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REVIEW PAPER

The role of auxin in shaping shoot architecture

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Abstract

The variety of plant architectures observed in nature is predominantly determined by vegetative and reproductive branching patterns, the positioning of lateral organs, and differential stem elongation. Branches, lateral organs, and stems are the final products of the activity of meristems, groups of stem cells whose function is genetically determined and environmentally influenced. Several decades of studies in different plant species have shed light on the essential role of the hormone auxin in plant growth and development. Auxin influences stem elongation and regulates the formation, activity, and fate of meristems, and has therefore been recognized as a major hormone shaping plant architecture. Increasing our knowledge of the molecular mechanisms that regulate auxin function is necessary to understand how different plant species integrate a genetically determined developmental programme, the establishment of a body plan, with constant inputs from the surrounding environment. This information will allow us to develop the molecular tools needed to modify plant architecture in several crop species and in rapidly changing environments.

Key words: Auxin, inflorescence development, maize, meristems, plant architecture, rice.

Introduction

One of the most remarkable features of plant development is the ability of plants to adapt their body plans according to ever-occurring changes in the surrounding environment. Availability of water, nutrients, and light, as well as the presence of pathogens, predators, and symbionts, are cues that plants must constantly interpret and respond to in order to survive and reproduce. The ability to adapt growth throughout the life cycle is due to meristems, groups of plant stem cells. During embryogenesis, the shoot and root apical meristems (SAM and RAM, respectively) are formed and establish the main apical–basal axis of growth that persists throughout development. Post-embryonically plants have the ability to form new meristems, termed axillary meristems, at the axils of true or modified leaves. These meristems are responsible for the formation of secondary axes of growth and, like the SAM, function by maintaining a core of pluripotent stem cells, as well as forming lateral primordia and the supporting stems (Weigel and Jurgens, 2002).

Plant shoot development is organized in morphological units or modules, called phytomers (Gray, 1879; Galinat, 1959). Each phytomer is composed of a stem segment (internode), a node, which bears a true or modified leaf, and one or more axillary meristems in each leaf axil. Variations in phytomer development greatly contribute to the broad diversity of plant shoot architecture observed in nature. In particular where and how many axillary meristems are formed during both vegetative and reproductive development, and, most importantly, if they terminate in differentiated structures (e.g. flowers), elongate to originate new branches, or simply remain quiescent, represent major determinants of plant architecture in all species (McSteen and Leyser, 2005). Differences in plant architectures are observed not only among different plant species but also within different individuals belonging to the same species. This is particularly true in natural environments, more so than in domesticated environments, where plant architecture is strictly controlled for the purpose of mechanization (e.g. in orchards).

In the model plant system *Arabidopsis thaliana*, the SAM produces a rosette of leaves subtending new axillary meristems at their axils. Upon transition to reproductive

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development, new phytomers are formed with elongated stems, smaller leaves (cauline leaves), and indeterminate axillary meristems that originate new lateral branches. These events are subsequently followed by phytomers with no visible leaves, elongated stems, and floral meristems, determinate axillary meristems that eventually form reproductive organs (Fig. 1A). This developmental plan is distinct from the one seen in maize (*Zea mays*), a member of the grass family, where during vegetative development the SAM forms leaves in alternating positions along one main axis. New axillary meristems are formed at the leaf axils and, in turn, form a few leaf primordia then generally become quiescent. With the transition to reproductive development, the internodes start to elongate, the SAM acquires an inflorescence meristem identity and eventually generates the apical male inflorescence, the tassel. Several lateral female inflorescences, called ears, are borne on the tip of lateral branches originating from previously formed axillary meristems. Generally only one or two ears, derived from the uppermost axillary meristems, develop to full maturity and provide fruits. This genetic programme results in the formation of unisexual inflorescences borne on different parts of the stem (Fig. 1B). Rice (*Oryza sativa*) plants, on the other hand, normally produce a proliferation of tillers, vegetative branches that arise as a result of the derepression of basal axillary buds and are composed of a series of new phytomers resembling the main shoot. Each individual tiller forms a new shoot that is eventually topped by a bisexual grain-bearing inflorescence (Fig. 1C). Tillers are considered a detrimental agronomic feature for cultivated modern maize and have been strongly selected against during maize genetic improvement, as well as during maize domestication from its wild relative teosinte (Doebley *et al.*, 1997). A phenomenon known as apical dominance refers to the inhibitory effect exerted by the main shoot apex on the activity of axillary buds in many plant species. Removal of the SAM usually results in an outgrowth of multiple shoots from derepressed axillary buds, and the plants appear bushier. The absence of tillers in modern maize, for example, represents a case of strong apical dominance

While differences in architecture are observed in the shoot, they are also evident in inflorescences (Prusinkiewicz *et al.*, 2007). Common variations occur in the arrangement of axillary meristems around the stem (phyllotactic pattern) as well as in internode elongation and branching patterns. For example, *Arabidopsis* produces raceme-type inflorescences, where flowers are borne laterally on main axes, whereas maize and rice inflorescences are panicles that consist of series of branches terminating in flowers. Differences are also evident between the architecture of the male and female inflorescences of maize due to indeterminate axillary meristems (called branch meristems) that are present in the tassel and give rise to a series of long basal branches, but are normally absent in ears. In rice, instead, all primary axillary meristems are indeterminate branch meristems, and these give rise to the highly branched rice inflorescence (Fig. 1, insets).

A pre-determined body plan is intrinsic to the genetic programme of every plant species, but the surrounding environment plays an important role in the final developmental outcome. Phytohormones, small molecules that can be produced and transported to different parts of the plant, are major determinants of how plants grow and thereby represent the first line of response to changes in environmental conditions (Vert and Chory, 2011). One of the hormones that has a major influence on many, if not all, aspects of development is auxin. In many cases, classic developmental mutants from different plant species that were first noted for their altered

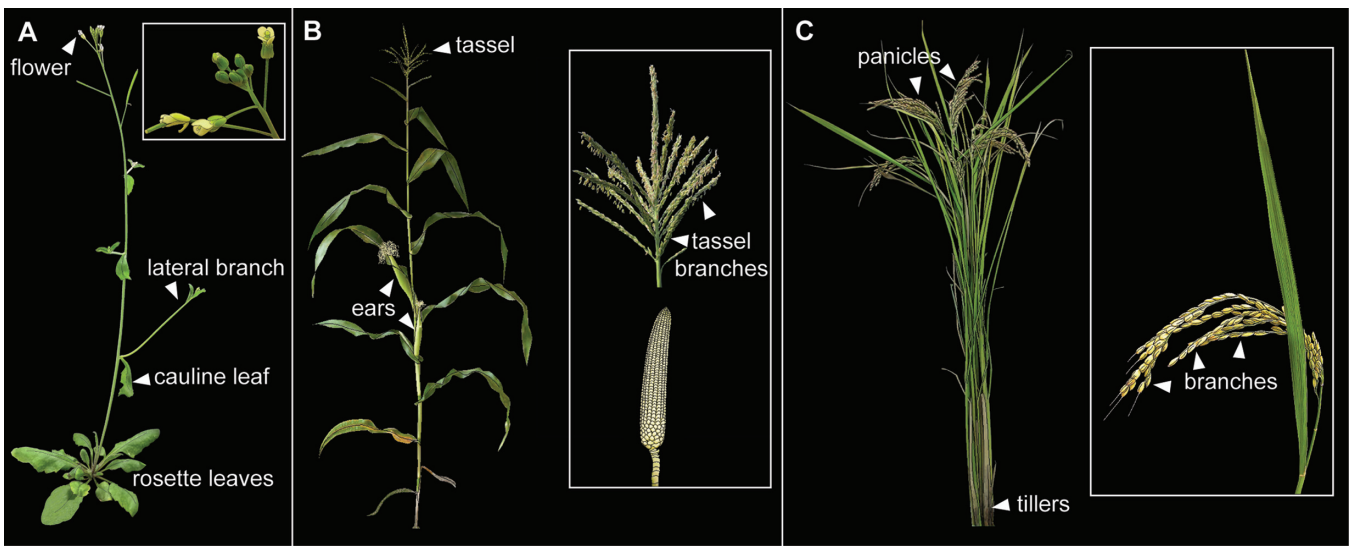

Fig. 1. *Arabidopsis*, maize, and rice shoot and inflorescence architectures. (A) *Arabidopsis* has a rosette of leaves and shoots carrying branches and flowers, arranged spirally along the shoot (inset). (B) Maize architecture is characterized by a single shoot, bearing leaves in alternate positions, and topped by a male inflorescence, the tassel. Borne at the axils of leaves are short branches tipped by female inflorescences, called ears. The tassel (inset, top) has several long branches and a long central spike, whereas the ear lacks long branches (inset, bottom). (C) Rice plants have multiple shoots all topped by a highly branched inflorescence (inset).

