This repository includes the detailed method (see below) and the code for the simulation of the medial longitudinal section of leaf and floral primordia models (see CODE SIMULATION file).
These models were published in … Current Biology.

## 1. Model Description

We created 2D medial longitudinal computer models for leaf and floral primordia using MorphoDynamX; the extension of the MorphoGraphX software (Barbier de Reuille et al., 2015). Briefly, we combined biomechanical properties such as strain with auxin flow dynamics (biochemical transport) to predict primordium morphology at subcellular level following early organogenesis. The initial model integrates experimental values of stiffness (measured by AFM) for the different domains. Model predicts resulting growth rates, organ morphology and patterns of auxin transport polarization. These model predictions were then tested against quantitative experimental measurements of the growth rates in the epidermal layer (Figures 1H and 1K), as well as qualitatively to experimentally observed domain geometry (Supplemental Figure 6A and 6B) and auxin drainage paths from the epidermis to inner tissues (Figures 6D′, 6E′ and 6G′ for leaf; and 6O′-6S′ for flower). This was obtained by performing high-throughput model simulations and parameter testing on a supercomputing cluster. Optimal parameters values that reproduce observed morphologies are listed in Methods S2.

### 1.1 Generation of the initial mesh for leaf and flower models

To create the starting 2D mesh for the flower and leaf simulations, we used MorphoGraphX (Barbier de Reuille et al., 2015) to segment a confocal microscope image of an incipient (I1) floral primordium (Figure S6C). In this incipient stage leaf and floral primordia show an equivalent shape. As a result, we obtained a connected mesh with cells individualized as triangulated polygons, defined by vertices, edges and faces. Each cell is delimited by edges that represent the cell boundaries (cell wall and cell membrane), while inner edges resemble the cytoskeleton. All these elements, with different attributes according to their dimension and biological meaning, integrate the biochemical and biomechanical processes that drive the organ growth at the cellular level and, therefore, determine the final shape of the primordia.

### 1.2 Mechanical component

A tissue growth combines two processes, cell expansion and cell division. In our model, cells are subjected to a constant internal turgor pressure that pushes the boundary vertices of the cells allowing for growth. This expansion force acts isotropically, in the

same way in all directions, and it is limited by constraint that describes the stiffness of the cell wall. In the model, various constraints were implemented using Position-Based Dynamics (PBD) (Marconi et al., 2021; Marconi and Wabnik, 2021).

Briefly, PBD is a recent modeling technique used to simulate dynamics systems that overcome the limitations of classical force-based approaches such as Mass-Spring systems, less realistic as they are highly dependent on the discretization of the tissue (de Boer et al., 1992); or Finite element methods, that are very accurate but computationally expensive and difficult to simulate subcellular events (Bidhendi and Geitmann, 2018; Zienkiewicz and Taylor, 2005). On the contrary, PBD is defined on a single-cell or sub-cellular levels to approximate physical forces whereas being fast and robust to numerical errors. PBD is characterized by avoiding the classical time integration scheme, due to the fact that this method is implicit and thus vertex positions are updated directly. The constraints projections are used to update the positions of the vertices in which a tissue has been discretized so that all constraints are satisfied. PBD source code is available at https://github.com/InteractiveComputerGraphics/PositionBasedDynamics. More details about how these constraints work and how they were adapted for biological tissue growth, together with an example of a 2D tissue modeling, can be found in Marconi et al., 2021.

For the floral and leaf primordia simulations shown in this work, the following constraints were applied:

**- Distance constraint** that mirrors the mass-spring system (de Boer et al., 1992). This constraint defines for each edge a compression and extension stiffness, which controls the distance between connected vertices (Müller et al., 2007). In this model, the compression stiffness is always maximum, avoiding cell shrinkage. On the other hand, the extension stiffness is minimal for the inner cell edges (these edges preserve the structure, but they do not modulate the growth), and for the boundary edges integrate the experimental observations of stiffness measured by AFM. In the internal layers, the extension stiffness is homogeneous between domains and set to low value (Figure S6D), recreating the restriction of inner growth by the epidermis (Zhou et al., 2020). In the epidermal layer the stiffness was set based on the experimental AFM measures and pectin methyl-esterification state shown in flower (Figure 5) and leaf (Figures 1 and 2 in Qi et al., 2017). In the flower model, since adaxial and abaxial have comparable patterns, we set the same extension stiffness for the epidermis in both domains, increasing its value until doubling it at the end of the simulation as it is observed experimentally. In the leaf model, we integrated a more complex dynamic with high stiffness in the adaxial domain in P1 and P2 that decreases until reaching P3 while increasing in the middle

