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REVIEW PAPER

Jasmonate regulates leaf senescence and tolerance to cold stress: crosstalk with other phytohormones

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Abstract

Plants are challenged with numerous abiotic stresses, such as drought, cold, heat, and salt stress. These environmental stresses are major causes of crop failure and reduced yields worldwide. Phytohormones play essential roles in regulating various plant physiological processes and alleviating stressful perturbations. Jasmonate (JA), a group of oxylipin compounds ubiquitous in the plant kingdom, acts as a crucial signal to modulate multiple plant processes. Recent studies have shown evidence supporting the involvement of JA in leaf senescence and tolerance to cold stress. Concentrations of JA are much higher in senescent leaves compared with those in non-senescent ones. Treatment with exogenous JA induces leaf senescence and expression of senescence-associated genes. In response to cold stress, exogenous application of JA enhances Arabidopsis freezing tolerance with or without cold acclimation. Consistently, biosynthesis of endogenous JA is activated in response to cold exposure. JA positively regulates the CBF (C-REPEAT BINDING FACTOR) transcriptional pathway to up-regulate downstream cold-responsive genes and ultimately improve cold tolerance. JA interacts with other hormone signaling pathways (such as auxin, ethylene, and gibberellin) to regulate leaf senescence and tolerance to cold stress. In this review, we summarize recent studies that have provided insights into JA-mediated leaf senescence and cold-stress tolerance.

Key words: Cold stress, crosstalk, ICE-CBF pathway, jasmonate, JAZ, leaf senescence.

Introduction

Jasmonate (JA) is a crucial hormone that modulates multiple physiological processes, such as root inhibition, anthocyanin accumulation, trichome initiation, male fertility, leaf senescence, and biotic and abiotic stress responses (Ueda and Kato, 1980; Staswick *et al.*, 1992; McConn and Browse, 1996; McConn *et al.*, 1997; Shan *et al.*, 2009; Zhu, 2014; Kazan, 2015; Yuan and Zhang, 2015; Zhu and Lee, 2015; Chini *et al.*, 2016; Goossens *et al.*, 2016*a*; Sharma and Laxmi, 2016; Valenzuela *et al.*, 2016). Previous investigations have revealed that the F-box protein COI1 (CORONATINE INSENSITIVE1) forms a co-receptor complex with JAZ (JASMONATE ZIM-DOMAIN) family proteins to perceive bioactive JA-Ile (Xu *et al.*, 2002; Chini *et al.*, 2007; Thines *et al.*, 2007; Fonseca *et al.*, 2009; Yan *et al.*, 2009; Sheard *et al.*, 2010). Perception of JA-Ile facilitates the ubiquitination of JAZ proteins and subsequently degradation by the 26S-proteosome (Chini *et al.*, 2007; Thines *et al.*, 2007; Yan *et al.*, 2009; Sheard *et al.*, 2010). In addition to acting as co-receptors, JAZ proteins also physically interact and repress multiple transcription factors (TFs), and JA-Ile-mediated

JAZ degradation releases these TFs to modulate various physiological processes (Chini *et al.*, 2007; Fernández-Calvo *et al.*, 2011; Niu *et al.*, 2011; Qi *et al.*, 2011, 2015; Song *et al.*, 2011, 2014; Zhu *et al.*, 2011; Hu *et al.*, 2013; Zhai *et al.*, 2013, 2015; Fonseca *et al.*, 2014; Jiang *et al.*, 2014; Boter *et al.*, 2015; Figueroa and Browse, 2015). In this review, we summarize current knowledge concerning the roles of JA in leaf senescence and tolerance to cold-stress.

Regulation of leaf senescence by JA

Leaf senescence is a developmental program that is regulated by intrinsic and environmental factors, such as hormones, developmental age, nutrients, temperature, light, and pathogens (Lim *et al.*, 2007). Although the effects of JA on leaf senescence have long been known, the molecular mechanisms underlying leaf senescence are not yet completely understood (Lim *et al.*, 2007). Recently, analysis of gene expression and function has provided further evidence for the involvement of JA in modulating this highly regulated developmental program (Balbi and Devoto, 2008). JA induces leaf senescence in a variety of plant species (Ueda and Kato, 1980; Parthier, 1990; He *et al.*, 2002; Yan *et al.*, 2009; Shan *et al.*, 2011; Lee *et al.*, 2015) and therefore is considered to be an important inducer of leaf senescence (Zhu *et al.*, 2015).

