

Cell Divisions Imprint Long Lasting Elastic Strain Fields in Epithelial Tissues

Ali Tahaei ¹, Romina Piscitello-Gómez,² S. Suganthan ¹, Greta Cwikla ³, Jana F. Fuhrmann ^{2,3},
Natalie A. Dye ^{2,3} and Marko Popović ^{1,3,4}

¹Max Planck Institute for the Physics of Complex Systems, Nöthnitzer Strasse 38, 01187 Dresden, Germany

²Max Planck Institute for Molecular Cell Biology and Genetics, Pfotenhauerstrasse 108, 01307 Dresden, Germany

³Excellence Cluster, Physics of Life, Technische Universität Dresden, Arnoldstrasse 18, 01307 Dresden, Germany

⁴Center for Systems Biology Dresden, Pfotenhauerstrasse 108, 01307 Dresden, Germany



(Received 23 August 2024; accepted 16 September 2025; published 5 November 2025)

A hallmark of biological tissues, viewed as complex cellular materials, is the active generation of mechanical stresses by cellular processes, such as cell divisions. Each cellular event generates a force dipole that deforms the surrounding tissue. Therefore, a quantitative description of these force dipoles, and their consequences on tissue mechanics, is one of the central problems in understanding the overall tissue mechanics. In this work, we analyze previously published experimental data on fruit fly *Drosophila melanogaster* wing epithelia to quantitatively describe the deformation fields induced by a cell-scale force dipole. We find that the measured deformation field can be explained by a simple model of wing epithelium as a linearly elastic sheet. This allows us to infer the magnitude and dynamics of the mechanical forces generated by the cell divisions. In particular, we find that cell divisions exert a transient isotropic force dipole field, corresponding to the temporary localization of the cell nucleus to the tissue surface during the division, and traceless-symmetric force dipole field that remains detectable from the tissue strain field for up to about 3.5 h after the division. This is the timescale on which elastic strains are erased by other mechanical processes and therefore it corresponds to the tissue fluidization timescale. In summary, we have developed a method to infer force dipoles induced by cell divisions, by observing the strain field in the surrounding tissues. Using this method we quantitatively characterize mechanical forces generated during a cell division and their effects on the tissue mechanics.

DOI: [10.1103/lh3v-v2c9](https://doi.org/10.1103/lh3v-v2c9)

I. INTRODUCTION

How mechanical forces influence biological tissues is one of the central problems in animal development and regeneration [1–5]. Biological tissues are often described as soft viscoelastic materials [6] that are elastic on sufficiently short timescales. Tissue fluidization on longer timescales arises from cell divisions, extrusions, and intercalations, which restructure the tissue over time [7–10].

Cellular processes, such as cell divisions and cell extrusions, are intrinsic sources of mechanical force generation within tissues, inducing deformation in the surrounding tissue [11–13], which can in turn lead to cellular rearrangements [14] and contribute to the overall tissue flow [7,15,16]. Furthermore, cell divisions have been indicated to control the glassy cell dynamics observed in cultured epithelial tissues [17,18] and explanted embryonic tissues [19]. However, despite the fact that cell divisions are a hallmark of biological matter and seem to be a major factor in controlling material properties of biological tissues, a quantitative understanding of the mechanical forces generated by cell divisions and their

immediate consequences on the surrounding tissue is still lacking.

To study the mechanics of cell divisions, we need to probe mechanical stresses in the surrounding tissue. One of the most common methods of probing mechanical stresses is the laser ablation experiment, in which one or multiple cells are destroyed by a laser. The resulting displacement of the ablated tissue boundary reflects the prestresses existing in the tissue before the ablation [8,20–24]. Prestresses are then characterized either by the initial dynamics of tissue boundary displacement [21,23] or from the final relaxed shape of the tissue [20,22,24,25]. Previous analyses focused on changes in the position or shape of specific points or outlines in the vicinity of the ablation. Therefore, they could not provide an insight into the strain field induced in the surrounding tissue, and they had to rely on assumptions about tissue mechanical properties. Furthermore, to extract information about all components of the prestress in the tissue, previous methods require a precise, circular laser ablation [22,25]. If we could instead infer force dipoles induced by cellular processes such as cell divisions, we would be able to extract information about tissue mechanics in a completely noninvasive way. Therefore, the starting point of our work, on the way to study cell division mechanics, is to develop a method that allows us to infer the force dipole in an epithelial tissue from the tissue strain field. The idea to infer properties of a localized force field from the strain field it induces was recently employed to characterize the properties of the core of plastic events in computer glasses

[26–28]. Here, we proceed independently of these results using an approach better suited for epithelial tissue data.

In this work, we first provide a detailed quantitative analysis of the tissue displacement field following a linear laser ablation performed in the pupal wing epithelium of the fruit fly *Drosophila melanogaster*. We show that the response of the fly wing epithelium to a laser ablation is consistent with that of a two-dimensional (2D) linear elastic sheet. This allows us to infer the force dipole induced by the ablation, normalized by the elastic constant, from the observed strain field. Furthermore, by analyzing the dynamics of the strain field, we show that the dominant mode of dissipation in the fly wing epithelium on short timescales less than 1 min is viscous dissipation, as opposed to frictional dissipation with a substrate.

Using our method to infer the force dipole from its strain field, we determine the force dipole generated by cell divisions in the *D. melanogaster* wing disc. We find that the isotropic component of the cell division force dipole tensor is only transient and vanishes almost immediately after the division, indicating that no net cell growth is accumulated during the cell division. Furthermore, we find that the traceless-symmetric component of the force dipole, imprinted by the cell division, remains visible in the surrounding tissue for up to about 3.5 h after the division. This provides a measurement of the fluidization timescale in the tissue and demonstrates the importance of elastic interactions in tissue-scale morphogenetic processes that take place on a comparable timescale, such as wing disc eversion [29].

II. THE FRUIT FLY WING EPITHELIUM IS A 2D LINEAR ELASTIC SHEET

A. Linear laser ablation experiments

In linear laser ablation experiments, a small linear segment of tissue is destroyed by a laser. In Ref. [24], laser ablation experiments have been performed in the *D. melanogaster* wing epithelium during pupal morphogenesis. Information about the prestresses in the tissue was inferred from the dynamics of the nearest cell-cell junction as it retracted away from the ablation. Here, we use laser ablation experiment data published in Ref. [24] to study the deformation field in the tissue surrounding the laser ablation. An image of a wing tissue before and after such a laser ablation experiment is shown in Fig. 1(a.i), where the red line indicates the line ablated by the laser. Ablations were performed in a plane thinner than 1 μm on the apical surface of the tissue [24]. Following the ablation, the tissue relaxes until reaching a mechanical equilibrium after about $T_e \approx 30$ s after ablation. We show an example of the relaxed state in Fig. 1(a.ii). The tissue remains in this state until after around 1 min. After this time, the tissue starts to exhibit additional flows, directed towards the line of ablation, which likely correspond to the wound-healing response of the tissue [see Sec. V and Supplemental Material (SM) [30]]. Note that in most experiments from Ref. [24] we could not segment and analyze a reasonably large circle of cells around the ablation (SM Fig. 1 [30]), and we selected four experiments that we could best analyze. In the main text we present analysis of the clearest one, denoted experiment

1. In experiment 2, such flows appear earlier so that no intermediate relaxed state can be identified. In experiments 3 and 4 the strain field generated by the ablation is also weaker than in experiment 1, so that the noise overcomes the signal already at small distances from the ablation. Analyses of these three experiments are presented in the SM [30].