domain based on our experimental measurements (Figure S6D). An integration of this dataset is critical because the model failed to produce a correct leaf morphology by applying the ubiquitous stiffness in all three domains (an overgrowth of the middle domain to the detriment of the abaxial one) (Figures S6E and S6E′). These predictions suggest that the final shape of the leaf is dependent on the complex mechanics of the epidermis.

**- Strain constraint** restricts expansion in a particular direction allowing that cells grow anisotropically (Bender et al., 2017). This restriction is critical to obtain leaf morphology, getting an axisymmetric shape equivalent to a floral primordium without it (Figures S6F and S6F′). Cell growth has been previously described to be favored in the direction orthogonal to the membrane-associated cortical microtubules (CMTs) (Lloyd and Chan, 2004). CMTs drive the oriented deposition of cellulose microfibrils, increasing the stiffness in specific directions (Ali et al., 2014). Therefore, this constraint emulates a directional restriction of the CMTs orientation. CMTs alignment correlates with the direction of maximal stress in the meristem (Bozorg et al., 2014; Burian et al., 2013). To implement this observation in our models, we defined the maximal stress direction for each cell as a vector by adding all the pressure forces acting on cell boundary vertices. Then, in the epidermal cells CMTs preferentially align with principal stress direction. This direction is perpendicular to the outer surface, in accordance with the CMTs orientation observed in leaf and leaf-like organs (Zhao et al., 2020). For the internal layers, the pressure force acting on the vertices is compensated by the tension created in the neighboring cells and, thus, the net force is always zero, therefore we are unable to measure a global maximum stress direction for an individual cell. Nevertheless, CMTs orientation correlates (although to a lesser extent) with the main strain direction (Bozorg et al., 2014). To calculate the principal direction of cell growth, the rectangle of minimum size covering the entire cell is defined, the deformation is then measured along the two dominant axes of the rectangle. Next, we defined CMTs deposition at each step parallel to each of these deformation axes and proportional to the deformation experienced in the orthogonal direction, according to the following formula:

$$\frac{d\,\overrightarrow{CMT}_{ax1|ax2}}{dt} = R_{CMT} * \sum_i^m u(\overrightarrow{CMT}_{ax1|ax2}) * abs((u(\overrightarrow{CMT}_{ax1|ax2}) \cdot (u(\overrightarrow{mem}_i)) * d_{mem}) * (-d_{CMT}) * \overrightarrow{CMT}_{ax1|ax2} \quad (1)$$

$\overrightarrow{CMT}_{ax1|ax2}$ are the two orthogonal vectors that resemble the microtubules deposition along the two axes of growth; $R_{CMT}$ denotes the microtubules deposition rate for each time step; $u(\overrightarrow{CMT}_{ax1|ax2})$ is the unit vector of the microtubules deposition; $u(\overrightarrow{mem}_i)$ is the unit vector orthogonal to membrane section *mem*; $d_{mem}$ is the deformation of the

membrane section *mem*; $d_{CMT}$ indicates the microtubules decay rate. Inner cells experience limited growth along only the axis that is occupied with CMTs.

Additional two constraints, shape and bending (Bender et al., 2017; Müller et al., 2007), were implemented to improve the stability of the simulations, but without any implications for the differential growth. These constraints preserve the initial morphology of the cell, mimicking the cytoskeleton dynamics that prevent cell collapse.

### 1.3 General cell division rule

Each cell divides after it reaches a maximum threshold for area (see $div_{threshold}$ in Methods S2.). The division line pass through the centroid of the cell and aligns with the maximal stress direction for the epidermis (reinforcing the tissue in the direction in which the cells suffer the highest tension; Louveaux et al., 2016) or with the shortest path for the internal layers, creating two daughter cells of equal size, following Errera's rule (Besson and Dumais, 2011).

