INTRODUCTION

During their lifespan, plants are exposed to continuously changing environmental conditions and pathogen threats. Various abiotic and biotic stresses, such as heat, cold, drought, high salinity, or pathogen attacks, can severely affect plant development, growth, fertility, and productivity. To survive, plants must be able to react rapidly to various stress signals, activate efficient defense responses, and adapt to new conditions. Plant hormones are key components of these defense and adaptation mechanisms. To mediate the responses and adaptations to stresses, different hormonal pathways are upregulated or downregulated. Modifications in the hormonal abundance and signaling will usually impact on the degree of resistance or susceptibility to the various stresses.

HORMONES AND ABIOTIC STRESSES

Plants can perceive and respond to environmental changes. For instance, seasonal variations in day/night length or in temperature might affect the reproductive cycle, flowering, and fruit set. However, unpredicted changes, such as flooding, extreme temperature, heavy metals, drought, or high salt levels, will be perceived as stress conditions and might have a strongly negative impact on grain yield, grain weight, and plant biomass. Likewise, the root system architecture will adapt in terms of growth and branching as a reaction to different stresses. Among the various stress conditions, salinity and drought are currently the major problems. Saline soils represent a total of 323 million hectares worldwide (Brinkman, 1980), whereas drought affects 1–3% of the land surface and is predicted to increase to up to 30% by 2090 (Burke et al., 2006). This altered hormonal balance also affects the plant development, with a direct impact on seed development, seed germination, dormancy, and overall plant growth (Finkelstein et al., 2002).

ABSCISIC ACID – THE ABIOTIC STRESS HORMONE

In response to abiotic stresses, such as drought and salinity, endogenous abscisic acid (ABA) levels increase rapidly, activating specific signaling pathways and modifying gene expression levels (Seki et al., 2002; Rabbani et al., 2003; Kilian et al., 2007; Goda et al., 2008; Zeller et al., 2009). In fact, up to 10% of protein-encoding genes are transcriptionally regulated by ABA (Nemhauser et al., 2006).

Abscisic acid is one of the most studied phytohormone because of its rapid response and prominent role in plant adaptation to abiotic stresses. In the meantime, the key components of the ABA signaling pathway have been characterized (Sreenivasulu et al., 2007; Cartler et al., 2010; Harayama and Shinomaki, 2010; Raghavendra et al., 2010; Debnath et al., 2011; Fujita et al., 2011). In Arabidopsis thaliana, the pyrabactin resistance1 (PYR1)/PYR1-LIKE (PYL)/regulatory components of ABA receptor (RCAR) proteins have been proposed as the main intracellular ABA receptors (Ma et al., 2009; Park et al., 2009; Santiago et al., 2009a; Nishimura et al., 2010). Multiple ABA receptor loss-of-function mutants, such as pyr1/pyr2/pyr4, pyr1/pyr1/pyr2/pyr, and pyr1/pyr2/pyr4, are insensitive to ABA, even at concentrations as high as 100 μM (Park et al., 2009; Gonzalez-Guzman et al., 2012). Particularly, the quadruple and sextuple mutants were less sensitive to the ABA-mediated inhibition of seed germination, root growth, stomata closure, and expression of ABA responsive genes (Park et al., 2009; Nishimura et al., 2010; Gonzalez-Guzman et al., 2012).
ABA and severely affects plant growth and seed yield (Fujii and Zhu, 2009). Similarly to the receptor mutants, mutants in the PP2C activity, such as abi1-1, are also insensitive to ABA (Fuji and Zhu, 2009; Cutler et al., 2010). PP2C repression activates downstream stress-related proteins, such as the protein kinases belonging to the sucrose non-fermenting 1-related subfamily2 SnRK2.2/D, SnRK2.3/I, and SnRK2.6/OST1/D, which trigger ABA-dependent gene expression and signaling (Umezawa et al., 2009; Vlad et al., 2009). Accordingly, the snrk2.2/snrk2.3/snrk2.6 triple mutant is highly insensitive to ABA and severely affects plant growth and seed yield (Fuji and Zhu, 2009).

**CYTOKinin in abiotic stress responses**

Besides ABA, other hormonal pathways, including cytokinin (CK), are activated when a plant is exposed to stress. The CK-dependent modulation of stress responses has been studied at various levels. The alteration of endogenous CK levels in reaction to stress suggests that this hormone is involved in stress responses. For instance, in response to drought, the in planta concentration and transport of trans-zeatin riboside decreases drastically, whereas the ABA levels increase (Hansen and Dörffling, 2003; Davies et al., 2005). Interestingly, when the partial root zone-drying approach was applied, the CK concentration decreased, not only in roots, but also in leaves, buds, and shoot tips, along with increased ABA levels (Stoll et al., 2000; Kudoyarova et al., 2007). These observations demonstrate that the local stress exerted on the root might trigger changes in the CK levels in various plant organs, including the shoot and, consequently, in developmental processes, such as the apical dominance (Hansen and Dörffling, 2003; Schachtman and Goedert, 2008). Typically, reduced CK levels would enhance the apical dominance, which, together with the ABA regulation of the stomatal aperture, aids to adapt to drought stress.

The negative CK-regulatory function in plants exposed to drought has been demonstrated in genetic studies in which the endogenous CK levels were modified, either by loss of the biosynthesis genes isopentyl transferase (IPT) or by overexpression of cytokinin oxidase (CKX1)-encoding degradation genes (Werner et al., 2010; Nishiyama et al., 2011b). A reduced CK content in the ipt3/ipt5/ipt7 quadruple and ipt single mutants or overexpression of CKX1 and its homologs correlates with an increased resistance to both salt and drought stresses.

In agreement with the increased abiotic stress resistance at low CK levels, mutants lacking the functional CK receptors are more resistant to abiotic stresses (Tran et al., 2007; Jeon et al., 2010; Kang et al., 2012). For example, the Arabidopsis histidine kinase (AHK) loss-of-function mutants ahk2/ahk3/ahk4 were significantly more resistant to freezing temperatures than the wild type (Jeon et al., 2010). Similarly, all ahk single and multiple mutants, with the exception of ahk4, showed an enhanced resistance to dehydration (Kang et al., 2012). Furthermore, like the CK-metabolic mutants ipt3/ipt5/ipt7, ipt8, and the CKX1-overexpressing plants, the ahk mutants affected dramatically the ABA sensitivity (Tran et al., 2007) and were hypersensitive to ABA treatments.

