Developmental Cell
Meeting Review

Plant Hormones and Signaling: Common Themes and New Developments

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DOI 10.1016/j.devcel.2008.03.013

About 200 plant biologists convened in Keystone, Colorado, for the "Plant Hormones and Signaling" symposium, which was organized by Joanne Chory, Joe Ecker, and Mark Estelle. The meeting was run concurrently with the "Plant Innate Immunity" symposium organized by Jonathan Jones and Jane Glazebrook. In this report, we summarize the progress in plant hormones and signaling.

Plant hormones are key regulators of plant growth and development. Great progress in understanding hormone biosynthesis, metabolism, transport, and signal transduction was reported at this meeting. All of the classic hormones including auxin, cytokinin (CK), brassinolide (BL), gibberellin (GA), ethylene, abscisic acid (ABA), jasmonate (JA), and salicylic acid (SA) were discussed, in addition to novel signaling molecules such as nitric oxide (NO), glucose, and the "branching hormone" produced by the MAX pathway. Now that almost all of the plant hormone receptors have been identified, it has become evident that common strategies are used to transmit different hormonal signals, although each hormone has its own unique way of exacting its regulatory effects. In this report, we highlight the key discoveries in the area of plant hormones and signaling reported at this meeting.

Hormone-Dependent Protein Degradation

A common strategy used to transmit hormonal signals is to remove a key transcription factor using an F-box-containing ubiquitin E3 ligase in a hormone-concentration-dependent manner. The transcription factor can be an activator, such as ETHYLENE INSENSITIVE 3 (EIN3) in the ethylene signaling pathway, but more often it is a transcriptional repressor, such as the Aux/IAA, DELLA, and JAZ proteins in the auxin, GA, and JA signaling pathways, respectively.

The importance of targeted protein degradation in plant hormone signaling was first described in the auxin signal transduction pathway (Abel et al., 1994; Guilfoyle and Hagen, 2007; Leyser et al., 1993). It has been well established that the Aux/IAA proteins, transcriptional repressors, are targeted for degradation in an auxin-dependent manner through the ubiquitin-related protein degradation machinery, thereby allowing the auxin response factors (ARFs) to activate a network of genes to regulate plant growth and development (Figure 1A). Recent crystallographic studies on the auxin receptor TRANSPORT INHIBITOR RESPONSE 1 (TIR1) in complex with auxin and an Aux/IAA peptide clearly show that auxin serves as "molecular glue" that brings together an Aux/IAA protein and the F-box protein TIR1 (Tan et al., 2007). Thus, the TIR1-dependent auxin signaling pathway is short, and auxin plays a direct role in facilitating the degradation of Aux/IAA proteins. At this meeting, Mark Estelle (Indiana University) extended his auxin signaling work in *Arabidopsis* to other systems, particularly *Physcomitrella patens* (moss). It appears that the TIR1-related auxin signaling pathway is highly conserved throughout the plant kingdom. Like the *Aux/IAA* genes in *Arabidopsis*, dominant mutations in moss *Aux/IAA* genes also lead to auxin resistance. In addition to the main auxin indole-3-acetic acid, other auxin-related molecules including indole-3-butyric acid, IAA conjugates, and IAA-esters have been identified in plants. The existence of multiple Aux/IAA proteins and TIR1/AFB proteins in *Arabidopsis* raises the possibility that some of the Aux/IAA-AFB combinations may respond to other endogenous auxins or modified auxins. Interestingly, Estelle reported that AFB4/5 appeared to be more specific for a synthetic auxin called picloram than to the natural auxin indole-3-acetic acid. These findings also make it likely that different plant species may differ in their Aux/IAA-AFB pairs that respond to a particular auxin.