### 1.4 Biochemical component

By applying only the mechanical components described above, we were able to fit well leaf, but not flower (Figures S6G and S6G′). To solve it, we modeled auxin flow dynamics according to the with-the-flux hypothesis (Stoma et al., 2008), in which cells sense the net flux of auxin and promote the polarization of PIN auxin carriers according to the flux. As a result, the existing fluxes of auxin are stabilized and amplified. In the meristem, the direction of auxin efflux transport largely depends on PIN-FORMED1 (PIN1) auxin efflux carrier (Zhou and Luo, 2018). In the models, auxin is exported to an intercellular space by PIN1. In turn, PIN1 production and trafficking to the plasma membrane is induced by intracellular auxin (Jönsson et al., 2006; Krogan et al., 2016), and PIN polarization is therefore favored in the direction of the net flux. Furthermore, auxin import is known to be mediated by AUXIN1/LIKE AUXIN1 family of auxin influx carriers in the meristem (Bainbridge et al., 2008). However, we simplified the model assuming that auxin import rates are balanced by the auxin export rates of the neighboring cells in the previous step. The particular equations that determine the auxin flow are detailed below. Parameter values are listed in Methods S2. The biochemical equations were numerically solved using the explicit Euler method.

As discussed above, the boundary edges delimit each cell from the neighbors (with a virtual intercellular space between them). Each of these edges denotes a section of the membrane (called *mem*) with its own chemical attributes. The change of auxin inside a cell ($dAuxin_{cell}$) depends on basal expression, passive diffusion from the intercellular space, active transport mediated by PIN1, and degradation. Similarly, the change of auxin in the intercellular space ($dAuxin_{mem}$) results from the combination of passive

diffusion from the cytoplasm, active transport, and degradation.

$$\frac{dAuxin_{cell}}{dt} = b_{auxin} + \sum_i^m Diffusion_{mem(i),cell} + \sum_i^m Transport_{mem(i),cell} - d_{auxinCell} * Auxin_{cell} \quad (2)$$

$$\frac{dAuxin_{mem}}{dt} = -\sum_i^m Diffusion_{mem(i),cell} - \sum_i^m Transport_{mem(i),cell} - d_{auxinMem} * Auxin_{mem} \quad (3)$$

$$Diffusion_{mem,cell} = P_{mem} * (Auxin_{mem} - Auxin_{cell}) * \frac{L_{mem}}{A_{cell}} \quad (4)$$

$$Transport_{mem,cell} = Import_{mem,cell} - Export_{mem,cell}$$

$$Export_{mem,cell} = K_{PIN1} * PIN1_{mem} * Auxin_{cell} * L_{mem};$$

$$Import_{mem,cell} = Export_{mem,n(t-1)} \quad (5)$$

$Auxin_{cell}$ is the amount of auxin inside the cell; $b_{auxin}$ is the constant auxin basal expression; $d_{auxinCell}$ is de degradation coefficient for auxin inside the cell; $Auxin_{mem}$ denotes the available auxin in the intercellular space on the membrane section *mem*; $d_{auxinMem}$ is the degradation coefficient for auxin in the intercellular space; $P_{mem}$ denotes the permeability coefficient of the membrane; $L_{mem}$ is the length of the membrane section *mem*; $A_{cell}$ is the cell area; $K_{PIN1}$ is the coefficient of auxin exporting rate; $PIN1_{mem}$ is the amount of PIN1 protein localized on the membrane section *mem*. For $Import_{mem,cell}$ , $Export_{mem,n(t-1)}$ refers to the amount of auxin exported by the neighbour cell (*n*) in the previous time step (*t-1*) throughout the membrane section *mem* shared by both cells.

