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Phyllotaxis — a new chapter in an old tale about beauty and magic numbers

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Phyllotaxis, the regular arrangement of leaves and flowers around the stem, is one of the most fascinating patterning phenomena in biology. Numerous theoretical models, that are based on biochemical, biophysical and other principles, have been proposed to explain the development of the patterns. Recently, auxin has been identified as the inducer of organ formation. An emerging model for phyllotaxis states that polar auxin transport in the plant apex generates local peaks in auxin concentration that determine the site of organ formation and thereby the different phyllotactic patterns found in nature. The PIN proteins play a primary role in auxin transport. These proteins are localized in a polar fashion, reflecting the directionality of polar auxin transport. Recent evidence shows that most aspects of phyllotaxis can be explained by the expression pattern and the dynamic subcellular localization of PIN1.

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Current Opinion in Plant Biology 2005, **8**:487–493

This review comes from a themed issue on
Cell signalling and gene regulation
Edited by George Coupland and Salome Prat Monguio

Available online 28th July 2005

1369-5266/$ – see front matter

DOI 10.1016/j.pbi.2005.07.012

Introduction

The leaves, flowers and floral organs of plants are arranged in regular patterns, a phenomenon referred to as phyllotaxis [1]. The prevalent phyllotactic patterns found in nature are distichous (alternate), decussate (opposite) and spiral. Spiral phyllotaxis is the most widespread pattern and is found in mosses, ferns, gymnosperms and angiosperms.

Spiral phyllotaxis has long been of interest to theoreticians because of its peculiarity in following mathematical rules characterized by the Fibonacci numbers [1,2]. By contrast, experimental approaches aimed at revealing the regulation of phyllotaxis have been hampered by the delicacy and the minute size of the meristem in which the organs are formed and their position (and hence the resulting phyllotactic pattern) determined [1]. Moreover, genetic approaches to study phyllotaxis have frequently been complicated by the pleiotropic phenotypes of many phyllotactic mutants [3].

During the past few years, however, thanks to the molecular-genetic tools developed in *Arabidopsis* and to improved experimental techniques, phyllotaxis has become more amenable to direct experimentation. Thus, it became possible to test directly biophysical and biochemical models of phyllotaxis by direct local application of growth regulators to the meristem [4] and by laser ablation of subdomains of the meristem [5]. Biophysical models have received little experimental support, whereas recent evidence supports a biochemical mechanism for phyllotaxis with auxin at its centre.

Leaves 'repel' each other

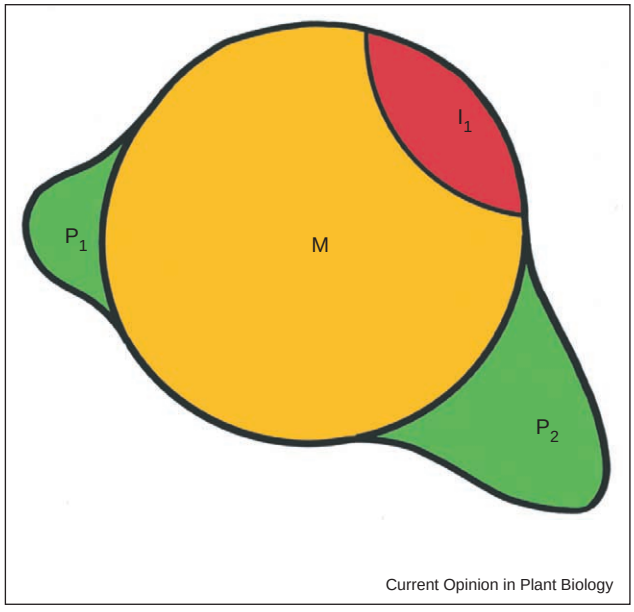
Leaves tend to be formed at a certain minimal distance from each other. This is obvious in distichous phyllotaxis, in which leaves are formed in two opposite vertical rows, as for example in maize. In this case, the divergence angle, that is the angle between successive leaves, is 180°. In decussate phyllotaxis, leaves are formed in pairs with opposite position and subsequent pairs diverge by 90°. Thus, in distichous and decussate phyllotaxis, leaves are formed at maximal distance from each other.

