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Phyllotaxis

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Summary

The precise arrangement of plant organs, also called phyllotaxis, has fascinated scientists from multiple disciplines. Whereas early work focused on morphological observations of phyllotaxis, recent findings have started to reveal the mechanisms behind this process, showing how molecular regulation and biochemical gradients interact with physical components to generate such precise patterns of growth. Here, I review new insights into the regulation of phyllotactic patterning and provide an overview of the various factors that can drive these robust growth patterns.

Key words: Phyllotaxis, Auxin, Cytoskeleton, Biomechanics, Meristem, Morphogenesis

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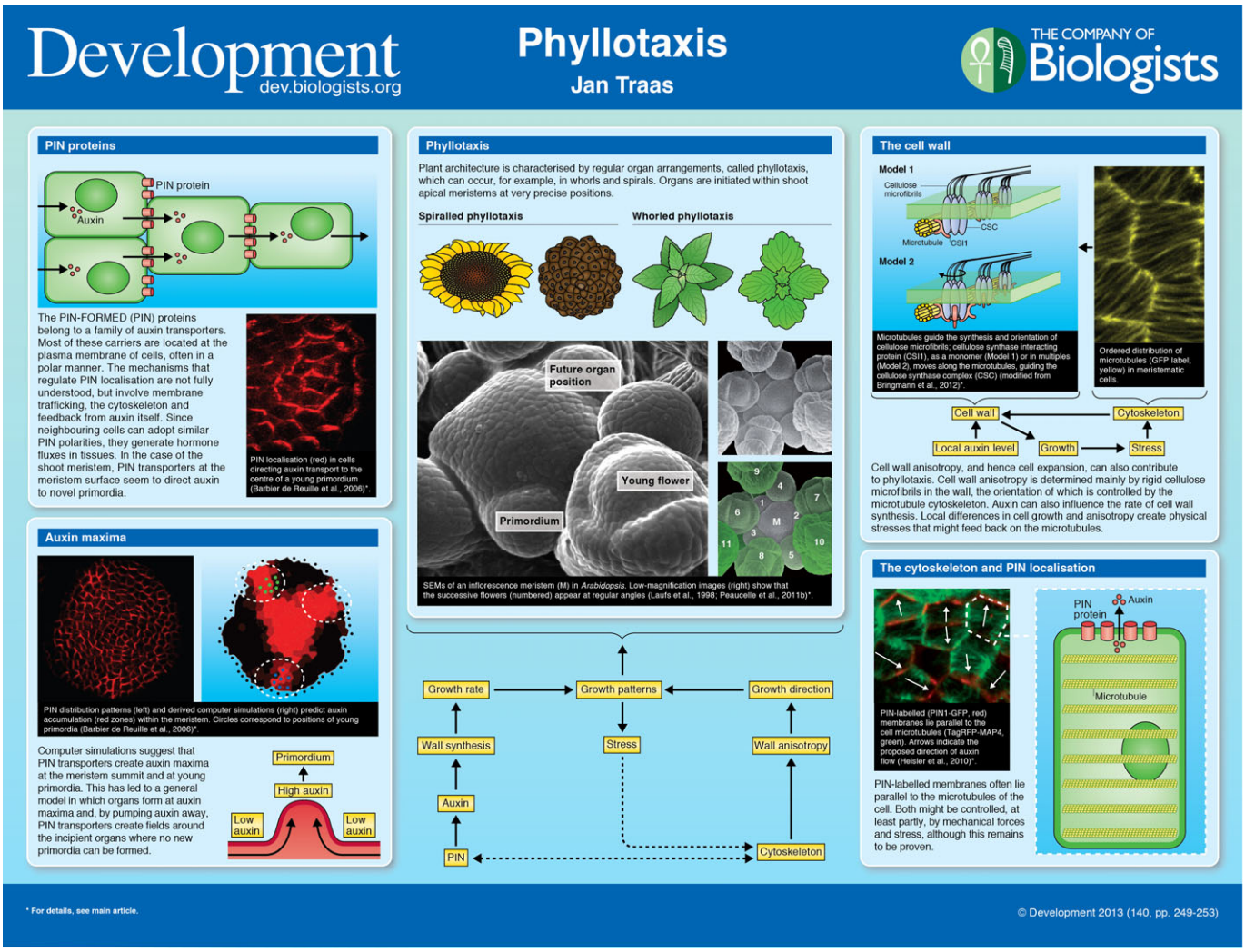
Introduction

