Auxin, Self-Organisation, and the Colonial Nature of Plants

Review

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Evolution has provided at least two particularly successful independent solutions to the problems of multicellularity — animals and higher plants. An obvious requirement for successful multicellularity is communication between different parts of the organism, both locally, for example between neighbouring cells, and over very long distances. Recent advances in understanding hormone signalling networks in plants are beginning to reveal how co-ordination of activity across the whole plant body can be achieved despite the lack of a control centre, typical of animal systems. Of particular importance in this distributed regulatory approach are the self-organising properties of the transport system for the plant hormone auxin. This review examines the integrative role of the auxin transport network in co-ordinating plant growth and development.

Introduction

Multicellularity usually involves specialisation of cell, tissue and organ types to perform delegated functions within an organism. The successful co-ordination of these functions is essential for the success of the organism and requires both short- and long-range communication systems. These systems are needed for the establishment of the different cell, tissue and organ types in the correct pattern during development, their coordinated growth, and the maintenance of homeostasis with appropriate adjustments in the face of environmental challenges.

In animal systems, as a gross generalisation, the three elements of long range signalling — patterning, growth and homeostasis — are somewhat separated. For example, there are relatively separate phases and regulatory systems for developmental vs. homeostatic/environmental regulation of cell, tissue and organ interactions. Embryogenesis can result in an organism with an essentially adult body plan, while post-embryonically, physiological and behavioural changes allow adaptation to the environment. In contrast, plant development is continuous, and tissue establishment and environmental adaptation are intimately connected. Although the basic axes of the plant body are laid down during embryogenesis, they are subsequently greatly elaborated post-embryonically to produce the adult plant body.

Post-embryonic development is driven by meristems — small groups of cells with stem cell-like properties [1,2]. During embryogenesis, a shoot apical meristem and a root apical meristem are established at the shoot and root poles, respectively (Figure 1). Post-embryonically, the meristems lay down new shoot and root tissues behind them, to extend the primary axes. In addition new, secondary meristems are established, allowing new axes of growth, i.e. branches, in both the root and shoot. Higher-order branches can follow.

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As a result, the final plant body plan is highly variable, depending on the number of branches produced, and how active they become. This flexibility is facilitated by the modular nature of plant development, with the body being assembled by the reiteration of basic developmental units.

This growth habit allows adaptation of form according to the prevailing environment — for example, modifying root and shoot system architecture to allow optimal nutrient, water and light capture — and continually rebalancing the allocation of resources between roots and shoots depending on whether carbon or nutrients are more limiting. In this way, regulation of post-embryonic growth and developmental events in plants is, in many ways, equivalent to behavioural responses in animals, which are similarly characterised by exquisite sensitivity to environmental and physiological status.

Long-Range Signalling and Coordination

In animal systems, the predominant post-embryonic long-range communication networks include the endocrine and nervous systems, which are connected at various levels. In both of these systems, there is some level of central coordination with, for example, production of the signals limited to unique specialised sites, which can act as integrators for multiple inputs. The prevailing model is one of central control. In contrast, plants operate with more distributed control systems. Indeed it has been argued that plants can be considered as colonial organisms with multiple redundant parts [3]. Plants generally have no unique parts or organs but, through their iterative growth habit, many of each type. Thus, while there is clear specialisation of roots to gather water and nutrients from the soil, and leaves to capture light and CO_2, no one root or leaf is essential.

These differences in organisation in both body plan and the co-ordination of activity across it are unsurprisingly accompanied by fundamental differences in signalling systems, but also many conceptual similarities, which are explored below.

Systemic Signalling via the Vascular System

As in animals, the vascular system is an obvious conduit for long-range signalling molecules capable of coordinating activity across the plant. In the vasculature there are two sorts of conducting tissue that run in parallel throughout the plant: xylem and phloem.

Xylem

Xylem vessels differentiate from files of cells that undergo cell wall lignification and apoptosis to generate empty tubes of water impermeable cell walls, which act like drinking straws to transport water and dissolved nutrients from the root to the shoot in the so-called transpiration stream (Figure 1). The energy for this transport is provided by the evaporation of water from leaves via stomatal pores, found predominantly in their lower epidermis.