Downstream of the AHK receptors, the Arabidopsis histidine phosphotransfer (AHP) proteins mediate stress signaling (Hwang et al., 2012). AHP proteins translocate into the nucleus and activate the type-B Arabidopsis response regulator (ARR) factors that trigger the transcription of specific genes in response to CK. A negative feedback loop is provided by type-A ARRs that inhibit the activity of type-B ARRs by a still unknown mechanism (Figure 1). Of all ARRs, type-A ARRs are the only ones of which the expression is altered under stress, e.g., ARR3, ARR6, ARR7, and ARR13 are upregulated under cold stress (Jeon et al., 2010; Jeon and Kim, 2013); ARR5, ARR7, ARR15, and type-C ARR22 are upregulated in response to dehydration (Kang et al., 2012); and ARR5 expression increases in response to salt stress (Mason et al., 2010). Stimulation of ARR5, ARR6, ARR7, and ARR15 expression in response to cold stress requires the activity of several components of the CK signaling pathway, including AHP2, AHP3, and AHP5, and also ARR1 (Jeon and Kim, 2013). Likewise, in response to salt stress, ARR5 upregulation depends on ARR1 and ARR12 (Mason et al., 2010). Furthermore, the negative regulatory role of AHP2, AHP3, and AHP5 during drought stress has been described recently (Nishiyama et al., 2013). Despite the clear indications that CK and the CK signaling components function in stress responses (Hwang et al., 2012), the high degree of redundancy in the CK signaling pathway, including three CK receptors, six AHPs, 10 type-A ARRs, and 11 type-B ARRs, makes it difficult to dissect the role of each specific component (Hwang et al., 2012). Interestingly, although CK levels are reduced, the type-A ARRs that belong to the early CK-responsive component (Hwang et al., 2012). Furthermore, a quadruple type-A ARR loss-of-function mutant arr3/arr4/arr5/arr8 is resistant to salt stress, which is unexpected because type-A ARRs act as CK signaling repressors (Mason et al., 2010). These observations imply that in stress responses the role played by the CK signaling pathway is more complex. In this context, AHKs might function as stress sensors that would activate the CK signaling pathway independently of CK levels (Urao et al., 1999; Tran et al., 2007; Jeon et al., 2010). In fact, another member of the histidine kinase family, AHK1, is able to sense and transduce changes in osmolarity to trigger downstream signaling pathways (Urao et al., 1999; Tran et al., 2007). However, unlike the CK receptors AHK2, AHK3, and AHK4, AHK1 positively regulates stress responses. Thus, it remains to be elucidated whether AHK2, AHK3, or AHK4 can sense abiotic stresses independently of CK, or whether AHK1 might cross-talk with a downstream CK signaling cascade.

Besides core components of the CK transduction cascade, downstream targets in stress responses have been disclosed as well. The cytokinin response factor (CRF) transcription factors of the APETALA2 (AP2) family have been identified as early CK response genes of which the expression is rapidly induced after CK application (Rusholt et al., 2006). Interestingly, the CRF6 homolog is also highly responsive to various abiotic stress...
O'Brien and Benková Cytokinin cross-talking during stress

FIGURE 1 | CK and crosstalks during abiotic stress responses. Under non-stress conditions, CK activates signaling mediated through AHK receptors, AHPs, and type-B response regulators ARRs. Type-B ARRs stimulate the expression of the early CK response genes, including type-A ARRs. ARRs also stimulate the expression of type-A ARRs, which provide a negative feedback loop of the CK signaling. Besides this negative feedback loop, type-A ARRs also repress the expression of the ABI5 gene and interfere with the ABA signaling, through the physical interaction with ABI5. In response to stress, ABA levels increase and, simultaneously, CK levels decrease. The recognition of ABA by the receptors PYR/PYL/RCAR promotes the interaction with PP2C proteins that will activate downstream responses through signaling components, including ABI5 and ABI4. At the same time, ABA interferes with the activity of CK and auxin and via ABI4 attenuates the expression of the PIN1 auxin efflux carrier and enhances the transcription of the CK signaling repressor ARR5. Interestingly, type-A ARRs, such as ARR5, are upregulated, despite the low CK levels, probably because of the indirect activation of the CK signaling pathway by alternative receptors of the histidine kinase family, such as AHK1.

treatments (Zwack et al., 2013) and, recently, its regulatory role has been characterized in leaf senescence control (Zwack et al., 2013).

HORMONAL CROSSTALKS AND ABIOTIC STRESS RESPONSES

The altered ABA sensitivity in plants with modified CK levels and signaling (Tran et al., 2007; Werner et al., 2010; Nishiyama et al., 2011; Wang et al., 2011b) hints at a crosstalk between ABA and CK. Interestingly, ARR4, ARR5, and ARR6 have been found to interact with ABI5 and also to regulate its expression levels. ABI5 is a basic leucine zipper protein that positively regulates the ABA signaling. The interaction with type-A ARRs attenuates the ABI5 activity and suppresses the ABA signaling (Figure 1; Wang et al., 2011b). Thus, type-A ARRs might, in addition to their regulation of the CK signaling, also control ABA signaling.

New insights into the ABA-CK crosstalk have been gained from the functional analysis of ABI4 (Shkolnik-Inbar and Bar-Zvi, 2010) and, similar to ABI5, also control ABA signaling. New insights into the ABA-CK crosstalk have been gained from the functional analysis of ABI4 (Shkolnik-Inbar and Bar-Zvi, 2010) and, similar to ABI5, also control ABA signaling. The interaction with type-A ARRs attenuates the ABI5 activity and suppresses the ABA signaling (Figure 1; Wang et al., 2011b). Thus, type-A ARRs might, in addition to their regulation of the CK signaling, also control ABA signaling.

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Vanstraelen and Benková, 2012; Zhai et al., 2013) and, recently, its role as a negative regulator of freezing tolerance has been demonstrated (Shi et al., 2012). The ET activity in stress responses is mediated by the downstream transcription factor of the ET signaling cascade, ethylene-insensitive 3 (EIN3). EIN3 suppresses the expression of the C-repeat/dihydropyrotoxin response element-binding factor 1 (CBF1), CBF2, and CBF3 genes, which mediate the response to cold stress, and also of the CK signaling repressors ARR5, ARR7, and ARR15 by direct binding to their promoters (Shi et al., 2012). Although ET interferes with the CK signaling output, its pathway is also affected by CK. Indeed, CK stabilizes 1-aminoacyclopropane-1-carboxylate synthase 3 (ACS3) and ACS9 (Vogel et al., 1998; Chae et al., 2003; Hansen et al., 2009) to high levels (Záradská et al., 2013). This stabilization might lead to an ET accumulation and, consequently, affect plant growth processes, such as root growth (Cary et al., 1995; Růžička et al., 2007). The complexity of the hormonal regulatory network underlying stress responses has been suggested (Lehotai et al., 2012) by the activation of both CK and ET signaling in response to senescence-induced stress by means of the ARR5 and ACS8 markers and decrease in the auxin levels.

Interestingly, the CK-ET and CK-ABA interactions exhibit tissue-specific features. CK treatments have been demonstrated to promote the ABA accumulation in shoots, but not in roots, in contrast to ET that accumulates predominantly in roots in response to high CK levels (Záradská et al., 2013).

**PLANT HORMONES IN RESPONSES TO BIOTIC STRESSES**

Hormones also tightly regulate plant responses against pathogens. The networks that control the immune responses in plants are highly complex and have been extensively reviewed (Teys and Parker, 2000; Broekaert et al., 2006; Robert-Seilaniantz et al., 2007; Nishimura and Dangl, 2010). The best characterized hormones that play a role in pathogen response/defense are salicylic acid (SA), jasmonate (JA), and ET. Depending on the lifestyle of the pathogens, a different response will be triggered by the plant. Against biotrophic pathogens, the resistance largely depends on SA-mediated responses and the principal defense strategy is programmed cell death (apoptosis) that restricts the biotrophic pathogen to the infection site, preventing its proliferation, and further spreading in the plant (Dangl and Jones, 2001; Jones and Dangl, 2006; Nishimura and Dangl, 2010; An and Mou, 2011). In contrast, for necrotrophic pathogens that feed on death tissue only, cell death is beneficial. These pathogens induce defense responses that depend on JA and ET to prevent cell death and that trigger the secretion of antimicrobial compounds and the accumulation of proteins with antimicrobial and antifungal activity, such as plant defensins (Overmyer et al., 2000; Andi et al., 2001; Alonso and Strepanova, 2004, Broekaert et al., 2006; Balbi and Devoto, 2008; Fonseca et al., 2009; Gellert et al., 2010). Because of their difference in the nature of the defense strategy, the JA-ET interaction tends to antagonize the SA responses (Petía-Cortés et al., 1993; Doares et al., 1995; Petersen et al., 2000; Kloeck et al., 2001), so that the stress-activated JA-ET signaling might suppress the SA-mediated resistance and vice versa. However, these two pathways might synergistically interact and be considered a fine-tuning mechanism to respond to biotic stresses (Cui et al., 2005; Mur et al., 2006; Truman et al., 2007).