Plant architecture is characterised by the regular spacing of lateral organs along stems and branches, an arrangement known as phyllotaxis (derived from the ancient Greek words *phýllon* meaning 'leaf' and *táxis* meaning 'arrangement'). Different types of phyllotaxis exist. In whorled phyllotaxis, for example, two or more organs are positioned at the same node. More complex organisations are found in phyllotactic patterns where organs are arranged in multiple clockwise and anticlockwise spirals. The type of phyllotaxis depends on the species. It can be constant throughout development, but this is not necessarily always the case; many dicotyledonous species start off with embryonic leaves that are positioned opposite to one another and then produce leaves and flowers in spiral arrangements and finally floral organs arranged in concentric whorls (reviewed by Steeves and Sussex, 1989).



(See poster insert)

Although growth patterns outside the shoot meristem can influence the relative position of organs (e.g. Peaucelle et al., 2007), phyllotaxis largely originates at shoot apical meristems. These are small populations of stem cells at the shoot tips that generate all the aerial parts of the plant. It is at the periphery of these meristems that organ primordia are initiated at very precise positions (see Laufs et al., 1998). Phyllotaxis has fascinated scientists since ancient times [for an excellent review on the history of phyllotaxis, see Adler et al. (Adler et al., 1997)]. Organ positions are often so regular that they can be described in very precise, mathematical terms. Therefore, phyllotaxis has been a multidisciplinary topic, studied by biologists, physicists, mathematicians and, more recently, computer scientists. This article and the accompanying poster provide a brief overview of our current understanding of phyllotaxis and underline the importance of chemical signals and biophysical processes in generating these very robust growth patterns. Relatively little is known about the molecular networks that play a role in this process and they will be mentioned only briefly.

Inhibitory fields: a widely accepted concept

Although phyllotaxis has been observed and studied since ancient times, more theoretical and mechanistic analyses of the processes that lead to these regular organ arrangements had to wait until the mid 19th century. In 1868, Hofmeister proposed a model in which new primordia appear periodically at the meristem periphery in the largest available space left by the preceding organs [for references see Adler et al. (Adler et al., 1997)]. Later, Snow and Snow initiated a set of elegant experimental studies of phyllotaxis. They and others showed that phyllotaxis can be altered by surgery or chemical treatments. This led them to propose (e.g. Snow and Snow, 1962) that the position of new leaf primordia is influenced by the pre-existing leaf primordia adjacent to the initiation sites, i.e. that each new leaf is inserted into the next available space at a minimum distance from the meristem tip [as reviewed and discussed by e.g. Snow and Snow (Snow and Snow, 1962)]. Earlier (1949), Wardlaw provided a more mechanistic interpretation (see Adler et al., 1997). Using surgical techniques on the fern *Dryopteris* he proposed an inhibitory effect of older primordia on young adjacent primordia. Subsequent analyses (e.g. Douady and Couder, 1996; see also Reinardt et al., 2003) supported both the ideas of Snow and Snow and the concept of an inhibitory field. As we will see, the findings of further molecular and cellular experimental analyses were also in line with the idea of an inhibitory field, which is now a widely accepted hypothesis.

The existence of an inhibitory field implies the presence of some type of interaction or signal that prevents the formation of a primordium next to an existing one. Below, recent findings are discussed concerning the nature of these interactions, which can be both chemical and physical.

Chemical signals: a central role for auxin

A major signal that is associated with phyllotaxis is the plant hormone auxin. The most abundant form, indole acetic acid (IAA), is actively transported from cell to cell. Since it is mostly present in its acid form in the cytoplasm, IAA is not able to diffuse freely across membranes and its transport throughout tissues is facilitated by auxin exporters localised at the cell membranes (reviewed by Grunewald and Friml, 2010). The pH in the extracellular space is more neutral and IAA can therefore more easily enter the cell. Nevertheless, even its import is facilitated by specific membrane proteins. In particular, the auxin exporters, which are transmembrane proteins of the PIN-FORMED or PIN family, show a polar localisation within individual cells. Since neighbouring cells often adopt similar PIN localisation, it has been assumed that these transporters create fluxes of auxin through the tissues, causing auxin maxima and minima to form. By contrast, auxin importers of the AUX family do not show a clear polar localisation at the shoot apical meristem and do not seem to play an important role in directing the fluxes there. Below, some of the experimental approaches are discussed that have helped to elucidate the role of auxin in phyllotaxis.

