Plant, Cell & Environment

Plant, Cell and Environment (2012) **35**, 53–60 doi: 10.1111/j.1365-3040.2011.02426.x

ABA signal transduction at the crossroad of biotic and abiotic stress responses

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ABSTRACT

Abscisic acid (ABA) regulates key processes relevant to seed germination, plant development, and biotic and abiotic stress responses. Abiotic stress conditions such as drought induce ABA biosynthesis initiating the signalling pathways that lead to a number of molecular and cellular responses, among which the best known are the expression of stress-related genes and stomatal closure. Stomatal closure also serves as a mechanism for pathogen defence, thereby acting as a platform for crosstalk between biotic and abiotic stress responses involving ABA action. Significant advances in our understanding of ABA signal transduction have been made with combination of approaches including genetics, biochemistry, electrophysiology and chemical genetics. Molecular components associated with the ABA signalling have been identified, and their relationship in the complex network of interactions is being dissected. We focused on the recent progress in ABA signal transduction, especially those studies related to identification of ABA receptors and downstream components that lead ABA signal to cellular response. In particular, we will describe a pathway model that starts with ABA binding to the PYR/PYL/RCAR family of receptors, followed by inactivation of 2C-type protein phosphatases and activation of SnRK2-type kinases, and eventually lead to activation of ion channels in guard cells and stomatal closure.

Key-words: drought; hormones; pathogens; stomata.

ABSCISIC ACID IN DROUGHT AND PATHOGEN RESPONSES

In the natural environment, plants are constantly challenged with biotic and abotic stresses, such as various pathogens, drought and high salinity. Plant hormone abscisic acid (ABA) serves as a chemical signal in response to these environmental factors and triggers changes in a number of plant physiological and developmental processes, leading to adaptation to the stress conditions (Finkelstein, Gampala & Rock 2002; Robert-Seilaniantz *et al*. 2007; Ton, Flors & Mauch-Mani 2009). As many review articles cover the topic of ABA action in abiotic stress responses (Chinnusamy, Gong & Zhu 2008; Popko *et al*. 2010; Sirichandra *et al*. 2010; Wilkinson & Davies 2010; Joshi-Saha, Valon & Leung 2011), we focus on recent studies on ABA action in the crosstalk of biotic and abiotic responses through regulation of stomatal movements.

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ABA-regulated stomatal closure in drought responses

Drought adversely affects plant growth and causes severe losses in crop production in agriculture. Plants lose water primarily through the stomata on the leaves. ABA is a key hormone that controls water status and stomatal function. Upon drought conditions, plants produce and accumulate more ABA that induces stomatal closure, thus conserving water. The cellular and molecular mechanisms underlying ABA-induced stomatal closure have been extensively studied and reviewed previously (Luan 2002; Assmann 2003; Wasilewska *et al*. 2008; Cutler *et al*. 2010; Hubbard *et al*. 2010; Popko *et al*. 2010; Wilkinson & Davies 2010). Typically, ABA level is modulated by the balance between ABA biosynthesis and ABA catabolism (Nambara & Marion-Poll 2005; Nilson & Assmann 2007). The 9-*cis*-epoxycarotenoid dioxygenase (*NCED*) genes encode key enzymes for ABA biosynthesis. Under drought stress condition, the *AtNCED3* gene is strongly induced and disruption of *AtNCED3* results in a decreased ABA level leading to higher transpiration rate in the mutant *Arabidopsis* plants (Iuchi *et al*. 2001). In contrast, overexpression of an *NCED* gene leads to higher levels of ABA accumulation and decrease in transpiration (Schwartz *et al*. 1997; Tan *et al*. 1997; Thompson *et al*. 2000; Iuchi *et al*. 2001; Schwartz, Qin & Zeevaart 2003). For catabolism, *CYP707A1* to *A4* genes encoding 8′-hydroxylase play a key role in ABA oxidation (Kushiro *et al*. 2004; Umezawa *et al*. 2006). Genetics analysis showed that *cyp707a3-1* mutant contained higher ABA levels, showed decreased transpiration rate, leading to drought-tolerant phenotype (Umezawa *et al*. 2006).