Once the pathogens or microbes have gained access to the plant tissues, they are sensed in each cell by pattern recognition receptors present in the plasma membrane of the host plant cells and bind to microbe-associated molecular patterns (MAMPs; Gómez-Gómez, 2004; Zépéd et al., 2006), the mechanism designated basal resistance or MAMP-triggered immunity (MTI). To overcome MTI, pathogens secrete effectors into the plant cytosol. In this manner, these proteins interfere with the plant immune responses (Chisholm et al., 2006) and modify the host proteins to evade detection and, hence, enhance their virulence, which is referred to as effector-triggered susceptibility. However, the coevolution of plants and microbes has led to the acquisition of the R proteins that specifically recognize these pathogen effectors or avirulence (avr) proteins in a characterized response known as gene-for-gene resistance or effector-triggered immunity (ETI; Hor, 1971). This specific resistance response is noticeable by localized cell death at the infection site and is known as the hypersensitive response (Hammond-Kosack and Jones, 1996; Greenberg and Yao, 2004).

**SAUCYCLIC ACID IN BIOTIC STRESSES**

During the hypersensitive response, different signal transduction pathways are activated. Tissues distal from the infection site develop an enhanced broad-spectrum resistance to secondary infections that is the systemic acquired resistance (SAR; Yarwood, 1960; Ross, 1961). Before SAR is triggered in remote leaves, SA, which is crucial for this defense strategy, accumulates (Malamy et al., 1990). When transgenic Arabidopsis plants express the bacterial SA hydroxylase gene nahG that disables the SA accumulation because of its fast turnover to catechol, they cannot develop SAR and induce the pathogen resistance (PR) gene expression (Gaffney et al., 1993; Delaney et al., 1994). Furthermore, lipid transfer proteins and SA-binding proteins might be involved in the SA accumulation-triggering signaling in SAR (Park et al., 2007). The non-expressor PR1 (NPR1) protein acts downstream of SA and transduces the signal to promote the PR gene expression (Durrant and Dong, 2004). During SAR induction, an oxidative burst occurs, followed by an increase in antioxidants to neutralize the harmful effects of reactive oxygen species. This reducing environment can then convert NPR1 from its inactive oligomeric form into its activated monomeric form that can be transported from the cytosol to the nucleus and activate transcription factors (Kazak et al., 2003; Mou et al., 2003), via protein-protein interactions between NPR1 and the TGACG sequence-specific (TGA) transcription factors (Zhang et al., 1999).

**JASMONIC ACID AND ETHYLENE IN BIOTIC STRESSES**

The defense response to an attack by necrotrophic pathogens and chewing insects is mediated through the JA pathway that commonly acts together with ET to mount a coordinated defense response. One of the best characterized components of the JA signaling pathway is the coronatine insensitive (COI1) receptor (Devoto et al., 2002; Xu et al., 2002). COI1 is part of the Skp1/Cullin/F-box (SCF) E3 ubiquitin-ligase protein degradation complex SCFCO1. High JA levels promote the interaction of the SCFCO1 complex with the JA ZIM (IAZ) domain repressors...
and activate the transcription of JA-responsive genes. The axr1 mutants that lack the functional JA receptor are more susceptible to infections by insects and necrotrophic pathogens, such as Botrytis cinerea, Pythium irregular, and Alternaria brassicicola (van Wees et al., 2003; Adie et al., 2007; Ferrari et al., 2007; Ye et al., 2012). Likewise, mutations that stabilize the JAZ proteins (JAZ1/3A) increase the susceptibility against herbivores, such as Spodoptera exigua (Chung et al., 2008), further supporting the significance of a functional JA signaling pathway in plant defense responses.

The JA-mediated responses against pathogens is strengthened by the ET activity. Ethylene is perceived in plants by the receptors ethylene resistant1 (ETR1), ETR2, ethylene-insensitive4 (EIN4), ethylene response sensor1 (ERS1), and (ERS2) that belong to a histidine kinase family (Bleecker et al., 1988; Chang et al., 1995; Hua et al., 1995, 1998; Saki et al., 1998). Mutations in these receptors not only confer ET insensitivity, but also increase susceptibility to necrotrophic pathogens (Geraats et al., 2003). Downstream from these receptors, the Raf-like kinase constitutive triple response 1 (CTR1) is active, which is a negative ET response regulator. In the presence of ET, the CTR1 repression activates EIN2 (Guzman and Ecker, 1990; Kieber et al., 1995; Chao et al., 1997) and, subsequently, stimulates the EIN3/EIL-like (EIL) transcription factors, whereas mutations in EIN2 confer ET insensitivity, in addition to an increased susceptibility to necropathic pathogens (Geraats et al., 2003).

Although both JA and ET contribute jointly to the plant’s fight against pathogen attacks, the molecular mechanisms of their crosstalk are not well understood, but new insights into the molecular mechanisms underlying their interactions have been provided (Zhu et al., 2011). The JA repressors of the JA signaling interact physically with the EIN3/EIL transcription factors and attenuate their ability to activate genes (Zhu et al., 2011). This interaction has a striking developmental impact, because it enables JA to contribute to the ET response regulation. Thus, besides the classical mechanism in which ET induces the EIN3/EIL stabilization (Gao and Ecker, 2003; Potuschak et al., 2003), EIN3/EIL is released from repression by JA through JA degradation, thereby triggering ET responses (Zhu et al., 2011).

The hormonal interplay between pathways that depend on JA–ET and SA is particularly important when plants are exposed to multiple pathogens of both biotrophic and necrotrophic types. Under such conditions, an effective defense requires only one of these pathways, but still they need to be tightly balanced with each other. This very complex crosstalk between JA and SA has been reviewed thoroughly (see Beckers and Spoel, 2006; Thaler et al., 2012).

**CYTOKININ AND ITS CROSSTALK WITH SALICYLIC ACID**

One of the first indications on the involvement of CK in biotic stress came from tobacco (Nicotiana tabacum) plants in which the S-adenosyl-homocysteine hydrolases (SAHHs) were downregulated. Originally, SAHHs have been studied in mammals because of their role in the regulation of transmethylation and mRNA 5′ capping during viral replication (De Clercq, 1998). Interestingly, the tobacco plants with low SAHH expression not only exhibited an enhanced resistance against the tobacco mosaic virus (TMV), cucumber mosaic virus, potato virus X, and potato virus Y (Masuta et al., 1995), but also increased CK levels and CK-related developmental defects.