JA plays a key role in plant defense and development, and it has been shown that the active form of JA is the JA-Isoleucine conjugate (JA-Ile) (Wasternack, 2007). The JA signaling pathway is analogous to the TIR1-mediated auxin signaling pathway (Figure 1B). Mutations in *CORONATINE INSENSITIVE 1* (*COI1*), which encodes an F-box protein, lead to insensitivity to coronatine (a chemical analog of JA) and disruption of the known JA responses. John Browse (Washington State University) reported that COI1 interacts with JASMONATE ZIM DOMAIN (JAZ) transcription factors in a coronatine-dependent and JA-Ile-dependent manner in a pull-down assay similar to that used to identify TIR1 as an auxin receptor. Both genetic and biochemical data indicate that COI1 is the JA receptor that promotes the degradation of the JAZ repressors (Chini et al., 2007; Thines et al., 2007). It will be interesting to investigate whether JA-Ile also serves as molecular glue to bridge the COI1 and JAZ proteins. Unlike auxin signaling, where ARF proteins bind to the auxin response elements to promote transcription, the transcriptional activator for JA signaling and the JA response elements have not been well defined, though the MYC2 transcription factor has been proposed to play a role.

GA signaling also shows some similarity to auxin and JA signaling (Figure 1C). Degradation of the DELLA proteins, transcriptional repressors, is a key step in GA signaling (Schwechheimer, 2008). In rice, the GA receptor GID1 is not an F-box protein, but GA binding to GID1 promotes the interaction between GID1 and

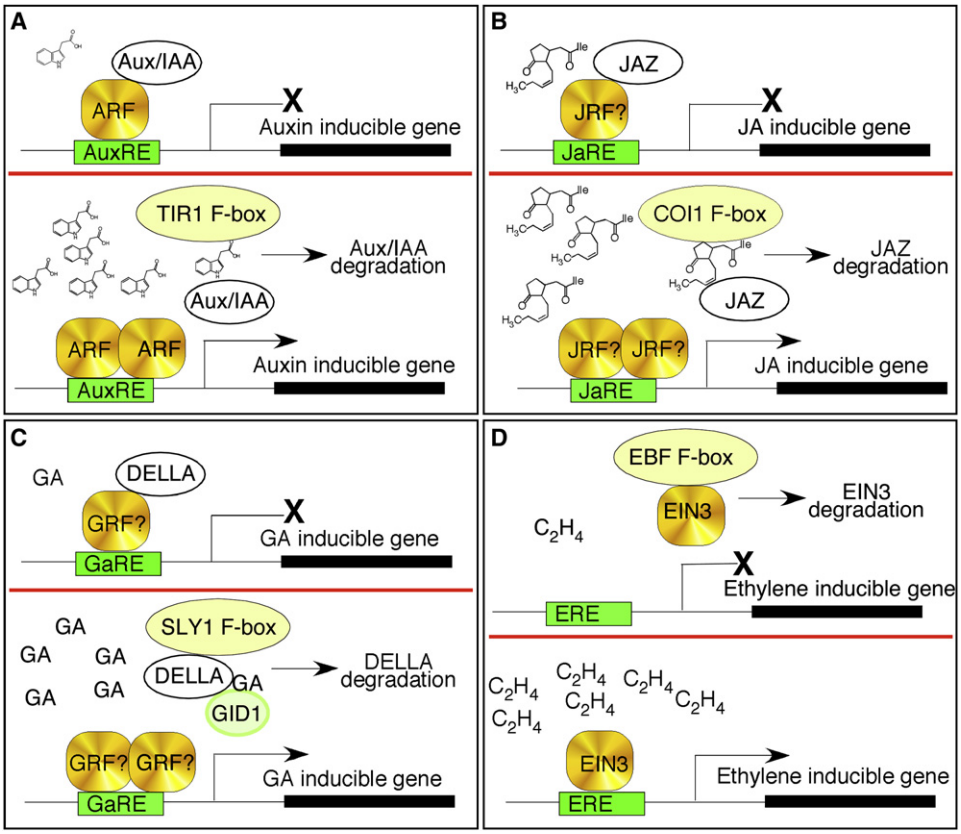

Figure 1. Hormone-Mediated Degradation of Transcription Factors Is a Key Step in Transmitting Hormonal Signals

(A) Auxin signaling. When auxin levels are low, Aux/IAA proteins form heterodimers with ARF, thus preventing ARF-mediated transcription of auxin-inducible genes (top panel). When auxin concentration is high, auxin serves as "molecular glue" that brings Aux/IAA and TIR1 together and promotes the degradation of Aux/IAA, therefore allowing ARFs to activate transcription (bottom panel).

