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REVIEW PAPER

Jasmonate action in plant growth and development

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Abstract

Phytohormones, including jasmonates (JAs), gibberellin, ethylene, abscisic acid, and auxin, integrate endogenous developmental cues with environmental signals to regulate plant growth, development, and defense. JAs are well-recognized lipid-derived stress hormones that regulate plant adaptations to biotic stresses, including herbivore attack and pathogen infection, as well as abiotic stresses, including wounding, ozone, and ultraviolet radiation. An increasing number of studies have shown that JAs also have functions in a remarkable number of plant developmental events, including primary root growth, reproductive development, and leaf senescence. Since the 1980s, details of the JA biosynthesis pathway, signaling pathway, and crosstalk during plant growth and development have been elucidated. Here, we summarize recent advances and give an updated overview of JA action and crosstalk in plant growth and development.

Key words: COI1, development, growth, jasmonate, JAZ, leaf senescence, reproductive development, root growth.

Introduction

The jasmonates (JAs), including jasmonic acid and its derivatives, are plant hormones that control plant defenses against herbivore attack and pathogen infection; confer tolerance to abiotic stresses, including ozone, ultraviolet radiation, high temperatures, and freezing; and regulate various aspects of development, including root growth, stamen development, flowering, and leaf senescence (Goossens *et al.*, 2016; Howe and Jander, 2008; Wasternack and Hause, 2013).

JAs are synthesized from α-linolenic acid (α-LeA/18:3) via the octadecanoid pathway (Fig. 1) (Browse, 2009; Wasternack and Hause, 2013). In plastids, α-LeA, produced via the coordinated actions of fatty acid desaturase (FAD) and phospholipase A1 (PLA), is sequentially converted to (13S)-hydroperoxyoctadecatrienoic acid (13-HPOT), 12,13(S)-epoxyoctadecatrienoic acid (12,13-EOT), and (9S,13S)-12-oxo-phytodienoic acid (OPDA) through the actions of 13-lipoxygenase (LOX), allene oxide synthase (AOS), and allene oxide cyclase (AOC), respectively. OPDA is transported to peroxisomes, where it is reduced to 3-oxo-2-(*cis*-2′-pentenyl)-cyclopentane-1-octanoic acid (OPC-8:0) by OPDA reductase (OPR). OPC-8:0 is then activated to OPC-8:0 CoA by OPC-8:0 CoA ligase (OPCL), and subsequently shortened to jasmonic acid by three rounds of β-oxidation catalyzed by three different enzymes: acyl-CoA oxidase (ACX), multifunctional protein (MFP), and 3-ketoacyl-CoA thiolase (KAT). Jasmonic acid is then exported to the cytoplasm, where it is conjugated with isoleucine to form bioactive (+)-7-*iso*-JA-Ile, which can be inactivated to 12-hydroxy-JA-Ile by CYP94B3, a cytochrome P450, or metabolized to other inactive forms via methylation, glucosylation, or sulfation (Wasternack and Strnad, 2016).

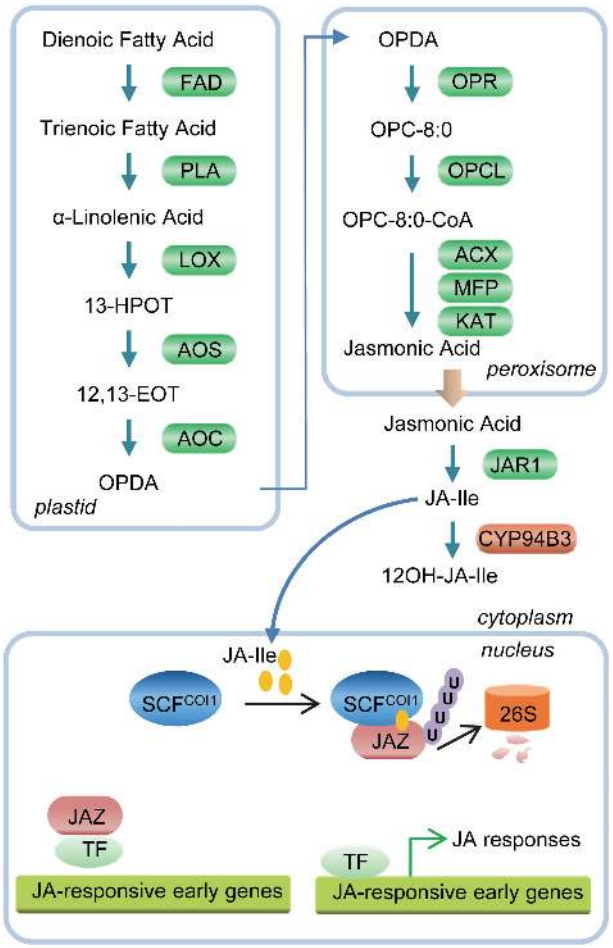

Fig. 1. A simplified view of jasmonate (JA) biosynthesis and signaling. JA-Ile, which is biosynthesized by JA biosynthetic enzymes in plastids, peroxisomes, and the cytoplasm, can be inactivated by CYP94B3. JA-Ile induces the interaction of CORONATINE INSENSITIVE1 (COI1) with JA ZIM-domain (JAZ) family proteins, leading to the ubiquitination and degradation of JAZ proteins via the 26S proteasome; as a result, downstream transcription factors (TFs) are de-repressed, allowing them to activate JA-responsive early genes and JA responses. ACX, acyl-CoA oxidase; AOC, allene oxide cyclase; AOS, allene oxide synthase; 12,13-EOT, 12,13(S)-epoxyoctadecatrienoic acid; FAD, fatty acid desaturase; 13-HPOT, 13-hydroperoxyoctadecatrienoic acid; JA-Ile, jasmonoyl-L-isoleucine; 12OH-JA-Ile, 12-hydroxy-JA-Ile; JAR1, JASMONATE RESISTANT 1/jasmonate-amido synthetase; KAT, 3-ketoacyl-CoA thiolase; LOX, 13-lipoxygenase; MFP, multifunctional protein; PLA, phospholipase A1; OPC-8:0, 3-oxo-2(cis-2′-pentenyl)-cyclopentane-1-octanoic acid; OPCL, OPC-8:0 CoA ligase; OPDA, (9S,13S)-12-oxo-phytodienoic acid; OPR, OPDA reductase.