Role of JA in promoting leaf senescence

In Arabidopsis, the endogenous JA level is four-fold higher in senescent leaves compared with that of fully expanded, non-senescent ones (He *et al.*, 2002). Consistent with the increased JA level in senescent leaves, certain genes associated with the JA biosynthesis pathway are differentially up-regulated during leaf senescence (He *et al.*, 2002). For example, the expression levels of *LOX1* (*LIPOXYGENASE 1*), *LOX3*, and *LOX4* are strongly increased with the progression of leaf senescence; the expression level of *AOS* (*ALLENE OXIDE SYNTHASE*) is only slightly increased during leaf senescence, partly because *AOS* is expressed at a relatively high level throughout leaf development. During leaf senescence, the expression level of *AOC1* (*ALLENE OXIDE CYCLASE 1*) is strongly increased and those of *AOC2* and *AOC3* are moderately increased; whereas the expression of *AOC4* is down-regulated. Exogenous application of methyl jasmonate (MeJA) promotes leaf senescence in attached and detached Arabidopsis leaves (He *et al.*, 2002). Expanding leaves of wild-type Arabidopsis plants display senescence symptoms after incubation with MeJA for 12 d. The detached wild-type leaves also display senescence symptoms as indicated by visible yellowing in response to exogenous MeJA treatment. Consistent with the visible yellowing symptoms, expression of the senescence-specific gene *SAG12* (*SENESCENCE-ASSOCIATED GENE 12*), which has been widely used as a molecular marker for leaf senescence, accumulates only in the leaves induced to undergo yellowing by MeJA (He *et al.*, 2002).

The JA biosynthetic gene *LOX2* is directly activated by the miR319- (microRNA319)-controlled TCP (TEOSINTE BRANCHED/CYCLOIDEA/PCF) TFs during leaf senescence (Schommer *et al.*, 2008). As a result, symptoms of leaf senescence are delayed in plants of the *jaw-D* mutant overexpressing *miR319a* grown under long days. An *in vitro* assay determined that the delayed senescence in *jaw*-D plants is potentially caused by JA deficiency or defective JA signaling. Consistent with the regulation of *LOX2* by TCP family TFs, exogenous MeJA restored the senescence response in *jaw*-D plants compared with wild-type plants (Schommer *et al.*, 2008).

Together, these results suggest a senescence-promoting role for JA.

Key components of the JA signaling pathway play roles in leaf senescence

A JA-insensitive mutant, *coi1* (*coronatine insensitive 1*), fails to display an early senescence phenotype in the presence of JA (He *et al.*, 2002), which suggests that an intact JA signaling pathway is required for JA-promoted leaf senescence. Xiao *et al.* (2004) conducted a screen for suppressors of the *coi1* mutant to better understand the molecular mechanism regulating JA responses via COI1. The *cos1* (*coi1 suppressor 1*) recessive mutant was identified in this study. As expected, JA-dependent senescence in the *coi1* mutant was restored in the *cos1* mutant genetic background, which further demonstrates an important role of the JA signaling pathway in JA-promoted leaf senescence (Xiao *et al.*, 2004). Moreover, COI1-dependent JA repression of Rubisco activase is suggested to be an essential mechanism for JA-induced leaf senescence (Shan *et al.*, 2011).

JAZ proteins are key transcriptional repressors of the JA signaling pathway and play important roles in plant defense, growth, and leaf senescence (Chini *et al.*, 2007; Thines *et al.*, 2007; Yan *et al.*, 2007; Figueroa and Browse, 2012; Jiang *et al.*, 2014; Zhang *et al.*, 2015). For example, we recently observed that JAZ4 and JAZ8 interact physically with the WRKY57 TF to negatively regulate JA-induced leaf senescence (Jiang *et al.*, 2014). The *jaz4* and *jaz8* mutants displayed more severe senescence phenotypes than the wild-type, whereas *JAZ4* and *JAZ8* overexpression plants displayed delayed senescence phenotypes under MeJA treatment. Therefore, as repressors of the JA signaling pathway, JAZ4 and JAZ8 negatively regulated JA-induced leaf senescence (Jiang *et al.*, 2014). JAZ7 plays a key role in delaying dark-induced leaf senescence. Compared with wild-type plants, the *jaz7* mutant displays a more severe leaf-senescence phenotype, such as more rapid chlorophyll degradation, more severe leaf yellowing, and enhanced accumulation of hydrogen peroxide in the dark. This typical dark-induced leaf-senescence phenotype can be rescued in *JAZ7*-overexpression lines. All of these results suggest that JAZ7 plays a negative role in dark-induced leaf senescence (Yu *et al.*, 2016).