(B) JA signaling is analogous to auxin signaling. JA-Ile promotes the degradation of JAZ repressors, thus allowing the expression of the JA-inducible genes. The JA response factors (JRFs) and JA response elements (JaRE) have not been defined. It is also not clear whether JA-Ile binds between JAZ and COI1.

(C) GA signaling. Unlike auxin and JA signaling, the GA receptor is not an F-box protein. However, GA binding to the receptor GID1 promotes the interaction between GID1 and the F-box protein GID2/SLY1, which catalyzes the degradation of DELLA proteins.

(D) Ethylene response. In the absence of ethylene, the transcription activator EIN3 is degradated by two F-box proteins (EBF1 and EBF2). When ethylene concentration is increased, the degradation of EIN3 is inhibited, allowing EIN3 to bind to ethylene response elements. The exact mechanisms by which ethylene regulates the stability of EIN3 are not understood.

SLR1, a DELLA protein functioning as a transcription repressor. Association of GID1 and SLR1 is GA concentration dependent and promotes the interaction between SLR1 and the F-box protein GID2/SLY1. Therefore, DELLA proteins can be degraded in a GA-dependent manner, although the GA receptor itself is not a ubiquitin E3 ligase. In GA signaling, the activator partners of DELLA and GA response elements have not been well defined. However, recent findings that PIF3 interacts with DELLA and that the removal of DELLA releases PIF3 for transcription regulation indicate that PIF3-DELLA may work analogously to Aux/IAA-ARF in auxin signaling (de Lucas et al., 2008; Feng et al., 2008). At the meeting, Nick Harberd (University of Oxford) described an analysis of the evolution of the GA signaling pathway (Yasumura et al., 2007). Modern land plants, including *Arabidopsis*, are thought to have evolved from a simple ancestor. Harberd's group chose several evolutionarily important plant species including moss, fern, and *Arabidopsis* to study the evolution of the GA signaling pathway. Although GA appears not to regulate the growth of moss, a nonvascular plant, moss has DELLA proteins that can function as transcription repressors independent of GA when expressed in *Arabidopsis*. In *Selaginella*, a type of fern and primitive vascular plant, GA is not a growth regulator. However, *Selaginella* DELLAs and GIDs interact with each other. Furthermore, *Selaginella* DELLAs can function as repressors in *Arabidopsis* in a GA-dependant manner. Because GA signaling appears to have evolved in a stepwise fashion in plants, these findings provide a unique opportunity to understand how plant hormone signaling may have evolved.

Regulation of EIN3 protein level plays an important role in ethylene signaling (Li and Guo, 2007). Unlike Aux/IAA, JAZ, and DELLA proteins, which are repressors, EIN3 is a transcriptional activator that directly induces the transcription of ethylene-inducible genes (Figure 1D). Ethylene treatment promotes the stabilization of EIN3, which interacts with two homologous F-box proteins (EIN3 BINDING F-BOX PROTEIN 1 and 2 [EBF1 and EBF2]). The exact mechanism by which ethylene regulates EIN3 stability still remains unsolved.

In addition to the signaling mechanisms of the four hormones discussed above, targeted protein degradation probably also plays important roles in other hormone signaling pathways. For