In the absence of JA, JA ZIM-DOMAIN (JAZ) proteins recruit NOVEL INTERACTOR OF JAZ (NINJA; an adaptor protein) and TOPLESS (TPL; a co-repressor) to repress various downstream transcription factors (TFs) via direct protein interactions (Fig. 1) (Chini *et al.*, 2007; Thines *et al.*, 2007; Yan *et al.*, 2007). Following the perception of bioactive JA, the JA receptor CORONATINE INSENSITIVE1 (COI1) (part of the SKP1/CULLIN-based SCF^{COI1} E3 ligase) (Yan *et al.*, 2009) mediates the ubiquitination and degradation of JAZ proteins via the 26S proteasome (Fig. 1). The resulting activation of TFs enables the expression of JA-responsive genes and JA responses (Fig. 1).

Recently, many excellent reviews of the JA signaling pathway as it applies to plant immunity and abiotic stress tolerance have been published (Chini *et al.*, 2016; Huot *et al.*, 2014; Kazan, 2015; Riemann *et al.*, 2015; Sharma and Laxmi, 2015; Wasternack and Strnad, 2016; Yan and Xie, 2015). In this review, we provide a comprehensive summary of recent advances in our understanding of JA action and crosstalk in plant growth and development.

Actions of jasmonates

Inhibition of seedling growth

The exogenous application of JAs inhibits various aspects of seedling growth, including primary root growth, leaf expansion, and hypocotyl elongation (Kim *et al.*, 2015; Song *et al.*, 2014*b*; Wasternack and Hause, 2013).

Inhibition of root growth

Arabidopsis thaliana (Arabidopsis) COI1, JAZs, and inositol pentakisphosphate ($InsP_5$) form a co-receptor for JA-Ile and the JA mimic coronatine (Sheard *et al.*, 2010). Mutations in COI1 lead to insensitivity to the inhibition of primary root growth by JAs (Xie *et al.*, 1998; Yan *et al.*, 2009). $InsP_5$ enhances the interaction of COI1 with JAZ9 and the inhibitory effect of JAs on root growth (Mosblech *et al.*, 2011). The JA competitive antagonist coronatine-O-methyloxime can impede the interaction of COI1 with JAZs and antagonize the inhibitory effect of coronatine on primary root growth (Monte *et al.*, 2014).

Most of the 13 JAZ members in Arabidopsis have no ERF-associated amphiphilic repression (EAR) domain; they must interact with NINJA and utilize its EAR domain to recruit the co-repressors TPL and TPL-related proteins (TPRs) to suppress JA responses, whereas a minority of non-canonical JAZs (e.g. JAZ8 and JAZ13) directly recruit TPL/TRRs through their EAR domain (Chini *et al.*, 2007, 2016; Pauwels *et al.*, 2010; Shyu *et al.*, 2012; Thines *et al.*, 2007; Thireault *et al.*, 2015; Yan *et al.*, 2007). The inhibitory effect of JAs on primary root growth was shown to be suppressed by the overexpression of NINJA or JAZ proteins carrying a deletion, mutation, or variation in the Jas domain (e.g. JAZ1Δ3A, JAZ3ΔC, JAZ10.3/JAS1, JAZ10.4, JAZ8, and JAZ13), but enhanced by the abolishment of NINJA/TPL or combined mutations in JAZ7, JAZ8, JAZ10, and JAZ13 (Chini *et al.*, 2007; Chung and Howe, 2009; Shyu *et al.*, 2012; Thatcher *et al.*, 2016; Thines *et al.*, 2007; Thireault *et al.*, 2015).

All basic helix-loop-helix (bHLH) subgroup IIIe TFs in Arabidopsis, including MYC2 and its homologs MYC3, MYC4, and MYC5, interact with JAZ proteins (Fig. 2) (Cheng *et al.*, 2011; Fernandez-Calvo *et al.*, 2011; Niu *et al.*, 2011; Qi *et al.*, 2015*a*). MYC2, MYC3, and MYC4, which are distributed in different layers of the primary root apex, function redundantly to mediate the inhibition of primary root growth by JAs (Fernandez-Calvo *et al.*, 2011; Gasperini *et al.*, 2015). The triple mutant *myc2 myc3 myc4* shows an obvious reduction in JA-dependent primary root growth