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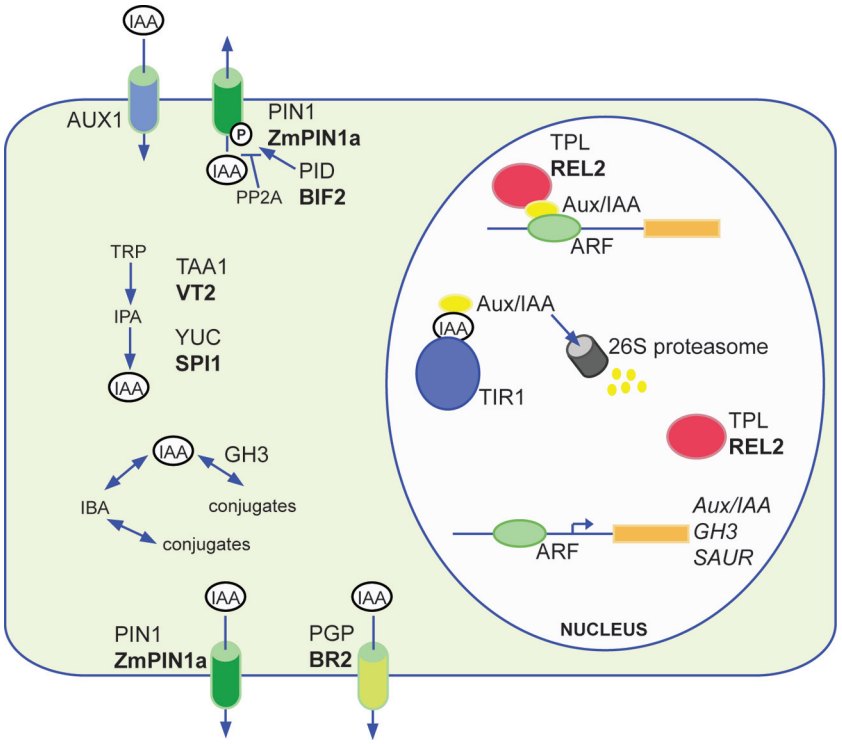

Fig. 2. Schematic representation of auxin biosynthesis, homeostasis, transport, and signalling. Only genes mentioned in the text and functioning in shoot development are included. In bold are genes functionally characterized in maize.

architecture have proven to be a gateway for the identification of a number of important genes related to auxin biology. Recent advances in deciphering the molecular mechanisms that regulate auxin function have greatly increased our understanding of the role that auxin plays in plant growth and development (Fig. 2), and have produced several tools to investigate how auxin affects the establishment and plasticity of plant morphology. Here the extensive evidence that connects auxin and its biology (homeostasis, transport, and signalling) with the determination of plant shoot architecture is reviewed, with particular emphasis on the most recent discoveries in two well-studied model plant systems, the dicotyledonous *Arabidopsis* and the monocot maize. Our current understanding of the molecular mechanisms and the role of auxin in the initiation of axillary meristems is also discussed. Throughout this review I will highlight only those components of auxin pathways that cause major phenotypic defects in plant architecture, and will use the respective nomenclatures of the different plant species mentioned.

Auxin biosynthesis

As suggested by the analysis of several auxin biosynthetic mutants in different species including *Arabidopsis*, petunia, maize, and rice, auxin biosynthesis is required for the formation of all primordia and is therefore essential to the development of a plant body plan (Tobena-Santamaria *et al.*, 2002; Cheng *et al.*, 2006; Woo *et al.*, 2007; Yamamoto *et al.*, 2007; Gallavotti *et al.*, 2008*a*; Phillips *et al.*, 2011). Defects in organ formation, internode elongation, and apical dominance are usually observed in the majority of these mutants. Several tryptophan-dependent and independent auxin biosynthetic pathways have been proposed over the years (reviewed in Woodward and Bartel, 2005; Zhao, 2010). For most of these pathways, single enzymes have been studied and characterized, but a comprehensive understanding of the sequential steps involved in each one is still lacking. A major breakthrough in the molecular events required for auxin biosynthesis in one of these pathways was recently reported in *Arabidopsis*. Two independent studies (Mashiguchi *et al.*, 2011; Won *et al.*, 2011) indicate that a major pathway for auxin biosynthesis in *Arabidopsis* involves the conversion of tryptophan to indole pyruvic acid (IPA) by the activity of tryptophan aminotransferase (*TAA*) genes (Stepanova *et al.*, 2008; Tao *et al.*, 2008) and the subsequent conversion of IPA to IAA (indole acetic acid, the most common natural auxin) by flavin monoxygenase enzymes belonging to the *YUCCA* (*YUC*) gene family. This finding was supported by genetic analyses of the interaction between *taa* and *yucca* mutants, as well as by measurement of IPA levels. Accumulation of IPA was indeed observed in *yucca* mutants, whereas reduced levels were found in *taa* mutants, consistent with the proposed two-step pathway (Mashiguchi *et al.*, 2011; Won *et al.*, 2011) (Fig. 2).

The *Arabidopsis YUCCA* genes were first identified thanks to *yuc1D*, a dominant gain-of-function auxin overproduction mutant that showed elongated hypocotyls when grown in white light (Zhao *et al.*, 2001). Other phenotypes observed included increased apical dominance and leaf epinastic

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growth. *YUC* genes are present in all plants, but phylogenetic analysis suggests that in monocots and eudicots independent expansion, diversification, and subfunctionalization may have occurred (Gallavotti *et al.*, 2008*a*). In *Arabidopsis*, *YUCCA1* belongs to a family comprising 11 members with specific expression patterns and functions (*YUC1*–*YUC11*). Subsequent studies on loss-of-function double and higher order mutant combinations helped to dissect the role of the various members of this family during development (Cheng *et al.*, 2006, 2007). These studies revealed developmental phenotypes undetected in single knockout mutants, such as loss of apical dominance and defects in leaf, vasculature, and flower formation, suggesting that the *YUC* genes have both overlapping and unique functions in development. Similar defects to those observed in *yuc* loss-of-function mutants are also seen in *taa* mutants. Whereas single mutants of different *TAA* genes in *Arabidopsis* are generally affected in hypocotyl elongation, triple *taa* mutants collectively display dramatic reductions in hypocotyl length and plant stature, decreased apical dominance, as well as vasculature defects and reduced organ numbers (Stepanova *et al.*, 2008).

