Modeling the emergence of polarity patterns in meristemic auxin transport

Silvia Grigolon [1,*], Peter Sollich [2] and Olivier C. Martin [1,3]

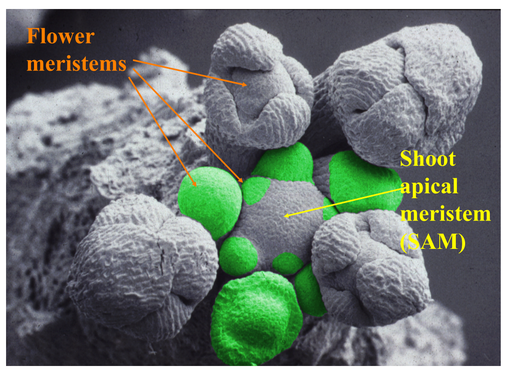
[1] LPTMS, Université Paris - Sud XI, 15, Rue Georges Clémenceau, 91405 Orsay CEDEX, France,
[2] Department of Mathematics, King's College London, Strand, London, WC2R 2LS UK,
[3] INRA, UMR 0320/UMR 8120, Génétique Végétale, Univérsité Paris - Sud XI, F-91190 Gif - sur - Yvette, France
[*] silvia.grigolon@lptms.u-psud.fr

Abstract

Morphogenesis is a key phase of multi-cellular life. A remarkable feature arising in plants is **phyllotaxis**, a highly ordered spatio-temporal program which drives the emergence of organs such as leaves and flowers. Their initiation takes place in **meristems**, tissue containing stem cells with an high potential to proliferate and differentiate into any of the plant's organs throughout the whole life of the plant. The choice of which organ to initiate depends on the cues received by the cells, be they from the environment or as signals produced by the plant itself. Among these signals, the **hormone auxin** plays a central role because its accumulation leads to the initiation of new organs. Like many other molecular species, auxin is actively transported and the resulting distribution of this hormone is key to the plant's architecture.
Here, we focus on the very early stages of meristem organization in *A. thaliana*, a model plant species, introducing a **robust toy model** aimed at reproducing the mechanisms mentioned above through a reaction - diffusion approach, enriched by active transport, carried out in this specific case by two different transporters, AUX1 and PIN1. These last transporters are found to form polarity patterns driving auxin flux throughout the meristem.
The aim of this work is to understand how these patterns can self - organize using as key ingredients what is known so far about PIN1 dynamics and auxin transport.

The Shoot Apical Meristem and auxin transport

The **Shoot Apical Meristem** (SAM) is a group of dividing cells that generate all the aerial parts of the plant (leaves, flowers, etc.). These organs are essential for **development** and **reproduction**. Focusing on flowers, one distinguishes 4 types of organs from the exterior towards the interior: ***sepals***, ***petals***, ***stamens***, ***carpels***. They arise following a very ordered spatio – temporal structure, generally corresponding to a spiral with an angle of gyration of 137.5, i.e., Fibonacci's spiral.



Microscopic picture of the meristem with highlighted organ initiation.

As pointed out in [2], the main morphogen in SAM differentiation is the **hormone auxin** that undergoes at the tissue level two different phenomena in and outside cells, e.g.:

- diffusion;
- active transport, i. e., carriers - mediated.

Carriers are specific for in and out - going flux, respectively called AUX1 and PIN1 [3, 4]. These last carriers are found to mainly direct morphogenesis throughout the SAM localizing on the plasma membrane via recycling between this one and the intracellular endosomal compartments in order to give a preferential direction (*polarization*) for auxin flow [6]. The deep mechanisms driving this phenomenon are still unclear but once polarity pattern is set it leads to auxin accumulation switching organ initiation [5].