How could the spacing be explained in the case of spiral phyllotaxis in which successive leaves diverge by 137.5°? One possibility is that leaf position is controlled by the two youngest primordia in an asymmetric fashion, with the youngest primordium (P_1) having a stronger repelling effect than the second youngest (P_2), thus forcing the new primordium (I_1) to be formed closer to P_2 than to P_1 (Figure 1; [6]. Indeed, the two youngest primordia (P_1 and P_2) are sufficient to determine the approximate position of I_1, and therefore the direction of the phyllotactic spiral (clockwise or counter clockwise) [7•]. Nevertheless, older primordia in the vicinity of I_1 and P_1 are important for their lateral delimitation [7•], thereby defining their exact final position. Although, the existence of a 'repelling mechanism' of some sort was first documented 70 years ago [8], the nature of the mechanism involved remained elusive until recently.

Auxin comes into the play

Several mutants that have defects in auxin production, transport, or perception exhibit defects in organ initiation, position, number, and delimitation as part of their pleiotropic phenotype [9–15]. This indicated an important role for auxin in organ formation and phyllotaxis. It has been

Figure 1



Schematic representation of an apex with spiral phyllotaxis. P_1 and P_2 are the youngest primordia, the yellow area represents the meristem (M) with the site of incipient leaf formation in red (I_1). P_1 and P_2 (green) diverge by 137.5°. I_1 is positioned in an asymmetric way closer to P_2 than to P_1.

shown that the natural auxin indole-3-acetic acid (IAA) acts as an inducer of organogenesis and influences organ position [4]. Moreover, the expression pattern of an auxin-responsive reporter gene showed high levels of auxin activity at the site of leaf and flower formation [16]. Together, this evidence suggests that auxin might be more than just a permissive factor that is required to promote growth (like a vitamin or an essential component of the cell cycle), but rather acts as an instructive signal: a morphogen [17].

Is auxin the phyllotactic morphogen?

The simplest mechanism to account for a 'repelling' influence of pre-existing primordia on new primordia would be the release of an inhibitor of leaf formation [6]. However, experimental support for such an inhibitory substance is missing. The observation that auxin influences leaf formation and positioning has opened exciting new possibilities for models of phyllotaxis. Could the pre-existing primordia determine the position of new primordia by influencing auxin distribution in the meristem? If primordia accumulate auxin from their vicinity, they could generate peaks in auxin concentration and, at the same time, lead to auxin depletion in their vicinity. Such a mechanism would be attractive because it explains the different aspects of phyllotaxis (i.e. organ positioning, initiation and lateral delimitation) through the activity of one signal, auxin. Furthermore, such a model would be consistent with the pleiotropic phenotypes observed in auxin-related mutants such as *pinformed1* (*pin1*), *pinoid*, and *monopteros*.