In planta analysis of auxin transport

That auxin efflux transport is important for phyllotaxis is clearly illustrated by the *pin1* mutant in *Arabidopsis*, in which transport at the meristem surface is impaired (e.g. Reinhardt et al., 2003). This mutant forms a naked inflorescence stem that is unable to generate flowers, thus demonstrating the importance of auxin and its transport in organ initiation, Interestingly, the *pin* phenotype can be rescued by simply adding high concentrations of hormone in a patch at the meristem periphery. This experiment led to the conclusions that high auxin concentrations are required for organ initiation and that auxin transport mediated by PIN1 is required to generate such local auxin maxima (Reinhardt et al., 2000). This conclusion was further supported by studies using several markers for auxin signalling, which indicated high auxin concentrations and signalling activity at the level of young organ primordia (e.g. Benková et al., 2003; Reinhardt et al., 2003; Barbier de Reuille et al., 2006; Heisler et al., 2005; Vernoux et al., 2011).

Whereas these experiments clearly established the importance of auxin and its transport in organ initiation, they did not provide information on the mechanism that positions the primordia. Therefore, the precise patterns of PIN1 and AUX transporters were analysed in detail. These studies revealed that both proteins are prominently present at the surface layer of the meristem. AUX1 is present on all membranes of the cells, whereas PIN1 shows complex patterns of polar localisation (e.g. Reinhardt et al., 2003). This strongly suggested that AUX proteins concentrate auxin at the meristem surface, whereas PIN1 proteins redistribute it there to create maxima and minima. However, it was not possible to deduce clear patterns of auxin fluxes at the meristem surface using this approach.

How are auxin fluxes coordinated at the meristem? Modelling auxin transport and phyllotaxis

More recent studies have used image analysis combined with modelling approaches to analyse the properties of the transport network suggested by PIN distributions (Barbier de Reuille et al., 2006). For this purpose, the images of PIN distribution were translated into ‘connection maps’ of the meristem surface. The properties of these maps were studied by injecting ‘virtual’ auxin into them. This simulation showed that primordia attract auxin fluxes from the surrounding cells, thus creating zones of influence from which auxin is attracted to the incipient organ. It was proposed that such ‘influence zones’ were equivalent to the inhibitory fields proposed by Wardlaw and others. These studies gave rise to the idea that, instead of producing an inhibitor, the primordium is rather removing a positive regulator, i.e. auxin (Reinhardt et al., 2003; Barbier de Reuille et al., 2006). An additional feature of the auxin transport network is the flux directed towards the meristem summit, suggesting an as yet undefined role of this area in auxin homeostasis at the meristem (Barbier de Reuille et al., 2006; Vernoux et al., 2011).

Whereas these studies provided a general picture of auxin transport-based organ initiation, it remained unclear how the cells coordinated their behaviour to generate patterned fluxes of hormone. Since auxin fluxes seemed to be directed towards auxin maxima, it was proposed that cells actually pump auxin against the auxin gradient ('against-the-gradient hypothesis'). To determine whether a simple scenario in which cells sense local auxin concentrations could generate phyllotactic patterns, Jönsson et al. (Jönsson et al., 2006) and Smith et al. (Smith et al., 2006) used models in the form of virtual tissues. These models consisted of a canvas of growing virtual cells that were able to 'sense' the auxin concentrations in their neighbours via an undetermined mechanism. Once these concentrations were determined, the cells transported some of their auxin to the cells with the highest auxin concentrations. This simple local behaviour was sufficient to generate different phyllotactic patterns. Some extra ad-hoc hypotheses were necessary to stabilise these patterns over time, but this might not be surprising, taking into account the very simple nature of the models. Nevertheless, these studies elegantly showed that a very simple stereotypic local behaviour is able to generate complex patterns at higher levels of organisation.