To measure the tissue strain field, we segment the apical cell surfaces over time and quantify area a and the elongation tensor Q_{ij} of each cell. The cell elongation tensor is defined such that the corotational changes in the elongation tensor Q_{ij} correspond to the accumulated pure shear strain [8,31]. Since no cell divisions or rearrangements appear in the region of interest following the laser ablation, we quantify the tissue strain field $U_{ij}(\mathbf{r}, t)$ from the change in cell shape and size relative to their values before the ablation (see Sec. V). The strain field tensor consists of the isotropic strain component U_{kk} and traceless-symmetric component $\tilde{U}_{ij}$ which we denote as the pure shear strain component. In Fig. 1(a.iv) we show the pure shear strain field component $\tilde{U}_{xy}$ at 50 s after the laser ablation, which exhibits a clear spatial pattern. See SM Fig. 3(a) [30] for the other traceless-symmetric component and the isotropic component.

Our goal is to find the relationships between the induced strain field and the stresses induced by the ablation. Since the process of ablation does not introduce any net force, the dominant component of the induced force field will be dipolar. Therefore, we aim to relate components of the strain field at distance $\vec{r}$ from the ablation to the components of the force dipole tensor, as illustrated in Figs. 1(b.i) and 1(b.ii).

We first consider tissue strain after it has reached the mechanical equilibrium after the time T_e and calculate the discrete angular Fourier spectrum $\tilde{U}_{ij}^{ss}(r, m)$, where $r = |\mathbf{r}|$ is the distance from the center of ablation and m is the angular mode index (see Sec. V). We find the strongest signal in the second and the fourth mode of the pure shear strain components [see Fig. 1(c.i) and SM Fig. 3(bii) [30]], which correspond to $\cos(2\varphi)$ and $\cos(4\varphi)$ angular Fourier modes. In the isotropic strain component, we find the strongest signal in the zeroth and second mode [see SM Fig. 3(b.i)], which correspond to the constant and $\cos(2\varphi)$ angular Fourier modes. We then quantify the radial dependence of the second angular mode and we find that its radial profile is consistent with r^{-2} , as shown in Fig. 1(c.ii). These results reminded us of the far field of Eshelby propagators in two-dimensional linear elastic sheets [26,32,33], and we next tested whether the linear elastic theory can indeed quantitatively account for our measurements.

We briefly present the linear elastic theory of a sheet in which a point force dipole has been inserted. A force dipole is represented by a tensor D_{ij} that can be decomposed into isotropic and traceless-symmetric contributions $D_{ij} = D_0\delta_{ij} + \tilde{D}_{ij}$ [34]. Inserting a force dipole in an elastic sheet induces a strain field that is determined by the force balance equation

$$\partial_j \sigma_{ij} + \partial_j (\delta(\mathbf{r}) D_{ij}) = 0 \quad (1)$$

where σ_{ij} is the stress tensor in the elastic sheet, which can be decomposed into pressure P and shear stress $\tilde{\sigma}$: $\sigma_{ij} =$

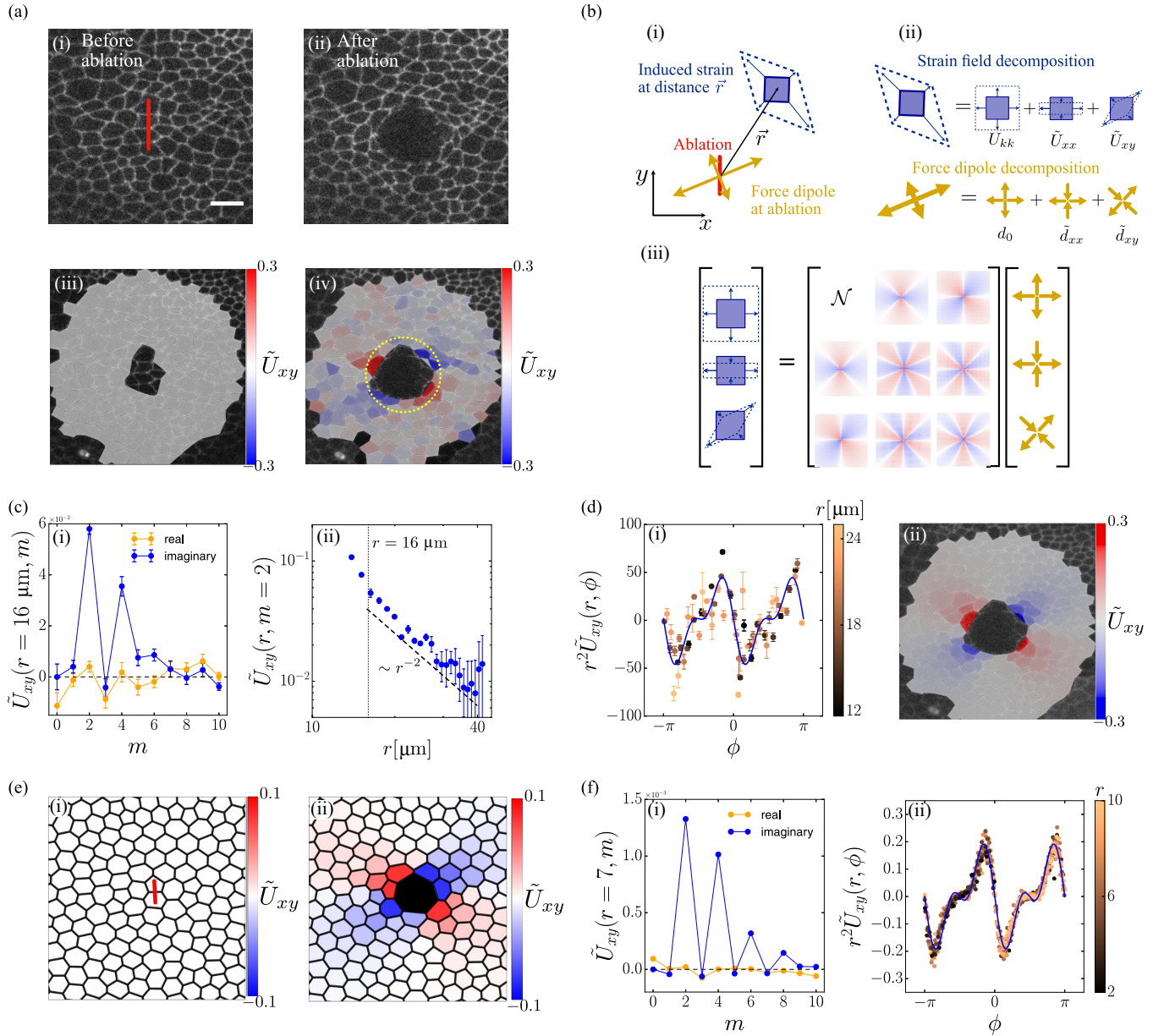


FIG. 1. (a.i) Fluorescently labeled cell outlines show the cellular packing before the laser ablation. The red bar indicates the location that will be ablated. (a.ii) Cell outlines 50 s after the laser ablation. (a.iii) Tracked and segmented cells in the region of interest are colored in white. (a.iv) The accumulated cell strain component $\tilde{U}_{xy}$, 50 s after the laser ablation. The scale bar is 10 μm . (b.i) A force dipole in a two-dimensional linear elastic system induces a strain in distance $\vec{r}$. (b.ii) Decomposition of two-dimensional strain field and force dipole tensors into isotropic and traceless-symmetric components. (b.iii) A Voigt representation of the relation between strain at position $\vec{r}$ induced by a force dipole at the origin. (c.i) Angular Fourier modes of $\tilde{U}_{xy}$ at distance $r = 16 \mu\text{m}$, which corresponds to the yellow circle (a.iv), show a clear signal in modes $m = 2$ and 4. (c.ii) The radial profile of the $m = 2$ angular mode shows a radial profile consistent with r^{-2} (dashed line). The dotted line indicates the distance $r = 16 \mu\text{m}$. Analysis in both (c.i) and (c.ii) is performed on the strain field averaged over the time interval from 30 to 70 s after laser ablation and the error bars show the standard deviation over this interval. (d.i) The fit of the linear elastic theory to the experimental data, shown in (c). The solid blue line is the fitted strain field with the corresponding fit parameters reported in the text. (d.ii) Visualization of the fitted strain field. (e.i) The cellular network in the vertex model simulation. The red bar indicates two cells removed by ablation. (e.ii) The cell strain field accumulated in the tissue after reaching the steady state following the simulated ablation. (f.i) Angular Fourier modes of the strain field pattern in (e.i) showing peaks in the second and fourth modes. (f.ii) The fit of the linear elastic theory to the cell strain field in the vertex model. The solid blue is the fitted strain field with the fit parameters $d_0 = 0.72 \pm 0.07A_0$, $\vec{d}_{xx} = 0.65 \pm 0.07A_0$, $\vec{d}_{yy} = 0.00 \pm 0.07A_0$, and $\mu = 1.3 \pm 0.1$, where A_0 is the cell preferred area in the model, see SM. The uncertainty intervals for fit parameters are obtained by bootstrapping the data (see Sec. V).

