_2^c = \eta/\tau$, (i.e. the flow threshold in the regions of zero friction), an homogeneous distribution of actomyosin is observed. Similarly, as an important control of the consistency of our numerical simulation, we tested that if friction is uniformly high everywhere, no accumulation takes place even for $\chi > \chi_2^c$ (provided of course contractility is below the global instability threshold with friction χ_1^c).

Next, we performed two-dimensional numerical simulations where we considered a non-uniform friction on the domains Ω_p and Ω_m. Starting from random initial conditions, we found a consistent MyoII accumulation at the base of the ingressing region (Fig. 4b and Extended Data Fig. 8i), mirroring the experimental data. As expected from the analytical expression above, we found that above a contractility χ_2^c, actomyosin flows formed and after a transient phase, created a gradient of MyoII concentration from the top to the base of the ingressing region. We did find some transient phases of actomyosin accumulation at the tips for some initial conditions, but these were unstable, with the steady-state accumulation re-localising at the base of the ingressing region. After such processes of relocalization, MyoII accumulation at the base was always the steady, stable state of the system. The vast majority of the MyoII feeding the accumulation came from the ingressing region, not from the neighbouring cortex, as shown by the fact that the horizontal velocity (Fig.4c and Extended Data Fig. 8j) v_x, in the direction of the cortex, was an order of magnitude smaller than the vertical velocity (Extended

Data Fig. 8k) v_y, in the direction of the ingressing region. This is because of the high friction, which prevents flows along the attached cortex from being long-ranged. Note that Extended Data Fig. 8i-m shows results for the boundary condition $\rho(x,0,t) = \rho_0$, while Extended Data Fig. 8n,o shows results for the boundary condition $\partial_y \rho(x,0,t) = 0$.

Moreover, the concentration of MyoII in the accumulation, as well as the magnitude of the flows, increased with the length of the ingressing region, at constant contractility (we show in Extended Data Fig. 8l respectively $h_0 = 0.75$, $h_0 = 1.25$, $h_0 = 2$ and $h_0 = 2.5$). In particular, the accumulation does not occur until a minimum length of the protrusion, dependent on the value of contractility, is reached. Importantly, we tested this prediction by analysing the frequency of F-Actin speckles as a function of the percentage of constriction of the dividing cell, and found that speckles could only be observed for constrictions larger than 35%, and that their frequency indeed increased with increasing constriction (Fig. 4g,h, Extended Data Fig. 9d and Supplementary Video 7c).

Similarly, the concentration of MyoII in the accumulation, as well as the magnitude of the flows, increased with contractility (we show in Extended Data Fig. 8m respectively $\chi = 0.9\chi^c$, $\chi = 1.1\chi^c$, $\chi = 1.7\chi^c$ and $\chi = 2.6\chi^c$), thereby confirming our previous conclusions from the one-dimensional simple criteria derived above. Note that Fig. 4a-c and Extended Data Fig. 8i-k,n-o shows results of simulations taking a length of ingressing region of $h_0 = 2$ and $\chi = 2\chi_c$. We confirmed experimentally this prediction by showing that loss of *rok* in the neighbouring cells (Fig. 4m-p, Extended Data Fig. 5p-r and Supplementary Video 8b) drastically reduced Actin flow velocity in the ingressing membrane and MyoII accumulation at the base. Importantly, this is unlikely due to a potential, indirect, effect of Rok on F-Actin polymerization, as we found that the ratio of F-Actin in the ingressing region and the medial pool (normalized by the Actin intensity at the remaining AJs) was not affected by loss of *rok* function (Extended Data Fig. 9j,k). Moreover, Rok targets that regulate F-actin dynamics, such as Shroom and FHOD1, are not essential for MyoII accumulation in the neighbours (Extended Data Fig. 7o-q and Extended Data Fig. 9l-n).

Finally, based on our theory, we would not expect a major contribution of F-Actin polymerization to the MyoII accumulation (as stated above, Actin serves as a substrate transporting MyoII, although it could have a secondary feedback effect on the value of contractility). As mentioned in the subsections above, we performed a thorough chracterization of the role of all *Drosophila* Formins, as well as Arp2/3, Enabled and Profilin (Extended Data Fig. 7i-r and Extended Data Fig. 8a,b). We found that none of these Actin nucleators abrogate MyoII accumulation in the neighbours and that only a drastic reduction of F-Actin nucleation in a triple *dia*, *arp2/3*, *profilin* mutant can abrogate MyoII accumulation (Extended Data Fig. 8a,b). This would correspond to the limit in which our theory, treating F-Actin as a substrate in large excess, would not be valid anymore.

For the Neumann boundary conditions, the results are qualitatively unchanged, although the peak of the accumulation forms at the boundary $y = 0$ (Extended Data Fig. 8n for $\chi = 1.2\chi^c$, $h_0 = 2$). On the other hand, if we assume that friction is low everywhere, for the same values of contractility and ingressing region height, MyoII can still accumulate via the formation of spontaneous flows, but the localisation of the accumulation becomes non-stereotypical, with several solutions coexisting for a given parameter set (example plotted on Extended Data Fig. 8o for $\chi = 1.2\chi^c$, $h_0 = 2$ with Neumann boundary conditions).

Therefore, our modelling of the actomyosin cortex as an active contractile gel is consistent with the idea that a local modification in its physical properties can, in the absence of any specific spatio-temporal cue in contractility or polymerization, create spontaneous actomyosin flows. In this Supplementary Note, we have explored the possibility that a local decrease in friction, due to the decrease in concentration of linker proteins such as E-Cad, both triggers and guides self-maintained actomyosin flows. Moreover, we show that, in the absence

of friction, the condition for flows to arise is fulfilled *in vivo*, and predict the correct order of magnitude for the velocity of such actomyosin flows. These actomyosin flows transmit the proliferative forces from the dividing cell to its neighbours, and serve as a previously unreported mode of mechano-sensing during cytokinesis.

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