Journal of Plant Biology, April 2007, 50(2) : 117-121 **REVIEW**

Recent Advances in ABA Signaling

Soo Young Kim*
Department of Molecular Biotechnology, College of Agriculture and Life Sciences,
Chonnam National University, Gwangju 500-757, Korea

Abscisic acid (ABA) is a major plant hormone that controls germination, seedling growth, and seed development. During the vegetative phase, ABA plays a key regulatory role in adaptive responses to common abiotic stresses, such as drought, high salinity, and cold. In seeds, ABA modulates the synthesis of storage components and prevents the precocious germination of embryos. ABA-regulated processes are critical for plant growth and survival, especially under unfavorable environmental conditions. Numerous genetic and biochemical studies to delineate signal transduction pathways have led to the identification of a large number of ABA signaling components. However, our knowledge about specific response pathways is still fragmentary. Over the past several years, significant progress has been made in identifying key regulators of early events in the ABA response. In this short review, new advances in ABA signaling research, especially those focused on ABA receptors, will be summarized.

Keywords: abiotic stress, abscisic acid, hormone receptor, signal-transduction

ROLE OF ABA AND BRIEF HISTORY OF ABA SIGNALING RESEARCH

Abscisic acid is a sesquiterpenoid with one asymmetric carbon at C-1 (Fig. 1; Zeevaart and Creelman, 1988). Its naturally occurring form is *S*-(+)-ABA, although *R*-(–)-ABA has biological activity. As a major phytohormone, ABA plays a key role in regulating numerous aspects of plant growth (Finkelstein et al., 2002; Xiong et al., 2002). During early stages, it controls seed germination and allows post-germination growth only in favorable environments (Lopez-Molina et al., 2001). In seeds, ABA modulates the biosynthesis of storage components such as lipids and proteins. Although it is generally known as a negative regulator, more recent studies have clearly demonstrated that ABA is required for normal growth (Cheng et al., 2002).

In seedlings, one of the major roles of ABA is to mediate adaptive responses (Xiong et al., 2002). Its level increases under water-deficit and other abiotic stress conditions, and it triggers several physiological processes necessary for sustained survival. For instance, when the water supply is limited, ABA promotes stomatal closure to prevent water loss via transpiration (Schroeder et al., 2001). This particular action is fast and does not require changes in gene expression patterns. Other ABA-dependent adaptive responses, however, generally do entail such alterations. More than 1000 ABA-responsive genes have been identified through genome-wide expression analyses (Hoth et al., 2002; Kreps et al., 2002; Takahashi et al., 2004). Approximately one-half of those genes are up-regulated by ABA, whereas the other 50% are down-regulated (Hoth et al., 2002). These genomics studies have also shown the variety of genes regulated by ABA, such as those involved in transcription, signaling, protein synthesis and destination, energy metabolism, cell rescue, and cell division.

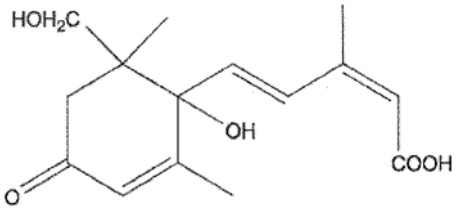

Figure 1. Structure of S-(+)-abscisic acid.

*Corresponding author; fax +82-62-972-5085
e-mail sooykim@chonnam.ac.kr

Numerous ABA signaling components have been identified by forward genetics screening (Finkelstein et al., 2002; Himmelbach et al., 2003). These include such signaling intermediates as secondary messengers, kinases, phosphatases, and transcription factors. ABA signaling mutants generally fall into two classes: ABA-insensitive and ABA-hypersensitive. Now that the entire genome sequences are available for *Arabidopsis* and rice, new components of ABA signal transduction are being identified at a remarkable speed (Xie et al., 2005, for the most recent review). Notwithstanding this rapid advancement, our knowledge about specific ABA response pathways is still fragmentary. Nevertheless, several key components of ABA-perception steps have been reported recently (Table 1), and these will be briefly summarized here.