For stomatal control, guard cell turgor is a major parameter that is regulated by ionic fluxes across the cell membranes through K^+ and anion channels. These ion channels in guard cells thus become the major targets for regulation by a number of environmental factors such as light/dark, drought, CO_2 levels and so on. In the case of drought, ABA serves as a primary chemical signal that induces stomatal

closure through second messengers such as reactive oxygen species, nitric oxide, Ca^{2+}, followed by activation and inactivation of protein kinases/phosphatases that further target the ion channels (Schroeder & Hagiwara 1989; Grabov & Blatt 1998, 1999; Hamilton *et al*. 2000; Pei *et al*. 2000; Garcia-Mata & Lamattina 2001; Garcia-Mata *et al*. 2003; Sokolovski *et al*. 2005; Negi *et al*. 2008; Vahisalu *et al*. 2008, 2010; Ward, Maser & Schroeder 2008; Lee *et al*. 2009; Chen *et al*. 2010;). The most recent model that depicts signalling pathway from ABA to stomatal closure will be discussed in the later sections.

ABA-regulated stomatal closure in pathogen response

The stomatal closure not only leads to water conservation during drought but also serves as a defence mechanism in preventing pathogen invasions. Recently, studies have demonstrated that ABA also plays an important role in pathogen response, and the signalling pathways overlap significantly between pathogen resistance and abiotic stress tolerance. In addition to ABA action in stomatal closure that limits pathogen access, this hormone affects pathogen responses by interacting with other hormones associated with plant defence mechanisms (Anderson *et al*. 2004; Melotto *et al*. 2006; Asselbergh, De Vleesschauwer & Hofte 2008; Melotto, Underwood & He 2008; Mosher *et al*. 2010).

Plants have innate physical and biochemical barriers that effectively protect them from a variety of pathogens. However, most pathogens have mechanisms that allow them to overcome or circumvent plant mechanical barriers, including the cell wall, leading to successful infection in plants. A pathogen entrance to plant interior is the first step in the effective establishment of infection. Some fungal pathogens can directly penetrate plant tissue by applying mechanical force or utilizing cell-wall degrading enzymes, but bacterial pathogens require passage to enter plant tissue (Mendgen, Hahn & Deising 1996; van Kan 2006; Ton *et al*. 2009). Generally, bacterial pathogens employ natural openings to enter into plant tissues, such as stomata, hydathodes and lenticels (Melotto *et al*. 2008). Among these, the stomata are a major entry point for bacterial pathogens. In addition to earlier studies, Melotto *et al*. (2006) also showed that stomatal closure directly restricts bacterial pathogenic invasion of plants. The pathogen-associated molecular pattern (PAMP) is recognized by plants and triggers plant innate immunity (Jones & Dangl 2006). Among the responses triggered by PAMPs is stomatal closure that restricts pathogen entrance (Melotto *et al*. 2006). By using *ost1* mutant that fails to respond to ABA (Mustilli *et al*. 2002) and ABA-deficient *aba3-1* mutant (Leon-Kloosterziel *et al*. 1996), studies showed that stomatal closure is not induced by PAMPs in these mutants (Melotto *et al*. 2006). It is thus proposed that PAMP-induced stomatal closure requires active ABA signal transduction pathway in guard cells. Therefore, ABA has a positive effect on disease resistance through induction of stomatal closure.

