The Plant Cell, S9–S14, Supplement 2002, www.plantcell.org

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Specificity and Cross-Talk in Plant Signal Transduction: January 2002 Keystone Symposium

More than 200 researchers convened in Tahoe City, CA, for this excellent symposium, which was organized by Julian Schroeder, Mark Estelle, and Masaki Furuya and sponsored by Monsanto Co. Many of the symposium speakers are coauthors of reviews presented in this Special Issue of *The Plant Cell*. In this report, I will not attempt to cover the entire symposium but instead will focus on a subset of my favorite presentations on topics that are not covered in detail elsewhere in this issue.

MITOGEN-ACTIVATED PROTEIN KINASE CASCADES AND PROTEIN–PROTEIN INTERACTIONS

Beverly Errede (University of North Carolina, Chapel Hill) delivered the keynote address of the symposium on mechanisms that contribute to the specificity of mitogen-activated protein (MAP) kinase cascade signaling pathways in *Saccharomyces cerevisiae*. The MAP kinase cascade is a highly conserved mechanism of intracellular signaling in eukaryotes, consisting of activation modules made up of kinases that work in sequence: a MAP kinase kinase kinase (MAPKKK) activates a MAP kinase kinase (MAPKK), which in turn activates a MAP kinase (MAPK) that ultimately affects the expression of one or more downstream genes. Errede emphasized that kinase cascades are networks and not linear pathways; a number of proteins are known to act in more than one pathway, and/or a particular pathway may have two different physical responses to different stimuli. For example, the transcription factor Ste12, which is activated by MAPK signaling, is involved in the different growth responses of yeast cells to nutrient deprivation (stress response) and to pheromone (mating response).

In the mating response, specific MAPKs are activated that lead to cell cycle arrest and to the derepression of Ste12, causing the expression of genes that bring about the formation of a mating projection. The response is transient; if mating does not occur within a short time, Ste12 is repressed again and the cell cycle resumes its normal progression. Pheromone induction of Ste12 involves the induction of proteasome-related factors, which appears to be related to the enhanced degradation of the Ste12 protein and the transient nature of the response. Under nutrient deprivation, cells are induced to alter their morphology to take on a pseudohyphal form, a growth response that is mediated by Ste12 as well. In this case, the cells do not arrest and the expression of Ste12 appears to continue throughout the cell cycle. As long as the cells remain under nutrient deprivation, Ste12 is expressed and cells continue to differentiate in a pseudohyphal form. The major lesson from Errede's presentation was that understanding protein–protein interactions is the key to understanding signaling networks.

Jen Sheen (Massachusetts General Hospital and Harvard Medical School, Boston) described the development of model mesophyll protoplast cell systems in maize and Arabidopsis to study MAPK signaling in plants. For example, Arabidopsis has about 20 MAPK genes, 10 MAPKK genes, and >25 MAPKKK genes, and it is known that many different signals activate MAPK cascades, including many hormones and biotic and abiotic stresses. The triple kinases typically are made up of different domains that affect different aspects of protein function (e.g., activity, specificity, and protein interactions); thus, domain function can be examined easily by genetic manipulation. The double kinases rely on phosphorylation for activation, and constitutively active and dominant-negative mutants can be created. Sheen's group is performing functional genomic analysis of the MAPKKKs, MAPKKs, and MAPKs in the protoplast system, together with reporter genes specific for various downstream responses, to investigate the details of MAPK signaling cascades. They recently used this system to identify a complete MAPK cascade that functions downstream of the flagellin receptor kinase and confers resistance to bacterial and fungal pathogens (Asai et al., 2002).

HORMONAL CROSS-TALK

Although "cross-talk" between pathways is likely to depend on specific (and multiple) protein–protein interactions, the term typically is used to refer to genetic interactions between various pathways. Peter McCourt (University of Toronto, Canada) described work on genetic interactions among various hormone signaling pathways in Arabidopsis. Screens for abscisic acid (ABA) signaling mutants that have turned up ethylene, gibberellic acid, and sugar signaling response genes provide evidence for genetic interactions between ABA signaling and other hormones. McCourt emphasized that one problem in the ABA signaling field is that the numerous existing mutants are found in different Arabidopsis accessions or

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ecotypes. To determine the complete function of a gene with multiple roles in signaling, it will be essential to generate numerous mutant alleles of single genes in a single accession.