Stomata are also the sites of CO_2 uptake for photosynthesis. Pore size is tightly regulated, balancing the need for CO_2 acquisition against excessive water loss [4]. Thus, the
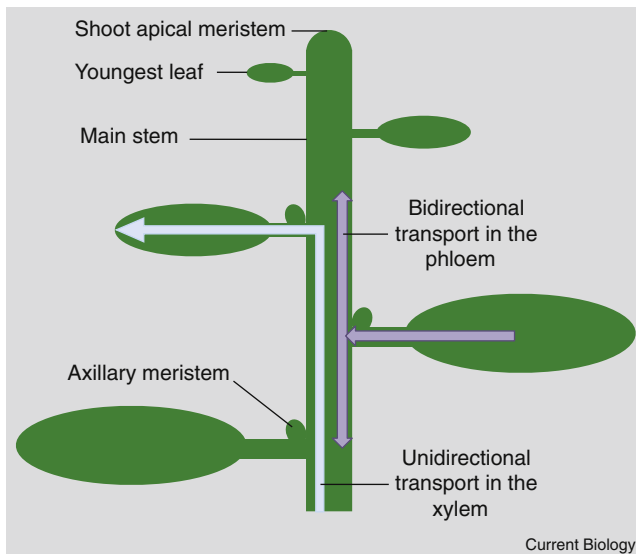
Figure 1. Transport systems in the modular shoot.

Cells in the primary shoot apical meristem divide to produce a stem below it and leaves around its circumference. Secondary shoot apical meristems are established in the axils of leaves (axillary meristems) with the same developmental potential as the primary shoot apex, but they may enter a dormant state and never fulfil this potential. Signals modulating the growth and development of the shoot are transported in the transpiration stream in the xylem, driven by evaporation of water from the leaves pulling water up from the roots, bringing along dissolved nutrients and signals. Signals can move bi-directionally in the phloem, which transports fixed carbon in the form of sucrose from source tissues, such as mature photosynthetically active leaves, to sink tissues, such as the roots or the growing shoot tip.

amount of water moving through the system can change dramatically depending on photosynthetic demand for CO_2 and water relations. Stomata are usually closed at night and partially closed in shaded parts of a plant, and they close in response to drought. These properties make the xylem rather interesting as a signal delivery system.

In the xylem, signals can only move upward, toward leaves with open stomata. Regulated synthesis of signals and loading into the transpiration stream can be used to influence the concentration of signal in the xylem, but the amount of signal arriving in the leaves will depend not only on concentration but also the rate of flow of water through the xylem. Recent evidence suggests that xylem sap is in much more dynamic equilibrium with neighbouring xylem parenchyma cells than one might expect, given the rapidity of xylem flow [5,6]. Thus, the concentration of signals in the sap may also be affected by uptake by xylem parenchyma cells. Thus, different parts of the shoot could in principle receive different amounts of signal depending on differential stomatal opening and differential uptake of xylem sap solutes by xylem parenchyma cells. In addition, new vascular strands can arise during growth and development. Thus, flow rates to any particular part of a plant can also vary over the long term due to differentiation of new vessels, or collapse of old ones.

A number of important regulatory signals are transmitted from root to shoot in the xylem. These include hormones involved in regulating root–shoot balance, such as cytokinins, which promote shoot branching, and strigolactones, which inhibit shoot branching. Both these hormones can report general root vigour, but both have also been implicated in communicating nutrient availability in the soil to the shoot [7–11]. Interestingly, soil water availability is also communicated in this way, via the hormone abscisic acid [12], the biosynthesis of which is up-regulated in roots by drought. Abscisic acid delivery to leaves via the xylem results in stomatal closure, reducing water loss, but simultaneously reducing xylem flux and abscisic acid delivery via this route, providing a feedback mechanism.

Phloem
Like xylem, the phloem consists of end-to-end files of cells connected to form tubes. However, unlike xylem, phloem cells remain alive and the connections are cytoplasmic through perforations in the end cell walls. The phloem delivers sucrose from source tissues, principally photosynthetically active leaves, to sink tissues, sites of sucrose removal through respiration, growth, or storage (Figure 1). Movement through the phloem can therefore be bidirectional, for example from a mature leaf up to the young growing shoot tip, or down to the roots. The movement is probably driven by bulk flow caused by loading and unloading of sucrose at the source and sink tissues, respectively [13].