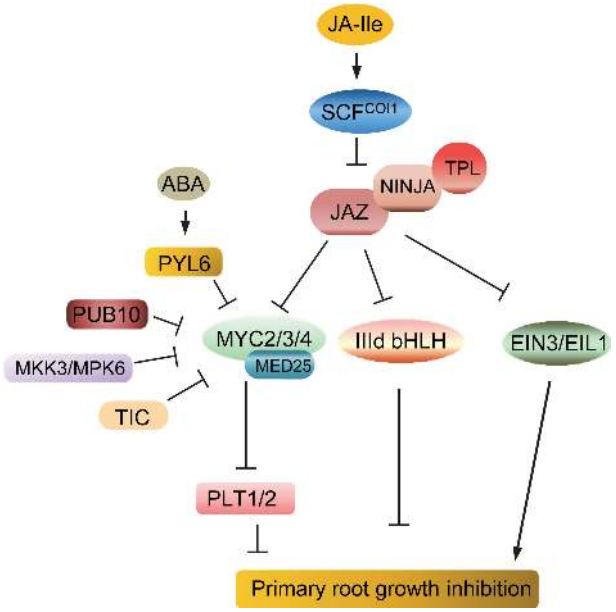

Fig. 2. Regulation of the inhibitory effect of JA on root growth. To attenuate the inhibitory effect of JA on root growth, JAZs recruit a co-repressor, NOVEL INTERACTOR OF JAZ (NINJA)/TPL, to inhibit downstream TFs. In response to JA-Ile, the E3 ligase SCF^{COI1} targets JAZs for ubiquitination and degradation via the 26S proteasome, while MYC2/3/4, subgroup IIId bHLHs (bHLH17/13/3/14), and ETHYLENE INSENSITIVE 3 (EIN3)/EIN3-LIKE1 (EIL1) are released to promote or repress JA-mediated root growth inhibition, respectively. PLANT U-BOX PROTEIN10 (PUB10), mitogen-activated protein kinase (MAPK) kinase 3 (MKK3)-MAPK 6 (MPK6), TIME FOR COFFEE (TIC), MEDIATOR25 (MED25), and the abscisic acid receptor PYRABACTIN RESISTANCE-LIKE 6 (PYL6) interact with MYC2 to modulate the inhibitory effect of JA on root growth. PLT1/PLT2, PLETHORA1 and 2.

inhibition (Fernandez-Calvo *et al.*, 2011). MYC2 interacts with MEDIATOR25 (MED25), a subunit of the Mediator complex, which serves as a bridge for TFs and RNA polymerase II (Cevik *et al.*, 2012; Chen *et al.*, 2012). MYC2 represses the expression of *PLETHORA* (*PLT*)*1* and *PLT2* to restrict root meristem activity and inhibit primary root growth (Chen *et al.*, 2011). JAZ9 incorporates its α-helical Jas domain into MYC3, impeding the interaction of MYC3 with MED25 (Zhang *et al.*, 2015). MYC2 ubiquitination by PLANT U-BOX PROTEIN10 (PUB10) and the phosphorylation of MYC2 by mitogen-activated protein kinase (MAPK) kinase 3-MAPK 6 affect JA-mediated root growth inhibition (Fig. 2) (Chico *et al.*, 2014; Sethi *et al.*, 2014).

JAZ proteins also interact with bHLH subgroup IIId TFs, including bHLH17/JASMONATE-ASSOCIATED MYC2-LIKE TFs (JAM1), bHLH13/JAM2, bHLH3/JAM3, and bHLH14 (Fig. 2) (Fonseca *et al.*, 2014; Nakata *et al.*, 2013; Song *et al.*, 2013*a*). However, these TFs function as transcriptional repressors; they antagonize transcriptional activators (e.g. MYC2) by binding competitively to and inactivating the mutual promoters of target genes (e.g. *TAT1*). Furthermore, they negatively regulate the inhibition of primary root growth by JAs (Fig. 2) (Qi *et al.*, 2015*b*; Song *et al.*, 2013*a*). Accordingly, the quadruple mutant *bhlh17 bhlh13 bhlh3 bhlh14* exhibits hypersensitivity to JA-induced root growth inhibition (Song *et al.*, 2013*a*).

Arabidopsis ETHYLENE INSENSITIVE (EIN)3 and EIN3-LIKE1 (EIL1), essential TFs in ethylene signaling, interact with JAZ proteins to positively mediate both JA-dependent primary root growth inhibition and JA-induced root hair formation (Fig. 2) (Zhu *et al.*, 2011). In rice (*Oryza sativa*), the inhibition of root growth in two JA biosynthesis-deficient mutants was decreased under conditions of salt stress compared with wild type (Hazman *et al.*, 2015). Meanwhile, a loss of function of RICE SALT SENSITIVE3, a nuclear factor that forms a ternary complex with bHLH094 and JAZ proteins, resulted in impaired root cell elongation and severely inhibited root growth under high salt conditions (Toda *et al.*, 2013).

JA affects the formation of lateral and adventitious roots. JA promotes lateral root formation in Arabidopsis by up-regulating the expression of ERF109, which binds to and activates the promoters of the auxin biosynthetic genes *ANTHRANILATE SYNTHASE A1* (*ASA1*) and *YUCCA2* (Cai *et al.*, 2014*b*; Sun *et al.*, 2009). In contrast, JA negatively regulates adventitious root formation in Arabidopsis via the COI1-MYC2/3/4 cascade (Gutierrez *et al.*, 2012). Auxin up-regulates the expression of the Gretchen Hagen3 (GH3) enzymes GH3.3, GH3.5, and GH3.6 to inactivate JA by conjugating Asp, Met, and Trp with JA, promoting adventitious root formation (Gutierrez *et al.*, 2012). However, JA enhances adventitious root formation in petunia cuttings (Lischweski *et al.*, 2015), suggesting that JA differentially regulates adventitious root formation in different species.

