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1 Strain- or Stress-sensing in mechanochemical 2 patterning by the phytohormone auxin

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7 **Abstract** Both chemical and mechanical fields are known to play a major
8 role in morphogenesis. In plants, the phytohormone auxin and its directional
9 transport are essential for the formation of robust patterns of organs, such
10 as flowers or leaves, known as phyllotactic patterns. The transport of auxin
11 was recently shown to be affected by mechanical signals, and conversely, auxin
12 accumulation in incipient organs affects the mechanical properties of the cells.
13 The precise interaction between mechanical fields and auxin transport, how-
14 ever, is poorly understood. In particular, it is unknown whether transport is
15 sensitive to the strain or to the stress exerted on a given cell. Here, we inves-
16 tigate the nature of this coupling with the help of theoretical models. Namely,
17 we introduce the effects of either mechanical stress or mechanical strain in a
18 model of auxin transport, and compare the patterns predicted with available
19 experimental results, in which the tissue is perturbed by ablations, chemical
20 treatments, or genetic manipulations. We also study the robustness of the pat-
21 terning mechanism to noise and investigate the effect of a shock that changes
22 abruptly its parameters. Although the model predictions with the two differ-
23 ent feedbacks are often indistinguishable, the strain-feedback seems to better
24 agree with some of the experiments. The computational modeling approach
25 used here, which enables us to distinguish between several possible mechani-
26 cal feedbacks, offers promising perspectives to elucidate the role of mechanics
27 in tissue development, and may help providing insight into the underlying
28 molecular mechanisms.

29 **Keywords** patterning · chemomechanical model · auxin transport · shoot
30 apical meristem

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1 Introduction

Understanding the formation of patterns in living organisms has long intrigued scientists [1]. This phenomenon has been investigated in a wide variety of species, such as zebrafish [23] or hydra [4]. We focus here on patterning in plants. The highly regular positioning of the organs in plant shoots, called phyllotaxis, relies on the patterns of the phytohormone auxin, whose local accumulation is necessary for the emergence of new organs [24, 18]. Auxin efflux is facilitated by the membrane-localized PIN FORMED1 (PIN1) protein [35, 51], which can be polarly (asymmetrically) distributed, inducing a directional transport of auxin, and resulting in well-defined auxin patterns. This transport is essential for the development of the plant. Indeed, knocking down the transporter activity, either genetically or chemically, leads to the absence of organ primordia at the shoot tip. Organ development can be then rescued by local application of exogenous auxin [34, 40, 41].

Several models have been proposed to describe the interaction between the hormone and its carrier in the context of the shoot apex, with the major assumption that the transporters allocation between the different sides of a cell is driven by auxin fluxes [49] or by auxin concentrations [48, 22, 44, 47]. Here we focus on the second assumption, which has received more attention, because how the auxin concentration regulates the transporters remains unclear.

Since morphogenesis relies on changes in the structural elements of the organism, biochemical patterns must also influence the mechanics of these structural elements, in plants [16] and in animals [19, 9, 39]. Conversely, mechanics can feedback on biochemical processes [16, 19, 20, 45], including gene expression [11] and cell fate [3]. Plants are well suited to study the coupling between biochemical and biophysical processes as hydrodynamic cell pressure generates tremendous forces and results in a high tension in the polysaccharide-based walls surrounding cells [13, 16]. Recent progress in computational approaches [8] and in the measurements of cell mechanics [28] has fostered a renewed interest in the mechanics of plant morphogenesis.

Notably, at the shoot apex, organogenesis is associated with a decrease in the stiffness of the cell wall [36] and likely with a reduced mechanical anisotropy [46]. In the context of phyllotaxis, the coupling between mechanics and chemistry was explored theoretically [33] and mechanics has been proposed to regulate the transport of auxin in the shoot apical meristem [17]. A mechanical feedback was postulated, that was capable of generating a local accumulation of auxin [17]. Based on preferential localization of PIN1 in the regions of the plasma membrane in contact with the wall with highest mechanical stress, this mechanical feedback is supported by several experiments [17, 32, 7]. However, whether PIN1 polarity is driven by the stress or by the strain of the cell wall remains unclear. More generally, the issue of stress- or strain-sensing calls for further investigation, especially in the context of plant development [31].

Here we address this issue by investigating a model of mechanical feedback on auxin transport, whose predictions can be compared with experimental results. We also study the robustness of the pattern formation mechanism with

76 respect to noise and abrupt changes in some of the biochemical parameters.
77 The predictions of the two mechanisms do not differ much. A feedback based
78 on strain nonetheless leads to more realistic predictions, and to a patterning
79 mechanism which is less sensitive to noisy inputs or sharp perturbations.

80 2 Results

81 2.1 A mechanochemical model of auxin patterning

82 We built a mechanochemical model of auxin transport and tissue mechanics
83 that incorporates the influence of auxin on the stiffness of the cell wall and
84 the mechanical feedback from the cell wall on auxin transport, as illustrated
85 in Fig. 1A. We briefly describe its main ingredients here, deferring further
86 details to the section "Model formulation" below. We model a single cell layer,
87 accounting for the epidermis of the shoot apex. We simplify tissue topology and
88 assume the cells to form a regular hexagonal lattice, following [17], because we
89 aim at stereotypic simulation results that would ease the comparison between
90 strain- and stress-sensing mechanisms. The tissue is under global isotropic
91 tension from turgor pressure, the cellular inner pressure that is assumed to
92 be uniform. Each edge of the lattice is made of two contiguous cell walls
93 associated with either of the two neighboring cells. Each cell wall is modeled
94 by a linear spring, whose stiffness decreases with auxin concentration in the
95 corresponding cell. This accounts for the positive effect of auxin on growth
96 in the shoot apex. Changes in auxin concentration are due to production,
97 degradation, diffusion and transport. We considered two hypotheses for the
98 mechanical feedback: PIN1 transporters are inserted preferentially facing the
99 cell walls that (i) undergo the highest elastic strain (elastic deformation) or
100 (ii) the highest stress (force).

101 We used two main observables: the polarity \mathcal{P} , defined as the ratio between
102 the highest and the lowest transporter concentrations along the cell edges, and
103 the membrane fraction \mathcal{F} , defined as the ratio between the number of trans-
104 porters localized at the plasma membrane and the total amount of transporters
105 in a cell (Fig. 1B). Typical patterns obtained by solving the models numeri-
106 cally are shown in Fig. 1C. In order to verify our simulations, we performed
107 an analytical linear stability analysis of the homogeneous state (supplemen-
108 tary text and Fig. S1) and predicted wavelengths in agreement with numerical
109 results for a broad range of parameter values.

110 For a given set of parameters, wavelengths are larger for strain- than for
111 stress-sensing (in agreement with linear stability analysis, see the supplemen-
112 tary note and Fig. S1). Because of this discrepancy in the wavelength, all anal-
113 yses performed hereafter were also carried out on 4 other sets of parameters
114 for the stress feedback, presented in the supplementary material (Fig. S2-6).
115 These additional simulations largely support the conclusions presented here.

116 2.2 Cell ablation induces radial polarity

117 When a single epidermal cell is laser-ablated at the shoot apex, the hypothesis
118 that the epidermis is in tension implies that the maximal stress orientation is
119 circumferential around the ablation [15]. The preferential localization of PIN1
120 transporters in the stretched walls would then makes the transport radial. A
121 model similar to ours was able to reproduce this observation with a stress-
122 driven feedback [17]. Here, we specifically asked whether our model can, with
123 a strain-driven feedback mechanism, reproduce the experimental results. As
124 in [17], we modeled the ablated cell by removing auxin, its transporters, and
125 cell walls, and by preventing the insertion of transporters in the membranes
126 adjacent to ablated cells. Our results, see Figure 2, show a radial reorientation
127 of the transporters in neighboring cells, as observed in experiments [17]. It
128 thus appears that ablation experiments do not allow us to distinguish between
129 strain- and stress-feedback mechanisms.

130 2.3 Variations of polarity with turgor pressure

131 Experiments with osmotic treatments show that PIN1 polarity \mathcal{P} is affected
132 by changes in turgor pressure [32]. By performing osmotic treatments, turgor
133 pressure was varied by approximately a factor 2 in these experiments. Polarity
134 was significantly smaller (-15%) with reduced turgor and slightly smaller (-5%)
135 with enhanced turgor.

136 We simulated the effect of a gradual increase or decrease of turgor pressure
137 by modulating tissue tension σ (see Eq.(1)) with respect to its reference value,
138 σ_0 , and monitored the change in the polarity of the transporters with the two
139 mechanical feedbacks. Our results, see Fig. 3, show that, irrespective of the
140 feedback mechanism, polarity increases over a range $0.5\sigma_0 \lesssim \sigma \lesssim 2\sigma_0$, as
141 observed experimentally.

142 The increase is sharper at small turgor pressure, consistent with experi-
143 ments. At higher values of σ ($\sigma \approx 2\sigma_0$), the two models slightly disagree with
144 experiments as they predict a slight increase in polarity while a small decrease
145 is observed. It is unclear whether this is due to a shortcoming of the model or
146 to a bias in experimental quantifications.

147 With the stress feedback, the sharp drop seen in Fig. 3 is due to a change
148 in the overall generated pattern of auxin peaks, which is not relevant in the
149 present context, and is followed by an increase in polarity; examples of tissues
150 and PIN1 distributions before and after this transition are shown in Figure
151 S2.

152 2.4 Global softening of the tissue increases polarity

153 Cellulose is the stiffest polysaccharide in plant cell walls. Accordingly, impair-
154 ing cellulose synthesis by chemical treatments leads to the softening of plant

155 tissues [42]. The consequence of such treatments on PIN1 localization was also
156 investigated in [17]. First, PIN1 polarity, \mathcal{P} , is amplified - the membrane con-
157 centration of PIN1 increases where it is high before treatment and decreases
158 elsewhere. Second, the amount of PIN1 decreases in the cytosol, or equivalently
159 the membrane fraction \mathcal{F} increases.

160 We simulated such treatments by decreasing the minimal stiffness, k_{min} , of
161 the cell walls (Fig. 4). The increase in polarity \mathcal{P} and in the membrane fraction
162 \mathcal{F} is reproduced with both feedbacks (Fig. 4). Therefore, these experiments
163 do not allow us to discriminate between the two models of feedback.

