J. Plant Biol. (2009) 52:277–282
DOI 10.1007/s12374-009-9034-x

REVIEW

Control of Plant Architecture: The Role of Phyllotaxy and Plastochron

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Received: 1 May 2009 / Accepted: 4 May 2009 / Published online: 28 May 2009

Abstract Plants display a wide variety of three dimensional forms, or architectures, that are critical for their survival in competitive environments or, in the case of crops, for their productivity. Architecture is generated after embryogenesis through the activities of shoot apical meristems and root apical meristems. Leaves are the principal lateral organ that determines the plant shoot morphology, and they normally develop in very regular patterns in time and space. The spatial pattern of leaf arrangement is called phyllotaxy, and the temporal pattern is determined by the plastochron, which is the time between successive leaf initiation events. Both programs involve many gene activities as well as the hormones auxin and cytokinin. Apparently, the mechanisms controlling phyllotaxy and plastochron share some regulatory components. In this review, the molecular mechanisms for both patterning programs will be discussed.

Introduction

Plant development has many unique features compared to that of animals. While animals mostly establish their morphology during embryogenesis, most of plant architecture is elaborated post-embryonically. This post-embryonic development is achieved by pools of stem cells in the shoot and root apical meristems (SAMs and RAMs). Through the activities of the SAM, plants generate lateral organs including leaves. The leaves are not initiated randomly; instead, they develop in very orderly patterns in both space and time. The spatial arrangement of leaves along the plant axis is called phyllotaxy, and the time period between initiations of two successive leaves is called the plastochron. These spatial (phyllotaxy) and temporal (plastochron) patterns of leaf initiation contribute to the unique architectures of specific plant species.

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Types of Phyllotaxy

In general, the type of phyllotaxy can be categorized according to the number of leaves at a given position along the stem (node) and the number of rows in the vertical arrangement along the stem (Fig. 1). Phyllotactic patterns having one leaf per node include “alternate” and “spiral.” Phyllotaxy with two leaves per node is called “opposite” and three or more leaves per node is termed “whorled.” The most common pattern is spiral phyllotaxy, where a single leaf grows out at each node and successive leaves form a spiral pattern with an approximately 137° displacement from the previous leaf. In the whorled arrangement, more than two leaves form at each node. While whorls of leaves can appear along the stem, a single predominant whorl can also form at the base of the shoot.

Other common patterns include “distichous,” with leaves arranged in two rows and “decussate” phyllotaxy with four rows of leaves arranged in opposite pairs. Distichous phyllotaxy is found both in alternate and opposite forms. In the case of decussate phyllotaxy, successive leaf pairs grow out perpendicularly to the previous leaf pair, leading to a four row arrangement.