Studies in other species lend support to the importance and conservation of the *TAA*/*YUC* pathway in plants. The recessive petunia *floozy* (*fzy*) mutant is affected in floral organ primordia and vasculature formation. *FLOOZY* encodes a *YUCCA-like* gene, and the observed developmental defects resemble those reported in *Arabidopsis* (Tobena-Santamaria *et al.*, 2002; Cheng *et al.*, 2006, 2007). Unlike in wild-type petunia, axillary meristems of *fzy* mutants are activated, resulting in a general loss of apical dominance in the inflorescence. Furthermore, when *FZY* is overexpressed, petunia plants accumulate excessive auxin, similar to the *yuc1D Arabidopsis* mutant, as indirectly observed by the ability of explants to form calli on media not supplemented with auxin (Tobena-Santamaria *et al.*, 2002). In maize, two loss-of-function mutants, *sparse inflorescence1* (*spi1*) and *vanishing tassel2* (*vt2*), led to the isolation of maize *YUCCA* and *TAA* genes, respectively (Gallavotti *et al.*, 2008*a*; Phillips *et al.*, 2011). Both *spi1* and *vt2* mutants are deficient in lateral organ formation, as both tassel and ear have a significantly reduced number of spikelets, the basic unit of grass inflorescences that bears the flowers, and those that do form ultimately lack floral organs (Gallavotti *et al.*, 2008*a*; Phillips *et al.*, 2011). *vt2* mutants, but not *spi1*, also show a major decrease in plant height and leaf number, suggesting that a smaller number of phytomers is formed. The phenotypic analysis of the *spi1;vt2* double mutant was one of the first pieces of evidence to hint that indeed flavin monoxygenase and tryptophan aminotransferase genes may work in the same auxin biosynthetic pathway, rather than in parallel pathways as originally thought (Phillips *et al.*, 2011). Double *spi1;vt2* mutants showed minor but significant enhancement of vegetative and reproductive developmental defects, but, nonetheless, they overall resembled *vt2* single mutant plants. Furthermore, measurements of auxin levels in leaves of *spi1;vt2* double mutants showed no decrease in free IAA, when compared with the levels detected in single *vt2* mutants (Phillips *et al.*, 2011).

The *Arabidopsis TAA1* gene was independently isolated in a mutant screen for the identification of genes involved in the shade avoidance response (Tao *et al.*, 2008). The shade avoidance syndrome consists of a series of developmental responses triggered by changes in the quality of light. One of the major changes commonly occurring in plants in reaction to shaded conditions is the elongation of the stem. This growth response is needed to compete for light with surrounding plants. After switching plants from white light to shade, an increase in the levels of free IAA due to increased biosynthesis was observed in normal seedlings but was impaired in *taa1*/*sav3* (*shade avoidance3*) mutants. A more recent report directly linked photoreceptor excitation in the shade avoidance response to changes in expression levels of auxin biosynthetic genes (Li *et al.*, 2012). In shaded conditions, the ratio of red (R) to far red (FR) light decreases, and this change can be perceived by phytochrome proteins. The main sensor of shaded light is phytochrome B, which in low R:FR conditions switches from an active to an inactive state. As a consequence, the basic helix–loop–helix (bHLH) transcription factor PHYTOCHROME-INTERACTING FACTOR 7 (PIF7), normally phosphorylated and degraded, is allowed to accumulate and bind to the promoters of *YUCCA8* and *YUCCA9*, and other genes. This cascade of events results in an overall increase in free IAA, and in the corresponding elongation of the hypocotyl in response to shade (Li *et al.*, 2012). Similarly, other PIF transcription factors (Leivar and Quail, 2011) were recently reported to regulate both auxin biosynthesis and signalling components directly (Hornitschek *et al.*, 2012).

In grasses, a major aspect of the shade avoidance response is the suppression of lateral bud outgrowth (Kebrom and Brutnell, 2007). Two maize mutants, *teosinte branched1* (*tb1*) and *grassy tillers1* (*gt1*), have been described as being affected in the regulation of this pathway. Both mutants are characterized by a marked increase in the number of tillers produced during vegetative development. The *tb1* gene is one of the founding members of the TCP transcription factor family (Doebley *et al.*, 1997), whereas *gt1* encodes a homeodomain leucine-zipper transcription factor that functions downstream of *tb1* (Whipple *et al.*, 2011). Domesticated maize varieties have been selected for repression of axillary bud outgrowth (Doebley *et al.*, 1997), and both *tb1* and *gt1* show evidence of selection during domestication (Doebley *et al.*, 1997; Studer *et al.*, 2011; Whipple *et al.*, 2011). It was recently shown that *gt1* transcripts are up-regulated in response to increased FR light in both teosinte, the wild progenitor of modern maize, and sorghum, two species that unlike maize show a strong shade avoidance response. This suggests that the regulation of axillary bud outgrowth by *tb1* and *gt1* is part of the shade avoidance response in grasses. It will be interesting to determine if any connection exists among *tb1*, *gt1*, and auxin, and if in grasses the response to shade also involves a direct regulation of auxin biosynthetic genes. Unfortunately, no auxin biosynthetic mutants have been described in sorghum or teosinte, while in rice antisense down-regulation of *OsYUC1* expression driven by a constitutive promoter produced plants with severe dwarfisms (Yamamoto *et al.*, 2007). According to the model presented by Whipple *et al.* (2011), and speculating

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that auxin biosynthesis in grasses is upstream of *tb1* and *gt1* function but downstream of phytochrome B activity, plants grown in shaded conditions could show an increase in auxin biosynthesis and lead to higher expression of *tb1* and *gt1* and a corresponding decrease in tiller outgrowth. In maize, genetic interactions between *tb1* and the two maize auxin biosynthetic mutants *spi1* and *vt2* indicated that whereas *spi1;tb1* double mutants showed a reduction in the number of tillers (Gallavotti *et al.*, 2008*a*), *vt2;tb1* plants formed a number of tillers comparable with *tb1* single mutants (Phillips *et al.*, 2011). The analysis of these apparently discordant effects of the two auxin biosynthetic mutants is complicated by the existence of redundancy in both gene families, and by the presence of a *vt2* duplicate gene (Phillips *et al.*, 2011). Furthermore, it is possible that the reduction in the number of tillers reported in *spi1;tb1* mutants is due to defective axillary meristem initiation during vegetative development, rather than a direct repression of axillary bud outgrowth, an observation that contrasts with the loss of apical dominance in *Arabidopsis yuc* mutants, since if axillary meristems form they still seem to elongate and form tillers (Gallavotti *et al.*, 2008*a*).

Another example of a direct connection between environmental influences on plant architecture and auxin is found in response to high temperatures (Franklin *et al.*, 2011; Sun *et al.*, 2012). *Arabidopsis* plants normally respond to high temperature by drastically increasing stem elongation, and this response is reduced in mutants affected in auxin biosynthesis, transport, or signalling. Additionally, it has been consistently shown that *Arabidopsis* seedlings grown at 28 °C produce higher levels of auxin. Two recent reports have begun to shed some light on these findings, showing that in response to elevated temperatures the expression of another phytochrome-interacting factor, *PIF4*, is up-regulated, and that PIF4 protein directly binds to the promoter of *YUCCA8* to stimulate hypocotyl growth (Franklin *et al.*, 2011; Sun *et al.*, 2012). These seminal studies involving *PIF* and *YUC* genes represent notable examples of mechanistic links between changes in the surrounding environment and plant architecture via modulation of auxin levels.

Auxin storage and degradation

The levels of auxin in different tissues need to be tightly controlled during development. This is accomplished not only by regulating auxin biosynthetic pathways as described above, but also by various mechanisms for degradation and storage. In general, auxin homeostasis involves the formation of conjugates with sugars, proteins/peptides, and amino acids. Some of these conjugates can later be hydrolysed to release active auxin or serve as tags for degradation pathways (recently reviewed in Ludwig-Muller, 2011). Formation of IAA–Ala, IAA–Leu, or IAA–Phe, for example, results in storage forms that can later be hydrolysed, whereas IAA–Asp or IAA–Glu targets auxin for degradation. IAA can also be converted by oxidation to IBA (indole-3-butyric acid) that can also be conjugated (Ludwig-Muller, 2011; Rosquete *et al.*, 2012) (Fig. 2).