Repression of Arabidopsis leaf expansion

JA treatment inhibits the expansion of true leaves and cotyledons. JA represses leaf expansion by inhibiting the activity of the mitotic cyclin CycB1;2 and cell division, rather than by affecting cell size. The Arabidopsis COI1-JAZ-MYC2 cascade mediates the JA-induced inhibition of leaf expansion (Zhang and Turner, 2008). Repeated wounding and touching inhibit leaf expansion via JA biosynthesis, contributing to the 'bonsai effect' in ornamental bonsai plants (Chehab *et al.*, 2012; Zhang and Turner, 2008). Moreover, in response to abscisic acid (ABA) signals, the ABA receptor PYRABACTIN RESISTANCE-LIKE 6 (PYL6) recruits and modulates the transcriptional function of MYC2 to attenuate JA/ABA-inhibited cotyledon expansion (Aleman *et al.*, 2016).

Inhibition of hypocotyl growth

JA acts through COI1 to inhibit hypocotyl elongation in Arabidopsis under all tested light conditions, including blue, red, and far-red wavelengths (Chen *et al.*, 2013). The JA-deficient mutant *jar1* shows an elongated hypocotyl under far-red light and a low red/far-red (R/FR) light ratio, and it is resistant to the far-red light-induced inhibition of hypocotyl growth (Robson *et al.*, 2010). The *coi1* mutant is insensitive to the JA-mediated inhibition of hypocotyl elongation in the dark and under far-red light (Chen *et al.*, 2013), and it exhibits a longer hypocotyl than wild type under red light and in the dark, and a low R/FR light ratio (Chen *et al.*, 2013; Robson *et al.*, 2010). The

myc2/jin1 mutant shows an elongated hypocotyl under far-red light and a low R/FR light ratio (Robson *et al.*, 2010), but a shortened hypocotyl under blue light (Yadav *et al.*, 2005), suggesting that MYC2 positively regulates the inhibition of hypocotyl elongation by red or far-red light and negatively regulates the inhibition of hypocotyl elongation by blue light. JA also inhibits coleoptile growth and plant height in rice and represses ear shoot growth in maize (*Zea mays*) (Biswas *et al.*, 2003; Riemann *et al.*, 2013; Riemann *et al.*, 2008; Yan *et al.*, 2012; Yang *et al.*, 2012).

Crosstalk in the JA-mediated repression of plant growth

The inhibitory effect of JA on growth enhances survival in natural environments by allowing plants to concentrate on defending themselves against various stresses. In Arabidopsis, JAs inhibit growth and promote defense, whereas gibberellin (GA) has the opposite effects. GA induces the degradation of DELLA proteins, thereby removing their repressive effect on TFs known as PHYTOCHROME INTERACTING FACTORs (PIFs) and promoting plant growth; GA also allows JAZs to inhibit MYC2 and JA-mediated plant defenses. Conversely, JAs promote JAZ degradation to activate MYC2 for the enhancement of plant defenses, and de-repress DELLA proteins to inhibit PIFs, suppressing growth (Hou *et al.*, 2010; Wild *et al.*, 2012; Yang *et al.*, 2012). Blue and red wavelengths stabilize MYC2, MYC3, and MYC4, inhibiting growth and enhancing plant defensive responses, whereas darkness and shade decrease the levels of these MYCs, suppressing plant resistance to pathogens (Chico *et al.*, 2014). The mutations of JAZ repressors and the photoreceptor phytochrome B, a PIF repressor, can de-repress MYCs and PIFs to enhance both growth and plant defenses against insects (Campos *et al.*, 2016). TIME FOR COFFEE, a key component of the circadian clock, binds and destabilizes MYC2; thus, it rhythmically controls plant growth and defensive responses (Shin *et al.*, 2012).

Regulation of plant reproductive development

Control of stamen development in Arabidopsis

Stamen development is essential for plant fertility. The Arabidopsis JA-deficient mutants *fad3 fad7 fad8*, *defective in anther dehiscence1* (*dad1*) (the mutant of the PLA DAD1), *lox3 lox4*, *aos*, and *opr3*; overexpression lines of the JA catabolism gene *CYP94B3*; the JA signaling mutant *coi1*; and transgenic lines of *JAZ1Δ3A* and *JAZ10.4* are male sterile due to arrested stamen development at anthesis (e.g. short filaments, indehiscent anthers, and non-viable pollen grains at the tricellular stage), with the exception of *acx1 acx5*, which shows an impairment only in pollen viability (Caldelari *et al.*, 2011; Chung and Howe, 2009; Ishiguro *et al.*, 2001; Koo *et al.*, 2011; McConn and Browse, 1996; Park *et al.*, 2002; Sanders *et al.*, 2000; Schilmiller *et al.*, 2007; Song *et al.*, 2013*b*; Stintzi and Browse, 2000; Thines *et al.*, 2007). JA treatment can restore stamen development in JA biosynthesis-deficient mutants, but not in JA signaling mutants. Restored COI1 expression in the epidermis of filaments and anthers can recover filament elongation, anther dehiscence, and pollen maturation in the *coi1* background (Jewell and Browse, 2016).

The R2R3-MYB TFs MYB21, MYB24, and MYB57 are direct targets of JAZs (Song *et al.*, 2011). They function in overlapping and specific manners to mediate JA-regulated stamen development (Fig. 3) (Cheng *et al.*, 2009; Mandaokar *et al.*, 2006). The strong double mutant *myb21 myb24* is completely male sterile due to short filaments, delayed anther dehiscence, and non-viable pollen grains. *MYB21* overexpression can restore stamen development in *coi1-1* plants (Song *et al.*, 2011). *MYB108* acts downstream of *MYB21* to regulate pollen maturation and anther dehiscence (Mandaokar and Browse, 2009).