$-P\delta_{ij} + \tilde{\sigma}_{ij}$. The resulting strain field can be written as

$$u_{ij}(\mathbf{r}) = G_{ijkl}(\mathbf{r})D_{kl}, \quad (2)$$

where G_{ijkl} is the elastic dipole propagator and $\mathbf{r}$ is the distance from the force dipole (see SM [30]). We graphically illustrate the components of Eq. (2) in Fig. 1(b.iii), using the Voigt tensor representation. In this representation, the force dipole propagator elements are organized in a matrix, which we plot in Fig. 1(b). From this point, we use the normalized force dipole defined as $d_{ij} = D_{ij}/2K$. The propagator elements depend only on $\cos(2\varphi)$, $\sin(2\varphi)$, $\cos(4\varphi)$, and $\sin(4\varphi)$ [see Fig. 1(b.iii) and SM], consistent with the observed second and fourth angular Fourier modes in the data. Furthermore, all components of G_{ijkl} decay in space as r^{-2} . This is consistent with the radial profile observed in the experimental data for $m = 2$ modes [see Fig. 1(c.ii) and SM Fig. 5]. The radial profiles of $m = 4$ modes are more noisy so that it is difficult to characterize the radial decay.

Therefore, the strain field measured in the laser ablation experiments is consistent with a linear elastic response of the tissue to the force dipoles generated by the ablation. However, to confirm this hypothesis, we need to show that linear elastic theory can quantitatively account for the observed strain fields.

We fit the steady state tissue strain field U_{ij}^{ss} with a strain field predicted by a linear elastic theory of a two-dimensional elastic sheet in Eq. (2). The fitting parameters are the three independent components of the force dipole tensor and the ratio μ of the two-dimensional shear and bulk elastic moduli. By analyzing the ablation experiment, we find that the data are well described by the fit, as shown in Fig. 1(d) and SM Fig. 6(a) [30]. The fitted parameter values are $d_0 = 160 \pm 10 \mu\text{m}^2$, $\tilde{d}_{xx} = 150 \pm 10 \mu\text{m}^2$, $\tilde{d}_{xy} = 20 \pm 10 \mu\text{m}^2$, and $\mu = 1.9 \pm 0.1$ (see Sec. V). Furthermore, the ratio $\mu \approx 2$, corresponding to the Poisson ratio of $\nu \approx -0.33$, is comparable with the value $\mu \approx 3$, corresponding to the Poisson ratio of $\nu \approx -0.5$, recently reported in the same tissue using a laser ablation method based on circular ablation shape [24].

To further test our analysis of the laser ablation experiments, we simulate a linear ablation in the vertex model of epithelial tissues [20]. We remove two cells from a prestressed cellular network, where the prestress represents the proximal-distal stress in the wing epithelium [8], and the ablation axis is perpendicular to the prestress orientation, recapitulating the orientation of ablation in experiments (see SM [30] for details, which includes Ref. [35]). We then quantify the resulting strain and find that the simulation shows the same pattern as in experiments, consistent with the linear elastic theory of a 2D sheet [Figs. 1(e) and 1(f) and SM Fig. 7 [30]]. We fit the linear elastic theory to the observed strain field pattern and infer the force density generated by the ablation from tissue deformation. Finally, we find Poisson's ratio of the vertex model cellular network to be $\nu = -0.13 \pm 0.04$. This finding is consistent with the value $\nu = -0.095 \pm 0.009$ found by a direct numerical measurement of the shear and bulk modulus of the same network (see SM [30]).

Our results show that the response of the *D. melanogaster* wing epithelium to a small linear laser ablation is consistent with that of a linearly elastic sheet with a negative two-dimensional Poisson ratio. This allows us to infer the force

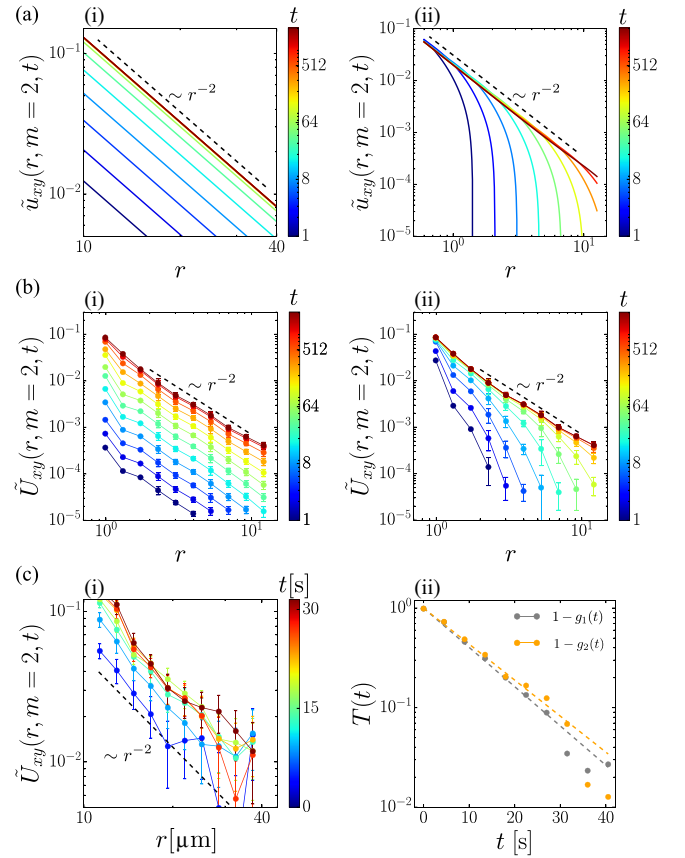


FIG. 2. Time evolution of the second Fourier mode $m = 2$ of the strain field xy component induced by a force dipole for a linear elastic sheet with (a.i) viscous dissipation and (a.ii) frictional dissipation and a vertex model simulation with (b.i) viscous dissipation and (b.ii) frictional dissipation. (c) Laser ablation experiment. (c.i) Time evolution of $\tilde{U}_{xy}$ shows a radial profile as r^{-2} , consistent with a viscous dissipation mechanism. (c.ii) Quantifying the viscous relaxation of strain as described in the text, which allows the measurement of viscous relaxation timescales. The black dashed line indicates the power-law r^{-2} decay for reference. Each data point represents the strain field average over five smaller bins and the errors are the corresponding standard deviations over those bins.

dipole tensor induced by the ablation from the tissue strain field, which we confirmed using the vertex model simulations.

B. Viscous dissipation determines tissue relaxation dynamics

Our analysis of the strain in the ablated tissue after it has relaxed allows us to study its elastic properties. Now, we explore the transient dynamics of tissue strain as it approaches the mechanical equilibrium, which will allow us to study dissipative processes in fly wing epithelium. We consider two possible dissipation mechanisms: viscosity and friction with a substrate.