The amidohydrolase family of proteins hydrolyses amino acid conjugates to produce free IAA *in vivo*. Single mutants of various *Arabidopsis* conjugate hydrolase genes do not show any visible phenotypes, but triple mutants have been shown to develop shorter hypocotyls, suggesting that the release of free IAA is necessary for normal hypocotyl growth. In accordance with these observations, overexpression of different members of the *Arabidopsis* family of auxin-conjugating synthase enzymes that reduce free IAA levels also affects hypocotyl growth and causes reduced apical dominance (Nakazawa *et al.*, 2001; Takase *et al.*, 2004). Ectopic expression of *WES1*/*GH3* (*GRETCHEN-HAGEN3*), an amino acid conjugase, for example, caused reduced hypocotyl growth, root defects, and altered leaf shape (Park *et al.*, 2007). The *GH3* gene that gives name to this family of enzymes was originally identified in soybean as an early auxin-responsive gene (Hagen *et al.*, 1984; Hagen and Guilfoyle, 1985).

Whereas in maize functional analysis has been carried out on some of these enzymes only in heterologous systems (Ludwig-Muller, 2011), in rice overexpression of a *GH3* gene, *OsMGH3*/*OsGH3-8*, altered overall plant architecture during vegetative development, reportedly causing reduced internode length and reduced apical dominance. Defects were also observed during reproductive development as panicles appeared to have reduced branching (Ding *et al.*, 2008; Yadav *et al.*, 2011). Rice loss-of-function mutants in this gene family seemed to affect predominantly fertility and disease resistance (Ding *et al.*, 2008). Similarly, overexpression of the rice enzyme *OsIAGLU*, an IAA–glucose synthase that is known to create IAA–glucose conjugates, generated rice plants with an increased number of tillers and reduced plant and inflorescence size, suggesting that decreasing free IAA levels enhances tillering and affects inflorescence development. Conversely, exogenous application of IAA significantly reduced tiller number in different rice cultivars (Choi *et al.*, 2012). It is possible that these seemingly contrasting phenotypes, increased vegetative branching (tillering) and decreased inflorescence branching, are due to pleiotropic effects on plant growth that manifest in later stages of development and therefore affect more severely reproductive organs. Alternatively, specific differences in auxin homeostasis during different phases of development may exist.

In summary, the mechanisms for auxin homeostasis and biosynthesis seem conserved in the different plant species mentioned. Auxin biosynthesis is required for all lateral organ formation, and defects are apparent in leaf formation, axillary meristem initiation, and outgrowth. The lack of maize mutants in auxin storage and degradation hinders a full comparison, but, as outlined above, in rice, reducing the pool of free IAA has similar effects to that observed in *Arabidopsis*, namely a general reduction in plant size, accompanied by a more bushy appearance.

Auxin transport

Auxin is actively transported over short or long distances throughout the plant by a complex network of active transporters, but can also move by passive distribution through

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vascular tissue. The isolation of active efflux and influx transporters operating in cell to cell movement of auxin has greatly increased our understanding of the process. Directional auxin transport is mediated by the PINFORMED (PIN) and the P-GLYCOPROTEIN (PGP)/MULTIDRUG RESISTANCE (MDR)/ATP-BINDING CASSETTE (ABC) family of efflux carriers, and the AUXIN1/LIKE AUXIN1 (AUX1/LAX) influx carriers (Fig. 2). Defective auxin transport, caused by means of either chemical transport inhibitors or specific genetic mutations, affects organogenesis and the formation of all primordia, including new axillary meristems. There is now overwhelming evidence supporting the fundamental role of auxin and in particular its transport in the positioning of new primordia at the SAM (see the review by Sassi and Vernoux, 2013).

The PIN family of polar auxin transporters functions in the efflux of auxin from cells and plays diverse roles in growth and development. PIN1, the first of eight *Arabidopsis* family members to be identified, has been directly shown to have a major effect on organ initiation in the shoot. *pin1* mutants indeed show strong defects in inflorescence development and generate pin-like inflorescences, hence the name *pin-formed* (Okada *et al.*, 1991; Galweiler *et al.*, 1998) (Fig. 3A). PIN1 is polarly localized in cells, and its subcellular localization is regulated by the contrasting functions of protein phosphatase 2A (PP2A) and PINOID (PID) (Friml *et al.*, 2004; Michniewicz *et al.*, 2007), a serine-threonine kinase that when mutated shows similar developmental defects to *pin1*;, namely the formation of naked inflorescences (Bennett *et al.*, 1995). In maize, mutants in the PIN family of auxin efflux transporters have not yet been reported. The most likely reason is functional redundancy caused by duplications in this family in maize and other grasses. Maize has 12 *ZmPIN* genes in its genome whose expression patterns have been recently characterized (Carraro *et al.*, 2006; Gallavotti *et al.*, 2008*b*; Forestan and Varotto, 2011; Forestan *et al.*, 2012). One member of the four *ZmPIN1* genes, *ZmPIN1a*, was shown to rescue the *pin1 Arabidopsis* mutant phenotype (Gallavotti *et al.*, 2008*b*). Currently, the only available information regarding auxin transport in maize comes from studies involving application of the auxin transport inhibitor NPA (*N*-1-naphthylphthalamic acid) to plants at different stages of development (Tsiantis *et al.*, 1999; Scanlon, 2003; Wu and McSteen, 2007; Gallavotti *et al.*, 2008*b*). In these experiments, the main effects reported were the formation of compressed internodes, the failure to initiate leaf primordia, and the absence of axillary meristem initiation during reproductive development.

In maize, developmental defects resembling the pin-formed phenotype of *Arabidopsis* are observed in what are referred to as the *barren* class of mutants (Fig. 3B). One of these mutants is called *barren inflorescence2* (*bif2*). In *bif2* mutants, both tassel and ear are severely impaired in the formation of axillary meristems and result in pin-shaped inflorescences that lack branches, spikelets, and flowers. *bif2* encodes a co-orthologue of the *Arabidopsis* PID serine-threonine kinase (McSteen *et al.*, 2007). Similarly to what was originally described for the *Arabidopsis* PID, BIF2 was

Fig. 3. An example of an *Arabidopsis* and a maize mutant affected in auxin transport and response, respectively. (A) The *Arabidopsis pin1* mutant inflorescence is reduced to a pin-formed structure. (B) The maize *barren stalk1* mutant tassel lacks all branches, spikelet, and flowers. The only primordia formed are suppressed bracts (arrowheads).

reported to phosphorylate ZmPIN1a *in vitro*. Consistent with these finding and those in *Arabidopsis*, the subcellular localization of ZmPIN1a was affected in *bif2* mutants (Skirpan *et al.*, 2009). Interestingly, the bHLH transcription factor BARREN STALK1 (BA1) was also found to be phosphorylated by BIF2 (Gallavotti *et al.*, 2004; Skirpan *et al.*, 2008). Strong *ba1* mutants are characterized by the failure to initiate axillary meristems throughout development, leading to maize plants composed only of a stem and leaves (Ritter *et al.*, 2002). Although the phosphorylation of BA1 by BIF2 has only been shown *in vitro*, the similar mutant phenotypes of *ba1* and *bif2* suggest that such an event may also occur *in vivo*. It was previously speculated that BA1 may function downstream of auxin transport (Wu and McSteen, 2007) and have a role in auxin signalling (Gallavotti *et al.*, 2004). The interaction with BIF2 seems indeed to support the existence of a connection with auxin pathways, though how the phosphorylation of BA1 affects its function has yet to be determined. Recently the orthologue of *ba1* in *Arabidopsis*, *ROX* (*REGULATOR OF AXILLARY MERISTEM FORMATION*), has been identified. Unlike in maize, single *rox* mutants have a very mild phenotype and reduce the number of axillary buds formed during vegetative development (Yang *et al.*, 2012). If and how the function of *ba1*/*ROX* genes intersects with auxin biology is currently unknown.

In rice, *OsPIN1* RNAi (RNA interference) lines mainly increase tiller angle, which is an important agronomic trait, since cultivated rice needs to grow upright to facilitate harvesting and high density growth (Xu *et al.*, 2005). Tiller angle

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in rice is also affected in other mutants such as the *lazy* mutant that was shown to increase polar auxin transport (Li *et al.*, 2007; Yoshihara and Iino, 2007). *LAZY* encodes a novel protein with no clear *Arabidopsis* homologue.