MYB21 and MYB24 associate physically with the IIIe bHLH factors MYC2, MYC3, MYC4, and MYC5, forming MYB-MYC complexes to control stamen development (Fig. 3) (Qi *et al.*, 2015*a*). Stamens from *myc2 myc3 myc4 myc5* quadruple mutant plants at anthesis exhibit the same defects as in *coi1-1* mutants, but they ultimately become fertile, resulting in partial male sterility (Qi *et al.*, 2015*a*). The expression of *MYB21*, *MYB24*, *MYB57*, and *MYB108* is repressed in flowers of *myc2 myc3 myc4 myc5* plants (Qi *et al.*, 2015*a*). Overexpression of *MYC5* fused with an EAR domain

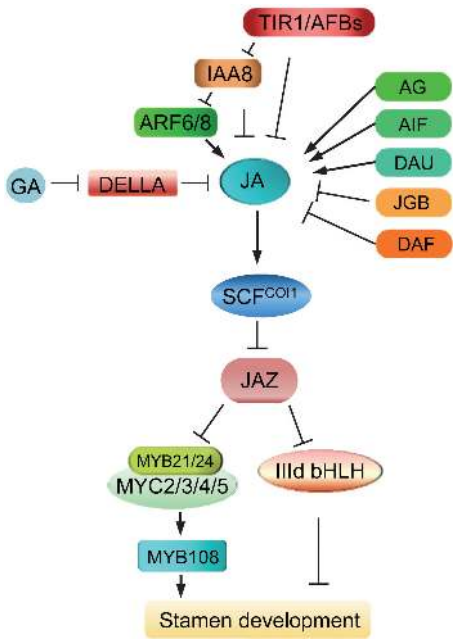

Fig. 3. JA signaling and crosstalk in stamen development. In response to developmental signals in the stamen, JA is biosynthesized to induce the degradation of JAZ proteins via SCF^{COI1}, and MYB21/24-MYC2/3/4/5 transcriptional complexes are activated to promote stamen development, whereas subgroup IIId bHLH factors (bHLH17/JAM1, bHLH13/JAM2, and bHLH3/JAM3) are released to antagonize the function of MYB-MYC in stamen development. AUXIN RESPONSE FACTOR (ARF)6/8, AGAMOUS (AG), ANTHER INDEHISCENCE FACTOR (AIF), and DAYU (DAU) activate JA biosynthesis to positively regulate anther dehiscence and pollen maturation, while DELLAs, TRANSPORT INHIBITOR RESPONSE1 (TIR1)/AUXIN SIGNALING F-BOX PROTEINs (AFBs), INDOLE-3-ACETIC ACID 8 (IAA8), DEFECTIVE IN ANTHER DEHISCENCE1-ACTIVATING FACTOR (DAF), and JINGUBANG (JGB) inhibit JA biosynthesis to repress stamen development.

inhibits stamen development and MYB expression (Figueroa and Browse, 2015; Qi *et al.*, 2015*b*). Overexpression of *MYC5* and *MYC3* can partially restore stamen development and fertility in *coi1-1* (Qi *et al.*, 2015*a*). $MYC2^{E165K}$, a constitutively active version of MYC2, was shown to restore the fertility of the JA-deficient mutant *aos* only in a *ninja* mutant background (Gasperini *et al.*, 2015). On the other hand, the overexpression of subgroup IIId bHLH/JAM TFs was found to inhibit stamen development (Nakata *et al.*, 2013; Nakata and Ohme-Takagi, 2013).

Recent studies suggest that the JA pathway is a hub for Arabidopsis stamen development (Fig. 3) (Song *et al.*, 2013*b*; Yuan and Zhang, 2015). GA suppresses DELLA repressor proteins to activate expression of the JA biosynthetic gene *DAD1* and JA biosynthesis, and triggers *MYB* expression to promote filament elongation (Cheng *et al.*, 2009). Auxin signaling components, including TRANSPORT INHIBITOR RESPONSE1(TIR1)/AUXIN SIGNALING F-BOX PROTEINs (AFBs), INDOLE-3-ACETIC ACID 8 (IAA8), and AUXIN RESPONSE FACTOR6 (ARF6) /ARF8, regulate JA biosynthesis and MYB expression to modulate filament elongation and anther dehiscence (Cecchetti *et al.*, 2008; Nagpal *et al.*, 2005; Wang *et al.*, 2013). The homeotic protein AGAMOUS (Ito *et al.*, 2007), E3 ligase DAD1-ACTIVATING FACTOR (Peng *et al.*, 2013), and the peroxisomal membrane protein DAYU (Li *et al.*, 2014) activate the expression of several JA biosynthetic genes and JA biosynthesis to influence anther dehiscence and pollen germination, while the NO APICAL MERISTEM/ARABIDOPSIS TRANSCRIPTION ACTIVATION FACTOR/CUP-SHAPED COTYLEDON (NAC) TF ANTHER INDEHISCENCE FACTOR (Shih *et al.*, 2014) and JINGUBANG (Ju *et al.*, 2016) repress anther dehiscence and pollen maturation, respectively, in Arabidopsis by suppressing JA biosynthesis (Fig. 3).