Interestingly, in a continuum model of tissue as an elastic sheet, these two dissipation mechanisms lead to qualitatively different relaxation dynamics upon insertion of a force dipole. In particular, in an elastic sheet with only viscous dissipation, all strain field components decay as $1/r^2$ with the distance from the force dipole throughout the relaxation with the

amplitude evolving in time [see SM [30] and Fig. 2(a.i)]. However, in an elastic sheet with only frictional dissipation, the strain field propagates through the sheet: at distances within the propagation front, the strain field converges to the steady state solution and rapidly decays beyond the front [see SM and Fig. 2(a.ii)].

To compare the strain field dynamics in viscous and frictional dissipation models within a cellular system, we performed vertex model simulations of laser ablation that include only the frictional dissipation and compare it with a viscous dissipation (see SM [30] for details). As discussed above, the signature difference between viscous and frictional dissipation is the power law decay of the shape $1/r^2$ in the former and the existence of a propagation front in the latter. With a frictional dissipation, we indeed find a clear propagation of the strain field that converges to the steady state value as the propagation front expands [Fig. 2(b.ii) and SM Fig. 12(b)], consistent with the analytical calculation [Fig. 2(a.ii)]. In contrast, with viscous dissipation, we observe that the strain field decays as $1/r^2$ in all time points [Fig. 2(b.i) and SM Fig. 12(a)].

To quantify the dynamics of tissue relaxation following the laser ablation, we measure the strain field in time intervals of 4.5 s, starting from 1 s after the ablation (see SM Fig. 8 and movies 1–3 [30]). We find that the angular strain pattern is the same as in the mechanical equilibrium one. We analyze the dynamics of the strain radial profile by calculating the second angular Fourier mode of the strain field at all measured time points [see Fig. 2(c.i) and SM Fig. 8 [30]]. We find that the strain field is consistent with $1/r^2$ decay and we do not see any signature of a propagating front (see Fig. 2). Therefore, we conclude that the viscosity is the dominant mode of dissipation in the fly wing epithelium on timescales below $T_e \approx 30$ s.

Our continuum model of an elastic sheet provides the dynamical strain propagator $G_{ij}(\mathbf{r}, t) = G_{ij}(\mathbf{r})T_{ij}(t)$, where no sum over repeated indices is performed. $G_{ij}(\mathbf{r})$ is the steady state propagator, and $T_{ij}(t)$ is the relaxation matrix the components of which contain two relaxation factors $g_1(t) = 1 - \exp(-t/\tau_1)$ and $g_2(t) = 1 - \exp(-t/\tau_2)$, where the two relaxation timescales

$$\tau_1 = \frac{\bar{\eta}}{K} \frac{1 + 2\eta/\bar{\eta}}{1 + \mu}, \quad (3)$$

$$\tau_2 = \frac{\eta}{K} \quad (4)$$

are related to the bulk and shear elastic constants, and bulk and shear viscosities $\bar{K}$, $2K$, $\bar{\eta}$, and 2η , respectively (see SM Sec. I.B.ii [30]). We extract τ_1 and τ_2 from experimental data (see Sec. V, SM, and SM Fig. 13 [30]). We find that the two relaxation timescales are very similar: $\tau_1 = 11 \pm 1$ s and $\tau_2 = 12 \pm 1$ s, where intervals of uncertainty correspond to the uncertainty of the fit (see Sec. V).

As mentioned above, experiment 1 presented here was selected based on our ability to segment and track cells in a radius of 40 μm surrounding the laser ablation, which was not possible for most of the other experiments. Here, we highlight that in experiment 2, the angular and radial pattern of the strain field are consistent with that of an elastic sheet with viscous dissipation on times comparable to the relaxation timescales τ_1 and τ_2 extracted above, as reported in the SM Fig. 13 [30].

Interestingly, on longer times radial profile of strain departs from r^{-2} and continues to evolve throughout the experiment. Similar additional motion is observed even in experiment 1 reported above, but only after about 2 min (see SM Fig. 11 [30]). This suggests that additional dynamics are due to a wound-healing response in the tissue and understanding this behavior would require a dedicated experimental exploration in the future.

In summary, our detailed analysis of the strain in the fly wing epithelium in response to the laser ablation shows that the tissue behaves as a two-dimensional viscoelastic solid. These results suggest that we can now infer force dipoles generated by cell divisions, and thereby study their mechanics, by simply observing the dynamics of the surrounding tissue strain caused by the division, without the need to directly perturb the tissue.

III. ELASTIC DEFORMATION IMPRINTED BY A CELL DIVISION

Cell division is a complex process during which the mother cell is split into two daughter cells. Here, we aim to characterize the mechanical forces generated by the dividing cell from the analysis of the strain field that the dividing cell induces in the epithelium. We analyze cell divisions in previously published experiments [25] on explanted *D. melanogaster* wing imaginal discs. We are interested in the generic features of the response of epithelial tissues to a mechanical perturbation, such as the laser ablation or a cell division. Therefore, we aim to extract normalized force dipoles in the surface plane generated by cell divisions using the same approach as with the laser ablation experiments. However, we perform the analysis independently of any results obtained from laser ablations in the pupal wing.

The wing disc developmental stage precedes the pupal stage in which linear laser ablations analyzed above were performed [24], and their mechanical moduli may be different. However, a quantification of the strain field generated by cell divisions is more convenient in the wing disc stage compared to the pupal wing stage. Cell divisions in the pupal wing differ from divisions in the wing disc in that they are patterned in time and space, with most cells dividing at least once within several hours, and the tissue exhibits strong tissue shear flows and active cell rearrangements [36]. Therefore, analysis of the strain field induced by individual divisions is practically impossible in the pupal wing due to additional dynamics in the tissue. However, in both tissues, the apical surfaces are mechanically special as they contain a high concentration of actomyosin cortex and adherens junctions between cells that are important for morphogenesis. Therefore, it is likely that apical mechanics plays a significant role in the overall tissue mechanics in both tissues.

Wing discs were cultured over about 13 h, during which the tissue was imaged every 5 min, and individual cells were segmented and tracked over time. The progress of a typical cell division is shown in Fig. 3(a). Before the actual cell division, the mother appears to inflate [Fig. 3(a.ii)]. This is not a real increase in cell size, but is rather a consequence of the cell nucleus arriving at the apical surface of the cell to divide and transiently stretching the cell apical surface

