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REVIEW PAPER

Jasmonate-regulated seed germination and crosstalk with other phytohormones

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Abstract

Seed plants have evolved mechanisms that maintain the dormancy of mature seeds until the time is appropriate for germination. Seed germination is a critical step in the plant life cycle, and it is an important trait in relation to agricultural production. The process is precisely regulated by various internal and external factors, and in particular by diverse endogenous hormones. Jasmonates (JAs) are one of the main plant hormones that mediate stress responses, and recent studies have provided evidence of their inhibitory effects on seed germination. In this review, we summarize our current understanding of the molecular mechanisms underlying the regulatory roles of JAs during the seed germination stage. We describe the crosstalk between JA and other phytohormones that influence seed germination, such as abscisic acid and gibberellic acid.

Keywords: Abscisic acid, crosstalk, jasmonate, jasmonic acid, gibberellic acid, phytohormones, seed germination.

Introduction

Plants are sessile organisms that have evolved developmental adaptations to survive in unfavorable environmental conditions. The plant life cycle starts with seeds that germinate to produce seedlings, in which the vegetative phase precedes the reproductive phase. The seed generally enables the embryo to survive the seed maturation stage and the seedling to survive the establishment stage, thereby ensuring the initiation of the next generation (Penfield, 2017). There are two vital seed development phases, namely zygotic embryogenesis and seed maturation. During the maturation phase, seeds are affected by various physiological processes (e.g. dormancy and germination) that will ultimately result in the establishment of a seedling (Graeber *et al.*, 2012; Righetti *et al.*, 2015). Seed germination is critical for the reproduction of plant species and it is precisely regulated by developmental and environmental signals. In addition, it is an important yield-related trait in crop species.

Germination marks the start of the life cycle of all higher plants, and it can affect the evolution of expressed traits (Donohue *et al.*, 2010). Seed germination occurs when dormant seeds take up water, after which part of the embryo breaks through the seed coat (Bewley, 1997). The emergence of the radicle signifies the completion of germination (Bewley, 1997). Because germination requires suitable conditions, the sensitivity

of seeds to environmental stimuli changes continuously as part of the adaptation to ambient conditions. Thus, seed germination is regulated by endogenous components (e.g. phytohormones) and by environmental signals (e.g. water, temperature, and light).

Seeds perceive external signals and then endogenous signaling pathways, especially those related to phytohormones, induce downstream responses (e.g. seed germination and dormancy) (Kendall *et al.*, 2011; Shu *et al.*, 2018). Phytohormones have key roles in affecting various processes throughout the plant life cycle, including seed maturation, germination, and the floral transition, and are also involved in responses to abiotic and biotic stress (Shu *et al.*, 2016; Yang and Li, 2017). For example, abscisic acid (ABA), gibberellic acid (GA), auxin, and ethylene have important functions related to seed dormancy and germination (Finkelstein *et al.*, 2008; Wilson *et al.*, 2014; H. Wang *et al.*, 2020). In addition, recent studies have also revealed the regulatory effects of jasmonates (JAs) on seed germination, either alone or with other phytohormones (Hou *et al.*, 2010; Dave *et al.*, 2011, 2016; Pauwels *et al*, 2015; Shu *et al.*, 2016; Barros-Galvão *et al.*, 2019; Varshney and Majee, 2021).

In general, ABA suppresses seed germination. The ABA receptors PYRABACTIN RESISTANCE (PTR)/REGULATORY COMPOMENT OF ABSICSIC ACID RECEPTOR (RCAR) recognize the ABA molecule (Ma *et al.*, 2009; Miyazono *et al.*, 2009; Nishimura *et al.*, 2009; Santiago *et al.*, 2009). When binding to ABA, these receptors cause the formation of a stable complex with type 2C protein phosphatases (PP2Cs), leading to the release of SNF1-related kinases 2 (SnRK2s) from PP2C–SnRK2 complexes (Cutler *et al.*, 2010). This complex helps the activated SnRK2s to subsequently phosphorylate downstream transcription factors, such as ABSCISIC ACID RESPONSIVE ELEMENT (ABRE) binding factors, ABSCISIC ACID-INSENSITIVE 5 (ABI5), ABI4, and ABI3 to mediate ABA responses (Nakashima *et al.*, 2009). These transcription factors in ABA signaling then bind to the promoters of ABA-responsive target genes involved in germination to regulate their expression, for example *EARLY METHIONINE-LABELED 6* (*EM6*) and *EM1*, which inhibit seed germination (Hu *et al.*, 2019).