ABA regulation of pathogen response through interplay with other hormones

A number of plant hormones, including salicylic acid (SA), jasmonic acid (JA) and ethylene, function in pathogen defence mechanisms. Typically, SA is associated with systemic acquired resistance (SAR) and resistance to biotrophic pathogens, whereas JA and ethylene are associated with induced systemic resistance (ISR) and resistance to necrotrophic pathogens (Alvarez *et al*. 1998; Glazebrook 2005; Pieterse *et al*. 2009). ABA is connected to the SA, JA and ethylene signalling pathways, and acts either synergistically or antagonistically with these hormones (Anderson *et al*. 2004; Mosher *et al*. 2010).

Studies suggest that ABA negatively regulate SA-mediated pathogen responses. For instance, overexpression of *NCED* genes, including *NCED2*, *NCED3* and *NCED5*, strongly induced accumulation of ABA and conferred a significant increase of bacterial growth (Fan *et al*. 2009). In addition, the *Arabidopsis* mutant *aba3-1*, which is impaired in ABA biosynthesis, showed a resistant phenotype to *Pseudomonas syringae* infection, whereas exogenous treatments with ABA led to increased susceptibility to virulent bacteria (Fan *et al*. 2009). ABA also suppressed the induction of SAR by inhibition of SA-induced gene expression (Yasuda *et al*. 2008). Collectively, the available results suggest that ABA appears to suppress SA-dependent defence mechanisms.

The negative effect of ABA on JA- and ethylene-dependent pathogen resistance has been previously assessed at gene transcription level (Anderson *et al*. 2004). The typical JA and ethylene marker genes, including *PDF1.2* and *CHI*, were induced more strongly in ABA-deficient mutants than in wild-type *Arabidopsis*, whereas these genes were down-regulated by exogenous ABA treatment (Anderson *et al*. 2004). Additionally, ABA suppressed ethylene response in rice (Xiong & Yang 2003). The *OsMPK5* gene encoding mitogen-activated protein kinase is induced by ABA and biotic and abiotic stresses (Xiong & Yang 2003). Its overexpression enhances ABA accumulation, but reduces ethylene levels, and renders plants more susceptible to the fungal pathogen *Magnaporthe oryzae* and the bacterial pathogen *Burkholderia glumae* (Xiong & Yang 2003; Asselbergh *et al*. 2008).

ABA SIGNAL TRANSDUCTION

ABA receptor

Earlier studies showed that ABA-binding proteins are present in several locations in the cell, such as plasma membrane and cytosol, suggesting that more than one ABA receptor may exist (Pedron *et al*. 1998; Zhang *et al*. 2001; Kitahata *et al*. 2005). Indeed, several ABA receptors have been identified so far, although the function of some receptors remains to be further demonstrated (Shen *et al*. 2006; Liu *et al*. 2007; Christmann & Grill 2009; Ma *et al*. 2009; Pandey, Nelson & Assmann 2009; Park *et al*. 2009). Here we focus on the pyrabactin resistance (PYR)-like (PYL)/

regulatory component of ABA receptor (RCAR) family ABA receptors that have been shown to functionally connect with the other known ABA regulators such as the PP2C-type protein phosphatases and the SnRK2-type protein kinases.

PYR/PYL or RCAR belongs to the family of the star-related lipid-transfer (START) protein and is homologous to the Bet v onefold protein (Ma *et al*. 2009; Park *et al*. 2009; Santiago *et al*. 2009b). We refer to this family of proteins as PYL/RCAR in this review. In the chemical genetics approach, pyrabactin was used as an ABA analogue to screen for mutants that were insensitive to this compound and genes encoding PYR/PYL proteins were isolated (Park *et al*. 2009). In a different approach, Ma *et al*. (2009) identified RCARs as direct interactors of ABI2. In both cases, ABA was shown to directly bind PYL/RCAR proteins, thus suggesting that they function as ABA receptors. Using triple (*pyr1:pyl1:pyl4*) and quadruple (*pyr1:pyl1:pyl2:pyl4*) mutants, genetic analysis demonstrated that these mutants displayed ABA insensitivity in germination, root growth and stomatal closure. However, ABA sensitivity was unaltered in a *pyr1* single mutant due to genetic redundancy, because the PYR/RCAR protein family includes 14 members (Park *et al*. 2009; Nishimura *et al*. 2010). Earlier genetic analysis provides crucial information for connecting PYL/RCAR function to the downstream components in the signalling process. Because ABI-like protein phosphatases, i.e. A-type PP2Cs, are negative regulators of ABA response (Gosti *et al*. 1999; Merlot *et al*. 2001; Schweighofer, Hirt & Meskiene 2004; Wasilewska *et al*. 2008) and PYL/RCAR proteins directly inhibit phosphatase activity of PP2Cs *in vitro* (Ma *et al*. 2009; Szostkiewicz *et al*. 2010), it is concluded that these PP2Cs are direct target of ABA receptors in the signalling pathway.