FCA

Despite many efforts, no ABA receptors have yet been identified by genetic screens for ABA response mutants. However, through biochemical means, two ABA receptors have been reported, including FCA, an RNA-binding protein that regulates flowering time (Macknight et al., 1997; Razem et al., 2006).

The protein was first isolated from barley aleurones by Razem et al. (2004), who screened a cDNA expression library with an antibody specific to ABA. The resultant ABA antibody-binding protein, named ABAP1, is a 412-amino acid polypeptide that binds ABA with high affinity. Its *in*

Table 1. ABA receptors and other ABA signaling components implicated in the ABA-perception step.

Gene	AGI number	Known functions	References
FCA	At4g16280	RNA-binding protein, flowering-time control	Macknight et al., 1997; Razem et al., 2006
ABAR/CHLH	At5g13630	Mg-chelatase H subunit, chlorophyll biosynthesis, plastid-nucleus signaling	Nott et al., 2006; Shen et al., 2006
RPK1	At1g69270	Leucine-rich repeat receptor-like kinase	Hong et al., 1997; Osakabe et al., 2005
GCR1	At1g48270	G protein-coupled receptor	Pandey and Assmann, 2004; Pandey et al., 2006

vitro activity is highly stereo-specific, i.e., it binds to the (+)-ABA, but not to (–)-ABA, trans-ABA, or other ABA analogs. The deduced amino acid sequence of ABAP1 is similar to that of FCA. Razem et al. (2006) have since then showed that FCA also binds ABA with high affinity (Kd = 19 nM) *in vitro*, and that this binding is specific to the physiologically active form of ABA, (+)-ABA.

Such ABA-binding affects FCA function *in planta* (Razem et al., 2006). Normally, this protein acts as a positive regulator of flowering time in an autonomous pathway (Macknight et al., 1997). Flowering is promoted by inhibiting mRNA accumulation of the floral repressor FLC, which is a MADS-box protein (Michaels and Amasino, 1999; Sheldon et al., 1999). To repress FLC expression, FCA associates with FY, an RNA-processing factor (Simpson et al., 2003). Razem et al. (2006) have revealed that ABA inhibits this interaction between FCA and FY, thus preventing any complex formation between the two (Fig. 2). The physiological significance of this ABA-dependent disruption has been demonstrated by investigating the effects of abscisic acid on FCA function in *Arabidopsis*. FCA auto-regulates its expression with a negative feedback loop, in which it prevents its own expression by promoting the premature cleavage of its pre-mRNA. In *fca* or *fy* mutants, production of this truncated FCA mRNA is therefore abrogated. This mutant phenotype is phenocopied via exogenous application of ABA to intact *Arabidopsis* plants, indicating that ABA disrupts the normal function of FCA *in vivo*. Moreover, ABA delays flowering in wild-type

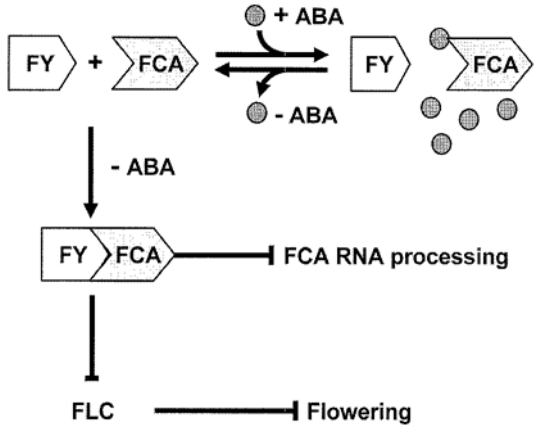

Figure 2. ABA inhibits functioning of FCA. Under normal conditions, FCA associates with FY to inhibit expression of FLC, a negative regulator of flowering time. Additionally, the FCA-FY complex regulates processing of FCA pre-mRNA to produce truncated FCA RNA. ABA-binding to FCA inhibits formation of FCA-FY complex. As a result, flowering is delayed and production of full-length FCA mRNA is enhanced.

plants, behavior that is also observed in *fca* mutants. Consistent with the *in vitro* binding data, the inhibitory effect of ABA is again stereo-specific, with the effect being exerted by (+)-ABA, but not by either (-)-ABA or trans-ABA.