164 2.5 Reducing auxin-driven softening disrupts polarity

165 It has been shown that de-methyl-esterification of the pectin homogalactur-
166 onan by pectin methyl-esterases (PMEs) is necessary for auxin to soften the
167 cells [37]. Indeed, in plants overexpressing an inhibitor of PME activity, PME
168 INHIBITOR3, patterns of auxin accumulation are absent and organ primor-
169 dia do not form. In addition, the application of auxin does not trigger any of
170 auxin response (no expression of the response reporter DR5), tissue softening,
171 or organ formation [7]. In these plant lines, polarity and membrane fraction
172 are both reduced.

173 To simulate such overexpression, we decreased the value of the parameter,
174 δk (see Eq.(3) in the method section), that controls the sensitivity of cell
175 wall stiffness to auxin. A vanishing δk means that auxin has no effect on wall
176 stiffness, and high values of δk imply that a small change in auxin level induces
177 a large change in stiffness. In parallel, we kept $k_{min} + \delta k$ constant so that the
178 reference stiffness in the absence of auxin remains unchanged. We measured
179 the polarity \mathcal{P} and the fraction \mathcal{F} of transporters in the cell walls with the
180 two types of feedbacks.

181 Fig. 5 shows that when δk decreases, the polarity converges towards 1, cor-
182 responding to a homogeneous state with no patterns. Thus the conclusion that
183 the polarity is lost as a result of tissue stiffening is independent of the nature
184 of the mechanical feedback. However, the variations of the membrane fraction
185 \mathcal{F} depend on the precise feedback mechanism. When stiffening the tissue, \mathcal{F}
186 decreases with the strain feedback, but remains approximately constant with
187 the stress-feedback, as shown in Fig. 5.

188 The behavior of the model can be understood analytically. In the homoge-
189 neous state, that is in the absence of auxin patterns as observed in the simula-
190 tions when δk is decreased, the membrane fraction can be computed exactly,
191 as shown in the supplementary material. The calculations predict that \mathcal{F}^{stress}
192 remains constant, whereas \mathcal{F}^{strain} decreases when δk decreases. Note that it
193 is relevant to assume a homogeneous state of the system because naked meris-
194 tems do not show auxin accumulation patterns. In view of these results, we
195 conclude that a strain feedback is more realistic to describe experiments where
196 PME activity is reduced.

197 2.6 Correlation between auxin level and polarity

198 Experiments showed that, at the initiation of a primordia, the concentration
199 of PIN1 transporters in the plasma membrane increases with the concentra-
200 tion of auxin [18], likely independently of the increase in PIN1 transcription in
201 response to auxin. Comparison of auxin and PIN1 concentrations in our simu-
202 lations show that with the strain-sensing mechanism, the model qualitatively
203 reproduces the experimental observations, whereas a stress-sensing mechanism
204 leads to the opposite behavior, see Fig. 6. Again, analytical calculations in the
205 limit of small auxin fluctuations around the homogeneous state confirm this
206 result, regardless of the choice of parameters (see supplementary material).
207 Accordingly, these results support a strain-sensing mechanism.

208 2.7 Robustness to noise

209 We now investigate the sensitivity of the pattern to noise. To this end, a ran-
210 dom term, temporally and spatially uncorrelated, is added to the production
211 rate of auxin or to the concentration of transporters, respectively s_a or P in
212 Eq. (2). We measured the wavelength of the pattern and its dependence on
213 the noise amplitude.

214 As shown by panel A in Fig. 7, the pattern is almost insensitive to the
215 effect of noise on the production of auxin. In contrast, see Fig. 7B, noise in the
216 concentration of transporters P alters significantly the pattern. With the stress
217 feedback, the wavelength is observed to increase by up to 200%, whereas the
218 increase in the wavelength of the pattern is only 20% with the strain feedback.
219 This difference in the sensitivity of the patterns to noise in the transporters,
220 PIN1, also discriminates between the two models. Although it is difficult to
221 assess the level of noise in plants, the striking robustness of phyllotaxis to
222 fluctuations in auxin levels [50] suggests that the feedback based on strain is
223 more plausible.

224 2.8 Robustness to sharp variations

225 Even though robustness to noise discriminates between the two models, noise
226 is difficult to manipulate experimentally. Robustness can also be tested by
227 applying a transient shock to the system, i.e. a sharp modification of the ex-
228 ternal parameters, and by observing whether the tissue returns to its pre-shock
229 state. Experimentally, this is feasible by transient chemical perturbation, such
230 as external auxin application or inhibition of auxin transport, as performed
231 in [18].

232 We simulated such perturbations as follows. In a tissue at equilibrium, we
233 modified the mean auxin level $\langle a \rangle$ (through the production rate s_a , as shown
234 in Fig. 8A) or the concentration of transporters P , as shown in Fig. 8B, and
235 let the system reach a new equilibrium. Then we reset the parameter to its

236 initial value and obtain a third equilibrium. We compared the wavelengths of
237 the first and third equilibria. Observing the same wavelength would mean that
238 the system exactly recovers its initial state.

239 Fig. 8 shows that large differences in wavelength can be observed between
240 the first and the third equilibria, with both feedbacks. The strain feedback,
241 however, is not as sensitive, compare the left and right columns in Fig. 8. This
242 is qualitatively consistent with its lower sensitivity to noise, documented in
243 the previous subsection.

244 3 Discussion

245 We developed here a mechanochemical model for auxin patterning in the plant
246 shoot apical meristem. The central question, addressed with our model, is
247 whether the insertion of the efflux facilitators (transporters) in the membrane
248 depends on the strain of the cell walls or by the stress applied to them. To this
249 end, we compared the predictions of the model with available experimental
250 results.

251 We found that both stress and strain-based feedbacks lead to similar predic-
252 tions when simulating cell ablation, turgor-induced changes in tissue tension,
253 or a global reduction of the stiffness of the cell walls, generally in agreement
254 with observations. Modeling *PMEI*-overexpressing plants allowed us to dis-
255 criminate between the two models: Assuming that auxin effect on cell wall
256 stiffness is reduced in such plant lines, we found that only the strain-based
257 feedback directly accounted for observations. Interestingly, organogenesis is
258 abolished in these lines, showing that it is useful to study the system be-
259 havior in the absence of patterns. We also compared patterns of auxin and
260 PIN1 concentrations, and show both numerically and analytically that only
261 a strain-sensing mechanism can explain their correlation, as observed in in-
262 cipient primordia, whereas a stress-sensing mechanism leads to anti-correlated
263 patterns. Note that we assume the amount of transporters per cell to be con-
264 stant, whereas experiments show that the application of auxin increases PIN1
265 concentration [18]. Although we cannot rule out that this effect compensates
266 for the observed anti-correlation with a stress-based feedback, a strain-sensing
267 mechanism seems more plausible. Finally, investigating the effect of noise, or
268 of a transient change in chemical parameters, on patterning strengthened our
269 conclusion: Patterns appear more robust with the strain-based feedback.

270 The model, however, partly failed to reproduce the observations concern-
271 ing the effect of tissue tension on polarity. Irrespective of the feedback chosen,
272 we found that polarity increased with tension, whereas, experimentally, po-
273 larity slightly decreases from isotonic to hypertonic conditions [32]. Again, a
274 possible explanation is that we assumed the amount of transporters per cell
275 to be constant, whereas PIN1 levels decrease in hypertonic conditions; other
276 processes might be triggered when plants react to such osmotic stress. Alter-
277 natively, the slight decrease observed could be due to a small experimental
278 bias, and the difference between experiments and our simulations may not be

279 very significant. In addition, the model yielded wavelengths that were smaller
280 for stress- than for strain-sensing. As we have found no explanation for this
281 difference, it is difficult to know whether the model is incomplete or whether
282 this result would also favor strain-sensing because auxin peaks are observed
283 to have a smaller spatial extent than inter-peak distance, which resembles
284 more simulated strain-sensing (Fig. 1C, right). Because of this difference in
285 wavelength, we also explored other values of parameters for the stress-based
286 feedback and found that all conclusions on the comparison with the strain-
287 based feedback hold, except for the conclusions on robustness that are more
288 sensitive to parameter values (supplementary text, Figures S2-S7).

289 The idealized hexagonal geometry of the cells and the absence of tissue
290 growth are important limitations of the model. The agreement of the analyti-
291 cal linear stability analysis with simulations (supplementary text, Figure S1)
292 makes it likely that cell topology has little effect on our conclusions. Nevethe-
293 less, plants respond to mechanical stimuli by altering their growth rates [31].
294 Increase in auxin levels induce cell growth [7], which induces tissue reorgani-
295 zation, changes in mechanical stress [2], and ultimately feeds back on auxin
296 transport. Although these processes are important in development, mechanical
297 signals are quasi-instantaneous and mostly depend on the current state of the
298 tissue, so that we we do not expect them to significantly our main conclusions.

299 Altogether, our results favor a feedback based on strain, though many of
300 the experimental configurations are insensitive to whether the mechanical feed-
301 back on auxin transport is provided by stress or strain. This raises the question
302 of the underlying molecular mechanisms. Many types of mechanosensors are
303 known [38]. In the case of PIN1, strain could shift the balance between en-
304 docytosis and exocytosis, accounting for the strain-based feedback, because
305 osmotic stress affects cell trafficking, in particular through clathrin-mediated
306 endocytosis [53]. The contact between the plasma membrane and the cell wall
307 is needed for PIN1 polarity [5,10], suggesting that the mechanical state of the
308 cell wall is relevant for polarity. However the role of the cell wall might just
309 be to reduce lateral diffusion of PIN1, helping to maintain the distribution of
310 PIN1 determined by cell trafficking [27].

311 The question of strain- and stress-sensing was raised in the different, but
312 related, context of plant cortical microtubules orientation by mechanical cues,
313 and the combination of experimental and theoretical approaches suggests that
314 stress-sensing is more likely to be involved [15,6]. The same conclusion has
315 been reached concerning the actomyosin cortex in the drosophila wing disc
316 [25]. However, the case of isolated animal cells has been debated. Early ex-
317 periments suggested force- [12] or deformation-sensing [43]. More recent ex-
318 periments showed that deformation-sensing occurred at low force, while force-
319 sensing occurred at high force [52], though other experiments showed that
320 cells could sense the stiffness of extracellular space [30], consistently with the
321 observation that cells can differentiate according to the stiffness of their envi-
322 ronment [3]. Actually, mechanosensing occurs at different scales [38], so that
323 the mechanical variable sensed may depend on the sensing molecule and on
324 the specific function associated with sensing.