In addition, structural analyses (Melcher *et al*. 2009; Miyazono *et al*. 2009; Nishimura *et al*. 2009; Yin *et al*. 2009; Santiago *et al*. 2009a) show that PYL/RCAR proteins harbour a ligand-binding pocket that may play a role as an ABA binding site. In the yeast two-hybrid assays, some interactions between PYL/RCARs and their target PP2Cs are enhanced by ABA; whereas other interactions are independent of ABA (Ma *et al*. 2009; Santiago *et al*. 2009b). These results suggest that two potential models can account for the function of ABA in the PYL/RCAR complexes. One explanation is that ABA binding induces structural changes in PYR/PYLs and ABA-bound PYR/PYL can interact tightly with PP2Cs. The other explanation is that ABA did not affect the structure of any proteins, and binds only to the PYR/PYL-PP2C complex. The structural analysis of PYR1, PYL1 and PYL2 demonstrated that PYR/PYLs bind to ABA and create a surface that recognizes PP2Cs and forms a complex with PP2Cs (Melcher *et al*. 2009; Miyazono *et al*. 2009; Yin *et al*. 2009). In this complex, ABA-bound PYR/PYLs interact with a phosphatase domain of PP2Cs and inhibit PP2C activity by covering the active sites of PP2Cs. Additionally, the tryptophan residue of PP2C stabilizes this ABA-PYR/PYLs-PP2C complex and makes contact with ABA (Melcher *et al*. 2009; Miyazono *et al*. 2009; Yin *et al*. 2009). These results suggest that PYR/PYLs inactivation of PP2Cs is mediated by ABA (Ma *et al*. 2009; Park *et al*. 2009; Santiago *et al*. 2009b; Szostkiewicz *et al*. 2010), consistent with a study showing that the binding affinity between RCAR1 (PYL9) and ABA was enhanced approximately 10-fold by the presence of ABI2 (Ma *et al*. 2009). Collectively, the genetic, physiological and structural analyses strongly support the hypothesis that PYR/PYLs/RCARs are bona fide ABA receptors.