PIN1 shows a basal expression level ($b_{PIN1}$) and auxin-inducible expression ($AuxinInduced_{cell}$):

$$\frac{dPIN1_{cell}}{dt} = b_{PIN1} + AuxinInduced_{cell} - d_{PIN1Cell} * PIN1_{cell}$$

$$AuxinInduced_{cell} = PIN1_{expr} * \frac{(\frac{Auxin_{cell}}{A_{cell}})^2}{{PIN1_K}^2 + (\frac{Auxin_{cell}}{A_{cell}})^2} * \frac{{PIN1_{MaxCell}}^8}{{PIN1_{MaxCell}}^8 + (\frac{PIN1_{cell}}{A_{cell}})^8} \quad (6)$$

$PIN1_{cell}$ denotes the amount of PIN1 inside the cell; $b_{PIN1}$ is the basal production rate for PIN1; $d_{PIN1Cell}$ is the degradation coefficient for PIN1 in the cytoplasm; $PIN1_{expr}$ is the maximal expression of PIN1 induced by auxin; $PIN1_k$ is the half concentration of auxin in the cell that promotes maximal PIN1 expression; $PIN1_{MaxCell}$ is the maximal PIN1 concentration allowed in the cell; $Auxin_{cell}/A_{cell}$ and $PIN1_{cell}/A_{cell}$ are the current auxin and

PIN1 concentrations inside the cell, respectively.

PIN1 trafficking to the section membrane *mem* is promoted by auxin and it depends on a sensitivity measure determined by the net auxin flow in the cell. The higher amount of auxin has been exported throughout this membrane section, the more likely it is to incorporate more PIN1 molecules at each step. To calculate the sensitivity, we measured the alignment between the cell auxin flux vector and the orthogonal vector to the section membrane *mem*. The final PIN1 sensitivity is obtained by dividing it by the arithmetic average of all raw PIN1 sensitivities:

$$\frac{dPIN1_{mem}}{dt} = PIN1_{cell} * PIN1_{tr} * \frac{Auxin_{cell}}{Auxin_{cell}+1} * PIN1Sensitivity_{mem,cell} * \frac{{PIN1_{MaxMem}}^{10}}{{PIN1_{MaxMem}}^{10} + (\frac{PIN1_{mem}}{L_{mem}})^{10}} - d_{PIN1Mem} * PIN1_{mem}$$

$$PIN1Sensitivity_{mem,cell} = KP * |Flux_{cell}| * \frac{FV^4_{mem,cell}}{K^4_{flux}+FV^4_{mem,cell}}$$

$$FV_{mem,cell} = u(\overrightarrow{Flux_{cell}}) * u(\overrightarrow{mem}); \qquad if\ FV_{mem,cell} < 0 \ \rightarrow \ FV_{mem,cell} = 0$$

$$\overrightarrow{Flux_{cell}} = \sum_i^m Transport_{mem(i),cell} * u(\overrightarrow{centroid_{cell}} - \overrightarrow{midpoint_{mem}}) * dt \qquad (7)$$

*PIN1*$_{mem}$ is the amount of PIN1 on the membrane section *mem*; *PIN1*$_{cell}$ and *Auxin*$_{cell}$ are the PIN1 and auxin amount available inside the cell, respectively; *PIN1*$_{tr}$ is a trafficking scaling factor; *PIN1*$_{MaxMem}$ denotes the maximal PIN1 concentration allowed on the membrane section *mem*; *PIN1*$_{mem}$/*L*$_{mem}$ is the current concentration of PIN1 on the membrane section *mem*; $d_{PIN1mem}$ is the degradation coefficient for PIN1 on the membrane; *KP* is a scaling factor for PIN1 sensitivity; $\overrightarrow{Flux_{cell}}$ is the auxin-flux vector (|*Flux*$_{cell}$| is the modulus of this vector); $K_{flux}$ is the half-activation coefficient of PIN1 sensitivity; $u(\overrightarrow{Flux_{cell}})$ is the unit vector parallel to the auxin-flux vector; $u(\overrightarrow{mem})$ is the unit vector orthogonal to the membrane section *mem*; $u(\overrightarrow{centroid_{cell}} - \overrightarrow{midpoint_{mem}})$ is the unit vector from the centroid of the cell to the midpoint of the membrane section *mem*.