Because the phloem is a living intracellular transport route, it is possible to regulate more directly and specifically the delivery of signals moving in the phloem to target tissues. Unloading can be limited to sites expressing specific transporters [14]. However, the fact that bulk flow in the phloem reflects tissue source/sink status provides opportunities for signals to be transmitted from source to sink tissues, hitching a ride on this flow and providing information about source status to sinks. This mechanism has been suggested for the small protein FT, which moves in the phloem and regulates shoot meristem determinacy and floral transition [15].

Auxin
Apart from the xylem and phloem, other systemic signal transmission mechanisms have been proposed, including hydraulic and electrical signalling. However, the only other known systemic transport system in plants is a dedicated network for the transport of auxin. Auxin (Figure 2) is a central regulator of plant growth and development, important for local patterning during embryogenesis [16], and post-embryonically in the iterative production of tissues and organs at both the root and shoot meristems [1,2]. In addition, auxin is an important mediator of long-distance signalling. This can be relatively direct — auxin moves over long distances, from shoot to root, influencing growth and development along this transport path, for example regulating elongation and branching [17–19]. It can also act more indirectly and over the long term in that a key role of auxin is in promoting the development of new vascular strands [20], thus affecting the capacity for transport of other systemic signals and of water, mineral nutrients and sugars.

The auxin transport system [21] is dependent on the weakly acidic nature of indole-3-acetic acid (IAA), the most prevalent naturally occurring auxin. In the extracellular environment of the cell wall, the apoplast, the pH is quite low (typically about 5.5), resulting in a proportion of auxin existing in an uncharged protonated state and thus able to cross the plasma membrane and enter cells. Active and potentially more rapid uptake of auxin is mediated by auxin import-proteins of the AUX/LAX family. In the cytoplasm, the pH is higher (typically 7), resulting in essentially complete

ionisation of auxin, trapping it in the cell. This means that auxin can accumulate to high levels inside cells and can continue to do so against a concentration gradient.

For auxin export, transporters are required. The two main classes of exporter are a family of ABCB transporters, members of the multidrug resistance transporter super-family, and the PIN family of plant-specific transporters. ABCB transporters have a major role in determining auxin flux through tissues, but much attention has focused on the PIN proteins because they are frequently polarly local-ised in cells, thereby determining the direction of auxin movement through tissues, and establishing the patterns of auxin distribution that direct development [22]. Further-more, PIN positioning is highly dynamic and PIN reposition-ing is crucial to the iterative production of organs during development [23–27] and to the modulation of patterns of growth and development in response to the environment, for example in gravitropism [28].

In the context of long distance auxin transport, more stable patterns of PIN localisation are observed (Figure 2). The unidirectional shoot-to-root transport of auxin mentioned above is polarised by the basal location of PIN transporters in files of parenchyma cells associated with the xylem [29] (Figure 2). This directional auxin flow is termed the 'polar auxin transport stream' (PATS) and it is remarkably stable [30]. Nonetheless, the stream can be rerouted if inter-rupted by wounding, illustrating the impressive self-organis-ing properties of the auxin transport network.

In an elegant series of experiments Tsvi Sachs [31,32] es-tablished that if a vascular strand in the stem is interrupted, for example by the insertion of a razor blade into the stem, the vasculature reroutes around the blade, reconnecting the upper and lower sections of the original strand. A very similar phenomenon is observed upon the application of an auxin source to a section of stem tissue. Vascular strands will differentiate, connecting the auxin source to existing vascular strands in the tissue.

Sachs proposed the canalisation hypothesis to account for these observations [32]. A central tenet of the hypothesis is a positive feedback between auxin flux and the accumula-tion of auxin transporters and their polarisation in the direc-tion of the flux. This can explain the wound healing process because when a vascular strand is cut, auxin in the PATS moving down the stem above the cut builds up at the cut site. Meanwhile auxin in the PATS moving down the stem below the cut is transported away, resulting in auxin deple-tion below the cut. This results in an auxin source above the cut and an auxin sink below it. Initial passive movement of auxin from the source to the sink is amplified by the canal-isation positive feedback process to produce files of cells with high auxin transport activity polarised toward the sink, and these files can differentiate into vascular strands. The canalisation hypothesis was proposed before the discovery of PIN proteins, but more recent observations have impres-sively demonstrated the accumulation and polarisation of PINs during this type of vascular reconnection process in exactly the predicted way [33]. ABCBs are likely to play an essential role in canalisation by providing a basal level of auxin export from cells, thereby keeping a proportion of auxin outside the cell, preventing stagnation of auxin flux through tissues in the absence of PINs, and amplifying it in the presence of PINs [34,35].

