Regulation of phyllotaxis by polar auxin transport

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The regular arrangement of leaves around a plant's stem, called phyllotaxis, has for centuries attracted the attention of philosophers, mathematicians and natural scientists; however, to date, studies of phyllotaxis have been largely theoretical. Leaves and flowers are formed from the shoot apical meristem, triggered by the plant hormone auxin. Auxin is transported through plant tissues by specific cellular influx and efflux carrier proteins. Here we show that proteins involved in auxin transport regulate phyllotaxis. Our data indicate that auxin is transported upwards into the meristem through the epidermis and the outermost meristem cell layer. Existing leaf primordia act as sinks, redistributing auxin and creating its heterogeneous distribution in the meristem. Auxin accumulation occurs only at certain minimal distances from existing primordia, defining the position of future primordia. This model for phyllotaxis accounts for its reiterative nature, as well as its regularity and stability.

A major determinant of plant architecture is the arrangement of leaves and flowers around the stem, known as phyllotaxis[1]. Leaves and flowers are formed by the shoot apical meristem in characteristic patterns such as alternate (distichous), opposite (decussate), or—most frequently—spiral[1,2]. Spiral phyllotaxis has received much attention from theoreticians because the divergence angle between successive leaves approaches the golden ratio of 137.5°, and the spiral arrangements are characterized by the Fibonacci numbers[1,3,4]. Models of phyllotaxis explain the regular spacing of leaf primordia with mechanisms based on "the first available space"[5], "biophysical forces" within the meristem[6], or "inhibitors of organogenesis" released from young leaf primordia[3,7]. Although mathematical modelling of phyllotactic mechanisms based on biophysical or biochemical regulation can recreate phyllotactic patterns[3,6,8], experimental support for such phyllotactic mechanisms is lacking.

Auxin has been implicated in various aspects of plant development, including embryogenesis[9–13], root development[14,15], and vascular differentiation[16]. Recently, auxin has been shown to induce leaf and flower formation at the shoot apical meristem (SAM)[17]. As a trigger of organ initiation, auxin could play a role in determining organ position and thereby phyllotaxis[18].

An instructive role of a signal molecule implies regulated distribution in the tissue. This could be achieved by defined sources of the signal, by regulated degradation, or by controlled transport of the signal to certain sites in the tissue. Auxin has its own polar transport route consisting of specific influx and efflux carriers in the plasma membrane[19]. Net auxin flux results from coordinated polar localization of the auxin efflux carrier in the auxin-conducting cells[20]. This routing of auxin is responsible for basipetal auxin transport from the young leaves towards the root. In the root tip, active polar transport generates an auxin maximum in the columella initials, which regulates meristem development and cell differentiation[14,15]. Furthermore, auxin efflux carriers mediate gravitropism by creating asymmetric auxin distribution, thereby causing anisotropic growth[21].

We have previously proposed that active auxin transport in the shoot apex could generate patterned auxin distribution, thereby determining the pattern of organ formation. To allow valid predictions about the transport and distribution of auxin within the shoot apex, we studied the expression pattern and subcellular localization of the putative *Arabidopsis* influx and efflux carriers, AUXIN RESISTANT1 (AUX1)[22] and PIN-FORMED1 (PIN1)[23]. In addition, we performed micro-application of auxin to *Arabidopsis* meristems to address the role of auxin in phyllotaxis.

PIN1 expression and localization in *Arabidopsis* apices

In vegetative apices, PIN1 protein was detected in the epidermis and the vasculature of developing leaf primordia, as well as in the meristem L_1 layer and, at lower levels, in L_2 (Fig. 1a). A marked induction at the meristem flank (Fig. 1a) corresponded with the site of incipient and young leaf primordia (Fig. 1b). PIN1 signal in the L_1 and L_2 layers was consistently localized to the apical side of the cells, that is, pointing to the meristem summit (Fig. 1c, arrowheads), except for the cells close to the site of incipient leaf initiation (I_1). In these cells, the signal pointed to I_1 (Fig. 1c, arrows). At the summit itself, the signal was more diffuse than at the flank (Fig. 1c). In the epidermis of young leaf primordia, PIN1 signal was restricted to the apical side of the cells (Fig. 1d, e), as in cotyledon primordia[24]. At the site of incipient organ formation, PIN1 protein in the inner cell layers was first localized with no obvious direction of polarization (Fig. 1f), but at the time of primordium outgrowth, PIN1 was detected at the side of the cells that points to the centre of the leaf primordia (Fig. 1g). This localization suggests accumulation of auxin in the centre of the young primordia.