One might be tempted to consider the outcome of these models as mathematical proof for the against-the-gradient hypothesis. However, the models rather showed that this hypothesis is plausible at the meristem surface. This is not necessarily the case in other parts of the plant. In roots, for example, auxin seems to move away from a stable auxin maximum, suggesting a flow down the gradient (e.g. Grieneisen et al., 2007). Therefore, several other scenarios were tested as well. Stoma et al. (Stoma et al., 2008), for example, investigated the so-called canalisation or flux-based hypothesis, which proposes that cells sense and amplify auxin fluxes passing through their membranes rather than sensing local auxin concentrations. Such a mechanism also has the potential to generate patterns, as was shown by Sachs (Sachs, 1969) for the patterning of veins in leaves and stems. Stoma et al. (Stoma et al., 2008) again used a virtual tissue to show that such a mechanism was indeed able to generate phyllotactic patterns. In their model, the cells sensed and amplified outgoing fluxes passing through their membranes. Models in which this amplification was relatively weak were able to produce diffuse auxin fluxes in the virtual tissue and patterned auxin maxima. Interestingly, realistic distributions of PIN transporter were obtained. Stronger amplifications led to canalised fluxes of auxin, reminiscent of the veination patterns in leaves. The canalisation-based model was also able to generate stable maxima, with auxin flowing in and out, as observed in the root meristem. Since, in contrast to the up-the-gradient model, it is able to explain the dynamic behaviour of auxin fluxes throughout the plant, canalisation provides a unifying concept for the control of auxin distribution.

It should be noted, however, that other concepts have been proposed as well, such as the hybrid model, in which cells, depending on the auxin concentration, can switch from one mechanism to the other (Bayer et al., 2009). Importantly, both the up-the-gradient and canalisation models are very abstract notions, as the cellular processes that lead to polarised PIN localisations remain poorly understood. PINs are membrane-associated proteins and there is strong evidence that the membrane trafficking machinery plays a central role in directing the transporters to specific membranes (Grunewald and Friml, 2010). In this context, auxin itself, by interfering with membrane properties, might feed back on its own transport. Finally, cytoskeletal proteins have also been implicated (Heisler et al., 2010).

From chemistry to physics

Above, we have seen how self-organising mechanisms could lead to the formation of local auxin maxima. There is strong evidence that these maxima provide positional information for organ outgrowth, but how are the auxin concentrations translated into morphogenetic responses? Obviously, the local hormone concentrations first interact with the perception and downstream signalling pathways. For auxin, this comprises a relatively complex network of receptors and transcriptional regulators. These regulators somehow control local growth rates, but how is this achieved? Recent studies, as discussed below, suggest that auxin might influence the physical and mechanical properties of cells, thus facilitating the controlled growth that can generate phyllotactic patterns.

Physical properties of plant cells

A growing biological system is not only a geometrical structure in which chemical gradients and molecular networks control growth rates and directions, but also a physical structure governed by mechanical cues. Therefore, if we want to understand how organs are initiated, we need to take into account the physical properties of the system. How do cells physically grow? Plant cells are under high internal turgor pressure and it is only the presence of a rigid extracellular wall that prevents them from bursting (reviewed by Keegstra, 2010). This cell wall is composed of a dense network of cellulose microfibrils that are crosslinked to each other by a network of polysaccharides. Cell expansion can only take place as long as the cells are under pressure, which has led to the concept of turgor pressure-driven cell growth. There is strong evidence that the irreversible yielding of the cell wall to this pressure causes cell growth (for reviews, see Kutchera, 1991; Hamant and Traas, 2010). This plastic deformation of the wall includes modifications both in cell wall elasticity and in cell wall synthesis. In other words, an increase in cell size can in principle be described in terms of turgor, elastic extensibility, followed by cell wall synthesis. The direction of this cell expansion depends on the anisotropic properties of the cell wall, which are mainly determined by the orientation of the rigid cellulose microfibrils. This orientation depends on the microtubule cytoskeleton (e.g. Hamant et al., 2008; Bringmann et al., 2012), but how this is precisely regulated is not known.

Biomechanical control of phyllotaxis

It is clear that to control the outgrowth of a primordium, the underlying molecular networks must interfere locally with the physical properties of the cell wall as well as with its anisotropic properties. Auxin can participate in this process in different ways (reviewed by Hamant and Traas, 2010). First, it could induce the expression of several cell wall remodelling proteins via transcriptional regulation. Peaucelle et al. (Peaucelle et al., 2011a), for example, presented evidence that early organ outgrowth involves pectin-modifying enzymes that reduce wall stiffness. Furthermore, auxin could act in a more direct manner by stimulating the secretion of protons into the cell wall, leading to a decrease in apoplastic pH, which in turn would lead to wall loosening. The situation is more complex, however, as not only auxin but also other signalling molecules could play a role and directly or indirectly control wall structure.

