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# Coupling water fluxes with cell wall mechanics in a multicellular model of plant development

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**The growth of plant organs is a complex process powered by osmosis that attracts water inside the cells; this influx induces simultaneously an elastic extension of the walls and pressure in the cells, called turgor pressure; above a threshold, the walls yield and the cells grow. Based on Lockhart's seminal work, various models of plant morphogenesis have been proposed, either for single cells, or focusing on the wall mechanical properties. However, the synergistic coupling of fluxes and wall mechanics has not yet been fully addressed in a multicellular model. This work lays the foundations of such a model, by simplifying as much as possible each process and putting emphasis on the coupling itself. Its emergent properties are rich and can help to understand plant morphogenesis. In particular, we show that the model can display a new type of lateral inhibitory mechanism that could contribute to the amplification of growth heterogeneities, essential for shape differentiation.**

Plant growth and morphogenesis | Biophysics | Mathematical modelling  
| Emergence | Lateral inhibition

Plants grow throughout their lifetime at the level of small regions containing undifferentiated cells, the meristems, located at the extremities of their axes. Growth is powered by osmosis that tends to attract water inside the cells. The corresponding increase in volume leads to simultaneous tension in the walls and hydrostatic pressure (so-called turgor pressure) in the cells. Continuous growth occurs thanks to the yielding of the walls to these stretching forces [1–3].

This interplay between growth, water fluxes, wall stress and turgor was first modelled by Lockhart in 1965 [4], in the context of a single elongating cell. Recent models focused on how genes regulate growth at more integrated levels [5–9]. To accompany genetic, molecular, and biophysical analyses of growing tissues, various extensions of Lockhart's model to multicellular tissues have been developed. The resulting models are intrinsically complex as they represent collections from tens to thousands of cells in 2- or 3-dimensions interacting with each other. To cut down the complexity, several approaches abstract organ multicellular structures as polygonal networks of 1D visco-elastic springs either in 2D [7, 10–12] or in 3D [6, 13] submitted to a steady turgor pressure. Other approaches try to represent more realistically the structure of the plant walls by 2D deformable wall elements able to respond locally to turgor pressure by anisotropic growth [8, 14, 15].

Most of these approaches consider turgor as a constant driving force for growth, explicitly or implicitly assuming that fluxes occur much faster than wall synthesis. Cells then regulate the tissue deformations by locally modulating the material structure of their walls (stiffness and anisotropy) [6, 16–20]. However, the situation in real plants is more complex: turgor heterogeneity has been observed at cellular level [21, 22], which challenges the assumption of very fast

fluxes. As a matter of fact, the relative importance of fluxes or wall mechanics as limiting factors to growth has fuelled a long standing debate [3, 23] and is still an open question. Moreover, from a physical point of view, pressure is a dynamic quantity that permanently adjusts to both mechanical and hydraulic constraints, which implies that a consistent representation of turgor requires to model both wall mechanics and hydraulic fluxes.

The aim of this article is to explore the potential effect of coupling mechanical and hydraulic processes on the properties of the "living material" that corresponds to multicellular populations of plant cells. To this end, we build a model that describes in a simple manner wall mechanics and cell structure, but do not compromise on the inherent complexity of considering a collection of deformable object hydraulically and mechanically connected.

The article is organized as follows (see Fig. 1): we first recall the Lockhart-Ortega model and its main properties. Then we explore two simple extensions of this model: first we relax the constraint of uniaxial growth in the case of a single polygonal cell; then we study how two cells hydraulically connected interact with each other. Finally we describe our multicellular and multidimensional model and numerically explore its properties.

## Significance Statement

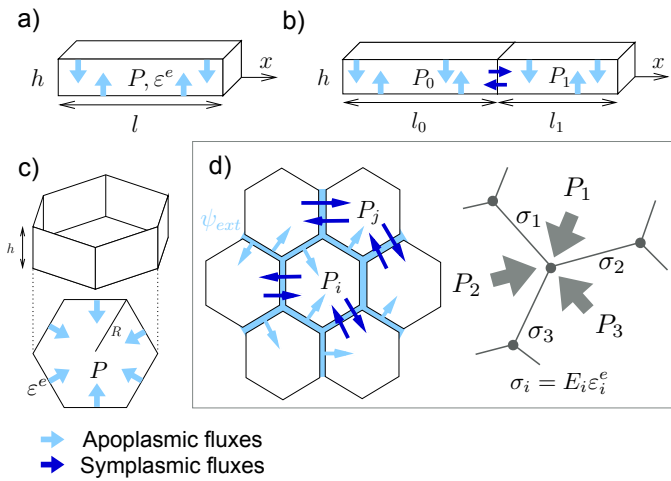
Plant cells are surrounded by a rigid wall that prevents cell displacements and rearrangements as in animal tissues. Therefore, plant morphogenesis relies only on cell divisions, shape changes, and local modulation of growth rate. It has long been recognized that cell growth relies on the competition between osmosis that tends to attract water into the cells and wall mechanics that resists to it, but this interplay has never been fully explored in a multicellular model. The goal of this work is to analyze the theoretical consequences of this coupling. We show that the emergent behavior is rich and complex: among other findings, pressure and growth rate heterogeneities are predicted without any ad-hoc assumption; furthermore the model can display a new type of lateral inhibition based on fluxes that could complement and strengthen the efficiency of already known mechanisms.

This study was initiated by C.G., M.G., and N.B. I.C. designed the model with the help of C.G. and M.G. performed the mathematical calculations with the help of C.G., designed the resolution algorithm, implemented it, ran simulations, and explored the parameters space. I.C. and C.G. analyzed the results and wrote the manuscript with inputs from other authors.

The authors declare no conflict of interest.

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**Fig. 1.** Hierarchy of models presented in this article. Main variables are turgor  $P$  and elastic deformation  $\varepsilon^e$ . **a)** Lockhart-Ortega model: uniaxial growth in the  $x$  direction of a cylindrical cell of length  $l$ ; the section perpendicular to  $x$  is a square of side  $h$ . **b)** two cells extension, both growing along  $x$ ; **c)** 2D extension of a single cell growth; **d)** Multicellular, multidimensional model; left: fluxes, right: mechanical equilibrium; the stress  $\sigma$  is proportional to the elastic deformation  $\varepsilon^e$ ;  $E$  is the elastic modulus.

## The Lockhart model

In 1965, Lockhart [4] derived the elongation of a cylindrical plant cell by coupling osmosis-based fluxes and visco-plastic wall mechanics. Ortega [24] extended this seminal model to include the elastics properties of the cell walls. We recall here the main properties of this model, see Fig. 1a for the geometrical configuration.

**Cell wall elongation.** It is expressed as a rheological law [4, 24]: the total strain rate of the walls  $\dot{\varepsilon}$  is decomposed into the sum of a plastic and an elastic strain rate:

$$\dot{\varepsilon} = \phi^w (P - P^Y)_+ + \frac{1}{\bar{E}} \frac{dP}{dt}, \quad [1]$$

where the extensibility  $\phi^w$  (inverse of a viscosity) describes the ability of the cell to synthesize wall material, and  $\bar{E}$  is an effective elastic modulus. Here,  $\phi^w$  and  $\bar{E}$  both depend on cell wall thickness. The notation  $(x)_+$  denotes  $x$  if  $x > 0$  and 0 otherwise for any real number  $x$ .

**Water uptake.** Lockhart described water uptake by the cell as a flux through a semi-permeable membrane characterized by its surface  $A$  and its permeability  $L^a$ . Assuming the membrane is perfectly impermeable to solutes, the rate of volume change is the result of a difference between the water potential  $\Psi$  of the cell and  $\Psi_{ext}$  of its exterior [25]:

$$\frac{dV}{dt} = AL^a (\Psi_{ext} - \Psi), \quad [2]$$

The cell water potential  $\Psi = P - \pi$  results from the antagonistic effect of the cell hydrostatic pressure  $P$  that tends to expel water from the cell and its osmotic pressure  $\pi$  that tends to attract water inside the cell. In the case of a single solute of concentration  $c$ , we have  $\pi = RTc$  where  $R$  is the ideal gas constant and  $T$  the temperature. Let us denote  $\phi^a = \frac{AL^a}{V}$  which has the same dimension as  $\phi^w$ . Assuming that the fluxes occur mostly on the lateral surface, the ratio  $A/V$  is constant

in the configuration of a cylindrical cell. After division by  $V$ , Eq. (2) turns into:

$$\dot{\gamma} = \phi^a (P^M - P). \quad [3]$$

where  $P^M = \Psi_{ext} + \pi$  quantifies the power of the osmotic pump: it is positive if  $\pi$  is high enough to overcome the negative water potential of the exterior of the cell. Growth ( $\dot{\gamma} > 0$ ) implies  $P < P^M$  and hence  $P^M$  is an upper bound for turgor, above which the cell would lose water to the exterior. The additional condition for growth  $P > P^Y$  (see above) requires  $P^M > P^Y$ : growth is possible only when the osmotic pump is able to overcome the mechanical resistance of the walls.

In order to keep the analysis as simple as possible, we take here and in the remaining of the article  $P^M$  constant with time and homogeneous among the cells, which corresponds for instance to constant  $\pi$  and  $\Psi_{ext}$ . This choice will be commented in the discussion section.

**Coupling hydraulics and mechanics for a single cell.** Equating the expressions of strain rate  $\dot{\varepsilon}$  from Eq. (1) and relative growth rate  $\dot{\gamma}$  from Eq. (3) ensures that the requirements for water uptake and yield of the cell wall are simultaneously satisfied. This means that turgor  $P$ , that is present in both equations, has to be adjusted to satisfy both hydraulic and mechanical constraints. The resolution of the model is detailed in Supplementary Information (SI), Eqs. (S3)-(S4). The time dependent solutions can be analytically determined and we find that  $P$  and  $\dot{\gamma}$  converge towards a stationary solution ( $P^*$ ,  $\dot{\gamma}^*$ ): first,  $P^*$  writes

$$P^* = \alpha^a P^M + (1 - \alpha^a) P^Y, \quad [4]$$

where

$$\alpha^a = \frac{\phi^a}{\phi^a + \phi^w} \in [0, 1] \quad [5]$$

measures the relative importance of  $\phi^a$  compared to  $\phi^w$ . In the limit  $\phi^a \ll \phi^w$  ( $\alpha^a = 0$ ), any excess of turgor above the threshold is relaxed by cell wall synthesis and turgor is minimal at  $P = P^Y$ . Conversely, in the limit  $\phi^w \ll \phi^a$  ( $\alpha^a = 1$ ), the wall synthesis is not able to relax turgor, which reaches then its maximal value  $P = P^M$ . Second, the expression of the relative growth rate is:

$$\dot{\gamma}^* = \frac{\phi^a \phi^w}{\phi^a + \phi^w} (P^M - P^Y), \quad [6]$$

or equivalently:  $P^M - P^Y = \left(\frac{1}{\phi^a} + \frac{1}{\phi^w}\right) \dot{\gamma}^*$ . This equation is the analog of Ohm's law  $\Delta U = (R_1 + R_2)I$  with two resistors  $R_1 = 1/\phi^a$  and  $R_2 = 1/\phi^w$  in series: growth can be limited by either hydraulic conductivity or wall synthesis.

**Link with wall rheology.** Wall expansion law (Eq. (1)) can be equivalently described as a function of wall stress  $\sigma$  rather than cell turgor  $P$ : in the cylindrical geometry of the Lockhart-Ortega model, we find (see SI for the calculations)  $P = \frac{2}{h} \sigma$ , where  $w$  is the width of the walls and  $h$  their height. Thanks to this relation, Eq. (1) translates into  $\dot{\varepsilon} = \frac{1}{\bar{E}} \frac{d\sigma}{dt} + \Phi^w (\sigma - \sigma^Y)_+$ , where  $E = \frac{h}{2w} \bar{E}$  (resp.  $\Phi^w = \frac{2w}{h} \phi^w$ ) is the intrinsic elastic modulus (resp. extensibility) of the walls. Let  $\varepsilon^e = \sigma/E$  be the so-called elastic deformation of the walls. It is dimensionless and can be measured from the image analysis

249 of experiments, without the knowledge of the elastic modulus.  
 250 The wall rheology is then described as follows:

$$251 \quad \dot{\varepsilon} = \frac{d\varepsilon^e}{dt} + \Phi^w E(\varepsilon^e - \varepsilon^Y)_+, \quad [7]$$

252 where  $\varepsilon^Y = \sigma^Y/E$  is the threshold elastic deformation. Note  
 253 that  $\frac{1}{\Phi^w E}$  can be interpreted as the characteristic time of wall  
 254 synthesis.

## 255 Multidimensional and multicellular models

256 A multicellular extension of the Lockhart-Ortega model  
 257 adapted to the study of morphogenesis requires first to relax  
 258 the constraint of uniaxial growth and allow multidimensional  
 259 geometries, and second is complexified by the possibility of  
 260 fluxes between cells. We study separately the effect of each of  
 261 these extensions before presenting the complete model.

