Auxin and Monocot Development

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Monocots are known to respond differently to auxinic herbicides; hence, certain herbicides kill broadleaf (i.e., dicot) weeds while leaving lawns (i.e., monocot grasses) intact. In addition, the characters that distinguish monocots from dicots involve structures whose development is controlled by auxin. However, the molecular mechanisms controlling auxin biosynthesis, homeostasis, transport, and signal transduction appear, so far, to be conserved between monocots and dicots, although there are differences in gene copy number and expression leading to diversification in function. This article provides an update on the conservation and diversification of the roles of genes controlling auxin biosynthesis, transport, and signal transduction in root, shoot, and reproductive development in rice and maize.

Auxinic herbicides have been used for decades to control dicot weeds in domestic lawns (Fig. 1A), commercial golf courses, and acres of corn, wheat, and barley, yet it is not understand how auxinic herbicides selectively kill dicots and spare monocots (Grossmann 2000; Kelley and Reichers 2007). Monocots, in particular grasses, must perceive or respond differently to exogenous synthetic auxin than dicots. It has been proposed that this selectivity is because of either limited translocation or rapid degradation of exogenous auxin (Gauvrit and Gaillardon 1991; Monaco et al. 2002), altered vascular anatomy (Monaco et al. 2002), or altered perception of auxin in monocots (Kelley and Reichers 2007). To explain these differences, there is a need to further understand the molecular basis of auxin metabolism, transport, and signaling in monocots.

Auxin, as we have seen in previous articles, plays a major role in vegetative, reproductive, and root development in the model dicot, *Arabidopsis*. However, monocots have a very different anatomy from dicots (Raven et al. 2005). Many of the characters that distinguish monocots and dicots involve structures whose development is controlled by auxin: (1) As the name implies, monocots have single cotyledons, whereas dicots have two cotyledons (Fig. 1B,C). Auxin transport during embryogenesis may play a role in this difference as cotyledon number defects are often seen in auxin transport mutants (reviewed in Chandler 2008). (2) The vasculature in leaves of dicots is reticulate, whereas the vasculature in monocots is parallel (Fig. 1). Auxin functions in vascular development because many mutants defective in auxin transport, biosynthesis, or signaling have vasculature defects

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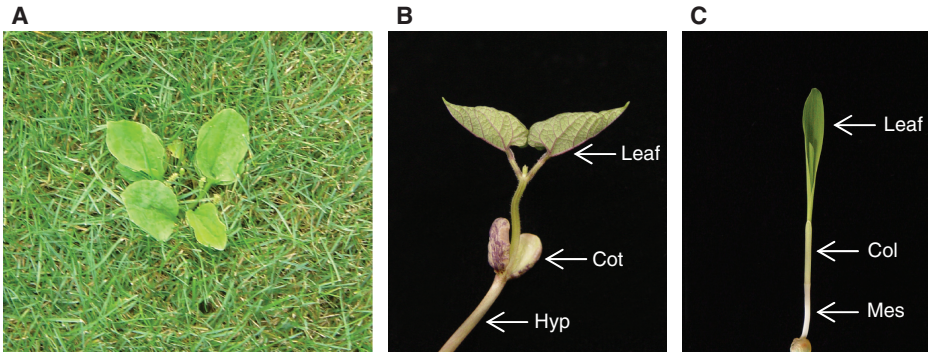

Figure 1. Differences between monocots and dicots. (*A*) A dicot weed in a lawn of grasses. Note the difference in morphology of the leaves. (*B*) Germinating dicot (bean) seedling. Dicots have two cotyledons (cot). Reticulate venation is apparent in the leaves. The stem below the cotyledons is called the hypocotyl (hyp). (*C*) Germinating monocot (maize) seedling. Monocots have a single cotyledon called the coleoptile (col) in grasses. Parallel venation is apparent in the leaves. The stem below the coleoptile is called the mesocotyl (mes).

(Scarpella and Meijer 2004). (3) Dicots often produce a primary tap root that produces lateral roots, whereas, in monocots, especially grasses, shoot-borne adventitious roots are the most prominent component of the root system leading to the characteristic fibrous root system (Fig. 2). Auxin induces lateral-root formation in dicots and adventitious root formation in grasses (Hochholdinger and Zimmermann 2008).

It is not yet clear if auxin controls the differences in morphology seen in dicots versus monocots. However, both conservation and diversification of mechanisms of auxin biosynthesis, homeostasis, transport, and signal transduction have been discovered so far. This article highlights the similarities and the differences in the role of auxin in monocots compared with dicots. First, the genes in each of the pathways are introduced (Part I, Table I) and then the function of these genes in development is discussed with examples from the monocot grasses, maize, and rice (Part II).

PART I—GENES CONTROLLING AUXIN BIOSYNTHESIS, TRANSPORT, AND SIGNAL TRANSDUCTION IN GRASSES

Auxin Biosynthesis

Maize has long been a model system for studies on the biochemistry of auxin biosynthesis (Kriechbaumer et al. 2006). Genes encoding enzymes required for the synthesis of auxin have been identified in maize, rice, and *Arabidopsis* (Table 1). Multiple pathways have been proposed, but the enzymes, genes that encode them, and intermediates are not fully known (Bartel 1997; Ljung et al. 2002; Cohen et al. 2003; Woodward and Bartel 2005). Recent advances have identified mutants and corresponding genes, which has led to revised pathways that still need to be defined further (Zhao et al. 2001; Zhao et al. 2002; Stepanova et al. 2008; Tao et al. 2008; Sugawara et al. 2009). Four tryptophan-dependent and one tryptophan-independent auxin biosynthetic pathways have been proposed. The revised tryptophan-dependent pathways proposed by Sugawara et al. (2009) will be used as a framework to introduce what is known of the genes controlling the pathway in the monocots, maize, and rice.