In attacked plants, the CK levels are coregulated with the SA levels (Kamada et al., 1992; Sano et al., 1994, 1996; Masuta et al., 1995). Tobacco plants that overexpressed the Ras-related small GTP-binding protein 1 (RGS1)-encoding gene exhibited higher levels of SA and of the acidic pathogenesis-related 1 (PR-1a) gene than those of wild-type plants, in correlation with an enhanced resistance against TMV infection. Interestingly, these transgenic plants also showed phenotypes typical for a high endogenous CK activity, such as reduced apical dominance and increased tillering (Kamada et al., 1992), as was, indeed, confirmed later (Sano et al., 1994, 1996). Furthermore, in both wild-type and RGS1-overexpressing plants, the CK perception inhibited by the use of the competitive inhibitor 2-chloro-4-cyclohexylamino-6-ethylamino-s-triazine interfered with the expression of the SA-dependent PR-1a and the basic JA-dependent PR-1 after wounding (Sano et al., 1996), thereby suggesting that CK contributes to the defense responses mediated by SA and JA.

As mentioned, the recognition of the pathogen Avr effector proteins by the resistance (R) proteins is an important part in plant defense responses. This interaction triggers ETL, which is characterized by the production of SA and the subsequent induction of PR genes and SAR. A dominant-positive mutant of the coiled-coil nucleotide-binding leucine-rich-repeat (CC-NB-LRR) protein UNI (uni-1D) that constitutively activates ETI (Igari et al., 2008) exhibits an enhanced expression of PR-1, PR-3, and of the type-A ARR CK-signaling repressors and increased endogenous CK levels, with phenotypic alterations typical for high CK activity as a consequence (Figure 2; Igari et al., 2008). In uni-1D plants, CK levels decreased by the CKX induction reduces both the PR-1 and of type-A ARR gene expression. However, in these uni-1D plants, overexpression of the bacterial SA hydroxylase encoding sahG gene prevents SA accumulation and interferes with the PR-1 expression, but without effect on the type-A ARR gene induction and the CK-like phenotypes (Igari et al., 2008). A similar CK-related phenotype has been observed in the knockdown mutant rin4-D of the resistance to Pseudomonas syringae pv. maculicola (RPM1)-interacting protein 4 (RIN4), which is a negative regulator of R proteins. In rin4-D plants, the R proteins Resistant to P. syringae 2 (RPS2) and RPM1 are constitutively active and trigger ETI, whereas both PR-2 and ARR5 transcript levels are upregulated and the phenotypic alterations are typical for high CK activity (Figure 2; Igari et al., 2008).

Another indication of the crosstalk between CK and SA has emerged from the characterization of the CRF 5 (Figure 2; Liang et al., 2010). Indeed, the CRFS expression is upregulated in response to Pseudomonas syringae pv. tomato DC3000 (Pst DC3000) and the transcript levels of SA-induced PR-1, PR-3, PR-4, and PR-5 are increased in the CRFS-overexpressing lines (Rudroff et al., 2006; Carcillo et al., 2011). This crosstalk mechanism between CK and SA has been elucidated (Choi et al., 2010) by showing that pretreatment of Arabidopsis plants with CK significantly increased the resistance against Pst DC3000 infection. Correspondingly, mutants defective in CK perception and signaling, such as ahk2/ahk3 and arr2, or plants with reduced
endogenous CK levels, such as 35S:CKX2 and 35S:CKX4, were more susceptible to Pst DC3000. In contrast, the plant resistance to Pst DC3000 was enhanced by high endogenous CK levels due to overexpression of the CK biosynthesis (IPT) genes or by CK signaling promoted by increased ARR2 expression (Choi et al., 2010). Therefore, CK has been proposed to affect priming, a defense-related response activation and might assist plants to cope with infections through the induced SA signaling and increased PR expression levels (Igari et al., 2008; Choi et al., 2010; Liang et al., 2010). This scenario is strongly supported by the findings that ARR2 interacts directly with the SA response factor TGA3, which binds the promoter regions of PR-1 and PR-2, and that this interaction is essential for the enhanced resistance of the 35S:ARR2 lines. Altogether, both the SA-triggered translocation of NPR1 into the nucleus and the formation of a complex with TGA3-ARR2 are seemingly necessary for the development of a full SA-mediated defense response (Choi et al., 2010, 2011). The impact of CK on the plant defense has been characterized in the Pst DC3000-Arabidopsis interaction model with the SA induction deficient 2 (sid2) mutant that fails to accumulate SA (Naseem et al., 2012). The increased susceptibility of sid2 toward Pst DC3000 can only be partially recovered by CK treatment (Naseem et al., 2012), thereby supporting that CK treatments enhance the immunity in an SA-dependent manner (Naseem and Dandekar, 2012).

Recently, the CK-promoted protection against pathogenic infections has been suggested to be involved in SA-independent mechanisms (Großkinsky et al., 2011). In the P. syringae pv. tabaci-tobacco interaction model, higher CK levels before infection increase the resistance of tobacco against P. syringae pv. tabaci and this resistance depends on increases phytoalexin levels, such as scopoletin and capsidiol, which accumulate in the presence of CK (Großkinsky et al., 2011). Thus, the mechanism underlying the CK-mediated resistance of tobacco differs from that in Arabidopsis that is based on an SA-dependent transcriptional control. In the solanaceous plant species, CK appears to promote primary defense responses through an increase of the phytoalexin-pathogen ratio.
in the early infection phases that then efficiently restricts the pathogen development.

**CYTOKININ AND ITS CROSSTALK WITH JASMONIC ACID**

Even though there is not much evidence for an interplay between JA and CK, these hormonal pathways might be linked directly (Ueda and Kato, 1982; Dermastia et al., 1994; Sano et al., 1996) and their interaction might be antagonistic (Naik et al., 2002; Stojanova-Bakalova et al., 2008). Typically, in wounded plants, the JA levels increase significantly, whereas the SA levels remain unchanged, but both CK applications and high endogenous CK levels accelerate the defense response to reach a faster maximum release of JA and methyl jasmonate (MeJA) than in control plants (Sano et al., 1996; Devi et al., 2010). In potato (Solanum tubera- nuni), JA treatments can induce the accumulation of CK ribosides (Dermastia et al., 1994), whereas they might strongly inhibit the CK-induced callus growth (Ueda and Kato, 1982). These observations hint at a very complex and unexplored interplay, in which the outcome probably depends not only on the CK-JA ratio, but also that of other hormones as well.

**CYTOKININ AND ITS CROSSTALK WITH AUXIN**

Crosstalk between CK and auxin has been widely studied over the years, particularly in a developmental context in which their interaction is primarily antagonistic (Bishopp et al., 2011; Vanstraelen and Benková, 2012), although a number of recent studies undoubtedly point toward a role of auxin in stress responses. Various pathogens can produce auxins or modulate auxin levels in planta to enhance the plant susceptibility to infection (Chen et al., 2007; An and Mao, 2011). In Arabidopsis plants lacking the functional IPR2 gene, the expression of the P. syringae type III effector AvrRpt2 decreased the resistance against Pst DC3000, and also show altered auxin levels and auxin-related phenotypes (Chen et al., 2007). This direct correlation between sensitivity and auxin levels implies that auxin promotes plant susceptibility. Also, a recent study in which PRI was used as a marker gene in the Pst DC3000–Arabidopsis interaction revealed that, whereas the immunity was positively promoted by CK and SA, it was negatively regulated by auxin, JA, and ABA (Naseem et al., 2012). Interestingly, the positive effect of CK pretreatments on the plant immunity can be repressed by a combined CK and auxin treatment (Naseem et al., 2012). Based on this evidence, CK and auxin might play a highly possible antagonistic role in plant defense responses, but the specific mechanisms that moderate this crosstalk are still unknown.