Another class of auxin efflux transporters is represented by the P-glycoproteins of the ABCB transporter family. In maize and sorghum, mutations in the *brachytic2* (*br2*) and *dwarf3* (*d3*) genes, respectively, have reduced auxin transport and cell elongation defects that result in shorter plants with smaller internodes (Multani *et al.*, 2003). *br2* and *d3* belong to the ABCB transporter family, similar to the *Arabidopsis ABCB1*/*PGP1* gene. In *Arabidopsis*, higher order *pgp* mutants give rise to a range of phenotypes. For example, *pgp1,pgp19* double mutants are shorter and display several vegetative and reproductive defects (Blakeslee *et al.*, 2007; Mravec *et al.*, 2008). In *Arabidopsis*, PGP proteins were reported to regulate auxin efflux coordinately with the PIN transporters (Blakeslee *et al.*, 2007; Mravec *et al.*, 2008).

Diffusion as well as active auxin transport are both mechanisms by which auxin can enter the cell. The best-characterized gene responsible for auxin uptake into the cell is *AUX1*, a member of the AUX1/LAX family of transporters, which consists of four members in *Arabidopsis*. Quadruple *aux1 lax1 lax2 lax3* mutants have major effects on shoot development and cause alterations in phyllotactic patterns (Bainbridge *et al.*, 2008). No functional analysis has yet been carried out on members of this family in maize and rice.

Most of the developmental plasticity of plants can be attributed to the regulation of axillary bud outgrowth. One of the major influences that auxin exerts on plant development that has long been studied is the phenomenon of apical dominance. It has long been known that auxin and its transport have an indirect inhibitory effect on bud outgrowth. The long search for the mechanisms by which auxin indirectly affects bud outgrowth has recently revealed a complex series of interactions involving various hormone pathways, and has led to the identification of a novel class of carotenoid-derived hormones, the strigolactones, that are involved in the regulation of shoot branching. Several highly tillered mutants that include *dwarf10*, *dwarf17*, and *dwarf27* in rice (Umehara *et al.*, 2008; Lin *et al.*, 2009) and *ramosus1* from pea (Gomez-Roldan *et al.*, 2008) were found to be deficient in the biosynthesis of strigolactones, and could be rescued by exogenous application of strigolactones. Another set of mutants affected in shoot branching, *dwarf3* and *ramosus4*, could not be rescued by exogenous strigolactone applications and were determined to be impaired in the strigolactone signalling pathway (Gomez-Roldan *et al.*, 2008; Umehara *et al.*, 2008). In *Arabidopsis*, genes involved in the biosynthesis and signalling of the strigolactone pathway belong to the *MAX* (*MORE AXILLARY MERISTEMS*) genes, and like their rice and pea counterparts were originally isolated as mutants exhibiting increased branching. A series of grafting experiments in *Arabidopsis max* mutants had previously suggested the existence of an inhibitory signal travelling from the root to the shoot to suppress axillary bud outgrowth (Leyser, 2003). Strigolactones were shown to reduce polar auxin transport, by decreasing the level of the auxin efflux transporter PIN1 at the plasma membrane in xylem parenchyma cells (Crawford *et al.*, 2010). Auxin is synthesized in young axillary buds, and its export is needed to establish directional transport in the future bud stem as postulated in the auxin transport canalization-based model (Scarpella *et al.*, 2006; Balla *et al.*, 2011). By dampening polar transport and therefore reducing the sink strength of the stem, strigolactones enhance the competition of various axillary buds for the export of auxin to the stem. The buds that activate first will export auxin and thus prevent other buds from activating. A comprehensive review on the current understanding of the interactions between auxin, strigolactones, and other hormones and their role in the control of shoot branching was recently published (Domagalska and Leyser, 2011).

Modern maize has been selected for strong apical dominance (Doebley *et al.*, 1997), and, accordingly, the majority of commonly used genetic backgrounds produce very few, if any, tillers. It is therefore less straightforward to assess the effect of auxin and other hormones in this process, and, as previously mentioned, auxin is also required for the initial formation of the axillary buds from which tillers develop. Nevertheless, a transposon insertion in the maize *CAROTENOID CLEAVAGE DIOXYGENASE8* (*CCD8*) gene, a component of the strigolactone biosynthesis pathway, produced mutants with mildly enhanced tillering. Furthermore, other pleiotropic phenotypes such as reduced plant and inflorescence size were also observed (Guan *et al.*, 2012). The *Arabidopsis CCD8* gene corresponds to one of the *MAX* genes (*MAX4*) (Sorefan *et al.*, 2003). Overexpression of the maize *CCD8* gene was capable of rescuing the reduced size and branching defects of the *Arabidopsis max4* mutant, suggesting that there is functional conservation in the strigolactone biosynthesis pathway between *Arabidopsis* and maize. One difference that was highlighted by Guan *et al.* is that in maize the expression of the *tb1* and *gt1* genes is unchanged in *Zmccd8* loss-of-function mutants. This contrasts with the current view that *BRC1*, the *Arabidopsis tb1* orthologue, acts downstream of strigolactone signalling to control branching (Aguilar-Martinez *et al.*, 2007). Together with genetic analysis, this evidence led the authors to conclude that in maize the activity of strigolactones is independent of the *tb1* pathway, though it cannot be completely ruled out that strigolactones are still made in *Zmccd8* mutants, hence the reported mild branching phenotype.

Auxin signalling

Our current knowledge of the auxin signalling pathway has greatly expanded in the last two decades, resulting in the establishment of a core model for auxin signal transduction (Chapman and Estelle, 2009). Auxin is perceived by co-receptor complexes formed by F-box TRANSPORT INHIBITOR RESPONSE1/AUXIN SIGNALING F-BOX (TIR1/AFB) and AUXIN/INDOLACETIC3 ACID (Aux/IAA) proteins (Dharmasiri *et al.*, 2005; Kepinski and Leyser, 2005). The stable interaction between auxin, TIR1/AFB, and Aux/IAA proteins triggers the ubiquitination of the transcriptionally

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repressive Aux/IAA proteins (Gray *et al.*, 2001; Tan *et al.*, 2007). Their subsequent degradation by the 26S proteasome ensures that the AUXIN RESPONSE FACTORS (ARFs), another class of transcriptional regulators which heterodimerize with the Aux/IAAs, can promote transcription of early auxin-responsive genes and therefore trigger a developmental response to auxin. The mechanism by which Aux/IAA repress downstream target genes entails physical interaction with a large transcriptional co-repressor called TOPLESS (TPL; Long *et al.*, 2006; Szemenyei *et al.*, 2008) that is believed to inhibit transcription by altering local chromatin structure (Long *et al.*, 2006; Krogan *et al.*, 2012; Wang *et al.*, 2013) (Fig. 2).