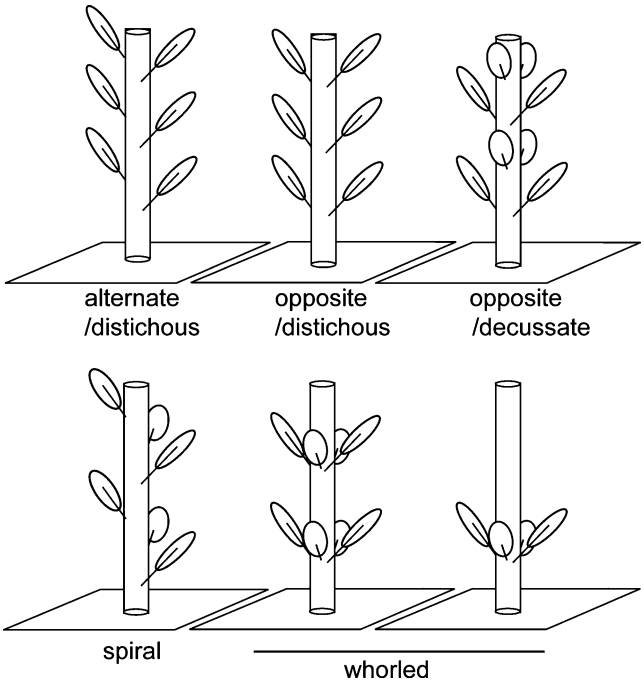

Fig. 1 Types of phyllotaxy

History of Phyllotaxy Studies, and Field Theory

In 1868, Hofmeister proposed a rule stating that new primordia develop periodically at the shoot apex boundary at the largest distance or space from the preceding primordia (Hofmeister 1868). This hypothesis is based on the idea that there is "communication" between primordia in the SAM. Snow and Snow investigated this "communication" using Lupin (*Lupinus albus*), which shows spiral phyllotaxy (Snow and Snow 1931). When the youngest visible leaf primordium (also known as Plastochron$_1$, or P_1) was surgically incised away from the apex, the next primordium to form (P_0 or incipient primordium, I_1) developed at its expected position. However, the subsequent primordium (known as P_{-1} or I_2), which normally forms close to P_1, shifted its position toward the isolated (P_1) primordium (Fig. 2). These results show that preexisting leaf primordia influenced the initiation sites of leaf primordia that are closest in space, not in time. Based on this experiment, Snow and Snow proposed the "first available space" model (Snow and Snow 1962). They saw space as a major limiting factor of primordium initiation.

However, Snow and Snow's results could also be explained by inhibitory field produced by the preexisting primordia. This possibility was tested by Wardlaw (1949) who performed similar surgical experiments with the fern *Dryopteris dilatata*. In this species, leaf primordia are widely spaced in a spiral phyllotaxy, ruling out the possibility of space limitation in primordium initiation. Surgical removal of I_1 caused a shift of the initiation site of I_4 (initiation site closest to I_1) toward the incision site, recapitulating Snow and Snow's observation. Because space is unlikely to be a limiting factor in *Dryopteris*, other factors appeared to be important in phyllotaxy regulation. In addition, Wardlaw observed that the shifted primordium (I_4) often grew faster than other normally positioned primordia. Together, these observations suggested an inhibitory effect of older primordia on younger primordia. This theory is called the "field theory" and was originally proposed by Schoute (1913). The field theory explains that the positioning of new primordia is regulated by chemical inhibitors released by older primordia to keep them from growing in their proximity. Thus, the removal of older primordia blocks the transport of these inhibitors, resulting in mispositioning of adjacent primordia. As mentioned above, this inhibitory field can also explain what Snow and Snow observed in the Lupin apex.

What is the nature of the inhibitor? Many data now suggest that the plant hormone auxin, which is involved in phyllotaxy regulation, is a good candidate. One of the features of auxin is its polar transport by PINFORMED (PIN) auxin efflux carriers. Pharmacological inhibition of polar auxin transport or mutations in the PIN1 auxin efflux carrier resulted in inhibition of lateral organ development and, hence, loss of phyllotaxy in plants including tomato,

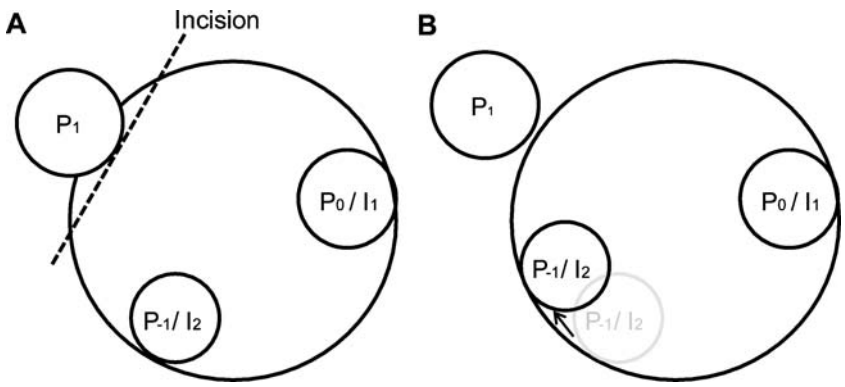

Fig. 2 Snow and Snow's microsurgical experiment. **A** Shoot apical meristems and leaf primordia (*top view*). **B** P_{-1} or I_2 position was shifted toward the incision site after incision around P_1