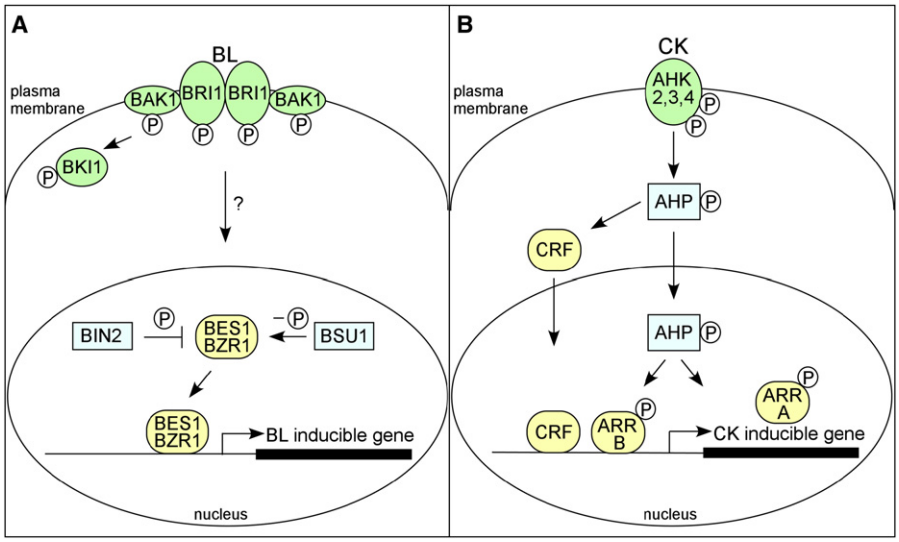

Figure 2. Perception of Hormones at the Plasma Membrane and Transmission of the Signal to Transcription Factors in the Nucleus using Phosphorylation

(A) Brassinosteroid signaling. Perception of BL at the plasma membrane by the BRI1 receptor kinase causes association with the receptor kinase BAK1 and dissociation of the phosphoprotein BKI1. By an unknown mechanism, BL signaling causes dephosphorylation and activation of the transcription factors BZR1/BES1 in the nucleus by inhibiting the kinase BIN2 and activating the phosphatase BSU1. BIN2 and BSU1 may interact with BZR1/BES1 in the nucleus or in the cytosol. (B) CK signaling. CK perception by the receptor kinases AHK2, AHK3, and AHK4 causes phosphorylation of the phosphotransfer proteins AHP1–5 and phosphorylation of the type A and B ARR transcription factors. CK binding also results in the rapid AHP-dependent nuclear translocation of the transcription factor CRF.

example, the *MAX2* gene encodes an F-box protein and plays a key role in regulating branching (Mouchel and Leyser, 2007). Ottoline Leyser (University of York) suggested that MAX2 probably mediates the degradation of a repressor in response to the branching hormone. Both the branching hormone and the MAX2 substrates remain to be identified.

Transmission of Signals from the Plasma Membrane to the Nucleus

Alteration of gene expression in the nucleus is the ultimate response elicited by hormone signals. As discussed above, perception of some hormones including auxin, GA, and JA can directly regulate the degradation of transcription factors, which are presumably located in the nucleus. Other hormones including BL, CK, and ethylene use the classical mechanism of signal transduction, which involves membrane-bound receptors. The hormone signal is transmitted from the receptors at the membrane, usually by phosphorylation, to transcription factors in the nucleus. There are still missing links in some of these hormone-signaling pathways. The role of phosphorylation and nuclear-cytoplasmic shuttling was a particularly hot topic at the meeting.

BL is perceived by the extracellular domain of BRASSINOSTEROID INSENSITIVE 1 (BRI1), a plasma-membrane-bound Leucine-rich-repeat (LRR) receptor kinase (Gendron and Wang, 2007). In the absence of BL, BRI1 association with the phosphoprotein BRI1 KINASE INHIBITOR 1 (BKI1) maintains it in an inactive state. Binding of BL to BRI1 causes dissociation of BKI1 from the plasma membrane and association of BRI1 with the receptor kinase BRI1 ASSOCIATED RECEPTOR KINASE 1 (BAK1). Joanne Chory (The Salk Institute) reported that BRI1 cycles between endosomes and the plasma membrane, but the function of BRI1 in the endosomes is still unknown (Geldner et al., 2007). The GSK1-like kinase BRASSINOSTEROID INSENSITIVE 2 (BIN2) phosphorylates the transcription factors BRASSINAZOLE RESISTANT 1 (BZR1) and BRI1-EMS-SUPPRESSOR 1 (BES1), whereas the phosphatase BRI1 SUPPRESSOR 1 (BSU1) dephosphorylates BZR1 and related proteins (Figure 2A). Phosphorylated BZR1/BES1 has lower affinity for the BL-responsive promoters than the dephosphorylated BZR1/BES1. The mechanism by which BL perception by BRI1 leads to activation of these transcription factors by negatively regulating BIN2 and positively regulating BSU1 is still not known. Zhi-Yong Wang (Carnegie Institution) reported that subcellular localization of BZR1 is regulated by a 14-3-3 protein (Gampala et al., 2007). His group determined that BIN2 phosphorylates the 14-3-3 binding site on BZR1 and that binding of phosphorylated BZR1 by a 14-3-3 protein retains BZR1 in the cytosol.