325 In order to disentangle the parameters involved in mechanosensing, it is
326 necessary to combine experimental perturbations of cells or tissues with analyt-
327 ical and computational studies of their behaviors. This is now made possible by
328 the improvement of micromechanical [28] and computational [8] approaches.
329 In this spirit, the present work provides insight on how the interaction be-
330 tween biomechanical and biochemical fields may contribute to the robustness
331 of morphogenesis.

332 Model formulation

333 *Tissue mechanics.* We model auxin transport through anticlinal cell walls in
334 the epidermis and thus we neglect the mechanical contribution of other cell
335 walls. We assume that the rest state of the tissue to be a regular hexagonal
336 tiling of the plane and we formulate the problem in terms of a vertex model
337 with periodic boundary conditions. The equilibrium positions of the vertices
338 are obtained by minimizing the mechanical energy of the N_C cells. The con-
339 tribution to the mechanical energy of the cell wall common to adjacent cells i
340 and j has a linear density

$$E_{i,j} = \frac{1}{2}k_{i,j}\varepsilon_{i,j}^2; \varepsilon_{i,j} = \frac{l_{i,j} - l^{(0)}}{l^{(0)}},$$

341 where $k_{i,j}$ is the stiffness of this wall, $l_{i,j}$ its length (with an equilibrium value
342 equal to $l^{(0)}$), and $\varepsilon_{i,j}$ its strain. The stress in the anticlinal wall is then given
343 by the derivative of its energy with respect to strain:

$$S_{i,j} \equiv \frac{\partial E_{i,j}}{\partial \varepsilon_{i,j}}.$$

344 The forces resulting from turgor pressure and tissue curvature are accounted
345 for by external stress with components σ_x , σ_y . The total energy of the tissue
346 then takes the form

$$E = -\sigma_x L_y^{(0)} (L_x - L_x^{(0)}) - \sigma_y L_x^{(0)} (L_y - L_y^{(0)}) + \sum_{\langle i,j \rangle} l^{(0)} E_{i,j} \quad (1)$$

347 where L_x and L_y are the tissue dimensions along the x and y directions and
348 $L_x^{(0)}$ and $L_y^{(0)}$ their values without external stress; the sum is over the pairs
349 of neighboring cells $\langle i, j \rangle$. Here we considered only the case of isotropic stress,
350 such that $\sigma_x = \sigma_y = \sigma$.

351 *Auxin dynamics and coupling with the mechanics.* We use the same assump-
352 tions as in previous studies [22,44]. Namely, we only model auxin concentra-
353 tions in the cytosol. We also assume that PIN1 dynamics occurs with a time
354 scale that is shorter than the time necessary for the transport of auxin through
355 cell walls. The latter is therefore the limiting step. These assumptions allow

356 us to reduce the model to only one chemical equation that describes the auxin
357 concentration, a_i , in cell i :

$$\frac{da_i}{dt} = s_a - d_a a_i - \frac{P}{A} \sum_{\langle j \rangle_i} l_{i,j} (t(a_i) p_{i,j} - t(a_j) p_{j,i}) - \frac{D}{A} \sum_{\langle j \rangle_i} l_{i,j} (a_i - a_j), \quad (2)$$

358 where s_a is auxin synthesis rate, d_a is auxin degradation rate by, and D is the
359 diffusion coefficient. $\langle j \rangle_i$ is the set of indices of the 6 cells adjacent to cell i . We
360 assume elastic strain to be small, so that the area of each cell is approximated
361 by its rest area A . The total amount of PIN1 proteins per cell, $P \sum_{\langle j \rangle_i} l_{i,j}$, is
362 assumed to be cell-independent; $p_{i,j}$ is the normalized linear concentration of
363 PIN1 proteins localised at the membrane of cell i and facing cell j (this PIN1
364 fraction is responsible for auxin efflux from cell i to cell j ; P is used as a unit
365 of linear concentrations). The rate of auxin transport by PIN1 proteins, $t(a)$,
366 has the following sigmoidal-dependence on auxin concentration:

$$t(a) = \frac{a^{H_t}}{K_t^{H_t} + a^{H_t}}$$

367 where K_t is a threshold in auxin concentration and H_t the Hill exponent.

368 Auxin controls tissue mechanics by softening the cell walls (Fig.1). The
369 stiffness of the walls decreases with the amount of auxin in the cells:

$$k_{i,j}(a_i) = k_{min} + \delta k \frac{K_k^{H_k}}{K_k^{H_k} + a_i^{H_k}} \quad (3)$$

370 where k_{min} is the wall stiffness in the absence of auxin, δk the variations in
371 stiffness, K_k the auxin threshold and H_k the Hill exponent.

372 The mechanical effects on the cell also affect auxin dynamics via its trans-
373 port. The amount of PIN1 transporters in a cell membrane is affected by the
374 strain or the stress, as illustrated in Fig. 1A. The normalized concentration,
375 $p_{i,j}$, of PIN1 proteins localised at the plasma membrane of cell i and facing
376 cell j reads

$$p_{i,j} = \frac{K_f^{H_f} \left(\left(\alpha + (1 - \alpha) \frac{k_{i,j}}{k_0} \right) \varepsilon_{i,j} \right)^{H_f}}{1 + \frac{\sum_{\langle k \rangle_i} l_{i,k} K_f^{H_f} \left(\left(\alpha + (1 - \alpha) \frac{k_{i,k}}{k_0} \right) \varepsilon_{i,k} \right)^{H_f}}{\sum_{\langle k \rangle_i} l_{i,k}}}$$

377 where $\alpha = 0$ for the stress feedback, $\alpha = 1$ for the strain feedback, and $\varepsilon_{i,j}$ and
378 $k_{i,j}$ stand for wall strain and wall stiffness as described above. The parameter
379 k_0 is set to k^* , the value of the stiffness in the homogeneous equilibrium state,
380 so that the contributions of stress and strain are of the same order of magnitude
381 in the homogeneous state. K_f sets the amplitude of the insertion function and
382 H_f is the corresponding exponent.

383 *Observables.* Two observables are relevant for the comparison between our
384 simulations and experimental observations, see Fig. 1B [32,7]. The first is
385 polarity, defined as the ratio of the PIN1 concentrations on the most and least
386 enriched membrane of cell i :

$$\mathcal{P}_i = \frac{p_i^{max}}{p_i^{min}} = \frac{\max_{\langle j \rangle_i} (p_{i,j})}{\min_{\langle j \rangle_i} (p_{i,j})},$$

387 and the second is the ratio of plasma membrane localized PIN1 to the total
388 amount in the cell:

$$\mathcal{F}_i = \frac{P_i^{wall}}{P_i^{in} + P_i^{wall}} = \frac{\sum_{\langle j \rangle_i} l_{i,j} p_{i,j}}{\sum_{\langle j \rangle_i} l_{i,j}}.$$

389 P does not appear in this equation because it was used to normalise $p_{i,j}$. In
390 practice we note \mathcal{P} and \mathcal{F} their distribution of the tissue.

391 *Implementation.* The model was programmed in C. The mechanical energy 1
392 is minimized thanks to the BFGS algorithm [26], implemented in the NLOpt
393 library [21]. The differential equations for the auxin concentration 2 are solved
394 iteratively thanks to the GNU GSL library with a Runge-Kutta (2, 3) method
395 [14]. We assume that the typical time-scale to reach the mechanical equilibrium
396 is much smaller than the time scale for auxin regulation. Consequently, we com-
397 pute the evolution of the system iteratively, by first computing the evolution
398 of the auxin concentration on a small time-step, and updating the configura-
399 tion of the tissue by minimizing its mechanical energy. The time-step $dt = 0.1$
400 for the evolution of auxin dynamics was chosen to limit the number of energy
401 minimization while generating the same results than smaller values. The code
402 is available on Github (github.com/JeanDanielJulien/auxinTransport). Sim-
403 ulations results were analyzed and figures were made with Matlab. Table 1
404 summarizes the values of the parameters used in the present work.

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Table 1 Values of the parameters used in the simulation: Parameters were estimated from previous models [22,44,17]. Stiffness dependence on auxin concentration was chosen to display 5-fold variations, in the range of realistic values [29]. The tension resulting from hydrostatic pressure was then chosen to yield a typical deformation of 4%, in the range estimated from osmotic treatments [32]. Finally, PIN1 insertion was tuned so that the fraction of transporters inserted in the membrane is around 0.7, similar to experimental estimates [32].

$l^{(0)}$	σ	s_a	d_a	D	P	K_t	H_t	K_f	H_f	k_0	k_{min}	δk	K_k	H_k
1	1.4	1	0.5	5	100	2	1	30	3	30	10	40	2	2

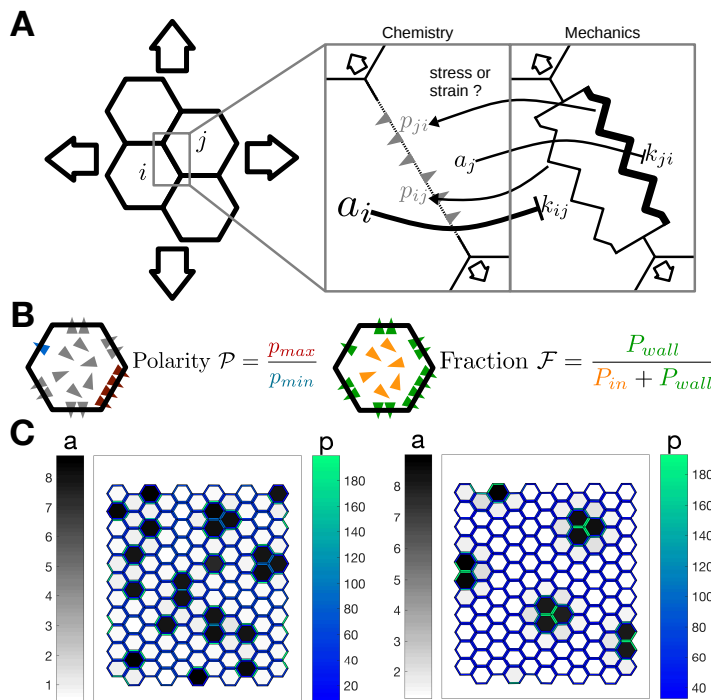


Fig. 1 Modeling assumptions and observables: (A) Schematic of the model: the tissue consists of hexagonal cells. The auxin transporters are localized at the plasma membranes and are influenced by the mechanical status of the corresponding cells walls. The biochemical and mechanical effects are presented in two different boxes, and their interactions are represented by arrows. The tissue is stretched by the turgor pressure. PIN1 proteins facilitate auxin movement out of cells. The stiffness of each cell wall is a decreasing function of the auxin concentration in the cell. The amount of effective transporters increases with the stress or strain at the cell wall. (B) Schematic definition of the two observables: the triangles represents the PIN1 transporters, colored according to the definition of the observables. The polarity \mathcal{P} is the ratio between the two extremal concentrations in the cell walls of a cell. The fraction \mathcal{F} is the ratio between the number of effective transporters and the total number of transporters. (C) An example of pattern predicted by the model, with each feedback (left: stress; right: strain). The cells are colored according to their auxin concentration (gray levels), the cell walls according to the density of PIN1 transporters (blue-green levels).