262 **First extension: Multidimensional growth.** In order to keep the  
 263 analysis as simple as possible, we study here the expansion  
 264 of a single 2D cell whose shape is a regular polygon with  $n$   
 265 edges (see Fig. 1c). This model allows to evaluate the effect  
 266 of a varying surface/volume ratio compared to the Lockhart-  
 267 Ortega model where this ratio is constant. The fluxes are  
 268 described in the same way as for Lockhart's model (Eq. (2))  
 269 but wall synthesis is described with Eq. (7), as a function  
 270 of elastic deformation instead of turgor. We find (see SI for  
 271 detailed calculations) that the relation between cell turgor and  
 272 wall stress becomes  $P = \frac{w}{R \cos(\pi/n)} \sigma$  where  $R$  is the cell radius.  
 273 In contrast with the Lockhart-Ortega model, the ratio  $P/\sigma$  is  
 274 no more constant as cell grows, and the turgor vanishes at long  
 275 times if the stress remains in the order of magnitude of the  
 276 threshold. Note also that for a given stress the turgor decreases  
 277 with the number of edges  $n$ . Therefore, the yield turgor  $P^Y$   
 278 depends both on  $n$  and  $R$  and is not a well defined parameter.  
 279 It suggests also that cells with less neighbours should have a  
 280 higher turgor, as experimentally observed in [21, 22].

281 The prediction of growth rate requires a numerical reso-  
 282 lution of the model (see SI). The parameters are chosen to  
 283 ensure a turgor of the order of 0.5 MPa and a relative growth  
 284 rate of the order of 2% per hour, using the predictions Eq. (4)  
 285 and Eq. (6). First let's examine the case of a cell of initial  
 286 radius  $R = 10\mu\text{m}$  for which wall synthesis is the limiting factor  
 287 to growth (case  $\alpha^a = 0.9$  in SI, fig. S2). We find that it results  
 288 initially in an accelerating growth (the bigger the cell, the  
 289 faster the growth), much faster than predicted by the Lock-  
 290 hart model, during which the elastic deformation of the walls  
 291 can reach values up to 20%. The ratio area/surface =  $1/R$   
 292 decreases with growth and there is less and less water available  
 293 compared to the volume; as a consequence, the relative growth  
 294 rate vanishes at long times after this initial accelerating phase.

295 In the case where the fluxes are already limiting in the initial  
 296 state (case  $\alpha^a = 0.1$  in SI, Fig. S2), the initial behaviour is  
 297 closer to the predictions of the Lockhart model but the relative  
 298 growth rate still vanishes at long times.

299 Altogether, these results show that a non constant sur-  
 300 face/volume ratio deeply modifies the behavior of the model  
 301 compared to the Lockhart model. In particular, flux and wall  
 302 synthesis as limiting factors for growth are no more equivalent.

303 **Second extension: Multicellular growth.** Then, we study a sim-  
 304 ple multicellular extension of the Lockhart-Ortega model where

311 two cylindrical cells  $i = 0, 1$  are in contact through one of their  
 312 wall (see Fig. 1b). The cells can absorb water from their lateral  
 313 surface and in the meantime exchange water with each other  
 314 through their common wall. We look for stationary solutions:  
 315  $\frac{dP_i}{dt} = 0$  and  $\frac{1}{V_i} \frac{dV_i}{dt} = \text{Cst}$ .

316 We set for both cells a common value of  $P^M$ ,  $L^a$  and  
 317  $\phi^w$ , while the value of the yield turgors  $P_i^Y$  can differ; this  
 318 corresponds for instance to a heterogeneity of wall elastic  
 319 modulus or yield deformation. For the sake of convenience, we  
 320 refer to fluxes between cells as symplasmic fluxes, characterized  
 321 by a water conductivity  $L^s$ , and to fluxes from the water source  
 322 as apoplasmic fluxes, characterized by a water conductivity  
 323  $L^a$ . Assuming that the symplasmic fluxes occur through  
 324 plasmodesmata that are permeable to both water and solutes,  
 325 the flux equation writes

$$326 \quad \frac{dV_i}{dt} = A_i L_i^a (P^M - P_i) + A_{01} L^s (P_j - P_i),$$

327 where  $j = 1 - i$ , and  $A_{01}$  is the surface of the common wall  
 328 of cells 0 and 1. We introduce the number  $\phi^s = 2A_{01}L^s/V_i$   
 329 which has the same dimension as  $\phi^a$  and  $\phi^w$ . In order to allow  
 330 an analytical resolution of this set of equations, we assume  $\phi^s$   
 331 to be constant with time, and consider it in this section as a  
 332 parameter of the model. Thus, we have

$$333 \quad \dot{\gamma}_i = \phi^a (P^M - P_i) + \frac{\phi^s}{2} (P_j - P_i). \quad [8]$$

334 We introduce the dimensionless number

$$335 \quad \alpha^s = \frac{\phi^s}{\phi^s + \phi^a} \in [0, 1]$$

336 which represents the relative importance of symplasmic fluxes  
 337 with respect to apoplasmic ones. We combine this flux equation  
 338 with the growth equation Eq. (1) and find analytical solutions  
 339 for any values of the parameters (see SI). We use here the  
 340 following set of control parameters:

$$341 \quad P^M, P_i^Y, \dot{\gamma}_0^*, \alpha^a, \alpha^s,$$

342 and fix the value  $\dot{\gamma}_0^* = 2\% \cdot \text{h}^{-1}$ ; this way, the parameters  
 343 space to explore is reduced to  $(P^M, P_i^Y, \alpha^a, \alpha^s)$ . When  $\alpha^s = 0$ ,  
 344 the cells are completely isolated one from another and reach  
 345 turgors  $P_i^*$  and growth rates  $\dot{\gamma}_i^*$  as predicted by the Lockhart  
 346 model (Eq. (4) and Eq. (6)). In particular, the condition  
 347  $P^M > P_i^Y$  ensures that each cell is growing. When  $\alpha^s > 0$ ,  
 348 the fluxes between cells modify this behaviour. We restrict  
 349 to the case  $P_0^Y < P_1^Y < P^M$ , which corresponds to less  
 350 mechanical constraints on cell 0 than cell 1; therefore we can  
 351 expect  $P_1 > P_0$  and  $\dot{\gamma}_1 < \dot{\gamma}_0$ . The calculations show a complex  
 352 non linear behaviour that is illustrated in Fig. 2, in which  
 353 the parameters subspace  $(\alpha^a, \alpha^s)$  is explored for given values  
 354 of  $P_i^Y$  and  $P^M$  (detailed calculations are provided in SI). Let  
 355  $\Delta P^Y = P_1^Y - P_0^Y > 0$  be the difference of the two yield turgors  
 356 and  $\bar{P}^Y = 0.5(P_0^Y + P_1^Y)$  their average; we also introduce the  
 357 dimensionless number

$$358 \quad \rho = \frac{\Delta P^Y}{2(P^M - \bar{P}^Y)}. \quad [9]$$

359 Note that the hypothesis  $P_0^Y < P_1^Y < P^M$  is equivalent to  
 360  $\rho \in ]0, 1[$ .

361 We find that the subspace  $(\alpha^a, \alpha^s)$  can be divided in two  
 362 main regions separated by the curve  $\alpha^s = \frac{1-\rho}{1-\alpha^a}$  (see Fig. 2a):

373 surprisingly, in the region  $\alpha^s > \frac{1-\rho}{1-\alpha^a}$ , only cell 0 is growing  
 374 ( $\dot{\gamma}_0 > 0$ ,  $\dot{\gamma}_1 = 0$ , and equivalently  $P_0 > P_0^Y$ ,  $P_1 < P_1^Y$ ). Hence,  
 375 the growth of cell 1 is inhibited by fluxes with cell 0. Conversely,  
 376 in the region  $\alpha^s < \frac{1-\rho}{1-\alpha^a}$  both cells are growing ( $\dot{\gamma}_i > 0$  and  
 377 equivalently  $P_i > P_i^Y$ ). The size of the region  $\alpha^s > \frac{1-\rho}{1-\alpha^a}$   
 378 increases with  $\rho$  and fills the whole square  $[0, 1] \times [0, 1]$  when  
 379  $\rho \rightarrow 1$ ; such values can be reached when  $\Delta P^Y$  is large and /  
 380 or  $P^M$  is close to  $\bar{P}^Y$ .

381 More quantitatively, Figs. 2d-e) show that  $\dot{\gamma}_1$  is always  
 382 below  $\dot{\gamma}_1^*$ , while  $\dot{\gamma}_0$  is always above  $\dot{\gamma}_0^*$  and can reach up to  
 383 twice this value. Furthermore, maximal values of  $\dot{\gamma}_0$  coincide  
 384 with minimal values of  $\dot{\gamma}_1$ : this confirms quantitatively that  
 385 the growth of the cell with less favorable mechanical condition  
 386 is slowed down if not inhibited by the growth of its neighbour.  
 387 This shows also that the growth rate heterogeneity is amplified  
 388 by fluxes.

389 Turgor heterogeneity is also affected by fluxes (see Figs. 2b-  
 390 c): when  $\alpha^s$  is close to zero, the cells are hydraulically isolated  
 391 and their turgors vary with  $\alpha^a$  as predicted by Lockhart model  
 392 (Eq. (4)), this is where the turgor heterogeneity is maximal.  
 393 Conversely, when  $\alpha^s$  is close to 1, there is no hydraulic resis-  
 394 tance between the two cells and the two turgors are equal.  
 395 Between these two limits,  $P_0$  is only slightly affected and re-  
 396 mains in the  $[P_0^Y, P^M]$  interval; conversely,  $P_1$  is dramatically  
 397 affected as it shifts from the interval  $[P_1^Y, P^M]$  when  $\alpha^s = 0$  to  
 398 the interval  $[P_0^Y, P^M]$  when  $\alpha^s = 1$ . Therefore, as  $P_0^Y < P_1^Y$ ,  
 399 there is a region where  $P_1 < P_1^Y$  which corresponds to the  
 400 region  $\alpha^s > \frac{1-\rho}{1-\alpha^a}$ , where cell 1 is not growing.

401 Finally, we have seen that intercellular fluxes tend to in-  
 402 crease (resp. decrease) growth rate (resp. turgor) hetero-  
 403 geneities; the cell with less mechanical constraints takes con-  
 404 trol over the other one and imposes its turgor, which can lead  
 405 the other one to stop growing. The growing cell then benefits  
 406 from the water resources of the other cell and its growth is all  
 407 the more increased.

409 **Generalization: a multidimensional and multicellular model**  
 410 **of growth.** We consider (see Fig. 1d) a collection of  $N$  cells  
 411 that form a (non necessarily regular) 2D mesh with a fixed  
 412 topology (distribution of neighbours) as is the case with plant  
 413 tissues when no division occurs.

414 The cell walls rheology is described by the visco-elasto-  
 415 plastic law (Eq. (7)) of the Ortega model and the fluxes toward  
 416 a cell  $i$  are described as in the simple multicellular model  
 417 presented above:

$$418 \frac{dV_i}{dt} = A_i L_i^a (P^M - P_i) + \sum_{j \in n(i)} A_{ij} L_{ij}^s (P_j - P_i), \quad [10]$$

419 where  $n(i)$  is the set of neighbours of cell  $i$ ,  $A_{ij}$  is the area  
 420 of the common wall with cell  $j$ ,  $L_{ij}^s$  its permeability (it is  
 421 symmetric:  $L_{ij}^s = L_{ji}^s$ ), and  $L_i^a$  is the permeability of the  
 422 lateral walls to the supply of water.

423 The last missing part to obtain a closed set of equation is  
 424 the mechanical equilibrium, that allows to link cells turgors,  
 425 walls tensions, and geometry. Contrary to the cases studied  
 426 above, no explicit expression of turgors as a function of stresses  
 427 can be obtained and the equilibrium has to be solved at each  
 428 time step. Let  $P_i$  be the turgor pressure in each cell  $i$ . The  
 429 tissue being at every moment in a quasi-static equilibrium,  
 430 pressure forces on wall edges and elastic forces within walls

balance exactly at each vertex  $v$ :

$$431 \frac{1}{2} \sum_{k \in f(v)} \Delta_k P S_k \mathbf{n}_k + \sum_{k \in f(v)} E_k \epsilon_k^e s_k \mathbf{e}_{k,v} = 0, \quad [11]$$

432 where  $f(v)$  is the set of faces adjacent to junction  $v$ ,  
 433  $\Delta_k P = P_{k_1} - P_{k_2}$  is the pressure jump across face  $k$ , with  
 434  $k_1 < k_2$  being indices of the cells across face  $k$ ,  $S_k = h l_k$  is  
 435 the area of the face  $k$  on which pressure is exerted,  $\mathbf{n}_k$  is the  
 436 normal vector to face  $k$ , oriented from cell  $k_1$  to cell  $k_2$ , and  
 437  $s_k = hw$  is the cross-section area of the face, on which the  
 438 elastic stress is exerted; finally,  $\mathbf{e}_{k,v}$  is the unit vector in the  
 439 direction of face  $k$ , oriented from junction  $v$  to the other end  
 440 of face  $k$ .