During primordium outgrowth, PIN1 became restricted to a narrow file of cells that comprised 2–3 cells in width, corresponding presumably to the provascular strand, and the PIN1 signal in these cells became gradually localized to the basal end of the cells (Fig. 1h). The gradual restriction to narrow cell files and the subcellular polarization of PIN1, first towards the centre and later towards the base of the leaf primordia, is suggestive of a canalization mechanism[25]. The canalization theory proposes that small local differences in auxin concentration are amplified by a self-reinforcing accumulation mechanism, resulting in local auxin elevation and auxin depletion in the surrounding tissue. As a

consequence, the cells that experience high auxin levels are restricted to narrow stripes that are determined to become auxin-conducting cell files[25].

In inflorescence apices, PIN1 expression followed a similar phyllotactic pattern as in vegetative meristems (Fig. 1i, compare with Fig. 1b). Notably, PIN1 induction was already clearly visible in incipient flower primordia at the I_3 stage, at least one plastochron earlier than the induction of the organ marker gene *LEAFY* (*LFY*)[26] (Fig. 1j). PIN1 was detected in the vasculature of the inflorescence stem as well as in young flower primordia (Fig. 1k). In the inflorescence meristem, the PIN1 signal was strongest in the L_1 layer, and lower in the interior layers with upregulation at the site of incipient organ formation (arrowhead in Fig. 1k). In young flower primordia, PIN1 expression was elevated at the flank, at the site of sepal induction (Fig. 1l), and later where the inner whorl organs were formed (Fig. 1m).

Organ formation in mutants with auxin-related defects

The similar expression patterns of PIN1 in vegetative and floral apices suggest a comparable function of PIN1 and auxin in the induction of leaves and flowers. However, *pin1* mutants can form leaves, although their size, shape, and position are aberrant[27,28]. To address the role of auxin in leaf formation, we treated young *pin1* plants with indole-3-acetic acid (IAA), resulting in the formation of normal leaves (Fig. 2a). In addition, we crossed *pin1* mutants with *lfy* mutants, which form leaves and axillary inflorescences instead of flowers[26]. Treatment of pin-shaped apices of *pin1;lfy* double mutants with IAA resulted in the formation of leaves instead of flowers (Fig. 2b). These leaves had abundant trichomes on their abaxial side (Fig. 2b), and formed pin-shaped lateral branches in their axils (not shown), indicating that they corresponded to cauline leaves. Hence, IAA can trigger the initiation of rosette leaves, cauline leaves, and flowers[17].
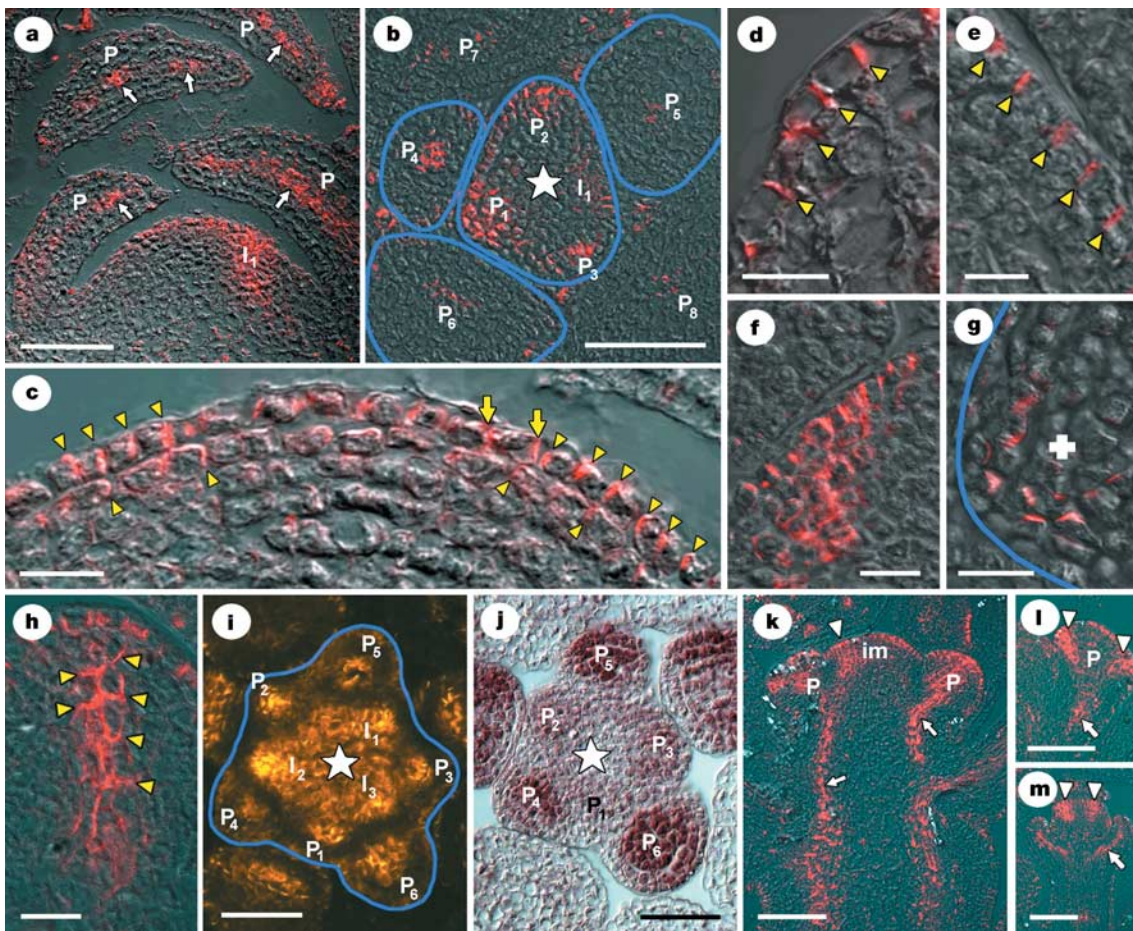