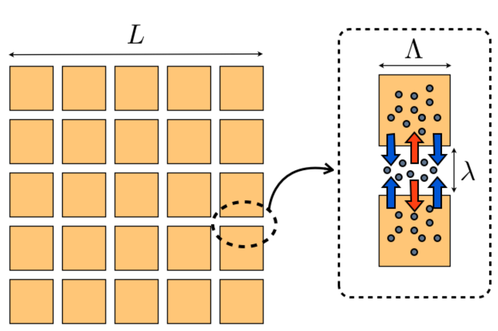
The model

SAM is here modeled as a *bipartite lattice*, i.e., is composed of two different types of sites corresponding respectively to **cells** and **apoplasts**, that is the space in between two adjacent cells. Apoplasts are normally much smaller than cells whose typical length is assumed to be $\Lambda \simeq 20\mu$m.

In cells but not in apoplasts, auxin can be produced with a constant rate β and degraded simply following the mass action law at a rate δ_A:

$$\emptyset \xrightarrow{\beta} A,$$

$$A \xrightarrow{\delta_A} \emptyset.$$



Auxin transport throughout the lattice takes place by **diffusion** induced by thermal fluctuations ($T_{env} \simeq 300K$) with diffusion constant D_A and by **active transport**.

• **Ingoing auxin flux:** on the cell membrane we assume there is a fixed number of AUX1, carriers aimed to facilitate auxin access into the cell, and the flux associated to this phenomenon is analogous to those of *enzymatic reactions* with an substrate and a product. Here, the role of substrate is played by auxin in apoplasts, A_a, while the role of product by auxin in cells, A_C, so that the flux is:

$$\phi_{IN} \propto N_{AUX1} \frac{A_a}{1 + \frac{A_a}{A^*} + \frac{A_C}{A^{**}}}.$$

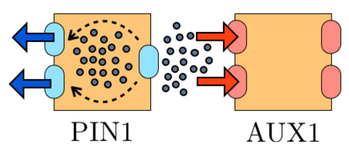
• **Outgoing auxin flux:** outgoing auxin flux is mediated by PIN1 molecules acting again on cell membrane as pumps. We assumed the same sort of process as above for AUX1 but with two main differences:

- the roles of substrate and product are now reversed and taken respectively by cell auxin, A_C, and apoplast auxin, A_a. Auxin outgoing flux through the $i-$th side of a cell is given now by:

$$\phi_{OUT-i} \propto NPIN_i \frac{A_C}{1 + \frac{A_a}{A^*} + \frac{A_C}{A^{**}}},$$

  where i =(Right, Left, Up, Down) and $NPIN_i$ is the amount of PIN1s on the $i-$th side of a cell.

- PIN1 are NOT FIXED on cell membrane but they undergo a dynamics and this phenomenon is known as *recycling*.



PIN1 recycling and polarization

Recycling phenomenon concerns PIN1s tendancy to flow within the cell according to the amount of auxin to pump out. We assumed this process to have a reaction rate depending somehow on the outgoing auxin flux tuned by a Hill - like coefficient h. The overall dynamics is furthermore constrained to ensure PIN1s conservation inside any given cell. Dynamics for PIN1s on the i-th face of the cell under consideration is then:

$$\frac{dNPIN_i}{dt} = -\frac{3}{4}\frac{NPIN_i}{1 + (\frac{\phi_{OUT-i}}{\phi^*})^h} + \frac{1}{4}\sum_{i \neq j}\frac{NPIN_j}{1 + (\frac{\phi_{OUT-j}}{\phi^*})^h}.$$

The *polarization of a cell* is a vector showing outgoing flux direction and defined as:

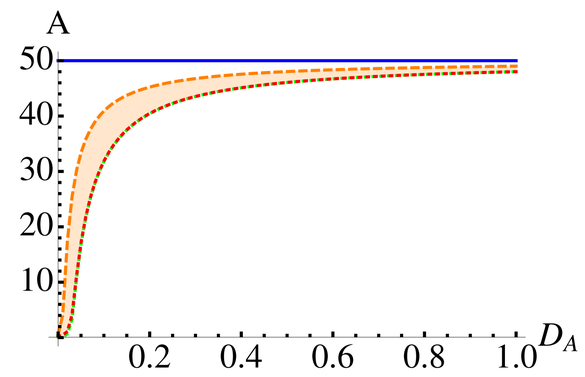
$$\vec{\delta} \equiv (\frac{NPIN_R - NPIN_L}{NPIN_{TOT}}, \frac{NPIN_U - NPIN_D}{NPIN_{TOT}}).$$

Its intensity takes values in $[0, 1]$: extremal values correspond respectively to the **unpolarized state** (equal number of PIN1s on each side of the cell) and to the **polarized state** (one side of the cell has a PIN1 amount much higher than the others).