441 **Coupling mechanical and hydraulic models.** In the Lockhart-  
 442 Ortega model, the compatibility between wall enlargement  
 443 and cell volume variation is automatically enforced through  
 444 the geometrical constraint of uni-directional growth that leads  
 445 to the identity between the relative growth rate of the cell and  
 446 the strain rate of the walls. In contrast, in the multicellular  
 447 model, this identity is no longer true. One has to solve the  
 448 closed set of equations Eq. (7)-Eq. (10)-Eq. (11) with respect  
 449 to the unknowns  $X$ ,  $P$ , and  $\epsilon^e$ .

450 Despite its apparent simplicity, the problem to be solved  
 451 is not straightforward as water fluxes induce potentially long  
 452 range interactions. In this respect, it differs from most vertex-  
 453 based models (e.g [11, 26]) where turgor is an input of the  
 454 model. The numerical resolution required the development  
 455 of an original algorithm (see SI) implemented in an in-house  
 456 code.

457 **Numerical experiments: growth of primordia in the shoot apical**  
 458 **meristem (SAM).** The properties of this model cannot be  
 459 as thoroughly studied as those of the simpler models presented  
 460 above, first because of the numerical cost of the resolution,  
 461 but above all because it allows an infinite variety of geometries  
 462 and spatial distribution of its parameters. We present here a  
 463 numerical experiment that illustrates on the one hand how the  
 464 properties of the simple multidimensional and multicellular  
 465 submodels are combined in the generalized model; in turn the  
 466 study of these models helps us to anticipate the properties  
 467 of the generalized model. And on the other hand, we show  
 468 that this model is readily applicable to the study of systems  
 469 of biological interest.

470 Growth heterogeneities can be triggered by the local mod-  
 471 ulation of the mechanical properties of the cell walls [27]. In  
 472 SAMs, new organs are initiated by a local increase in growth  
 473 rate that leads to the appearance of small bumps. Measure-  
 474 ments show that physico-chemical properties of walls are mod-  
 475 ified so that mechanical anisotropy and elastic modulus are  
 476 decreased. In our 2D model, we can explore what effect a  
 477 local softening of the walls has on growth rate and turgor  
 478 heterogeneities; based on our previous analysis of the model  
 479 in simple configurations, we expect that the growth hetero-  
 480 geneities will be maximal for parameters such that the growth  
 481 is restricted by fluxes rather than wall synthesis (low  $\alpha^a$ ),  
 482 cell-cell conductivity is large, and the walls deformations are  
 483 just above the growth threshold, which can be enforced by a  
 484 low value of the osmotic pressure (yet large enough to ensure  
 485 growth). The set of parameters (REF) is chosen according  
 486 to these criteria; then we explore the effect of a higher  $\alpha^a$   
 487 ((ALPHA+) set) and lower cell-cell conductivity ((CC-) set)

497 that should both decrease the growth heterogeneities, and also  
498 test the effect of a lower osmotic pressure ((PM-) set) that  
499 should conversely increase the growth heterogeneity. See table  
500 1 in SI for the values of the parameters corresponding to these  
501 sets and SI for more precise explanations.

502 We build a mesh made primarily of hexagons (see Fig. 3a)  
503 and first let it grow with homogeneous parameters until the  
504 elastic regime ends and plastic growth occurs. Then we di-  
505 vide by two the elastic modulus of a small group of cells  
506 (marked with a white star in Fig. 3a) that will be referred to  
507 as “bump cells” thereafter. All the details of the computations  
508 are presented in SI. First, Fig. 3b shows that the multicellular  
509 system grows globally in the same way as the single hexagonal  
510 cell studied above; it diverges from the Lockhart predictions  
511 because the ratio  $A/V$  of the cells is not constant: the (AL-  
512 PHA+) simulations exhibit a very large initial growth rate  
513 that decreases only when the cells are so large that water  
514 fluxes become limiting. The (PM-) set leads to a roughly  
515 twice lower growth rate than (REF). The set (CC-) leads to  
516 the same dynamics at the tissue level as (REF), because the  
517 total influx of water is not affected by fluxes between cells in  
518 this setup.

519 Then we turn to the observation of heterogeneities: we focus  
520 on the differences between the bump region and the rest of the  
521 tissue. For all the parameters sets, Fig. 3c shows that turgor  
522 is in general lower in bump cells, but the gap varies depending  
523 on the parameters, as it has been predicted by the study of  
524 the two-cells model: compared to (REF), the heterogeneity  
525 in turgor is increased by a lower cell-cell conductivity (set  
526 CC-), and decreased by a larger value of  $\alpha^a$  (set ALPHA+).  
527 Decreasing the value of  $P^M$  (set PM-) does not alter much  
528 the turgor heterogeneity compared to (REF). The maps of  
529 turgor (Figs. 3e,g,i,k) confirm visually these observations.

530 Fig. 3d shows the time evolution of  $\dot{\gamma}/\dot{\gamma}^*$  where  $\dot{\gamma}^*$  is the  
531 relative growth rate predicted by the Lockhart model (see  
532 Eq. (6)); its value is  $2\% \text{ h}^{-1}$  for (REF), (CC-) and (ALPHA+),  
533 and  $0.5\% \text{ h}^{-1}$  for (PM-). In the considered time frame, the  
534 relative growth rate of bump cells is always higher except for  
535 (ALPHA+): after an initial fast increase where bump cells  
536 grow faster, the tendency is inversed at  $t \approx 20\text{h}$  because the  
537 bump cells have grown so much that fluxes become limiting. In  
538 the (REF) simulation, while the growth rate of non bump cells  
539 is almost constant and close to  $\dot{\gamma}^*$ , the growth rate of the bump  
540 cells is up to 6 times  $\dot{\gamma}^*$  at the beginning of the simulation and  
541 progressively decreases toward  $\dot{\gamma}^*$ . As a result of this large  
542 discrepancy, the bump region can be clearly distinguished from  
543 the rest of the tissue (Figs. 3e-f). In (CC-), the growth rate of  
544 the non bump cells is close to that of (REF), but the growth  
545 rate of the bump cells is much lower (Fig. 3d). As a result,  
546 the global shape remains convex and the bump is not clearly  
547 detached from the rest of the tissue (Figs. 3i-j). Note that  
548 (CC-) corresponds to a lower value of  $\alpha^s$  compared to (REF),  
549 which corresponded to a lower growth heterogeneity with the  
550 two-cells model studied above; this is also confirmed by the  
551 lower cell-cell fluxes towards the bump cells for (CC-), see  
552 the arrows in Figs. 3e,i. The (ALPHA+) simulation exhibits  
553 also a convex shape (Fig. 3k-l); it corresponds to a larger  
554 value of  $\alpha^a$  than (REF), and similarly to the two-cells model  
555 studied above, the growth rate heterogeneity is lower than  
556 (REF). Finally, the set (PM-) corresponds to an increase of  
557 the dimensionless parameter  $\rho$  (see Eq. (9)), and accordingly

558 to an increase in growth rate heterogeneity as can be seen  
559 with Fig. 3d. Consequently, the bump region can clearly  
560 distinguished from the rest of the tissue, even better than  
561 (REF) (Fig. 3g-h); moreover, the growth of the cells close to  
562 the bump seems to be inhibited by fluxes as explained in the  
563 two-cells model described above and further explored below.

564 **Flux-based lateral inhibition predicted by the model.** As we saw,  
565 cells that benefit from better mechanical conditions for growth  
566 (in the present case a lower elastic modulus) have a lower turgor  
567 than the other cells, and therefore attract water from them.  
568 Not only does it amplify their growth but it also inhibits  
569 the growth of their neighbours. Such a lateral inhibition  
570 mechanism is important for morphogenesis, as it allows very  
571 large growth rate heterogeneities and the appearance of well  
572 differentiated shapes (in the present case the appearance of a  
573 bump on the surface of the meristem). The efficiency of this  
574 mechanism varies depending on the position in the parameters  
575 space: for instance it is increased if the cell-cell conductivity  
576  $L^s$  (or equivalently  $\alpha^s$ ) is increased (see Fig. 4a-d); even  
577 the whole tissue can be inhibited. Inhibited cells can also  
578 relax the tension of their walls and decrease their volume (see  
579 Fig. 4a). To further explore and quantify the spatial range of  
580 this inhibition process, we extended our two-cells model (see  
581 SI for detailed equations) to a chain of  $2N + 1$  cells where  
582 the central cell has twice softer walls. We numerically solved  
583 the corresponding system of differential equations for the set  
584 (REF) and then for a large range of values of  $L^s$ . Fig. 4e shows  
585 that the number  $2N_i$  of inhibited cells scales with  $\sqrt{L^s}$ . We  
586 computed the prefactor  $c$  (such that  $N_i \approx c\sqrt{L^s}$ ) for values  
587 of  $(\alpha^a, P^M) \in [0.05, 0.35] \times [0.51, 0.85]$  (the interval for  $P^M$  is  
588 in MPa) and plotted its value in the  $(\alpha^a, P^M)$  space (Fig. 4f).  
589 This shows that the inhibition is favored by low values of  $\alpha^a$   
590 and  $P^M - P^Y$ .

## 591 Discussion

592 **A minimal model with a complex and rich behavior.** The model  
593 proposed in this article is a minimal multicellular and multidimensional extension of the Lockhart 1-D single cell model; it  
594 can be regarded as a conceptual tool to study the interplay  
595 between fluxes and wall mechanics in a multicellular tissue.  
596 Wall expansion is modeled with a visco-elasto-plastic rheological law, while fluxes derive from water potential gradients.  
597 These two contributions are integrated into the mechanical  
598 equilibrium and interact through the pressure term. Contrary  
599 to most previous approaches, turgor is not an input of the  
600 model but a variable that adjusts simultaneously to mechanical,  
601 hydraulic, and geometrical constraints. First of all, this  
602 leads to a physically consistent representation of turgor: for  
603 instance, the model predicts that cells with softer walls have  
604 a lower turgor. Moreover, this has deep implications at tissue  
605 level: in the previous example, lower turgor is associated with  
606 a faster growth which can be itself amplified by fluxes that  
607 follow decreasing pressure gradients.

608 Thanks to the simplicity of the model, the predicted behavior  
609 can be analyzed and interpreted with two submodels built  
610 from the Lockhart model: first, a 1-D multicellular submodel  
611 was build with two or more side-by-side cells; it was used to  
612 study the growth of competing cells with heterogeneous prop-  
613 erties. Key ingredients here are the wall synthesis threshold,  
614 the fact that fluxes and growth can relax turgor, and cell to  
615 cell fluxes that allow long range interactions. Second, in a  
616

621 1-D system, cells are considered essentially as cylinders and  
622 their surface-to-volume ratio is constant. We thus extended  
623 also the Lockhart model in two dimensions, where cells have  
624 more degree of freedom to change their shape. In particular  
625 their allometric surface-to-volume ratio may then vary. This  
626 new possibility induces additional complexity in the tissue  
627 development as the rate of growth of cell surfaces may become  
628 a limiting factor for growing cells.

629 **A potentially new type of lateral inhibition mechanism.** Depend-  
630 ing on mechanical and hydraulic parameters of tissue regions,  
631 the model exhibits different growth regimes corresponding to  
632 either uniform or differential growth. One unexpected conse-  
633 quence of such an hydraulic-mechanical coupling at the tissue  
634 level is the observation that in certain regions of the parameter  
635 space where cell-to-cell hydraulic exchanges are non-limiting,  
636 growing tissue may exert an inhibiting influence on the growth  
637 of neighboring regions. This may be interpreted as a lateral  
638 inhibition mechanism. It has for long been recognized that  
639 lateral inhibitory mechanisms play a key role in setting some  
640 morphogenetic patterns in procaryotes (e.g. [28]), animals (e.g.  
641 [29, 30]) or plants (e.g. [31, 32]). Lateral inhibition operates  
642 in these systems via chemical signals, such as delta-notch in  
643 animals or auxin in plants. Our model predicts the existence  
644 of a novel type of lateral inhibition mechanism based on the  
645 coupling between mechanics and water fluxes. Previous obser-  
646 vations of tissue growth suggest that such a phenomenon may  
647 occur in real tissues. In the shoot apical meristem for instance,  
648 detailed quantification of growth with cellular resolution indi-  
649 cates that the region surrounding primordia growth may have  
650 a negative growth rate [33], Figs. 2G and 3K. According to  
651 our model, this decrease of volume in boundary regions might  
652 be due to the primordium growth attracting locally most of  
653 the water supply and depriving lateral regions from water, and  
654 thus confornts the hypothesis of a new hydraulic-mechanical  
655 component of primordium lateral inhibition, beyond already  
656 identified auxin and cytokinin signals [34].  
657

658 **Model simplifications and further potential extensions.** Throug-  
659 hout the development of the model, we made several key choices  
660 concerning the abstraction of a multicellular plant tissue. First,  
661 our model was developed in 2-D for reasons of computational  
662 efficiency. In principle, it can be extended in 3-D, though at  
663 the expense of more complex formalism and implementation.  
664 Second, the current model considers that water transport is  
665 performed in the plant tissue through two conceptually differ-  
666 ent pathways ([1]). Water can first move within the apoplastic  
667 compartment between the cells and finally enter a cell. Water  
668 can also move locally from cell to cell. This movement includes  
669 itself conceptually both symplasmic movements (water circ-  
670 ulates between cells through plasmodesmata without crossing  
671 membranes) and movements from cell to cell with intermedi-  
672 ate steps in the wall (water is for example exported locally  
673 out of the cell by water transporters like aquaporins into the  
674 wall and immediately re-imported by water transporters into  
675 neighboring cells). For sake of simplicity in this first analysis,  
676 we represented the apoplasm as a single abstract compartment  
677 able to exchange water with every cell. To analyze precisely  
678 the effect of water transporters and their genetic regulation or  
679 to assess the impact of wall resistance to water movement in  
680 the processes, explicit spatial representation of the apoplasm,  
681 of plasmodesmata and of membrane water transporters could  
682 be integrated into the model in the future.