Jasmonates are a class of lipid-derived, natural, and widely distributed hormones in plants. Their metabolic derivatives include jasmonic acid–isoleucine (JA-Ile) and methyl jasmonate (MeJA). The bioactive form of JA is recognized by the receptor CORONATINE-INSENSITIVE PROTEIN 1 (COI1) and the JASMONATE DOMAIN (JAZ) transcriptional repressor, which results in the degradation of JAZ proteins and the release of transcription factors, including MYC2/3/4. The transcription factors subsequently activate the expression of JA-related genes (Chini *et al.*, 2007; Thines *et al.*, 2007). Research has indicated that JA signaling mediates plant defenses against herbivores and pathogens, while also conferring abiotic stress tolerance (Nahar *et al.*, 2011; Yan and Xie, 2015). In addition to its regulatory roles related to defense responses, JA is required for plant reproduction, growth, and developmental processes (Wasternack and Hause, 2013).

The effects of several phytohormones on seed dormancy and germination have been extensively studied, especially ABA and GA. Several studies have been conducted to clarify how the JA-mediated signaling pathway influences seed germination (Dave *et al.*, 2011, 2016; Lackman *et al.*, 2011; Aleman *et al.*, 2016; Barros-Galvão *et al.*, 2019; Ju *et al.*, 2019; Pan *et al.*, 2020; Y. Wang *et al.*, 2020); however, the mechanisms underlying the regulation of seed germination by JA have not been comprehensively determined. In this review, we summarize recent advances in the characterization of the effects of JA on seed germination and its associated crosstalk with other phytohormones.

Overview of JA biosynthesis and signaling

The JA biosynthesis pathway was first described in *Vicia faba* by Vick and Zimmerman (1983). Research over the past 40 years has demonstrated that JA is synthesized in different subcellular compartments via the octadecanoid pathway, in which octadecatrienoic acid (18:3n-3) is oxidized by 13-lipoxygenase (13-LOX) to form 13-hydroperoxylinolenic acid in the plastids or chloroplasts. In the plastids, 13-LOX catalyses the oxidation of α-LeA to produce (13S)-hydroperoxy octadecatrienoic acid, which is a substrate for cytochrome P450 enzymes from the CYP74 family, allene oxide synthase (AOS), hydroperoxide lyase, epoxyalcohol synthase, and divinyl ether synthase. Subsequently, 13-hydroperoxylinolenic acid is converted to *cis*-(+)-12-oxo-phytodienoic acid (*cis*-OPDA) by different enzymes, including AOS and allene oxide cyclase (AOC) (Wasternack and Hause, 2013). The genes encoding four AOCs in Arabidopsis are expressed locally and systemically (Delker *et al.*, 2006). The ATP-binding cassette transporter COMATOSE (CTS) helps transport *cis*-OPDA through the cytosol to the peroxisome, where it is converted to JA via a reduction and three cycles of β-oxidation (Bussell *et al.*, 2013). The reduction of OPDA is catalysed by OPDA reductase (OPR). In Arabidopsis, only OPR3 contains a peroxisome-targeting sequence. Moreover, OPR3 is activated by carboxyl-CoA ligase, resulting in the production of OPC-8:0. The three cycles of β-oxidation are catalysed by acyl-CoA oxidase (ACX), a multifunctional protein (MFP), and L-3-ketoacyl-CoA thiolase (KAT). Finally, JA is exported into the cytoplasm and then modified by JA carboxyl methyltransferase or JA conjugate synthase (JAR1) to form MeJA or JA-Ile, respectively (Staswick and Tiryaki, 2004). An alternative pathway has been revealed to produce JA from dinor-OPDA [2,3-dinor-12-oxo-10, 15(*Z*)-phytodienoic acid; dn-OPDA)] (Chini *et al.*, 2018), which is the bioactive ligand in *Marchantia polymorpha* (Monte *et al.*, 2018, 2019). Therefore, OPDA, dn-OPDA, and their derivatives are also considered as JAs (Park *et al.*, 2013; Howe, 2018; Fig. 1).