Inhibition of petal expansion in Arabidopsis

The Arabidopsis JA-deficient mutants *aos* and *opr3* and the JA perception mutant *coi1* exhibit larger petals than wild-type plants at anthesis, suggesting that JA restricts petal expansion (Brioudes *et al.*, 2009; Reeves *et al.*, 2012). MYB21 and MYB24 are required for petal expansion (Reeves *et al.*, 2012). In Arabidopsis, from the final stage of flower bud opening to the final spreading of the petals after anthesis, JA represses the expression of *MYB21* in petals, resulting in restricted petal growth; in contrast, the expression level of *MYB21* is increased in the petals of *aos* and *coi1* plants, leading to persistent petal expansion and large petals (Reeves *et al.*, 2012). Expression of the bHLH TF BIGPETALp, which limits petal size by controlling post-mitotic cell expansion, is increased in the petals of *opr3* plants, leading to larger petals and cells (Brioudes *et al.*, 2009).

Sex determination in maize

In maize, formation of the tassel (male inflorescence) and ear (female inflorescence) results from the abortion of pistil and stamen development, respectively. In the maize mutants *tasselseed1* (*ts1*) (the mutant of a LOX), *ts2*, and *opr7 opr8*, the tassels are converted to fertile ears that are able to set seeds; JA treatment can restore tassel development, demonstrating that JA controls sex determination in maize (Acosta *et al.*, 2009; Yan *et al.*, 2012). However, the molecular role of TS2 in the JA pathway is unknown.

Control of stamen and spikelet development in rice

The rice JA-deficient mutants *coleoptile photomorphogenesis* (*cpm*)*1* (Biswas *et al.*, 2003), *cpm2*/*hebiba* (Riemann *et al.*, 2013), *osjar1* (Xiao *et al.*, 2014), and *extra glume* (*eg*)*1* (Li *et al.*, 2009); the JA signaling mutant *eg2-D* (with a dominant mutation in OsJAZ1) (Cai *et al.*, 2014*a*); RNAi lines of *OsCOI1a* and *OsCOI1b* (Yang *et al.*, 2012); transgenic lines of truncated JAZs (Hori *et al.*, 2014); and rice transgenic lines expressing an Arabidopsis jasmonic acid carboxyl methyltransferase gene (Kim *et al.*, 2009) exhibit complete or partial male sterility due to abnormalities in spikelet organs, including abnormal or reduced stamens, reiterative glume-like structures, stigma-like organs, and impaired anther dehiscence (except for *cpm1* and COI1a/b RNAi lines, which show defects only in anther dehiscence). Rice MYC2 interacts with JAZ1 and acts through the floral identity genes *OsMADS5*, *OsMADS6*, and *OsG1* to regulate spikelet development (Cai *et al.*, 2014*a*; Zhang *et al.*, 2016*a*).

Regulation of embryo/seed development

Embryo development in the tomato JA-deficient mutant *prosystemin-mediated responses2*, which carries a mutation in FAD7, is delayed or arrested owing to increased programmed cell death (Goetz *et al.*, 2012). The *jasmonic acid-insensitive1* (*jai1*) mutant, which exhibits a loss of function of the tomato homolog of COI1, cannot set viable seeds (Li *et al.*, 2004). Interestingly, a wound-induced endogenous rise in OPDA can slightly restore seed development in *jai1*, suggesting a role for OPDA signaling in the maternal control of seed development (Goetz *et al.*, 2012). Moreover, the tomato mutant *acx1a*, which produces OPDA and a residual amount of JA, sets viable seeds. However, *SiOPR3*, a tomato transgenic line in which the *OPR3* gene is silenced, contains a similar amount of OPDA to wild type and sets only a few viable seeds; methyl-JA treatment can restore the seed-setting of *SiOPR3* (Scalschi *et al.*, 2015), suggesting that JA but not OPDA has a major role in the maternal control of seed development. Overall, the roles of JA and OPDA in seed development require further study.

Induction of leaf senescence

JA promotes leaf senescence in a COI1-dependent manner (Qi *et al.*, 2015*b*). JAZ7 represses JA-regulated dark-induced leaf senescence (Yu *et al.*, 2016). MYC2, MYC3, and MYC4 mediate JA/dark-induced leaf senescence by up-regulating the expression of senescence-associated genes (e.g. *SENESCENCE-ASSOCIATED GENE29* [*SAG29*]) and chlorophyll catabolic enzyme genes (CCGs) (e.g. *Pheophorbide A Oxygenase*), as well as by down-regulating photosynthesis-related genes (e.g. *Chlorophyll A*/*B Binding Protein1*; Fig. 4) (Qi *et al.*, 2015*b*; Zhu *et al.*, 2015). The NAC TFs NAC019, NAC055, and NAC072 act downstream of

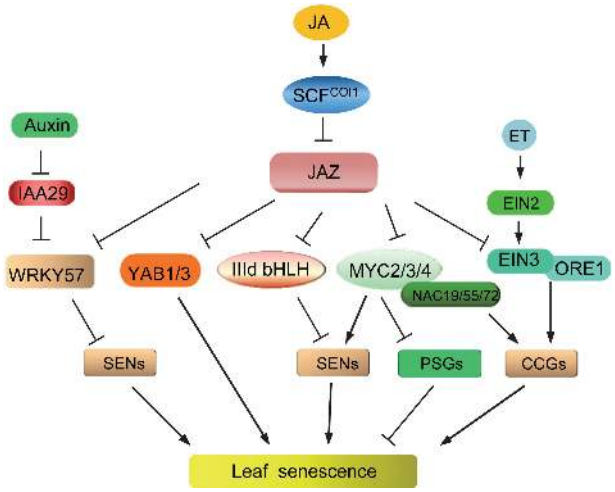

Fig. 4. Regulation of leaf senescence by JA. In response to JA-induced JAZ degradation, the TFs MYC2/3/4, subgroup IIId bHLH factors (bHLH17/13/3/14), WRKY57, YABBY1/3 (YAB1/3), and EIN3 are released to accelerate or delay leaf senescence via the regulation of senescence-associated genes (SENs), photosynthesis-related genes (PSGs), and chlorophyll catabolic enzyme genes (CCGs). WRKY57 interacts with IAA29 and JAZ4/8 to mediate the antagonism of auxin and JA in leaf senescence. Ethylene (ET) acts through EIN2 to activate EIN3, which interacts with ORE1 and JAZ proteins to affect JA-induced leaf senescence.