Mathematical and computational models based on the canalisation hypothesis are capable of reproducing a range
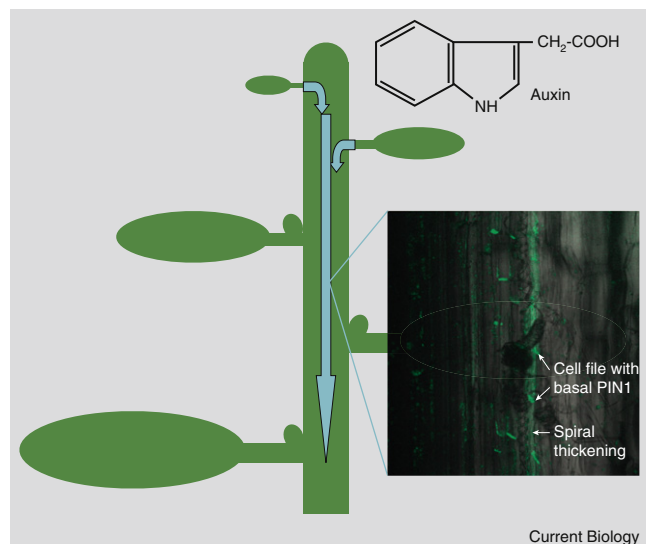

Figure 2. The polar auxin transport stream.

The chemical structure of indole-3-acetic acid, the most common natural auxin, is shown top right. Auxin synthesised in young expand-ing leaves is transported rootward in files of xylem parenchyma cells running parallel to the xylem vessels. Directionality for this transport is provided by the basal location of PIN family auxin efflux carriers. The inset shows GPF-tagged PIN1 protein basally localised in xylem parenchyma cell files (as indicated) imaged by confocal microscopy of hand sectioned *Arabidopsis* stems [64]. The spiral thickening of adjacent cell files is typical of differentiating xylem.

of phenomena observed in plant patterning, such as vascular connection between new leaves and existing vasculature in the stem, and many aspects of venation patterning in leaves [36]. Despite this useful explanatory and predictive power, the canalisation hypothesis has remained controversial because the mechanistic basis for the feedback at its heart is unknown. With increasing insight into the mechanisms of auxin signalling, and particularly its effects on auxin-trans-porter biology, hypotheses concerning this feedback are beginning to emerge.

Auxin Signalling

There are multiple auxin signalling pathways. Best under-stood is the pathway that regulates transcription, which is remarkably short [37]. Auxin binds directly to a small clade of nuclear receptors of the F-box protein family. F-box proteins are the substrate-selection subunit of a class of ubiquitin–protein ligase complexes, which polyubiquitinylate their substrates, marking them for proteasomal degradation. The degradation targets, in this case, are members of the Aux/IAA family of transcriptional repressor proteins. These can be considered as co-receptors with the F-box proteins because they dock with the F-box proteins at the auxin binding pocket, with auxin acting as a molecular glue keeping the two together [38]. The consequent ubiquitinyla-tion and degradation of the Aux/IAAs can de-repress transcription.

There is good evidence that this transcriptional pathway contributes to feedback between auxin and its transport in a way consistent with canalisation. An ever-increasing collection of genes involved in PIN accumulation and target-ing are regulated by this pathway, either directly or indirectly. Of particular relevance for canalisation are the PIN proteins

themselves [23,39], which are up-regulated by auxin, and ICR1 [40], a protein involved in polar targeting of PINs to the plasma membrane. ICR1 interacts with ROP/RAC GTPases, which are involved in cytoskeletal organisation and targeted vesicle trafficking [41].