**1.5 Connection between chemical and mechanical model**

Auxin is known to promote or inhibit cell wall loosening and, thus, modulate the extensibility of plant cell walls (Rayle and Cleland, 1992). To integrate this effect into our mechano-biochemical framework, we considered auxin as a growth factor which is able to modulate the rest length of the distance constraint. Thus, the increase in rest length depends on the amount of auxin, combined with the strain (see equation 8), based on

the Lockhart's model (Lockhart, 1965). A threshold of strain is used to recreate the elasticity of the cell walls:

$$\frac{RestLength_{wall}}{dt} = G * AuxinEffect * Strain$$

$$AuxinEffect = (k * [Auxin])^{n1} * \frac{[Auxin]^{n2}}{(km+[Auxin])^{n2}} + b \quad (8)$$

*G* denotes a constant growing scaling factor; *AuxinEffect* is the contribution of auxin to wall extension; *Strain* is the predicted deformation of the wall in the current time step; *[Auxin]* is concentration of auxin in the cell delimited by the given wall or the average concentration of auxin of the two cells adjacent to that wall; *k* indicates the coefficient of auxin-induced growth; $k_m$ is the half activation factor of auxin-induced growth; *b* refers to the effect over growth of a cell without auxin; *n1* and *n2* are exponential coefficients. Optimal values for *k*, *km*, *n1*, *n2* and *b* are shown in Methods S2.

**1.6 Model setup**

Before model simulations each cell is associated with a particular domain (adaxial, middle or abaxial) and a layer (either epidermis or internal). All the progeny generated from one of these initial stem cells inherit the domain definition from the mother cell to get a realistic leaf primordium morphology. Furthermore, we set the same initial auxin concentration for leaf and floral primordia (Figure S6C). At the incipient stage both primordia act as auxin sinks, creating an auxin maximum in the epidermis of the meristem (Benková et al., 2003; Reinhardt et al., 2003). When outgrowth begins, our starting point, PIN1 polarity reverses, triggering the depletion of auxin from this maximum through the incipient provascular tissue (Heisler et al., 2005). Finally, an auxin import from the meristem to the growing primordia occurs through the epidermal layer (Benková et al., 2003). Accordingly, the two cells at the ends of the epidermis (one in adaxial and the other in abaxial) in contact with the meristem tissue produce an additional auxin amount. This production rate increases over time:

$$Source = s0 * (\frac{t(h)}{s1})^{s2} + s3 \quad (9)$$

*t(h)* denotes current time in hours; *s0*, *s1*, *s2* and *s3* parameter values are listed in Methods S2.

**1.7 Additional model assumptions predicted from the analysis of high throughput model simulations**

To optimize the fit of the model predictions to the experimental observations and isolate the best-performing models that are shown in Figure 7, we incorporate additional

assumption based on the analysis of prediction coming from high throughput model simulations run on the supercomputing cluster:

**a**. the strain constraint of the epidermis of the middle domain in leaf: the growth is limited in the direction orthogonal to the maximal stress direction (instead of parallel as in the adaxial and abaxial domains), based on the behavior already described for the central zone of the shoot apical meristem, in which the main strain and stress directions align (Bozorg et al., 2014). This assumption significantly improved the model fit to experimental measurements (Figures S6H and S6H′).

**b**. the strain constraint of the internal layers in leaf: CMTs deposition is favored in the adaxial-abaxial axis to reproduce the correct leaf primordium morphology, as experimentally reported in Zhao et al., 2020. Relaxing this assumption, either in the middle or the abaxial domain, led to aberrant leaf morphologies (Figures S6I and S6I′). This finding highlights the importance of the anisotropic cell growth for establishing the leaf bilateral symmetry.

**c**. cell division: the threshold area for division was increased by factor of 2 for the abaxial domain, allowing larger sizes of abaxial cells. Although a good fit for the growth rates can be found without considering this assumption (Figures S6J and S6J′), however, cell morphologies did not match the experimental observations shown in Figures S6A and S6B.

**d**. Epidermis is less sensitive to the auxin-mediated cell elongation (parameter *AuxinEffect* in equation 8). Therefore, the same relative auxin concentrations trigger less elongation of the walls in the epidermis than in the internal layers, strengthening the epidermal restriction of growth (Zhou et al., 2020). Lack of this assumption leads to high concentration of auxin in the epidermis, which causes an unrestricted growth of the cells in this layer towards the outer space. Also, the auxin flux hardly internalizes into the underlying layers (Figures S6K and S6K′).