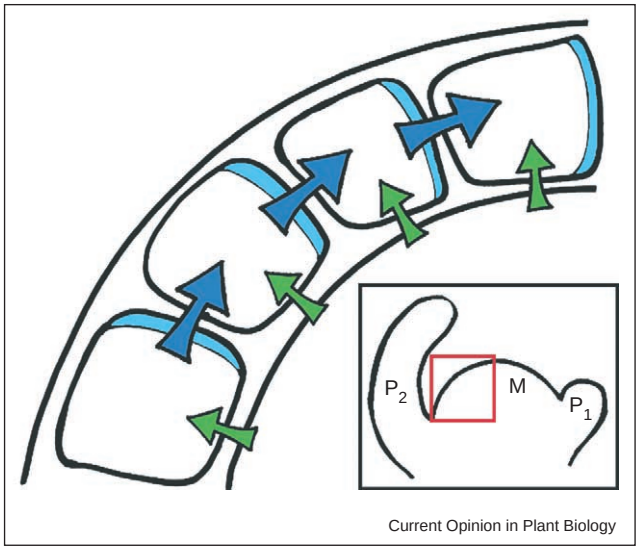
An instructive function implies that differences in auxin concentration are perceived and interpreted by the meristem cells that are deciding which identity to acquire (i.e. organ or non-organ). In a tissue as small as the meristem, the size of which ranges between 50 and 150 μm, however, a small molecule such as IAA (M_r = 175.2) would be expected to diffuse rapidly, thus dissipating concentration differences within minutes. Therefore, the generation and maintenance of auxin concentration gradients in the meristem would require active directional transport. A central component of the active and selective auxin transport system is represented by the PIN protein family, which is involved in auxin efflux from cells [18].

An auxin-based model of phyllotaxis

Polar auxin transport, the directional transport of auxin within a tissue, is mediated by cellular influx and efflux carriers that frequently exhibit an asymmetric subcellular localization [10,19–22]. Auxin can also be transported in the phloem from the sites of synthesis in young organs to sink tissues such as the root [19]. Auxin transport via phloem cannot occur in the meristem, however, because functional vascular strands are not present in this tissue. The shoot meristem of *Arabidopsis* expresses AUXIN RESISTANT1 (AUX1), the founding member of the family of putative auxin influx carriers (AUX/LAX family) [23], as well as PIN1, a member of the efflux regulator family (PIN family) [18]. AUX1 is expressed in the epidermal L_1 layer of the meristem, indicating that cells within this layer accumulate auxin [24]. PIN1 is expressed in the same cells, with an asymmetrical localization on the upper end of the cells (pointing towards the meristem centre), suggesting that auxin is transported upwards into the meristem through the L_1 layer ([24]; Figure 2).

Importantly, PIN1 expression is also induced very early in organogenesis, when its subcellular localization indicates that auxin accumulates in the centre of the young organs ([24]; Figure 3). Taken together, these observations support the following model (Figure 4): first, auxin is delivered uniformly throughout the meristem with no preferred position around the periphery (Figure 2). Second, auxin accumulates in primordia and is depleted in their vicinity (Figure 3). Third, by default, auxin accumulates at a certain minimal distance from the pre-existing primordia (which is beyond the 'reach' of the primordia) (Figure 4a). At this point, auxin induces PIN1 expression and early founder cell identity. This initiates the active accumulation of auxin, resulting in a sharp auxin peak and leading to delimitation of the future organ and auxin depletion from the surrounding cells (Figure 4b). Finally, a new primordium grows out at the site of the auxin peak (Figure 4c).

Figure 2



Auxin transport in the epidermal L_1 layer of the shoot meristem. L_1 cells express the presumptive auxin influx carrier AUX1, leading to the accumulation of auxin in L_1 (green arrows). PIN1 is also expressed in the L_1 layer, where it is localized to the upper side of the cells (light blue). This results in the acropetal transport of auxin towards the meristem centre (blue arrows). Inset: the depicted area in the context of the apex.

This model explains the reiterative nature of phyllotaxis and is consistent with the results of microsurgical and laser ablation studies. Remarkably, ablations of the meristem centre do not affect phyllotaxis [5,25], whereas isolation or ablation of young primordia leads to a shift in position of the subsequent primordia [7•,8]. These observations are compatible with a function of primordia as auxin sinks.