A model for the CK–auxin interplay in plant defense has been proposed (Naseem and Dandekar, 2012). After infection, pathogens will modulate the auxin levels and the signaling that will diminish the responses mediated by SA and CK, whereas CK pretreatments will prevent the auxin-based susceptibility, due to the known effect of CK on auxin transport and signaling.

**CONCLUSIONS AND FUTURE PERSPECTIVES**

Nowadays, one of the major objectives of plant biologists is to improve plant performances under less favorable environmental conditions. By enhancing plant defense responses against biotic and abiotic stress, non-cultivable land might be used, the losses due to flooding and infections be decreased, and the amount of applied fertilizers and pesticides in the fields be reduced. However, because the crosstalk between stress-related and developmental hormones is largely unknown, and uncharacterized, usually unforeseen problems occur when the stress resistance is modified. Ideally, plants with enhanced resistance to stress or pathogen attacks should not be affected in growth or developmentally hampered. In this context, it is crucial to understand the hormonal crosstalks underlying plant responses to various stresses, because the modification of one single hormonal pathway will very probably alter the activity of other hormonal pathways as well.

The complexity of the impact of hormones on the resistance to stress can be nicely illustrated with examples of plants with altered CK levels. Due to the importance of CK in stress responses, several genes involved in the regulation of CK levels have been proposed as possible targets to enhance stress resistance, such as IPT and CTK genes (Werner et al., 2010; Nishiyama et al., 2011; Wang et al., 2011b). However, the benefit of the stress-tolerant phenotype of the IPT loss-of-function mutants or of CKX-overexpressing plants was counteracted by developmental defects caused by low bioactive CK levels, such as Nb-Δ2-isopentenyl)adenine and trans-zeatin (Nishiyama et al., 2011). To overcome this drawback, it is necessary to control the CK activity either in an organ or in a tissue-specific manner, an approach that has already been used in several species (McCabe et al., 2001; Sykorová et al., 2008; Ghanem et al., 2011; Qin et al., 2011). For instance, as a consequence of downregulated CK levels in root tissues only (Werner et al., 2010), root length, branching, and biomass increased and the plants were also more resistant to abiotic stress treatments, such as severe drought or heavy metal contaminations (Werner et al., 2010). Furthermore, modulation of CK-mediated defense to stress might at the same time attenuate the input provided by other signaling pathways, such as ABA (Wang et al., 2011b). A reduced CK content leads to a decrease in ABA content and hypersensitivity to ABA treatments (Nishiyama et al., 2011), in contrast to the stressed plants in which the ABA levels are upregulated (Stoll et al., 2000; Hansen and Dorfling, 2003; Kudoyarova et al., 2007). Correspondingly, overexpression of IPT genes results in insensitivity to ABA treatments and prevents the induction of ABI1 and ABI5 in seedlings (Wang et al., 2011b). These examples clearly show that a good knowledge of the molecular mechanisms underlying the hormone-mediated responses and of the mutual communication among hormonal pathways might be very rewarding in the targeted modulation of specific hormonal pathways and, hence, in the effective plant adaptation to concrete environmental conditions.

Extended studies on the genes that mediate the crosstalk between CK and other developmental and stress-related hormones might identify novel targets for the stress tolerance improvement of crop species. Importantly, the identification of molecular components and mechanisms that mediate the phytohormonial interplay might enable us to dissect the stress-related from the developmental functions.

Finally, to increase the plant resistance against various stresses, new alternative approaches should take in account the specific features of the plant species and the distinct mechanisms that underlay their stress responses (Cho et al., 2010; Großkinisky et al., 2011). A nice example of such a strategy is the enhanced
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REFERENCES

Aike, B. T., Pérez-Pérez, J., Pérez-Pérez, M. M., Godoy, M., Sánchez-Serrano, J.,-S., Schmidt, E. A., et al. (2007). ABA is an essential signal for plant resistance to pathogens affecting JA hypersensitivity and the activation of defenses in Arabidopsis. Plant Cell 19, 1665–1681. doi: 10.1105/tpc.107.054414

Alonso, J. M., and Stepanova, N. A. (2004). The ethylene signalling pathway. Science 300, 1513–1515. doi: 10.1126/science.1084812

An, C., and Mou, Z. (2011). Salicylic acid and its function in plant immunity. Annu. Rev. Plant Biol. 62, 405–424. doi: 10.1146/annurev-arplant-042809-112122

Andújar, C., Sánchez, M., Prat, P., Marqués, J., and Sánchez-Martínez, C. (2005). Effect of methyl jasmonate on hynpin-induced hypersensitive cell death, generation of hydrogen peroxide and expression of PAL mRNA in tobacco suspension cultured BY-2 cells. Plant Cell Physiol. 42, 446–449. doi: 10.1093/pcp/peh150

Balls, V., and Dewey, A. (2000). Jasmonate signalling network in Arabidopsis thaliana: crucial regulatory nodes and new physiological functions. New Phytol. 177, 303–315. doi: 10.1046/j.1469-8137.2000.00220.x

Beckers, G. J. M., and Spalding, S. H. (2008). Fine-tuning plant defence signalling: a post-genome perspective. Plant Biol. 10, 1–13. doi: 10.1111/j.1744-7909.2007.00496.x

Bocchini, D., Mancino, G., Pastorino, P., Asimonti, R., and Chicoine, S. (2012). Cytokinin responses in tomato: a mathematical model of CK-overproducing plants responding to water and salt stress. BMC Syst. Biol. 15, 545–559. doi: 10.1186/1752-0509-6-146

Bürkle, A., Benková, E., and Helariutta, Y. (2011). Sending mixed messages: cytokinin cross-talking during stress responses. J. Plant Growth Regul. 30, 1513–1515. doi: 10.1111/j.1744-7909.2011.01043.x

Broekaert, W. F., Delauré, S. L., De Bolle, M. F. C., and Cammue, B. P. A. (2006). Salicylate versus jasmonate. Plant Cell Environ. 29, 241–246. doi: 10.1111/j.1365-3040.2006.01532.x

Bleecker, A. B., Estelle, M. A., Somerville, C., and Kende, H. (1988). Insensitivity to auxin of nuclear protein ethylene-insensitive3 and related proteins. Proc. Natl. Acad. Sci. U.S.A. 85, 8920–8924. doi: 10.1073/pnas.85.24.8920

Chen, Z.-S., Wang, Y.-Y., Tong, B., Wang, Y., Zhang, R., Wang, Y., et al. (2010). COI1 links jasmonate signalling and fertility to the SCF ubiquitin-ligase complex. Nature 464, 91–95. doi: 10.1038/nature08786

Chung, H. S., Koo, A. J., Kim, Y. Y., Shin, D. J., and Jeong, I. (2010). Cytokinin-activated transcription factor ARR2 promotes plant immunity via TGA2/NPR1-dependent salicylic acid signaling in Arabidopsis. Proc. Natl. Acad. Sci. U.S.A. 107, 840–845. doi: 10.1073/pnas.0911846107

Chung, J., Kim, J., Kim, J., and Jeong, I. (2010). The cytokinin-activated transcription factor ARR2 promotes plant immunity via TGA2/NPR1-dependent salicylic acid signaling in Arabidopsis. Proc. Natl. Acad. Sci. U.S.A. 107, 840–845. doi: 10.1073/pnas.0911846107

Curtis, K. R., Rodriguez, P. L., Finkelstein, R. R., and Abrams, S. R. (2010). Abscisic acid emergence of a core signaling network. Proc. Natl. Acad. Sci. U.S.A. 107, 1701–1706. doi: 10.1073/pnas.0910510106