For example, *ABI3* (*ABA INSENSITIVE3*) encodes a seed-specific transcription factor that is involved in the regulation of seed dormancy and that has a number of different domains. The generation of a set of mutant alleles in the Columbia ecotype having mutations in different domains showed that ABI3 is a complex protein that is involved in responses to a number of different signals: *abi3-9* has a mutation in the B2 domain and is resistant to exogenous ABA application; *abi3-10* has a different mutation in the B2 domain and shows a sugar signaling response; and the *abi3-11* allele has a mutation between the B2 and B3 domains and shows a somewhat different sugar signaling response from that of *abi3-10*. Work of this type will help determine the protein–protein interactions that underlie genetic interactions between pathways.

THE SEARCH FOR AN ABA RECEPTOR

McCourt also touched on the search for an ABA receptor, which has remained elusive despite concerted efforts toward this goal. The lack of an ABA receptor mutant could be attributable to lethality, redundancy, or simply bad luck (i.e., not enough mutants). Another approach might be to use analogs of ABA. For example, the stereoisomer (−)-ABA has some, but not all, of the same effects as the wild-type (+)-ABA when applied exogenously. A screen for (−)-ABA–insensitive mutants (Nambara et al., 2002) has revealed some new and interesting mutant loci (if not the desired ABA receptor) in a gene designated *CHO1* that encodes a member of the AP2 family of transcription factors. The generation of multiple new alleles of known mutant loci, and of new mutants through the use of innovative screens, should help determine the details of ABA signaling and cross-talk with other hormones.

Mike Blatt (University of Glasgow, Scotland) also is involved in efforts to identify an ABA receptor, in this case with the use of a *Xenopus* oocyte system. *Xenopus* oocytes readily translate foreign (injected) mRNA. Blatt and colleagues cloned *Nt-SYR1* from tobacco by searching for a plant-derived mRNA whose product activated an ABA-induced Ca^{2+}-dependent chloride channel in the oocyte system (Leyman et al., 1999). *Nt-SYR1* encodes a syntaxin-related SNARE, which is a family of proteins that are essential for membrane trafficking in all eukaryotes. A dominant-negative mutant of Nt-SYR1 prevented potassium and chloride ion channel responses to ABA in isolated guard cells. It also caused stunting, altered leaf morphology, and cessation of root growth when expressed in tobacco plants, and it disrupted the secretion of a secreted green fluorescent protein marker in transgenic tobacco, suggesting that Nt-SYR1 functions in vesicle trafficking to the plasma membrane and plays a role in an ABA signaling cascade (Leyman et al., 1999; Geelen et al., 2002). Blatt and colleagues are continuing to investigate the link between vesicle trafficking and stomatal movement to determine protein partners that interact with Nt-SYR1 and how this SNARE protein regulates ion channels.

Fedora Sutton (South Dakota State University, Brookings) also is using the *Xenopus* oocyte system to search for an ABA receptor and decipher this signaling pathway. Sutton offered the valuable reminder that two very similar cells lying in close proximity can have very different—even opposite—functions at one time. Coming from a background in neurobiology, she gave the example of the activity of G-protein gated ion channels in presynaptic and postsynaptic nerve cells, which exhibit completely opposite behavior as a nerve impulse is perceived and propagated from cell to cell. She suggested that guard cells and mesophyll cells in plant leaves could be likened to presynaptic and postsynaptic cells. For example, during dehydration, ABA levels increase and channels are activated that cause potassium ions to leave guard cells and enter adjacent mesophyll cells.

Sutton's group is using mRNA isolated separately from guard cells and mesophyll cells in the heterologous *Xenopus* expression system to look for signal transduction components that are associated specifically with the inward-rectifying K^+ channel in guard cells and the outward-rectifying K^+ channel in mesophyll cells (Sutton et al., 2000). They have cloned a guard cell cDNA library that they believe contains a cDNA that encodes an ABA receptor; now they have to isolate the cDNA and demonstrate that it functions as an ABA receptor in planta.

ABA SIGNALING AND RNA PROCESSING

RNA processing is a critical feature of post-transcriptional gene expression in all eukaryotes. Because the majority of genes (~80% of plant genes) contain introns, alternative mRNA splicing has the potential to generate tremendous diversity at the level of the proteome. Evidence is accumulating that RNA processing plays an important role in the regulation of ABA signaling (McCourt, 2001). This is sure to become a hot topic in plant research in the near future.