Figure 1 Localization of the PIN1 protein in *Arabidopsis* apices. **a**, Median longitudinal section through a vegetative apex. Note PIN1 signal (red) in the vasculature of leaf primordia and at the site of incipient organ formation (I_1). **b**, Cross-section of an apex as in **a**. The outline of primordia (blue) is highlighted for clarity. Note PIN1 expression in young primordia (P_1, P_2 and P_3) and the developing central vein of older primordia (P_4–P_8). **c**, Near-median longitudinal section of the same meristem as depicted in **a**. Yellow arrows indicate discontinuity of PIN1 localization at I_1. **d**, Longitudinal section through a young leaf primordium showing PIN1 signal on the distal end of cells in the abaxial epidermis. **e**, Tangential longitudinal section through a vegetative apex showing PIN1 signal in the lateral margin of a young leaf primordium. Leaf tip at the top of the image. **f**, Close-up at I_1 of a section as in **a**. **g**, Close-up of the same section as depicted in **b**, corresponding to the P_1 position, imaged with reduced pinhole size. The centre of the leaf primordium is denoted by a cross. **h**, Tangential longitudinal section of a leaf primordium showing localization of PIN1 in the central provascular strand. **i**, Cross-section through an inflorescence apex with PIN1 signal in the centres of flower primordia (P_1–P_8), and at the site of incipient flower formation (I_1–I_3). **j**, Section as in **i** hybridized with an antisense probe against *LEAFY* mRNA. **k**, Longitudinal section through an inflorescence apex. Note PIN1 signal in the vasculature, in the L_1 layer of inflorescence and flower meristems, and at the site of incipient organ formation (arrowhead). **l**, Longitudinal section through an early flower primordium. Signals coincide with the position of incipient sepals (arowheads) and the developing vascular strand. **m**, Longitudinal section through a later flower primordium. Signal coincides with the position of incipient anthers (arrowheads) and with the central vascular strand of sepals. P_1–P_8, leaf primordia in **b** and flower primordia in **i** and **j**, with P_1 being the youngest; I_1–I_3, sites of incipient organ formation. im, inflorescence meristem; P, leaf primordia in **a** and flower primordia in **k** and **l**. White arrows indicate the vasculature, yellow arrowheads indicate polar PIN1 signal. Stars denote the meristem centre. Scale bars, 50 μm (**a**, **b**, **i**–**m**); 10 μm (**c**–**h**).