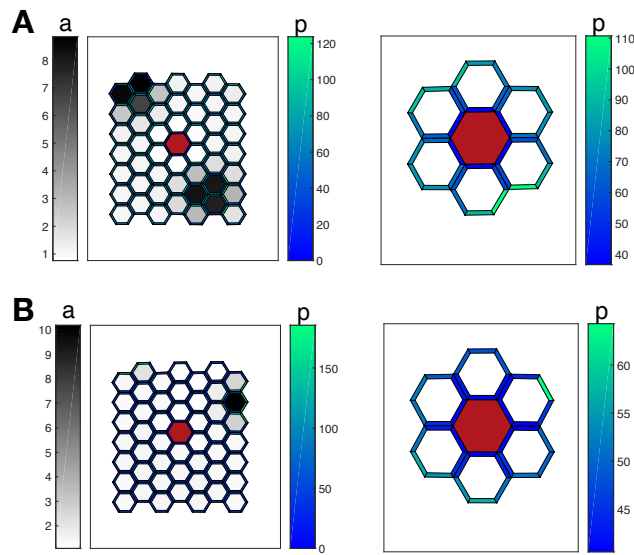


Fig. 2 Radial polarity around a cell ablation: The red hexagon corresponds to the ablated cell, whose walls, auxin and transporters have been removed from the simulation. The transporters pointing toward this cell are also removed. The cells are colored according to their auxin concentration, the cell walls according to the concentration of transporters. The insertion of transporters is driven either by stress (A) or by strain (B). Auxin is not represented on the close-ups. Parameters for the strain feedback are indicated in Table 1. Parameters for the stress feedback are identical, except for $H_k = 1.06$, which was chosen to increase wavelength and better visualize the effect of an ablation on polarity (results are independent of the value of H_k).

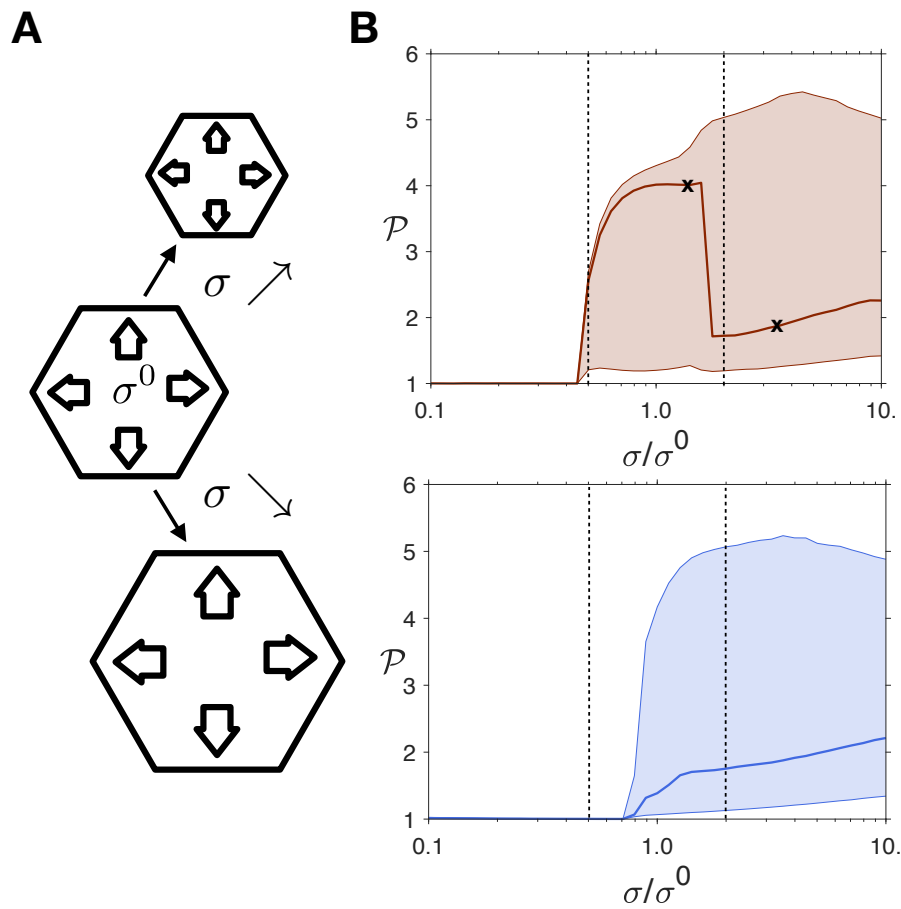


Fig. 3 Dependence of polarity on turgor pressure: (A) Schematic of the simulations: starting from $\sigma^{(0)}$, the pressure is gradually increased or decreased, for pressures ranging from $0.1 \times \sigma^{(0)}$ and $10 \times \sigma^{(0)}$. (B) For each cell the polarity \mathcal{P} is measured. The curves represent the median value of \mathcal{P} and the shaded areas the interval between the 15th and 85th percentiles in a tissue of 3600 cells. The results are shown in red (left) for the stress-based feedback, in blue (right) for the strain-based feedback. The dashed lines show the range of pressure investigated experimentally [32] and the black crosses indicate the parameters for which tissues are shown in Fig. S8.

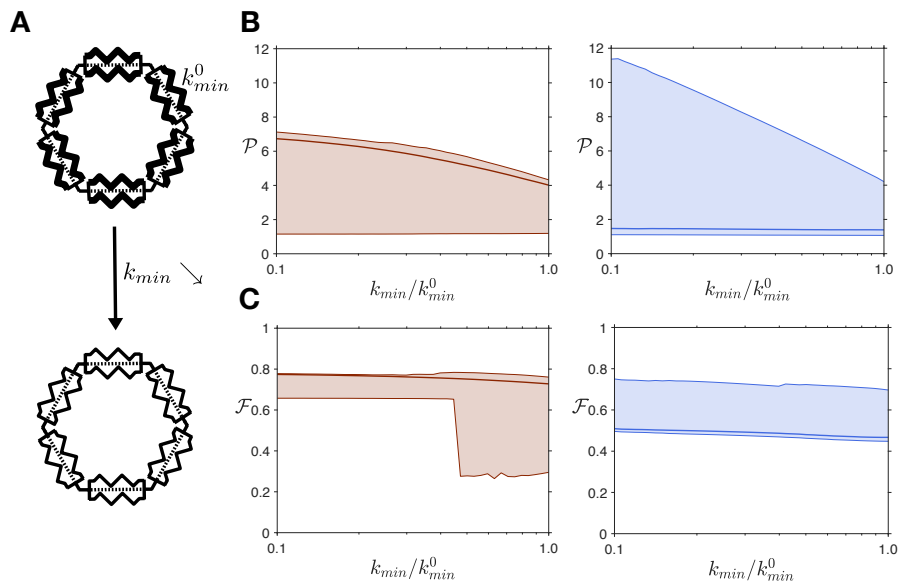


Fig. 4 Global softening of the tissue increases polarity: (A) Schematic of the simulations: starting from $k_{min}^{(0)}$, the minimal stiffness of the tissue is gradually decreased, ranging from $k_{min}^{(0)}$ to $k_{min}^{(0)}/10$. This corresponds to the stiffness at high levels of auxin. We quantified (B) the polarity \mathcal{P} and (C) the membrane fraction \mathcal{F} of transporters. The curves represent the median and the shaded areas the interval between the 15th and 85th percentiles in a tissue of 3600 cells. The results are shown in red (left) for the stress-based feedback, in blue (right) for the strain-based feedback.

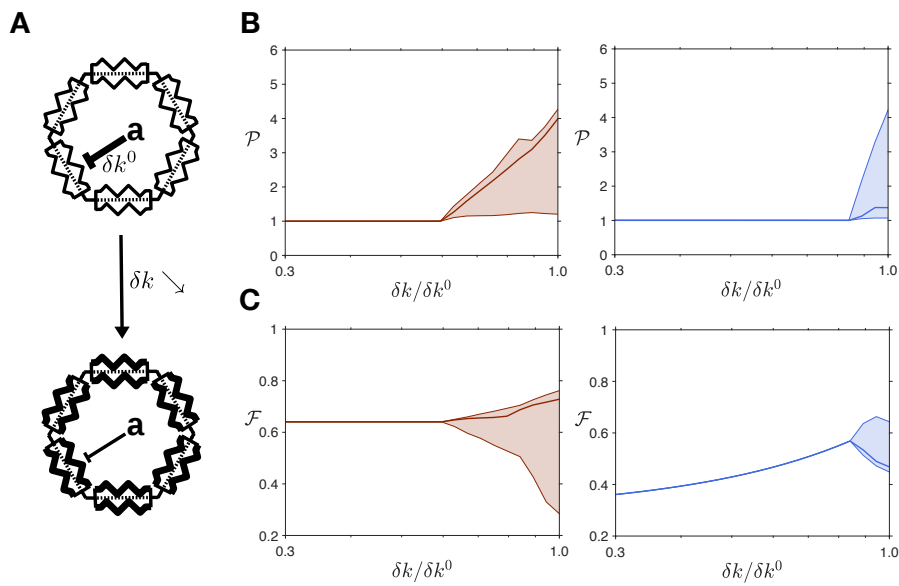


Fig. 5 Reducing auxin effects on the cell wall disrupts polarity: (A) Schematic of the simulations: starting from $\delta k^{(0)}$, the amplitude of stiffness variations is gradually decreased, from $k_{min}^{(0)}$ to $0.3 \times k_{min}^{(0)}$. We quantified (B) the polarity \mathcal{P} and (C) the membrane fraction \mathcal{F} of transporters. The curves represent the median and the shaded areas the interval between the 15th and 85th percentiles in a tissue of 3600 cells. The results are shown in red (left) for the stress-based feedback, in blue (right) for the strain-based feedback.