SnRK2 kinases and PP2C A-type phosphatases

Protein phosphorylation and dephosphorylation events in ABA signalling involve several protein kinases and phosphatases in plants (Lee *et al*. 2009; Vlad *et al*. 2009; Geiger *et al*. 2009). The first reported SnRK2-type kinase was PKABA1, which was isolated from wheat (Anderberg & Walker-Simmons 1992). The activity of PKABA1 is induced by ABA and is involved in the phosphorylation of the transcription factor TaABF1 (Anderberg & Walker-Simmons 1992; Johnson *et al*. 2002). Studies indicate that TaABF1suppresses ABA-induced gene expression in wheat and barley (Gomez-Cadenas *et al*. 1999; Johnson, Shin & Shen 2008). In a subsequent work, an ABA-activated serine-threonine protein kinase (AAPK) was identified in *Vicia faba*, using gel kinase assays (Li *et al*. 2000). AAPK is also an SnRK2-type kinase and is involved in stomatal closure via the regulation of plasma membrane slow anion channels (Li *et al*. 2000). An orthologue of AAPK in *Arabidopsis* was identified as Open Stomata 1 (OST1) by a genetics approach (Mustilli *et al*. 2002). The kinase activity of OST1 is prompted by ABA, but its expression is not regulated by ABA (Mustilli *et al*. 2002; Yoshida *et al*. 2002). The *ost1* mutant exhibits an ABA-insensitive phenotype and increases leaf water loss by keeping the stomata open, even under drought conditions (Mustilli *et al*. 2002; Yoshida *et al*. 2002). The OST1 also interacts with and phosphorylates ABF2 and ABF3, which bind to ABA-responsive elements (ABRE) and control ABA-regulated gene expression (Furihata *et al*. 2006; Fujii *et al*. 2009; Fujita *et al*. 2009; Sirichandra *et al*. 2010). In addition to OST1, two other SnRK2-type kinases, SnRK2.2 and SnRK2.3, are induced by ABA (Boudsocq, Barbier-Brygoo & Lauriere 2004). A *snrk2.2*/*snrk2.3* double mutant shows ABA-insensitive phenotypes in seed germination and root growth, whereas each of the single mutants has no distinguishable phenotype, due to functional redundancy (Fujii, Verslues & Zhu 2007). Each of these SnRK2-type kinases is involved in ABA signalling and is activated by ABA. A triple mutant, lacking SnRK2.2, SnRK2.3 and OST1, shows severe phenotypes indicative of defects in ABA signalling and water stress responses (Fujii & Zhu 2009; Fujita *et al*. 2009; Nakashima *et al*. 2009). The SnRK2.2 and SnRK2.3, as well as OST1, thus function as positive regulators of ABA signalling.

Genetic evidence revealed that A-type PP2Cs, such as ABI1 and ABI2, are negative regulators of ABA signalling in *Arabidopsis* (Gosti *et al*. 1999; Merlot *et al*. 2001;

Wasilewska *et al*. 2008; Rubio *et al*. 2009). Additionally, mutants in HAB1, HAB2, AHG1 and PP2CA were identified in seed germination screen as hypersensitive to ABA (Nishimura *et al*. 2004; Saez *et al*. 2004, 2006; Kuhn *et al*. 2006; Robert *et al*. 2006; Yoshida *et al*. 2006b; Rubio *et al*. 2009). The PP2C functions related to ABA were initially suggested by the dominant mutants *abi1-1* and *abi2-1*, which were associated with an ABA-insensitive phenotype in seed germination, seedling growth and stomatal closure (Koornneef, Reuling & Karssen 1984; Finkelstein & Somerville 1990; Leung, Merlot & Giraudat 1997; Allen *et al*. 1999). These dominant mutants appear to have a gain of function effect to make the protein phosphatases constitutively active, thereby leading to ABA insensitivity, a phenotype opposite to the loss-of-function mutants. The A-type PP2Cs function with certain degree of specificity and redundancy. While each mutant displays detectable ABA hypersensitivity, some double mutants show a more profound hypersensitive phenotype to ABA as compared with single mutants (Merlot *et al*. 2001; Rubio *et al*. 2009).

The PP2C–SnRK2 relationship

Several studies demonstrate functional and physical interactions between PP2C A-type phosphatases and SnRK2-type kinases. Earliest indication of such interactions came from a study by Yoshida *et al*. (2006a) who identified a physical interaction between the ABI1 and ABI2 proteins and SnRK2.6/OST1. More recent studies have established several SnRK2–PP2C interactions that clearly function in ABA signalling (Yoshida *et al*. 2006a; Fujii *et al*. 2009; Lee *et al*. 2009; Umezawa *et al*. 2009; Vlad *et al*. 2009). For example, the interaction of A-type PP2Cs with OST1 plays a role in ABA-induced stomatal closure through the control of phosphorylation status and activity of the SLAC1 anion channel in guard cells (Lee *et al*. 2009; Geiger *et al*. 2009). The physical interaction between the PP2Cs and OST1 renders inactivation of OST1 kinase and down-regulation of the SLAC1 channel required for stomatal closure. The same interactions between PP2Cs and SnRK2s also function in ABA-induced gene expression (Yoshida *et al*. 2006a; Nishimura *et al*. 2010). In this context, studies have established that PP2Cs, including ABI1 and PP2CA, inactivate OST1 by physical interaction as well as dephosphorlyation (Fujii *et al*. 2009; Lee *et al*. 2009; Umezawa *et al*. 2009).