The *monopteros* (*mp*) and *pinoid* (*pid*) mutants display a defect in organ formation similar to that in *pin1*, that is, they fail to induce flowers but are normal with respect to stem growth and meristem maintenance[28,29]. Hence, *PIN1*, *MP* and *PID* may function in the same pathway in flower formation. *PID* encodes a protein kinase that has been implicated both in negative regulation of auxin response and in positive regulation of polar auxin transport[30,31]. *MP* encodes an ARF-type auxin response transcription factor[11]. While these mutants are similar with respect to their inflorescence phenotype, they can be distinguished by their response to auxin microapplication. *mp* mutant meristems were unresponsive to

1 mM IAA (Fig. 2c), or to 10 mM IAA in the lanolin paste (*n* = 25, data not shown), suggesting a function of MP in the response to auxin. However, the stems of *mp* plants still responded with anisotropic growth to lateral IAA application (Fig. 2d), showing that *mp* plants are not generally auxin-insensitive. *pid* mutant apices, like those of *pin1* mutants, responded to exogenous IAA with the formation of flower primordia (Fig. 2e). Hence, the defect in *pid* is likely to be upstream of auxin action. Apices of *pin1;pid* double mutants resembled *pin1* and responded to IAA with organ formation (Fig. 2f), although at lower frequency than the *pin1* and *pid* single mutants.

Expression of PIN1 in *mp*, *pid* and *pin1*

The mutant phenotypes of *pin1*, *pid* and *mp* show that auxin transport and perception are critical for flower initiation. To see whether the patterning of the inflorescence meristem was also affected in these mutants we analysed PIN1 expression, which exhibits a phyllotactic pattern in wild-type plants[32] (Fig. 1b, 1i).

In *mp* mutants, PIN1 was expressed in the L_1 layer, and was localized in a polar fashion (Fig. 2g, h). However, PIN1 was uniformly expressed at the flank of the meristem and lacked a phyllotactic pattern. In the *pid* mutant, PIN1 protein did not accumulate to normal levels, and did not show a phyllotactic pattern (Fig. 2i). PIN1 protein could not be detected in *pin1* mutants;
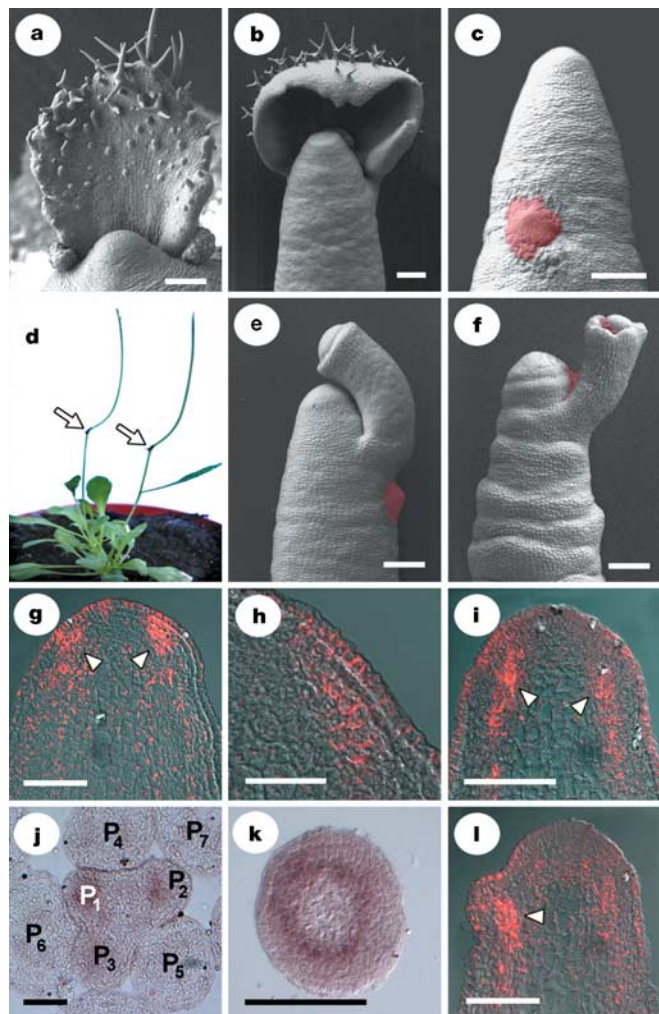