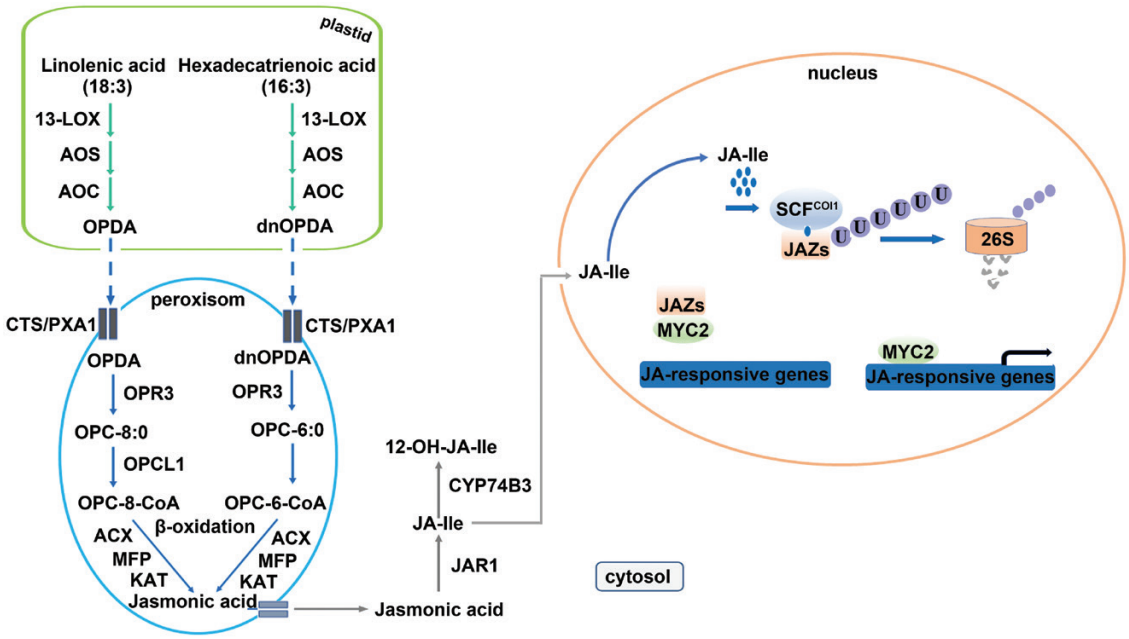

Fig. 1. A simplified model for jasmonate (JA) biosynthesis and signaling. JA-Ile, which is synthesized by JA biosynthetic enzymes in plastids, peroxisomes, and the cytosol, can be inactivated by CYP94B3. JA-Ile induces the interaction of CORNATINE INSENSITIVE1 (COI1) with JA ZIM-domain (JAZ) proteins, leading to the ubiquitination and degradation of JAZs via the 26S proteasome, and thus the downstream transcription factor MYC2 is de-repressed, allowing it to activate early JA-responsive genes and hence leading to JA responses. Abbreviations: 13-LOX, 13-lipoxygenase; AOS, allene oxide synthase; AOC, allene oxide cyclase; dnOPDA, dinor-oxo-phytodienoic acid; CTS, COMATOSE ABC transporter; OPR3, 12-oxophytodienoate reductase 3; OPC-8, 8-[3-oxo-2{cis-2´-pentenyl}-cyclopentase]-1-octanoic acid; OPCL1, OPC-8:0 CoA ligase 1; ACX, acyl-CoA oxidase; MFP, multifunctional protein; OPC-6, 6-[3-oxo-2{cis-2´-pentenyl}-cyclopentase]-1-octanoic acid; KAT, L-3-ketoacyl-CoA thiolase; JAR1, JA-amido synthetase; CYP74B3, JA-Ile-12-hydroxylase; JA-Ile, jasmonoyl-*L*-isoleucine; JAZs, jasmonate ZIM-domain proteins.