Like BL, CK is also perceived outside the cell by the extracellular domain of the three CK histidine kinase receptors ARABIDOPSIS HISTIDINE KINASE 4 (AHK4) (also known as CYTOKININ RESPONSE 1 [CRE1] or WOODEN LEG 1 [WOL1]), AHK2, and AHK3 (To and Kieber, 2008) (Figure 2B). The binding of CK sets up a phospho-relay similar to the two-component systems in bacteria. CK binding leads to autophosphorylation of a histidine residue in the cytoplasmic domain of the CK receptors. Subsequently, the phosphate is transferred from the histidine to an aspartic acid residue in the receptor. From there, the phosphate is transferred to a histidine residue in one of the five ARABIDOPSIS HIS PHOSPHOTRANSFER PROTEINS (AHP1–5), which translocate to the nucleus and transfer the phosphate group to an aspartic acid residue on the 10 type A or 11 type B ARABIDOPSIS RESPONSE REGULATORS (ARR). The type A ARRs are negative regulators of CK response, and type B ARRs are positive regulators. Recent work reported by Joe Kieber (University of North

Carolina, Chapel Hill) has heroically determined the functions of many of these components by simultaneously inactivating multiple gene family members. This analysis is beginning to reveal extensive functional diversification of these genes in vascular development, gametophyte development, light and circadian signaling, cell expansion, and shoot and root meristem development, as well as in biotic and abiotic stress.

The Kieber lab has recently shown that the AP2-like transcription factors CYTOKININ RESPONSE FACTORS (CRFs) rapidly translocate to the nucleus in response to CK signaling and induce the expression of target genes that are a subset of the targets of the type B ARRs (Rashotte et al., 2006). It has been reported that AHPs also translocate to the nucleus in response to CK. However, Kieber reports that AHP translocation is too slow to account for the CK-activated transcription response. In addition to cytosol/nucleus trafficking, phosphorylation also regulates transcription factor activity in CK signaling. Kieber reported that phosphorylation of type A ARR transcription factors stabilizes the proteins and is required for their activity (To et al., 2007).