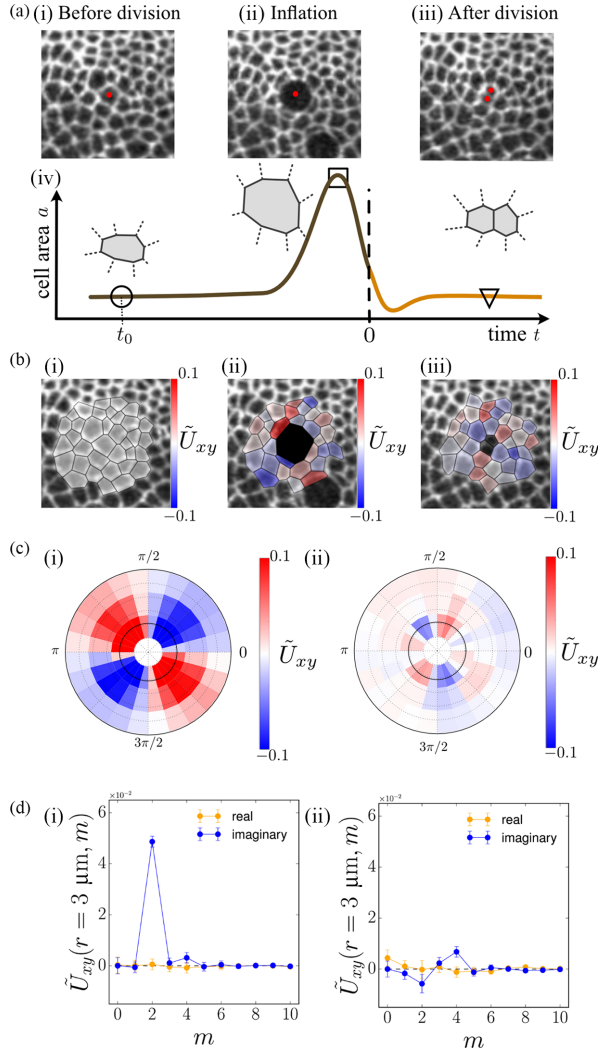


FIG. 3. (a) Snapshots of a cell division in three stages. (a.i) As the mother cell starts the division process, (a.ii) it rounds up, corresponding to the inflation, and then (a.iii) it shrinks and divides into the daughter cells. The red dots show the mother and the daughter cells. (a.iv) A schematic representation of the cell area of the mother cell (brown) and the daughter cells (orange) during the division process. The symbols highlight the stage of the dividing cell before the division, at the peak of inflation, and after the division. The new bond forms at $t = 0$, and t_0 is the reference state. (b) Strain $\tilde{U}_{xy}$ of the cells near a typical cell division at the three stages shown in (a) with $t_0 = -60$ min. (c) Ensemble average ($N = 682$) of $\tilde{U}_{xy}$ around the dividing cell: (c.i) at the peak of the inflation $t = -20$ min [corresponding to (a.ii) and (b.ii)] and (c.ii) after the division $t = 30$ min [corresponding to (a.iii) and (b.iii)]. The radial extent of the plots is $6 \mu\text{m}$. (d) The angular Fourier transform of the $\tilde{U}_{xy}$ at a distance of $r = 3 \mu\text{m}$ from the center of the mother cell at $t = -20$ min (d.i) and at $t = 30$ min (d.ii).

[11,37]. This transient inflation is followed by the creation of the new cell boundary, and the two daughter cells become visible [Fig. 3(a.iii)]. For each cell division, we define the time of the new bond creation as the reference cell division time $t = 0$.

Figure 3(a.iv) qualitatively summarizes the experimental observations. For each cell division, we quantify the tissue

strain field in the vicinity of the dividing cell, as described in Sec. II, relative to the starting time point $t_0 = -60$ min before the cell division time. We show an example of the cell strain field induced by a dividing cell in Fig. 3(b). The timescale on which the cell division forces evolve, which is on the order of tens of minutes, is much larger than the minutes relaxation timescale reported in the wing disc [25]. Therefore, the strain field dynamics observed over tens of minutes is largely due to changes of the force dipole generated by the cell division, and not due to much faster stress propagation through the tissue. Although it is possible that on these longer timescales cells adapt to the experienced stress and adapt their reference shape, a clear positive correlation between stress and strain has been reported in our past work [25]. Therefore, it is reasonable to interpret the change in cell elongation as a measure of elastic strain, even if the adaptation to stress effectively renormalizes the elastic constants, as discussed in Ref. [25].

We find that a single cell division generates weaker forces compared to those induced by laser ablation [compare Figs. 1(a) and 3(b)], and the corresponding strain field is more susceptible to noise. Therefore, we average the strain field from multiple cell divisions ($N = 682$). We first align the centers of the cell division, defined as the area-weighted average of daughter cell centers at the cell division time ($t = 0$). We next rotate each division such that the orientation of the line connecting daughter cell centers is oriented along the x -axis at the cell division time. We show the resulting strain field component $\tilde{U}_{xy}$ in Fig. 3(c) and the corresponding angular Fourier modes in Fig. 3(d) at times $t = -20$ min, corresponding to the peak of the cell inflation, and $t = 30$ min after the cell division. In Fig. 3(c.i), the twofold symmetry of the angular strain pattern indicates that the isotropic force dipole is dominant, consistent with the isotropic cell inflation. In Fig. 3(c.ii), the pattern is closer to a fourfold symmetric one, such as would be induced by an anisotropic force dipole. Finally, we find that radial profiles of the Fourier modes are consistent with r^{-2} decay (SM Fig. 15 [30]), as was the case with laser ablation experiments above.

We fit the linear elastic theory Eq. (2) to the experimentally measured strain field at each time point and extract the resulting force dipole $d_{ij}(t)$. Comparison between the fit and the data is shown for the xy component of the strain field at $t = -20$ and 30 min in Fig. 4(b). We find that the isotropic force dipole component d_0 rises sharply during the mother cell inflation and peaks at about $t = -20$ min [magenta line in Fig. 4(b)]. However, by the time of the new bond formation, the isotropic component has disappeared and remains negligible at later times. This result shows that cells do not noticeably grow during the division, but rather that the division reorganizes the available material into the two daughter cells. The traceless part of the force dipole $\tilde{d}_{xx}$ grows as the new bond is formed and persists in the tissue multiple hours after the division [green line in Fig. 4(b)], indicating that the division-induced strains remain imprinted in the tissue long after the division has been executed. Over time, $\tilde{d}_{xx}$ slowly decays while its fluctuations increase, representing the effect of other cell divisions and rearrangements that appear in the tissue. We estimate the tissue fluidization time from the decay of $\tilde{d}_{xx}$ in time by fitting an exponential function to it. We find

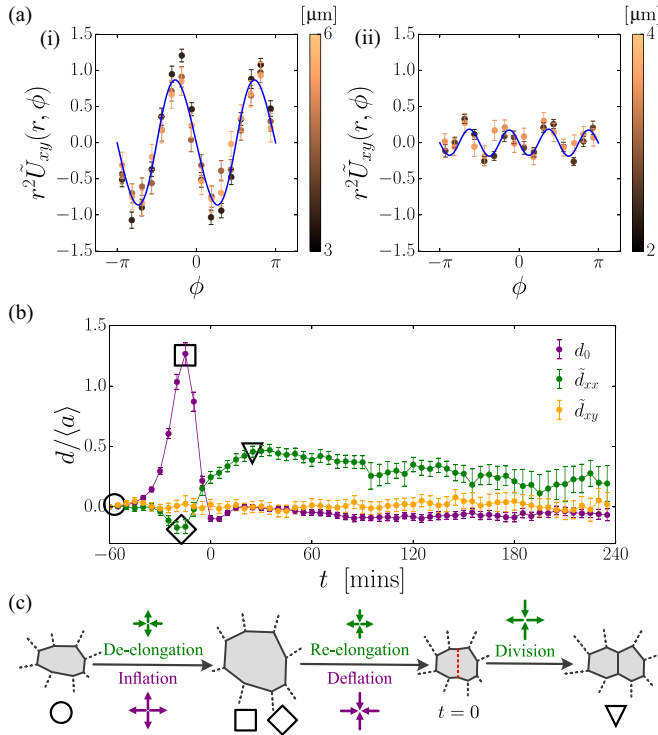


FIG. 4. (a) The fit of linear elastic theory to the measured strain field around cell divisions (a.i) at the peak of inflation $t = -20$ min and (a.ii) after division $t = 30$ min. Each data point shows the average strain field in polar bins and the error bars are the standard deviation over these bins. (b) The force dipole generated by a cell division as a function of time, normalized by the average cell area in the tissue. (c) A schematic summary of the force dipoles during stages of a cell division.

the fluidization timescale to be $\tau_\alpha \simeq 3.5 \pm 0.7$ h (see Sec. V), over which $\tilde{d}_{xx}$ vanishes and, therefore, the strain field induced by the division is erased. Note that component $\tilde{d}_{xy}$ remains negligible at all times.

Finally, we note that at the time of the peak of d_0 , there is a small but clear negative peak in $\tilde{d}_{xx}$. To interpret this observation, we have to remember that our strain measurements are relative to the state of the cells at $t_0 = -60$ min. Since the mother cell attains an almost perfectly circular shape during the inflation, any initial anisotropy of the mother cell at time t_0 gives rise to an anisotropic force dipole component during the inflation. Therefore, the observed negative value of $\tilde{d}_{xx}$ indicates that mother cells are elongated along the future division axis, consistent with the well-known Hertwig's rule [11,38–41]. We summarize these results in Fig. 4(c).