Debnath, M., Pandey, M., and Bisen, P. S. (2011). An omics approach to understand drought stress tolerance of alfalfa (Medicago sativa) by metabolic profiling and pathway analysis. J. Exp. Bot. 62, 4461–4470. doi: 10.1093/jxb/err113

Delessert, S., Larhrib, S., Guerin, N., Medana, H., and David, A. (2005). Tobacco pathogens and integrated defense responses to infection. Natture 431, 826–835. doi: 10.1038/nature03163

Deppenmeier, W., Kudla, J., and Hirt, H. (2005). Long-distance ABA-signaling and its relation to other signaling pathways in the detection of soil drying and the mediation of the plant’s response to drought. J. Plant Growth Regul. 24, 285–295. doi: 10.1007/s10995-005-0103-1

De la Cruz, E. F. (1998). Cytokinin-induced growth hormone analogues in Sadenosylmethionine hydrolases inhibit growth and auxin-induced auxin-resistant advances. Nucleosides Nucleotides Nucleic Acids 17, 625–634. doi: 10.1080/07432869808200520

Deppenmeier, W., Kudla, J., and Hirt, H. (2011). An enzmys approach to understand the plant abiotic stress. OMICS 15, 739–762. doi: 10.1089/omi.2010.0146

Doléans, T., Pélissé, O., Semmouzi, S., Friedrich, L., Weymann, K., Nogrette, D., et al. (1994). A central role of salicylic acid in plant disease resistance. Science 266, 1247–1250. doi: 10.1126/science.266.5188.1247

Dormer, M., Ravnkilde, M., Villar, B., and Korsø, M. (1994). Increased level of cytokinin elavocin in jasmonic acid (induced by tomato stem beetle) stem node culture. Physiol. Plant. 92, 241–246. doi: 10.1111/j.1399-3054.1994.241._x

Domingo, J., Codina, I., Sánchez, M., Prat, P., and Marqués, J. (2005). COI1 links jasmonate signalling and fertility to the SCF ubiquitin-ligase complex in Arabidopsis. Plant J. 37, 457–466. doi: 10.1111/j.1365-3102.2005.02152.x

Durão, K. S., Vogel, M. O., and Viñals, A. (2012). Ap2/Ereb/ERF transcription factors are part of gene regulatory networks and integrate metabolic, hormonal and environmental signals in stress acclimation and retrograde signalling. Protoplasma 249, 3–14. doi: 10.1007/s00709-011-0412-8

Duran, S. H., Núñez-Vásquez, J., Compean, A., and Ryan, C. A. (1995). Salicylic acid inhibits synthesis of protease inhibitors in tomato leaves induced by systemic and jasmonic acid. Plant Physiol. 108, 1741–1746. doi: 10.1104/pp.108.4.1741

Durant, W. E., and Dong, X. (2004). Systemic acquired resistance. Annu. Rev. Plant Biol. 55, 201–225. doi: 10.1146/annurev.arplant.55.031503.135352

Ferrari, S., Galletti, R., Denoux, C., De Lorenzo, G., Ausubel, F. M., and Ausubel, F. M. (1995). Jasmonate-signaling network in Arabidopsis. J. Exp. Bot. 46, 284–295. doi: 10.1037/2010.07.011

Fujii, H., Kihara, N., and Kato, S. (1998). The cytokinin-activated transcription factor ARR2 promotes plant immunity via TGA2/NPR1-dependent salicylic acid signaling in Arabidopsis. Proc. Natl. Acad. Sci. U.S.A. 95, 1791–1796. doi: 10.1073/pnas.95.4.1791

Gallego, R., Donoso, C., De Lorenzo, G., Assendel, F. M., and制度改革, J. (2007). Resistance to Botrytis cinerea inoscula in Arabidopsis by elicitors is independent of salicylic acid, ethylene, or jasmonic signalling

GBR:00940012
but requires PHYTOALEXIN DEFICIENT3. Plant Physiol. 146, 567–579. doi: 10.1104/pp.107.120594

Ferry, B. L. and Parker, J. E. (2000). Interplay of signaling pathways in plant disease resistance. Trends Genet. 16, 449–453. doi: 10.1016/S0168-9525(00)02107-7

Finkelman, B. R., Campile, S. L. and Rock, C. D. (2002). Abscisic acid signaling in roots and seedlings. Plant Cell 14, 513–545.

Flor, H. H. (1971). Current status of the gene-for-gene concept. Annu. Rev. Phytopathol. 9, 275–296. doi: 10.1146/annurev.py.09.090171.001425

Forrester, S., Chao, J. M., and Solano, R. (2009). The jasmonate pathway: the legend, the receptor and the core signalling module. Curr. Opin. Plant Biol. 12, 537–546. doi: 10.1016/j.pbi.2009.07.013

Fujii, H., and Zhu, J. K. (2009). Abscisic acid mutant deficient in 3 abscisic acid-activated protein kinase reveals critical roles in growth, reproduction, and stress. Proc. Natl. Acad. Sci. U.S.A. 106, 4880–4885. doi: 10.1073/pnas.0809441106

Fujita, Y., Fujita, M., Shimazaki, K., and Nakada, K. (2011). ABA-mediated transcriptional regulation in response to osmotic stress in plants. J. Plant Res. 124, 508–525. doi: 10.1007/s10265-011-0421-5

Gaffney, T., Friedrich, L., Vernooij, B., Negrotto, D., Nye, G., Uknes, S., et al. (1993). Identification of a high-copy-number T-DNA insertional mutant that is virulent on tomato. Plant Cell 5, 1045–1058. doi: 10.1105/tpc.5.6.1045

Gómez-Gómez, L. (2004). Plant perception systems for pathogen recognition and defense. Annu. Rev. Plant Biol. 65, 335–380. doi: 10.1146/annurev-annureeva-022108-103053

Igloi, G., Endre, R., Hörner, K.-L., Aala, M., Sakhalina, H., Kinnunen, T., et al. (2008). Confirmation of activity of a COI-1-NBE-EAR protein alters morphogenesis through the cytokinin pathway in Arabidopsis. Plant J. 55, 14–27. doi: 10.1111/j.1365-313X.2008.03711.x

Jørgensen, J. O., and Kim, J. (2013). Arabidopsis response regulator and Arabidopsis halothane phosphoprotein pseudoprotien (AHPP), MHP, and AHPP function in cold stress. Plant Physiol. 161, 418–424. doi: 10.1104/pp.111.207621

Jong, J., Kim, N. Y., Kim, S., Kang, N. Y., Novik, O., Ka, S.-I., et al. (2010). A subset of cytokinin two-component signaling system plays a role in cold temperature stress response in Arabidopsis. J. Biol. Chem. 285, 23771–23786. doi: 10.1074/jbc.M109.096644

Jones, J. D. G., and Deng, J. L. (2006). The plant immune system. Nature 444, 325–329. doi: 10.1038/nature05208

Kamalak, U., Yamando, S., Fransen, S., and Sims, H. (1992). Transgenic tobacco plants expressing rpg1, a gene encoding a ras-related GTP-binding protein from rice, show distinct morphological characteristics. Plant J. 2, 399–407. doi: 10.1111/j.1365-313X.1992.tb00149.x

Kang, N. Y., Cho, C., Kim, N. Y., and Kim, J. (2012). Cytokinin receptor-dependent and receptor-independent pathways in the dehydration response of Arabidopsis thaliana. Plant Cell Environ. 35, 1179–1194. doi: 10.1111/j.1365-3040.2012.02447.x