Tryptophan-dependent Auxin Biosynthesis

(1) IAM Pathway

Genes controlling the conversion of tryptophan (TRP) to indole-3-acetamide (IAM) have not been detected in plants, although in bacteria the *iaaM* gene catalyzes this reaction (Comai and Kosuge 1982). The conversion of IAM to indole-3-acetic acid (IAA) is proposed to occur

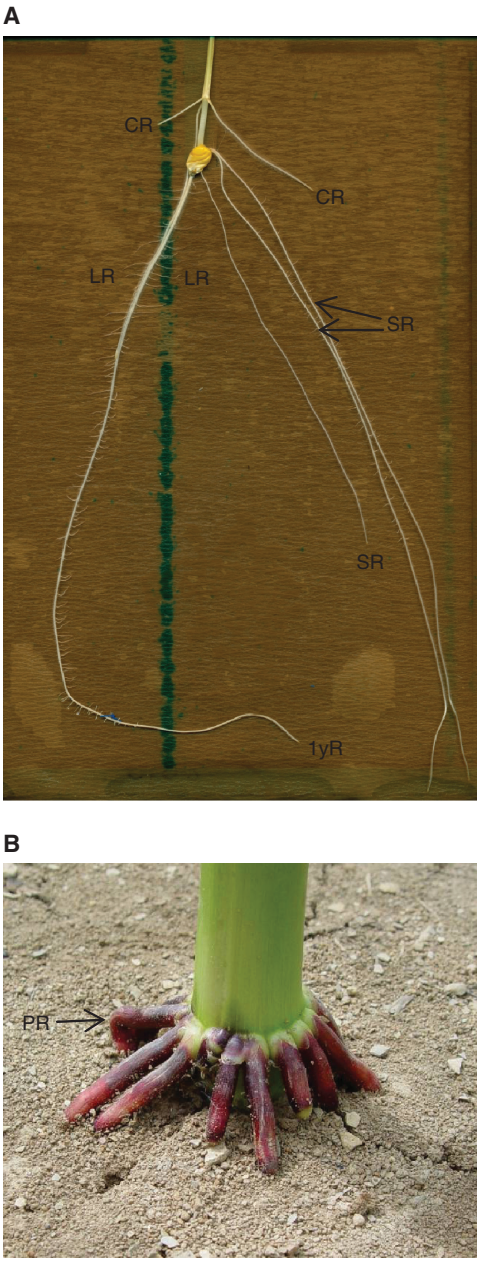

Figure 2. The root system in monocots. (*A*) Maize seedling showing the primary root (1yR), which has many lateral roots (LR). The seminal roots (SR) are a type of adventitious root produced during embryonic development. Crown roots (CR) are produced from stem tissue. (*B*) The base of a maize plant showing prop roots (PR), which are adventitious roots produced from basal nodes of the stem later in development.

through the action of the amidase gene, *AMI1*, in *Arabidopsis* (Pollmann et al. 2003). Amidase genes have not yet been reported in monocots, though an IAM hydrolase activity has been detected from rice callus, so it is likely that this pathway is used in grasses (Arai et al. 2004).

(2) IPA Pathway

There is evidence that the indole-3-pyruvic acid (IPA) pathway is important in both monocot and dicot development. The conversion of TRP to IPA occurs through the action of the tryptophan aminotransferase genes *TAA1*, *TAR1*, and *TAR2,* which play a role in embryogenesis, root, vascular, and inflorescence development in *Arabidopsis* (Stepanova et al. 2008; Tao et al. 2008). *TAA1*-like genes from monocots have not yet been reported but the cloning of the *vanishing tassel2 (vt2)* mutant in maize provides evidence that this enzyme also functions in vegetative and inflorescence development in monocots (K. Phillips and P. McSteen, unpubl.). It has been proposed that IPA is converted to indole-3-acetaldehyde (IAAld) by an IPA decarboxylase activity although the gene encoding this enzyme has not been definitely identified in dicots or monocots (Woodward and Bartel 2005). The conversion of IAAld to IAA has been proposed to be catalyzed by an aldehyde oxidase encoded by the *AAO1* gene of *Arabidopsis* (Sekimoto et al. 1998). A rice homolog, *OsAO1* (Yamamoto et al. 2007) and maize homologs, *ZmAO-1* and *ZmAO-2* (Sekimoto et al. 1997), have been reported but not functionally characterized. However, from the phenotype of the *taa1* and *vt2* mutants, it is apparent that this pathway significantly contributes to vegetative and reproductive development in both dicots and monocots.

(3) TAM Pathway

There is good evidence that the tryptamine (TAM) pathway plays a significant role in development of both monocots and dicots. It is not known which genes convert TRP to TAM although it is thought to occur by a tryptamine decarboxylase activity (Woodward and Bartel

Table 1. *Arabidopsis* genes required for auxin biosynthesis, transport, and signal transduction, and their homologs that have been functionally characterized in maize and rice

Protein	*Arabidopsis*	Maize	Rice	Maize/Rice References
Biosynthesis				
Tryptophan synthase β	*TRP2*	*ORP1,2*		Wright et al. 1991
Amidase	*AMI1*			
Trytophan aminotransferase	*TAA1 TAR1, 2*	*VT2*		K. Phillips and P. McSteen, unpublished
Aldehyde oxidase	*AAO1*	*ZmAO1*	*OsAO1*	Sekimoto et al. 1997
Flavin monoxygenase	*YUC1-11*	*SPI1*	*OsYUC1* *COW1/NAL7*	Fujino et al. 2007; Woo et al. 2007; Yamamoto et al. 2007; Gallavotti et al. 2008a
Cytochrome P450	*CYP79B2/3*			
Nitrilase	*NIT1-4*	*ZmNIT1,2*	*OsNIT*	Park et al. 2003; Kriechbaumer et al. 2007
Transport				
Auxin influx transporter	*AUX1*	*ZmAUX1*		Hochholdinger et al. 2000; Brooks et al. 2009
Auxin efflux carrier	*PIN1*	*ZmPIN1a* *ZmPIN1b* *ZmPIN1c*	*OsPIN1*	Xu et al. 2005; Carraro et al. 2006; Gallavotti et al. 2008b
Serine threonine kinase	*PID*	*BIF2*	*OsPID/OsBIF2*	McSteen et al. 2007; Morita and Kyozuka 2007
ABC transporter	*ABCB1,19*	*BR2*		Multani et al. 2003
			RCN1	Yasuno et al. 2009
ARF-GAP	*VAN3/SFC*	*ZmSFC*	*OsAGAP*	Zhuang et al. 2006; Zhang et al. 2007
Signal transduction				
Aux/IAA transcription factor	*IAA1-25*	*RUM1*	*IAA1* *IAA1/3* *IAA3/31*	Thakur et al. 2001; Nakamura et al. 2006; Song et al. 2009b
Auxin response factor	*ARF1-23*	*ZmARF1* *ZmARF2* *ZmMP*	*OsARF1*	Waller et al. 2002; Attia et al. 2009; Brooks et al. 2009
F box	*TIR1, AFB*	*ZmTIR1*		Zhang et al. 2007
Other				
Auxin binding protein	*AtABP1*	*ZmABP1,4*		Im et al. 2000
SAUR	*SAUR*	*ZmSAUR2*	*OsSAUR*	Knauss et al. 2003; Jain et al. 2006c
LOB transcription factor	*LBD16/29*	*RTCS*	*ARL1/CRL1*	Inukai et al. 2005; Liu et al. 2005; Taramino et al. 2007
BHLH transcription factor		*BA1*	*LAX1*	Komatsu et al. 2003; Gallavotti et al. 2004