683 Finally, we considered a simplified situation here by impos-  
684 ing constant cell osmolarity. Allowing osmolarity variations  
685 (for instance higher values in faster growing regions) may  
686 impact turgor distribution (e.g [35]). However, this should  
687 not affect the ability of the system to build up growth het-  
688 erogeneities. Similarly, we further simplified our model by  
689 keeping constant the apoplastic water potential. Relaxing  
690 this hypothesis would increase cell-cell water fluxes (via the  
691 apoplasm) and could also shift the model in the direction of  
692 the flux-limiting regime. This would therefore favor regimes  
693 where growth heterogeneities are amplified by fluxes.  
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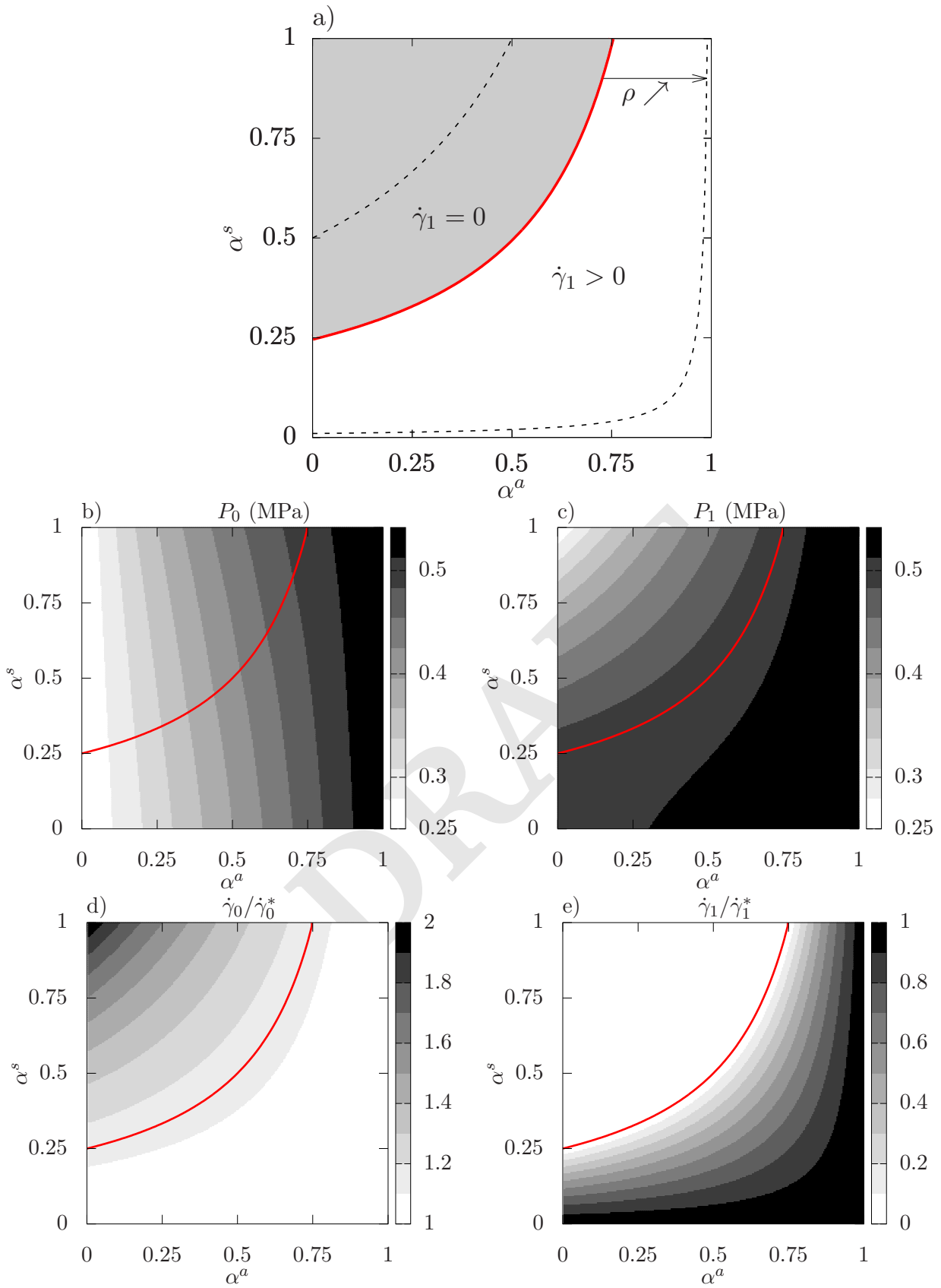
695 **ACKNOWLEDGMENTS.** This work has been carried out within  
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697 foundation in Montpellier, France.  
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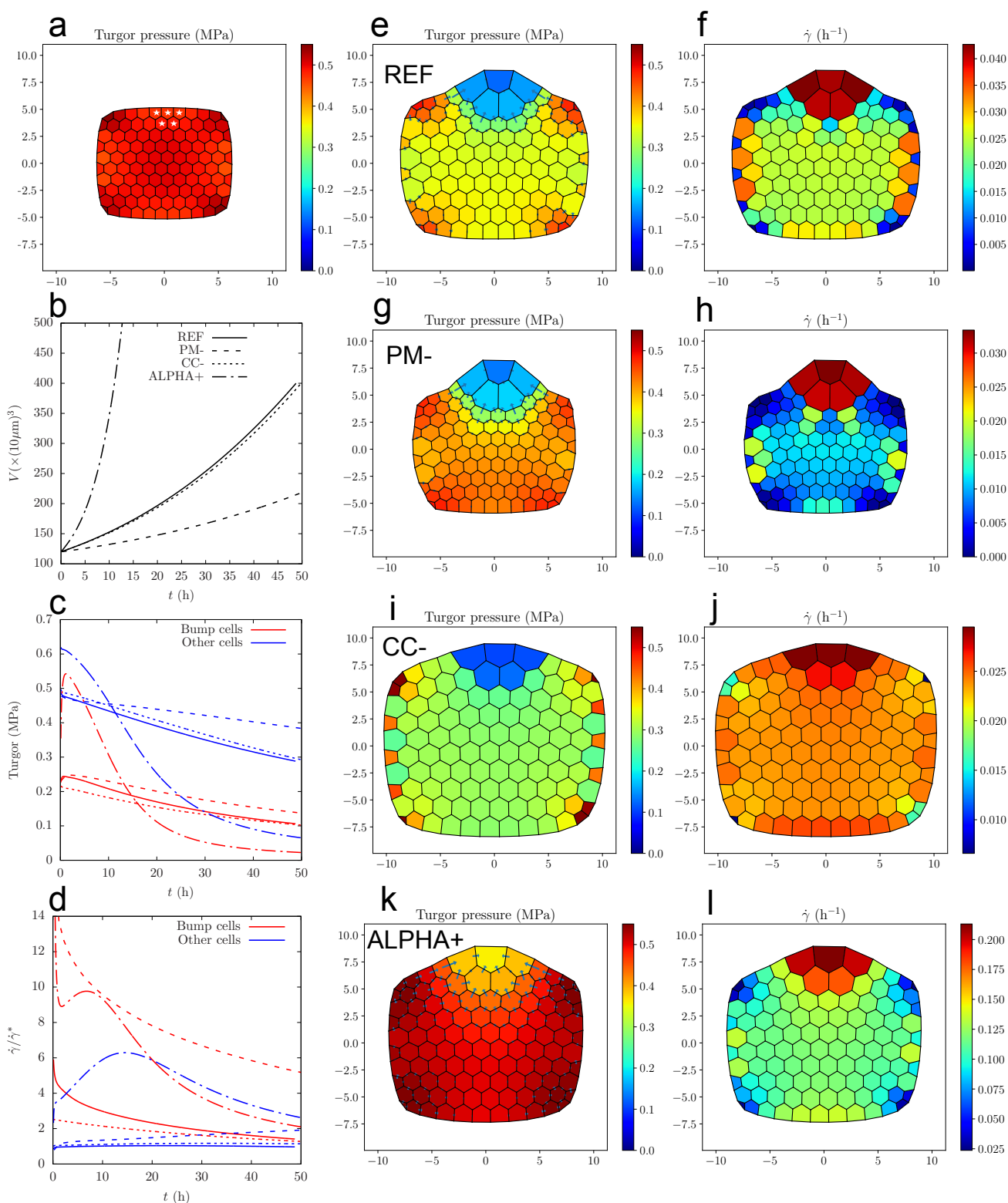
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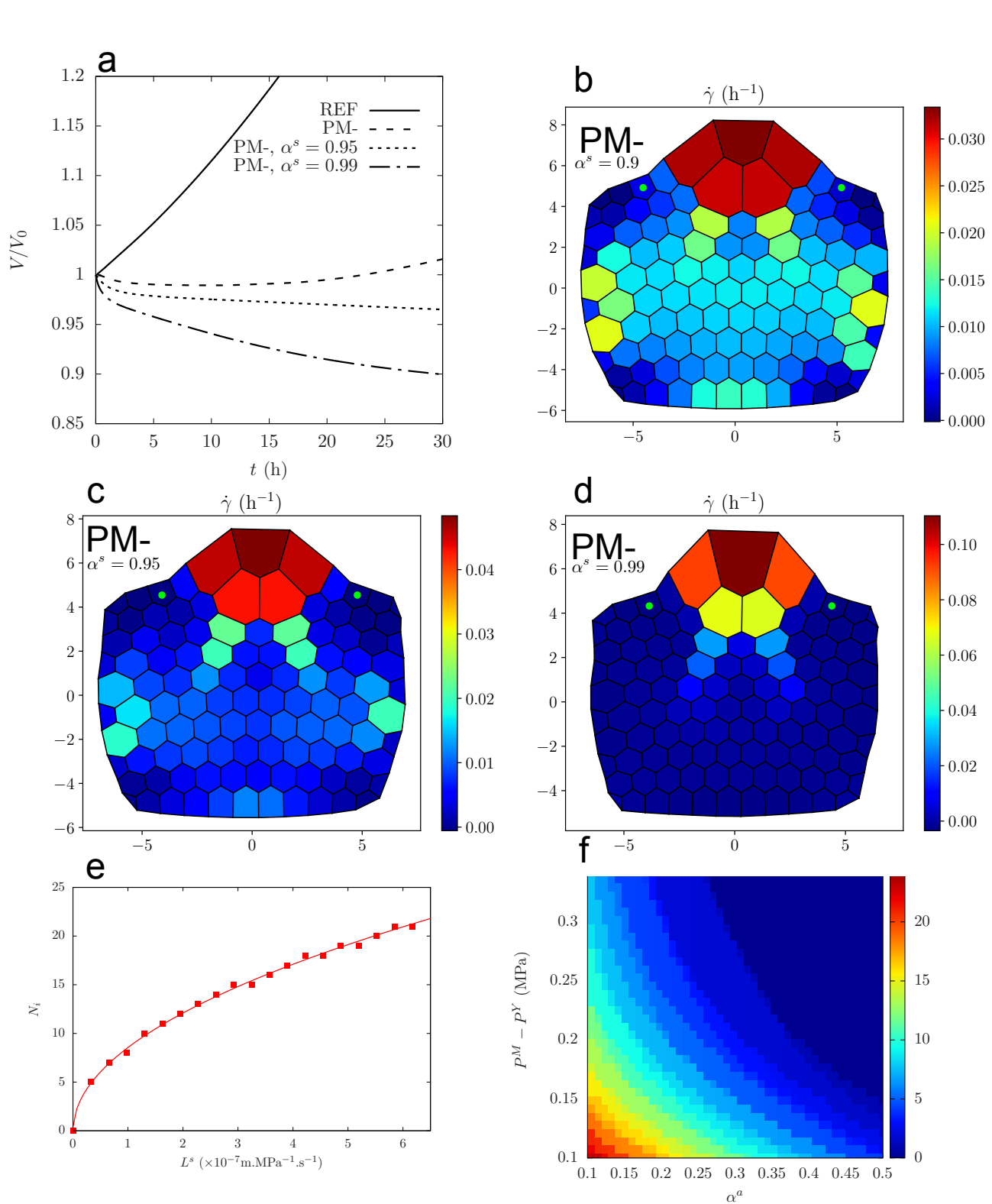
**Fig. 2.** Analytical resolution of the two cells model, properties of the solution in the parameters space  $\alpha^a \times \alpha^s$ ; **a)** delimitation of the two zones  $\dot{\gamma}_1 = 0$  and  $\dot{\gamma}_1 > 0$ : the red thick solid line  $\alpha^s(\alpha^a) = \frac{1-\rho}{1-\alpha^a}$  corresponds to  $\rho = 0.75$ . The two black thin dashed lines correspond to the values  $\rho = 0.5$  and  $0.99$ . **b-c)** Turgors  $P_0$  and  $P_1$  for  $\rho = 0.75$ . **d-e)** relative growth rates  $\dot{\gamma}_i/\dot{\gamma}_i^*$  for  $\rho = 0.75$ .