In addition to the transcriptional pathway, with its nuclear receptors, additional signalling pathways involving extracellular and cytoplasmic receptors have been proposed [42]. The best characterised extracellular receptor was initially identified as an auxin-binding protein in maize and named ABP1. Loss of ABP1 function in *Arabidopsis thaliana* results in early embryo lethality [43]. Progress in understanding the post-embryonic roles of ABP1 has been greatly helped by conditional knock-down approaches and by abp1-5, an allele which is predicted specifically to reduce the auxin binding affinity of the protein [44–47]. ABP1 has an endoplasmic reticulum (ER) retention signal, but there is good evidence that some is secreted and that it can act in extracellular auxin perception [48]. Recently, a role for ABP1 in PIN accumulation at the plasma membrane has been demonstrated [46]. ABP1 promotes PIN endocytosis, but binding of auxin inhibits this activity. This effect is likely to be driven from an extracellular location because over-expression of ABP1 lacking the ER retention signal results in increased endocytosis. This then represents another mechanism by which auxin can increase auxin flux, by keeping PINs on the membrane. In addition, through this mechanism, there is the possibility for differential auxin distribution in a tissue to influence the polarity of PIN accumulation. A computational model in which ABP1 is allocated to the membrane of one or the other of two adjacent cells, depending on the auxin gradient across the cell wall between them, has been shown to be capable of reproducing canalisation phenomena when combined with the transcriptional pathway increasing PIN expression [49]. Experimental evidence to support the ABP1-dependent local accumulation of PINs comes from studies of differential PIN accumulation and cell expansion in leaf pavement cells, an effect apparently mediated by ABP1-dependent auxin-mediated ROP/RAC activation [47]. Taken together, these data suggest that ABP1 could be an important component of the canalisation feedback machinery. However, plant lines where ABP1 is significantly down-regulated, to the point that it is no longer detectable with antibodies, show severe morphological defects, but essentially normal vascular patterning, suggesting that canalisation is not greatly disrupted [44]. This may be due simply to redundancy in the system, with other components being able to compensate for lack of ABP1. Alternatively, ABP1 may act in other aspects of PIN positioning, and may be less relevant for canalisation.

The above raises a more general question about the universality of PIN positioning systems. Based on current evidence, it is unlikely that a single mechanism can account for the many different behaviours of PINs. For example, in addition to the mechanisms described above, there is evidence that PINs can be positioned and repositioned according to physical forces [50] and that the polarity of PIN localisation can be dramatically flipped in response to PIN phosphorylation by a family of AGC kinases [51,52]. Because of the diverse contexts in which PIN polarity is modulated and the many different inputs that influence these events, including of course auxin itself, it is necessary to study PIN localisation in the specific context of interest to be sure of analyzing the relevant mechanisms. This is quite difficult in the context of canalisation because it occurs in some of the less accessible parts of plants. To date, most work has focused on local patterning events in root and shoot tips, and it will be interesting to discover how much overlap there is between the mechanisms revealed in these studies and the systems that drive canalisation.

Canalisation and Long-Range Signal Integration

Whatever its detailed biochemical basis, the phenomenon of canalisation has a sound observational foundation. Assuming that this process functions in some form across much of the systemic auxin transport network, the resulting system has extremely interesting properties. Long-range signalling between distant plant parts can be achieved based on interactions between auxin flow along the interconnected auxin transport routes. Conceptually, this long-range communication is similar to that arising on road networks where changes in traffic flow in one area can cause either hold-ups or increased flow in other parts of the road system. The auxin transport network, therefore, has the potential to integrate information over the whole plant and could be the mainstay of the distributed processing system mentioned above, and the key to understanding the colonial properties of plants [53].

Auxin Transport and Shoot Branching

The power of the systemic auxin transport network as a distributed processing system is well illustrated by its role in the control of shoot branching. The importance of auxin in the regulation of shoot branching was first identified through the phenomenon of 'apical dominance' [19]. As every gardener knows, removal of the leading shoot apex encourages the activation of previously dormant buds in the axils of subtending leaves. If auxin is applied to the decapitated stump, the activation of these axillary meristems can be prevented. As the young expanding leaves at the shoot apex are known to be highly active auxin sources [54], and as described above, the auxin is transported from them down the stem in the PATS, this suggests that apically derived auxin moves down the stem and inhibits the activation of more basal buds [55–57]. Consistent with this idea, pharmacological inhibitors of PATS prevent bud inhibition by apical auxin.