In addition to JAZ proteins, MYC2, MYC3, and MYC4, three bHLH (basic helix-loop-helix) TFs that are targets of JAZ repressors, are essential regulators of most aspects of the JA signaling pathway in Arabidopsis (Goossens *et al.*, 2016*b*). Recently, MYC2, MYC3, and MYC4 have been demonstrated

to function redundantly to activate JA-induced leaf senescence, and it has been shown that bHLH subgroup IIId TFs (bHLH03, bHLH13, bHLH14, and bHLH17) attenuate MYC2/MYC3/MYC4-activated JA-induced leaf senescence (Qi *et al.*, 2015). In addition, Arabidopsis MYC2, MYC3, and MYC4 proteins are shown to bind the *PAO* (pheophorbide a oxygenase) promoter, driving expression of a key enzyme that participates in chlorophyll degradation during green organ senescence (Zhu *et al.*, 2015). These results demonstrate that the JA signaling pathway plays an essential regulatory role during leaf senescence.

JA and salicylic acid crosstalk in leaf senescence

Genetic and genomic studies have provided further evidence for the involvement of multiple phytohormone pathways in the regulation of leaf senescence (Fig. 1; Buchanan-Wollaston *et al.*, 2005; van der Graaff *et al.*, 2006). Salicylic acid (SA) is an important hormone that regulates leaf senescence (Morris *et al.*, 2000; Buchanan-Wollaston *et al.*, 2005; van der Graaff *et al.*, 2006). Antagonistic interactions between SA- and JA-dependent signaling pathways have been widely documented during pathogen infection (Cui *et al.*, 2005; Glazebrook, 2005; Spoel *et al.*, 2007). Miao and Zentgraf (2007) demonstrated that a senescence-responsive TF, WRKY53, is antagonistically regulated by JA and SA in Arabidopsis, suggesting that these two phytohormone pathways interact during senescence. Expression of *WRKY53* is strongly induced by SA, and mutation of *WRKY53* affects SA-mediated responses (Dong *et al.*, 2003; Miao *et al.*, 2004; Hu *et al.*, 2012). WRKY53 interacts with the JA-inducible protein ESR (EPITHIOSPECIFYING SENESCENCE REGULATOR) in the nucleus and this interaction negatively impacts on the DNA-binding activity of WRKY53. These results suggest that the negative JA/SA crosstalk is mediated by the interaction between ESR and WRKY53 proteins during leaf senescence, which is most likely governed by the JA and SA equilibrium (Miao and Zentgraf, 2007).

JA and ethylene crosstalk in leaf senescence

Recent research on JA and ethylene signaling pathways has shed light on the regulatory mechanisms by which these two senescence-associated phytohormones control the timing of leaf senescence. HDA6 (HISTONE DEACETYLASE 6), which deacetylates histone H3, is required for gene silencing and cytosine methylation maintenance (Lippman *et al.*, 2003; Earley *et al.*, 2006). Wu *et al.* (2008) showed that HDA6 positively regulates JA-mediated leaf senescence as well as flowering time. Compared with wild-type plants, *axe1-5*, an Arabidopsis *hda6* mutant, and *HDA6*-RNAi plants displayed increased leaf longevity. Expression of the senescence-associated genes *SEN4* (*SENESCENCE 4*) and *SAG12* is down-regulated in *axe1-5* and *HDA6*-RNAi plants (Wu *et al.*, 2008).

Another study confirmed a functional relationship between ethylene and JA in regulating the timing of both