2005). The *YUCCA* (*YUC*) genes catalyze the rate-limiting step in the pathway by the conversion of TAM to *N*-hydroxyl tryptamine (HTAM) (Zhao et al. 2001). It is not known how HTAM is converted to IAA, though a recent report provides evidence that this does not occur through an indole-3-acetaldoximine (IAOx) intermediate (Sugawara et al. 2009).

There are a total of 10 *YUC*-like genes in *Arabidopsis* (Zhao et al. 2001). Each of the genes is expressed in a specific pattern and is proposed to function redundantly to produce auxin in a localized manner during embryogenesis, leaf, vascular, and inflorescence development (Zhao et al. 2001; Cheng et al. 2006; Cheng et al. 2007a) There are 13 *YUC*-like genes in

rice, although functional information has been reported for only two of them. Seven *OsYUC* genes with similarity to *AtYUC1-8* were reported by (Yamamoto et al. 2007) and another gene, *OsYUC8/COW1/NAL7*, was subsequently reported (Fujino et al. 2007; Woo et al. 2007). Five additional genes that are more closely related to *AtYUC10* and *11* were included in a phylogenetic analysis reported in Gallavotti et al. 2008a. There is proposed to have been expansion, subfunctionalization, and diversification of this gene family in monocots and dicots (Gallavotti et al. 2008a).

The rice gene most closely related to *AtYUC1* and *4* is *OsYUC1* (Yamamoto et al. 2007). Overexpression and antisense inhibition of *OsYUC1* causes opposite defects in root development (described in Part II) (Yamamoto et al. 2007). The ortholog of *OsYUC1* based on sequence, phylogenetic analysis, and synteny is the *SPARSE INFLORESCENCE1* (*SPI1*) gene of maize, yet loss of function of this gene causes defects in vegetative and reproductive development (Gallavotti et al. 2008a), indicating that there has been diversification of gene function even within the grasses. *OsYUC1* and *SPI1* belong to a monocot-specific rather than grass-specific clade as evidenced by the isolation of a *SPI1* homolog from the monocot *Joinvillea* (Gallavotti et al. 2008a). A very similar phenotype to *spi1* is seen in *Arabidopsis* plants with loss of function of four *YUC* genes, indicating that the *YUC* family is more redundant in *Arabidopsis* than in maize (Cheng et al. 2006). Although the basic biochemical function of the *YUC* genes may be conserved, the biological function of these genes appears to have diversified due to changes in copy number and expression pattern (Gallavotti et al. 2008a).

A gene more distantly related to *AtYUC1* and *4* has been functionally characterized in rice. *OsYUC8/COW1/NAL7* is sister to the *Arabidopsis* genes in clade *AtYUC1-9* and clade *AtYUC10-11* (Gallavotti et al. 2008a). *constitutively wilted1 (cow1)* mutants have defects in root mass, leading to leaf rolling and the wilty phenotype after which the mutant is named (Woo et al. 2007). The same gene was identified as a natural variant, *narrow leaf7 (nal7)*, with narrow leaves (Fujino et al. 2007). More research is needed to uncover the functions of the other monocot *YUC*-like genes and to determine if they have additional functions masked by redundancy as seen for the *Arabidopsis yuc* mutants.

(4) IAOX Pathway

The indole-3-acetaldoximine (IAOx) pathway has been proposed to occur only in cruciferous species such as *Arabidopsis*, in which glucosinolate secondary metabolism occurs (Sugawara et al. 2009). However, there is conflicting evidence suggesting that this pathway may also occur in monocots (Kriechbaumer et al. 2006; Sugawara et al. 2009). TRP is converted to IAOx by genes encoding the cytochrome P450 enzymes CYP79B2 and CYP79B3 in *Arabidopsis* (Zhao et al. 2002). However, no orthologs of CYP79B2 and CYP79B3 have been found in noncruciferous species, including maize and rice, raising doubts as to whether this pathway occurs in other species. In support of this, Sugawara et al. (2009) were unable to detect IAOx in seedlings of maize, rice, or tobacco. In the next step of the proposed pathway, enzymes catalyzing the conversion of IAOx to indole-3-acetonitrile (IAN) are not known, but conversion of IAN to IAA occurs through the action of nitrilase genes in *Arabidopsis* (Pollmann et al. 2006). Nitrilase genes have also been well characterized in maize. For example, *ZmNIT2* hydrolyses IAN to IAA in maize kernels and seedlings (Park et al. 2003; Kriechbaumer et al. 2007). Genes similar to *ZmNIT2* have been reported but not characterized in rice (Yamamoto et al. 2007).