Considering that auxin perception by TIR1 is a nuclear event, how does the cell know what is happening with auxin influx and auxin efflux at the membrane? How does the cell know what is happening in neighboring cells? Moreover, is there any connection between events at the plasma membrane and events in the nucleus? The auxin influx (AUX1) and efflux (PIN1 and PGP) carriers are present at the plasma membrane, and it has been reported that PIN1 localization depends on the TIR1 signal transduction pathway (Sauer et al., 2006). Furthermore, like BRI1, PIN proteins cycle between the plasma membrane and endosomes (Geldner et al., 2001; Vieten et al., 2007). Jiri Friml (Flanders Institute for Biotechnology, University of Ghent) reported at the meeting that establishment of PIN polar localization is a two-step process and an important mechanism for targeting PINs to specific membranes. A plant homolog of the endocytosis regulator RAB5 plays an important role in this process. In addition, Friml reported on the surprising discovery that PIN5 is localized in the ER, where it may play a role in auxin influx into the ER. AUXIN BINDING PROTEIN 1 (ABP1) is also present in the ER, highlighting that this subcellular location may be a site of auxin perception or degradation. Another protein that regulates PIN1 localization, perhaps directly, is the serine threonine protein kinase PINOID (PID) (Michniewicz et al., 2007). Paula McSteen (Penn State University) reported that a maize ortholog of *PID*, called *BARREN INFLORESCENCE 2* (*BIF2*), is localized both at the plasma membrane and in the nucleus (in heterologous systems) where it phosphorylates a bHLH transcription factor required for inflorescence development (McSteen et al., 2007). Does this represent multiple roles of the BIF2 kinase or a potentially direct mechanism to transduce the auxin signal from the membrane to the nucleus? Yunde Zhao (University of California, San Diego) reported on the identification of NAKED PINS IN YUC MUTANTS (NPY) as a new signaling component in the auxin perception pathway (Cheng et al., 2007b; Furutani et al., 2007). NPY has homology to NON PHOTOTROPIC HYPOCOTYL 3 (NPH3), a BTB/POZ-containing protein involved in phototropism signaling. In this pathway, light is perceived by PHOTOTROPIN 1 (PHOT1), a plasma-membrane-localized serine threonine kinase from the same family as PID, leading to the dephosphorylation of NPH3 by an unknown phosphatase and the activation of the ARF-like transcription factor NON PHOTOTROPIC HYPOCOTYL 4 (NPH4) in the nucleus (Pedmale and Liscum, 2007). This signaling pathway appears to parallel the situation in auxin signaling, where *pid*, *npy*, and *monopteros*/*arf5* (*mp*) have similar phenotypes and synergistic genetic interactions. Is it possible that PID-NPY-MP acts similarly to PHOT1-NPH3-NPH4, with signaling from PID at the membrane through NPY to turn on MP in the nucleus? There are still missing links and missing mechanisms for both the auxin and phototropism signaling pathways.

Similar to auxin, where there may be multiple sites of perception, multiple receptors and multiple sites of ABA recognition have been reported. Ligeng Ma (National Institutes of Biological Sciences, Bejing) reported on the isolation of G PROTEIN COUPLED RECEPTOR 2 (GCR2) as a plasma-membrane-localized ABA receptor (Liu et al., 2007). This discovery was quite controversial (Gao et al., 2007) and generated much discussion at the meeting. There appear to be several ABA receptors that act in different subcellular compartments and regulate different responses to ABA (Wang and Zhang, 2008). Although much is known of the signal transduction pathway, second messengers, and transcription factors involved in ABA signaling, how the signal is transduced into the nucleus to regulate ABA-induced genes is not known, though phosphorylation is proposed to play a role.

The importance of nuclear translocation of transcription factors was also emphasized in the presentation by Xinnian Dong (Duke University) on SA signaling. The transcription factor NON EXPRESSOR OF PATHOGENESIS RELATED GENES (NPR1) is a key component in SA signaling (Loake and Grant, 2007). SA induces the translocation of NPR1 into the nucleus, but the mechanism is quite different from those used by other hormone signaling pathways. SA induces a burst of reactive oxygen species followed by redox changes that cause the reduction of disulfide bonds between cysteine residues of NPR1. As a result, NPR1 changes from an oligomeric form to a monomer that enters the nucleus. Once inside the nucleus, NPR1 interacts with TGA1 transcription factors and induces the expression of WRKY transcription factors and PATHOGENESIS RELATED (PR) genes, which play a role in plant defense.

Glucose has been proposed as a signaling molecule as well as a carbon source (Rolland et al., 2006). Low glucose promotes growth, but high glucose represses growth. Jen Sheen (Harvard Medical School) reported on the role of HEXOKINASE 1 (HXK1) in glucose sensing. HXK1 has two roles as a kinase that converts glucose to glucose-6-phosphate and a role in glucose signaling. HXK1 is localized to the outer membrane of mitochondria and is present in a high molecular weight complex in the nucleus. In the nucleus, HXK1 affects gene expression by repressing photosynthetic gene expression. At the meeting, Sheen reported on the identification of some of the proteins that interact with HXK1 in the nucleus, HXK1 UNCONVENTIONAL PARTNER (HUP1) and HUP2, and their effect on gene expression (Cho et al., 2006). In addition, Sheen also talked about the energy sensors Snf1 related protein kinases (SNRK1), KIN10, and KIN11 involved in starvation response (Baena-Gonzalez et al., 2007). The energy status of the cell is sensed by KIN10/11, which signals through bZIP transcription factors to globally regulate the transcription response in the nucleus. However, where in the cell the energy status is sensed is not known.