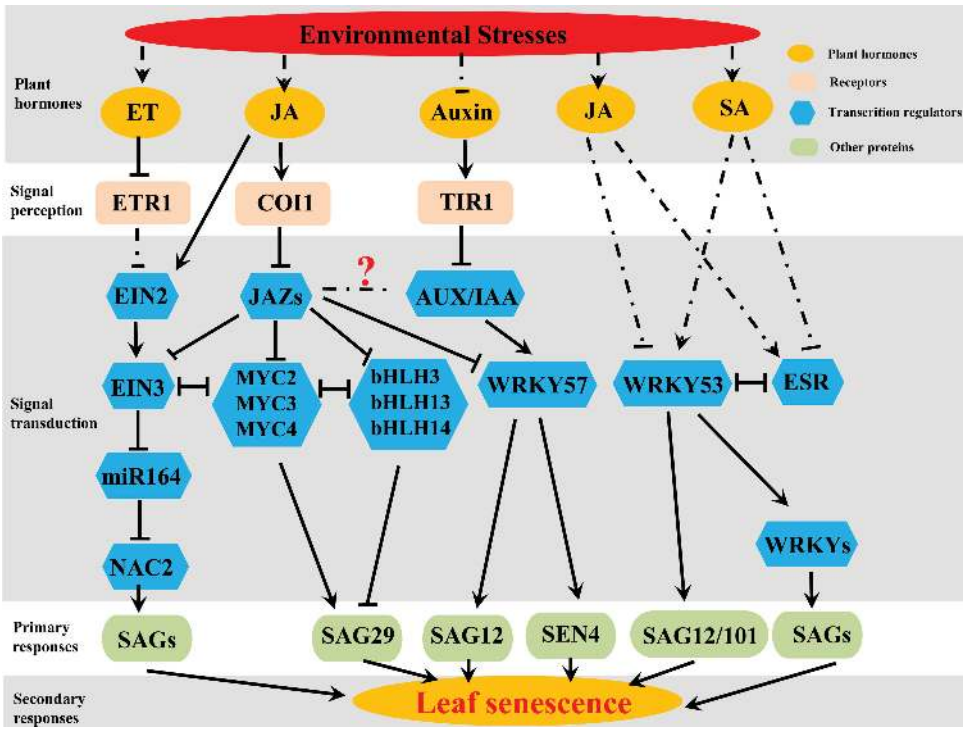

Fig. 1. Proposed model for JA-mediated regulation of leaf senescence in Arabidopsis. Arrows and lines ending in bars indicate activation and suppression processes, respectively. Solid lines represent direct interactions and dotted lines indicate indirect interactions. See the text for additional details. JA, jasmonate; ET, ethylene; SA, salicylic acid; ETR1, ethylene response 1; COI1, coronatine insensitive 1; TIR1, transport inhibitor response 1; JAZ, jasmonate ZIM-domain protein; EIN, ethylene insensitive; AUX/IAA, auxin/indole-3-acetic acid protein; bHLH; basic helix-loop-helix; ESR, epithiospecifying senescence regulator; miR164, microRNA164; NAC2, NAC domain containing protein 2; SAG, senescence-associated gene; SEN4, senescence 4.

leaf senescence and floral organ abscission (Kim *et al.*, 2013). EIN2 (ETHYLENE INSENSITIVE 2) is a central component of ethylene signaling and performs multiple roles in the ethylene signaling pathway (Alonso *et al.*, 1999; Binder *et al.*, 2004; Kim *et al.*, 2012). In *ein2-1* plants, floral organ abscission, a typical response to ethylene, is unaffected by application of exogenous ethylene. The male-sterile and JA-related phenotype of the *dde2-2* mutant containing a mutation in *AOS* can be rescued by exogenous MeJA (von Malek *et al.*, 2002). The *dde2-2 ein2-1* double-mutants, however, respond to exogenous ethylene with accelerated floral organ abscission similar to that observed in wild-type plants. These results suggest that a low concentration of JA can rescue the ethylene response of *ein2-1* mutants, and demonstrate that there is a unique interplay between JA and ethylene.

Li *et al.* (2013) demonstrated that JA-induced leaf senescence is dependent on ethylene signaling, especially the key ethylene-signaling components EIN2 and EIN3. Temporary activation or constitutive overexpression of *EIN3* is sufficient to accelerate leaf-senescence symptoms. By contrast, *ein3*, a loss-of-function mutant, displays delayed JA-induced leaf senescence. Furthermore, EIN3 directly binds to the promoters of *miR164* (*microRNA164*), and overexpression of *miR164* represses EIN3-induced early senescence phenotypes. Li *et al.* (2013) also observed that EIN3 acts downstream of EIN2 to repress expression of *miR164* and up-regulate expression of *NAC2* (*NAC DOMAIN CONTAINING PROTEIN 2*), a target of *miR164*. Collectively, these results define a signaling pathway involving EIN2–EIN3–*miR164*–NAC2 in the regulation of leaf senescence and provide a mechanistic insight into the crosstalk between the JA and ethylene signaling pathways.