The *ARF* and *Aux/IAA* gene families each contain many members (23 and 29 members, respectively, in *Arabidopsis*), a feature which is shared by most flowering plants sequenced to date. The affinities of the auxin co-receptor complexes for auxin is largely determined by the Aux/IAA proteins, and the combinatorial interaction of TIR1/AFBs and Aux/IAAs may result in different sensitivities to auxin according to the dynamic range of the hormone concentration and to different developmental contexts (Dreher *et al.*, 2006; Villalobos *et al.*, 2012). In *Arabidopsis*, only a subset of the *ARF* genes appear to function as true transcriptional activators in the manner outlined above (Guilfoyle and Hagen, 2007). One of these is *ARF5* or *MONOPTEROS* (*MP*). *MP*/*ARF5* was originally identified as a recessive mutant with severe defects in embryogenesis. *mp* mutants are also unable to form lateral organs, resulting in the formation of a pin-like inflorescence (Przemeck *et al.*, 1996; Hardtke *et al.*, 2004). Interestingly the same defects are also observed in *MP*/*ARF5* overexpression lines, suggesting that auxin signalling in the shoot must be precisely balanced in order to promote organogenesis (Hardtke *et al.*, 2004). The absence of lateral organ formation in the inflorescence meristems of *mp* mutants cannot be rescued by localized auxin application (Reinhardt *et al.*, 2003), a finding which supports the role of *MP*/*ARF5* downstream of auxin transport. Although the auxin signalling pathway involving MP/ARF5 has been extensively detailed during embryogenesis (Weijers *et al.*, 2006; Schlereth *et al.*, 2010), it is unclear if similar mechanisms and players are also acting during post-embryonic development to regulate organogenesis at the flanks of the SAMs. ARFs that function as repressors have also been identified, but their contribution to the auxin signalling pathway is currently less clear, and their activity is seemingly regulated by auxin-independent mechanisms (Guilfoyle and Hagen, 2007; Paponov *et al.*, 2009; Vernoux *et al.*, 2011).

Dominant or semi-dominant mutants in different *Aux/IAA* genes in *Arabidopsis*, caused by mutations in the degron domains that affect their ability to interact with the TIR1/AFB receptor, are generally associated with various shoot developmental defects, such as decreased apical dominance, stem elongation defects, agravitropic hypocotyl growth, and upcurling of leaves (Reed, 2001). More recently, dominant mutations in *IAA18* were reported to disturb apical patterning in the embryo, post-embryonic leaf development, fertility, and stem elongation. These phenotypes could be suppressed by overexpressing MP/ARF5, suggesting an antagonistic interaction between IAA18 and MP proteins. Furthermore, PIN1–green fluorescent protein (GFP) localization was also perturbed in *iaa18-1* mutants, thus affecting the correct patterning of lateral organs (Ploense *et al.*, 2009).

TIR1/*AFB* proteins comprise a family of six members in *Arabidopsis*. *tir1* mutants are defective in auxin-mediated hypocotyl elongation (Ruegger *et al.*, 1998). When combined with *axr1* mutants, a synergistic interaction is observed in shoot development, and plants are smaller and diplay slightly reduced apical dominance. *AXR1* encodes an E1 ubiquitin-activating enzyme functioning with the $TIR1^{SCF}$ complex in ubiquitin-mediated protein degradation (Leyser *et al.*, 1993).

Some well-known early auxin-responsive genes include *GH3*, *SAUR* genes, and the *Aux/IAA* genes themselves. *GH3* and *Aux/IAA* genes have been previously discussed, and a few recent reports have begun to shed light on the function of SAURs and their role in plant development. SAURs (SMALL AUXIN UPREGULATED PROTEINS) are small proteins that, as suggested by their name, are up-regulated shortly after auxin induction. Recent studies have highlighted their role in internode and stem elongation in *Arabidopsis*. When fused to GFP, but not to a shorter epitope tag such as hemagglutinin (HA), SAURs cause elongation of stems and twisted growth. Subcellular localization of these fusion proteins seems to indicate that they mainly localize at the plasma membrane. Plants expressing SAUR–GFP proteins have higher basipetal auxin transport, indicating that they may directly or indirectly affect auxin transport. Loss-of-function artificial microRNA lines show shorter hypocotyls and stamen filaments, suggesting that *SAUR* genes promote auxin-induced growth (Chae *et al.*, 2012).

In maize, very few mutants affected in known components of the auxin signalling pathway have been reported so far. The maize genome encodes anywhere from 31 to 36 ARFs (Xing *et al.*, 2011; Wang *et al.*, 2012), 31 Aux/IAAs (Wang *et al.*, 2010), four TIR1/AFBs, and four TPL/TPL-LIKEs (auxinevodevo.org). Recently a maize mutant in an *Aux/IAA* gene, called *rootless with undetectable meristem1*, was reported and shown to reduce lateral root development, but no alterations in shoot architecture were reported (von Behrens *et al.*, 2011). The maize orthologue of the transcriptional co-repressor *TOPLESS*, called *ramosa1 enhancer locus2* (*rel2*), was recently identified by a mutagenesis screen designed to investigate the regulation of meristem determinacy in maize inflorescences (Gallavotti *et al.*, 2010). The reported phenotype of single *rel2* mutants is mild, mainly affecting the angle at which tassel branches are borne on the central spike, but the severity of the mutant phenotype is strictly background dependent (AG, unpublished). The contribution of *rel2* to the auxin signalling pathway has not yet been investigated, but, given the high degree of homology between REL2 and TPL, REL2 likely directs transcriptional repression of maize Aux/IAAs in a manner similar to its *Arabidopsis* homologues.

Functional characterization of auxin signalling pathway genes in grasses has been pursued mainly in rice, and

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mostly by transgenic approaches. The rice genome encodes 31 OsAux/IAA and 25 OsARF proteins (Wang *et al.*, 2007). A stabilized version of *OsIAA3* expressed by an inducible system affects the overall growth of rice plants and, in particular, causes reduced leaf and stem size, as well as root defects (Nakamura *et al.*, 2006). Similarly, overexpression of *OsIAA1* causes a significant reduction in plant height, due to the development of shorter internodes (Song *et al.*, 2009). In rice, overexpression of *OsSAUR39* causes altered shoot morphology and reduced plant growth. Auxin levels and polar transport are also reduced in these lines, and these defects can be rescued by exogenous auxin application. This implies that *OsSAUR39* functions as a negative regulator of auxin synthesis and transport (Kant *et al.*, 2009).

Auxin and axillary meristem initiation

The main driver of body plan diversification is represented by the formation and activity of axillary meristems, processes that as described above are highly connected to auxin biology. Auxin biosynthesis, transport, and signalling are all required for axillary meristem initiation, as demonstrated by different mutants defective in these pathways in various species, i.e. *yuc*, *pin1*, and *mp* in *Arabidopsis*, and *spi1*, *bif2* in maize, that fail to form new meristems. Whereas axillary meristem outgrowth has received much attention in the past few years with the discoveries of the complex interaction among auxin, strigolactones, and cytokinins (Domagalska and Leyser, 2011), still relatively little is known about how auxin promotes the formation of new axillary meristems, despite the large number of characterized mutants that are affected in their initiation. Axillary meristem initiation can thereby serve as a model example to study how auxin pathways interconnect among themselves and with other developmental pathways.

Axillary meristem initiation involves the correct positioning of the meristem anlagen, followed by the delineation of the boundaries for primordium separation and the establishment of the meristem proper. Computational and modelling evidence suggests that the regular arrangement of primordia around the main SAM is the result of dynamic interactions between existing and incipient primordia mediated by the activity of the auxin efflux transporter PIN1 (de Reuille *et al.*, 2006; Jonsson *et al.*, 2006; Smith *et al.*, 2006). It is notable though that auxin biosynthesis, visualized by the expression patterns of different *YUCCA-like* genes in different plant species, is taking place in very specific domains of the apical meristems (Tobena-Santamaria *et al.*, 2002; Cheng *et al.*, 2006; Gallavotti *et al.*, 2008*a*). Genetic evidence also points to a crucial role for auxin biosynthesis in the formation of new axillary meristems. It is therefore likely that auxin biosynthesis acts in concert with the activity of auxin transporters in this process. Synergistic interactions between auxin transport and biosynthetic mutants have been reported in both maize and *Arabidopsis*. In *spi1;bif2* and *vt2;bif2* mutants, plants are severely reduced in size and the tassels are completely lacking branches and spikelets (Gallavotti *et al.*, 2008*a*; Phillips *et al.*, 2011). Severe defects in organogenesis are also observed in the *Arabidopsis yuc1;yuc4;pid* and *yuc1;yuc4;pin1* triple mutants and *yuc1;yuc2;yuc4;yuc6;aux1* quintuple mutant (Cheng *et al.*, 2007, 2008). In *Arabidopsis*, genetic screens aimed at identifying enhancers or suppressors of *yuc1;yuc4* and *pid* mutants led to the identification of the NPY/MAB/ENP family of BTB-NPH3-like proteins that enhance both *yuc1yuc4* and *pid* phenotypes (Cheng *et al.*, 2007; Furutani *et al.*, 2007). Recently it was reported that several members of this family function redundantly to regulate the polarity and endocytosis of PIN proteins (Furutani *et al.*, 2011). In maize, similar screens on *spi1*, *vt2*, and *bif2* may lead to the discovery of new genes that could help clarify the relative contributions and possible molecular connections between auxin biosynthesis and transport. Since the level of redundancy in auxin pathways clearly differs in different plant species (see, for example, *spi1* and *YUC* genes, or *PIN* genes in maize and *Arabidopsis*), it is crucial to carry out such screens in both monocot and dicot species as they may lead to different gene discoveries.