Hormone Biosynthesis: Unexpected Complexity

The biosynthesis of most classic hormones has been well defined with the exception of auxin. At this meeting, Yunde Zhao (University of California, San Diego) reported on the roles of YUCCA (YUC) flavin monooxygenases in auxin biosynthesis. Overexpression of the *YUC* genes leads to auxin overproduction, whereas disruption of *YUC* genes causes defects in embryogenesis, seedling, vascular, and flower development (Cheng et al., 2006, 2007a). The *yuc* mutants can be rescued by the expression of the bacterial auxin biosynthesis gene *iaaM* under the control of the *YUC* promoters. Previous studies have shown that YUC catalyzes the hydroxylation of tryptamine, a rate-limiting step in tryptophan-dependent auxin biosynthesis. *Arabidopsis* has 11 *yuc* genes that have distinct and overlapping expression patterns. *YUC1* and *YUC4* are expressed in discrete groups of cells that often mark the incipient cites for lateral organ formation. Hence, the analysis of *YUC* genes indicates that localized auxin biosynthesis plays an important role in controlling many aspects of plant development. Andrea Gallavotti (Schmidt lab, University of California, San Diego) reported that the maize *sparse inflorescence 1* (*spi1*) mutant is defective in axillary meristem and vasculature formation, similar to the phenotype of *bif2* mutants discussed above (McSteen et al., 2007). *SPI1* encodes a *YUC*-like flavin monooxygenase that is only expressed in localized domains during axillary meristem and lateral organ initiation. Together with previous studies on *YUC* genes in *Arabidopsis*, petunia, tomato, and rice, the analysis of *spi1* further demonstrates that the *YUC* pathway is a highly conserved auxin biosynthesis pathway, and that localized auxin biosynthesis plays an important role in plant development. Julin Maloof (University of California, Davis) reported that the *YUC* genes in *Arabidopsis* also play an important role in shade avoidance.

Jose Alonso (North Carolina State University) reported on the identification of another tryptophan-dependent auxin biosynthesis pathway and its role in plant development. Alonso conducted a genetic screen for mutants that display weak ethylene insensitivity (*wei* mutants), but interestingly, several mutants including *wei8* turn out to encode components in auxin pathways. It is known that many auxin-resistant mutants, such as *axr1* and *aux1*, display weak ethylene insensitivity, suggesting that normal auxin functions are necessary for ethylene responses. Alonso reported that *WEI8* encodes a PLP-dependent amino transferase that can convert tryptophan to indole-3-pyruvate in vitro, a presumed step in a tryptophan-dependent auxin biosynthesis pathway. Inactivation of *WEI8* and its two closely related homologs leads to defects in embryogenesis, vascular patterning, and flower development, phenotypes similar to the *yuc* mutants. Interestingly, *WEI8* is also only expressed locally, further supporting the concept that localized auxin biosynthesis is essential for plant development. It is not clear why plants use several nonredundant pathways to synthesize auxin. Apparently, *WEI8* and *YUC* genes have overlapping expression patterns, but they do not have overlapping functions. Perhaps the two pathways may contribute auxin to different intracellular auxin pools.

Sakis Theologis (Plant Gene Expression Center) reported the systematic analysis of the *1-AMINOCYCLOPROPANE-1-CARBOXYLIC ACID SYNTHASE* (*ACS*) family in *Arabidopsis* by biochemical, genetic, and physiological analysis. ACS catalyzes the rate-limiting step in the ethylene biosynthesis pathway in plants. There are nine *ACS* genes in the *Arabidopsis* genome. Theologis' group demonstrated that the *ACS* genes have unique and overlapping expression patterns and ACS proteins can form homodimers and heterodimers that may or may not have enzymatic activities (Tsuchisaka and Theologis, 2004a; Tsuchisaka and Theologis, 2004b; Yamagami et al., 2003). Analysis of *Arabidopsis* plants in which multiple *ACS* genes are compromised demonstrated that *ACS* genes play a key role in many aspects of plant growth and development.