JA and auxin crosstalk in leaf senescence

Auxin plays an essential role in the suppression of leaf senescence (Lim *et al.*, 2003). An inverse correlation between auxin concentration and senescence has been reported in several classic studies (Sexton and Roberts, 1982; Nooden and Leopold, 1998) and auxin concentrations decline with leaf age (Shoji *et al.*, 1951). Auxin-modulated JA signaling is essential for development in plants. However, crosstalk between JA and auxin signaling has not been well studied during leaf senescence.

Recently, we provided a mechanistic understanding of how the interaction between the JA and auxin signaling pathways are fine-tuned by WRKY57 during JA-induced leaf senescence (Fig. 1; Jiang *et al.*, 2014). *WRKY57* null mutants display typical JA-induced leaf senescence symptoms, such as more severe yellowing of leaves, higher cell death rates, and lower chlorophyll content, compared with those of wild-type plants. Furthermore, *in vivo* and *in vitro* assays confirmed that the AUX/IAA protein IAA29 and JAZ4/8 competitively interact with WRKY57. The *jaz4* and *jaz8* mutants display more severe leaf-senescence phenotypes in response to exogenous JA application compared with that of wild-type plants. Similarly, *IAA29* overexpression lines display a more severe senescence phenotype compared with the wild-type following application of exogenous JA. These results suggest that JAZ4/8 and IAA29 display opposite functions in JA-induced leaf senescence, which is consistent with the opposing functions of JA and auxin in JA-induced leaf senescence. Most importantly, the JA-induced leaf-senescence symptoms in wild-type leaves are rescued by application of exogenous auxin and this process is dependent on WRKY57. Collectively, these results demonstrate that the JA-induced leaf-senescence process is antagonized by auxin via WRKY57 (Jiang *et al.*, 2014).

JA and leaf senescence

In summary, the underlying molecular mechanisms involved in JA-mediated regulation of leaf senescence are beginning to be understood, implying crosstalk among different phytohormone signaling pathways (Fig. 1). Much work is needed to fully understand the crosstalk of multiple phytohormone pathways that mediate aging and the timing of senescence in both organs and whole plants.

Regulation of cold-stress tolerance by JA

Extreme low temperature beyond a plant's optimal tolerance range is a major environmental stress that severely limits growth and development. Plants have evolved sophisticated mechanisms involving altered biochemical and physiological processes to tolerate low-temperature stress. These altered biochemical and physiological processes involve changes in gene expression patterns. For example, the ICE (INDUCER OF CBF EXPRESSION)–CBF transcriptional regulatory cascade pathway plays a central role in the cold-response pathways (Thomashow, 1999; Chinnusamy *et al.*, 2007; Zhu, 2016). In Arabidopsis, two bHLH TFs, ICE1 and ICE2, directly bind to CANNTG cis-elements present in the CBF promoters and positively regulate their expression (Chinnusamy *et al.*, 2003; Kim *et al.*, 2015). After activation of ICE pathway by cold, three CBF TFs (CBF1, CBF2, and CBF3) induce expression of a large subset of *COR* (*COLD-REGULATED*) genes, finally leading to an enhanced cold (freezing) stress tolerance (Thomashow, 1999; Chinnusamy *et al.*, 2007; Jia *et al.*, 2016; Zhao *et al.*, 2016). In addition, several important regulators, such as OST1 (OPEN STOMATA 1), SIZ1 [scaffold attachment factor A/B/acinus/PIAS (SAP) and MIZ SUMO E3 ligase], HOS1 (HIGH EXPRESSION OF OSMOTICALLY RESPONSIVE GENES 1), CAMTA3 (CALMODULIN BINDING TRANSCRIPTION ACTIVATOR 3), and COLD1, have been shown to modulate the ICE–CBF pathway and cold-responses in plants (Dong *et al.*, 2006; Miura *et al.*, 2007; Doherty *et al.*, 2009; Ding *et al.*, 2015; Ma *et al.*, 2015; Eremina *et al.*, 2016).