Jasmonates (including the biosynthetic precursor *cis*-OPDA) are signaling molecules related to plant stress responses, physiological reactions, and developmental processes, and are perceived by a component of the E3 ubiquitin ligase complex SCF^{COI1} (Xie *et al.*, 1998; Xu *et al.*, 2002; Yan *et al.*, 2009; Sheard *et al.*, 2010), which recruits JAZ proteins for the ubiquitination and degradation via the 26S proteasome pathway (Chini *et al.*, 2007; Thines *et al.*, 2007; Yan *et al.*, 2007). In the absence of JA, the degradation of JAZ proteins eliminates the inhibition of the downstream transcription factors, such as MYC2 (Pauwels *et al.*, 2010; Zhang *et al.*, 2015; Wasternack and Song, 2017), which initiates JA signaling cascades by activating the transcription of some genes encoding transcription factors, including ETHYLENE RESPONSE FACTOR1 (ERF1) and OCTADECANOID-RESPONSIVE ARABIDOPSIS59 (ORA59) (Lorenzo *et al.*, 2004; Fig.1).

Diverse JA derivatives mediate seed germination

Jasmonates, such as MeJA and JA-Ile, and their precursor OPDA, are involved in biotic and abiotic stress responses (Wasternack and Kombrink, 2009; Hu *et al.*, 2017) while also regulating plant growth and development (e.g. reproduction, pollen and embryo development) (Fey *et al.*, 1994; Balbi and Devoto, 2008; Wasternack and Kombrink, 2009). MeJA and OPDA can also affect seed germination (Preston *et al.*, 2009; Dave *et al.*, 2011).

Application of exogenous JAs inhibits seed germination

Thines *et al.* (2007) reported that JA-Ile, but not JA, MeJA, or OPDA, has vital roles in JA responses. For example, only JA-Ile promotes the interaction between the COI1 and JAZ proteins that results in the ubiquitin-dependent degradation of the latter. Although different JA derivatives or their precursor might have different functions in the responses, application of exogenous JA or MeJA can inhibit seed germination in *Arabidopsis thaliana*, *Brassica napus*, *Linum usitatissimum*, *Lupinus luteus*, *Solanum lycopersicum*, and *Zea mays* (Wilen *et al.*, 1991; Preston *et al.*, 2002, 2009; Miersch *et al.*, 2008; Oh *et al.*, 2009; Zalewski *et al.*, 2010; Dave *et al.*, 2011). In addition, exogenous OPDA also inhibits seed germination, and approximately 10-times more efficiently than JA. The synergistic inhibitory effects of OPDA and ABA on the germination of Arabidopsis seeds are similar to those of MeJA and ABA (Wilen *et al.*, 1991; Ellis and Turner, 2002; Nambara *et al.*, 2010; Pan *et al.*, 2020). In a recent study, we observed synergism between coronatine, which

Table 1. Germination potential in Arabidopsis mutants with disrupted fatty acid β-oxidation

AGI gene code	Protein	Mutant allele	Disrupted process	Germination potential	Genetic background	References
At4g39850	COMATOSE ABC transporter	*cts-1*	β-oxidation	Compromised in germination potential	Ler	Russell *et al.* (2000)
At4g39850	COMATOSE ABC transporter	*cts-2*	β-oxidation	Compromised in germination potential	Ws	Footitt *et al.* (2002); Dave *et al.* (2011)
At4g39850	COMATOSE ABC transporter	*pxa1-1*	β-oxidation	Impaired germination phenotype	Col-0	Zolman *et al.* (2001)
At2g06050	OXOPHYTODIENOIC ACID REDUCTASE 3	*opr3-1*	Jasmonate biosynthesis	Impaired germination phenotype	Ws	Stintzi and Browse (2000); Dave *et al.* (2016)
At4g16760, At5g65110	Long-chain acyl-CoAs	*acx1-2*, *acx2-1*	β-oxidation	Compromised in germination potential	Ws, Col-0	Pinfield-Wells *et al.* (2005); Dave *et al.* (2011)
At2g33150	Thiolase	*kat2-1*	β-oxidation	Impaired germination phenotype	Ws	Germain *et al.* (2001); Dave *et al.* (2011)
At3g58750, At2g42790	Peroxisomal citrate synthases	*csy2-1*, *csy3-1*	β-oxidation	Impaired germination phenotype	Col-0	Pracharoenwattana *et al.* (2005)