The Importance of Deactivating Hormones: To Grow or Not to Grow

Plant hormones such as brassinosteroids, auxin, and GA promote growth, while ethylene and ABA suppress growth. Joanne Chory (The Salk Institute) showed that brassinosteroids act in the epidermis to regulate growth (Savaldi-Goldstein et al., 2007). Auxin has also been proposed to be transported and to regulate polar growth in the epidermis (Reinhardt et al., 2003). One of the mechanisms to control growth is to use regulated protein degradation to remove the repressors (e.g. DELLA and Aux/IAA), as was discussed above. Another important mechanism that is gaining recognition is the deactivation of the hormones themselves. Enzymatic processes for deactivation and conjugation of hormones are known, but new components have recently been identified. For example, Michael Neff (Washington State University) reported on a role for cytochrome P450s in brassinosteroid inactivation (Turk et al., 2005), and Zhen-Ming Pei (Duke University) reported that NO can interact with CK in vivo and in vitro.

Moreover, a role for regulated hormone deactivation in plant development has recently been discovered. Brassinosteroids, auxin, and GA, being growth hormones, need to be removed when the plant wants to suppress growth. One place where the plant needs to suppress growth is at organ boundaries. It was previously reported that GA is removed at the boundary between the apical meristem and lateral organs by activation of *GA 2 OXIDASE*, which deactivates GA at the boundary (Jasinski et al., 2005). Patty Springer (University of California, Riverside) reported that brassinosteroids are downregulated in organ boundaries through direct action of the LATERAL ORGAN BOUNDARIES (LOB1) protein, which was recently shown to be a transcription factor (Husbands et al., 2007). Moreover, Lars Ostergaard (John Innes Centre) reported that formation of an auxin-response minimum is necessary for valve margin differentiation in *Arabidopsis* fruit. The mechanism for auxin removal at the boundaries may involve regulated auxin transport controlled by valve margin identity factors.

Crosstalk between Hormones

Crosstalk between hormones is an active area of research. Most excitingly, the field is now getting to the actual molecular mechanism of crosstalk. As discussed above Jose Alonso (North Carolina State University) reported on a direct interaction between ethylene and auxin biosynthesis. Jennifer Nemhauser (University of Washington) discussed interactions between brassinosteroids and auxin in seedling growth, Christian Hardtke (University of Luasanne) reported on auxin-BL interaction in root vascular development, and Stacey Harmer (University of California, Davis) discussed the interaction between circadian and auxin signaling.

A novel aspect of the meeting was that one day was devoted to a joint session with the "Plant Innate Immunity" symposium so that the recently identified links between hormone signaling and defense could be discussed in more detail. Recent advances in the regulatory interaction between JA and SA in plant defense were discussed by Corne Pieterse (Utrecht University) and Jane Glazebrook (University of Minnesota). The newly discovered role of auxin and GA in defense was discussed by Jonathan Jones (Sainsbury Laboratory). There were multiple cases of people screening for hormone mutants and ending up identifying components in plant defense or vice versa, as shown by Bonnie Bartel (Rice University) and Christina Dixelius (Swedish University of Agricultural Sciences). Pathogens are known to produce hormones, and in fact some hormones were first isolated from pathogens (e.g., GA). Perhaps it is not surprising that pathogens have coopted plant hormone signaling pathways in the arms race for control of plant growth.

Perspectives

Tremendous progress has been made in the past few years in the area of plant hormones and signaling. Future trends in this area will undoubtedly use new approaches including live cell imaging as discussed by Marcus Heisler (Meyerowitz lab, California Institute of Technology) and chemical genetics as discussed by Peter McCourt (University of Toronto) and TaeHoun Kim (Schroeder lab, University of California, San Diego). Computer-aided mathematic modeling of hormonal signaling pathways may lead to the development of new hypotheses. Investigation of the evolution of hormone pathways will provide insights into how hormone pathways have adapted to regulate complex and diverse developmental processes.

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