Initially it was proposed that auxin in the PATS moves upwards into buds and inhibits them directly, but this was discounted because application of auxin directly to buds does not inhibit them, and radiolabelled auxin applied apically moves down the stem and inhibits the buds without the radiolabel accumulating in the bud [57]. It was then proposed that a second messenger was involved in relaying the auxin signal into the bud. Consistent with this idea, auxin, signalling through its transcriptional pathway, can down-regulate the accumulation of transcripts encoding cytokinin biosynthetic enzymes, leading to reduced cytokinin levels [58,59], and up-regulate those encoding strigolactone biosynthetic enzymes [60,61]. As the direct application of cytokinins to buds activates them [62], and the direct application of strigolactones to buds inhibits them [63,64], this second messenger idea would seem to offer an excellent explanation for the indirect mode of auxin action. In further support, in the case of strigolactones where viable mutants are available, loss of strigolactone synthesis or signalling results in increased shoot branching and reduced response of buds to apically applied auxin [56,65,66]. However,

despite the attractions of the second messenger idea, the situation seems to be much more interesting than it would first seem. At least in the case of strigolactones, their ability to inhibit bud activation is highly context-dependent [67,68]. Strong bud inhibition is only observed in the presence of an active auxin source exporting auxin into the stem on which the bud is carried (Figure 3). Thus, on *Arabidopsis* stem segments bearing a single bud, strigolactone application has no effect on bud growth, whereas when apical auxin is simultaneously applied, strigolactone treatment results in increased bud inhibition (Figure 3A–D). Perhaps even more strikingly, when two buds are present, strigolactone treatment results in the focusing of growth into one bud or the other (Figure 3E–F).

The above results can be explained by considering the evidence that a primary mode of action for strigolactone in shoot branching control consists of reducing the accumulation of PIN proteins on the plasma membrane [66,68]. Plants mutant in strigolactone signalling or biosynthesis show over-accumulation of PIN proteins in xylem parenchyma cell files in the stem. Consistent with this, these mutant stem segments transport more applied radiolabelled auxin than wild-type controls. For both PIN accumulation and auxin transport, wild-type levels can be restored by application of strigolactone for the biosynthetic mutants but not for the signalling mutant. These responses are quite rapid, with a strong reduction in PIN accumulation visible after six hours of treatment. Furthermore, reducing PIN activity in other ways, for example with low level treatment with pharmacological inhibitors, results in restoration of normal shoot branching levels and normal response of buds to apical auxin [66,69,70]. These last results are particularly significant because, as described above, application of auxin transport inhibitors to wild-type plants promotes shoot branching and prevents inhibition of buds by apical auxin. Thus, increased shoot branching can be associated with either very high or very low levels of auxin transport in the main stem, and in either case, this can render buds resistant to the effects of apically applied auxin.

If these effects on auxin transport are the cause of the branching phenotype of strigolactone mutants, as the evidence suggests, then auxin transport must be central to bud activity. Consistent with this idea, it has previously been demonstrated that there is a strong correlation between the activity of a bud and its ability to export auxin into the main stem [71,72]. If it is the case that such export is required for bud activity, this can be used as the basis for understanding how auxin moving in the main stem can inhibit bud activity, and how strigolactones can modulate this effect.

The previously established correlation between bud activity and bud auxin transport has recently been extended to PIN polarisation. In the stems of inactive buds PINs are not polarised, but an early event in activation involves their polarisation toward the main stem [73,74]. Furthermore, auxin transport along the main stem is apparently able to inhibit the establishment of polar auxin transport out of the bud and associated PIN polarisation — a phenomenon that has been termed 'auxin-transport auto-inhibition' [72]. This has led to speculation about mechanisms by which buds can detect auxin transport in the stem, but consideration of the canalisation hypothesis makes such mechanisms unnecessary. Active auxin transport in the main stem can, by itself, prevent canalisation of auxin transport from the bud to the
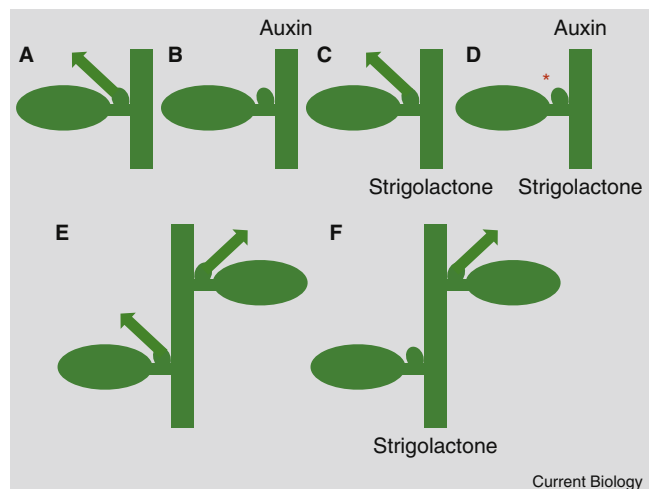