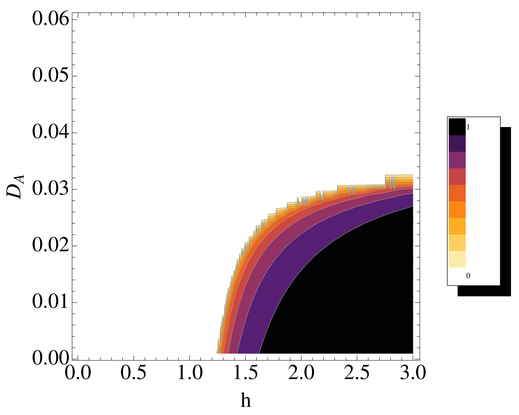
Results

Assuming the *homogeneous configuration* for auxin both in cells and in apoplasts and *uniform polarization* for PIN1s, auxin concentrations as a function of diffusion constant can be obtained.

- auxin in cells does not vary as a function of diffusion constant (blue thick line);
- apoplasts are more and more enriched in auxin as D_A increases (orange and red dotted lines).



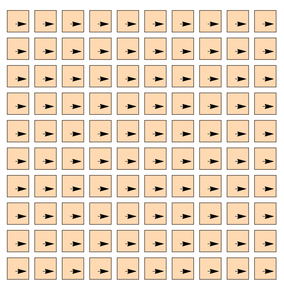
Auxin concentration in cells and apoplasts. For apoplasts, the two bounds are set by the global conditions on PIN1s, i.e., either polarized (lower) or unpolarized (upper).



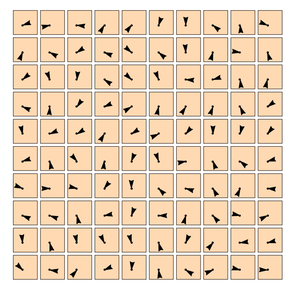
Studying PIN1 dynamics, using the values previously computed for auxin, a well defined region for polarized patterns arises. In the low diffusion regime ($D_A \lesssim 0.03$ $\frac{\mu m^2}{s}$) and high Hill – like coefficients ($h \gtrsim 1.2$), PIN1s are localized on the cell membrane.

• **Typical auxin configurations in presence of molecular noise**
Stochasticity within the model is given by molecular noise ensuing chemical reactions and Poissonian distribution plausibly characterizes noisy dynamics:

$\mathcal{P}(\Delta n_k(t)) = \frac{1}{\Delta n_k(t)!} e^{-\Delta n_k(t)}$, where $\Delta n_k(t) = n_k(t+1) - n_k(t)$, say $n_k(t)$ the number of k-th species molecules at time t, and k =auxin, PIN1 .



Typical steady state configuration for $D_A \simeq 10^{-4}$ $\frac{\mu m^2}{s}$ (low diffusion regime) and $h = 3$. PIN1 are localized on the right - hand side of the cell as they have been initialized.



Typical steady state configuration for $D_A \simeq 0.6$ $\frac{\mu m^2}{s}$ (high diffusion regime) and $h = 3$. PIN1 are not anymore localized although they have been initialized in that way.

References

[1] S. Grigolon, P. Sollich, O. C. Martin, *in preparation.*
[2] D. Reinhardt et al., *The Plant Cell*, 12(507), 2000.
[3] D. Reinhardt et al., *Nature*, 426(255), 2003.
[4] J. Kleine - Vehn et al., *The Plant Cell*, 18:3171, 2006.
[5] E. Benková et al., *Cell*, 26, 115(5), 2003.
[6] J. Wisniewska et al., *Science*, ;312(5775), 2006.