Arabidopsis, and maize (Okada et al 1991; Reinhardt et al. 2000; Scanlon 2003). When auxin was locally applied to pin-like shoot apices of *pin1* mutant, lateral organ (leaf) outgrowth was initiated at the application site (Reinhardt et al. 2000, 2003b) strongly suggesting the involvement of auxin in leaf initiation. If auxin is the instructive signal in leaf initiation and positioning, what is the inhibitory chemical produced by older primordia that inhibits the function of auxin in leaf initiation? After careful observation of PIN1 protein subcellular localization in the *Arabidopsis* SAM, Reinhardt et al. (2003a, b) concluded that instead of existing primordia being the source of an inhibitor, they function as a sink for the activator, auxin. Auxin distribution in the SAM can be investigated indirectly by PIN1 protein localization, as auxin flow will be determined by PIN1 subcellular localization and concentration (Petrasek et al. 2006; Wisniewska et al. 2006). In the *Arabidopsis* SAM, PIN1 is expressed mainly in the L1 layer of the SAM, and its subcellular localization suggests that auxin moves toward the future primordium. These subcellular patterns of PIN1 protein suggest that auxin maxima form at the site of primordium initiation. The local high concentration of auxin initiates primordium outgrowth at the peripheral region of the SAM. Once the primordium forms, the PIN1 expression domain expands inward in two to three cell files, suggesting downward auxin flow into the stem. In this way, the growing primordium functions as a sink to withdraw auxin from the region around the growing primordium. As a result, only distant regions will be able to accumulate enough auxin to initiate a new primordium. Similar patterns of PIN1 expression and localization are also observed in maize (Carraro et al. 2006; Lee et al. 2009), suggesting that similar mechanisms of phyllotaxy regulation by auxin and PIN1 likely function in diverse plant species.

The distribution of auxin in the SAM suggests a different interpretation of the field theory; it is not an inhibitor released from the preexisting primordium but depletion of an activator that creates an "inhibitory field" around a primordium. Using this theory, several computer simulations have been successfully developed (de Reuille et al. 2006; Jonsson et al. 2006; Smith et al. 2006), which may help predict the alterations of phyllotaxy in response to changes of auxin concentration, its movement, and other important factors.

Biophysical Theory for Phyllotaxy

Although the field theory is generally accepted, there is an alternative model to explain the generation and propagation of phyllotaxy. Biophysical theory describes how biophysical tensions and compression within the SAM might give rise to regular patterns of lateral organ initiation (Green 1985; Green et al. 1996). According to this theory, cells in the outer or L1 layer of the SAM mainly divide anticlinally, resulting in differences in biophysical forces between the L1 layer and the layers below it. This can result in bulges, which develop into lateral organs. Thus, this theory places cortical microtubules and cell wall cellulose microfibrils as the central players in phyllotaxy regulation. The biophysical model is supported by at least two experiments. Local application of exogenous expansin, a cell wall loosening enzyme onto the SAM, leads to primordium outgrowth at the application site (Fleming et al. 1997). In addition, expansin mRNA localization is associated with leaf primordium development in tomato (Reinhardt et al. 1998). These results suggest that cell wall loosening is the initial step for leaf initiation and is consistent with the observations that increased local cell division rate (Wyrzykowska et al. 2002), and disrupted cell division in the SAM (Wyrzykowska and Fleming 2003) did not alter leaf initiation. However, some experimental evidence argues against the biophysical model.

As this model is based on tension in the L1 layer and compression in layers below, it would be critical to have the whole L1 layer intact to enforce biophysical stress on the SAM surface. However, when the L1 layer of the tomato SAM was surgically removed, the effect on the leaf initiation program was localized only to the affected region (Reinhardt et al. 2003a). Furthermore, treatment of the SAM with the microtubule-depolymerizing drug oryzalin did not affect SAM patterning, including phyllotaxy (Hamant et al. 2008). This suggests that organ outgrowth at the SAM can be uncoupled from a microtubule-based mechanism.

Despite these data, cell wall structure appears to be crucial in leaf initiation and phyllotaxy. For example, the methylesterification status of cell wall pectins affects phyllotaxy in *Arabidopsis* (Peaucelle et al. 2008). Thus, an open question is how this cell-wall-based control and auxin-mediated regulation interplay in phyllotaxy patterning.