is a JA analog that is structurally similar to the active conjugate JA-Ile, and ABA during the inhibition of Arabidopsis seed germination (Pan *et al.*, 2020). These results indicate that exogenous JAs and ABA have synergistic effects that lead to delayed germination.

The content of endogenous JAs varies during seed development

The contents of diverse endogenous JA derivatives in Arabidopsis seeds have been quantified in order to clarify the molecular mechanisms by which JAs regulate seed germination. Preston *et al.* (2009) reported that JA and JA-Ile contents vary between non-dormant Columbia (Col-0) seeds and dormant Cape Verde Island (Cvi) seeds. An examination of dry seeds indicated that the non-dormant Col seeds contain 10-fold more JA and JA-Ile than the dormant Cvi seeds, but that the contents in the Col-0 seeds decrease during imbibition. Moreover, Dave *et al.* (2011) observed that JA, JA-Ile, and OPDA accumulate during the early seed development period in Col-0. It therefore seems that the content of endogenous JAs is related to the seed development stages, and that the levels decrease during germination; however, the detailed relationship between endogenous JA content and germination remains unclear.

Peroxisomal β-oxidation mutants show affected seed germination

JA synthesis is dependent on peroxisomal β-oxidation (Baker *et al.*, 2006; Graham, 2008), and hence its possible involvement in the regulation of germination can be investigated in mutants where this process is disrupted. The Arabidopsis mutants *acx1 acx2*, *csy2 csy3*, *cts*, *kat2*, *pxa1*, and *opr3*, which are unable to properly catabolyse fatty acids derived from storage oils and to synthesize JA, reportedly exhibit defective early seedling establishment and require the addition of exogenous sucrose (Russell *et al.*, 2000; Footitt *et al.*, 2002, 2006; Adham *et al.*, 2005; Pinfield-Wells *et al.*, 2005; Pracharoenwattana *et al.*, 2005; Table 1). These mutants for peroxisomal β-oxidation are severely compromised in seed germination (Table 1), suggesting that this biochemical process is involved in regulating germination potential. The impaired germination phenotype in peroxisomal β-oxidation mutants might be caused by changes in the contents of different endogenous JA derivatives. For instance, in the *cts-1* and *cts-2* mutants, there is an increase in the JA, JA-Ile, and OPDA contents (Dave *et al.*, 2011). Compared with wild-type seeds, those of the *aos* mutant, in which JA, JA-Ile, and OPDA accumulation is inhibited, are less dormant, whereas the seeds of the *cts1-1* and *opr3-1* mutants, which over-accumulate OPDA but are deficient in JA and JA-Ile, are more dormant (Park *et al.*, 2002; Chehab *et al.*, 2011; Dave *et al.*, 2011, 2016). However, the germination rate is unaffected in some seed mutants with disruption of storage-oil breakdown, including the triacylglycerol lipase mutant *sdp1* and the peroxisomal long-chain acyl CoA synthetase double-mutant *lacs6 lacs7* (Russell *et al.*, 2000; Fulda *et al.*, 2004; Eastmond, 2006; Quettier *et al.*, 2008).