These results lead to the question of how IAN is produced in maize if there are no CYP79B2/3 genes and no detectable IAOx. On the other hand, enzyme activity that converts TRP to IAOx and converts IAOx to IAN and IAAld has been detected in maize tissues (reviewed in Kriechbaumer et al. 2006). Therefore, IAOx must be made by a different pathway in maize, although genes catalyzing this process have yet to be identified. Hence, although CYP79B2/3

genes are not present in monocots, a pathway related to the IAOx pathway may occur.

Tryptophan-independent Auxin Biosynthesis

Studies of the *orange pericarp* mutant, which fails to make tryptophan (because of a deficiency in tryptophan synthase β, which converts indole to tryptophan) but still makes auxin, provided the first evidence that tryptophan-independent biosynthesis occurs in maize (Wright et al. 1991). Studies of analogous mutants provided evidence that tryptophan-independent auxin biosynthesis also occurs in *Arabidopsis* (Normanly et al. 1993). When auxin is not made from tryptophan it is thought to be made via indole-3-glycerol phosphate or indole, although genes in the pathway have not been identified from monocots or dicots (Woodward and Bartel 2005).

In the future, it will be of interest not just to identity the genes in the entire biosynthetic pathway but also to understand why there are multiple pathways for auxin biosynthesis. Are different pathways used in different cells at different times in response to different conditions? Are different pathways used more in some species that others?

Auxin Homeostasis

Much of the auxin found in the plant is stored as conjugates to amino acids or sugars (Woodward and Bartel 2005). Auxin can also be produced by β oxidation of indole-3-butyric acid (IBA). These processes are thought to occur in monocots and some genes controlling the process have been identified. For example, *ZmIAGLU,* which conjugates IAA to glucose, has been characterized in maize (Ludwig-Muller et al. 2005). *OsGH3*-like genes, which conjugate IAA to amino acids, have been discovered in rice (Jain et al. 2006b). Furthermore, IAA has been found to be converted to IBA in maize (Ludwig-Muller 2000). Thus, it appears that there are conserved mechanisms of auxin homeostasis in monocots. However, more research needs to be done on the molecular basis for homeostasis. As the synthetic auxin herbicide 2,4,D is rapidly degraded in maize, auxin homeostasis has been proposed to be one of the mechanisms of selectivity of auxinic herbicides (Gauvrit and Gaillardon 1991; Monaco et al. 2002).

Auxin Transport

Evidence of the role of auxin transport in monocot development is illustrated by experiments in which monocots (in particular grasses) are treated with polar auxin transport inhibitors. Treatment with *N*-1-naphthylphthalamic acid (NPA) causes defects in embryogenesis (Fischer and Neuhaus 1996), leaf initiation and growth (Tsiantis et al. 1999; Scanlon 2003), root development (Morita and Kyozuka 2007), and reproductive development (Wu and McSteen 2007). Many of these effects are similar to the effects when dicots are treated with auxin transport inhibitors except for differences in morphology and in sensitivity to certain types of auxin transport inhibitors (Wu and McSteen 2007). The role of auxin transport in development is further discussed in Part II. In this section, genes controlling auxin transport in maize and rice are described.

AUX1

A homolog of the auxin influx carrier *AUX1* has been identified in maize, although no functional evidence has been reported (Hochholdinger et al. 2000). *ZmAUX1* is expressed in roots at the tip of all types of root and in the endodermis, pericycle, and epidermis, indicating it may play a role similar to *Arabidopsis* in roots. A recent analysis using laser capture microscopy and microarray analysis showed that *ZmAUX1* is up-regulated at the site of leaf primordia initiation and down-regulated on treatment with NPA, indicating that *ZmAUX1* may also function in shoots (Brooks et al. 2009).

PIN1

Three homologs of the *PIN1* auxin efflux carrier have been reported in maize and rice. *ZmPIN1a* is expressed in a manner similar to *AtPIN1* as it is up-regulated at the site of initiation of all primordia during shoot and reproductive

development (Carraro et al. 2006; Gallavotti et al. 2008b; Brooks et al. 2009; Lee et al. 2009) and is induced by auxin (Lee et al. 2009). *ZmPIN1a* can rescue the *Arabidopsis pin1* mutant, indicating that it may play a similar role in maize as in *Arabidopsis* (Gallavotti et al. 2008b). Knockdown of a homolog in rice, *OsPIN1*, does not produce the characteristic pin phenotype seen in *Arabidopsis*, perhaps because of redundancy with the three rice *PIN1*-like genes (Xu et al. 2005). However, knockdown of *OsPIN1* does exhibit a phenotype somewhat similar to treatment of rice plants with NPA as there is a decrease in the number of crown roots. Furthermore, there is an increase in the number of tillers and an increase in tiller angle, phenotypes that differ from *pin1* mutants. Therefore, there may be some differences in function that need to be further analyzed.

PID

Maize and rice orthologs of *PID* have been reported. Analysis of function shows that there are similarities and differences. *OsPID* is auxin induced and is expressed in lateral organs and axillary meristems (McSteen et al. 2007; Morita and Kyozuka 2007). No loss-of-function *OsPID* phenotype has been reported, but overexpression gives a root phenotype similar to treatment of plants with NPA (Morita and Kyozuka 2007).

One of the orthologs of *PID* in maize is *BARREN INFLORESCENCE2 (BIF2)* (McSteen et al. 2007). *bif2* mutants have phenotypes very similar to *pid* mutants, indicating that function is conserved. Furthermore, BIF2 phosphorylates ZmPIN1a in vitro and affects ZmPIN1a localization in vivo similar to the function of PID (Skirpan et al. 2009). On the other hand, there may also be differences in function, as BIF2 has been reported to be nuclear localized and to phosphorylate a nuclear localized bHLH transcription factor, BARRENSTALK1 (BA1) (Skirpan et al. 2008). In the future, it would be interesting to determine the subcellular localization of OsPID and to determine whether other nuclear-localized PID-like kinases have taken over the role of BIF2 in *Arabidopsis*.