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**Fig. 3.** Growth of tissue with heterogeneous mechanical parameters, see table 1 in SI. **(a)** Initial state for (REF): walls are under tension because of turgor and have reached their yield deformation. At  $t = 0$ , the walls of the cells marked with a white star are softened (the elastic modulus is divided by two). **(b)** Time evolution of the total volume. The dashtype of the lines distinguishes the parameters sets; the same dashtype convention is used in **(c)** and **(d)**. **(c)** Time evolution of turgor pressure of bump cells (red) and other cells (blue). **(d)** Time evolution of relative growth rate of bump cells (red) and other cells (blue). **(e-l)** Turgor and relative growth rate maps of parameters sets (REF) (**(e-f)**), (PM-) (**(g-h)**), (CC-) (**(i-j)**), and (ALPHA+) (**(k-l)**), at the time when the volume of the bump cells has increased by a factor 5:  $t = 51\text{h}$  for (REF),  $t = 33\text{h}$  for (PM-),  $t = 80\text{h}$  for (CC-),  $t = 14.8\text{h}$  for (ALPHA+). The arrows represent the intensity and direction of cell-cell water fluxes; the scale for arrows is the same for (REF), (PM-) and (CC-) and close to 4 times higher for (ALPHA+).

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**Fig. 4.** Evidence of lateral inhibition: left: **a**) time evolution of the volume of two cells on the boundary of the bump (marked with a green dot on the maps **b, c, d**) with the sets of parameters (REF), (PM-), (PM-) with  $\alpha^s = 0.95$ , (PM-) with  $\alpha^s = 0.99$ .  $V_0$  is the volume of the cells at  $t = 0$ . **b, c, d**) maps of relative growth rate at  $t = 33$ h for (PM-),  $t = 20$ h for (PM-) and  $\alpha^s = 0.95$ ,  $t = 10$ h for (PM-) and  $\alpha^s = 0.99$ . **e-f**) Results for a chain of  $2N + 1$  cells with  $N = 50$ , where the central cell has twice softer walls; **e**) number  $N_i$  of cells that are inhibited on each side of the central cell, for different values of  $L^s$ ; the line is a fit with a square root function, in the form  $c\sqrt{L^s}$ . **f**) Values of the prefactor  $c$  in the space  $(\alpha^a, P^M)$ .

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**Supplementary Information for**

**Coupling water fluxes with cell wall mechanics in a multicellular model of plant development**

**Ibrahim Cheddadi, Michel Génard, Nadia Bertin, Christophe Godin**

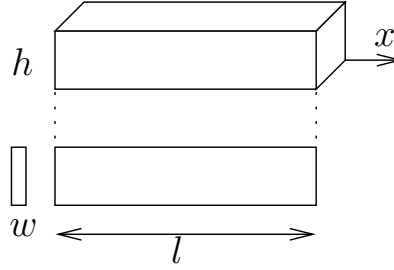
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**This PDF file includes:**

- Supplementary text
- Figs. S1 to S4
- Table S1
- References for SI reference citations

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139  
140 **Fig. S1.** Geometrical parameters of Lockhart-Ortega models: height  $h$  and length  $l$  of the cell, thickness  $w$  of the walls. The two faces orthogonal to the  $x$  axis are referred to  
 141 as base faces while the four other faces are referred to as lateral faces.

142  
143 **Lockhart-Ortega models.** The equations of cell wall elongation (Eq. (1) in main text) and of water uptake (Eq. 2 in main text) 205  
 144 can be linked thanks to the geometry of the cell and the mechanical equilibrium. See Fig. S1 for the geometrical description. 206

145 First, the cell volume is  $V = h^2 l$  and therefore we find that the relative growth rate of the cell is equal to the strain rate of 207  
 146 the walls: 208

$$147 \quad \dot{\gamma} = \frac{1}{V} \frac{dV}{dt} = \frac{1}{l} \frac{dl}{dt} = \dot{\epsilon}. \quad [S1] \quad 209$$

149 Then, we consider the balance of forces on the base faces (see Fig. S1 for the nomenclature); their area is  $h \times h$  and they are 211  
 150 submitted to a total pressure force  $Ph^2$  in the direction of the main axis of the cell, balanced by the tension from the lateral 212  
 151 walls. Let  $\sigma$  be the common (scalar) stress in the walls; the wall thickness is  $w$  so their cross section is  $h \times w$  and therefore 213  
 152 they each exert a force  $\sigma hw$  on the base faces. To be coherent with the bidimensional model we propose, we consider that the 214  
 153 top and bottom lateral faces bear no stress and the balance of forces leads to 215

$$154 \quad Ph^2 = 2\sigma hw \quad 216$$

155 and therefore the balance of forces leads to  $P = 2\frac{w}{h}\sigma$ . Finally, thanks to this equation and the identity Eq. (S1), the 217  
 156 Lockhart-Ortega model (eqs. (1), (3) in main text) is reduced to the following differential equation for  $P$ : 218

$$157 \quad \frac{1}{E} \frac{dP}{dt} + \phi^w (P - P^Y)_+ = \phi^a (P^M - P), \quad [S2] \quad 219$$

160 where  $\phi^a = \frac{AL^a}{V}$  has been introduced in the main text; in order to keep the calculations as simple as possible, Lockhart made 221  
 161 the assumption that the area of the base faces is negligible compared to the area  $A = 4hl$  of the lateral faces (see Fig. S1). 222  
 162 Note that the cell volume is  $V = h^2 l$  and therefore the ratio  $A/V = 4/h$  is constant. 223

163 Let's study the transient behaviour of equation Eq. (S2), from an initial condition  $P(t = 0) = 0$ : 224  
 164 225

- 165 • Elastic regime: first,  $P$  is below  $P^Y$  and the plastic rate is zero; Eq. (S2) becomes 226  
 166 227

$$167 \quad \lambda^a \frac{dP}{dt} + P = P^M, \quad 228$$

168 where  $\lambda^a = \frac{1}{\phi^a E}$  is a characteristic time. The solution is 229  
 169 230

$$170 \quad P = P^M (1 - \exp(-t/\lambda^a)). \quad 231$$

171 The relative growth rate is 232  
 172 233

$$173 \quad \dot{\gamma} = \phi^a P^M \exp(-t/\lambda^a). \quad 234$$

- 174 • Plastic regime: the plastic regime starts when  $P = P^Y$ , at  $t^0 = \lambda^a \log\left(\frac{P^M}{P^M - P^Y}\right)$ . The equation Eq. (S2) becomes: 235  
 175 236  
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$$179 \quad \frac{1}{E} \frac{dP}{dt} + (\phi^a + \phi^w)P = \phi^a P^M + \phi^w P^Y, \quad 240$$

180 and equivalently 241  
 181 242

$$182 \quad \lambda^{aw} \frac{dP}{dt} + P = \alpha^a P^M + (1 - \alpha^a) P^Y, \quad 243$$

249 where  $\lambda^{aw} = \frac{1}{(\phi^a + \phi^w)\bar{E}}$  is a characteristic time. The solution is

$$250 \quad P = \alpha^a P^M + (1 - \alpha^a) P^Y - \alpha^a (P^M - P^Y) \exp((t^0 - t)/\lambda^{aw}), \quad [S3]$$

$$251 \quad \dot{\gamma} = \frac{\phi^a \phi^w}{\phi^a + \phi^w} (P^M - P^Y) - \frac{(\phi^a)^2}{\phi^a + \phi^w} (P^M - P^Y) \exp((t^0 - t)/\lambda^{aw}). \quad [S4]$$

256 The stationary solution is

$$257 \quad P^* = \alpha^a P^M + (1 - \alpha^a) P^Y \quad [S5]$$

$$258 \quad \dot{\gamma}^* = \frac{\phi^a \phi^w}{\phi^a + \phi^w} (P^M - P^Y). \quad [S6]$$

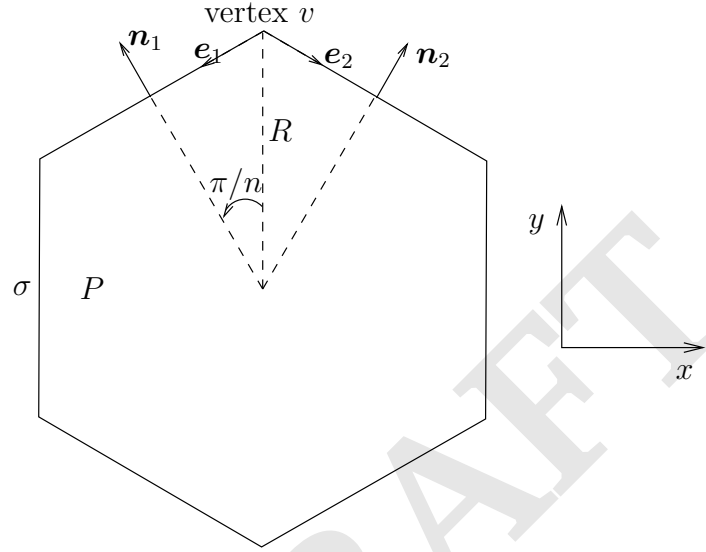


Fig. S2. Geometrical parameters for the single polygonal cell model.

287 **Single polygonal cell.** We consider a regular convex polygon of radius  $R$  with  $n$  edges that represents a cell (see Fig. S2).

288 **Mechanical equilibrium.** Let  $\sigma$  be the stress in the walls and  $P$  the pressure inside the cell; the outside pressure is set to zero.  
 289 The length of the edges is  $2R \sin(\pi/n)$ , and the walls are given a height  $h$  and a thickness  $w$ ; therefore the stresses are exerted  
 290 on a surface  $hw$ ; the contribution of pressure on vertex  $v$  is  $\frac{1}{2} P 2hR \sin(\pi/n)(\mathbf{n}_1 + \mathbf{n}_2)$ . Therefore, the balance of forces on  
 291 vertex  $v$  writes:

$$292 \quad \frac{1}{2} P 2hR \sin(\pi/n)(\mathbf{n}_1 + \mathbf{n}_2) + \sigma hw(\mathbf{e}_1 + \mathbf{e}_2) = 0.$$

295 The normal vectors are

$$296 \quad \mathbf{n}_1 = (-\sin(\pi/n), \cos(\pi/n)) \quad \text{and} \quad \mathbf{n}_2 = (\sin(\pi/n), \cos(\pi/n)).$$

298 The tangent vectors are

$$299 \quad \mathbf{e}_1 = (-\cos(\pi/n), -\sin(\pi/n)) \quad \text{and} \quad \mathbf{e}_2 = (\cos(\pi/n), -\sin(\pi/n)).$$

302 By symmetry, the  $x$  component of the resulting force is zero; the projection of the balance of forces on  $y$  axis yields