IV. DISCUSSION

In this work, we devised a full spatiotemporal analysis of tissue strain induced by two types of force dipole fields: linear laser ablations and cell divisions. In this way, we have shown that the developing *Drosophila* wing epithelium can be described as a two-dimensional elastic solid with viscous dissipation. We also measured its two-dimensional Poisson ratio, which we find to be negative, consistent with the values reported in the literature [24,25]. Our analysis was based on

laser ablation of the wing tissue during pupal development, at which time the wing consists of two apposed epithelial layers. Although the experimental observations are consistent with a model of the wing apical surface as a linear elastic sheet, in the future it might be interesting to probe the three-dimensional structure response of the tissue with deeper ablations and in wings where the two epithelial layers have been separated [42].

We characterized the dynamics of mechanical forces generated by a dividing cell throughout the cell division process. We found that the strain fields imprinted by a cell division are slowly erased on the timescale of $\tau_\alpha = 3.5 \pm 0.7$ h, which can be understood as a fluidization timescale of the *Drosophila* wing tissue. Furthermore, our analysis shows that there is no cell growth during a division, and stresses induced by the cell division correspond to those of a pure shear force dipole. Finally, we found that Hertwig's rule applies in the *D. melanogaster* wing disc [11,12].

An important implication of our work is that the *Drosophila* wing epithelium is a solid the fluidization time τ_α of which is not negligible on the developmental timescales. For example, large-scale shape changes that the wing undergoes during eversion are executed on a similar timescale [29]. A possible physical picture of the *Drosophila* wing epithelium that could account for our observations is that of a glassy material, or as was put forward in the glass literature, a solid that flows [43–45]. In other words, the long-range elastic interactions between cells would lead to correlated glassy dynamics, which has been reported in tissue cultures [17,18], and it remains to be seen whether they indeed play a role in developing tissues. Furthermore, active biological processes in the wing tissue make it an active glass which might exhibit mechanical properties distinct from passive ones [10,46–49]. In summary, our results quantify force dipoles generated by dividing cells in developing *D. melanogaster* wing epithelia, as well as elastic interactions between cells, which paves the way to the development of tissue mechanics theory and motivates further experimental study of collective cell dynamics.

V. METHODS

A. Cell segmentation and tracking

The linear ablation experiments were performed during the pupal morphogenesis stage and were previously published in Ref. [24], where the authors analyzed the initial retraction velocity of the ablated bonds. Here, we reanalyzed movies of those experiments, and fully segmented and tracked cells in the entire field using the Fiji plugin, TissueAnalyzer [50], using a bond cutoff of 2 pixels. Then, after the manual correction of segmentation errors, we use TissueMiner [36] to analyze changes in the cell shape and size.

The cell division experimental data came from the wing disc stage and were previously segmented, tracked, and analyzed in Refs. [25,51].

B. Tissue relaxation after a laser ablation

After the ablation, nearby tissue relaxes to a new mechanical equilibrium. In experiment 1 reported in the main text, we estimate equilibrium to be reached after about 30 s after

ablation, when the tissue does not visibly evolve any more. However, this equilibrium state is not permanent, as at about 1 min after the ablation, we observe additional motion of the tissue, which we interpret as the onset of the wound-healing response. In this work, we are interested in the stress response to the ablation, not the wound-healing process, and therefore we limit our analysis to times below 1 min after the laser ablation. Note that in experiments 2–4, reported in detail in SM [30], the additional patterns appear to emerge earlier and we could not extract as clear an intermediate regime as in experiment 1. However, even in these experiments the profiles of strain observed during the relaxation are consistent with the linear elastic theory, thereby further strengthening our results from experiment 1.

C. Tissue strain field measurement

The strain field consists of isotropic strain $U_{kk}(\mathbf{r}, t)/2$ and shear strain $\tilde{U}_{ij}(\mathbf{r}, t)$. To determine the strain field from the changes of cell shape and area, we use the method based on the cell triangulation [8,31,52] in which the corotational change of the cell elongation tensor Q_{ij} corresponds to the tissue shear rate, and changes in the logarithm of cell area correspond to the isotropic strain rate. Here, we approximate the accumulated tissue strain by the change in cell elongation tensor and in the logarithm of cell area for pure shear and isotropic components, respectively. To define the cell elongation tensor Q_{ij} , we choose the subcellular triangulation of the tissue obtained by connecting two adjacent vertices to the geometric center of cells, as described in Ref. [52]. We estimate the accumulated tissue shear strain field in a cell from the difference between the cell shape tensors:

$$\tilde{U}_{ij}(t) \simeq Q_{ij}(t) - Q_{ij}(0). \quad (5)$$

The only approximation is that we neglect to account for changes in cell elongation due to local rotations. We determined the exact cell shear tensor at several time points, and we found that the differences from our approximation are small. Therefore, we use the approximate expression for simplicity. The isotropic strain field in a cell is defined as the accumulated relative area change rate [8,31], which we measure relative to the initial cell state as

$$U_{kk}(t) = \ln \left(\frac{a(t)}{a(0)} \right), \quad (6)$$

where a is the cell area.

Finally, we define the tissue strain field $U_{ij}(\mathbf{r}, t)$ at position $\mathbf{r}$ to have the value of the strain tensor obtained for the cell inside of which point $\mathbf{r}$ lies.

D. Discrete angular Fourier transform

We examine the angular discrete Fourier transformation of the tissue strain field to investigate its angular pattern and study its radial profiles. The strain in position $\mathbf{r}$ is represented by $U_{ij}(r, \varphi)$, where r is the distance to the center of the cut and φ is the polar angle. We use the notation $U_{ij}(r, m)$ for the m th angular mode of discrete angular Fourier transform at a distance r , and it is defined as

$$U_{ij}(r, m) = \frac{1}{N} \sum_{i=1}^N U_{ij}(r, \varphi_i) e^{im\varphi_i}, \quad (7)$$

where φ_i are N uniformly distributed numbers between 0 and 2π . In rare cases a point with coordinates (r, φ_i) does not belong to a cell, making $U_{ij}(r, \varphi)$ undefined at that point. To handle such cases, we modify the Fourier transform as follows:

$$U_{ij}(r, m) = \frac{\sum_{i=1}^N U_{ij}(r, \varphi_i) e^{im\varphi_i} \iota(r, \varphi_i)}{\sum_{i=1}^N \iota(r, \varphi_i)}, \quad (8)$$

where $\iota(r, \varphi_i) = 1$ if the point belongs to a cell and zero otherwise.

For the analyses of cell division data, a point can belong to multiple cells due to simultaneous analysis of multiple cell divisions. In such cases, we replace $U_{ij}(r, \varphi_i)$ with its average $\langle U_{ij}(r, \varphi_i) \rangle$ where $\langle \dots \rangle$ is the average over all divisions.

E. Fit of the elastic theory to data

We measure the force dipole, the ratio of shear moduli to bulk moduli, and viscosity by fitting the strain field pattern to the elastic and viscoelastic theories. First, we define a cost function:

$$E = \sum_{\alpha \in \text{cells}} \{ [U_{kk}^\alpha - u_{kk}(\mathbf{r}^\alpha)]^2 + [\tilde{U}_{xx}^\alpha - \tilde{u}_{xx}(\mathbf{r}^\alpha)]^2 + [\tilde{U}_{xy}^\alpha - \tilde{u}_{xy}(\mathbf{r}^\alpha)]^2 \}, \quad (9)$$

where U_{ij}^α is the cell strain measured from data, $\mathbf{r}^\alpha$ is the position of the center of each cell, and $u_{ij}(\mathbf{r}^\alpha)$ is the strain field predicted from theory at position $\mathbf{r}^\alpha$. By minimizing E , we find the magnitude of the force dipole, the ratio of the shear modulus to bulk modulus, and when fitting the relaxation dynamics the relaxation timescales.

When fitting the steady state strain field measured in experiment 1, we wanted to take into account the fluctuations in the steady state. For this, we sampled the strain field at 5 time points uniformly spaced between 36 and 58.5 s after ablation. We constructed datasets by bootstrapping the sampled data ten times, and we fit the linear elastic theory for each dataset. The reported parameter values and uncertainties are mean values and standard deviations of these ten fit outcomes.