In *Arabidopsis*, live confocal imaging of multiple fluorescent transcriptional and translational reporters revealed the dynamic role of various genetic factors involved in the earliest events of floral meristem formation. At first, up-regulation of PIN1 protein expression is observed. PIN1 is polarized towards the newly forming primordium, and concomitantly auxin-induced transcription occurs at sites where primordia will form, marked by the expression of the synthetic reporter *DR5-VENUS*. Subsequently, PIN1 polarity is reversed and auxin is thought to be transported away from the developing primordium to initiate newer primordia. At this stage, the expression of organ boundary genes, such as *CUP SHAPED COTYLEDON2* (*CUC2*), becomes apparent. Organ polarity is established by the concerted activity of abaxial (*FILAMENTOUS FLOWER*; *FIL*) and adaxial (*REVOLUTA*; *REV*) determinants, and, as the primordium grows, the two domains are pushed apart from the cell proliferation that characterizes the establishment of the new meristem. Finally, the expression of *LEAFY* (*LFY*), a transcription factor that specifies floral meristem identity, is established (Heisler *et al.*, 2005) (Fig. 4A).

In maize, axillary meristem initiation during inflorescence development has received much attention in recent years with the isolation of a series of mutants affected in their formation. Nonetheless, a high resolution analysis such as that described above for *Arabidopsis* has not yet been achieved, despite the availability of auxin transport and response reporter lines (Gallavotti *et al.*, 2008*b*). This is primarily due to inherent difficulties in carrying out live imaging of maize inflorescences, since at early stages of development inflorescences are not exposed but are surrounded by leaf tissue. Nonetheless, these lines have shown that similar up-regulations of ZmPIN1a as well as expression of the *DR5-RFP* reporter are observed at the flanks of the inflorescence meristem where the newly developing primordia will form (Gallavotti *et al.*, 2008*b*) (Fig. 4A). The analysis of auxin transport dynamics during axillary meristem initiation at the flanks of the inflorescence meristem is complicated by the concurrent formation

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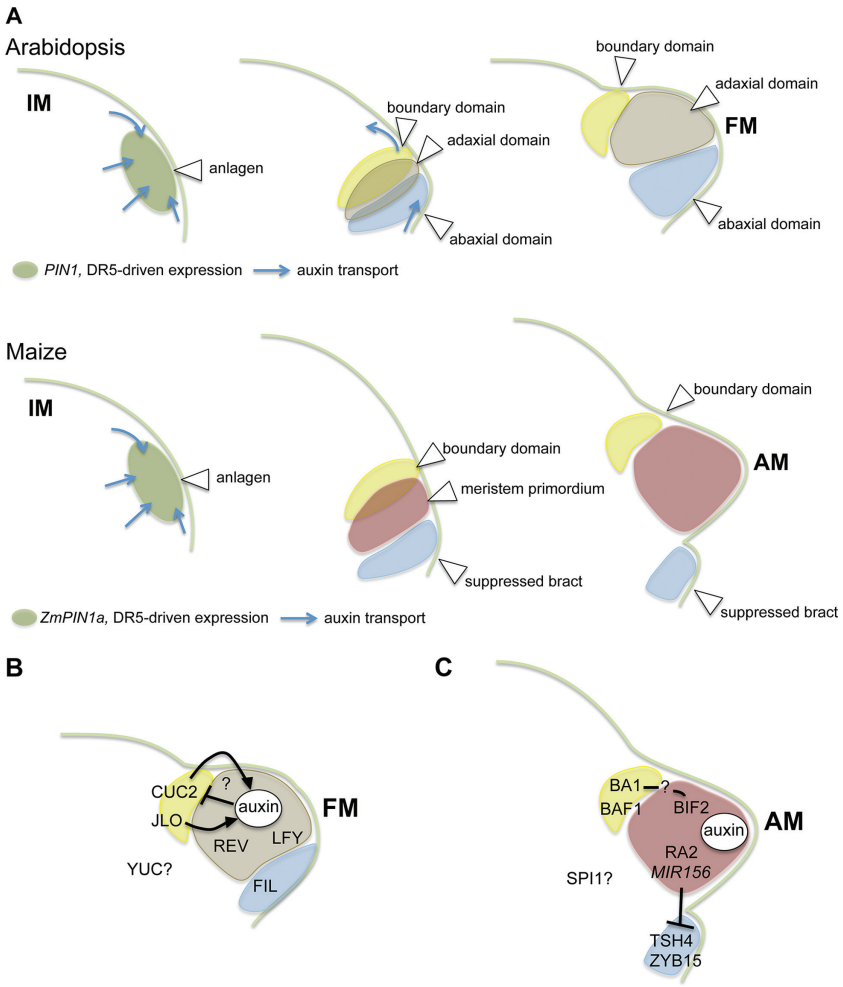

Fig. 4. Schematic representation of axillary meristem initiation in both *Arabidopsis* and maize. Based on data from Heisler *et al.* (2005), Rast and Simon (2012), Bilsborough *et al.* (2011), Chuck *et al.* (2010), and Gallavotti *et al.* (2008*b*, 2011). (A) Schematic steps involved in floral meristem formation and axillary meristem formation in *Arabidopsis* and maize, respectively. (B, C) Genes involved in axillary meristem initiation discussed in the text for *Arabidopsis* (B) and maize (C). *zyb15* is the maize orthologue of *FIL*. IM, inflorescence meristem; FM, floral meristem; AM, axillary meristem.

of visible suppressed bracts. In *Arabidopsis*, cryptic bracts subtend axillary meristems and show expression of the *FIL* gene, but remain morphologically indistinguishable (Siegfried *et al.*, 1999; Dinneny *et al.*, 2004). Suppressed bract formation is also regulated by auxin transport and biosynthesis (Gallavotti *et al.*, 2008*a*, *b*; Phillips *et al.*, 2011). In immature *ba1* mutant tassels, where the only primordia formed consist of suppressed bracts (Fig. 3B), treatments with NPA indeed block their formation, and the tassels have a smoother surface (Gallavotti *et al.*, 2008*a*). Similarly, *spi1* and *vt2* tassels do not show the formation of suppressed bracts on the main inflorescence axis. Mutants where the suppressed bracts are no longer suppressed have been recently isolated, and have provided new insights into the axillary meristem initiation pathway.