As mentioned above, ABA affects several physiological processes, including germination, root growth, seed development, stomatal movement, and stress responses. The results of Razem et al. (2006) indicate that FCA affects only a subset of these. They observed that ABA-inhibition of lateral root formation (de Smet et al., 2003) is abnormal in *fca* mutants. However, other aspects of the ABA response, i.e., seed germination and stomatal-opening, are normal in those mutants. In addition, FCA function is normal in *abi-1* and *abi-2* mutants, indicating that FCA works in a pathway different from that of ABI1 or ABI2.

Mg-CHELATASE H SUBUNIT

Zhang et al. (2002) have isolated an ABA-binding protein from the leaf epidermis of the broad bean. This protein binds ABA stereo-specifically: (+)-ABA, but not (–)-ABA. In addition, when guard cell protoplasts are pre-treated with an antibody raised against the protein, ABA-induced phospholipase D activity is decreased. This suggests that the ABA-binding protein may function as an ABA receptor.

More direct evidence that the ABA-binding protein is an ABA receptor has been provided by the study of its *Arabidopsis* homolog (Shen et al., 2006). ABAR (for ABA receptor) is the putative H subunit (CHLH) of the magnesium-protoporphyrin-IX chelatase (Mg-chelatase) in *Arabidopsis*. CHLH has a chloroplast-to-nucleus signaling function in addition to its enzymatic role in chlorophyll biosynthesis (Nott et al., 2006). Studies carried out with both recombinant and natural proteins have shown that ABAR binds ABA stereo-specifically, with high affinity (Kd=32 nM).

The *in vivo* proof for the involvement of ABAR in the ABA response has come from analyses of its overexpression and RNAi lines (Shen et al., 2006). Overexpression of ABAR results in hypersensitivity to ABA during seed germination and seedling growth, and in stomatal movement, transpiration, and drought tolerance. In contrast, ABAR RNAi lines display opposite phenotypes, i.e., ABA insensitivity and drought sensitivity. ABA content is normal in transgenic plants, suggesting that their phenotypic alterations do not result from changes in their levels of ABA. Furthermore, homozygous ABAR knockout plants are lethal, and *abar* seeds are deficient in lipid and protein bodies. Together, these data indicate that ABAR is a positive regulator of ABA signaling throughout all stages of plant development.

CHLH is one of the three subunits that constitute Mg-chelatase, which catalyses the insertion of magnesium into protoporphyrin IX to form Mg-protoporphyrin IX (Walker and Willows, 1997). This subunit is also involved in plastid-to-nucleus signaling functions (Nott et al., 2006). ABA binds to CHLH independently of the protoporphyrin IX and, thus, the ABA signal-perception is distinct from its enzymatic activity (Shen et al., 2006). In addition, analyses of various mutants defective in this plastid-to-nucleus signaling have shown that the ABA signal-perception function of ABAR is distinct from retrograde plastid-to-nucleus signaling.

The ABAR/CHLH gene, encoded by a single-copy gene in the *Arabidopsis* genome, is highly conserved among plants and even bacteria. In *Arabidopsis*, this gene is expressed in most tissue types, except dry seed. Reduced expression of ABAR results in a decrease in the expression of many ABA-responsive genes, such as *RD29A* (Yamaguchi-Shinozaki and Shinozaki, 1994), *MYB2* (Abe et al., 2003), *MYC2* (Abe et al., 2003), *ABI4* (Finkelstein et al., 1998), *ABI5* (Finkelstein and Lynch, 2000), and *OST1* (Mustilli et al., 2002). Similarly, seed-specific genes are down-regulated in ABAR RNAi plants, further indicating that ABAR has the regulatory role of ABA-regulated genes.