Current evidence shows that mutations in JA signaling genes (including *COI1*, *JAZ*s, and *MYC2*) do not exhibit impaired germination phenotypes under normal conditions (Ellis and Turner, 2002; Abe *et al.*, 2003; Dave *et al.*, 2011; R. Chen *et al.*, 2012; Ju *et al.*, 2019; Pan *et al.*, 2020), while mutating or overexpressing certain genes does affect seed germination rates following ABA treatment (Abe *et al.*, 2003; Dave *et al.*, 2011; Goossens *et al.*, 2015; Ju *et al.*, 2019; Pan *et al.*, 2020). Impaired germination phenotypes exhibited by JA synthesis mutants might be caused by the changes in content of endogenous JA, and JA signaling can directly or indirectly regulate other phytohormone biosynthesis (mainly ABA) and signaling pathways to affect germination rates. Hence, there are no

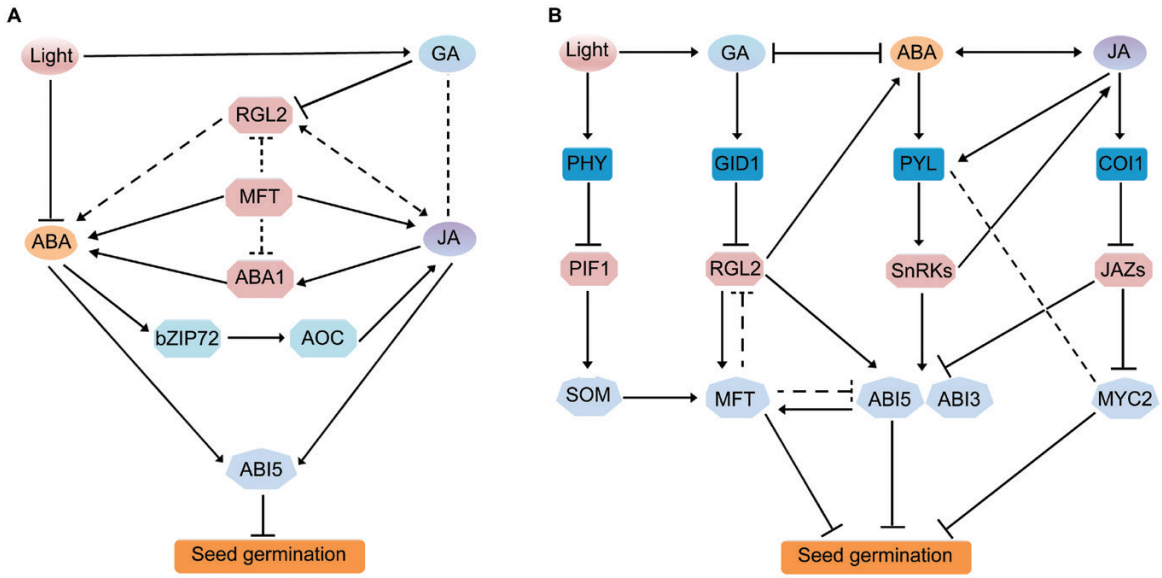

Fig. 2. Jasmonate-mediated crosstalk affecting seed germination. Jasmonate-mediated germination is regulated by (A) hormone biosynthesis and (B) hormone-mediated seed sensitivity. (A) Light induces GA accumulation and inhibits ABA synthesis in the process of germination. GA effects the content of JA through the DELLA protein member RGL2, and JA also regulates the expression of *ABA1* to enhance ABA accumulation. In addition, ABA modulates JA synthesis through the bZIP72–AOC pathway. (B) JA-inhibited germination in seeds is regulated by alterations in ABA and GA synthesis and/or signaling. JA activates ABA signaling via degrading JAZs, releasing the transcription factors ABI3 and ABI5, thereby delaying germination. JA also interacts with GA and light signaling, influencing the ABA:GA ratio through the RGL2–ABI5–MFT and PIF1–SOM–MFT pathways. Arrows represent activation and lines ending in bars indicate suppression. Abbreviations: JA, jasmonate; ABA, abscisic acid; GA, gibberellin; ABA1, ABA deficient 1; ABI3, abscisic acid insensitive 3; ABI5, abscisic acid insensitive 5; MFT, mother of FT and TFL 1; RGL2, RGA-like 2; GID1, GA insensitive dwarf 1; PYL, regulatory component of ABA receptor; COI1, coronatine insensitive 1; SnRKs, serine/threonine kinases; PHY, phytochromes; PIF1, PHYTOCHROME INTERACTING FACTOR 1; SOM, SOMNUS.

obvious germination phenotypes associated with different JA signaling components.