Julian Schroeder (University of California–San Diego, La Jolla) and colleagues isolated a new recessive ABA-hypersensitive mutant, *abh1*, which shows ABA-hypersensitive inhibition of seed germination, ABA-hypersensitive

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increases in cytosolic calcium in guard cells, stomatal closure, and reduced wilting under drought stress. The *ABH1* gene was found to encode a nuclear mRNA cap binding protein that functions in RNA processing (Hugouvieux et al., 2001). ABH1 is proposed to act as a modulator of ABA signaling by influencing transcript processing of early ABA signaling regulatory factors.

Another ABA signaling protein with a putative function in RNA splicing is SAD1, which was discussed by Jian-Kang Zhu (University of Arizona, Tucson). The *sad1* (supersensitive to ABA and drought) mutant shows ABA-hypersensitive seed germination and increased expression of the luciferase reporter under the control of the ABA-responsive promoter *rd29A*. *SAD1* encodes a protein that is similar to a component of the spliceosome in humans, suggesting that SAD1 may affect ABA responses via effects on RNA metabolism (Xiong et al., 2001).

A FLAGELLIN RECEPTOR?

Elicitors are microbial compounds that evoke an active defense response in plants. Among these elicitors is bacterial flagellin. Various plant species have a highly sensitive perception system for the most highly conserved part of bacterial flagellin, a 22–amino acid peptide (flg22). Thomas Boller (University of Basel, Switzerland) discussed genetic and biochemical approaches to identify the flagellin receptor in plants. Flagellin activates a MAPK cascade in Arabidopsis that includes AtMPK6. The Wassilewskija ecotype is insensitive to flagellin as a result of a mutation at the *FLS1* locus, which remains unidentified. Flagellin-insensitive mutants were recovered from the sensitive ecotype Landsberg *erecta*, which led to the identification of another gene, *FLS2*, which encodes a Leu-rich repeat (LRR) receptor kinase. Binding studies using a radiolabeled derivative of flg22 showed that flagellin binding was abolished in insensitive ecotypes that carry a mutation in *FLS1* and in *fls2* mutants of sensitive ecotypes, suggesting that these two genes encode components of a flagellin receptor (Bauer et al., 2001).

ABIOTIC STRESS SENSORS

Norio Murata (National Institute for Basic Biology, Okazaki, Japan) described a systematic approach to the identification of cold, salt, and osmotic stress sensors in the cyanobacterium *Synechocystis*. To search for a cold sensor, Murata's group undertook systematic mutagenesis of each of 43 putative His kinases in the *Synechocystis* genome and screened for cold-sensor mutants by monitoring the expression of the luciferase reporter driven by the cold-inducible *desB* promoter. Mutations in several genes produced mutants that did not respond to cold, including *Hik33* and *Hik19*. *Hik33* encodes a protein with a His kinase domain and a domain that might be involved in dimer formation; thus, it is a good candidate for a cold sensor. *Hik19*, on the other hand, may be a good candidate for a cold signal transducer. In addition to 43 His kinases, the *Synechocystis* genome contains 40 response regulators and 11 Ser/Thr kinases as well as numerous transcription factors that may play roles in signaling. Murata's group plans to continue with a systematic genomics approach to inactivate all of these genes to search for various kinds of signal sensors and transducers.

DEFENSE SIGNALING

Plant biologists now talk about "innate immunity" in plants, which is likened to immune responses in mammals and insects. A shared characteristic of innate immunity in animals is the recognition of infectious, "nonself" agents by Toll/Interleukin receptor (TIR) or TIR-like proteins with extracellular LRR domains and the activation of MAPK cascades (Asai et al., 2002). Numerous plant disease resistance (*R*) genes encode nucleotide binding site (NBS)–containing LRR proteins that contain either a TIR domain or a coiled-coil (CC) domain and function as receptors of pathogen-encoded effector molecules (e.g., avirulence proteins and other elicitors of plant defense) (Dangl and Jones, 2001).

RPM1 is a CC-NBS-LRR protein in Arabidopsis that confers resistance to *Pseudomonas syringae* strains carrying either of the avirulence (*avr*) genes *avrRpm1* or *avrB*. RPM1 was found to be membrane localized and to be degraded coincident with the onset of the hypersensitive response (HR), suggesting the involvement of a negative feedback loop controlling the resistance response (Boyes et al., 1998). Jeff Dangl (University of North Carolina, Chapel Hill) reported on the isolation of RIN4, an RPM1-interacting protein that also appears to be localized to the plasma membrane and is required for *RPM1*-mediated HR after infection with *P. syringae* (Mackey et al., 2002). RIN4 also interacted with the AvrRpm1 and AvrB proteins in plant cells, and this interaction was associated with the phosphorylation of RIN4. These data suggest that RIN4 functions as a bridge or part of a receptor complex between Avr proteins and RPM1.