The triple mutant *abi1-2/hab1-1/pp2ca-1* exhibits constitutive activation of SnR2.2, SnRK2.3 and OST1 (Fujii *et al*. 2009), indicating that the ABA-dependent activation of SnRK2-type kinases results from removal of the inhibitory effect of PP2Cs. Hubbard *et al*. (2010) suggests that the earliest processes occurring in the ABA signal transduction pathway comprise the interaction among PYL/RCARs, PP2Cs and SnRK2s. The interaction between PP2C (negative regulator) and SnRK2 (positive regulator) causes inactivation of SnRK2 and suppression of the signalling pathway. The ABA-dependent (and sometimes ABA-independent) binding of PYL/RCARs with the PP2Cs breaks the physical interaction and inhibition of SnRK2s by PP2Cs, leading to activation of SnRKs and de-repression of the signalling pathway (Fujii *et al*. 2009; Lee *et al*. 2009; Ma *et al*. 2009; Park *et al*. 2009; Umezawa *et al*. 2009).

ABA signalling to ion channel regulation and stomatal closure

ABA-induced stomatal closure involves regulation of several ion channels at the plasma membrane and tonoplast (Schroeder, Kwak & Allen 2001a). Typically the S-type anion channels and outward K-channels are activated by ABA, whereas inward K^+ channels are inactivated, leading to net ionic efflux and decrease in guard cell turgor (Schroeder *et al*. 2001b; Nilson & Assmann 2007; Ward *et al*. 2008; Wasilewska *et al*. 2008; Kim *et al*. 2010). When ABA levels increase upon drought or other stress conditions, anion efflux via the anion channels induces depolarization and activation of outward K-channels (Ward *et al*. 2008; Kim *et al*. 2010). Reduced ionic concentration in the cell causes water efflux and reduces guard cell volume, thereby leading to stomatal closure (Ward, Pei & Schroeder 1995; Wasilewska *et al*. 2008).

The ABA signalling components ABI1 and ABI2 and OST1/AAPK have long been implicated in the regulation of guard cell ion channels (e.g. Pei *et al*. 1997; Li *et al*. 2000). In the context of current understanding of ABA signalling described previously, a model is proposed to connect ABA signal to ion channel regulation and stomatal closure (Fig. 1). Normally, the levels of ABA and PYL/RCAR proteins are low. The A-type PP2C phosphatases bind to the SnRK2-type kinases and inhibit their activity. The SLAC1 and other anion channels may be dephosphorylated and their activity remains low. In response to drought or pathogen or other stress factors, ABA levels are up-regulated to reach certain threshold that bind to PYL/RCAR-type receptors and enhances the interaction between PYL/RCAR with PP2Cs, thereby activating SnRK2s that in turn interact with and phosphorylate SLAC1 and other channels resulting in activation of the channels and efflux of the anions. Depolarization of guard cell membrane activates outward K-channels leading to drop in guard cell turgor and stomatal closure (Pei *et al*. 1997; Lee *et al*. 2009; Geiger *et al*. 2009, 2010). Through stomatal closure, the ABA-induced signalling pathway intersects with both abiotic stress factors (e.g. drought) and biotic stress factors (such as pathogen).

