Modeling leaf venation patterns

Anne-Gaelle Rolland-Lagan

Introduction

Relatively little is known about the regulation of leaf development, leaf vein pattern formation, and the relationship between them (Dengler & Kang, 2001). Analyses of vascular pattern and leaf shape mutants indicate that common mechanisms regulate both of these aspects, transport of the plant hormon auxin playing a central role (Dengler & Kang, 2001). In particular auxin transport and leaf development have been widely investigated in the weed *Arabidopsis thaliana*. The growing amount of molecular data on auxin transport mechanisms and leaf venation patterns in *Arabidopsis* suggests that enough information may now be available to model these patterns in a realistic way. In this context, we have been working on biologically realistic models of auxin transport and venation formation, and plan to incoporate growth in the models in order to generate venation patterns on a developing leaf.

Models of venation formation in response to auxin

Two biologically plausible hypotheses have been proposed to explain how veins are formed. The first one, named the canalization hypothesis (Sachs, 1981), was based on a series of careful experiments (Sachs, 1981). It predicts that as auxin flows through a tissue, cells that transport more auxin than the others become more efficient in transporting auxin. As a result, strands of increased auxin flux emerge. These can then differentiate into veins. Mathematical models were proposed by Mitchison (1980,1981), which conformed to the canalization hypothesis and created channels of increased flux. The second hypothesis is based on activator-inhibitor systems (Meinhardt, 1984). The canalization hypothesis has been supported by many experimental observations: in particular the venation patterns observed when auxin transport is inhibited qualitatively fits with the idea of auxin canalization (Mattsson et al., 1999). However, the discovery of mutants with disconnected venation patterns (e.g. Koizumi et al., 2000) casted doubts on the canalization hypothesis as it was unclear whether canalization could be compatible with these discontinuities.

We reproduced Mitchison's models of canalization (Rolland-Lagan et al., submitted), and extended them to incorporate current biological knowledge, by simulating the production of auxin efflux carriers to generate polar transport (Figure 1). We also extended the models to three dimensions (Figure 2). Our results showed that canalization could proceed in such a way that zones of increased flux would be discontinuous before forming continuous strands (Rolland-Lagan et al., submitted). Therefore canalization seems the model best supported by experimental data.

Perspectives

The next step will be to incorporate growth into the models and generate realistic vein patterns. To do so, we will use the VV programming environment (Smith and Prusinkiewicz, submitted), and test our models against experimental data. We may also model growth in 3D to study venation formation in the plant stem.

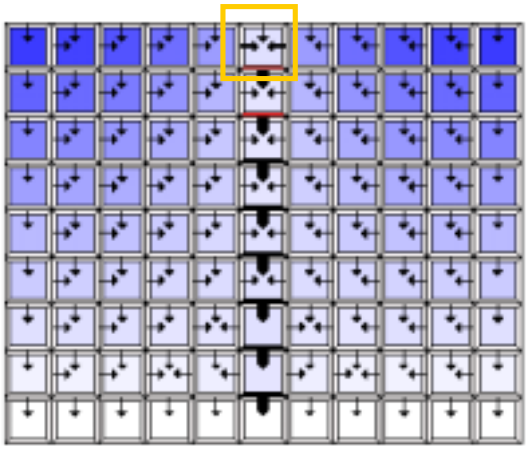

Figure 1. Pattern of fluxes, efflux carriers localisation and auxin concentrations after 10000 steps of simulation. A cell producing auxin is highlighted by an orange square, and an incoming flux of auxin arrives at the top of the tissue. The bottom row of the tissue is a sink for auxin (concentration = 0). Auxin concentration is shown in blue (darker blue for higher concentrations), amount of efflux carriers at the cell wall is shown in red (darker red for high concentrations), up to saturation of carriers which is shown in black. Fluxes of auxin are shown as black arrows (thicker arrows for higher fluxes).

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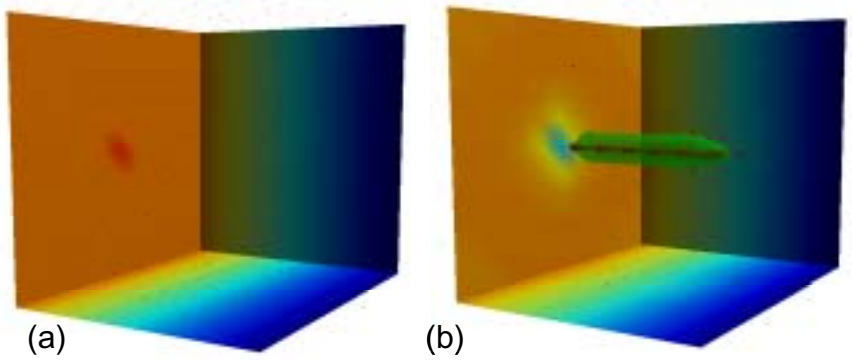

Figure 2. Example of auxin canalization in 3D. Auxin transport is modulated by efflux transporters located at the cell walls, as indicated by biological evidence (Steinmann et al., 1999). The production of efflux transporters is dependent on the direction and magnitude of auxin flux between cells. Hot colors represent higher auxin concentrations. Red cones represent auxin flux from cell to cell (large cones represent large fluxes). in (a), an extra source of auxin is placed at the top of the tissue (red dot signals high auxin concentration), whereas the bottom of the tissue is a sink for auxin. (b) With time, auxin transport away from the source becomes more and more efficient. As the number of efflux transporters increases, a strand of increased auxin flux appears, and concentrations in the strand drop. The zone of high flux is represented by a green isosurface.

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