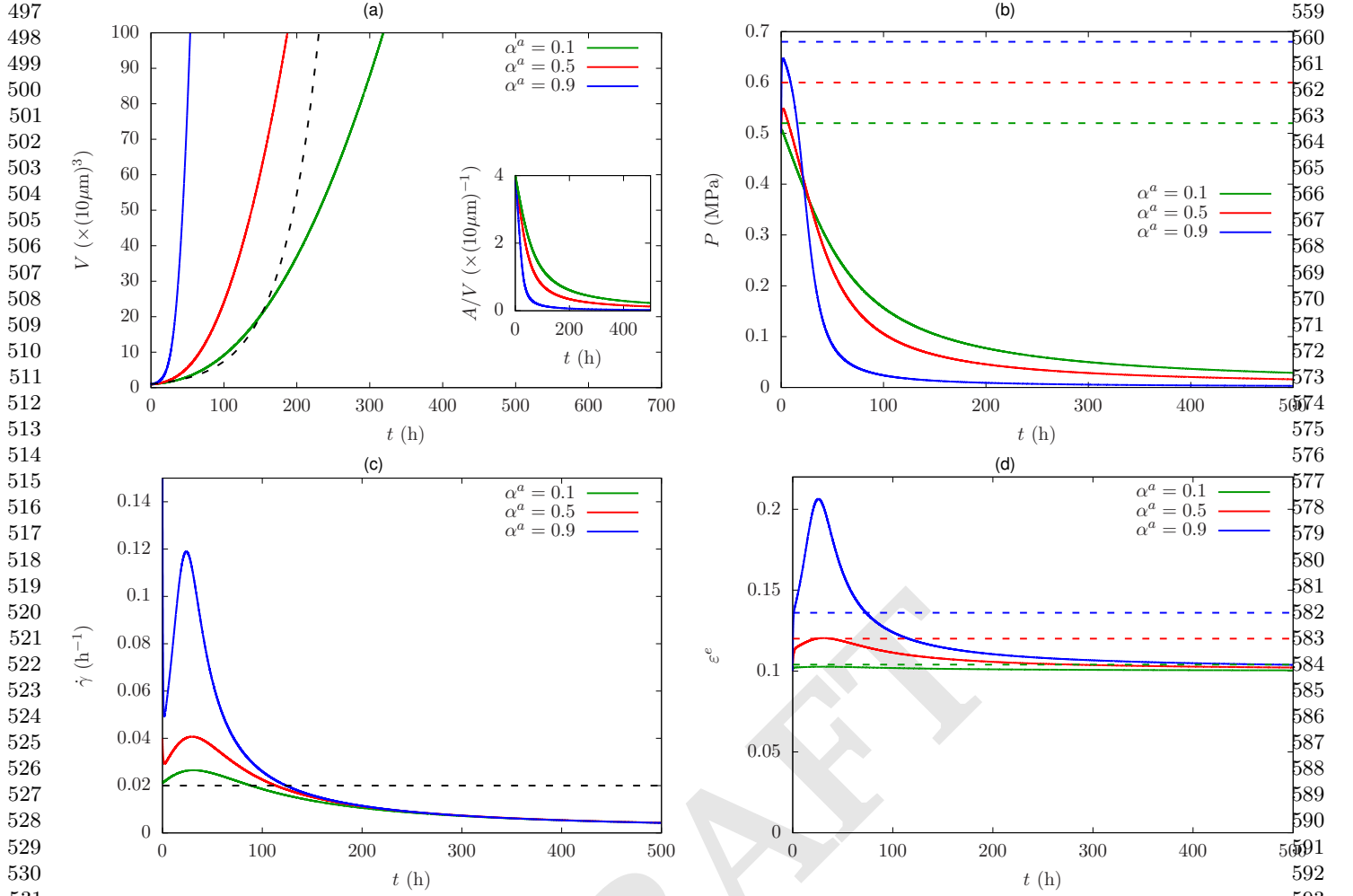
$$303 \quad 2PhR \sin(\pi/n) \cos(\pi/n) - 2\sigma hw \sin(\pi/n) = 0,$$

306 and

$$307 \quad P = \frac{w}{R \cos(\pi/n)} \sigma. \quad [S7]$$

309 When  $n \rightarrow \infty$ ,  $\cos(\pi/n) \rightarrow 1$  and we recover the Laplace law.

373 **Flux equation.** The surface of the polygon is 435  
374 436  
375  $S_n = n \times 2R \sin(\pi/n) R \cos(\pi/n)/2 = R^2 n \sin(\pi/n) \cos(\pi/n).$  437  
376 438  
377 The volume of the cell is  $V = S_n h$ , so the volume variation is 439  
378 440  
379  $\frac{dV}{dt} = 2hR \frac{dR}{dt} n \sin(\pi/n) \cos(\pi/n).$  441  
380 442  
381 The perimeter of the polygon is  $n \times 2R \sin(\pi/n)$  so the lateral area of the cell is 443  
382 444  
383  $A = 2nhR \sin(\pi/n).$  445  
384 446  
385 Note that the ratio  $A/V$  is not constant: 447  
386 448  
387  $\frac{A}{V} = \frac{2}{R \cos(\pi/n)}.$  449  
388 450  
389 Finally, the flux equation writes 451  
390 452  
391  $2hR \frac{dR}{dt} n \sin(\pi/n) \cos(\pi/n) = n2hR \sin(\pi/n) L(P^M - P),$  453  
392 454  
393 which yields 455  
394  $\frac{dR}{dt} = \frac{L}{\cos(\pi/n)} (P^M - P)$  [S8] 456  
395 457  
396 **Wall rheology.** Let  $\varepsilon^e$  be the elastic deformation of the walls; it is related to the stress by the constitutive equation  $\sigma = E\varepsilon^e$  458  
397 where  $E$  is the elastic modulus. The length of the edges is  $l = 2R \sin(\pi/n)$  and therefore the strain rate of the edges is 459  
398  $\frac{1}{l} \frac{dl}{dt} = \frac{1}{R} \frac{dR}{dt}$ . The rheological behaviour of the walls is given by 460  
399 461  
400  $\frac{1}{R} \frac{dR}{dt} = \frac{d\varepsilon^e}{dt} + \Phi^w E \max(0, \varepsilon^e - \varepsilon^Y),$  [S9] 462  
401 463  
402 or equivalently 464  
403 465  
404  $\frac{1}{R} \frac{dR}{dt} = \frac{1}{E} \frac{d\sigma}{dt} + \Phi^w \max(0, \sigma - \sigma^Y),$  [S10] 466  
405 467  
406 where  $\varepsilon^Y$  (resp.  $\sigma^Y$ ) is a yield elastic deformation (resp. stress). 468  
407 469  
408 **Numerical results.** The problem to solve is reduced to a set of two differential equations. It is numerically solved with the 470  
409 `odeint` routine from the `python` library `scipy`. 471  
410 We study the growth of a hexagonal cell ( $n = 6$ ) growing from an initial state where the elastic deformation of the walls is 472  
411 set to the threshold value, in order to bypass the pure elastic regime; computations are run over a long time scale. We want to 473  
412 study how this models compares to Lockhart-Ortega when the relative importance of fluxes and wall synthesis varies; to this 474  
413 end, we run three simulations with  $\alpha^a = 0.1, 0.5, 0.9$ . Let  $R_0 = 10\mu\text{m}$  be the initial radius of the cell, then  $P^Y = \frac{w}{R_0 \cos(\pi/6)} E\varepsilon^Y$  475  
414 is a representative value for the yield turgor of a hexagonal cell. The value  $\varepsilon^Y = 0.1$  is chosen accordingly to experimental 476  
415 observations where wall deformations can be of the order of 10%; then we choose  $E$  such that  $P^Y = 0.5$  MPa, which sets an 477  
416 order of magnitude for the initial turgor of the cell, close to observed experimental data. We choose  $P^M = 0.7$  MPa so that it 478  
417 is above  $P^Y$ . Finally, we can use the Lockhart's prediction Eq. (S6) as an order of magnitude of the relative growth rate; we 479  
418 choose  $\dot{\gamma}^* = 2\% \cdot \text{h}^{-1}$ . Then, a given value of  $\alpha^a$  (evaluated with the initial area of the cell) sets a unique value of  $L^a$  and  $\phi^w$ . 480  
419 At the onset of the simulation, walls start to extend irreversibly and plastic growth occurs. Fig. S3a,c shows that the volume 481  
420 increases faster for large values of  $\alpha^a$ , although we have chosen the parameters so that the Lockhart model predicts a constant 482  
421 and common value of  $\dot{\gamma}$ . Fig. S3b shows that  $P$  is initially close to Lockhart predictions  $P^*$  but decreases fastly to zero; the 483  
422 fast decrease of  $P$  coincides with peaks of  $\dot{\gamma}$  (Fig. S3c) above the value  $\dot{\gamma}^*$  with a higher peak for larger values of  $\alpha^a$ ; the elastic 484  
423 deformation  $\varepsilon^e$  (Fig. S3d) is not constant either, with a large peak above the Lockhart-Ortega prediction for  $\alpha^a = 0.9$ . For all 485  
424 values of  $\alpha^a$ ,  $\varepsilon^e$  converges toward the threshold  $\varepsilon^Y$ . 486  
425 487  
426 **Two-cells model.** The geometry and notations of the two-cells model is recalled in Fig. S4. Gathering the flux equation (Eq. 8 488  
427 from main text) and the wall mechanics equation (Eq. 1 from main text) with  $\frac{dP}{dt} = 0$ , we get 489  
428 490  
429  $\phi^a (P^M - P_0) + \frac{\phi^s}{2} (P_1 - P_0) - \phi^w (P_0 - P_0^Y)_+ = 0$  [S11] 491  
430 492  
431  $\phi^a (P^M - P_1) - \frac{\phi^s}{2} (P_1 - P_0) - \phi^w (P_1 - P_1^Y)_+ = 0.$  [S12] 493  
432 494  
433 495  
434 First, we assume that both cells are growing ( $P_i > P_i^Y, i = 0, 1$ ). 496



**Fig. S3.** Growth of a single hexagonal cell for three different values of  $\alpha^a$ : time evolution of volume (inset: ratio area/volume) (a), turgor (b), relative growth rate (c), and elastic deformation of the walls (d). The dashed lines correspond to the solution of the Lockhart model; note that the chosen sets of parameters lead to the constant and equal value  $\dot{\gamma}^* = 2\% \cdot \text{h}^{-1}$ , and to the same evolution of volume.

**First regime:**  $P_i > P_i^Y, i = 0, 1$ . Adding Eq. (S11) and Eq. (S12) we get:

$$\bar{P} = \alpha^a P^M + (1 - \alpha^a) \bar{P}^Y, \quad [\text{S13}]$$

where  $\alpha^a = \frac{\phi^a}{\phi^a + \phi^w}$ ,  $\bar{P} = \frac{P_0 + P_1}{2}$ . With Eq. 1 from main text, we get

$$\bar{\gamma} = \frac{\phi^a \phi^w}{\phi^a + \phi^w} (P^M - \bar{P}^Y), \quad [\text{S14}]$$

where  $\bar{\gamma} = \frac{\dot{\gamma}_0 + \dot{\gamma}_1}{2}$ . Therefore, the gathering of two cells behaves the same as one cell if one considers the mean values.

Then, we examine the heterogeneities in turgor and growth rate. Subtracting Eq. (S11) to Eq. (S12), we get

$$\Delta P = \frac{\phi^w}{\phi^a + \phi^s + \phi^w} \Delta P^Y.$$

Let

$$\alpha^s = \frac{\phi^s}{\phi^s + \phi^a}.$$

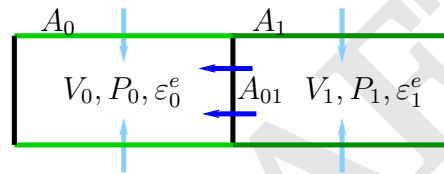
Then the previous expression becomes

$$\Delta P = \frac{(1 - \alpha^a)(1 - \alpha^s)}{1 - \alpha^s + \alpha^a \alpha^s} \Delta P^Y. \quad [\text{S15}]$$



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**Fig. S4.** Two cells model: symplasmic flows (dark blue arrows) occur through the contact surface  $A_{01}$ ; apoplasmic flows (light blue arrows) occur through the surfaces  $A_0$  and  $A_1$ . Growth is restricted to the green edges: cell 0 (in dark green) has stiffer walls than cell 1 (in light green).