Kleinhofs, A., Johnston, R., Mohr, G., Feldmann, K. A., and Ecker, J. R. (1993). CTR1, a negative regulator of the ethylene response pathway in Arabidopsis, encodes a member of the Raf family of protein kinases. Cell 72, 427–431. doi: 10.1016/0092-8674(93)90619-B

Kim, J., Whitcomb, D., Herik, J., Wanka, D., Weid, S., Batista, O., et al. (2007). The Arabidopsis global stress expression data set: evaluation and model data analysis of UV-B light, drought and cold stress responses. Plant J. 50, 347–361. doi: 10.1111/j.1365-313X.2007.03032.x

Kloos, A. P., Verbist, M. L., Sharma, S. B., Schoedt, J. E., Vogel, J., Klessig, D. F., et al. (2001). Resistance to Pseudomonas syringae conferred by an Arabidopsis halorthane phosphoprotein pseudoprotein. Proc. Natl. Acad. Sci. U.S.A. 98, 39–43. doi: 10.1073/pnas.98.10.00391

Komor, A., Muller-Wille, L., Vrebalov, J. A., Grotewold, E., and Ecker, J. R. (2007). Effect of partial root drying on the concentration of statin-type cytokinins in tomato (Solanum lycopersicum L.) system sap and leaves. J. Exp. Bot. 58, 161–168. doi: 10.1093/jxb/erm316

Krushnshine, S., Jones, A. M., and Lamm, A. (2011). Cytokinin interplay with ethylene, brassinosteroids, and abscisic acid under abiotic stress conditions. Plant Cell Physiol. 52, 351–363. doi: 10.1093/pcp/pcr039

Kushwah, S., Jones, A. M., and Laxmi, A. (2011). Cytokinin interplay with ethylene, brassinosteroids, and abscisic acid under abiotic stress conditions. Plant Cell Physiol. 52, 351–363. doi: 10.1093/pcp/pcr039

Kwak, Y. S., Kim, C., Kim, N. Y., and Kim, J. (2012). Cytokinin receptor-dependent and receptor-independent pathways in the dehydration response of Arabidopsis thaliana. Plant Cell Environ. 35, 1179–1194. doi: 10.1111/j.1365-3040.2012.02447.x

Lehoczki, N., Kolbert, Z., Patai, A., Fügös, G., Oslog, A., Kutnar, D., et al. (2012). Sekoi-induced hormonal and signaling mechanisms during root growth of Arabidopsis thaliana. J. Exp. Bot. 63, 5977–5987. doi: 10.1093/jxb/erq202

Li, H., Yang, S., Ermakova, N., Chat, V. V., Jhang, M. H., Achilli, M., Kim, M. C., et al. (2010). Overexpression of an AP2/ERF-type transcription factor CRFS confers pathogen resistance to Arabidopsis plants. J. Korean Soc. Appl. Biol. Chem. 53, 142–148. doi: 10.5392/kacs.2010.00134

Min, Y., Smokovska, I., Kotera, A., Moss, D., Yang, Y., Christmann, A., et al. (2009). Regulators of PDPhosphate acid phosphatase activity function as abscisic acid sensors. Science 324, 1044–1048.

Moos, J., Carr, J. P., Klessig, D. F., and Rauser, I. (1990). Salicylic acid is a likely endogenous signal in the resistance of tobacco to viral infection. Science 250, 1102–1104. doi: 10.1126/science.250.4983.1102

*<fplis-04-00451> — 2013/11/18 — 17:11 — page 9 — #9 *
Petersen, M. G., Bae, D., Salch, D. E., Tester, M., Hill, K., Klober, J. J., et al. (2018). Type-B response regulators ARR1 and ARR7 regulate expression of MEsteL1 and accumulation of selenum in Arabidopsis shoots. Plant J. 94, 756–765. doi: 10.1111/tpj.14060

Munna, C., Tamura, H., Ushiki, K., Korona, S., Kevei, A., and Niino, M. (1995). Broad resistance to plant virus in transgenic plants conferred by antisense inhibition of a host gene encoding a 5′-adenosine/thymine-dependent transcriptional repressor. Proc. Natl. Acad. Sci. U.S.A. 92, 6137–6142. doi: 10.1073/pnas.92.15.6137

McCabe, M. S., Garrot, J. C., Schepers, F., Jordan, W. J. R. M., Stoopen, G. M., D’Août, U., et al. (2001). Effects of PtdAG2−IP7 gene expression on development and senescence in transgenic lettuce. Plant J. 27, 505–516. doi: 10.1046/j.1365-313X.2001.01244.x

Miao, Z., Fan, W., and Dong, X. (2003). Inducers of plant systemic acquired resistance regulate NPR1 function through redox changes. Cell 115, 935–944. doi: 10.1016/S0092-8674(03)00429-X

Miki, J. A., Katayose, A., Abe, T., Miquel, O., and Wasternack, C. (2006). The outcomes of concentration-specific interactions between salicylate and jasmonate signaling include synergy, antagonism, and oxidative stress leading to cell death. Plant Physiol. 140, 249–262. doi: 10.1104/pp.105.072448

Naik, G. B., Malikheeva, I., and Reid, D. M. (2002). Influence of cytokinins on the methyl jasmonate-promoted senescence in Petunia axenic cotyledons. Plant Growth Regul. 38, 61–68. doi: 10.1007/s10729-002-0233-y

Nassem, M., and Davidson, T. (2012). The role of auxin-cytokinin antagonism in plant-pathogen interactions. Mol. Plant Pathol. 13, 909–924. doi: 10.1111/j.1364-3703.2012.00805.x

Overmyer, K., Tuominen, H., Kettunen, R., Betz, C., Langebartels, C., Sandermann, H., et al. (2013). Arabidopsis AHP2, AHP3, and AHP5 histidine phosphotransfer proteins function as redundant negative regulators of drought stress response. Plant J. 75, 753–763. doi: 10.1111/tpj.12367

Park, S.-W., Kaimoyo, E., Kumar, D., Mosher, S., and Klessig, D. F. (2007). Methyl salicylate is a critical mobile signal for plant systemic acquired resistance. Proc. Natl. Acad. Sci. U.S.A. 104, 10556–10561. doi: 10.1073/pnas.0701550104

Park, S.-Y., Fung, P., Nishimura, N., Jensen, D. R., Fujii, H., Zhao, Y., et al. (2009). MAP kinase 4 negatively regulates systemic acquired immunity reveals multiple crosstalk for cytokinin. Science 323, 113–116. [Erratum Science 321, 342]. doi: 10.1126/science.1147113

Rabbani, M. A., Maruyama, K., Abe, H., Khan, M. A., Katsura, K., Ito, Y., et al. (2008). The abscisic acid receptor PYR1 in complex with abscisic acid. The abscisic acid receptor PYL5 through inhibition of clade A PP2Cs. Plant J. 51, 1627–1634. doi: 10.1111/j.1365-313X.2007.03107.x

Raghavendra, A. S., Gonugunta, V. K., Christmann, A., and Grill, E. (2010). ABA perception and signaling. Trends Plant Sci. 15, 395–401. doi: 10.1016/j.tplants.2010.04.006

Rashotte, A. M., Masson, M. G., Hulshoff, C. E., Forrer, I., Schaller, G. E., and Keller, J. (2006). A subset of Arabidopsis J2P transcription factors mediates cytokinin response in concert with a two-component pathway. Proc. Natl. Acad. Sci. U.S.A. 103, 11081–11085. doi: 10.1073/pnas.0602031103