Barbara Baker (U.S. Department of Agriculture Plant Gene Expression Center/University of California–Berkeley, Albany, CA) reported on *N* gene–mediated resistance to *Tobacco mosaic virus* (TMV) in solanaceous plants (e.g., tobacco, tomato, and potato). Tobacco plants that carry a functional *N* gene, which encodes a TIR-NBS-LRR protein, produce a classic HR to TMV infection and are resistant to disease, whereas tobacco plants lacking *N* do

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not show HR and display disease characteristics. Tomato plants normally sensitive to TMV were made resistant by introducing three copies of the tobacco *N* gene. *NNN* tomato plants were subjected to fast neutron mutagenesis, and progeny were screened for *N* suppressor mutants. One of the interesting mutants isolated was *son1*, which showed enhanced susceptibility to TMV and to fungal pathogens such as *Verticillium* and *Fusarium*. SON1 thus has a possible role as a shared signaling component of a number of defense response pathways. Further characterization and cloning of this gene should provide valuable information on defense signaling networks.

Baker and colleagues also are investigating MAPK signaling in disease resistance in solanaceous plants by silencing selected MAPK genes with the use of a virus-induced gene-silencing system. Silencing of *NPK1*, a MAPKKK required for cytokinesis in tobacco (Nishihama et al., 2001), resulted in the attenuation of the *N*-mediated and *Bs2*-mediated disease resistance pathways, suggesting an additional role in plant defense responses.

Another class of *R* gene is represented by the tomato Cf proteins, which are membrane-anchored glycoproteins that carry a transmembrane domain and extracellular LRR domains. The fungus *Cladosporium fulvum* secretes Avr peptides that elicit a *Cf*-dependent defense response. Jonathan Jones (John Innes Centre and Sainsbury Laboratory, Norwich, UK) discussed a genetic approach to investigating *RCR* (*Required for Cf Resistance*) genes. *RCR3*, which is required for *Cf2* but not *Cf5* or *Cf9* function, encodes a secreted Cys protease of the papain family, and its expression is increased in older plants and in response to infection by *C. fulvum*. RCR3 was found to be a functional protease, and domain-swapping experiments between *Cf2* and *Cf9* suggested that it interacts with the LRR domain of Cf2. Interestingly, the *C. fulvum* Avr9 peptide has similarity to carboxypeptidase inhibitors, suggesting that some Avr proteins might be proteinase inhibitors.

LIGHT SIGNALING

Peter Quail (U.S. Department of Agriculture Plant Gene Expression Center/University of California–Berkeley) is investigating the transcriptional network of phytochrome signaling pathways using microarray analysis of *phyA* and *phyB* mutants of Arabidopsis. Work to date has been conducted using microarrays containing sequences corresponding to ~8000 genes; the data can be examined at http://www.pgec.usda.gov/quail/phyA.html. The experiments eventually will be repeated and extended with "full-genome" arrays. Experiments with the *phyA* and *phyB* mutants led to the identification of a set of genes involved in the switch from heterotrophic to autotrophic growth coincident with the deetiolation of seedlings. Quail's group is interested in identifying the phytochrome signal transduction "master regulators," genes that interact with phytochromes and regulate the major transcription factors that control downstream pathways (Tepperman et al., 2001).

Klaus Harter (University of Freiburg, Germany) reported on the interaction of the response regulator protein ARR4 with phyB. Response regulators are part of a conserved multistep phosphorylation signaling mechanism in eukaryotes. ARR4 was found to interact with the extreme N terminus of phyB and to modulate phyB response in a phosphorylation-dependent manner. Plants overexpressing ARR4 displayed enhanced sensitivity to red light, whereas *Spm*-insertion ARR4 knockout plants and plants expressing a nonphosphorylable ARR4 showed reduced red light sensitivity. ARR4 expression was induced by cytokinin as well as red light, suggesting its involvement in regulating the interactions between light and hormonal signaling pathways.