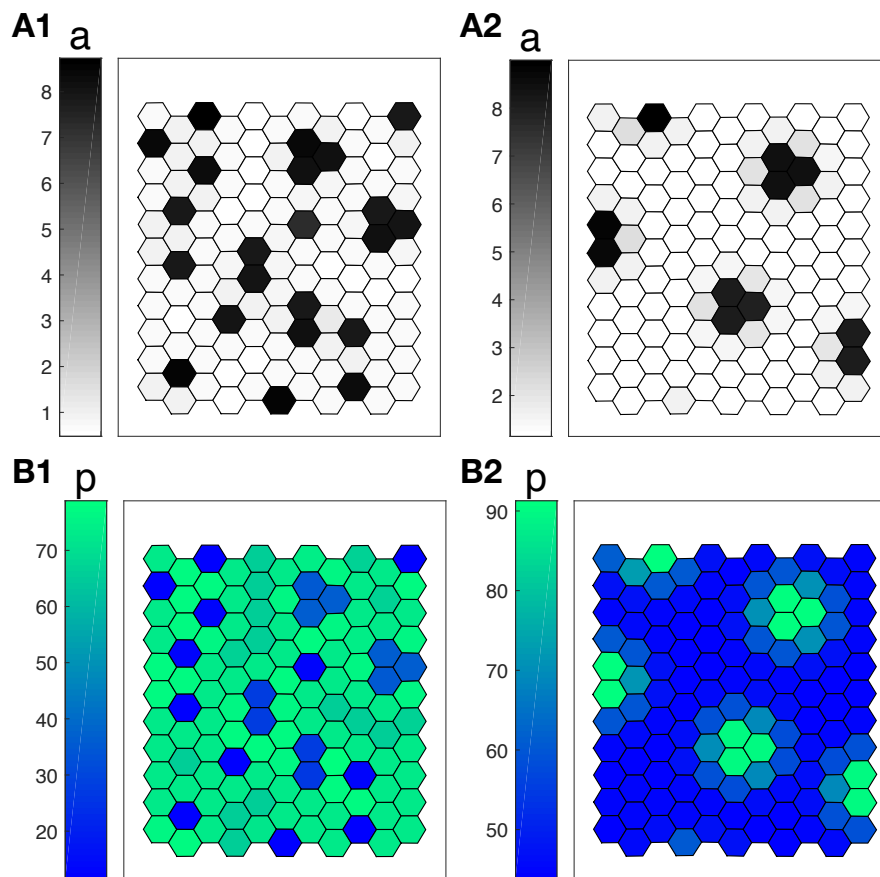


Fig. 6 Correlation between auxin and PIN1 concentrations: Examples of pattern predicted by the model, with each feedback (A/B1: stress; A/B2: strain). The cells are colored according to their auxin concentration (gray levels, A1/2), or according to the density of PIN1 transporters, averaged over their walls (blue-green levels, B1/2).

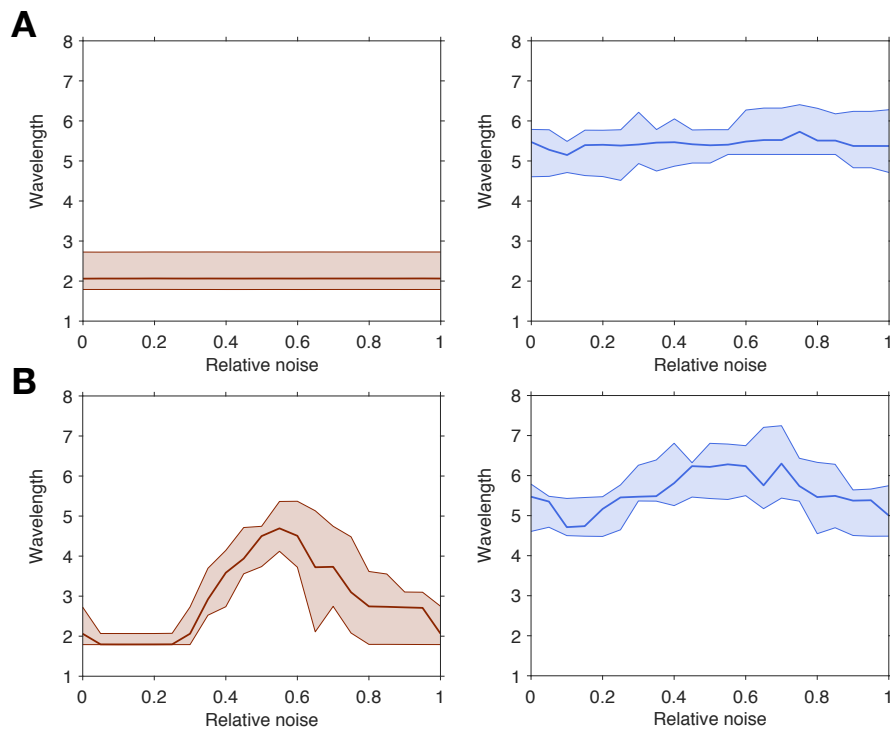


Fig. 7 Robustness to noise: The auxin production rate s_a (A) or the PIN1 concentration P (B) is spatially and temporally random, with a uniform distribution centered around the value of these parameters without noise. The relative noise amplitude is half the ratio between the width of this interval and the average value of the variable. The wavelength is measured as the average distance between a peak and its nearest neighbor. The prediction of the model with the stress feedback is shown in red (left), while the one with the strain feedback is represented in blue (right). The curves represent the median and the shaded areas the interval between the 15th and 85th percentiles.

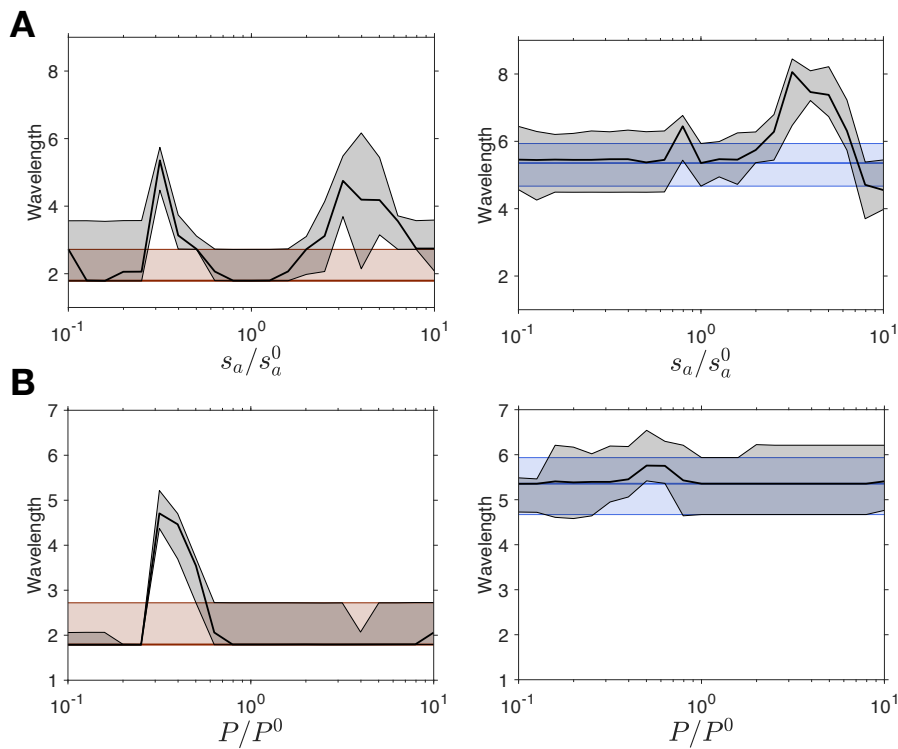


Fig. 8 Robustness to sharp variations: The auxin production rate s_a (A) or the PIN1 concentration P (B) is transiently modified over the entire tissue. Once the tissue reaches equilibrium, the original set of parameters is restored. The wavelength is measured at equilibrium before the shock and, and at equilibrium after resetting the original parameters. The wavelength before the shock is plotted in red for the stress feedback (left) and in blue for the strain feedback (right). The wavelength after the recovery is plotted in black. The curves represent the median, the shaded areas the interval between the 15th and 85th percentiles before the shock, and the hatched areas the same percentiles after the resetting.

Supplementary material

Strain- or Stress-sensing in mechanochemical patterning by the phytohormone auxin

Jean-Daniel Julien Alain Pumir Arezki Boudaoud

Linear stability analysis

The analytical study of the system is performed in the case of isotropic external stress such that $\sigma_x = \sigma_y = \sigma$.

The system is linearised by considering small variations of auxin around the homogeneous stable state $a^* + \delta_i$ where $a^* = s_a/d_a$. In the homogeneous stable state we can write the deformation of the walls, their stiffness and the rate of PIN1 insertion as:

$$\varepsilon^* = \frac{l^* - l_0}{l_0} = \frac{\sqrt{3}\sigma l_0}{2k(a^*)},$$

$$k^* = k(a^*) = k_{min} + \Delta k \frac{K_k^{H_k}}{K_k^{H_k} + (a^*)^{H_k}},$$

$$p^* = \frac{K_f^{H_f} \left[\left(\alpha + (1 - \alpha) \frac{k^*}{k_0} \right) \varepsilon_{i,j} \right]^{H_f}}{1 + K_f^{H_f} \left[\left(\alpha + (1 - \alpha) \frac{k^*}{k_0} \right) \varepsilon_{i,j} \right]^{H_f}}.$$

We assume that the local variations in the mechanical tension are small compared with the variations of auxin. Consequently we can write:

$$\frac{l_{i,j} - l_0}{l_0} = \frac{\sqrt{3}\sigma l_0}{k(a_i) + k(a_j)}$$

We then linearise the system:

$$\frac{d\delta_i}{dt} = -d_a \delta_i - [A - (1 - \alpha)B + 2(1 - \alpha)C] \sum_{\langle j \rangle_i} (\delta_i - \delta_j) + \frac{C}{N} \sum_{\langle j \rangle_i} \left[\sum_{\langle k \rangle_i} (\delta_i + \delta_k) - \sum_{\langle l \rangle_i} (\delta_j + \delta_l) \right].$$

The constants A , B and C are defined as:

$$A = P \frac{l^*}{A^*} t'(a^*) p^* + D \frac{l^*}{A^*},$$

$$B = P \frac{l^*}{A^*} t(a^*) p^* \left(-\frac{H_f}{k^*} k'(a^*) \right),$$

$$C = P \frac{l^*}{A^*} t(a^*) (p^*)^2 \left(-\frac{H_f}{2k^*} k'(a^*) \right).$$

The system can be diagonalised by applying a Fourier transform $\delta_{\vec{k}} = \frac{1}{2\pi} \sum_i \delta_i e^{-i\vec{k} \cdot \vec{x}_i}$, where \vec{x}_i is the position of the cell i . Thus we obtain:

$$\dot{\delta}_{\vec{k}} = \left\{ -d_a - N [A - (1 - \alpha)B] (1 - S_{\vec{k}}) + NC \left[2 (S_{\vec{k}} - 1) (1 - \alpha) + 1 - S_{\vec{k}}^2 \right] \right\} \delta_{\vec{k}}$$

$$\dot{\delta}_{\vec{k}} = \left\{ [-d_a - NA + N(1-\alpha)B + N(2\alpha-1)C] + [NA - N(1-\alpha)B + 2N(1-\alpha)C] S_{\vec{k}} - NC S_{\vec{k}}^2 \right\} \delta_{\vec{k}}$$

where $S_{\vec{k}} = \frac{1}{N} \sum_p e^{i\vec{k} \cdot \vec{e}_p}$ is the form factor of the tissue.