A feature of this model is the conceptual separation of polar auxin transport into an acropetal (i.e. upward) component that is responsible for the delivery of auxin to the meristem (with no phyllotactic information), and a component that mediates the redistribution of auxin in the organogenic periphery (and represents the phyllotactic component of auxin transport). This aspect of the model is in agreement with the observation that inhibition of auxin transport can lead to seemingly contradictory consequences: depending on the stage of plant development and on the experimental conditions, it leads either to the production of bigger and fused organs or to a complete block in organ formation [4,9]. I suggest that in the latter case, the patterning defect is hidden because of the lack of auxin in the meristem, whereas in the former, enough auxin is supplied to the meristem to reveal the patterning defect. The fact that *pin1* mutants are capable of organ formation under certain conditions suggests either that redundant functions of related auxin efflux carriers are active at these stages or that the sources of auxin are close enough to the meristem to allow auxin to reach the meristem by diffusive transport. In this context, it is interesting to note that the cessation of organogenesis in *pin1* coincides with the onset of bolting, whereby the shoot apical meristem becomes progressively more distant from the rosette leaves.

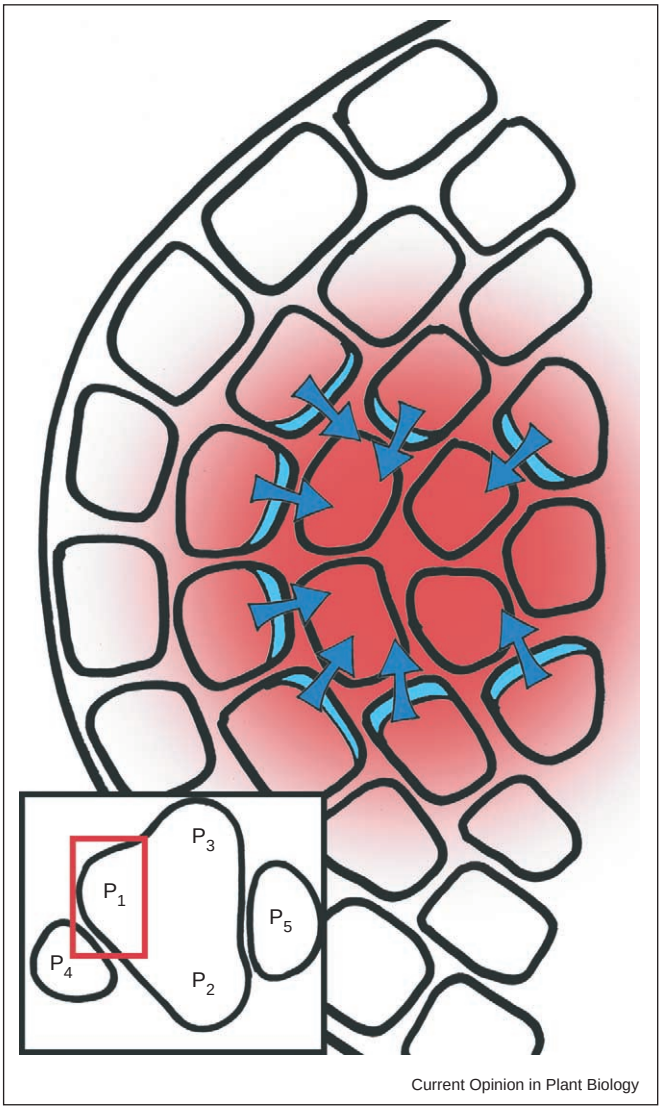
Where does auxin come from?

In general, young tissues such as growing leaves and developing flowers produce auxin [26,27], but we do not know at which stage of primordium development auxin biosynthesis commences. The lack of organ formation in *pin1* mutants and in apices that have been treated with auxin transport inhibitors suggests that auxin is not produced in the meristem itself [4]. However, small apical explants that retain only the meristem and one primordium with a little bit of submeristematic tissues are capable of organ formation without a delay, suggesting that the sources of auxin are close to the meristem [7•]. Similarly, when meristems of pin-shaped apices were relieved from transport inhibition, they resumed leaf formation despite the absence of pre-existing leaves [4]. This suggests that stem tissues are capable of sufficient auxin production to promote organ formation. However, the relative contributions of the different tissues in the apex (i.e. primordia, stem and meristem) to auxin biosynthesis remain largely unknown.