ABC Transporters

The adenosine triphosphate (ATP)–binding cassette (ABC) transporters, *ABCB19/PGP19/MDR1*, *ABCB1/PGP1*, and *ABCB4/PGP4*, play important roles in auxin transport in *Arabidopsis* (Bandyopadhyay et al. 2007). An ABC transporter also functions in auxin transport in maize and sorghum (Multani et al. 2003). The mutations, *brachytic2 (br2)* in maize and *dwarf3 (d3)* in sorghum, are caused by loss of function of an ABC transporter with high homology to *ABCB1/PGP1* (Multani et al. 2003). *br2* plants have short internodes because of reduced cell elongation and have defects in auxin transport (Multani et al. 2003). In contrast, knockout of *ABCB1/PGP1* does not have a significant phenotype in *Arabidopsis* but double mutants with *ABCB19/PGP19/MDR1* are short and have additional defects in vegetative and reproductive development. Further phylogenetic analysis is required to determine if there is less redundancy in the ABCB gene family in maize.

An ABC transporter of a different class, ABCG, has been identified in rice through the cloning of the *reduced culm number1* (*rcn1*) mutant (Yasuno et al. 2009). *rcn1* mutants, as the name implies, have fewer tillers (Takamure and Kinoshita 1985). The defect is caused by a defect in tiller outgrowth rather than tiller initiation. *rcn1* is induced by auxin, indicating that it may play a role in auxin-mediated development.

ARF GAP

The ADP-ribosylation factor–GTPase-activating protein (ARF-GAP), *VAN3/SCARFACE*, functions in auxin-mediated vascular development in cotyledons, leaves, and roots of *Arabidopsis* (Koizumi et al. 2005; Sieburth et al. 2006). A maize *VAN3/SFC* homolog is reported to be expressed in leaf primordia (Zhang et al. 2007). Overexpression of *OsAGAP*, a rice ARF-GAP, in *Arabidopsis* and rice leads to defects in root development (Zhuang et al. 2005; Zhuang et al. 2006). The *OsAGAP* transgenics have defects in auxin transport, proposed to be caused

by defects in auxin influx (Zhuang et al. 2005; Zhuang et al. 2006).

Homologs of other genes involved in auxin transport such as the guanine-nucleotide exchange factors for ADP-ribosylation factor GTPases (ARF-GEF) *GNOM*, the immunophilin-like *TWISTED DWARF1* (*TWD1*), and the calossin-like *BIG*, have not yet been functionally characterized in monocots. However, from what has been discovered so far, there is conservation in the molecular mechanisms controlling auxin transport in monocots. Monocot-specific functions of auxin transport in controlling crown-root initiation and tiller angle is discussed in Part II.

Auxin Signal Transduction

Aux/IAA

Although there are 25 *Aux/IAA* genes in *Arabidopsis*, there are 31 members in the *Aux/IAA* gene family in rice (Jain et al. 2006a), 24 of which are regulated by auxin (Song et al. 2009a) and three of which have been functionally characterized. The first, *Aux/IAA* reported from rice was called *OsIAA1* but is now renamed *OsIAA3. OsIAA1/3* transcript is induced by auxin and suppressed by light (Thakur et al. 2001) and the protein has a short half life and is degraded by the proteosome (Thakur et al. 2005) similar to the stability of AUX/IAA proteins in *Arabidopsis*.

A very important finding in this area is the seminal paper by Nakamura et al. (2006), reporting the production of auxin-insensitive rice by expressing a degradation-resistant version of *OsIAA3,* now called *OsIAA31. OsIAA3/31* transgenic plants have diverse phenotypes reminiscent of auxin-insensitive mutants in *Arabidopsis*, such as defects in shoot and root gravitropism, root length, and lateral-root initiation (Nakamura et al. 2006). This provided the first functional evidence that auxin signaling may occur similarly in rice as in *Arabidopsis*.

Much work remains to be done to characterize the specific functions of other *Aux/IAA* genes. Overexpression of *OsIAA1* causes defects in root development and an additional defect of enlarged leaf angle (Song et al. 2009b). A transposon insertion into *OsIAA25* is reported to be "dwarf with reduced fertility" but the phenotype has not been characterized in detail (Jain et al. 2006a). In maize, there has been very little characterization of auxin signal transduction. The *rum1* gene of maize, which is a mutant with very specific defects in root development (discussed in Part II), has recently been reported in a patent application to be an *Aux/IAA* gene (Taramino et al. 2008). Additional auxin-induced genes such as homologs of the *SAUR* gene family have been identified in rice and maize (Knauss et al. 2003; Jain et al. 2006c). Therefore, it is likely that there is conservation in signal transduction of auxin in both maize and rice.

ARF

A gene family of *AUXIN RESPONSE FACTOR* (*ARF*) transcription factors has been described in rice with 25 *OsARF* genes compared with 23 *ARFs* in *Arabidopsis* (Sato et al. 2001; Wang et al. 2007). *OsARF* genes have diverse expression patterns but significant functional analysis has only been reported for *OsARF1* (Waller et al. 2002; Attia et al. 2009), which is closely related to *AtARF1* and *AtARF2* in *Arabidopsis*.

OsARF1 is auxin induced and is expressed in coleoptiles, callus, and young panicles with weaker expression in leaves and roots (Waller et al. 2002; Attia et al. 2009). Antisense knockdown of *OsARF1* in rice has defects in vegetative development, producing dwarf plants with small curled leaves and defects in reproductive development, as the plants either fail to flower or flower late and are sterile. Therefore, there are similarities and differences with *arf1*;*arf2* double mutants in *Arabidopsis,* which have delayed flowering, sterility, and delayed senescence (Ellis et al. 2005). Transposon insertions in *OsARF5, 11,* and *12* are reported to have low fertility, whereas insertions in *OsARF11, 19*, and *24* are reported to be dwarf but the phenotypes have not been characterized in detail (Wang et al. 2007). Maize homologs of *AtARF1*, *2*, and 5 are reported to be expressed at a higher level in leaf primordia than in the meristem (Brooks et al. 2009).