Then we can write the conditions of instability for the two models:

$$(1-\alpha)B + 2\alpha C > A,$$

$$N \frac{[(1-\alpha)B + 2\alpha C - A]^2}{4C} > d_a,$$

$$A + 2(2-\alpha)C > (1-\alpha)B.$$

If these conditions are true, we can show that the most unstable wave vectors $\{\vec{k}_0\}$ fulfils

$$S_{\vec{k}_0} = \frac{A - (1-\alpha)B + 2(1-\alpha)C}{2C}.$$

Note that $S_{\vec{k}}$ is a decreasing function of \vec{k} , and therefore an increasing function of the wavelength. Since $2C = p^*B < B$, the wavelength for the stress feedback ($\alpha = 0$) is always smaller than the wavelength for the strain feedback ($\alpha = 1$), for any set of parameters.

We compared these results with the wavelength measured in simulations for 2-fold changes in the value of each parameter, see Fig. S1. Overall, the linear stability analysis presented here provides a semi-quantitative explanation for the results of our numerical simulations.

Investigation of different parameters for the stress feedback

The most parsimonious set of parameters, presented in the main manuscript, leads to significantly different wavelengths for the stress- and strain-sensing mechanisms. In order to evaluate the robustness of the results, we present here the same analysis with 4 different parameter changes of the stress feedback. We first tried modifying k_0 , the only parameter that distinguishes the two models. Decreasing k_0 increases the wavelength, but does not allow to reach wavelength of the strain-based feedback. Additionally, it increases the typical fraction of transporters inserted in the membrane p^* to 1, inconsistent with experimental observations. We present the results for $k_0 = k_{min}$, close to the wavelength saturation (panels 1 in all figures 2 to 7). We then tried to modify 3 parameters that are not involved in the definition of p^* , and which perturb neither the typical deformation of the cells ϵ^* nor their typical stiffness variations $\Delta k/k_{min}$. The three parameters values (panels 2: $H_k = 1.06$; panels 3: $D = 17$; panels 4: $P = 30$) were tuned by using the linear stability analysis to match the wavelength of the strain-based feedback. For figures 2 to 7, transporters concentration is regulated by stress. The curves all represent median values and the shaded areas the interval between the 15th and 85th percentiles in a tissue of 3600 cells. Parameters are the same as in Table 1 of the main manuscript, except for one parameter, modified in order to get comparable wavelengths for both feedbacks (1: $k_0 = k_{min}$; 2: $H_k = 1.06$; 3: $D = 17$; 4: $P = 30$).

The parameters sets presented here support the conclusion of the main manuscript. In particular, the qualitative differences between the stress-feedback and experiments, namely the absence of internalisation of transporters in simulated PME1 mutants (Fig. 4) and the anti-correlation of auxin and PIN1 patterns (Fig. 5), are valid for all other parameters investigated, consistent with the calculations in the limit of small fluctuations. Results regarding patterns robustness to noise (Fig. 6) and perturbations (Fig. 7) are mitigated, with very different sensitivities of the different parameters sets.

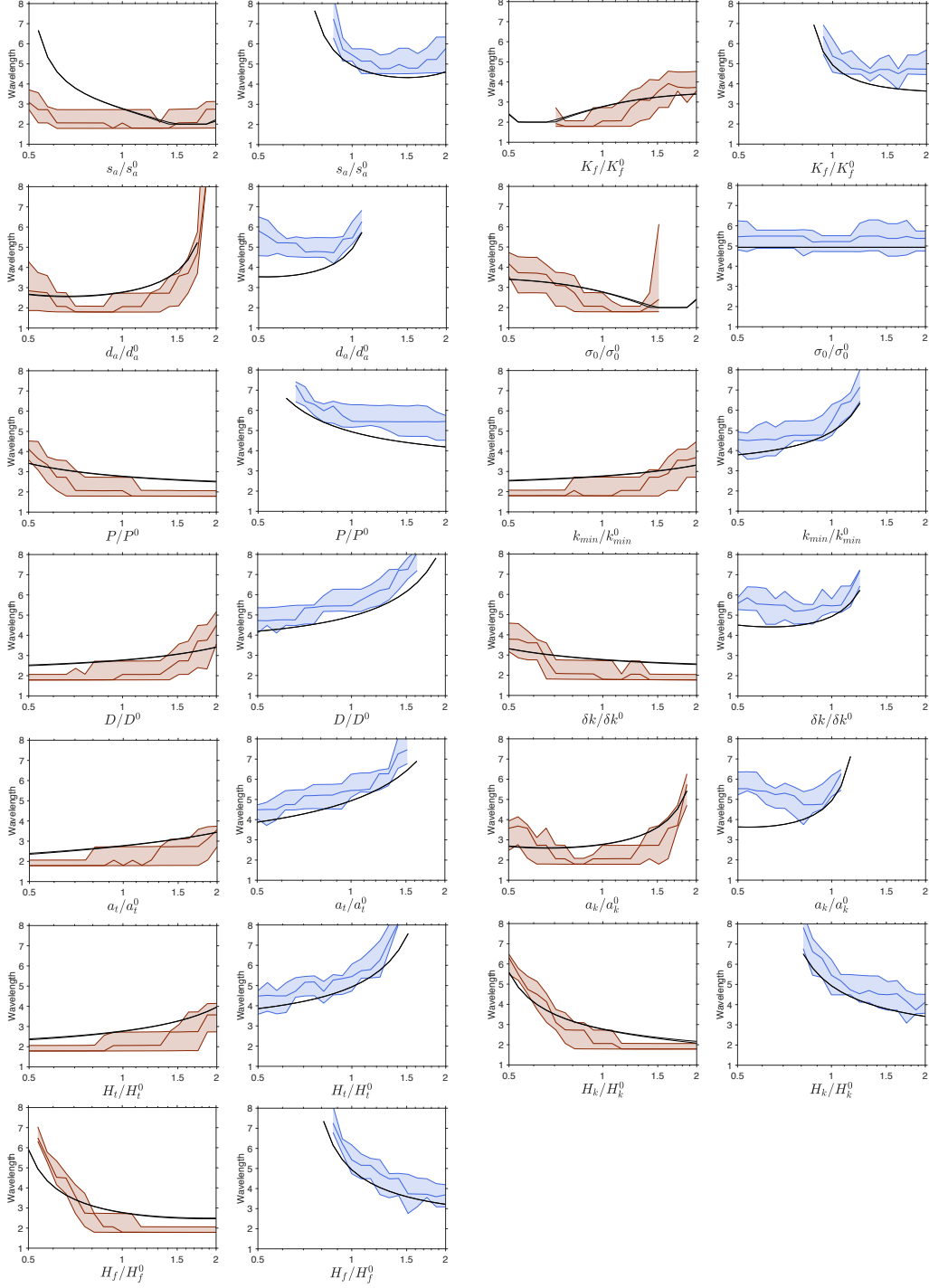


Figure 1: **Dependence of the wavelength on each parameter of the model:** The wavelength is measured for 2-fold changes in each parameter around the values given in Table 1 of the main text. Results are shown in red for the stress-based feedback, and in blue for the strain-based feedback. The black lines indicate the predictions from the linear stability analysis, in the two directions of the wave vector \vec{k} where the prediction is minimal and maximal. Note that these lines almost overlap, except for the smallest wavelengths.

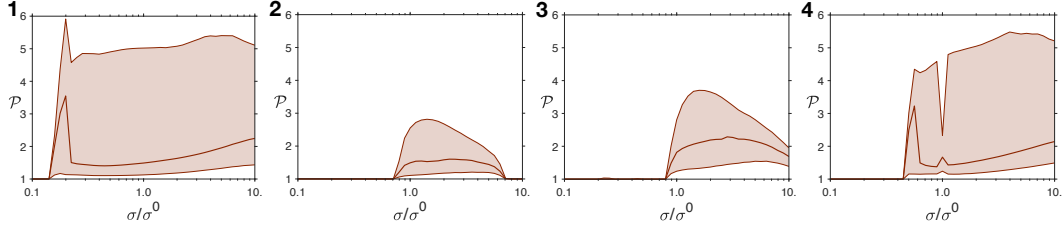


Figure 2: **Dependence of polarity on turgor pressure:** Starting from $\sigma^{(0)}$, the pressure is gradually increased or decreased, for pressures ranging from $0.1 \times \sigma^{(0)}$ and $10 \times \sigma^{(0)}$. (1-4) Polarity of transporters for different parameters changes.

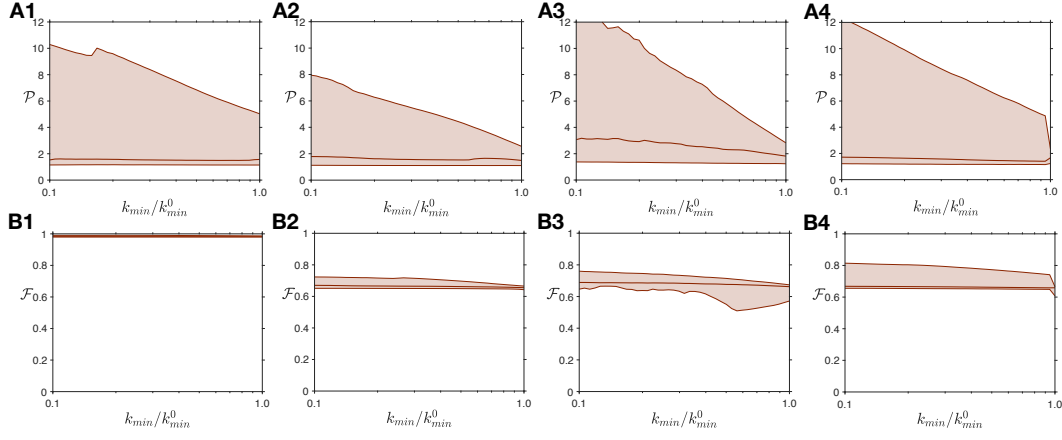


Figure 3: **Global softening of the tissue increases polarity:** Starting from $k_{min}^{(0)}$, the minimal stiffness of the tissue is gradually decreased, ranging from $k_{min}^{(0)}$ to $k_{min}^{(0)}/10$. (A1-4) Polarity and (B1-4) fraction of transporters for different parameters changes.