What is upstream of PIN1?

A central feature of our phyllotactic model is the regulation of PIN1 localization. How is the polar localization of the efflux carriers controlled? Although little is known about the mechanism of initial polarization, we have started to understand how PIN1 localization is regulated in the polarized cell. PIN1 is continuously endocytosed to endosomal compartments, and recycled back to the plasmalemma, thus providing a dynamic cellular mechanism that enables rapid changes in PIN1 localization [28]. Hence, it appears that the site of integration of PIN1-containing vesicles into the plasmalemma determines the steady-state subcellular localization. The recycling of PIN1 requires the vesicular trafficking system as a workhorse; but how does the cell know where to deliver its cargo? An important determinant of PIN1 localization is the protein kinase PINOID (PID) [29••]. Although PID might not be involved in the polarization of the cells *per se*, it influences the way in which cells interpret this information and allocate the PIN1 protein. PID decides at which end of the polarized cell PIN1 will accumulate. Consequently, PID can act as a switch that funnels auxin in one direction or the other. This effect has been observed in several cell types in the root and the shoot apex, including the meristem L_1 layer [29••]. A probable explanation for the similarity of the *pin1* and the *pid* phenotypes is that, in *pid* mutants, auxin transport in L_1 goes in the wrong direction, away from the meristem. Is PID also involved in the patterning aspect of phyllo-

Figure 3

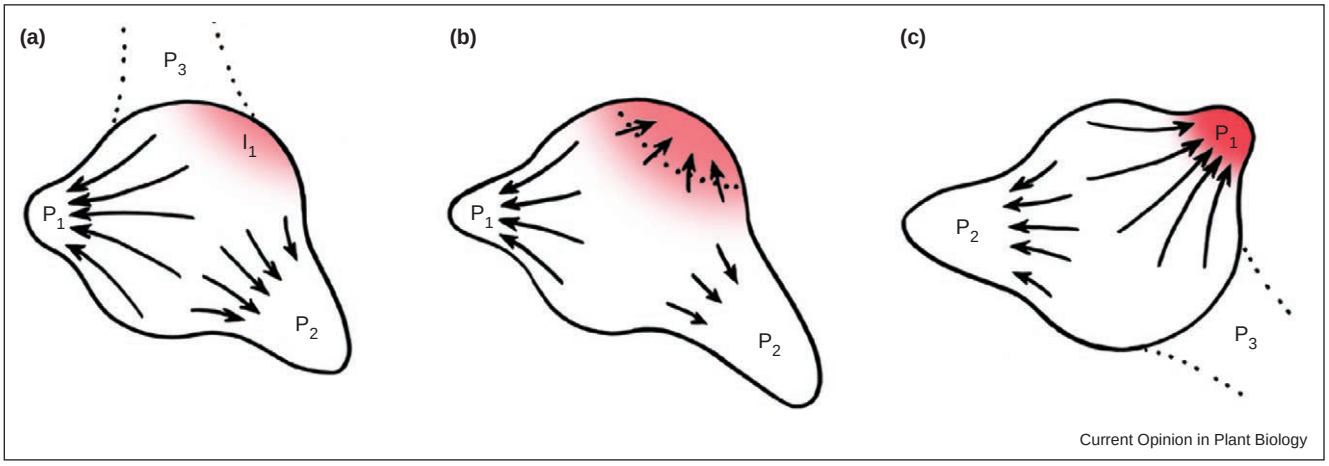


Auxin accumulation in young primordia. PIN1 is induced in young primordia. It becomes localized to the side of the cells that points to the centre of the primordium (light blue). This results in the accumulation of auxin in the primordium and its withdrawal from the surrounding cells (blue arrows). The resulting auxin gradient (red) confers positional information to the cells allowing them to establish organ and boundary identity. Inset: Location of the P_1 position in the context of the apex.

taxis? Although *pid* mutants exhibit patterning defects during embryogenesis and flower formation, they appear more normal than *pin1* during vegetative development [11], and they retain at least part of their patterning capacity in the inflorescence meristem [24]. Hence, not all aspects of PIN1 subcellular localization appear to be regulated by PID. Redundant functions of PID-related proteins might be involved in regulating PIN1 localization in these cases.