745 As  $(1 - \alpha^a)(1 - \alpha^s) = 1 - \alpha^a - \alpha^s + \alpha^a \alpha^s < 1 - \alpha^s + \alpha^a \alpha^s$ , we find that turgor difference  $\Delta P$  cannot exceed the value  $\Delta P^Y$ . 807  
 746 When  $\alpha^s = 0$  (symplasmic fluxes negligible with respect to apoplasmic ones), then  $\Delta P = (1 - \alpha^a)\Delta P^Y$ ; when  $\alpha^s > 0$ , 808  
 747 symplasmic fluxes tend to reduce the turgor heterogeneity between cells. 809

748 With Eq. 7 from main text we get then 810

$$749 \quad \Delta \dot{\gamma} = \frac{(\phi^a + \phi^s)\phi^w}{\phi^a + \phi^s + \phi^w} \Delta P^Y, \quad [S16] \quad 811$$

750 where  $\Delta \dot{\gamma} = \frac{\dot{\gamma}_0 - \dot{\gamma}_1}{2}$ . Note that this expression is valid iff  $P_1 > P_1^Y$  or equivalently  $\dot{\gamma}_1 > 0$ . The limit  $\dot{\gamma}_1 = 0$  corresponds to the 812  
 751 situation where cell 0 is growing in such a way that it prevents cell 1 to grow because of the symplasmic fluxes between them. 813  
 752 We examine how this situation can occur depending on the values of the sumplasmic conductivity  $\phi^s$  and the other parameters. 814  
 753 We find that 815

$$754 \quad P_1 > P_1^Y \iff \frac{\phi^a + \phi^s}{\phi^a + \phi^s + \phi^w} \frac{\Delta P^Y}{P^M - \bar{P}^Y} < \frac{\phi^a}{\phi^a + \phi^w} \quad 816$$

$$755 \quad \iff \frac{\alpha^a}{1 - (1 - \alpha^s)\alpha^a} \rho < \alpha^a \quad 817$$

$$756 \quad \iff \alpha^s < \frac{1 - \rho}{1 - \alpha^a}. \quad 818$$

757 For instance,  $P_0^Y = 0.25$  MPa,  $P_1^Y = 0.5$  MPa, and  $P^M = 0.625$  MPa yields  $\rho = 0.5$ . The hypothesis of this study 819  
 758 ( $P_0^Y < P_1^Y < P^M$ ) corresponds to the condition  $\rho \in [0, 1]$ . Note that if  $\alpha^a > \rho$ , then  $\frac{1 - \rho}{1 - \alpha^a} > 1$ , and the condition is verified 820  
 759 whatever the value of  $\alpha^s$ ; if  $\alpha^s = 1 - \rho$ , the condition is equivalent to  $\alpha^a > 0$ , which is also always verified. Fig. S4a) 821  
 760 recapitulates the regions of the parameters space  $\alpha^a \times \alpha_s$  where the condition is verified, for different values of  $\rho$ . The size of 822  
 761 the region  $\dot{\gamma}_1 = 0$  increases as  $\rho$  gets closer to 1. 823

762 **Second regime:  $P_0 > P_0^Y$  and  $P_1 < P_1^Y$ .** In this case, eqs. Eq. (S11) and Eq. (S12) turn into 824  
 763 825

$$764 \quad \phi^a(P^M - P_0) + \frac{\phi^s}{2}(P_1 - P_0) - \phi^w(P_0 - P_0^Y) = 0 \quad [S17] \quad 826$$

$$765 \quad \phi^a(P^M - P_1) - \frac{\phi^s}{2}(P_1 - P_0) = 0. \quad [S18] \quad 827$$

766 Eq. (S18) leads to 828

$$767 \quad P_1 = (1 - \tilde{\alpha}^s)P^M + \tilde{\alpha}^s P_0, \quad [S19] \quad 829$$

768 where  $\tilde{\alpha}^s = \frac{\phi^s}{2\phi^a + \phi^s}$ . Adding eqs. Eq. (S17) and Eq. (S18) leads to 830  
 769 831

$$770 \quad P_0(\phi^a + \phi^w) = 2\phi^a P^M + \phi^w P_0^Y - \phi^a((1 - \tilde{\alpha}^s)P^M + \tilde{\alpha}^s P_0), \quad 832$$

771 then, 833

$$772 \quad P_0(\phi^a(1 + \tilde{\alpha}^s) + \phi^w) = \phi^a(1 + \tilde{\alpha}^s)P^M + \phi^w P_0^Y, \quad 834$$

773 and finally 835

$$774 \quad P_0 = \alpha^{as} P^M + (1 - \alpha^{as}) P_0^Y, \quad [S20] \quad 836$$

775 where 837

$$776 \quad \alpha^{as} = \frac{\phi^{as}}{\phi^{as} + \phi^w} \quad \text{and} \quad \phi^{as} = \phi^a(1 + \tilde{\alpha}^s). \quad 838$$

777 Hence, thanks to the symplasmic fluxes from its neighbour cell 1, cell 0 benefits from an enhanced access to the apoplasmic 839  
 778 fluxes by a factor  $\phi^{as}/\phi^a = 1 + \tilde{\alpha}^s$ . Then, from Eq. 1 in main text, the relative growth rate of cell 0 is 840  
 779 841

$$780 \quad \dot{\gamma}_0 = \frac{\phi^{as}\phi^w}{\phi^{as} + \phi^w} (P^M - P_0^Y). \quad [S21] \quad 842$$

781 By hypothesis, the growth rate of cell 1 is zero, and we can compute the heterogeneity in turgor: from Eq. (S19), we find that 843  
 782 844

$$783 \quad \Delta P = \frac{1 - \tilde{\alpha}^s}{2} (P^M - P_0), \quad 845$$

784 and hence 846

$$785 \quad \Delta P = \frac{1}{2} (1 - \tilde{\alpha}^s)(1 - \alpha^{as})(P^M - P_0^Y). \quad [S22] \quad 847$$

869	<b>2. Numerical resolution of the 2D multicellular model</b>	931
870	<b>Structure of the mathematical problem.</b> Thanks to the geometrical constraint of uni-directional growth, the Lockhart-Ortega	932
871	is very simple to resolve. The identity between the relative growth rate of the cell and the strain rate of the walls allows to	933
872	couple the equation that describes fluxes, and the equation that describes walls synthesis. Then the stress in the walls and the	934
873	pressure inside the cell are linked by the mechanical equilibrium. Finally there is only one independent variable (pressure for	935
874	instance) and the model can be solved analytically.	936
875	Conversely, in the bidimensionnal model we propose, the properties of a given wall (elongation rate and elastic deformation)	937
876	cannot be directly linked to the properties of the adjacent cells (growth rate and pressure). Hence a new strategy has to be	938
877	developped. First, we emphasize the strong coupling between fluxes and mechanics: the motion of the vertices is prescribed by	939
878	the mechanical equilibrium (Eq. 11 from main text) between pressure forces and elastic forces; meanwhile, a displacement	940
879	of the vertices can cause a variation of volume of several cells, which has to be balanced by water fluxes (Eq. 10 from main	941
880	text); water fluxes are limited by the finite permeability of the walls, which sets a constraint on possible variations of volume.	942
881	Similarly, any variation in the length of the walls leads to a modification of their elastic deformation (Eq. 7 from main text).	943
882	Another way to understand this problem is to consider it as the minimization of mechanical energy (mechanical equilibrium	944
883	Eq. 11 from main text) under two constraints on the position of the vertices, through the volumes of the cells (Eq. 10 from	945
884	main text) and the lengths of the edges (Eq. 7 from main text). This kind of problem is often encountered in mechanics, <i>e.g.</i>	946
885	solid friction, contact mechanics, or incompressible fluid mechanics; a powerfull theoretical and practical tool to solve this is the	947
886	method of lagrangian multipliers. For instance, in the context of incompressible fluid mechanics, the constraint of volume	948
887	conservation is relaxed by pressure that acts as a lagragian multiplier. Physically, the pressure adjusts itself so that both the	949
888	constraint and the mechanical equilibrium are satisfied. The model we propose exhibits the same structure, as pressure will	950
889	adjust to both fluxes and mechanical constraints. However, the system here is discrete, and the flux equation (Eq. 10 in main	951
890	text) is linear with respect to pressure, so it can be reduced to a linear system. We will take advantage of this for the resolution	952
891	of the model.	953
892		954
893	<b>Resolution algorithm.</b>	955
894		956
895	<b>Volumes and lengths as functions of the positions of the vertices.</b> First, we express volumes and lengths as functions of	957
896	the positions of the vertices. Let $N_v$ be the number of vertices and $\mathbf{X} \in \mathbb{R}^{2N_v}$ the vector of the positions of all the vertices.	958
897	The volume of a cell $i$ is $V_i = S_i h$ where $S_i$ is its surface. As cells are non intersecting polygons, their signed surface is given by	959
898	the general formula	960
899		961
900	$S_i = \frac{1}{2} \sum_{k=0}^{n_i-1} (x_k y_{k+1} - x_{k+1} y_k),$	[S23] 962
901		963
902	where $n_i$ is the number of vertices of cell $i$ , $(x_k, y_k)_{k=0, \dots, n_i-1}$ are the coordinates of the vertices of the cell $i$ in counterclockwise	964
903	order, and we set $(x_{n_i}, y_{n_i}) = (x_0, y_0)$ . Let $N_c$ be the number of cells and $\mathbf{V} \in \mathbb{R}^{N_c}$ the vector of all the cells volumes; thanks	965
904	to Eq. (S23), it can be expressed as a function of $\mathbf{X}$ and its gradient $\nabla_{\mathbf{X}} \mathbf{V}$ with respect to $\mathbf{X}$ can be computed. Then the time	966
905	derivative of $\mathbf{V}$ expresses as	967
906	$\frac{d\mathbf{V}}{dt} = \nabla_{\mathbf{X}} \mathbf{V} \frac{d\mathbf{X}}{dt}.$	968
907		969
908	Note here that $\nabla_{\mathbf{X}} \mathbf{V}$ is a $N_c \times 2N_e$ matrix and $\frac{d\mathbf{X}}{dt}$ is a $2N_e$ vector, so their product is well defined and has the correct	970
909	dimension.	971
910	Similarly, the length of a segment $k$ with two vertices $v_1 = (x_1, y_1)$ and $v_2 = (x_2, y_2)$ at its ends is	972
911		973
912	$l_k = \sqrt{(x_1 - x_2)^2 + (y_1 - y_2)^2}.$	[S24] 974
913	Let $N_e$ be the number of edges and $\mathbf{l} \in \mathbb{R}^{N_e}$ the vector of all the edges lengths; thanks to Eq. (S24), it can be expressed as a	975
914	function of $\mathbf{X}$ and its gradient $\nabla_{\mathbf{X}} \mathbf{l}$ with respect to $\mathbf{X}$ can be computed. Then the time derivative of $\mathbf{l}$ expresses as	976
915		977
916	$\frac{d\mathbf{l}}{dt} = \nabla_{\mathbf{X}} \mathbf{l} \frac{d\mathbf{X}}{dt}.$	978
917		979
918	<b>Time discretisation.</b> Time is discretized using a fixed time step $\Delta t$ and the time derivatives are approximated by the 1st	980
919	order Euler scheme, for instance:	981
920		982
921	$\frac{d\mathbf{X}}{dt}(t) \approx \frac{\mathbf{X}(t + \Delta t) - \mathbf{X}(t)}{\Delta t}.$	983
922	Let $\boldsymbol{\varepsilon} \in \mathbb{R}^{N_e}$ be the vector of all the elastic deformations of the edges. Let $\mathbf{X}^0 = \mathbf{X}(0)$ and $\boldsymbol{\varepsilon}^0 = \boldsymbol{\varepsilon}(0)$ be some initial conditions.	984
923	We construct successive approximations of the solution at times $t_n = n\Delta t$ for $n > 0$ by solving at each time step the mechanical	985
924	equilibrium (Eq. 11 from main text) along with the discretized versions of flux (Eq. 10 from main text) and wall rheology (Eq. 7	986
925	from main text) equations: let $\mathbf{P} \in \mathbb{R}^{N_c}$ be the vector of all the cells pressures; these equations can be written in a matrix form:	987
926		988
927	$\nabla_{\mathbf{X}} \mathbf{V}(\mathbf{X}^{n+1}) \frac{\mathbf{X}^{n+1} - \mathbf{X}^n}{\Delta t} = M_P \mathbf{P}^{n+1} + \mathbf{b}_P,$	[S25] 989
928		990
929	$\frac{\boldsymbol{\varepsilon}^{n+1} - \boldsymbol{\varepsilon}^n}{\Delta t} + \beta^n \boldsymbol{\varepsilon}^{n+1} = \frac{1}{l(\mathbf{X}^{n+1})} \nabla_{\mathbf{X}} \mathbf{l}(\mathbf{X}^{n+1}) \frac{\mathbf{X}^{n+1} - \mathbf{X}^n}{\Delta t}.$	[S26] 991
930		992

993 where  $M_P$  is a  $N_i \times N_i$  matrix, with the following non-zero coefficients: 1055

$$994 \quad M_P(i, i) = A_i L_i^a - \sum_{j \in n(i)} A_{ij} L_{ij}^s, \quad \forall i = 1, \dots, N_c, \quad 1056$$

$$995 \quad M_P(i, j) = A_{ij} L_{ij}^s, \quad \forall i = 1, \dots, N_c, \quad \forall j \in n(i), \quad 1057$$

996  
997  
998 with  $\mathbf{b}_P \in \mathbb{R}^{N_c}$  is defined by its coefficients 1061

$$1000 \quad \mathbf{b}_p(i) = A_i L_i^a P^M, \quad \forall i = 1, \dots, N_c. \quad 1062$$

1001 Note here that the model implies no time derivative of the pressure, so that  $\forall n > 0$ ,  $\mathbf{P}^{n+1}$  can be computed without the 1063  
1002 knowledge of  $\mathbf{P}^n$ , and the initial value of the pressure is not needed. 1065