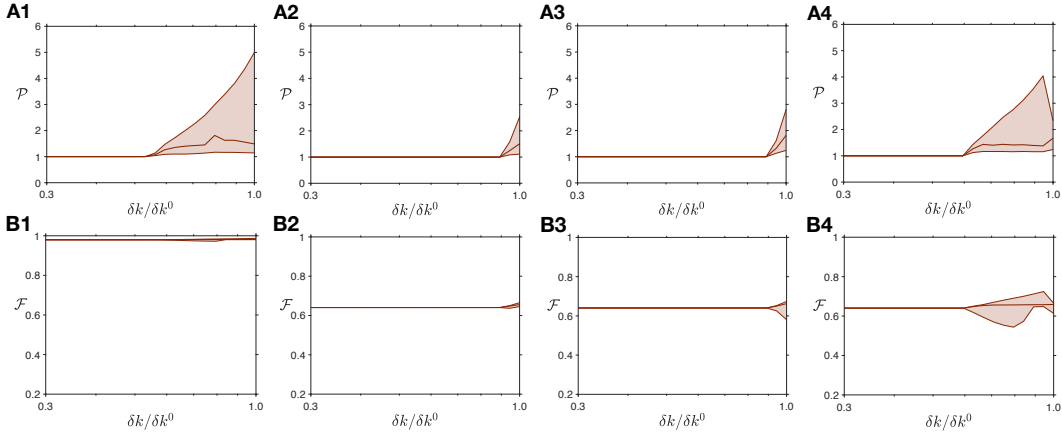


Figure 4: **Reducing auxin effects on the cell wall disrupts polarity:** Starting from $\delta k^{(0)}$, the amplitude of stiffness variations is gradually decreased, from $k_{min}^{(0)}$ to $0.3 \times k_{min}^{(0)}$. (A1-4) Polarity and (B1-4) fraction of transporters for different parameters changes.

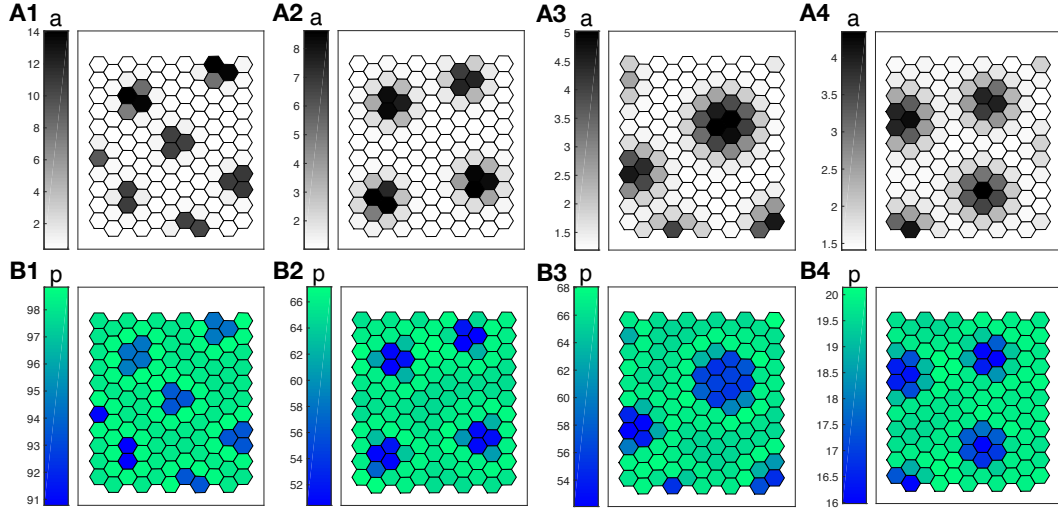


Figure 5: **Anticorrelation between auxin and PIN1 concentrations:** Examples of pattern predicted by the model, with a stress feedback, for different parameters changes. The cells are colored according to their auxin concentration (gray levels, A1-4), or according to the density of PIN1 transporters, averaged over their walls (blue-green levels, B1-4).

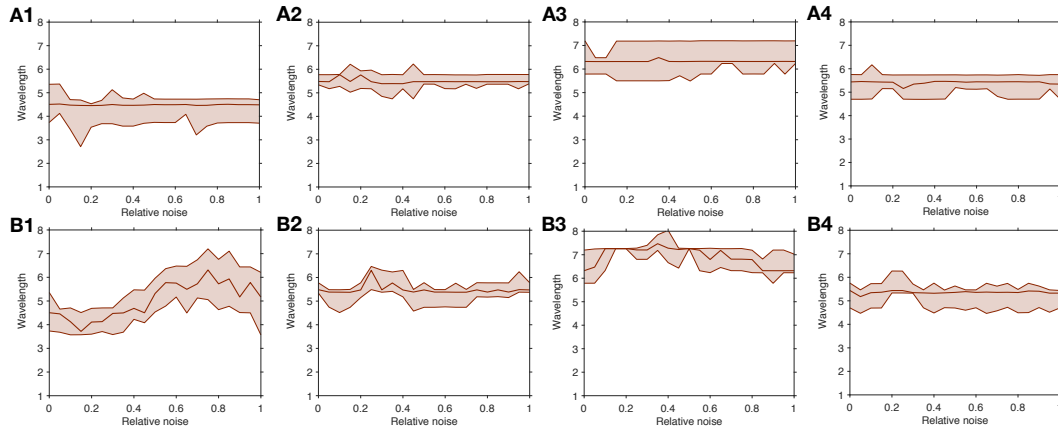


Figure 6: **Robustness to noise:** The auxin production rate s_a (A) or the PIN1 concentration P (B) is spatially and temporally random, with a uniform distribution centered around the value of these parameters without noise. The relative noise amplitude is half the ratio between the width of this interval and the average value of the variable. The wavelength is measured as the average distance between a peak and its nearest neighbor, for different parameters changes (1-4).

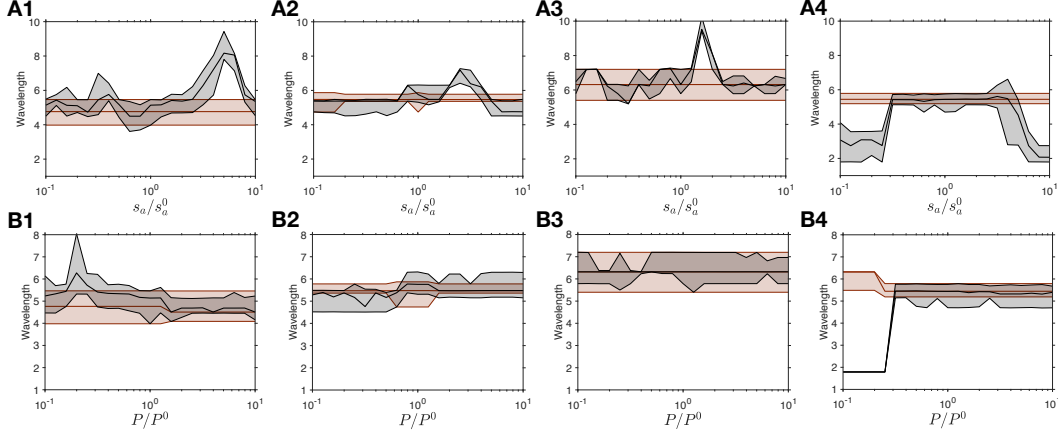


Figure 7: **Robustness to sharp variations:** The auxin production rate s_a (A) or the PIN1 concentration P (B) is transiently modified over the entire tissue. Once the tissue reaches equilibrium, the original set of parameters is restored. The wavelength is measured at equilibrium before the shock and, at equilibrium after resetting the original parameters. The wavelength before and after the shock is plotted in red and black respectively, for different parameters changes (1-4).

Change of pattern type with increased pressure for the stress-feedback

A sharp decrease of the median polarity \mathcal{P} was predicted with a stress-based feedback when tension is increased above $\sigma/\sigma_0 = 2.3$ (see Fig. 2C). This decrease is due to the change of pattern type. At low tension, the auxin peaks are limited to one or two cells. They become larger as tension increases. This enlargement corresponds to smaller auxin gradients between neighboring cells and accordingly to weaker polarities (see Fig. S8).

Dependence of the membrane fraction \mathcal{F} on δk

To simulate the effect of pectin methyltransferase inhibition, we decrease the parameter δk , with the constraint that $k_{min} + \delta k$ remains constant. By doing so, the dependence of cell wall stiffness with auxin concentration is decreased but the stiffness in the absence of auxin remains the same. The simulations, presented in Fig. 3, show that with the two feedback mechanisms the polarity \mathcal{P} and the membrane fraction of PIN1 \mathcal{F} decrease until the auxin patterns vanish. After the disappearance of the pattern, the simulation results depend on the feedback mechanism. With a stress-based feedback, the fraction \mathcal{F} remains constant, whereas with a strain-based feedback it keeps decreasing. These different behaviors can be explained analytically. In the homogeneous state, the membrane fraction \mathcal{F} of transporters is:

$$\mathcal{F}^{stress} = \frac{K_f^{H_f} \left(\frac{k^*}{k_0} \varepsilon^* \right)^{H_f}}{1 + K_f^{H_f} \left(\frac{k^*}{k_0} \varepsilon^* \right)^{H_f}}$$

$$\mathcal{F}^{strain} = \frac{K_f^{H_f} (\varepsilon^*)^{H_f}}{1 + K_f^{H_f} (\varepsilon^*)^{H_f}}$$

where the star indicates the values in the homogeneous state. k^* is given by

$$k^* = k_{min} + \delta k \frac{K_k^{H_k}}{K_k^{H_k} + a^{*H_k}} = (k_{min} + \delta k) - \delta k \frac{a^{*H_k}}{K_k^{H_k} + a^{*H_k}}$$