TIR1

The F box gene family, including homologs of the *TRANSPORT INHIBITOR RESPONSE1 (TIR1)* auxin receptor gene family have been catalogued in rice (Jain et al. 2007). A maize homolog *ZmTIR1* is expressed in young leaf primordia (Zhang et al. 2007). However, no *TIR1*-like genes have been functionally characterized in maize and rice. It has been proposed that differences in selectivity to herbicides are caused by differences in the functions of *TIR1* auxin receptors because different TIR1-like proteins have different affinities for different auxins in *Arabidopsis* (Walsh et al. 2006; Kelley and Reichers 2007).

ABP1

Another auxin-binding protein, AUXIN BINDING PROTEIN1, ABP1, was initially discovered in maize and subsequently in *Arabidopsis* (reviewed in Timpte 2001; Christian et al. 2006). In maize, knockouts of *ABP1* and *4* did not have a phenotype, whereas *Arabidopsis* knockouts of *ABP1* were embryo lethal, indicating that there may be greater redundancy in the gene family in maize (Im et al. 2000; Chen et al. 2001). Alternately, there could be differences in the role of *ABP1* in maize, which has also been proposed as a possible mechanism for herbicide selectivity (Kelley and Reichers 2007).

PART II—ROLE OF AUXIN IN MONOCOT DEVELOPMENT

Root Development

The root systems of monocots and dicots differ in architecture, yet recent work suggests that auxin plays an equivalent role in maize and rice as in *Arabidopsis* (Hochholdinger and Zimmermann 2008). In most dicots, including *Arabidopsis*, there is a central primary root and the root system branches through the production of lateral roots. In grasses, there is often a fibrous root system such that much of the branching occurs through adventitious roots called crown roots (Raven et al. 2005). In maize and in rice, there is an embryonic and postembryonic root system (Hochholdinger et al. 2004). The embryonic root system consists of a short-lived primary root called a primary root in maize and a seminal root in rice, and embryonic adventitious roots called seminal roots in maize and crown roots in rice (Fig. 2A). The postembryonic root system consists of shoot-borne adventitious roots and lateral roots, which arise on all root types. Maize has three types of adventitious roots—seminal roots, which are embryonic adventitious roots, crown roots, which arise from stem tissue underground, and prop roots, which arise from stem tissue above ground (Fig. 2B). Rice also has the same three types of adventitious root but they are all called crown roots. For the sake of simplicity, all types of adventitious roots in maize and rice are referred to as crown roots in this section. From the analysis of mutants defective in auxin biosynthesis, transport, or signaling, auxin is required to inhibit root elongation and promote lateral-root and crown-root initiation in grasses. The role of auxin in each of these processes will be considered separately.

Crown-root Initiation

Crown-root initiation is of tremendous interest as adventitious roots are much more important for root architecture in grasses than in dicots. It appears that crown-root initiation is controlled somewhat similarly to lateral-root initiation even though these two root types develop from shoot and root tissues, respectively.

Evidence that auxin biosynthesis is required for crown-root formation comes from rice plants overexpressing *OsYUC1,* which have increased crown-root number (Yamamoto et al. 2007). Furthermore, application of exogenous IAA increases crown-root number (Inukai et al. 2005; Xu et al. 2005). Auxin transport is important as inhibition of auxin transport through either treatment with NPA, antisense inhibition of *OsPIN1,* or overexpression of *OsPID* reduces the number of crown roots (Inukai et al. 2005; Xu et al. 2005; Morita and Kyozuka 2007). In addition, auxin signal transduction plays a role because overexpression of *OsIAA1* or degradation

resistant *OsIAA3/OsIAA31* abolishes crown-root initiation (Nakamura et al. 2006; Song et al. 2009b). Therefore, auxin biosynthesis, transport, and signaling are required for crown-root initiation.

Mutants that lack crown roots have been used to identify genes functioning in crown-root initiation. *adventitious rootless1 (arl1)/crown rootless1 (crl1)* mutants in rice lack crown roots, have fewer lateral roots, and have altered root gravitropsism, though they have no defects in the embryonic root system (Inukai et al. 2005; Liu et al. 2005). The *ARL1/CRL1* gene encodes an auxin inducible *LATERAL ORGAN BOUNDARY* (*LOB*) domain containing transcription factor expressed in lateral- and crown-root primordia as well as floral tissue (Inukai et al. 2005; Liu et al. 2005). In maize, the ortholog of *ARL1/CRL1* is *ROOTLESS CONCERNING CROWN AND SEMINAL ROOTS (RTCS)* (Taramino et al. 2007). *rtcs* mutants do not produce crown roots or seminal adventitious roots but can still produce an embryonic primary root with lateral roots (Hetz et al. 1996; Hochholdinger et al. 2004).

In rice, *CRL1* has been shown to be an in vitro target of an *OsARF1*-like transcription factor (Inukai et al. 2005). However, *OsARF1* is predicted to be a repressor ARF and overexpression does not affect root development, so the functional significance of this is unknown (Inukai et al. 2005). In *Arabidopsis*, the LOB domain, containing transcription factors *LBD16* and *LBD29* have been shown to be bona fide downstream targets of *ARF7* and *ARF19* (Okushima et al. 2005a; Okushima et al. 2007). *arf7;arf19* double mutants fail to initiate lateral roots and *lbd16* mutants have a mild defect in lateral-root initiation, whereas *lbd29* knockouts have not been identified (Okushima et al. 2005b). *ARF7* and *ARF19* are activating ARFs, so perhaps homologs of these *ARFs* induce *CRL1* in rice.

Recently, another transcription factor was found to play a role in crown-root formation in rice (Zhao et al. 2009). The *WOX11* gene is a *WUSCHEL*-like homeodomain-containing transcription factor that is auxin and cytokinin inducible. Loss-of-function mutations have fewer crown roots, whereas overexpression causes an increase in the number of crown roots. *WOX*-like genes also play a role in lateral-root initiation in *Arabidopsis*, indicating that gene function is conserved (Deveaux et al. 2008).

Lateral-root Initiation

Auxin has been shown to play a role in lateral-root initiation in maize and rice as in *Arabidopsis*. However, mutant analysis has found that the processes of lateral-root and crown-root formation can be genetically separable.