Crosstalk between JA and other phytohormones modulates seed germination

JA and ABA

Abscisic acid represses seed germination by inhibiting cell-wall loosening and expansion, which is a key step for initiating germination (Gimeno-Gilles *et al.*, 2009). There is considerable genetic evidence for the inhibitory effect of ABA on germination. Interestingly, some studies have indicated JA biosynthesis is stimulated by exogenous ABA and by stress-induced increases in endogenous ABA (Adie *et al.*, 2007; Ju *et al.*, 2019; Pan *et al.*, 2020). Jasmonates are generally believed to function synergistically with ABA in most biological processes, including seed germination (Krock *et al.*, 2002; Dave *et al.*, 2011, 2016; Liu *et al.*, 2015; Barros-Galvão *et al.*, 2019; Ju *et al.*, 2019; Pan *et al.*, 2020; Tang *et al.*, 2020; Y. Wang *et al.*, 2020). However, JA and ABA appear to have antagonistic effects in a few plant responses to biotic stress (Garcia-Andrade *et al.*, 2011; Xie *et al.*, 2018). The relationship between JA and ABA has been confirmed by examining mutants in which ABA or JA biosynthesis and signaling are disrupted (Staswick *et al.*, 1992; Berger *et al.*, 1996; Ellis and Turner, 2002; Abe *et al.*, 2003; Lorenzo *et al.*, 2004; Kanai *et al.*, 2010; Lackman *et al.*, 2011; Nakata *et al.*, 2013; Goossens *et al.*, 2015; Pauwels *et al.*, 2015; Aleman *et al.*, 2016).

The role of the JA pathway in regulating seed germination through ABA signaling.

Peroxisomal β-oxidation genes regulate both the transcript and protein levels of ABSCISIC ACID INSENSITIVE5 (ABI5). Kanai *et al.* (2010) first detected increased transcription of the gene encoding the bZIP-type transcription factor ABI5 in the *ped3* mutant (an allele of *cts*). Genetic analyses showed that the *abi5* mutation is able to rescue the defective germination of *ped3*, and thus the activation of *PED3* expression by imbibition reduces ABI5 transcripts and promotes seed germination. Moreover, application of 10 μM OPDA results in an increase in ABI5 levels (Dave *et al.*, 2011). Therefore, the JA biosynthesis pathway might regulate ABI5 levels to mediate seed germination (Fig. 2A).

Apart from the JA biosynthesis mutant *ped3*, other JA signaling components are also involved in ABA responses during seed germination. Seeds with a mutation in the JA receptor F-box protein COI1 exhibit a much higher percentage of germination than wild-type seeds when ABA is applied (Pan

et al., 2020). Seeds overexpressing *JAZ*s with a deleted Jas domain (e.g. *JAZ1*, *JAZ3*, *JAZ5*, and *JAZ8*) are less insensitive to ABA, while the mutant *jazQ* with T-DNA insertion mutations in *JAZ1/3/4/9/10* and the mutant *jazD* that is defective in *JAZ1–7/9/10/13* is more sensitive than the wild type to ABA during seed germination (Ju *et al.*, 2019; Liu *et al.*, 2019; Pan *et al.*, 2020). The transcription factor MYC2 has been shown to be a positive regulator of the ABA signaling associated with ABA-mediated inhibition of germination. Several studies have demonstrate that *MYC2*-overexpressing plants are hypersensitive to ABA, whereas plants in which this gene is knocked out (i.e. *myc2* mutant) exhibit decreased ABA sensitivity (Abe *et al.*, 2003; Lorenzo *et al.*, 2004; Yadav *et al.*, 2005; Gangappa *et al.*, 2010). In addition, MYC2 can interact with the ABA receptor PYRABACTIN RESISTANCE (PYR)/PYR1-LIKE (PYL)/REGULATORY COMPONENT OF ABSCISIC ACID RECEPTOR 6 (PYL6) (Fig. 2B). The *pyl6* receptor mutant is more sensitive to JA and ABA together than to ABA alone (Aleman *et al.*, 2016). These findings confirm that JAs regulate seed germination in a process that may depend on ABA signaling.