Overall, the available data reveal a relatively straightforward series of events in which cells can generate patterned auxin maxima by pumping the hormone against gradients or along fluxes. These accumulations of hormone could lead to local cell wall loosening and increased growth rates. In reality, however, the situation is more complex. In multicellular tissues, this scenario is

further complicated because we are dealing with different cell types with different cell wall properties and that are firmly linked to each other. Therefore, local differences in growth lead to tensions within tissues. Recent findings suggest that the resulting patterns of forces might feed back on growth via the cytoskeleton. For example, Hamant et al. (Hamant et al., 2008) found strong indications that microtubules orient along stress fields. Since microtubules, in turn, orient the cellulose microfibrils, this stress-based feedback could cause the cells to resist the forces exerted on them (Hamant et al., 2008; Uyttewaal et al., 2012). As such, instead of releasing the tension in tissues, cells would tend to amplify them, causing tissues to fold and buckle while they grow.

Furthermore, Heisler et al. (Heisler et al., 2010) found that this feedback mechanism could also operate in the localisation of the PIN auxin transporter. Indeed, there is a significant correlation between PIN localisation and microtubule orientation, as the transporter tends to be localised on membranes that lie parallel to the microtubules and thus along predicted stress patterns. Thus, by causing differential growth patterns and stresses in tissues, auxin would feed back on its own flux. Computer simulations have shown that such a mechanism would be able to generate the phyllotactic patterns observed *in vivo* (Heisler et al., 2010; Jönsson et al., 2012). The mechanism that leads to these precise organisations of the cytoskeleton, however, remains unknown. Recent studies in *Arabidopsis* suggest that microtubule dynamics play an important role, as organ initiation and separation are perturbed if *KATANIN*, a gene involved in tubulin severing and polymerisation, is knocked out (Uyttewaal et al., 2012).

Conclusions and perspectives

In summary, a scenario emerges in which a self-organising auxin transport system leads to precise patterns of hormone distribution. By influencing local cell wall properties, these distributions are then translated into growth rates and directions. There is evidence that these growth patterns generate tensions within the tissue that, in turn, feed back via the cytoskeleton on microtubule orientation and auxin transport. There are, however, many important perspectives and open questions that remain to be addressed in the field.

First, little is known about the downstream events that are elicited once auxin has accumulated. In fact, very little is known about the molecular network involved in phyllotaxis. Recent studies have started to unravel the transcriptional determinants that play a role in organ formation (e.g. Vernoux et al., 2011; Prasad et al., 2011), but substantial efforts are still required. Another question concerns the precise nature of the inhibitory fields that surround the organs. Although there is little doubt that they exist and that auxin is an important player, it is not the only one, and the inhibitory fields could be formed by multiple components. Additional chemical factors could be involved, but, as we have seen, mechanical forces are also likely to play a role.

Although the correlation between microtubules, PIN localisation and predicted stress patterns is strong, the precise mechanisms involved in coordinating these factors are largely unknown. How do the cells sense directional stress? What are the receptors involved? How important are stresses in guiding patterning events? What are the precise mechanical properties of the meristem? Answering these questions will require the development of novel approaches – for instance to probe the biomechanical properties of the cells. The first studies to address these issues have revealed that different parts of the meristem do indeed seem to have different mechanical characteristics (Milani et al., 2011; see also Peaucelle et al., 2011b; Kierzkowski et al., 2012), but we are only at the beginning of these analyses.

In addition, studying patterned growth as an output of genetic regulation requires a detailed and quantitative knowledge of the way tissues grow. This, in turn, requires novel techniques to follow growth with cellular resolution. As we have seen, modelling is becoming an important tool in developmental biology. The first models in the form of virtual tissues have been developed (Smith et al., 2006; Jönsson et al., 2006; Hamant et al., 2008), allowing hypotheses regarding both chemical and physical processes to be put forward. These modelling tools will now have to be made more sophisticated in order that processes such as phyllotaxis can be analysed at multiple levels.

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Competing interests statement

The authors declare no competing financial interests.

Development at a Glance

A high-resolution version of the poster is available for downloading in the online version of this article at http://dev.biologists.org/content/140/2/249.full

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