The *tassel sheath (tsh)* mutants show the formation of leaves at the base of tassel branches and of spikelet pairs (Whipple *et al.*, 2010). These leaves are the results of the outgrowth of the suppressed bracts. This outgrowth generally occurs at the expense of tassel branch formation, such that fewer long branches are indeed observed in *tsh1-5* mutants (Whipple *et al.*, 2010). Based on phenotypic characterization and careful expression analysis of one of the TSH proteins, TSH4, an SBP-box transcription factor, a model was proposed in which the anlagen at the flanks of the inflorescence meristem is marked by co-expression of both boundary genes and TSH4, but, as the primordium develops, separate domains emerge. One domain corresponds to the suppressed bract domain, and the other one to the spikelet-pair meristem, the first type of determinate axillary meristem formed in maize inflorescences (Chuck *et al.*, 2010). Competition between the two domains is regulated by the antagonistic relationship between meristem and suppressed bract determinants. This may be accomplished by the activity of a microRNA, miRNA156, that restricts the expression of TSH4 protein to the suppressed bract domain, and by the antagonistic interactions between *tsh4* and RAMOSA2 (RA2), a LATERAL ORGAN BOUNDARY (LOB) transcription factor that is required for spikelet-pair meristem identity (Bortiri *et al.*, 2006) (Fig. 4C). In support of

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this model is the observation that in *ba1* tassels the suppressed bracts seem indeed enlarged relative to those present in normal tassels (Ritter *et al.*, 2002; Gallavotti *et al.*, 2008*b*). The rice orthologue of *ba1*, *LAX (LAX PANICLE)*, was shown to be required for maintaining an initial cell proliferation event for the establishment of a new axillary meristem (Oikawa *et al.*, 2009). If *ba1* behaves similarly, it is possible that the initial cell proliferation is incorporated in the newly developing suppressed bract primordia and that may be the reason why these bracts appear larger than normal. Both *ba1* and *LAX* have a very distinct expression pattern and their transcripts are localized in a very narrow boundary domain adaxial to newly forming meristems. Another interesting aspect reported in rice is that the LAX protein seems to move from the boundary domain into the developing meristem, and this movement may be required for its function (Oikawa *et al.*, 2009). Given the orthology relationship between *LAX* and *ba1*, it is likely that the BA1 protein also moves from the place of its transcription into the newly forming meristem, though this has yet to be reported. Since BA1 may be phosphorylated by BIF2 (Skirpan *et al.*, 2008), and *bif2* is expressed in the meristem rather than in the boundary domain (McSteen *et al.*, 2007), the phosphorylation of BA1 may be required to prevent its function in the meristem proper. Testing how BA1 proteins behave in a *bif2* mutant is crucial to investigate this hypothesis. Other possible links between auxin transport and signalling with the genes mentioned above are still to be identified.

Of pivotal importance for axillary meristem initiation is the establishment of a boundary domain that separates the development of adjacent organs. In both *Arabidopsis* and maize, several genes with a striking localized boundary domain expression pattern have been reported, and loss-of-function mutations in the majority of these genes lead to organ fusion and axillary meristem initiation defects. Among these are the already mentioned *CUC* genes as well as *RAX1*, *JLO*, *LOF*, and *LOB* genes in *Arabidopsis*, and *ba1* and *baf1* in maize (Hibara *et al.*, 2006; Keller *et al.*, 2006; Borghi *et al.*, 2007; Lee *et al.*, 2009; Bell *et al.*, 2012; Gallavotti *et al.*, 2004, 2011). There is an intimate relationship between organ boundary formation and auxin transport, and some of the boundary expressed genes have been shown to be involved in the regulation of PIN protein expression. Among these, *JAGGED LATERAL ORGANS* (*JLO*), a gene encoding a LOB domain transcription factor, was recently shown to function in the regulation of PIN efflux carriers during both shoot and root development in conjunction with *ASYMMETRIC2*, another LOB-domain transcription factor encoding gene (Rast and Simon, 2012). The relationship between the *CUC* genes and auxin transport was also recently investigated during the development of leaf margins in *Arabidopsis*, and a computational model was proposed in which a feedback loop exists between auxin and *CUC2* expression, where *CUC2* promotes the generation of auxin response maxima by the activity of PIN1 proteins, which in turn repress CUC2 activity (Bilsborough *et al.*, 2011). Whether a similar interaction between auxin transport and *CUC2* may also occur during organogenesis at the SAM has yet to be determined, but it provides an intriguing scenario in which auxin excludes *CUC2* from the meristem proper and by doing so delineates a boundary domain of low growth (Fig. 4B). Auxin is not the only hormone implicated in the formation of boundary domains. Recently the brassinosteroid-activated transcription factor BZR1 was shown to regulate *CUC* genes negatively in the meristem centre, and, conversely, the LOB transcription factor was shown to regulate brassinosteroid accumulation negatively in the boundary domain (Bell *et al.*, 2012; Gendron *et al.*, 2012). A complex picture of interactions occurring between high growth regions such as meristems and primordia, and the low growth boundary domain regions, involving auxin, brassinosteroids, and a plethora of transcriptional regulators, is therefore emerging.

Conclusions and future challenges

As highlighted with several examples, many connections exist between auxin function and the regulation of plant architecture. Given the key role that auxin plays in the patterning and formation of lateral organs, stem elongation, and the regulation of branching, it has been suggested that auxin is crucial to the evolution of different plant architectures (Finet and Jaillais, 2012). We have now identified a number of major players in auxin function, and a concerted effort is needed to establish the functional conservation or divergence of the various auxin pathways in both eudicot and monocot species with different plant and inflorescence architectures.

The data gathered so far predominantly point to a functional conservation in auxin biosynthesis, homeostasis, transport, and signalling pathways between dicot and monocot species. Nonetheless, it is commonly known that monocotyledonous and dicotyledonous species respond differently to auxin-based herbicides, and this selectivity may lie in the metabolism and perception of auxin herbicides in different plant species (Grossmann, 2010). Since architectural differences are observed in monocot and dicot species, it is tempting to speculate that they may be attributed to specific variations existing in the common pathways described above. For example, considering the expansion of transcriptional regulator families such as ARFs and Aux/IAAs during plant evolution (Paponov *et al.*, 2009), it is possible that changes in the expression levels and/or interaction profiles of these various proteins are major contributors to differences in developmental outcomes. Furthermore, as recently shown, Aux/IAA and TIR co-receptors have different affinities depending on the type of auxin and the Aux/IAA involved (Villalobos *et al.*, 2012), and Aux/IAA proteins also have very different half-lives (Dreher *et al.*, 2006). Genes involved in auxin biosynthesis and homestasis may be expressed at different levels in specific tissues, and this would also contribute to changes in local auxin concentrations. All these levels of regulation of auxin function have the potential to determine a differential response to specific auxin stimuli, depending on the concentration of auxin in a specific cell type or the presence of a specific Aux/IAA protein (Vernoux *et al.*, 2011).

Unfortunately, functional analyses of genes involved in auxin metabolism and perception in monocot species are not abundant. One way to approach the gap in our knowledge of certain species is to intensify the search for new mutants

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affected in auxin function. In maize, for example, there are still many developmental mutants with auxin-related defects that await further characterization; for example *Dvd1*, *sem1*, and *Bif1* (Scanlon *et al.*, 2002; Barazesh and McSteen, 2008; Phillips *et al.*, 2009) (auxinevodevo.org). Also reverse genetic approaches need to be systematically pursued in maize and rice for some of the gene families involved in auxin biology. To this end, it must also to be kept in mind that it may be difficult to assess if differences exist among different species because of the problem of redundancy in the majority of the gene families involved. Only when functional characterization is carried out for most of these genes in maize, rice, or other species can we start answering the question of how auxin influences the establishment and evolution of different body plans.

Recent examples in rice and maize showed how genes involved in inflorescence architecture can be used to improve crop yield (Jiao *et al.*, 2010; Miura *et al.*, 2010; Bommert *et al.*, 2013). As demand for biomass increases, it is crucial to devise strategies for bioenergy crops that can allow growth at higher density by, for example, increasing tillering via modulation of genes controlling bud outgrowth (Whipple *et al.*, 2011). In *Arabidopsis*, recent research has started to link environmental stimuli mechanistically with corresponding changes in growth and development. Since a basic understanding of the major regulators in the function of auxin and other plant hormones has been achieved (Santner *et al.*, 2009), we now have the tools to pursue this exciting new research avenue that has major implications for applied crop science, especially considering the challenges that agriculture has begun to face with the increasing occurrence of drastic changes in the environment. Understanding how plants interpret environmental cues to adapt their growth is a major challenge that plant scientists will hopefully be able to tackle in the coming years.

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