MYC2/3/4 to coordinately promote chlorophyll degradation by activating CCGs (Fig. 4) (Zhu *et al.*, 2015). Subgroup IIId bHLH TFs antagonize MYC2/3/4 by binding competitively to their mutual target promoters (e.g. *SAG29*) and inhibiting leaf senescence (Fig. 4) (Qi *et al.*, 2015*b*). JA-induced leaf senescence is delayed in the mutants *myc2 myc3 myc4* and *nac019 nac055 nac072*, but accelerated in *bhlh17 bhlh13 bhlh3 bhlh14* (Qi *et al.*, 2015*b*; Zhu *et al.*, 2015).

WRKY57 interacts with JAZ4/8 to negatively regulate JA-induced leaf senescence, and it functions as a target of IAA29 to mediate the antagonism of JA and auxin in leaf senescence (Fig. 4) (Jiang *et al.*, 2014). Ethylene acts through EIN2 to stabilize EIN3, which is repressed by JAZs, and EIN3 activates CCGs with ORE1, accelerating JA-induced leaf senescence (Fig. 4) (Li *et al.*, 2013; Qiu *et al.*, 2015; Zhang *et al.*, 2016*b*). YABBY1 and YABBY3 interact with JAZs to promote chlorophyll degradation (Boter *et al.*, 2015).

Effects on seed germination

JA application delays the ABA-mediated inhibition of seed germination in Arabidopsis; however, *jar1* and *coi1-16* show increased sensitivity to the inhibition of seed germination by ABA (Ellis and Turner, 2002; Staswick *et al.*, 1992). OPDA inhibits seed germination in Arabidopsis in a COI1-independent manner (Dave *et al.*, 2011). During the cold-stimulated germination of wheat (*Triticum aestivum*) seeds, JA biosynthesis-related gene expression and JA biosynthesis increase rapidly in the dormant embryos after transfer to room temperature, and JA suppresses ABA biosynthesis to promote cold-stimulated germination (Xu *et al.*, 2016).

Inhibition of apical hook formation in Arabidopsis

The apical hook in dark-grown etiolated seedlings protects the shoot from damage as it emerges from soil. JA inhibits apical hook formation through the COI1-JAZs-MYC2/3/4 cascade (Song *et al.*, 2014*a*). JA activates MYC2, MYC3, and MYC4, which interact with and repress the transcriptional activity of EIN3/EIL1, leading to down-regulation of the apical hook formation gene *HOOKLESS1* and the inhibition of apical hook curvature (Song *et al.*, 2014*a*; Zhang *et al.*, 2014; Zhu, 2014). Moreover, MYC2 activates the expression of EIN3 BINDING F-BOX PROTEIN1 to degrade EIN3 (Zhang *et al.*, 2014). In contrast, ethylene stabilizes EIN3/EIL1 to inhibit the transcriptional function of MYCs, and it attenuates MYC-mediated plant defenses against insect attack (Song *et al.*, 2014*a*).

Promotion of trichome formation

Trichomes, epidermal cell structures on the aerial parts of plants, protect plants from herbivore attack by acting as sensors or barriers, or by releasing volatile compounds. Deficiencies in JA biosynthesis and perception block wound-induced trichome formation in Arabidopsis (Yoshida *et al.*, 2009). Further analysis has shown that, in the absence of JA, Arabidopsis JAZ proteins interact with WD-repeat/bHLH/MYB complexes, repressing their formation and transcriptional activity (Qi *et al.*, 2011). Following wounding or an insect attack, however, JA biosynthesis is triggered, leading to the turnover of JAZ proteins; under these conditions, WD-repeat/bHLH/MYB complexes are able to promote trichome formation. Subgroup IIId bHLH TFs antagonize WD-repeat/bHLH/MYB complexes by binding competitively to the promoters of their mutual target genes to inhibit trichome formation (Nakata *et al.*, 2013; Song *et al.*, 2013*a*). JA and GA synergistically enhance trichome formation. DELLA repressors in the GA signaling pathway also directly target and inhibit WD-repeat/bHLH/MYB complexes (Qi *et al.*, 2014). JAZ and DELLA proteins are depleted in response to JA and GA, respectively, eliminating the repression of WD-repeat/bHLH/MYB complexes to promote trichome formation (Qi *et al.*, 2014).