RECEPTOR-LIKE PROTEIN KINASE

More than 600 receptor-like protein kinase (RLK) genes occur in *Arabidopsis* (Shiu and Bleecker, 2003; Tichtinsky et al., 2003). Among these, greater than 400 are for transmembrane proteins with C-terminal cytoplasmic serine/threonine kinase domains and N-terminal extracellular domains. RLKs are involved in various cellular processes, e.g., hormone responses, cell differentiation and development, and self-incompatibility. A large portion is found in a putative leucine-rich repeat (LRR) subfamily of receptors. These LRR-RLKs include cell-fate determinant CLAVATA1 (Clark et al., 1997), brassinosteroid receptor BRI1 (Li and Chory, 1997), and flagellin receptor FLS2 (Gomez-Gomez and Boller, 2000).

One of these LRR-RLKs, RPK1, is involved in the early steps of ABA-signaling, probably in its perception (Osakabe et al., 2005). RPK1 is an *Arabidopsis* LRR-RLK that is induced not only by ABA but also by abiotic stresses, such as dehydration, high salinity, and cold (Hong et al., 1997). Its hormonal control is ABA-specific, i.e., it is not induced by other growth regulators (Osakabe et al., 2005). Subcellular localization experiments employing a GFP fusion protein have indicated that the RLK1 protein is localized to the plasma membrane.

More direct evidence that RPK1 may be involved in ABA signaling has been provided by its mutant analysis (Osakabe et al., 2005). RPK1 knockout mutants grow normally under standard conditions, although their germination rates are slightly higher than those of wild-type plants. These mutants display reduced sensitivity to ABA throughout all developmental stages. For example, ABA inhibition of root elongation is reduced. ABA insensitivity during germination and post-germination growth is also observed with antisense-RPK1 transgenic plants. In addition, growth of suspension-cultured cells derived from RPK1 antisense plants is less sensitive to ABA inhibition. Compared with the wild type, shoot growth and stomatal closure in RPK1 knockout plants also are partially insensitive to ABA. At the molecular level, ABA-induced expression of numerous ABA-responsive genes is decreased, suggesting that they are regulated by RPK1. The results clearly show that this protein is a positive regulator of the ABA response in seedling growth and stomatal movement.

Involvement of the LRR of RPK1 in ABA signaling has been demonstrated by investigating the overexpression effect of truncated RPK1 (Osakabe et al., 2005). Overexpression of the N-terminal portion of RPK1, which contains the LRR and transmembrane domain but lacks the kinase domain, leads to ABA insensitivity during germination and seedling growth, suggesting a dominant negative effect on RPK1 function. Moreover, overexpression of a chimeric construct, which consists of the LRR of RPK1 and the kinase domain of BRI1 (the BR receptor RLK), results in a higher frequency of dwarf phenotypes. These observations suggest that the chimeric protein suppresses normal BR signaling. Significantly, this dwarf phenotype depends upon ABA concentration.

The research summarized above demonstrates that RPK1 is a major component of ABA signaling. It affects various aspects of the hormonal response, including germination, post-germination seedling growth, cell growth rates, stomatal closure, and the expression of ABA-responsive genes. Although it is not yet known whether RPK1 binds ABA, its membrane localization and the *in planta* function of the LRR of RPK1 suggest that this protein is involved in an early phase of ABA signaling, perhaps in the ABA-perception step.

G PROTEIN-COUPLED RECEPTOR

A key function of abscisic acid is to regulate the opening and closing of stomata, which consist of two guard cells (Schroeder et al., 2001; Fan et al., 2004). ABA promotes stomatal closure and inhibits their opening under water-deficit conditions. Because guard cells are amenable to study at the single-cell level, they provide a good model system for identifying signaling intermediates.