Robert-Seilaniantz, A., Narváez, B., Barry, J., and Jones, J. D. G. (2007). Pathological hormone intercages. Curr. Opin. Plant Biol. 10, 372–379. doi: 10.1016/j.pbi.2007.06.003

Ross, A. F. (1961). Localized acquired resistance to plant virus infection in hypersensitive hosts. Virology 14, 329–339. doi: 10.1016/0042-6822(61)90318-X

Rüdiger, K., Kuying, V., Vannov, S., Dolnikov, R., Büchtemann, T., Fromi, I., et al. (2007). Ethylene regulates root growth through effects on auxin biosynthesis and transport-dependent auxin distribution. Plant Cell 19, 2197–2212. doi: 10.1105/tpc.107.057124

Salah, H., Iba, J., Chen, Q. G., Chang, C., Mehrotra, L. J., Bleecker, A. B., et al. (1998). ETI2 is an ETR1-like gene involved in ethylene signaling in Arabidopsis. Proc. Natl. Acad. Sci. U.S.A. 95, 5812–5817. doi: 10.1073/pnas.95.12.5812

Sano, H., Seki, S., Orudzen, E., Sowzuda, K., and Ohad, Y. (1994). Expression of the gene for a small GTP binding protein in transgenic tobacco deli- vore endogenous cytokinin levels, abnormally induces salicylic acid in response to wounding, and increases resistance to tobacco mosaic virus infection. Plant Physiol. 104, 1057–1060. doi: 10.1104/pp.104.3.710

Sano, H., Seki, S., Kozutsumi, N., Niki, Y., Iwashina, H., and Ohad, Y. (1996). Regulation of cytokinin of endogenous levels of jasmonic and salicylic acids in mechanically wounded tobacco plants. Plant Cell Physiol. 37, 782–789. doi: 10.1093/pcp/37.6.782

Sanjuy, J., Depuydt, F., Beaud, A., Antoni, R., Park, S.-Y., Jamin, M., et al. (2009a). The abscisic acid receptor PTB1 is in complex with abscisic acid. Nature 462, 665–668. doi: 10.1038/nature08591

Sanjuy, J., Kühler, A., Saur, A., Rabie, S., Antuni, R., Depuydt, F., et al. (2008b). Modulation of drought resistance by the abscisic acid recep- tor PTBS through inhibition of clade A ABA receptors. Plant Cell 20, 575–588. doi: 10.1105/tpc.107.055816

Schmidt, D. P., and Greulich, J. D. (2008). Chemical root to shoot signalling under drought. Trends Plant Sci. 13, 281–287. doi: 10.1016/j.tplants.2008.04.003

Seki, M., Narusaka, M., Ishii, N., Tajima, T., Mitra, M., Osono, Y., et al. (2002). Monitoring the expression profiles of 7000 Arabidopsis genes under drought, cold and high-salinity stresses using a full-length cDNA microarray. Plant J. 31, 279–292. doi: 10.1046/j.1365-313X.2002.01330.x

Shi, Y., Tian, S., Houx, L., Huang, X., Zhang, X., Gao, H., et al. (2012). Ethyl- 0-sulfonyl signaling negatively regulates freezing tolerance by repressing expression of CBF and type-A ARA genes in Arabidopsis. Cell 148, 2578–2589. doi: 10.1016/j.cell.2011.12.034

Slusarenko, A., and Bar-Zvi, D. (2010). ABI4 mediates abscisic acid and cytokinin inhibition of lateral root formation by reducing polar auxin transport in Arabidopsis. Cell 142, 550–561. doi: 10.1016/j.cell.2010.08.005

Stover, J., Lloyd, R., and Dry, P. (2008). Hormonal changes induced by partial root drying of irrigated grapevine. J. Exp. Bot. 59, 1627–1634. doi: 10.1093/jxb/ern098

“fips-04-00451” — 2013/11/18 — 17:11 — page 10 — #10

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Stenvora-Bakalova, E., Piotov, P. I., Gjergja, L., and Bäurki, T. I. (2008). Differen-
tial effects of methyl jasmonate on growth and division of etiolated rachis-
cornett. Plant Cell Environ. 31, 475–486. doi: 10.1111/j.1365-3090.2008.01694.x

Sylvestre, B., Kureková, G., Dikoula, S., Terecký, M., Heyrovský, K., Rašimová,
I., et al. (2008). Senescence-induced repression of the A. thaliana apo
2c gene in wheat delay of senescence increases cytokinin content, nitrate influx,
and nitrate reductase activity, but does not affect grain yield. J. Exp. Bot. 59,
377–387. doi: 10.1093/jxb/ern415

Sroka-Kiewicz, I., Rudzik, K., Kepka, M., Demrou, S., Milo, Y., Korte, A., et al.
(2018). Closely related receptor complexes differ in their ABA selectivity and sensi-
tivity. Plant J. 61, 25–35. doi: 10.1111/tpj.13550.10025.x

Thaler, J. S., Humphrey, P. T., and Whiteman, N. K. (2012). Evolution of aux-
in. Plant Cell Physiol. 53, 1559–1583. doi: 10.1093/pcp/psr106

Tran, L. N. S., Urao, T., Qin, F., Maruyama, K., Kakimoto, T., Shinozaki, K., et al.
(2008). Enforced ectopic expression of the AtERF7 gene in wheat delays leaf senescence, increases cytokinin content, nitrate influx, and nitrate reductase activity, but does not affect grain yield. J. Exp. Bot. 59, 377–387. doi: 10.1093/jxb/ern415

Vandepoele, I., Bullens, K., De Veylder, L., Vandenbussche, F., De Smet, F., et al.
(2004). A global view on hormone signaling synergy in Arabidopsis. Plant Cell
16, 918–930. doi: 10.1105/tpc.111.002635

Wang, Y., Li, L., Ye, T., Zhao, S., Liu, Z., Feng, Y.-Q., et al. (2011). Roles of cytokinin
in rice increase susceptibility to below-ground insects and impairs inducible defense.
PLoS ONE 6:e21384. doi: 10.1371/journal.pone.0021384

Wend, J. J., Prvšian, A., Stel, B., Hansen, J., and Smukler, S. C. (2013). ARF4: versatile activator and repressor. Trends Plant Sci. 18, 125–132. doi: 10.1016/j.tplants.2012.10.004

Xu, X., Li, X.-L., and Luo, L. (2012). Effects of unsaturated S-adenosyl-methionine on cytokinin synthesis and tolerance of alfalfa to antoxygenic drought stress. Appl. Environ. Microbiol. 78, 8056–8061. doi: 10.1128/AEM.01726-12

Yamada, K., Usuda, D., Kato, K., and Takeda, S. (2002). Drought resistance in rice increases susceptibility to below-ground insects and impairs inducible defense. PLoS ONE 7:e36214. doi: 10.1371/journal.pone.0036214

Yau, M., Luo, S. M., Xie, J. F., Li, Y. F., Xu, T., Liu, Y., et al. (2012). Silencing COI1 as a potential conflict of interest. The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest. Received 27 January 2013; accepted 22 October 2013; published online: 19 November 2013

Citation: O’Brien JA and Benková E (2013) Cytokinin cross-talking during stress and molecular characterization of GmERF7, a soybean ethylene-response fac-
tor that increases salt stress tolerance in tobacco. Genes 5:174–185. doi: 10.3390/genes50100174

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