Does auxin influence PIN1 expression and localization? Auxin triggers the differentiation and patterning of vascular strands [30]. These contain the PIN1-expressing xylem parenchyma cells, the site of polar auxin transport [10]. Thus, besides acting downstream of PIN1 as a transport substrate, auxin can also function upstream of PIN1 by determining the direction and capacity of its own transport routes. This dual role of auxin is also a feature of phyllotaxis, in which PIN1 expression is induced at the site of organ formation [24,31] or by exogenous auxin [24]. Moreover, the subcellular localization of PIN1 also responds to phyllotactic information, providing a feedback mechanism for the reiterative progression of phyllotaxis ([24]; Figures 3 and 4). It remains to be seen, however, whether PIN1 localization is influenced directly by auxin, or whether a secondary signal released from founder cells and young primordia can relay the phyllotactic information provided by auxin gradients.

Figure 4



Progression of organ positioning and outgrowth during the phyllotactic cycle. **(a)** As a result of the sink function of P_1 and P_2, auxin that is delivered to the meristem by acropetal auxin transport becomes diverted into the primordia (arrows). As a result, auxin (red) can accumulate only slowly at a defined minimal distance from P_1 and P_2, which corresponds to the site of incipient organ formation (I_1). **(b)** At a certain threshold level of auxin, PIN1 becomes induced and begins to actively accumulate auxin at I_1. At the same time, the sink activity of P_1 and P_2 decreases. **(c)** Auxin has been focused to a sharp peak at I_1, leading to the outgrowth of a new organ. Thus, the apex has progressed by one plastochron compared to that shown in (a) (note the change in the nomenclature of primordia). Arrows represent the direction of polar auxin transport; auxin distribution is represented in red. For clarity, only auxin at I_1 is depicted.

Downstream events in organ formation

Little is known about auxin perception and signal transduction in the meristem. Although a candidate for an auxin receptor has been characterized [32], its role in auxin perception in the meristem is not known. Recent evidence shows a prominent role for the F-box protein TRANSPORT INHIBITOR RESPONSE1 (TIR1), a subunit of the SKP1 CULLIN F-BOX/RING-H2 (SCF) complex, in auxin perception [33••,34••]. In the absence of auxin, AUX/IAA proteins (i.e. proteins expressed by genes that are induced by auxin and IAA) prevent an auxin response by binding, and thereby inhibiting, auxin response factors (ARFs), which are transcriptional regulators of downstream auxin responses [35]. In the presence of auxin, AUX/IAA proteins become ubiquitinated by the SCF^{TIR1} complex and subsequently degraded by the 26S proteasome [36], thus leading to the release of the ARFs and the activation of the auxin response. TIR1, a component of the ubiquitin ligase complex SCF^{TIR1}, directly binds auxin and therefore acts as an auxin receptor [33••,34••]. Mutations in the ARF gene *MONOPTEROS* (*MP*) lead to a block in organogenesis similar to that observed in the *pin1* mutant [14]; in contrast to *pin1*, however, the meristem of *mp* mutants does not respond to exogenous auxin [24]. These results document the role of *MP* in the downstream signal transduction of the auxin signal in organ formation.