Figure 2 Auxin-induced leaf and flower formation and PIN1 expression in mutants of *Arabidopsis*. **a**, Leaf formation triggered on a vegetative *pin1* meristem by exogenous IAA (66% induction, *n* = 24). **b**, Leaf formation on a *pin1;lfy* double mutant treated with exogenous IAA (20% leaves, *n* = 64 versus 0% leaves in *pin1* single mutant). **c**, Application of IAA to *mp* meristems did not induce organ formation (*n* = 45). **d**, Auxin response (arrows) in the stem of *mp* plants (80% bending, *n* = 25). **e**, Organ formation on *pid* induced with IAA (92% induction, *n* = 24). **f**, Organ formation on *pin;pid* induced with IAA (70% induction, *n* = 50). **g**, Longitudinal section through a *mp* mutant apex, showing PIN1 signal on both sides of the meristem flank (arrowheads), and in the L_1 layer. **h**, Section as in **g** showing polar localization of PIN1 in the L_1 layer. **i**, Longitudinal section through a *pid* mutant apex, showing PIN1 signal at the flank, but weaker and in cells more basal than in *mp* (compare with g). **j**, Cross-section of a wild-type inflorescence apex showing *PIN1* mRNA accumulation following the phyllotactic pattern. **k**, Cross-section of a *pin1* apex with *PIN1* mRNA accumulation in the entire periphery. **l**, Longitudinal section of a *pid* apex with PIN1 induction in a flower primordium triggered by exogenous IAA. White arrowheads indicate PIN1 signal. Lanolin paste in SEM images was pseudo-coloured red in **c**, **e** and **f** for clarity. Scale bars, 100 µm (**a–f**); 50 µm (**g**, **i–l**); 25 µm (**h**).
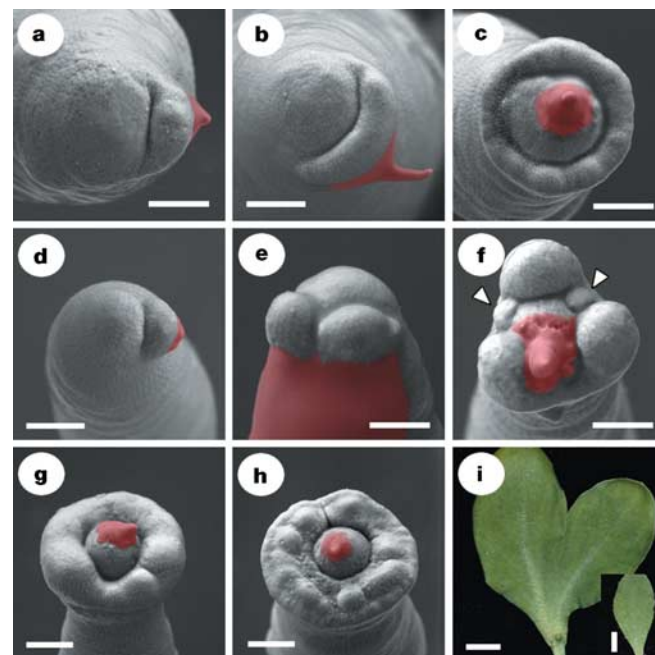

Figure 3 The role of PIN1 in organ separation and delimitation. **a**, Small flower primordium induced by small droplet of exogenous IAA at the flank of a *pin1* meristem (90% induction; *n* = 20). **b**, Wide flower primordium induced by larger droplet of exogenous IAA at the flank of a *pin1* meristem (93% induction; *n* = 27). **c**, Ring-shaped flower primordium induced by IAA application to the meristem centre of a *pin1* meristem (60% complete ring, 40% incomplete ring; *n* = 20). **d**, Small flower primordium induced by a small droplet of exogenous IAA at the flank of a *pid* meristem (88% induction; *n* = 25). **e**, Pair of separated small flower primordia induced by large amount of exogenous IAA at the flank of a *pid* meristem (63% pairs, 21% single organ; *n* = 24). **f**, Multiple small flower primordia induced by IAA application on the meristem centre of a *pid* meristem. Note the formation of a second whorl of organs (arrowheads) in the gaps between primordia of the previous whorl (95% induction; *n* = 20). **g**, Treatment of *pid* as in **f** but with 2,4-D instead of IAA resulted in the formation of ring-shaped flower primordia (50% ring; 6% incomplete ring; *n* = 30). **h**, Treatment of a *pin1;pid* double mutant on the meristem centre resulted in the formation of a ring (50% ring; 20% incomplete ring; *n* = 20). **i**, Fused leaf of a *pin1* mutant, compared to a leaf of *pid* (inset). Scale bars, 100 µm (**a–h**); 1 mm (**i**).

 257

however, the messenger RNA still accumulated, allowing us to determine the expression pattern of *PIN1* in *pin1* mutants by *in situ* hybridization. In wild-type apices, *PIN1* mRNA was expressed in a phyllotactic pattern similar to the PIN1 protein (Fig. 2j), but, in *pin1* mutants, *PIN1* mRNA was expressed throughout the periphery of the meristem (Fig. 2k). This shows that *PIN1* expression not only responds to phyllotactic information, but that functional PIN1 protein is necessary to create such patterns. Treatment of *pid* mutants with IAA resulted in the induction of PIN1 in the emerging organs (Fig. 2l), as in wild-type organ formation (Fig. 1k). Thus, PIN1 expression responds to auxin, directly or indirectly.