**e**. Initial conditions: we assumed that the amount of auxin coming from the meristem to the primordia depends both on the type of primordium (leaf or flower), and the source site, either in adaxial or abaxial domains. Introducing the same amount of auxin through both source cells resulted in a good fit of the leaf model, but not the flower model. In the flower model, this results in the excessive growth of the adaxial domain with respect to the abaxial one which is not observed experimentally. Even more, the shift in the auxin drainage path from the boundary to the adaxial domain is advanced, being completed 42h after starting the simulation (Figures S6L and S6L′). On the contrary, the auxin drainage path in the adaxial domain does not dominate until P5 (60h of growing; see Figure 6O′-6S′). However, by assuming different amounts of auxin for both auxin sources (equation 9), the new auxin drainage path is correctly established between P4 and P5

(Figure 7A and Video S2).

### 1.8 Parameter sensitivity analysis

To test the robustness of the model we varied ±10% the value of some critical parameters using high-throughput simulations on the supercomputing cluster. Then, we compared the predicted average cellular growth rates for the epidermis in each domain, by measuring the deviation between the predictions and the experimental results (and also the best-fit simulations shown in Figures 7C and 7D). The general conclusion is that both floral and leaf primordia models are generally robust. In particular, the models show a very high stability when we modified $R_{CMT}$, the reorientation rate of the CMTs in the internal layers based on the deformation that the cell undergoes in each step (Figure S7K). Similar results were obtained, for the chemical model of auxin transport, by changing $P_{mem}$ (membrane permeability for passive diffusion) (Figure S7M); *KP*, which modulates the capacity of auxin to promote PIN1 location on the membrane (Figure S7N); or $K_{PIN1}$ (auxin export rate) in flower (Figure S7L). Remarkably, we found that $K_{PIN1}$ in leaf shows a higher sensitivity in the adaxial domain, with variations of up to 1.25-fold from the experimental reference. $K_{PIN1}$ alterations mimic an overexpression/downregulation of PIN1 transporter and presented prediction of growth rates for PIN1 knock-down mutant or overexpressor could be experimentally tested in the future. Regarding the link between mechanical and chemical models, we observed higher overall growth in all domains as the wall elongation rate G increases (Figure S7O). Finally, when we modified the output value for *AuxinEffect* (equation 8), the total amount of auxin coming from the meristem to the primordia, again we got high stability either by altering both sources or just one of them (Figures S7P-S7R).

## 2. References


- Ali, O., Mirabet, V., Godin, C., & Traas, J. (2014). Physical models of plant development. *Annu. Rev. Cell Dev. Biol. 30*, 59-78.
- Bainbridge, K., Guyomarc'h, S., Bayer, E., Swarup, R., Bennett, M., Mandel, T., & Kuhlemeier, C. (2008). Auxin influx carriers stabilize phyllotactic patterning. *Genes Dev. 22*(6), 810-823.
- Barbier de Reuille, P., Routier-Kierzkowska, A.L., Kierzkowski, D., Bassel, G.W., Schupbach, T., Tauriello, G., Bajpai, N., Strauss, S., Weber, A., Kiss, A.*, et al.* (2015). MorphoGraphX: A platform for quantifying morphogenesis in 4D. *eLife 4*, 05864.
- Bender, J., Müller, M., & Macklin, M. (2017). A survey on position based dynamics, 2017. In *Proceedings of the European Association for Computer Graphics:* Tutorials (pp. 1-31).
- Benková, E., Michniewicz, M., Sauer, M., Teichmann, T., Seifertová, D., Jürgens, G., and Friml, J. (2003). Local, efflux-dependent auxin gradients as a common module for plant organ formation. *Cell 115*, 591-602.
- Besson, S., & Dumais, J. (2011). Universal rule for the symmetric division of plant cells. *Proc. Natl. Acad. Sci. USA. 108*(15), 6294-6299.
- Bidhendi, A.J., & Geitmann, A. (2018). Finite element modeling of shape changes in plant cells. *Plant Physiol.*, 176(1), 41-56.
- Bozorg, B., Krupinski, P., & Jönsson, H. (2014). Stress and strain provide positional and