To fit the relaxation dynamics and extract the relaxation timescale, we consider the strain field at each time point t_i , spaced 4.5 s apart, during the relaxation and use the steady state parameters and force dipole to fit the elements of the matrix $\mathbf{T}(t_i)$. Finally, we fit an exponential function to the obtained matrix element values at different time points [Fig. 2(c.ii)] and report the relaxation timescale values and their uncertainties obtained by this exponential fit.

To analyze cell divisions, we first reduce noise in the strain field by applying a moving average over a 10-min window. Next, we select the time point $t = 30$ min and systematically vary the reference time t_0 from -120 to -40 min. For each t_0 within this range, we fit the steady state linear elastic theory to the measured strain field to extract the corresponding set of $\mu(t_0)$ values. The parameter μ is then determined as the average $\langle \mu(t_0) \rangle$, which remains fixed for the subsequent strain field analyses (see SM Fig. 14 [30]). This is done to more precisely measure the parameter μ , which cannot be extracted independently of d_0 when traceless symmetric components of the force dipole vanish. Namely, in that case it is impossible

to factorize the product μD_0 as the two factors only appear in the form of the product [see SM Eqs. (9) and (10) [30]]. Finally, to compute the force dipole at each time point t we set $t_0 = -60$ min, and we perform the fit at each time point using the bootstrapping algorithm over the ensemble of all divisions, obtaining the force dipole values and their uncertainties at each time point, while using the previously obtained values of μ as a constant.

F. Estimation of the tissue fluidization time

To measure the decay timescale of the anisotropic force dipole $\tilde{d}_{xx}$, we fit an exponential function to normalized force dipole $y = \tilde{d}_{xx}(t)/\langle a \rangle$ as $f(y) = \tilde{d}_{xx}(t=0) \exp[-t/\tau_\alpha]$ to observed data in SM Fig. 16 [30]. From that, we obtain the fluidization timescale $\tau_\alpha = 3.5 \pm 0.7$ h and $\tilde{d}_{xx}(t=0)/\langle a \rangle =$

0.53 ± 0.01 , normalized by the average cell area in the tissue before ablation. The reported errors correspond to the range of parameters over which an exponential function successfully fits the data.

ACKNOWLEDGMENTS

We would like to thank Michael Staddon, Frank Jülicher, and Jonas Neipel for useful discussions and comments. Funding for the biological experiments was supported by the Max Planck Society and the Deutsche Forschungsgemeinschaft (Cluster of Excellence Physics of Life, Grants No. EXC-2068-390729961 and No. SPP1782/EA4/10-2). N.A.D. additionally acknowledges funding from Deutsche Krebshilfe/MSNZ Dresden. M.P. and N.A.D. acknowledge funding by Deutsche Forschungsgemeinschaft Projects No. 544201605 DY180/2-1 and PO3023/2-1.

-
- [1] C.-P. Heisenberg and Y. Bellaïche, Forces in tissue morphogenesis and patterning, *Cell* **153**, 948 (2013).
 - [2] A. Brugués, E. Anon, V. Conte, J. H. Veldhuis, M. Gupta, J. Colombelli, J. J. Muñoz, G. W. Brodland, B. Ladoux, and X. Trepat, Forces driving epithelial wound healing, *Nat. Phys.* **10**, 683 (2014).
 - [3] L. LeGoff and T. Lecuit, Mechanical forces and growth in animal tissues, *Cold Spring Harbor Perspect. Biol.* **8**, a019232 (2016).
 - [4] F. Jülicher and S. Eaton, Emergence of tissue shape changes from collective cell behaviours, *Semin. Cell Dev. Biol.* **67**, 103 (2017).
 - [5] R. J. Tetley, M. F. Staddon, D. Heller, A. Hoppe, S. Banerjee, and Y. Mao, Tissue fluidity promotes epithelial wound healing, *Nat. Phys.* **15**, 1195 (2019).
 - [6] B. Aigouy, R. Farhadifar, D. B. Staple, A. Sagner, J.-C. Röper, F. Jülicher, and S. Eaton, Cell flow reorients the axis of planar polarity in the wing epithelium of *Drosophila*, *Cell* **142**, 773 (2010).
 - [7] J. Ranft, M. Basan, J. Elgeti, J.-F. Joanny, J. Prost, and F. Jülicher, Fluidization of tissues by cell division and apoptosis, *Proc. Natl. Acad. Sci. USA* **107**, 20863 (2010).
 - [8] R. Etournay, M. Popović, M. Merkel, A. Nandi, C. Blasse, B. Aigouy, H. Brandl, G. Myers, G. Salbreux, F. Jülicher *et al.*, Interplay of cell dynamics and epithelial tension during morphogenesis of the *Drosophila* pupal wing, *eLife* **4**, e07090 (2015).
 - [9] S. Curran, C. Strandkvist, J. Bathmann, M. de Gennes, A. Kabla, G. Salbreux, and B. Baum, Myosin II controls junction fluctuations to guide epithelial tissue ordering, *Dev. Cell* **43**, 480 (2017).
 - [10] S. Kim, M. Pochitaloff, G. A. Stooke-Vaughan, and O. Campàs, Embryonic tissues as active foams, *Nat. Phys.* **17**, 859 (2021).
 - [11] W. T. Gibson, J. H. Veldhuis, B. Rubinstein, H. N. Cartwright, N. Perrimon, G. W. Brodland, R. Nagpal, and M. C. Gibson, Control of the mitotic cleavage plane by local epithelial topology, *Cell* **144**, 427 (2011).
 - [12] Y. Mao, A. L. Tournier, A. Hoppe, L. Kester, B. J. Thompson, and N. Tapon, Differential proliferation rates generate patterns of mechanical tension that orient tissue growth, *EMBO J.* **32**, 2790 (2013).
 - [13] D. Roellig, S. Theis, A. Proag, G. Allio, B. Bénazéraf, J. Gros, and M. Suzanne, Force-generating apoptotic cells orchestrate avian neural tube bending, *Dev. Cell* **57**, 707 (2022).
 - [14] J. Firmino, D. Rocancourt, M. Saadaoui, C. Moreau, and J. Gros, Cell division drives epithelial cell rearrangements during gastrulation in chick, *Dev. Cell* **36**, 249 (2016).
 - [15] D. A. Matoz-Fernandez, E. Agoritsas, J.-L. Barrat, E. Bertin, and K. Martens, Nonlinear rheology in a model biological tissue, *Phys. Rev. Lett.* **118**, 158105 (2017).
 - [16] L. Bocanegra-Moreno, A. Singh, E. Hannezo, M. Zagorski, and A. Kicheva, Cell cycle dynamics control fluidity of the developing mouse neuroepithelium, *Nat. Phys.* **19**, 1050 (2023).
 - [17] T. E. Angelini, E. Hannezo, X. Trepat, M. Marquez, J. J. Fredberg, and D. A. Weitz, Glass-like dynamics of collective cell migration, *Proc. Natl. Acad. Sci. USA* **108**, 4714 (2011).
 - [18] J.-A. Park, J. H. Kim, D. Bi, J. A. Mitchel, N. T. Qazvini, K. Tantisira, C. Y. Park, M. McGill, S.-H. Kim, B. Gweon *et al.*, Unjamming and cell shape in the asthmatic airway epithelium, *Nat. Mater.* **14**, 1040 (2015).
 - [19] E.-M. Schotz, M. Lanio, J. A. Talbot, and M. L. Manning, Glassy dynamics in three-dimensional embryonic tissues, *J. R. Soc. Interface* **10**, 20130726 (2013).
 - [20] R. Farhadifar, J.-C. Röper, B. Aigouy, S. Eaton, and F. Jülicher, The influence of cell mechanics, cell-cell interactions, and proliferation on epithelial packing, *Curr. Biol.* **17**, 2095 (2007).
 - [21] M. Mayer, M. Depken, J. S. Bois, F. Jülicher, and S. W. Grill, Anisotropies in cortical tension reveal the physical basis of polarizing cortical flows, *Nature (London)* **467**, 617 (2010).
 - [22] I. Bonnet, P. Marcq, F. Bosveld, L. Fetler, Y. Bellaïche, and F. Graner, Mechanical state, material properties and continuous description of an epithelial tissue, *J. R. Soc. Interface* **9**, 2614 (2012).
 - [23] K. E. Kasza, D. L. Farrell, and J. A. Zallen, Spatiotemporal control of epithelial remodeling by regulated myosin phosphorylation, *Proc. Natl. Acad. Sci. USA* **111**, 11732 (2014).
 - [24] R. Piscitello-Gómez, F. S. Gruber, A. Krishna, C. Duclut, C. D. Modes, M. Popović, F. Jülicher, N. A. Dye, and S. Eaton, Core