Genetic, biochemical, and pharmacological studies have shown that many signaling components, such as G-proteins, phospholipases, kinases, and calcium, are involved in guard cell ABA signaling (Fan et al., 2004). Among the intermediates, heterotrimeric G proteins and a putative G protein-coupled receptor have been implicated in ABA-perception or the early steps of its signaling. Wang et al. (2001) have provided the first genetic evidence that G proteins are involved in guard cell ABA signaling. In the *Arabidopsis* genome, the G protein α subunit is encoded by a single gene, *GPA1* (Assmann, 2002). Disrupting that gene by T-DNA insertions results in an impaired ABA response in the guard cells. Consequently, stomatal opening is not inhibited by ABA in mutant plants. Similarly, K^+ uptake, which occurs during stomatal opening, is not blocked by ABA in the *gpa1* mutants, indicating that GPA1 is required for ABA inhibition of both stomatal opening and inward K^+ channels. Another

aspect of ABA function in stomatal movement, i.e., the activation of slow anion channels, is weakened in *gpa1* mutants. However, knockout mutations in *GPA1* exert opposite effects during germination. Unlike in the guard cells, *gpa1* mutant seeds are hypersensitive to ABA and sugars (Ullah et al., 2002; Lapik and Kaufman, 2003). Taken together, these results demonstrate that GPA1 is involved in guard cell ABA signaling and that its function is cell type-specific.

Heterotrimeric G proteins consist of three subunits: α, β, and γ (Assmann, 2002; Perfus-Barbeoch et al., 2004). In general, the signals perceived by G protein-coupled receptors (GPCR) are relayed to downstream signaling effectors. In the *Arabidopsis* genome, Gα is, as mentioned above, encoded by a single-copy gene, *GPA1*, while the Gβ subunit is encoded by another single-copy gene, *AGB1*. In contrast, the Gγ subunit is encoded by two genes, *AGG1* and *AGG2*. For G protein-mediated signaling, the G protein α subunit associates with GPCR. Pandey and Assmann (2004) have reported that GPA1 indeed interacts with GCR1, i.e., an *Arabidopsis* protein with a 7-transmembrane domain that is typical of non-plant GPCRs. They have also demonstrated that *gcr1* knockout mutants exhibit ABA hypersensitivity in stomatal responses and root growth. Their results indicate that GCR1 acts as a negative regulator of the ABA response in guard cells, probably by inhibiting GPA1 function. This antagonistic effect on GPA1, however, is not observed during germination, indicating that it is specific to the guard-cell ABA response. Indeed, more recent genetic research by Pandey et al. (2006) has shown that, unlike in the guard cells, GCR1, GPA1, and AGB1 act within the same pathway during seed germination and early seedling development. Single or double and triple mutants all display ABA hypersensitivity. Thus, the components of the G protein complex are negative regulators of the ABA response. Although GCR1 regulates ABA response, it is not yet known whether it binds ABA directly. In addition, their functions are not specific to ABA, with both GCR1 and the heterotrimeric G proteins being involved in other hormonal signaling (Assmann, 2002). It remains to be seen what the ligands of GCR1 might be.

CONCLUDING REMARKS

Several lines of evidence, including the stereo-specificity of the ABA structure, suggest the presence of multiple ABA-perception sites, both intracellular and extracellular (Finkelstein et al., 2002; Razem et al., 2006). Furthermore, the ABA response pathways downstream of those sites are diverse, depending upon tissue type and physiological or environmental cues. Therefore, it would not be surprising to find multiple types of ABA receptors. FCA, the first reported ABA receptor, exemplifies this expectation. It regulates a subset of ABA responses, i.e., lateral root growth and flowering-time control, whereas other ABA-regulated processes are not affected. ABAR function is more general, regulating germination, seedling growth, stomatal movement, and seed maturation. Both receptors are unusual in several respects. They are not membrane-localized. They each have additional functions, as either a RNA-binding protein or a biosynthetic enzyme. FCA and ABAR proteins are encoded by unique genes, whereas receptors are generally encoded by a small family of related genes. Thus, new classes are defined by these two receptors. The function of FCA has been well-established in previous studies. However, in the case of ABAR, none of its downstream signaling components are known, and the nature and mode of their actions remain to be identified. Based on several reports that point to the plasma membrane as an ABA-perception site (Yamazaki et al., 2003), we might speculate on the existence of additional, still-unidentified ABA receptors.

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