The uniform expression patterns of PIN1 at the periphery in *pin1*, *mp* and *pid* are reminiscent of the ring-shaped expression domains of organ and boundary marker genes in the periphery of *pin1* meristems[32]. Similarly, in the *pid* mutant, the organ marker LFY as well as the boundary marker CUC2 were expressed around the entire periphery (data not shown). Taken together, these results show that PIN1, and auxin, play a central role in phyllotactic patterning. On the other hand, PIN1 responds to phyllotactic patterning information, indicating that phyllotaxis involves a feedback mechanism.

Role of *PIN1* in organ separation and delimitation

During embryogenesis, auxin efflux carrier activity is critical to establish bilateral symmetry and cotyledon separation[9,10]. To test the role of PIN1 in organ separation and delimitation in the shoot meristem, we applied lanolin droplets of different sizes containing IAA to the flank of the meristem of *pin1* and *pid* mutants. In *pin1* mutants, the amount of applied IAA determined the size of the induced organs (Fig. 3a, b). When IAA was applied to the tip of the meristem, a ring-shaped organ was induced, indicating that, in the absence of PIN1 function, IAA could diffuse through the meristem and that the entire periphery of the *pin1* meristem was capable of organ formation (Fig. 3c). In contrast, *pid* mutant apices formed organs of a defined size, and application of larger amounts of IAA increased the number of organs instead of their size (Fig. 3d, e). Application of auxin to the tip induced a whorl of separated organs instead of a ring (Fig. 3f). Interestingly, such whorls were often symmetrically organized, and sometimes, a second whorl of organs was initiated, with the individual organs always being formed between the organs of the previous whorl (Fig. 3f, arrowheads).

It could be argued that auxin placed on the tip of *pid* mutants merely revealed a prepattern present before the treatment. To test this possibility, we treated *pid* apices with 2,4-D, a synthetic auxin analogue which is a poor substrate for the efflux carrier[33] but which can induce organ formation. 2,4-D treatments on the top of the meristem of *pid* mutants induced ring-shaped primordia (Fig. 3g), similar to those formed on IAA-treated *pin1* mutants (Fig. 3c). Thus, no prepattern had been present at the time of the treatment, which is in agreement with the lack of a visible phyllotactic expression pattern of PIN1 in *pid* meristems (Fig. 2i). It follows that patterns like those seen in Fig. 3e and f must have been generated after auxin application. We hypothesize that PIN1-dependent patterning was triggered by the exogenous auxin and resulted in the generation of separate auxin peaks from an initially even auxin distribution around the periphery of the meristem. According to this scenario, auxin plays an instructive role in the patterning of the apex, and is not just a permissive factor in primordium outgrowth. IAA treatment of *pin1;pid* double mutant meristems on the meristem centre induced the formation of rings similar to those in *pin1* single mutants (Fig. 3h). Thus, *pin1* is epistatic to *pid*, indicating that the patterning induced by IAA in *pid* mutants requires the function of *PIN1*. The defect of *pin1* mutants in flower primordium separation correlates well with defects in delimitation and separation of leaves and cotyledons[17,23,27,28] (Fig. 3i).

Role of AUX1 in phyllotaxis

Our data demonstrate a prominent role of the putative efflux carrier PIN1 in the phyllotaxis of *Arabidopsis*. However, in tomato, pharmacological studies had revealed an additional role for auxin influx carriers in the lateral delimitation of leaves[34]. Mutations in the putative auxin influx carrier *AUX1* of *Arabidopsis* did not visibly affect leaf formation (data not shown), suggesting that either influx carrier function is not limiting in *Arabidopsis* meristems, or that redundancy of function with the three related *Like-AUX1* genes[35] (*LAX* genes) hides the loss-of-function phenotype of *aux1* mutants. To explore these possibilities, we crossed *aux1* into the *pin1* background, reasoning that in the absence of the efflux carrier, the role of AUX1 could become limiting. When the apices of such double mutants were treated with IAA at the flank, organs were formed that were much wider than in *pin1* single mutants (Fig. 4a, compare with Fig. 3a). Thus, auxin influx carrier activity contributes to organ delimitation, but in the presence of active PIN1, the contribution of AUX1 is masked.