Figure 3. Context-dependent action of strigolactone in bud inhibition. Top row, solitary buds on isolated nodal stem segments: (A) Activation of untreated bud to produce a branch. (B) Inhibition of bud by apical auxin application. (C) No effect on bud of basal strigolactone application. (D) Super-inhibition of bud (red asterisk) by simultaneous application of basal strigolactone and apical auxin. Bottom row, stem segments with two buds: (E) Activation of both buds when untreated. (F) Activation of only one bud when treated with basal strigolactone.

stem by presenting a weak sink for auxin flux out of the bud. If the background auxin flux from the bud to the stem is low, then the positive feedback loop that drives canalisation will not be activated and PINs will remain unpolarised, as is observed. Upon decapitation, the amount of auxin in the main stem drops, whilst its ability to transport any auxin away to the root is, at least in the first instance, maintained, thus generating an excellent sink for auxin, and increasing background flux from the bud into the stem. This initial flux would activate positive feedback and thus canalised auxin transport out of the bud, establishing a bud PATS.

Computational modelling has demonstrated the plausibility of this switching mechanism [49,73], but perhaps the most compelling argument for its operation is that it can explain the observations of strigolactone mutant behaviour under the hypothesis that strigolactones regulate, for example, the rate of PIN removal from membranes [68,73]. If strigolactones enhance the rate of PIN removal from the plasma membrane, then high strigolactone would reduce the effectiveness of the canalisation feedback loop by rapid removal of inserted PINs coupled with a simultaneous reduction in main stem sink strength by reducing its ability to transport auxin away to the root. Similarly, low strigolactones would enhance the feedback loop by reducing PIN removal. In this way, the auxin-transport switch is on a hair trigger in strigolactone-defective mutant buds, but it is more difficult to activate buds in the presence of high strigolactone. This explains the correlation of high PIN accumulation and high auxin transport with auxin resistant hyperactivation of buds in strigolactone mutants.

This model also explains why strigolactone is only effective at inhibiting bud activation in the presence of an alternative auxin source. If there is only one bud present on a stem segment, and thus no auxin in the main stem, there will be sufficient auxin flux from the bud to the stem to activate the positive feedback loop. In the presence of a competing auxin source, where strigolactone is high, the bud will

be inhibited more easily, as described above. Effectively, strigolactones enhance the competition between auxin sources for a common auxin transport path in the main stem.

This regulatory system has very interesting properties from the point of view of systemic signalling and co-ordination of growth across the plant. For example, as mentioned above, strigolactones are transported up the plant in the xylem and their synthesis in roots is greatly up-regulated during nitrate and phosphate starvation [7–9]. According to the model, the delivery of strigolactones to the shoot in the xylem results in perfusion of the shoot with strigolactones, dampening canalisation. The result will be that fewer axillary buds can activate, reducing shoot growth to a level appropriate for the nutrients available. However, the generally higher levels of strigolactone do not dictate which axillary buds will become active. They reduce the total number of active buds, but local inputs will determine which buds win the competition to establish auxin transport into the main stem. Those that become active first will prevent activation of latecomers. In this way, the most 'vigorous' buds, for example those in the best light environment [75], will become active, while more feeble buds will remain dormant. In more favourable nutrient environments, more buds will be active, but even then, these will be the most 'vigorous'.

This model is consistent with the idea that the shoot system can be considered as a collection of competing meristems [3], each of which depends for its activity on the export of auxin into the main stem. The main stem ultimately connects all the shoot tips on the plant, however ramified its branching pattern. The feedback systems in the auxin transport network thus allow growth to be balanced across the entire shoot system, through local competition, with the possibility of global modulation of the strength of that competition, for example mediated by strigolactones. Given that auxin transport routes ultimately trigger vascular differentiation, a longer term positive feedback is inherent in the system as the branches that export the most auxin will also develop the most vascular connectivity with the stem and thus attract the most water, nutrients, etc. This system therefore provides a mechanistic framework for the consideration of plants as colonial organisms with competing populations of redundant organs, as envisaged by Sachs and co-workers [3]. Interestingly, recent evidence from moss suggests that an early role for strigolactones was as a quorum sensing molecule, mediating competition between adjacent colonies [76]. It will be interesting to track the evolutionary events that co-opted this system to mediate competition within a plant.

Acknowledgements

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