

Experiment-based models of phyllotaxis

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Phyllotaxis, the regular arrangement of leaves or flowers around a plant stem, is an example of developmental pattern formation and organogenesis. Phyllotaxis is characterized by the divergence angles between the organs, the most common angle being 137.5° , the golden angle. Models of phyllotaxis must explain its de novo establishment in the radially symmetric embryo, the stable maintenance of the different arrangements and the observed transitions between phyllotactic patterns. Most importantly, they must explain the specific divergence angles of 180° , 90° , 137.5° and in rarer cases other angles as well [1].

This quantitative aspect makes phyllotaxis an unusual developmental problem. It has traditionally attracted the interest of mathematicians and computer scientists, who have constructed a wide variety of simulation models. To the biologist it is surprising that only minimal assumptions about the underlying molecular mechanisms are sufficient to arrive at mathematical models that correctly and robustly recreate phyllotactic patterns. In this presentation I will give an overview of the experimental work on phyllotaxis and how these experiments form the basis for a new generation of simulation models. The interplay between modeling and experiment will be discussed.

Early experimental work [2] showed that phyllotactic patterns can be disrupted by experimental interference, but that they will quickly recover and reestablish the original arrangement. Apparently, aberrant positioning can be somehow corrected. On the other hand, transitions between patterns, for instance from decussate to spiral, occur frequently during the life of a single plant, indicating that developmental switches can override the self-correction mechanism. In our own work we showed that inhibition of polar auxin transport specifically inhibits organ formation but not stem growth or meristem maintenance. Local application of a microdroplet of auxin to the flank of the naked meristem could rescue the defect [3]. The subcellular localization of the auxin transporters was consistent with auxin accumulation at the site of organ initiation. The ensuing model postulates that auxin is an inducer of lateral organ formation, that preexisting organs serve as auxin sinks and that the position of a new organ is determined by active transport of auxin [4].

This conceptual model formed the basis for a new generation of computer simulations that were constructed in collaboration with our colleagues at the University of Calgary [5]. The three key assumptions of the model are that patterning occurs in the L1 surface layer of the meristem, that auxin is readily available within the patterning tissue but is redistributed by a combination of diffusion and active transport, and that auxin transport proteins are positioning towards the neighboring cell with the highest auxin concentration (“up-the-gradient” polarization). Such a model can be tested by experiment, guide further experiments and lead to further development of the model.

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