Regulation of stomatal closure and reopening in Arabidopsis

Stomata, leaf epidermal pores bordered by pairs of guard cells, regulate water loss, gas exchange, and plant immunity to pathogens. Methyl-JA acts through COI1 to activate a plasma membrane H^+-ATPase; it also induces the efflux of H^+, influx of Ca^{2+}, generation of reactive oxygen species, activation of Cl^- channels, efflux of Cl^-, activation of outward K^+ channels, and efflux of K^+, leading to a loss of turgor in guard cells and stomatal closure (Munemasa *et al.*, 2007; Suhita *et al.*, 2004; Yan *et al.*, 2015). JA interacts with the ABA pathway to induce stomatal closure in Arabidopsis through the protein kinase OPEN STOMATA1, but not through ABA receptors (Yin *et al.*, 2016). Moreover, drought

induces the biosynthesis of the JA precursor OPDA, rather than JA; OPDA functions more efficiently than methyl-JA, and it interacts with ABA to promote stomatal closure in Arabidopsis (Savchenko *et al.*, 2014).

Stomatal closure is triggered by *Pseudomonas syringae* infection as a means of limiting bacterial invasion (Melotto *et al.*, 2006). As a counter-attack, some *P. syringae* strains produce coronatine, which hijacks COI1-JAZ-MYC signaling and inhibits salicylic acid accumulation via NAC019/NAC055/NAC072 and JA2L, promoting stomatal reopening to facilitate bacterial infection (Du *et al.*, 2014; Zheng *et al.*, 2012). In addition, the *P. syringae* effectors HopX1 and HopZ1a interact with and deplete JAZ proteins in a COI1-independent manner, and maintain stomatal apertures to promote infection (Gimenez-Ibanez *et al.*, 2014; Jiang *et al.*, 2013). It will be interesting to elucidate why both methyl-JA and coronatine/effectors utilize COI1 but produce different stomatal behaviors.

Delay of flowering in Arabidopsis

JA inhibits flowering—the transition from vegetative to reproductive growth—in Arabidopsis. The *coi1* mutant and *JAZ1Δ3A* transgenic plants display early flowering, demonstrating that JA acts through COI1-JAZ to inhibit flowering (Zhai *et al.*, 2015). The APETALA2/ERF domain TFs TARGET OF EAT (TOE)1 and TOE2 interact with JAZ proteins and inhibit flowering by inactivating transcription of the florigen gene *FLOWERING LOCUS T* (Zhai *et al.*, 2015). Overexpression of *TOE1* and *TOE2* suppresses the early flowering phenotype of *coi1* (Zhai *et al.*, 2015). Subgroup IIId bHLH factors negatively regulate JA-delayed flowering (Song *et al.*, 2013*a*). Flowering time is delayed in *bhlh17 bhlh13 bhlh3 bhlh14* mutant plants, but accelerated in *bHLH17* and *bHLH13* overexpression lines (Song *et al.*, 2013*a*).

Gravitropism in plants

Gravitropism affects root and shoot architecture. JA affects lateral auxin redistribution in Arabidopsis root tips after gravistimulation, and it interferes with the root gravitropic response in a COI1-dependent manner (Sun *et al.*, 2011). Trp conjugates of jasmonic acid and indole-3-acetic acid inhibit the agravitropic root growth of Arabidopsis seedlings in a COI1-independent manner (Staswick, 2009). During the gravitropic response in rice coleoptiles, JA accumulates to a higher level in the upper flank and to a lower level in the lower flank (Gutjahr *et al.*, 2005). JA treatment delays gravitropic bending, and the JA-deficient mutant *hebiba* exhibits a slower gravitropic bending response, suggesting that JA is required for normal gravitropism in rice coleoptiles (Gutjahr *et al.*, 2005).

Regulation of cotton fiber elongation

Cotton fibers are the most important natural material in the textile industry. JA positively regulates cotton fiber elongation. The class I TCP TF GbTCP promotes JA biosynthesis, activates the expression of genes required for fiber elongation, and controls fiber elongation and quality (Hao *et al.*, 2012). Cotton GhJAZ2 suppresses fiber initiation by interacting with GhMYB25-like, GhGL1, GhMYC2, GhWD40, and GhJI1 (Hu *et al.*, 2016). *GhJAZ2* overexpression inhibits lint and fuzz fiber initiation, as well as fiber elongation (Hu *et al.*, 2016).

Other developmental roles of JA

JA induces tendril coiling in *Bryonia dioica*. OPDA might be the endogenous hormone responsible for tendril coiling, owing to its dramatic increase during the initiation and progression of coiling (Falkenstein *et al.*, 1991). In rice, the JA-responsive receptor-like protein Root Meander Curling (OsRMC) regulates root curling, and the knockdown of OsRMC enhances root coiling in response to JA treatment (Jiang *et al.*, 2007). JA induces tuber formation in potato; an antisense mutant of potato *LOX* showed decreased tuber formation (Kolomiets *et al.*, 2001). Ectopic expression of JAZ1 or the mutation of MYC2 induces indole-3-acetic acid biosynthesis and promotes somatic embryogenesis in Arabidopsis (Mira *et al.*, 2016). Moss produces *cis*-(+)-OPDA but not JA, and the mutation of PpAOC1 or PpAOC2 results in aberrant sporophyte morphology, defective sporogenesis, and reduced fertility (Stumpe *et al.*, 2010).

Conclusions

Since the 1980s, enormous effort has been made to investigate the functions of JAs. As a result, researchers have uncovered key roles for JAs in various developmental and growth-related processes, demonstrating that JAs are an essential growth regulator. JAs display conservative functions among different plant species, including the inhibition of seedling growth and induction of leaf senescence. JAs also regulate different reproductive and developmental processes across plant species, including stamen development in Arabidopsis, spikelet and stamen development in rice, tassel development in maize, and maternal control of embryo development in tomato. With the development of advanced genetic and biochemical methods, novel JA functions, key components, and the molecular basis of JA action in plant development and growth will be revealed.

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