1003 In addition,  $\beta^n$  is the  $N_e \times N_e$  diagonal matrix with components  $\beta^n(k, k) = \frac{2w}{h} \phi_k^w E_k \max\left(0, \frac{\varepsilon_k^n - \varepsilon_k^Y}{\varepsilon_k^n}\right)$  for  $k = 1, \dots, N_e$ , 1066  
1004 and for the purpose of notation,  $\frac{1}{l}$  is the  $N_e \times N_e$  diagonal matrix with components  $1/l_k$ . Note here that the variables  $\beta^n$  are 1068  
1005 taken at time step  $n$  so that they are considered as constants at time step  $n + 1$  and the equation Eq. (S26) is linear with 1069  
1006 respect to the unknown  $\varepsilon^{n+1}$ . 1070

1007 **Pressure and elastic deformation as functions of the position of the vertices.** Thanks to this time discretization, we see 1072  
1008 that at each time step, the unknown pressure  $\mathbf{P}^{n+1}$  and elastic deformation  $\varepsilon^{n+1}$  are defined through the linear equations 1073  
1009 Eq. (S25) and Eq. (S26) which can be easily inverted, which allows to express both these variables as functions of the spatial 1074  
1010 unknown  $\mathbf{X}^{n+1}$ . 1075

1011 First, from equation Eq. (S25): 1076

$$1012 \quad P(\mathbf{X}^{n+1}) = \frac{1}{\Delta t} M_P^{-1} \nabla_{\mathbf{X}} \mathbf{V}(\mathbf{X}^{n+1}) \mathbf{X}^{n+1} - M_P^{-1} \left( \frac{1}{\Delta t} \nabla_{\mathbf{X}} \mathbf{V}(\mathbf{X}^{n+1}) \mathbf{X}^n - \mathbf{b}_P \right). \quad 1077$$

1013 Then, using Eq. (S26): 1080

$$1014 \quad \varepsilon(\mathbf{X}^{n+1}) = \frac{1}{\Delta t} M_\varepsilon^{-1} \frac{1}{l(\mathbf{X}^{n+1})} \nabla_{\mathbf{X}} l(\mathbf{X}^{n+1}) \mathbf{X}^{n+1} - \frac{1}{\Delta t} M_\varepsilon^{-1} \left( \frac{1}{l(\mathbf{X}^{n+1})} \nabla_{\mathbf{X}} l(\mathbf{X}^{n+1}) \mathbf{X}^n - \varepsilon^n \right), \quad 1081$$

1015 where  $M_\varepsilon = \frac{1}{\Delta t} I_{N_e} + \beta^n$ . 1082

1016 **Structure of the resolution algorithm** Thanks to the two previous steps, we are now able to propose a algorithm for the resolution 1086  
1017 of the model. 1087

- 1018 • Initialization: Define  $\mathbf{X}^0 \in \mathbb{R}^{2N_v}$  and  $\varepsilon^0 \in \mathbb{R}^{N_e}$  1088
- 1019 •  $\forall n \geq 0$ , assuming  $\mathbf{X}^n$  and  $\varepsilon^n$  are known, let  $\mathbf{F}^n : \mathbb{R}^{2N_v} \rightarrow \mathbb{R}^{2N_v}$  be the function such that  $\forall v = 0, \dots, N_v - 1$ , 1089

$$1020 \quad \begin{pmatrix} F_{2v+1}^n(\mathbf{X}) \\ F_{2v+2}^n(\mathbf{X}) \end{pmatrix} = \frac{1}{2} \sum_{k \in f(v)} \Delta_k P(\mathbf{X}) A_k(\mathbf{X}) \mathbf{n}_k(\mathbf{X}) + \sum_{k \in f(v)} E_k \varepsilon_k^e(\mathbf{X}) a_k(\mathbf{X}) \mathbf{e}_{k,v}(\mathbf{X}), \quad 1090$$

1021 where  $F_k^n$  is the  $k$ -th component of  $\mathbf{F}^n$ , and with the same notations as in Eq. 11 from main text;  $\mathbf{P}(\mathbf{X})$  and  $\varepsilon(\mathbf{X})$  are 1091  
1022 the functions of  $\mathbf{X}$  given by Eq. (S27) and Eq. (S28). Then, the new position of the vertices  $\mathbf{X}^{n+1}$  is the solution of the 1092  
1023 equation 1093

$$1024 \quad \mathbf{F}^n(\mathbf{X}) = 0. \quad 1094$$

1025 **Resolution of Eq. (S29).** This is the last and most critical step of the resolution algorithm. The problem of computing the roots 1100  
1026 of a multidimensional non linear function is often encountered in the mechanical modelling of complex multibody systems, 1101  
1027 and a method of choice for the resolution is the Newton algorithm [1]. It is a iterative process which derives from a Taylor 1102  
1028 expansion about a current point  $\mathbf{u}^k$ : 1103

$$1029 \quad \mathbf{F}^n(\mathbf{u}^{k+1}) = \mathbf{F}^n(\mathbf{u}^k) + J(\mathbf{u}^k)(\mathbf{u}^{k+1} - \mathbf{u}^k) + o(\mathbf{u}^{k+1} - \mathbf{u}^k), \quad 1104$$

1030 where  $J(\mathbf{u}^k)$  is the jacobian matrix of function  $\mathbf{F}^n$ . The new value  $\mathbf{u}^{k+1}$  is obtained by setting the right-hand side to zero and 1105  
1031 neglecting the high order term, and then solving the linear system: 1106

$$1032 \quad J(\mathbf{u}^k) \delta \mathbf{u}^k = -\mathbf{F}^n(\mathbf{u}^k), \quad \mathbf{u}^{k+1} = \mathbf{u}^k + \delta \mathbf{u}^k. \quad 1107$$

1033 With the initial value  $\mathbf{u}^0 = \mathbf{X}^n$ , iterations are run until a stopping criterium is met, for instance 1108  
1034 1109

$$1035 \quad \frac{\|\mathbf{F}^n(\mathbf{u}^k)\|}{\|\mathbf{F}^n(\mathbf{u}^0)\|} \leq tol_{res}, \quad 1110$$

1036 where  $tol_{res} > 0$  is a fixed value. Then one can set  $\mathbf{X}^{n+1} = \mathbf{u}^k$ . 1111

The computation of the jacobian matrix  $J(\mathbf{u}^k)$  is non trivial here because of the numerous non-linearities of function  $\mathbf{F}^n$ . Therefore we have chosen to use the Newton-Krylov variant of this algorithm, that avoids the computation of the jacobian without loosing efficiency [1].

However, Newton methods in general have only local convergence properties, which means that they need an initial guess close enough to the solution to be able to converge. This is critical for instance in the first time step of the simulation, because the initial conditions might be far from equilibrium, but also for further time steps. This lack of global convergence properties is often dealt with by adding a friction term proportional to the velocity and hence to the time derivative of the positions. With this method, the problem to solve at each time step becomes after time discretization: find  $\mathbf{X}$  such that

$$\mathbf{G}(\mathbf{X}) = \mathbf{F}^n(\mathbf{X}) - c \frac{\mathbf{X} - \mathbf{X}^n}{\Delta t} = 0,$$

where  $c > 0$  is a friction coefficient. This new problem is easier to solve with the Newton method, all the more that  $c$  is large. However, the root of  $\mathbf{G}$  might not satisfy the condition Eq. (S30), and in addition its value depends on the value of  $c$ . Therefore, instead of applying the Newton method to the function  $\mathbf{G}$ , we perform the following iterative process:

- Initialization:  $\mathbf{u}^0 = \mathbf{X}^n$
- Assuming  $\mathbf{u}^k$  is known, compute  $\mathbf{u}^{k+1}$  as the solution of

$$\mathbf{G}^k(\mathbf{u}^{k+1}) = 0, \quad [\text{S31}]$$

where  $\mathbf{G}^k(\mathbf{u}^{k+1}) = \mathbf{F}^n(\mathbf{u}^{k+1}) - c^k \frac{\mathbf{u}^{k+1} - \mathbf{u}^k}{\Delta t}$ , and the value  $c^k > 0$  will be adjusted to ensure a robust convergence (see below). This solution is computed thanks to the Newton method, with the tolerance  $tol_{res}/10$  in the stopping criterium.

- The iterations are stopped when  $\frac{\|\mathbf{F}^n(\mathbf{u}^k)\|}{\|\mathbf{F}^n(\mathbf{u}^0)\|} \leq tol_{res}$ . Then the choice  $\mathbf{X}^{n+1} = \mathbf{u}^k$  is an approximate solution of Eq. (S29).

In this algorithm, the choice of the friction coefficient  $c^k$  is not straightforward: a large value would ensure the convergence of subproblem Eq. (S31), but it would also slow down the convergence toward the solution of problem Eq. (S29). To avoid this, we choose a large initial value  $c^0$  and decrease it with the law  $c^{k+1} = c^k/2$ . This choice ensures a robust behaviour of the algorithm.

### 3. Sets of parameters used for the bump simulations

Let  $R_0 = 10\mu\text{m}$  be the initial radius of the cell, then  $P^Y = \frac{w}{R_0 \cos(\pi/6)} E \varepsilon^Y$  is a representative value for the yield turgor of a hexagonal cell. However we have observed that the effective threshold pressure is approximately twice lower in multicellular tissues and we have adapted the value of  $E$  accordingly: we choose  $E$  such that  $P^Y = 0.5$  MPa and multiplied this value by two to obtain a an order of magnitude for the initial turgor of the cell close to the target value 0.5 MPa. The value  $\varepsilon^Y = 0.1$  is chosen accordingly to experimental observations where wall deformations can be of the order of 10%. We choose two values for  $P^M$ : 0.55 MPa close to the threshold, and 0.7 MPa. Finally, we can use the Lockhart's prediction  $\dot{\gamma}^*$  (Eq.6 from main text) as an order of magnitude of the relative growth rate; we choose  $\dot{\gamma}^* = 2\% \cdot \text{h}^{-1}$ . Then, a given value of  $\alpha^a$  (evaluated with  $R = R_0$ ) sets a unique value of  $L^a$  and  $\phi^w$ . The table S1 recapitulates the sets of parameters used in this article, either with the control parameters

$$\varepsilon^Y, P^M, P^Y, \dot{\gamma}^*, \alpha^a, \quad [\text{S32}]$$

or equivalently with the actual parameters of the model

$$\varepsilon^Y, P^M, E, \Phi^w, L^a. \quad [\text{S33}]$$

The correspondance has been obtained with  $R_0 = 6.5\mu\text{m}$ .

## References

1. Knoll DA, Keyes DE (2004) Jacobian-free newton-krylov methods: a survey of approaches and applications. *J Comp Phys* 193:357–397.

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**Table S1. Parameters used for the bump simulation (see Fig. 3 in main text). The top part of the table refers to the control parameters Eq. (S32), and the bottom part to the actual parameters Eq. (S32) used in the 2D model. The rightmost parameters after the vertical double bar are specific to multicellular models as they quantify the water conductivity between neighbour cells. The geometrical parameters are  $h = 10\mu\text{m}$  and  $w = h/20$ .**

Control parameters	$\varepsilon^Y$	$P^M$ (MPa)	$P_6^Y$ (MPa)	$\dot{\gamma}^*$ ( $\text{h}^{-1}$ )	$\alpha^a$	$\alpha^s$
(REF)	0.1	0.7	0.5	$2 \cdot 10^{-2}$	0.1	0.9
(CC-)	0.1	0.7	0.5	$2 \cdot 10^{-2}$	0.1	0.1
(ALPHA+)	0.1	0.7	0.5	$2 \cdot 10^{-2}$	0.9	0.9
(PM-)	0.1	0.55	0.5	$0.5 \cdot 10^{-2}$	0.1	0.9
Actual parameters	$\varepsilon^Y$	$P^M$ (MPa)	$E$ (MPa)	$\Phi^w$ ( $\text{MPa}^{-1} \cdot \text{s}^{-1}$ )	$L^a$ ( $\text{m} \cdot \text{MPa}^{-1} \cdot \text{s}^{-1}$ )	$L^s$ ( $\text{m} \cdot \text{MPa}^{-1} \cdot \text{s}^{-1}$ )
(REF)	0.1	0.7	112.6	$2.8 \cdot 10^{-5}$	$8.7 \cdot 10^{-11}$	$7.8 \cdot 10^{-10}$
(CC-)	0.1	0.7	112.6	$2.8 \cdot 10^{-5}$	$8.7 \cdot 10^{-11}$	$9.6 \cdot 10^{-12}$
(ALPHA+)	0.1	0.7	112.6	$3.1 \cdot 10^{-6}$	$7.8 \cdot 10^{-10}$	$7.0 \cdot 10^{-9}$
(PM-)	0.1	0.55	112.6	$2.8 \cdot 10^{-5}$	$8.7 \cdot 10^{-11}$	$7.8 \cdot 10^{-10}$