Concluding remarks

The plant hormone ABA activates a complex signalling network that regulates numerous physiological processes including seed dormancy, development, and responses to biotic and abiotic stresses. We discuss recent literature and attempt to place ABA signalling in the crossroads of biotic and abiotic stress responses. Although the crosstalk between the signalling pathways may be too complex to dissect at this point, it is certain that ABA-induced stomatal

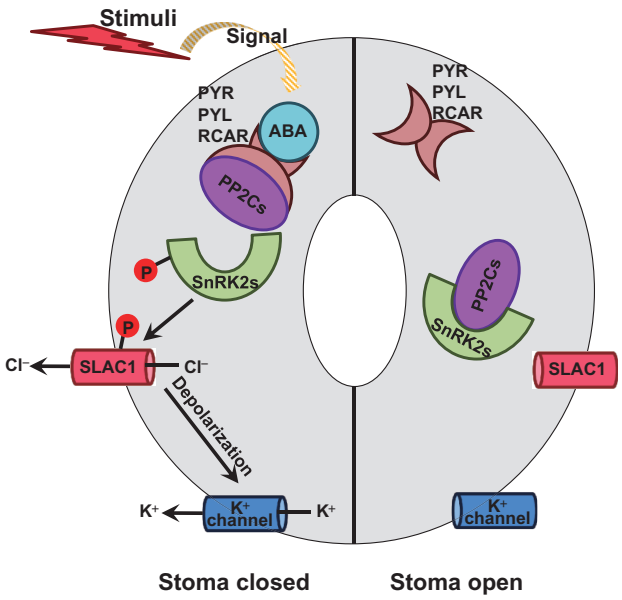

Figure 1. Summary of abscisic acid (ABA) signalling pathway. This model focuses on regulation of stomatal closure by ABA signal transduction. The ion channels are regulated by protein–protein interaction relay involving PYR/PYL/RCAR-PP2Cs-SnRK2s-Channels. In the presence of ABA, PYR/PYL/RCARs bind to PP2Cs and inhibit phosphatase activity, leading to activation of SnRK2s. Active SnRK2s interact with and phosphorylate ion channels (such as SLAC1) and lead to stomatal closure. In the absence of ABA, PP2Cs bind to SnRKs and inhibit kinase activity, which prevents subsequent phosphorylation and activation of ion channels, and stomata keep open. PYL, PYR-like; PYR, pyrabactin resistance; RCAR, regulatory component of ABA receptor.

closure serves as a centre stage for such crosstalk between biotic and abiotic stress signals. On one hand, stomatal closure functions to conserve water in response to abiotic stress such as drought; on the other hand, closed stomata act as a barrier for pathogen invasion. In both cases, ABA signalling pathway is critical for integrating signals to cellular responses. In this regard, recent work on the signalling mechanism in response to ABA is particularly relevant to agriculture as it provides insights into molecular events that function in both pathogen resistance and abiotic stress tolerance. The molecular components in ABA signalling, including PYR/PYL/RCAR, PP2Cs, SnRKs and SLAC1 channel, will be tentative targets for genetic engineering of stress-tolerant crops. They also provide a starting point for further dissecting the complexity of the signalling events. For instance, there are 14 members of PYR/PYL/RCAR receptors, 6–9 members of the A-type PP2Cs and at least 3 members of the SnRK2s involved in ABA signalling. Therefore, more than 200 possible combinations exist to form different PYL/RCAR-PP2C-SnRK2 complexes; each combination may regulate the same or different downstream targets depending on factors such as cell type, ABA concentrations and subcellular locations of the targets, resulting in enormous complexity in fine-tuning of the regulation. Future research on this fine-tuning of signalling pathways will help us further understand crosstalk between biotic and abiotic stress responses.

ACKNOWLEDGMENTS

The work in authors' laboratories was supported by a grant from the Biogreen21 Program (PJ008222) Korean Rural Development Administration (to S.C.L.) and the US National Science Foundation and Korean WCU Program of National Research Foundation (to S.L.).

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Received 19 June 2011; received in revised form 6 September 2011; accepted for publication 9 September 2011