JA plays a positive role in modulating tolerance to cold stress

In a recent study, we showed that JA positively regulates the ICE–CBF pathway to enhance freezing tolerance in Arabidopsis (Hu *et al.*, 2013). Exogenous application of MeJA significantly improved plant freezing tolerance with

or without cold acclimation. In contrast, blocking JA biosynthesis and signaling pathways resulted in hypersensitivity to freezing stress. Consistent with the positive role of JA by modulating the freezing stress tolerance, biosynthesis of endogenous JA was activated under cold stress. Further analysis of the mechanism revealed that several JAZ repressors physically interact with the ICE1 TF to inhibit its transcriptional activity. Consistent with these results, overexpression of *JAZ1* or *JAZ4* represses the ICE–CBF signaling pathway and freezing responses in Arabidopsis. Our research thus indicated that JA acts as an upstream signal of the ICE–CBF pathway to positively modulate freezing responses in Arabidopsis (Fig. 2). In addition to the ICE–CBF cascade, our results revealed that JA also regulates expression of some ICE–CBF pathway-independent genes. For example, a number of genes encoding enzymes for biosynthesis of secondary metabolites (such as polyamine, glutathione, and anthocyanins) were regulated by JA signaling under cold stress (Hu *et al.*, 2013).

Other studies indicate that JA is also positively involved in cold tolerance in other plant species. For example, Lee *et al.* (1997) reported that exogenous application of MeJA remarkably enhances the survival ratio of chilled rice (*Oryza sativa*) seedlings. Du *et al.* (2013) showed that the concentration of endogenous JA increases in rice under cold stress. Furthermore, this study revealed that several JA biosynthesis-related genes, such as *OsLOX2*, *OsAOC*, *OsAOS1*, and *OsAOS2*, were up-regulated in response to cold exposure. In addition, JA signaling genes, such as *OsCOI1a* and *OsbHLH148*, were up-regulated in response to cold stress. Very recently, Wang *et al.* (2016) reported that JA activates the CBF pathway and positively modulates the response to cold in tomato (*Solanum lycopersicum*). The *suppressor of prosystemin-mediated responses 2* (*spr2*) mutant, which is partially deficient in JA when the JA pathway is active (Li *et al.*, 2003), shows increased cold sensitivity compared with the wild-type.

JA is also relevant to the cold storage (e.g. 5 or 7 °C) of several tropical or subtropical fruits, such as banana, mango, papaya, and avocado (González-Aguilar *et al.*, 2000, 2004; Cao *et al.*, 2009; Mohammad *et al.*, 2011; Zhao *et al.*, 2013; Siboza *et al.*, 2014; Ba *et al.*, 2016). For example, application of exogenous MeJA induces chilling (7 °C) tolerance in banana (*Musa acuminata*) fruit (Zhao *et al.*, 2013). Further analysis showed that two homologs of Arabidopsis *MYC2* genes (*MaMYC2a* and *MaMYC2b*) of banana are rapidly induced following MeJA treatment during cold storage. Moreover, yeast two-hybrid analysis indicated that MaMYC2 physically interacts with MaICE1. Consistent with this, expression of ICE–CBF cold-responsive pathway genes, such as *MaCBF1*, *MaCBF2*, *MaCOR1*, *MaRD2*, *MaRD5*, and *MaKIN2*, is significantly induced in MeJA-treated banana fruit during cold storage (7 °C). Very recently, the same research group showed

