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An auxin transport model for regulation of plant organ initiation

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A plant continuously develops new organs throughout its adult life. New leaf and flower primordia, for instance, are initiated in the shoot at the flanks of a tissue called the Shoot Apical Meristem (SAM). The spatial placement of new primordia (the phyllotactic pattern) is very distinct and results in astonishingly regular patterns. A common phyllotactic pattern, found in *e.g.* the model plant *Arabidopsis thaliana*, is a spiral where the angular divergence between consecutive primordia is close to the golden angle. We have recently described a mechanism based on experimental findings that provides a plausible explanation for these patterns [2]. Our approach is to combine *in-vivo* confocal imaging and mathematical modeling, and here we further develop the model to include additional details. The work is part of the Computable Plant effort (http://www.computableplant.org).

The plant hormone auxin is essential for the initiation of new organs. It has long been known that auxin is transported within plants in a polarized fashion leading to auxin gradients involved in regulating different aspects of plant development. Polar auxin transport is mediated by the asymmetric localization of cellular influx and efflux carriers transporting auxin across cell membranes, which determines the directions of auxin flow within the plant. An important protein for primordia initiation at the SAM flank is the auxin efflux mediator PIN1, which has a loss of function phenotype where no flowers are developed resulting in a pin-formed plant. Localized addition of auxin to the SAM flank in the *pin1* mutant is sufficient to initiate new primordia. In the SAM, PIN1 is mainly expressed in the epidermal layer of cells and is mainly localized at cell membranes towards newly forming and away from older primordia [1]. This leads to a model where PIN1 dynamically reorients and directs auxin transport towards specific positions in the SAM where new primordia are initiated. This has been verified by extracting PIN1 localization from confocal data and simulation of a detailed auxin transport model using experimental estimates of transport parameters and PIN1 localization. The model is sufficient to localize auxin accumulation at positions predicting new primordia [2]. Auxin has the ability to regulate its own efflux and influx mediators, and a feedback model where auxin in neighboring cells influences the PIN1 localization is able to create regular spatial patterns of auxin concentration peaks. When coupled with a growing shoot tissue this provides a plausible mechanism for how phyllotactic patterns are generated in plants [2].

In our previous model, the known promotion of PIN1 expression by auxin was not included and influx mediators were not explicitly modeled. Auxin influx is mediated by members of the AUX protein family. Although no phyllotactic phenotype for any single *aux* mutant has been seen so far, the influx carriers are known to be important for auxin transport. Here we introduce these features in the model and also include experimentally estimated parameters for auxin degradation and metabolic rates. This leads to a detailed reaction-transport model for auxin including membrane transport as well as apoplastic diffusion. The model is simulated on an epidermal layer shoot tissue including cells, membranes, and cell walls. Among other things, we show that auxin-induced PIN1 expression can lead to destabilization of the patterning mechanism, while auxin-induced AUX1 expression stabilizes patterning.

References

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