Many genes in the meristem are expressed in a phyllotactic pattern [3] (i.e. their expression responds to the phyllotactic patterning machinery). At the site of incipient organ formation, members of the KNOX (KNOTTED-like homeobox) gene family, which confer meristem identity, are downregulated, reflecting the acquisition of founder cell identity [37]. Other genes play a crucial role in the elaboration of the organs by defining organ boundaries (*NO APICAL MERISTEM* and *CUP-SHAPED COTYLEDON*) [38,39], organ identity (*LEAFY*) [40], and patterning of the developing organs (*PHANTASTICA* and *PHABULOSA*) [41,42]. In the *pin1* mutant, genes that are normally expressed in a phyllotactic pattern loose this pattern [31], indicating their position downstream of auxin. However, the transport regulators PIN1 and PID themselves loose their phyllotactic expression patterns in the *pin1* mutant, demonstrating their dependence on patterning by auxin. This apparent paradox is due to the fact that phyllotaxis is not regulated by a pathway that is organised into a linear hierarchy but by a mechanism that involves feedback regulation (see also previous section). Therefore, downstream genes that are expressed in developing organs might modulate their auxin sink activity, thereby influencing auxin distribution in the meristem and, as a consequence, the positioning and development of future primordia. In this way, genes that are downstream of auxin (considering the developing primordium P_1) could, indirectly, function upstream of auxin relative to the position of the next primordium (I_1). For further discussion of founder cell identity and downstream regulatory mechanisms in primordium development, the reader is referred to two recent reviews [43,44].

Factors independent of auxin

Besides the range, capacity and rate of auxin transport, other parameters such as meristem size, growth rate and

apical–basal extension growth can be expected to influence phyllotaxis because they indirectly influence auxin transport and the size of the field in which auxin operates. Indeed, changes in cytokinin homeostasis that affect meristem size in maize can lead to a change from distichous to decussate phyllotaxis [45•]. It is conceivable that merely increasing meristem size leads to this dramatic transition, simply by creating space for two leaves to be formed at a time instead of one. Likewise, in the *ultrapetala* mutation of *Arabidopsis*, an increase in organ number is associated with an increase in meristem size [46].

Nevertheless, increased organ number and changes in phyllotactic patterning can also occur in the absence of changes in meristem size: as observed, for example, in the mutants *perianthia* and *bellringer* [47,48]. The homeobox protein BELLRINGER and members of the KNOX family of homeobox transcription factors are involved in a regulatory network that controls the biosynthesis of, and response to, the phytohormones gibberellin and cytokinin in the meristem [49]. Thus, cytokinin, gibberellin, and perhaps other signals can influence phyllotaxis independently of auxin by modulating tissue growth and cell differentiation in the apex.

Conclusions

At the centre of phyllotaxis is auxin, together with PIN1 and PID, which regulate its transport, and MP, which is involved in signal transduction. However, additional upstream and downstream components remain to be identified. Many of the biochemical and molecular details of auxin biosynthesis, transport and perception are still elusive. In particular, the feedback loop that is a central component of the reiterative phyllotactic mechanism remains to be characterized.

Computer modelling has traditionally been employed to develop and test mechanisms of phyllotaxis [2,50–53]. Because of the lack of information on the molecular/cellular basis of phyllotaxis, however, most of these models remained largely abstract. Some of them also failed to express characteristic features such as the robustness and the self-correcting properties of phyllotaxis. With the detailed information from experimental work that is now available, we can build models that are closer to reality. Starting from simulations on the level of small populations of cells [54], it will be possible to generate integrated models that exhibit the known features of phyllotaxis. Such models will help to design future experiments and will direct our attention to crucial questions that remain to be answered. Furthermore, such models might provide hints as to how developmental phenomena, such as the transition from decussate to spiral phyllotaxis in dicotyledonous plants, could be regulated. Ultimately, the combination of experimental and theoretical approaches promises to reveal the origin of phyllotaxis.

Acknowledgements

I thank Jiri Friml, Cris Kuhlemeier, Przemek Prusinkiewicz and Sam Zeeman for critical reading of the manuscript. This work was supported by a grant from the Swiss National Science Foundation (SNF 31-55540.98).

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