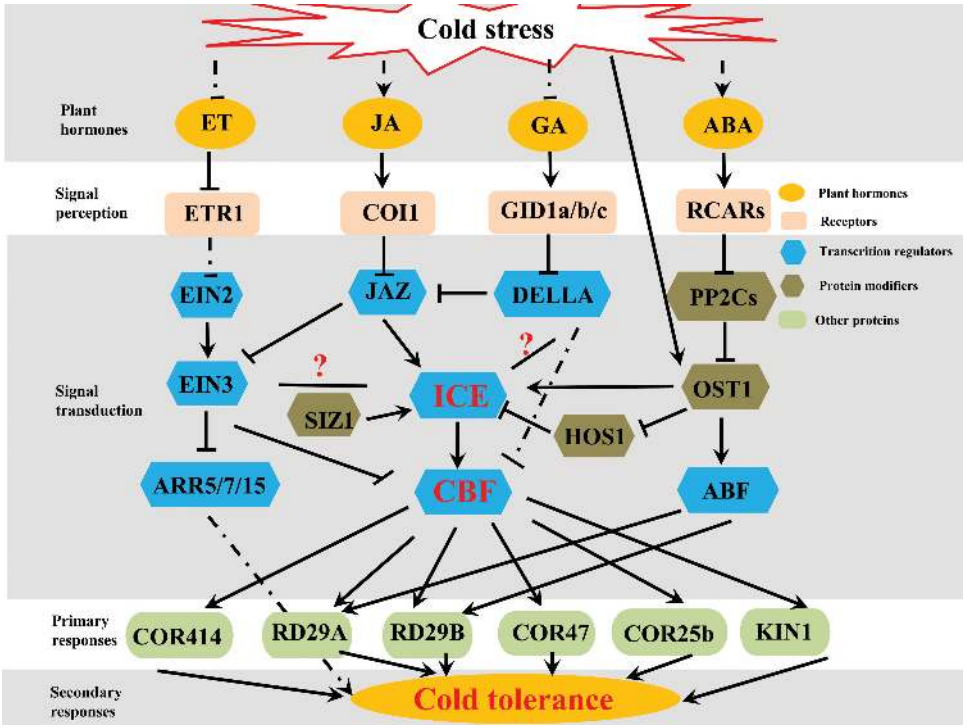

Fig. 2. Proposed model for JA-mediated regulation of cold-stress tolerance in Arabidopsis. Arrows and lines ending in bars indicate activation and suppression processes, respectively. Solid lines represent direct interactions and dotted lines indicate indirect interactions. See the text for additional details. JA, jasmonate; ET, ethylene; ABA, abscisic acid; GA, gibberellin; COI1, coronatine insensitive 1; ETR1, ethylene response 1; GID1, GA insensitive dwarf 1; RCAR, regulatory component of ABA receptor; JAZ, jasmonate ZIM-domain protein; EIN, ethylene insensitive; PP2C, phytochrome-associated protein phosphatase type 2C; OST1, open stomata 1; ICE, inducer of CBF expression; SIZ, scaffold attachment factor A/B/acinus/PIAS [SAP] and MIZ SUMO E3 ligase; HOS1, high expression of osmotically responsive genes 1; ARR, Arabidopsis response regulator; CBF, C-repeat binding factor; ABF, abscisic acid responsive element-binding factor; COR, cold regulated protein; RD, responsive to desiccation.

that one lateral-organ boundaries domain (LBD) protein, designated MaLBD5, is also involved in JA-mediated cold tolerance of banana fruit (Ba *et al.*, 2016). Accumulation of MaLBD5 is induced by cold temperature and MeJA treatment. Furthermore, MaLBD5 physically interacts with MaJAZ1 (a potential repressor of JA signaling of banana), and transactivated expression of *MaAOC2*. These results suggest that MaLBD5 may be partially associated with the biosynthesis of JA to mediate JA-induced cold tolerance of banana fruit. In addition, several physiological studies indicate that JA-induced cold tolerance of tropical and subtropical fruits may also be associated with increased accumulation of cryoprotective compounds, such as polyamine, glutathione, and anthocyanins (González-Aguilar *et al.*, 2000, 2004; Cao *et al.*, 2009; Mohammad *et al.*, 2011; Siboza *et al.*, 2014; Ji *et al.*, 2015).

JA and ethylene crosstalk in modulating tolerance to cold stress

Recent studies suggest that JA interacts with other phytohormones to modulate cold-stress responses (Fig. 2). Ethylene, a gaseous plant hormone, is also involved in plant adaptation to low temperature (Shi *et al.*, 2012, 2015; Catalá *et al.*, 2014). Recently, Shi *et al.* (2012) demonstrated that ethylene negatively modulates freezing tolerance in Arabidopsis. Further genetic and biochemical analysis in their study showed that EIN3 binds to the *CBF* promoters and negatively regulates expression of these cold-responsive genes. Interestingly, EIN3 is a direct target of JAZ repressors that mediate JA-regulated responses (Zhu *et al.*, 2011). Consequently, JA signaling appears to be integrated with ethylene signaling to modulate the *CBF* pathway and cold tolerance via EIN3. Further studies are needed to reveal the relationship between EIN3 and ICE1, as these two important TFs directly regulate the expression of *CBF* genes (Chinnusamy *et al.*, 2003; Shi *et al.*, 2012). Taking into account that EIN3 physically interacts with ICE1 homologs, such as MYC2 and FIT1 (Lingam *et al.*, 2011; Song *et al.*, 2014; Zhang *et al.*, 2014), it is possible to propose that EIN3 and ICE1 interact to regulate *CBF* expression and cold tolerance.