Exogenous application of auxin promotes lateral-root initiation (Xu et al. 2005). Moreover, defects in auxin transport affect lateral-root initiation, for example, overexpression of *OsPIN1* increases the number of lateral roots, whereas overexpression of *OsAGAP* decreases the number of lateral roots (Xu et al. 2005; Zhuang et al. 2006). Auxin signaling is also required because overexpression of *OsIAA1* decreases the number of lateral roots (Song et al. 2009b).

Mutants that affect crown-root initiation can also affect lateral-root initiation. For example, *crl1/arl1* have defects in both crown-root and lateral-root initiation and the gene is expressed in the primordia of both types (Inukai et al. 2005). On the other hand, the *rootless with undetectable meristems 1* (*rum1*) mutant of maize does not initiate lateral roots or embryonic adventitious roots but crown roots initiate as normal, indicating that these two processes are separable (Woll et al. 2005). Hence, this mutant has been used for analysis of the proteome of different root types (Liu et al. 2006; Saleem et al. 2009). Analysis of the transcriptome indicates no change in the levels of *ZmPIN1* or *ZmAUX1* transcripts (Woll et al. 2005). However, there is reduced auxin transport in mutant roots. *rum1* is reported to be caused by a mutation in an *Aux/IAA* gene (Taramino et al. 2008). This indicates conservation in auxin signal transduction mechanisms in both monocots and dicots, and indicates conservation in function during root development as several dominant mutants in *Arabidopsis Aux/IAA* genes produce no lateral roots (Fukaki et al. 2005; Uehara et al. 2008).

Root Length

Defects in auxin biosynthesis, transport, or signaling also cause defects in root length.

Exogenous auxin inhibits root elongation in monocots and dicots (Zhuang et al. 2006). Decreased auxin biosynthesis caused by *cow1* mutants or antisense *OsYUC1* in rice and *ZmNIT2* mutants in maize have short roots (Kriechbaumer et al. 2007; Woo et al. 2007; Yamamoto et al. 2007). Changes in auxin transport, such as overexpression of *OsPIN1*, increased root length, whereas overexpression of *OsAGAP* decreased root length (Xu et al. 2005; Zhuang et al. 2006). In addition, the degradation-resistant *OsIAA3/31* lines have longer roots on auxin (Nakamura et al. 2006).

In conclusion, despite the differences in root morphology, with *Arabidopsis* having a tap-root system and maize and rice having a fibrous-root system, auxin inhibits root length and promotes lateral-root formation in *Arabidopsis* and inhibits root length and promotes lateral-root and crown-root formation in maize and rice.

Vegetative Development

Stem Development

Dwarfism is a common defect in mutants defective in auxin transport in maize and rice as in *Arabidopsis*. The *br2* mutant in maize has reduced auxin transport and short internodes (Multani et al. 2003). Another mutant in maize that has reduced auxin transport and short internodes is the *semaphore* (*sem*) mutant (Scanlon et al. 2002). The mutant has not yet been cloned but it has additional defects such as defects in vasculature in leaves and a reduction in the number of lateral roots, which implies it plays a role in an auxin-regulated process. The *Developmental disaster* (*Dvd1*) mutant of maize has short internodes without affecting leaf number (Phillips et al. 2009). *Dvd1* mutants also have a pin inflorescence phenotype, indicating that *Dvd1* may control an auxin-regulated process. In rice, *narrow leaf1 (nal1)* mutants have reduced auxin transport and short stems (Qi et al. 2008). Hence, auxin is required for stem elongation in monocots as well as in dicots.

Leaf Development

Genes required for auxin biosynthesis and transport also play a role in leaf initiation. *spi1*, *bif2,* and *Bif1* mutants have a mild reduction in the number of leaves as single mutants (Barazesh and McSteen 2008a; Gallavotti et al. 2008a). However, in double-mutant combinations *spi1;bif2* or *Bif1;bif2*, there is a synergistic effect such that about half the number of leaves as normal are initiated (Barazesh and McSteen 2008a; Gallavotti et al. 2008a). Leaf number defects are also seen in *pin1* mutants in *Arabidopsis* (Bennett et al. 1995). Therefore, auxin biosynthesis and transport play a role in leaf initiation.

Mutants defective in auxin biosynthesis or transport sometimes have narrow leaves, indicating that auxin is required to promote leaf expansion. *nal7* mutants have narrow leaves (Fujino et al. 2007; Woo et al. 2007), and overexpression causes the production of wider leaves (Fujino et al. 2007). *nal1* mutants have narrow leaves as well as short stems and have vasculature defects in the stem and leaves (Qi et al. 2008). *NAL1* encodes a protein with unknown biochemical function with homologs in maize and *Arabidopsis*.

Tiller Angle

A unique role of auxin in rice is in the control of tiller angle. This is a form of stem gravitropism and is critical for the ability of rice to grow at high density. In fact, through breeding programs, rice has been selected for increased tiller angle (Wang and Li 2008). Wild rice has a prostrate growth habit, whereas cultivated rice has a more upright growth habit.

The gravity sensing tissue in grass stems is the pulvinus region at the base of the stem. Leaves also sense gravity independently in a region known as the leaf sheath pulvinus. In fact, in the tiller-angle mutants, it is difficult to separate leaf gravitropism from stem gravitropism as the leaves ensheath the stem. It is

likely that auxin functions in gravity sensing in the pulvinus as auxin differentially accumulates in the pulvinus on gravity stimulation (Long et al. 2002)

The first indication of a role for auxin transport in controlling tiller angle came from RNAi of *OsPIN1,* which increased tiller angle (Xu et al. 2005). The next evidence came from the cloning of *LAZY1* (*LA1*) of rice, which has defects in auxin transport and tiller angle (Li et al. 2007; Yoshihara and Iino 2007). *la1* mutants have defects in stem, leaf, and coleoptile gravitropism, but do not affect root gravitropism. *LA1* encodes a novel gene that appears to be grass specific, as there is no clear homolog in *Arabidopsis* (Li et al. 2007; Yoshihara and Iino 2007). *LA1* is expressed in the leaf sheath pulvinus, the coleoptile, and adjacent to vascular bundles in the stem and leaf (Li et al. 2007; Yoshihara and Iino 2007). *la1* mutants have altered auxin distribution in the stem on gravity stimulation and have increased auxin transport in the coleoptile, indicating that LA1 is a negative regulator of auxin transport (Li et al. 2007).