- PCP mutations affect short-time mechanical properties but not tissue morphogenesis in the *Drosophila* pupal wing, *eLife* **12**, e85581 (2023).
- [25] N. A. Dye, M. Popović, K. Venkatesan Iyer, J. F. Fuhrmann, R. Piscitello-Gómez, S. Eaton, and F. Jülicher, Self-organized patterning of cell morphology via mechanosensitive feedback, *eLife* **10**, e57964 (2021).
- [26] A. Moriel, Y. Lubomirsky, E. Lerner, and E. Bouchbinder, Extracting the properties of quasilocalized modes in computer glasses: Long-range continuum fields, contour integrals, and boundary effects, *Phys. Rev. E* **102**, 033008 (2020).
- [27] R. N. Chacko, F. P. Landes, G. Biroli, O. Dauchot, A. J. Liu, and D. R. Reichman, Elastoplasticity mediates dynamical heterogeneity below the mode coupling temperature, *Phys. Rev. Lett.* **127**, 048002 (2021).
- [28] A. Moriel, D. Richard, E. Lerner, and E. Bouchbinder, Elementary processes in dilatational plasticity of glasses, *Phys. Rev. Res.* **6**, 023167 (2024).
- [29] J. F. Fuhrmann, A. Krishna, J. Pajmians, C. Duclut, G. Cwikla, S. Eaton, M. Popović, F. Jülicher, C. D. Modes, and N. A. Dye, Active shape programming drives *Drosophila* wing disc eversion, *Sci. Adv.* **10**, adp0860 (2024).
- [30] See Supplemental Material at <http://link.aps.org/supplemental/10.1103/lh3v-v2c9> for additional figures and details of the simulations, which contain Refs. [20,24,36].
- [31] M. Merkel, R. Etournay, M. Popović, G. Salbreux, S. Eaton, and F. Jülicher, Triangles bridge the scales: Quantifying cellular contributions to tissue deformation, *Phys. Rev. E* **95**, 032401 (2017).
- [32] J. D. Eshelby, The determination of the elastic field of an ellipsoidal inclusion, and related problems, *Proc. R. Soc. A* **241**, 376 (1957).
- [33] A. Nicolas, E. E. Ferrero, K. Martens, and J.-L. Barrat, Deformation and flow of amorphous solids: Insights from elastoplastic models, *Rev. Mod. Phys.* **90**, 045006 (2018).
- [34] We consider only force dipoles that do not exert a net torque.
- [35] M. F. Staddon, A. Hernandez, M. J. Bowick, M. Moshe, and M. C. Marchetti, The role of non-affine deformations in the elastic behavior of the cellular vertex model, *Soft Matter* **19**, 3080 (2023).
- [36] R. Etournay, M. Merkel, M. Popović, H. Brandl, N. A. Dye, B. Aigouy, G. Salbreux, S. Eaton, and F. Jülicher, TissueMiner: A multiscale analysis toolkit to quantify how cellular processes create tissue dynamics, *eLife* **5**, e14334 (2016).
- [37] P. J. Strzyz, M. Matejcic, and C. Norden, Heterogeneity, cell biology and tissue mechanics of pseudostratified epithelia: Coordination of cell divisions and growth in tightly packed tissues, in *International Review of Cell and Molecular Biology*, International Review of Cell and Molecular Biology Vol. 325, edited by K. W. Jeon (Academic, New York, 2016), Chap. 3, pp. 89–118.
- [38] O. Hertwig, Das problem der befruchtung und der isotropiedes eies: Eine theorie der vererbung, *Jenaische Zeitschrift für Naturwissenschaften*, Jena: Gustav Fischer. (1884).
- [39] M. Théry, A. Jiménez-Dalmaroni, V. Racine, M. Bornens, and F. Jülicher, Experimental and theoretical study of mitotic spindle orientation, *Nature (London)* **447**, 493 (2007).
- [40] A. Lisica, J. Fouchard, M. Kelkar, T. P. J. Wyatt, J. Duque, A.-B. Ndiaye, A. Bonfanti, B. Baum, A. J. Kabla, and G. T. Charras, Tension at intercellular junctions is necessary for accurate orientation of cell division in the epithelium plane, *Proc. Natl. Acad. Sci. USA* **119**, e2201600119 (2022).
- [41] T. C. Middelkoop, J. Neipel, C. E. Cornell, R. Naumann, L. G. Pimpale, F. Jülicher, and S. W. Grill, A cytokinetic ring-driven cell rotation achieves Hertwig’s rule in early development, *Proc. Natl. Acad. Sci. USA* **121**, e2318838121 (2024).
- [42] T. Sun, Y. Song, D. Teng, Y. Chen, J. Dai, M. Ma, W. Zhang, and J. C. Pastor-Pareja, Atypical laminin spots and pull-generated microtubule-actin projections mediate *Drosophila* wing adhesion, *Cell Rep.* **36**, 109667 (2021).
- [43] J. C. Dyre, Solid-that-flows picture of glass-forming liquids, *J. Phys. Chem. Lett.* **15**, 1603 (2024).
- [44] M. Ozawa and G. Biroli, Elasticity, facilitation, and dynamic heterogeneity in glass-forming liquids, *Phys. Rev. Lett.* **130**, 138201 (2023).
- [45] A. Tahaei, G. Biroli, M. Ozawa, M. Popović, and M. Wyatt, Scaling description of dynamical heterogeneity and avalanches of relaxation in glass-forming liquids, *Phys. Rev. X* **13**, 031034 (2023).
- [46] L. Berthier and J. Kurchan, Non-equilibrium glass transitions in driven and active matter, *Nat. Phys.* **9**, 310 (2013).
- [47] D. Bi, X. Yang, M. C. Marchetti, and M. L. Manning, Motility-driven glass and jamming transitions in biological tissues, *Phys. Rev. X* **6**, 021011 (2016).
- [48] L. M. C. Janssen, Active glasses, *J. Phys.: Condens. Matter* **31**, 503002 (2019).
- [49] F. Brauns, N. H. Claussen, M. F. Lefebvre, E. F. Wieschaus, and B. I. Shraiman, The geometric basis of epithelial convergent extension, *eLife* **13**, RP95521 (2024).
- [50] B. Aigouy, D. Umetsu, and S. Eaton, Segmentation and quantitative analysis of epithelial tissues, in *Drosophila: Methods and Protocols*, edited by C. Dahmann (Springer, New York, 2016), pp. 227–239.
- [51] N. A. Dye, M. Popović, S. Spann, R. Etournay, D. Kainmüller, S. Ghosh, E. W. Myers, F. Jülicher, and S. Eaton, Cell dynamics underlying oriented growth of the *Drosophila* wing imaginal disc, *Development* **144**, 4406 (2017).
- [52] C. Duclut, J. Pajmians, M. M. Inamdar, C. D. Modes, and F. Jülicher, Nonlinear rheology of cellular networks, *Cells Dev.* **168**, 203746 (2021).