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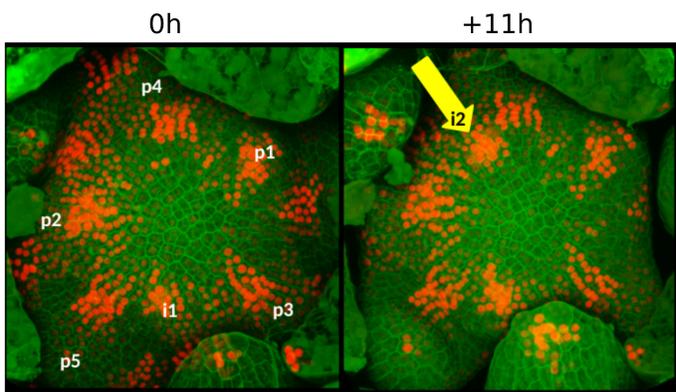
TOWARD A 3D MODEL OF PHYLLOTAXIS

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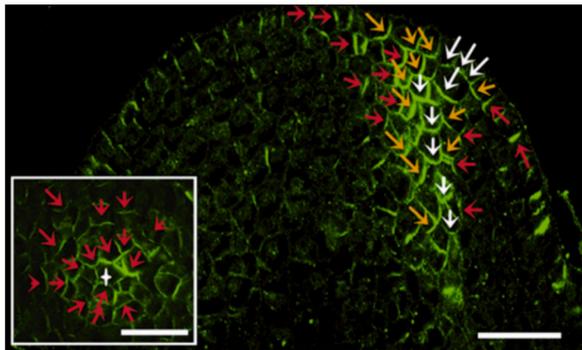
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AUXIN AND PIN1 DYNAMICS IN THE SAM



In the epidermal cell layer (L1) of the shoot apical meristem, auxin accumulates at several points, from which primordia subsequently emerge. (Confocal imaging with DR5 reporter, by Agata Burian.)

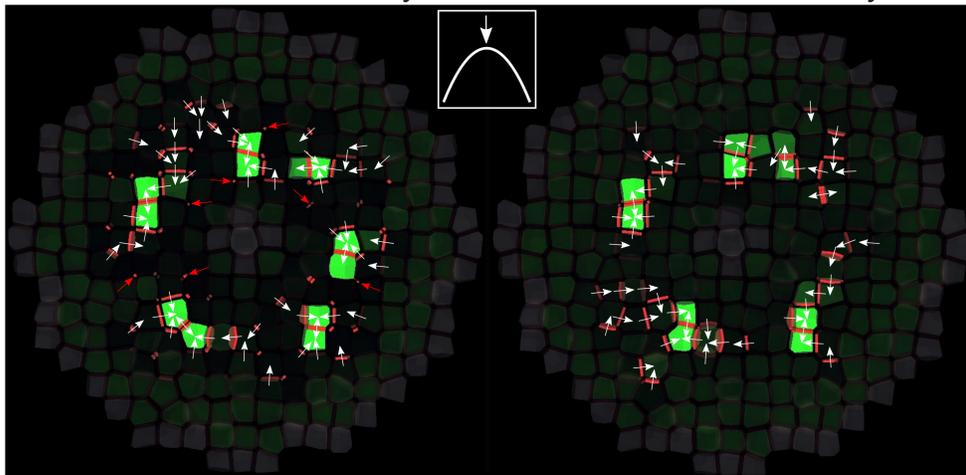


In the L1, PIN1 are polarized toward new primordia (convergence). In the inner tissue, PIN1 canalize auxin from primordia to pre-existing vasculature (venation). From Bayer *et al.* (2009).

LATERAL PIN MOBILITY

without lateral mobility

with lateral mobility

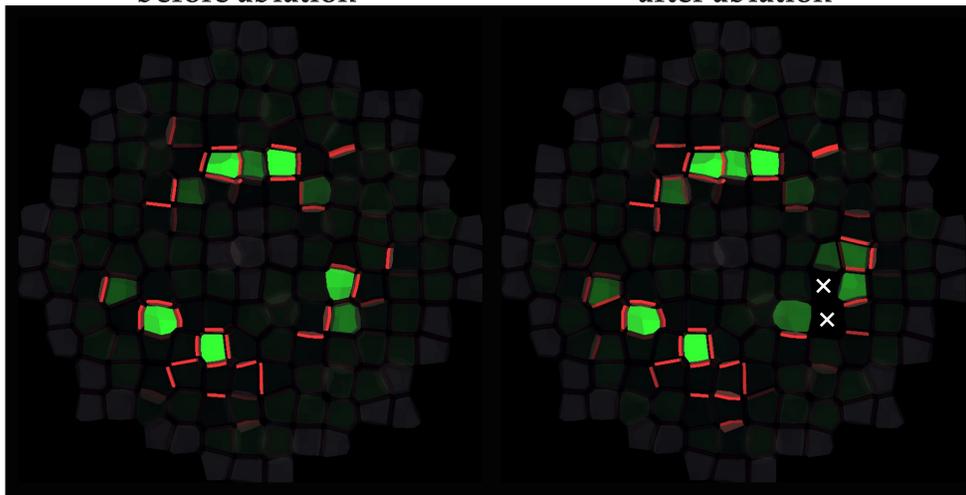


An artifact of 3D discretization is that PINs tend to accumulate on membrane elements with a very small area. This aberration disappears by assuming PIN lateral mobility.