To determine the expression pattern and subcellular localization of AUX1, we performed immunofluorescent detection of a haemagglutinin-tagged version of AUX1 (referred to as HA-AUX1)[36]. In vegetative apices, HA-AUX1 was detected in the abaxial epidermis of all leaf primordia, and in the meristem L_1 layer (Fig. 4b), where the signal was stronger at the flank compared to the meristem centre (Fig. 4c). On the basis of this expression pattern, we think the function of AUX1 could be to promote intracellular auxin
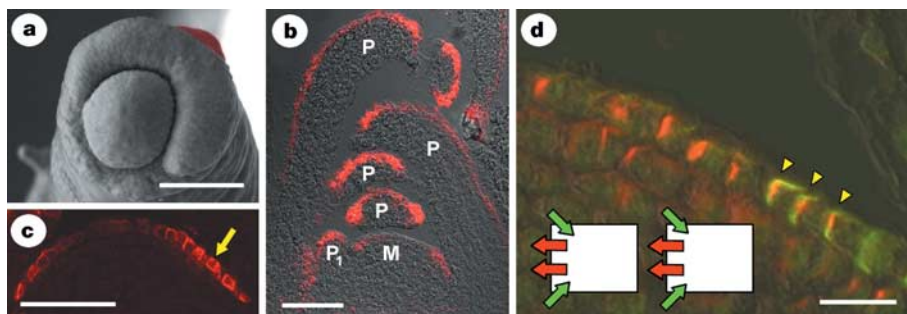

Figure 4 Localization and function of AUX1 in the meristem. **a**, In the *pin1;aux1* double mutant, small droplets of auxin induced the formation of oversized flower primordia (compare to Fig. 3a) (46% ring; 11% incomplete ring; 17% local organ; $n = 120$). **b**, Expression of HA-tagged AUX1 in a 6-week-old vegetative meristem. Signal was strongest in the abaxial epidermis of P_1 and older leaf primordia (P). **c**, At 6 weeks, AUX1 was expressed in the L_1 cells of the meristem with the strongest expression at the periphery. Localization was to the plasmalemma with hints of subcellular polarity (arrow). **d**, Double labelling on a median section of a 4-week-old plant with antibodies against HA-AUX1 (green) and PIN1 (red). Localization of AUX1 and PIN1 partially overlapped (arrowheads). Inset, possible net auxin fluxes inferred from the localization and relative levels of AUX1 and PIN1. M, meristem; P, primordia. Scale bars, 50 µm (**a**–**c**); 10 µm (**d**).

accumulation in the L_1 layer and abaxial epidermis.

In 6-week-old plants, AUX1 signal was clearly localized to the plasmalemma, and showed only hints of subcellular polarity (Fig. 4c). During early vegetative development, subcellular localization of HA-AUX1 signal was more evident, and frequently coincided on the same side of the cells as PIN1 signal, that is, on the distal side of the cells (Fig. 4d). However, while the PIN1 signal was restricted to the anticlinal membrane towards the meristem summit, AUX1 signal was also observed at the edges of the cells and in the outer and inner periclinal membrane of the L_1 cells (Fig. 4d).

The coexpression of AUX1 and PIN1 in the L_1 layer could point to a common function in auxin trafficking in these cells. We propose that AUX1 restricts auxin to the outer cell layer (by counteracting loss of auxin via diffusion to inner layers), and that PIN1 promotes its directional transport towards the tip of the meristem and of leaf primordia. After extrusion of auxin by PIN1, AUX1 could retrieve apoplastic auxin before it diffuses away to the inner layers. A parallel could be drawn with the vertebrate brain, where the neurotransmitter glutamate is removed from the synaptic cleft by re-uptake into presynaptic terminals, in order to return to a low resting concentration after exocytotic release of glutamate[37].