Involvement of Cytokinin in Phyllotaxy

Recent cloning of the maize *ABERRANT PHYLLOTAXY 1 (ABPH1* also known as *ABPHYL1*) gene suggests involvement of another plant hormone, cytokinin, in phyllotaxy regulation (Giulini et al. 2004). In fact, auxin and cytokinin crosstalk is involved in many plant developmental programs. For example, different ratios of auxin and cytokinin in tissue culture results in different developmental outcomes—high auxin/cytokinin causes root regeneration and low auxin/cytokinin leads to shoot regeneration. Thus, the isolation of *ABPH1* suggests that auxin and cytokinin signaling also interact in phyllotaxy, since *ABPH1* encodes

a cytokinin-inducible type A response regulator (Giulini et al. 2004). These genes are predicted to act in the cytokinin signal transduction pathway (Hwang and Sheen 2001). Mutations in *ABPH1* result in decussate maize phyllotaxy, which is extremely unusual in the grasses where phyllotaxy is usually alternate or distichous (Jackson and Hake 1999). It is unusual that a mutation in a single type A response regulator gene gives rise to a dramatic phenotype, since similar mutations in *Arabidopsis* do not have a phenotype (To et al. 2004). However, multiple *Arabidopsis* type A response regulator mutants do show some phyllotactic changes (Leibfried et al. 2005). It must be noted that there are some *Arabidopsis* mutants and lines that show sporadic phyllotaxy alterations. These include *bellringer* (Byrne et al. 2003), plants expressing miR164-resistant *CUP-SHAPED COTYLEDON2* (Peaucelle et al. 2007), *argonaute1-27* (Wang et al. 2008), and *stabilized1-1* (Lee et al. 2006, B.-h. Lee, unpublished observation). Many of these affect RNA processing including miRNA production, as well as SAM activity. However, maize *abph1* mutants are unique in that there are no such *Arabidopsis* mutants that show conversion from one regular phyllotactic pattern to another. Interestingly, *abph1* mutants have bigger SAMs than wild type. Thus, as many type A response regulators function as negative regulators of cytokinin signaling, *ABPH1* may negatively regulate cytokinin-induced growth of the SAM. One could say that the phyllotactic changes in *abph1* are a secondary effect of its enlarged SAM. However, *ABPH1* expression, which was all over the SAM in the embryos, becomes localized to the incipient primordium in the SAM during vegetative development, which suggests that *ABPH1* plays a direct role in determining the site of leaf initiation. Unexpectedly, *PIN1* gene expression in the *abph1* SAM is lower than in normal plants (Lee et al. 2009), suggesting that *ABPH1* is a positive regulator of *PIN1* expression. In *abph1* embryos, leaf initiation is delayed by about one day in comparison to wild type, and this may also be due to the reduced *PIN1* expression (Lee et al. 2009). Consistent with the effect of *ABPH1* on *PIN1* expression, their expression domains overlap in the SAM (Lee et al. 2009). These results suggest an interesting dual function of *ABPH1*-as a negative regulator in cytokinin signaling and as a positive regulator in auxin signaling, with both functions contributing to phyllotaxy regulation. It will be interesting to see if such interactions occur in *Arabidopsis*, as in maize.

Plastochron Regulation

Relatively few studies have been reported regarding plastochron regulation. Current results suggest that similar to the inhibitory field in phyllotaxy, preexisting primordia non-autonomously inhibit successive leaf initiation events in time. The rice mutants, *plastochron1* (*pla1*) and *plastochron2* (*pla2*) have a shortened plastochron (Itoh et al. 1998; Miyoshi et al. 2004; Kawakatsu et al. 2006). *PLA1* encodes a cytochrome P450, *CYP78A11*, which is expressed on the abaxial side of young leaf primordia, the bracts of the primary rachis branches, and peripheral cells of internodes in the stem and rachis but not in the SAM (Miyoshi et al. 2004). The expression of *PLA2*, encoding a MEI2-like RNA binding protein, is confined to leaf primordia (Kawakatsu et al. 2006). These results suggest that both *PLA1* and *PLA2* are negative regulators of leaf initiation and function at a distance to regulate SAM activity in determining the plastochron (Fig. 3).

Molecular players in plastochron regulation in *Arabidopsis* also include the *ALTERED MERISTEM PROGRAM1* (*AMP1*) gene, a cytochrome P450 gene (*CYP78A5*), miR156, and miR156's targets, the *SQUAMOSA PROMOTER BINDING PROTEIN-LIKE* (*SPL*) genes (Fig. 3). Down-regulation of *SPL9* expression by overexpressing miR156 reduces plastochron length, resulting in increased leaf numbers in *Arabidopsis* (Wang et al. 2008). Consistently, reduced miR156 levels cause increased *SPL9* RNA levels and a longer plastochron (Wang et al. 2008). *SPL9* is expressed in leaf initiation sites (anlagen) and developing leaf primordia, but not in the SAM. Restricted expression of miR156 only in leaf primordia also shortened the plastochron and increased leaf number. Therefore, noncell-autonomous regulation of leaf initiation by plastochron genes hold true in *Arabidopsis*, as in rice.