directional cues in development. *PLoS Comput. Biol. 10*(1), e1003410.
- Burian, A., Ludynia, M., Uyttewaal, M., Traas, J., Boudaoud, A., Hamant, O., & Kwiatkowska, D. (2013). A correlative microscopy approach relates microtubule behaviour, local organ geometry, and cell growth at the Arabidopsis shoot apical meristem. *J. Exp. Bot. 64*(18), 5753-5767.
- de Boer, M.J., Fracchia, F.D., & Prusinkiewicz, P. (1992). A model for cellular development in morphogenetic fields. In *Lindenmayer Systems* (pp. 351-370). Springer, Berlin, Heidelberg.
- Heisler, M.G., Ohno, C., Das, P., Sieber, P., Reddy, G.V., Long, J.A., and Meyerowitz, E.M. (2005). Patterns of auxin transport and gene expression during primordium development revealed by live imaging of the *Arabidopsis* inflorescence meristem. *Curr. Biol. 15*, 1899-1911.
- Jönsson, H., Heisler, M.G., Shapiro, B.E., Meyerowitz, E.M., & Mjolsness, E. (2006). An auxin-driven polarized transport model for phyllotaxis. *Proc. Natl. Acad. Sci. USA. 103*(5), 1633-1638.
- Krogan, N. T., Marcos, D., Weiner, A. I., & Berleth, T. (2016). The auxin response factor MONOPTEROS controls meristem function and organogenesis in both the shoot and root through the direct regulation of PIN genes. *New Phytol. 212*(1), 42-50.
- Lloyd, C., & Chan, J. (2004). Microtubules and the shape of plants to come. *Nat. Rev. Mol. Cell Biol. 5*(1), 13-23.
- Lockhart, J.A. (1965). An analysis of irreversible plant cell elongation. *J. Theor. Biol. 8*(2), 264-275.
- Louveaux, M., Julien, J.D., Mirabet, V., Boudaoud, A., & Hamant, O. (2016). Cell division plane orientation based on tensile stress in *Arabidopsis thaliana*. *Proc. Natl. Acad. Sci. USA. 113*(30), E4294-E4303.
- Marconi, M., Gallemi, M., Benková, E., and Wabnik, K. (2021). A coupled mechano-biochemical model for cell polarity guided anisotropic root growth. *eLife 10*, e72132.
- Marconi, M., & Wabnik, K. (2021) Shaping the organ: a biologist guide to quantitative models of plant morphogenesis. *Front. Plant Sci. 12*, 2171.
- Müller, M., Heidelberger, B., Hennix, M., & Ratcliff, J. (2007). Position based dynamics. *J. Vis. Commun. Image Represent.*, *18*(2), 109-118.
- Qi, J., Wu, B., Feng, S., Lü, S., Guan, C., Zhang, X., Qiu, D., Hu, Y., Zhou, Y., Li, C.*, et al.* (2017). Mechanical regulation of organ asymmetry in leaves. *Nat. Plants 3*, 724-733.
- Rayle, D.L., and Cleland, R.E. (1992). The Acid Growth Theory of auxin-induced cell elongation is alive and well. *Plant Physiol. 99*, 1271-1274.
- Reinhardt, D., Pesce, E.R., Stieger, P., Mandel, T., Baltensperger, K., Bennett, M., Traas, J., Friml, J., and Kuhlemeier, C. (2003). Regulation of phyllotaxis by polar auxin transport. *Nature 426*, 255-260.
- Stoma, S., Lucas, M., Chopard, J., Schaedel, M., Traas, J., & Godin, C. (2008). Flux-based transport enhancement as a plausible unifying mechanism for auxin transport in meristem development. *PLoS Comput. Biol. 4*(10), e1000207.
- Zhao, F., Du, F., Oliveri, H., Zhou, L., Ali, O., Chen, W., Feng, S., Wang, Q., Lü, S., Long, M.*, et al.* (2020). Microtubule-mediated wall anisotropy contributes to leaf blade flattening. *Curr. Biol. 30*, 3972-3985.
- Zhou, J. J., & Luo, J. (2018). The PIN-FORMED auxin efflux carriers in plants. *Int. J. Mol. Sci. 19*(9), 2759.
- Zhou, L., Du, F., Feng, S., Hu, J., Lü, S., Long, M., & Jiao, Y. (2020). Epidermal restriction confers robustness to organ shapes. *J. Integr. Plant Biol. 62*(12), 1853-1867.
- Zienkiewicz, O.C., & Taylor, R.L. (2005). The finite element method for solid and structural mechanics. Elsevier.