It is not clear from this analysis of *LA1* and *OsPIN1* if auxin transport promotes or inhibits the prostrate growth habit. *LA1* inhibits auxin transport and mutants have increased tiller angle, whereas *OsPIN1* promotes auxin transport and loss of function causes increased tiller angle (Xu et al. 2005; Li et al. 2007). It should be noted that auxin transport was tested in *la1* coleoptiles rather than tillers, so more research is needed to clarify this. What is clear though is that differential auxin distribution is important for stem and leaf gravitropism.

Two additional genes, *TILLER ANGLE CONTROL1* (*TAC1*) and *PROSTRATE GROWTH1 (PROG1)*, that have opposite effects on tiller angle have been identified as QTL. It is not known if these genes have an auxin connection, but it is clear that they have played a critical role in rice domestication. *TAC1* encodes an unknown protein that is present in single copy in the rice genome and appears to be present only in grasses (Yu et al. 2007). Loss of function of *TAC1* leads to upright tillers, whereas increased expression leads to wider tiller angle. Sequence analysis indicates that the *tac1* mutation is associated with upright tiller angle in *japonica* rice varieties, whereas wild rice and *indica* varieties have wild-type *TAC1* and spread out tiller angle. Similar to *LA1*, *TAC1* is expressed at the base of tillers and in the leaf sheath pulvinus.

PROG1, which is responsible for prostrate growth in the wild rice *Oryza rufipogon*, was identified independently by two groups (Jin et al. 2008; Tan et al. 2008). Loss of function of *PROG1* in cultivated lines led to more erect growth habit and reduced tiller number. In addition, the mutants also have increased panicle branch number and yield. *PROG1* encodes a novel CYS_2 HIS_2 zinc finger in the EBF class. *PROG1* is expressed at the base of tillers in the vascular bundles of the leaf sheath pulvinus and in the lamina joint of the leaf. Whether the absence of *LA1*, *TAC1*, or *PROG1* homologs in *Arabidopsis* indicates a lack of function of auxin in controlling branch angle in *Arabidopsis* requires further analysis.

Reproductive Development

Auxin plays a fundamental role in axillary meristem initiation in the inflorescence (Cheng and Zhao 2007; Barazesh and McSteen 2008b). Axillary meristems give rise to flowers in *Arabidopsis* and branches and spikelets (branches containing the florets) in grasses (McSteen et al. 2000; Barazesh and McSteen 2008b). Defects in auxin biosynthesis, transport, or signal transduction cause defects in flower initiation characterized by the pin inflorescence phenotype in *Arabidopsis* (Bennett et al. 1995; Przemeck et al. 1996; Cheng et al. 2006; Cheng et al. 2007b). The equivalent phenotype in maize is the barren inflorescence phenotype seen in *bif2* and *spi1* mutants, which have defects in auxin transport and biosynthesis (McSteen and Hake 2001; Ritter et al. 2002; Gallavotti et al. 2008a). *Bif1* and *Dvd1* are semidominant mutants that are proposed to have auxin-related defects (Barazesh and McSteen 2008a; Phillips et al. 2009). An additional eight mutants in maize have similar phenotypes but have not been characterized in detail (www.AuxinEvoDevo.org). Furthermore, DR5 and PIN marker lines show an auxin maximum as lateral primordia and axillary meristems

are initiated in maize (Gallavotti et al. 2008b). Thus, despite the differences in morphology between *Arabidopsis* and grasses, auxin still plays a critical role in inflorescence development.

A bHLH transcription factor that functions in axillary meristem formation has been discovered in maize and rice but its role in *Arabidopsis* has not yet been reported (Komatsu et al. 2003; Gallavotti et al. 2004). *ba1* mutants in maize and *lax1* mutants in rice do not produce axillary meristems during inflorescence development but the bract leaves that subtend axillary meristems are unaffected (Ritter et al. 2002; Komatsu et al. 2003). Recent analysis has shown that *lax1* mutants have defects in axillary meristem outgrowth rather than initiation (Oikawa and Kyozuka 2009). BA1 has been proposed to function either upstream or downstream of auxin (discussed in Barazesh and McSteen 2008b).

An exciting recent development was the discovery that the LAX1 protein may traffic between cells (Oikawa and Kyozuka 2009). As *BA1/LAX1* RNA expression is adjacent to rather than in axillary meristems, *BA1/LAX1* was proposed to control the movement of a signal to promote axillary-meristem development (Komatsu et al. 2003; Gallavotti et al. 2004). It turns out that the signal may be the LAX1 protein itself because the protein is present throughout the axillary meristem (Oikawa and Kyozuka 2009). This discovery leads to the question of what controls the movement of LAX1. This will be an exciting area of future research considering how little is known about how transcription factors move between cells.

CONCLUDING REMARKS

The research described here illustrates the power of the comparative approach. In many cases, genes controlling auxin biosynthesis, transport, and signaling were cloned first in *Arabidopsis* and then used to identify homologs in maize and rice. However, in some cases, for example *ABP1*, genes were isolated first in maize and then homologs identified in *Arabidopsis*. Some gene families, for example *PIN1*, are more redundant in maize and rice, whereas others, for example *YUC*, are more redundant in *Arabidopsis*. in some cases, genes such as CYP79B2/3 are found only in species related to *Arabidopsis*, whereas, in other cases, genes such as *LA1* are grass specific. In general, the mechanisms of auxin biosynthesis, transport, and signal transduction are conserved in monocots and dicots, emphasizing the fundamental importance of auxin in development. In the future, it will be interesting to discover how the genes mediating the effects of auxin have diversified, which eventually may lead to an understanding of what makes monocots different from dicots.

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