JA and abscisic acid crosstalk in modulating tolerance to cold stress

Abscisic acid (ABA) is another important phytohormone that modulates plant cold-stress responses. Low temperature induces an increase in endogenous ABA, and exogenous ABA treatment enhances plant cold tolerance (Lång *et al.*, 1994; Mäntylä *et al.*, 1995). Kreps *et al.* (2002) showed that ~10% of ABA-related genes are also responsive to cold stress. Although both ABA and JA positively regulate plant cold-stress responses, the crosstalk between ABA and JA signaling has not been well studied. Wang *et al.* (2016) recently revealed that JA functions downstream of ABA to activate the CBF pathway in cold tolerance mediated by light quality. The authors observed that application of MeJA significantly improved cold tolerance in the wild-type and an ABA-deficient mutant. However, application of ABA did not enhance cold tolerance in the JA-deficient mutant *spr2*. Consistent with these findings, ABA application induced expression of genes associated with JA biosynthesis and, ultimately, an increase of JA accumulation (Wang *et al.*, 2016). Ding *et al.* (2015) reported that ABA-related OST1 physically interacts with ICE1. Cold-activated OST1 phosphorylates ICE1 to promote its transcriptional activity under cold stress. Taking into account that JA also regulates the transcriptional function of ICE1 (Hu *et al.*, 2013), ABA/cold-activated OST1 may function synergistically with JA signaling to modulate the ICE–CBF pathway and cold tolerance in plants.

JA and gibberellin crosstalk in modulating tolerance to cold stress

Interestingly, gibberellin (GA) is also involved in plant responses to low-temperature stress. GA-deficient or GA-insensitive mutants show altered cold tolerance in Arabidopsis and rice (Achard *et al.*, 2008; Richter *et al.*, 2010, 2013). For example, the *gai-t6 rga-24* knockout mutant of DELLA genes, which are repressors of the GA pathway, is hypersensitive to freezing stress (Achard *et al.*, 2008). By contrast, the *ga1* mutant, a GA-deficient mutant, shows increased freezing tolerance compared with the wild-type plants. Consistently, the expression level of cold-responsive genes, such as *CBF1*, *CBF2*, and *COR15A*, are up-regulated in *ga1* compared with the wild-type (Richter *et al.*, 2010, 2013). Hou *et al.* (2010) reported that JAZ repressors physically interact with DELLA proteins to integrate JA and GA signaling in Arabidopsis. Further analysis by these authors showed that GA induces degradation of DELLA proteins, allowing JAZ1 to bind MYC2 and suppress JA signaling outputs. Taking into account the finding that JAZs interact with DELLA *in vivo*, it is possible to propose that GA signaling may partially regulate cold tolerance through the JAZ and JA pathway. Further characterization of the crosstalk between JA and GA signaling may reveal the molecular basis of the tight regulation during cold tolerance.

JA and cold stress

In summary, accumulating evidence indicates that JA positively regulates cold tolerance in plants. Mechanistic studies have revealed that JA positively modulates the CBF pathway leading to accumulation of cryoprotective compounds, such as polyamine, glutathione, and anthocyanins. Moreover, JA may interact with other phytohormones, such as ethylene, ABA, and GA, to mediate cold-stress responses. However, the molecular mechanisms underlying these crosstalks remain poorly known. Identification and analysis of the novel components that mediate crosstalk between JA and other regulatory signals will contribute to dissecting the molecular mechanisms involved in tolerance to cold stress.

Conclusions and prospects

JA is a critical hormone that regulates plant developmental processes and tolerance against biotic and abiotic stresses. Recent studies have provided evidence for the involvement of

JA in leaf senescence and cold responses. However, the molecular basis of JA-mediated leaf senescence and cold responses remains limited. Although several studies have reported that JAZ repressors physically interact with some TFs (such as WRKY57, MYC2, and ICE1) to modulate leaf senescence or cold-stress responses in Arabidopsis (Figs 1 and 2), the exact mechanisms underlying the crosstalks between JA and other related hormones are still largely unknown. Moreover, there are few studies regarding JA-regulated leaf senescence and cold responses in crops. Considering the important functions of JA, further studies are needed to identify more critical components mediating JA-signaled leaf senescence and cold tolerance in Arabidopsis and crops. Thus, it is also important to determine the key components that integrate JA and other hormone pathways. Further molecular studies will contribute to underpinning the complex mechanisms of signaling and transcriptional reprogramming controlled by JA and other hormones. We anticipate that this new knowledge will help to improve crop yields in the future under ongoing worldwide climate change scenarios.

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