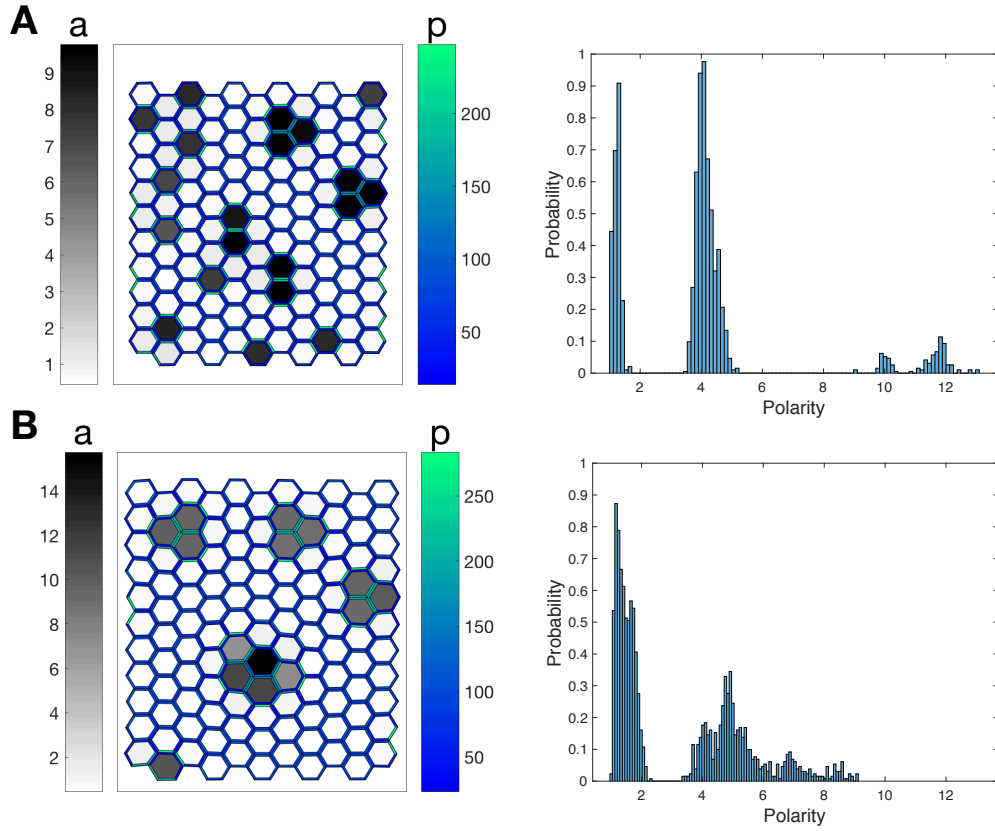


Figure 8: **Change in the overall pattern when tension increases:** (A) Tissues are represented for $\sigma/\sigma_0 = 1.41$ (left) and $\sigma/\sigma_0 = 3.55$ (right). Each hexagon is a cell that is colored according to its auxin concentration. The two tissues correspond to the parameters marked by black crosses in Fig. 2B. (B) Probability distributions of the polarity \mathcal{P} computed on the respective tissues.

If we decrease the value of δk from an initial value δk^0 with the constraint that $k_{min} + \delta k$ remains constant, equal to its initial value $k_{min}^0 + \delta k^0$, k^* is then:

$$k^* = k_{min}^0 + \delta k^0 - \delta k \frac{a^{*H_k}}{K_k^{H_k} + a^{*H_k}}$$

Thus k^* is a decreasing function of δk . The deformation of a regular hexagonal lattice of springs with rest length l_0 , stiffness k^* and under a tension σ is given by $\varepsilon^* = \frac{\sqrt{3}\sigma l_0}{2k^*}$.

In conclusion, the fractions are

$$\mathcal{F}^{stress} = \frac{K_f^{H_f} \left(\frac{\sqrt{3}\sigma l_0}{2k_0} \right)^{H_f}}{1 + K_f^{H_f} \left(\frac{\sqrt{3}\sigma l_0}{2k_0} \right)^{H_f}}$$

$$\mathcal{F}^{strain} = \frac{K_f^{H_f} \left(\frac{\sqrt{3}\sigma l_0}{2k^*} \right)^{H_f}}{1 + K_f^{H_f} \left(\frac{\sqrt{3}\sigma l_0}{2k^*} \right)^{H_f}}$$

These formulas show that \mathcal{F}^{stress} does not depend on δk whereas \mathcal{F}^{strain} actually decreases because k^* increases, The second behavior is consistent with available experimental results [?].

Correlation between auxin and transporters concentrations

We want to study the variation of the mean concentration of transporters in a cell's membrane, $\bar{p} = (\sum_{\langle j \rangle_i} l_{i,j} P p_{i,j}) / (\sum_{\langle j \rangle_i} l_{i,j})$, as a function of small fluctuations of auxin δ_i around the homogeneous equilibrium. Since we assume that the total concentration of transporters is the same in each cell, this is equivalent to the variations of the fraction $\mathcal{F}_i = (\sum_{\langle j \rangle_i} l_{i,j} p_{i,j}) / (\sum_{\langle j \rangle_i} l_{i,j})$.

The change in fraction $\delta \mathcal{F}_i$ is given by

$$\delta \mathcal{F}_i = \delta \left[\frac{\sum_{\langle j \rangle_i} l_{i,j} p_{i,j}}{\sum_{\langle j \rangle_i} l_{i,j}} \right] = \frac{1}{N} \sum_{\langle j \rangle_i} \delta p_{i,j}.$$

Recall the expression for $p_{i,j}$:

$$p_{i,j} = \frac{K_f^{H_f} \left[\left(\alpha + (1 - \alpha) \frac{k_{i,j}}{k_0} \right) \varepsilon_{i,j} \right]^{H_f}}{1 + \frac{\sum_{\langle k \rangle_i} l_{i,k} K_f^{H_f} \left[\left(\alpha + (1 - \alpha) \frac{k_{i,k}}{k_0} \right) \varepsilon_{i,k} \right]^{H_f}}{\sum_{\langle k \rangle_i} l_{i,k}}}$$

We use the same assumption than for the linear stability analysis to write the fluctuations in strain and wall length as

$$\varepsilon_{i,j} = \frac{l_{i,j} - l_0}{l_0} = \frac{\sqrt{3}\sigma l_0}{k(a_i) + k(a_j)},$$

$$\delta \varepsilon_{i,j} = \varepsilon^* \left(\frac{-k'(a^*)}{2k^*} \right) (\delta_i + \delta_j),$$

$$l_{i,j} = l_0 (1 + \varepsilon_{i,j}),$$

$$\delta l_{i,j} = l_0 \delta \varepsilon_{i,j}.$$

Plugging these equations back in the PIN1 concentration, we obtain:

$$\delta p_{i,j} = p^* \frac{H_f \delta \varepsilon_{i,j}}{\varepsilon^*} - (p^*)^2 \frac{H_f \sum \delta \varepsilon_{ik}}{\langle k \rangle_i N \varepsilon^*} - (p^*)^2 \frac{\sum \delta l_{ik}}{N l^*} + (p^*)^2 \frac{\sum \delta l_{i,k}}{N l^*} + (1 - \alpha) p^* \frac{H_f \delta k_{i,j}}{k^*} - (1 - \alpha) p^{*2} \frac{H_f \sum \delta k_{ik}}{N k^*},$$

from which we get

$$\begin{aligned} \delta p_{i,j} &= p^* H_f \left(\frac{-k'(a^*)}{2k^*} \right) (\delta_i + \delta_j) - (p^*)^2 H_f \left(\frac{-k'(a^*)}{2k^*} \right) \frac{1}{N} \sum_{\langle k \rangle_i} (\delta_i + \delta_k) \\ &\quad - (1 - \alpha) p^* H_f \left(\frac{-k'(a^*)}{k^*} \right) \delta_i + (1 - \alpha) p^{*2} H_f \left(\frac{-k'(a^*)}{k^*} \right) \delta_i. \end{aligned}$$

Summing over the neighbours gives the change in fraction $\delta \mathcal{F}_i$:

$$\delta \mathcal{F}_i = \frac{1}{N} p^* H_f \left(\frac{-k'(a^*)}{2k^*} \right) \left((2\alpha - 1) N \delta_i + \sum_{\langle j \rangle_i} \delta_j - p^* \sum_{\langle k \rangle_i} ((2\alpha - 1) \delta_i + \delta_k) \right)$$

We define the Fourier transform of $\delta \mathcal{F}$, $\Delta_{\vec{k}} = \frac{1}{2\pi} \sum_i \delta \mathcal{F}_i e^{-i \vec{k} \cdot \vec{x}_i}$, with the Fourier transform of the auxin fluctuations denoted as $\delta_{\vec{k}}$, and the form factor of the tissue $S_{\vec{k}} = \frac{1}{N} \sum_p e^{i \vec{k} \cdot \vec{e}_p}$.

$$\begin{aligned} \Delta_{\vec{k}} &= \frac{1}{N} p^* H_f \left(\frac{-k'(a^*)}{2k^*} \right) [(2\alpha - 1) N \delta_{\vec{k}} + N S_{\vec{k}} \delta_{\vec{k}} - p^* ((2\alpha - 1) N \delta_{\vec{k}} + N S_{\vec{k}} \delta_{\vec{k}})] \\ \Delta_{\vec{k}} &= p^* H_f \left(\frac{-k'(a^*)}{2k^*} \right) ((2\alpha - 1) + S_{\vec{k}}) (1 - p^*) \delta_{\vec{k}} \end{aligned}$$

We thus obtain the ratio between transporters and auxin fluctuations, for each feedback mechanism ($\alpha = 0$ corresponds to stress and $\alpha = 1$ to strain):

$$\begin{aligned} \left(\frac{\Delta_{\vec{k}}}{\delta_{\vec{k}}} \right)_{stress} &= p^* H_f \left(\frac{-k'(a^*)}{2k^*} \right) (S_{\vec{k}} - 1) (1 - p^*) < 0 \\ \left(\frac{\Delta_{\vec{k}}}{\delta_{\vec{k}}} \right)_{strain} &= p^* H_f \left(\frac{-k'(a^*)}{2k^*} \right) (S_{\vec{k}} + 1) (1 - p^*) > 0 \end{aligned}$$

As a conclusion, fluctuations in auxin and PIN1 concentrations have the same sign for the strain feedback, and opposite signs for the stress feedback (we remind that $k' < 0$). This result supports a feedback from mechanical strain, since observations show that both auxin and PIN1 accumulate in incipient primordia. Simulations are in agreement with this result, see Figure 5.