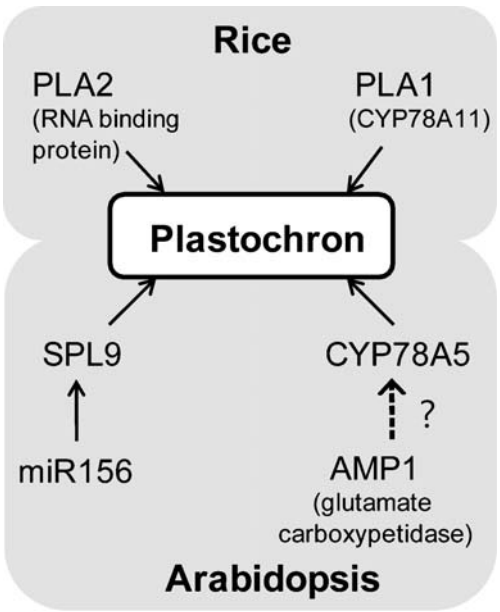

Fig. 3 Plastochron regulation. Genes involved in rice and *Arabidopsis* are shown. *Arrows* indicate hierarchical relationship, not positive or negative controls, between genes. PLA1 (CYP78A11) is a likely ortholog of *Arabidopsis* CYP78A5

Arabidopsis amp1 mutants develop more leaves, again due to a shortened plastochron (Chaudhury et al. 1993; Lee 2009). *AMP1* encodes a putative glutamate carboxypeptidase, and mutations in *AMP1* caused overexpression of *CYP78A5*, the likely ortholog of rice *PLA1*, which appears to be correlated with the increased SAM size (Helliwell et al. 2001). SAM size does not, however, seem to be the important factor that controls the leaf initiation rate. Rather, the rate of cell division appears to play a major role in plastochron regulation (Itoh et al. 1998; Cockcroft et al. 2000; Ikeda et al. 2005; Kawakatsu et al. 2006).

The *miR156-SPL* pathway and the *CYP78A5*-mediated pathway appear to act in parallel in plastochron regulation (Wang et al. 2008). Analysis of rice *pla1 pla2* double mutants also suggests that these genes act independently in rice plastochron regulation (Kawakatsu et al. 2006).

While rice *pla1* and *pla2* mutants only affect plastochron without altering phyllotaxy, many plastochron mutants, including *Arabidopsis amp1* and maize *terminal ear1* (*te1*; a possible ortholog of rice *PLA2*), also cause phyllotaxy alterations to some extent (Veit et al. 1998). Thus, it appears that phyllotaxy and plastochron may share some regulatory mechanisms. One possible link is cytokinin. Studies with maize *abph1* mutants suggest that cytokinin is involved in phyllotaxy regulation. Cytokinin might also be involved in plastochron regulation since *amp1* mutants have higher cytokinin levels (Chaudhury et al. 1993) and *CYTOKININ OXIDASE1* (*CKX1*) overexpressors, which have lower cytokinin levels, show a longer plastochron (Werner et al. 2001, 2003). The delay in initiation of the first leaf in *abph1* mutant could be due to altered cytokinin levels or low auxin levels (Lee et al. 2009). Thus, modulation of cytokinin as well as auxin distribution or absolute levels in the SAM could be responsible for crosstalk between regulatory mechanisms for phyllotaxy and plastochron.

Acknowledgement We thank Robyn Johnston and Joo-hyuk Park for valuable comments on this manuscript. This work was supported by Korea Research Foundation (C00251). S.-i.Y. and B.-h.L. are thankful for their funding by the Brain Korea 21 Fellowship from the Korean Ministry of Education, Science, and Technology and the Sogang University Research Grant of 2008 (200810022.01), respectively. Work in DJ lab on phyllotaxy is supported by the National Science Foundation grant, IOB-0642707.

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