PRIMORDIUM ABLATION

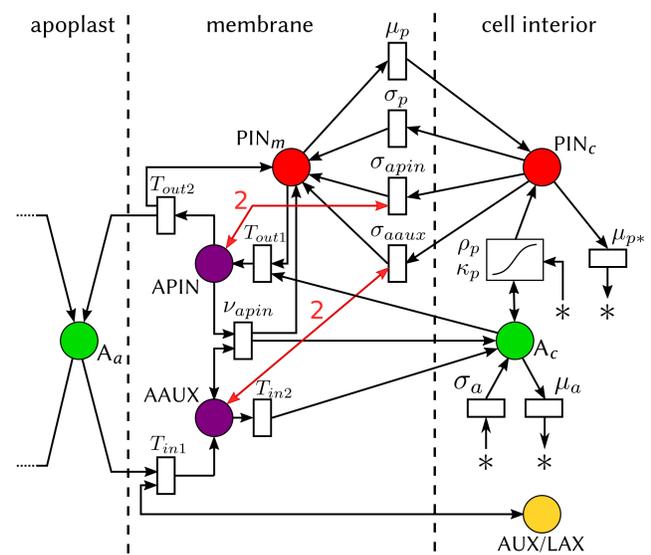
before ablation

after ablation



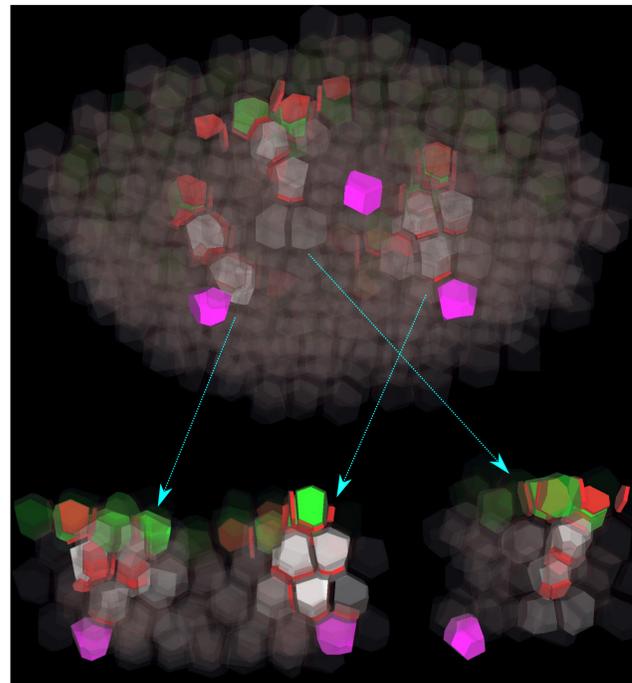
Simulating the affect of the laser ablation of a primordium: A new primordium forms near the ablated site.

BIOCHEMICAL MODEL OF PIN POLARIZATION

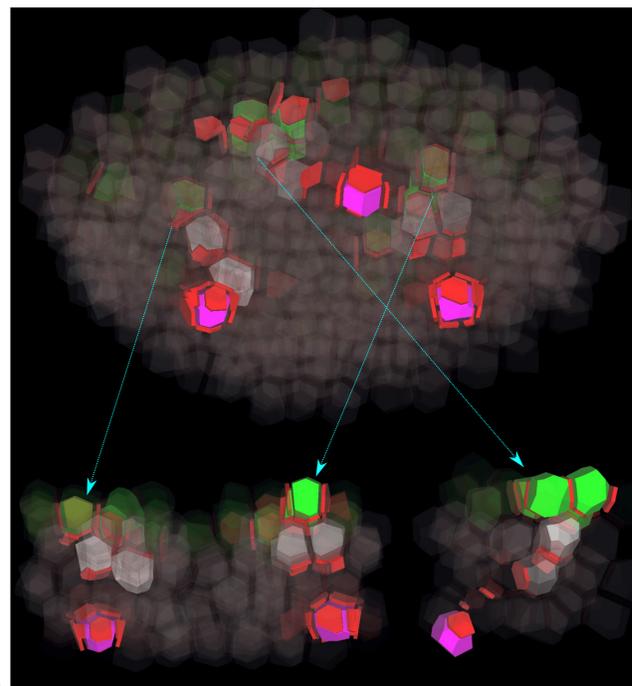


Our simulations are based on a biochemical mechanism of auxin/PIN dynamics by Cieslak *et al.* (2015), which produces both convergence and canalization.

VENATION FROM THE L1 TO THE SINKS



The canalization mechanism in 3D is not very efficient for connecting primordia the the main vasculature: Not all veins eventually connect to a sink.



We hypothesize that already-existing vasculature releases a **Vein Attraction Factor (VAF)** which diffuses within the apoplast. When VAF binds a membrane, it favors PIN allocation to that medmbrane. This ensures that progressing veins reach the main vasculature.

REFERENCES

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- E. M. Bayer, R. S. Smith, T. Mandel, N. Nakayama, M. Sauer, P. Prusinkiewicz, and C. Kuhlemeier, Genes & Development 23, 373 (2009).
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Videos here!