Discussion

Phyllotaxis is one of the most conspicuous examples of patterning in plants, and our results reveal a central role of auxin in its molecular regulation. The accumulated evidence clearly indicates an instructive rather than a permissive role for auxin in phyllotaxis. First, genetic and pharmacological interference with auxin transport or response leads to a wide range of phyllotactic patterning defects at all stages of development. Second, the phyllotactic expression patterns of several regulatory genes were abolished in *pin1*, as well as in *pid* and *mp*. Third, if *PIN1* just transmitted phyllotactic information from an upstream regulator, then the *PIN1* mRNA should be expressed in the normal phyllotactic pattern in *pin1*. Thus, the lack of phyllotactic expression of *PIN1* in the *pin1* mutant shows that *PIN1* not only responds to the phyllotactic signal (auxin) but also itself creates phyllotactic pattern. Fourth, exogenous auxin application does not only restore organogenesis, but the observed effects depend on the particular mutant, on the concentration of auxin and on the site of application. Fifth, application of the natural auxin IAA, but not the synthetic auxin 2,4-D (which is a poor substrate for the efflux carrier), can induce the PIN1-dependent generation of *de novo* phyllotactic pattern on the *pid* mutant (Fig. 3). Sixth, the localization of the putative auxin transporters is highly informative and predicts auxin distribution in a dynamic pattern, with auxin accumulating each plastochron at the site of incipient organ initiation.

Based on the experimental data, we propose a model for the regulation of phyllotaxis in *Arabidopsis* (Fig. 5). Auxin is acropetally transported towards the meristem. In the meristem, auxin becomes absorbed by the primordia, which function as sinks. As a result, auxin is depleted from the surroundings of the primordia and reaches the organogenetic peripheral zone only at a certain minimal distance from P_1 and P_2 (corresponding to the I_1 position). Auxin accumulation at I_1 induces a new primordium, which, in the course of the plastochron, will grow out and itself become a sink for auxin. This mechanism represents a combination of positive feedback (auxin accumulation) and lateral inhibition (withdrawal of auxin from adjacent tissues), that could conceptually be compared with the short-range activator and the long-range inhibitor in reaction–diffusion mechanisms[8].

The described system would require dynamic regulation of PIN1 during the course of the plastochron. Indeed, cellular redistribution of the PIN1 protein can be extremely rapid[21,38] and could easily allow for the 137° shift in orientation required during each plastochron. In the root, the subcellular localization of PIN1 is dynamically regulated by GNOM, which is a component of the vesicular cycling mechanism[24,39]. A similar mechanism could be active in the meristem. We also note that genes such as *PINHEAD/ZWILLE* are expressed in a phyllotactic pattern well below the meristem, and might also contribute to phyllotactic patterning[40]. However, the lack of patterned expression of all genes tested in *pin1*, *pid* and *mp* show that phyllotactic patterning in the *Arabidopsis* inflorescence cannot proceed independently of auxin. Essential downstream effectors that are required to translate auxin-based patterning information into actual growth patterns are likely to include factors such as *CUP-SHAPED COTYLEDON1* and *2* (*CUC1* and *CUC2*)[12,41] for organ separation, and expansin for organ outgrowth[42].

Our model accounts for the reiterativity and the stability of organ positioning. But how are the divergence angles determined? We speculate that if only P_1 is effective as a sink, a distichous phyllotaxis (divergence angle 180°) would result. Higher-order phyllotactic systems will result from the contribution of increasing numbers of primordia. Important parameters influencing relative sink strength are likely to be the size of the central zone relative to the organogenetic zone (plastochron ratio) and the growth rate of the apex along the apical–basal axis[43]. In addition, the timing of the transition of primordia from auxin sinks to auxin sources will influence phyllotaxis. Our work addresses the maintenance of phyllotactic pattern. The challenge now is to elucidate how phyllotactic patterns are generated *de novo*, and how they change under natural conditions, for example when dicotyledonous plants undergo the transition from decussate to spiral phyllotaxis. □
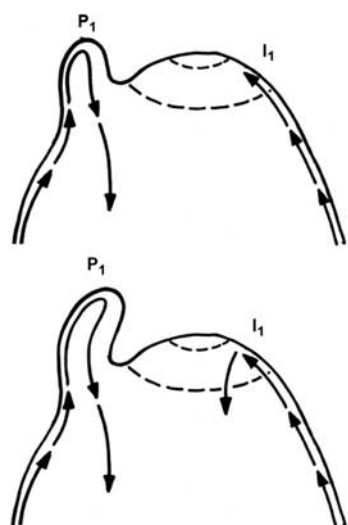

Figure 5 Model for the role of polar auxin transport in phyllotaxis. Schematic representation of an apex in longitudinal section through P_1 and I_1 at an early (top) and a later stage (bottom) of incipient primordium formation. Polar auxin flux is indicated with arrows. Top, acropetal auxin flux is diverted by P_1 preventing auxin accumulation on the left flank of the meristem, while auxin can reach the right flank (I_1). Bottom, accumulation of auxin at I_1 promotes primordium formation, and establishment of a new auxin sink.

Methods

Plant material and growth conditions

articles

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