The chromophore domain of phytochrome (in particular the N-terminal region, where ARR4 binds phyB) undergoes major structural change during photoconversion. Hiroko Hanzawa (Hitachi Advanced Research Laboratory, Saitama, Japan), together with Masaki Furuya and colleagues, is investigating the hypothesis that chromophore structure determines the photosensory specificity of phyA and phyB. Synthetic phytochromobilin (corresponding to native phyA chromophore) and phycocyanobilin were supplied exogenously to *phyA phyB* chromophore-deficient double mutants; it was found that phyB-mediated responses were restored by both synthetic chromophores, whereas phyA-mediated responses occurred only when phytochromobilin was supplied. Phycocyanobilin appears to lack the capacity for interaction with a specific amino acid of the phyA apoprotein (Hanzawa et al., 2002).

Hanzawa also reported on the translocation of phyA and phyB into the nucleus during the light response, which appears to be a common step in phytochrome signaling. Hanzawa and colleagues detected some immunocytochemical differences between the phyA and phyB responses. In the dark, both phyA and phyB are distributed throughout the cytoplasm. When exposed to continuous red light, nuclear translocation of phyA begins within a few minutes and then disappears by 4.5 hr, whereas translocation of phyB to the nucleus reaches a maximum only after 6 hr.

CAMELEONS REVEAL THE IMPORTANCE OF CALCIUM OSCILLATIONS

Many studies have shown that various stimuli are correlated with oscillations

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in cytosolic free calcium [$(Ca^{2+})_c$], which help to define specific "calcium signatures." For example, Peter Hepler (University of Massachusetts, Amherst) presented data—and wonderful movies—showing a correlation between distinct growth pulses and $(Ca^{2+})_c$ oscillations during pollen tube growth. However, a fundamental question regarding calcium oscillations is whether they are required for specific responses.

Gethyn Allen (University of California–San Diego), along with Julian Shroeder and colleagues, is beginning to address this question with the use of "cameleons" and various mutants of Arabidopsis to follow calcium oscillations in guard cells. Cameleons are chimeric proteins consisting of cyan fluorescent protein linked via calmodulin and the calmodulin binding M13 peptide to yellow fluorescent protein (Miyawaki et al., 1999). On binding to $(Ca^{2+})_c$, the cameleon undergoes a conformational change that brings the two fluorescent moieties into close proximity and changes the emission spectrum. Binding is reversible and allows for real-time measurements of changes and oscillations in $(Ca^{2+})_c$. For example, in the vacuolar H^+-ATPase mutant *deetiolated3* (*det3*), transformation with cameleon showed that $(Ca^{2+})_c$ failed to oscillate in isolated guard cells and that stomatal closure was abolished under a range of external calcium concentrations. However, exogenous ABA induced both $(Ca^{2+})_c$ oscillations and stomatal closure in *det3* and wild-type cells, suggesting that oscillations may be required for the response.

Further evidence was provided by imposing calcium "clamps" on isolated guard cells, which consist of a depolarizing (high-potassium, low-calcium) or a hyperpolarizing (low-potassium, high-calcium) buffer (Allen et al., 2001). $(Ca^{2+})_c$ oscillations were induced in *det3* guard cells by oscillatory changes in the buffer clamps, and the imposed oscillations were sufficient to bring about stomatal closure. The guard cells even seemed to have the ability to "count," because increasing the number of oscillations increased the degree of stomatal closure. The frequency of oscillations also had a significant effect, with a clear optimum of maximal stomatal closure corresponding to a period of ~10 min. Mutants such as *gca2*, which affect the frequency of oscillation and the stomatal response, also have been identified. This exciting work is illustrative of how innovative techniques, coupled with genetic, biochemical, and physiological analysis, are beginning to reveal the secrets of signaling networks.

SPECIAL NOTE: IN MEMORIAM OF GETHYN ALLEN

Gethyn Allen, whose work appears on the cover and is discussed above, died on March 31, 2002. Allen was a postdoc and senior research associate with Julian Schroeder (University of California–San Diego) for the last 4 years and previously had worked with Dale Sanders (University of York, UK). As is apparent from his numerous publications in the last few years, Allen was performing innovative and exciting work in plant cell signaling. His creative research opened new doors in understanding how calcium oscillations can encode specific outputs, with scientific implications reaching beyond plant biology. In his most recent research, he obtained results that provided experimental support for a long-standing model in eukaryotic calcium signaling. To quote Julian, Gethyn was "one of the very most creative scientists" he had met in his career and also "so caring and brought joy to all who knew him." His presence in the plant science community will be sorely missed.

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