JA acts synergistically with ABA signaling to delay seed germination.

Dave *et al.* (2011) first reported the effects of the crosstalk between JA and ABA on seed germination. Specifically, they observed that OPDA combined with ABA inhibits seed germination. Exogenously applied OPDA induces the expression of *ABI5* in plants. In addition, the ABI5 content is more stable in the presence of both OPDA and ABA than in the presence of either one separately. However, in a more recent study, OPDA was found to be ineffective when germination assays were performed using the ABA biosynthesis mutant *aba1-1*, but it regulated the expression of *ABA1* and *ABI5*, which are involved in ABA production (Barros-Galvão *et al.*, 2019). Hence, OPDA induces ABA accumulation by increasing *ABA1* expression, while it influences ABA sensitivity by increasing *ABI5* expression and stabilizing the ABI5 protein (Kanai *et al.*, 2010; Dave *et al.*, 2011; Barros-Galvão *et al.*, 2019) (Fig. 2A).

The JAZ proteins that negatively regulate ABA responses during the germination of wheat and Arabidopsis seeds have been functionally characterized by Ju *et al.* (2019). First, the interaction between JAZ and ABI5 was confirmed. Consistent with this, we have recently shown that seed germination percentage decreases after treatment with ABA combined with coronatine, which is structurally similar to JA-Ile. This inhibition is indicative of a physical interaction between ABI3 and JAZ1/JAZ5/JAZ8, with the JAZ proteins suppressing the transcriptional activation by ABI3 and ABI5, thereby inhibiting the expression of the downstream partners (Pan *et al.*, 2020). Interestingly, the rice SnRK2 member SAPK10 together with ABA participate in the phosphorylation and stabilization of bZIP72, which activates the expression of the JA biosynthesis gene *AOC* by binding to the G-box motif. Thus, the SAPK10–bZIP72–AOC pathway inhibits seed germination (Y. Wang *et al.*, 2020).

In summary, ABA can promote JA synthesis via the SAPK10–bZIP72–AOC pathway to synergistically inhibit seed germination (Fig. 2). Simultaneously, jasmonate activates ABA signaling through a process involving the physical interaction between JAZ and ABI3/ABI5, which suppresses their transcriptional ability (Fig. 2B). Moreover, the perception of ABA triggers JA biosynthesis and signaling, leading to the degradation of JAZ proteins and the activation of ABI3/ABI5, thus inducing the expression of *EM6* and *EM1* that leads to the repression of seed germination. The resulting decrease in the expression and function of downstream genes leads to delayed seed germination. Thus, there is evidence that JA-mediated seed germination is mainly associated with ABA.

JA and GA

Together with ABA, GA forms a pair of classic phytohormones that antagonistically mediate several development-related processes and modulate the transition between dormancy and germination (Yazaki and Kikuchi, 2005; Finch-Savage and Leubner-Metzger, 2006; Finkelstein *et al.*, 2008). Moreover, GAs substantially affect various aspects of plant development, including flowering and seed germination (Sun, 2008; Yamaguchi, 2008). Growth-repressing DELLA proteins are integral to GA signal transduction. Gibberellic acid can bind to the receptor GA INSENSITIVE DWARF1 (GID1) and enhances the GID1–DELLA interaction, leading to the degradation of DELLA by the ubiquitin–proteasome pathway (Murase *et al.*, 2008; Sun, 2010). Moreover, DELLA proteins repress germination. Of the five DELLA members in Arabidopsis, RGA-LIKE2 (RGL2) is the major one involved in seed germination (Lee *et al.*, 2002; Tyler *et al.*, 2004; Cao *et al.*, 2005; Penfield *et al.*, 2006; Piskurewicz *et al.*, 2008; Piskurewicz and Lopez-Molina, 2009). In contrast to seeds of the *aba1-1* mutant, seed germination of *rgl2-1* is not inhibited by OPDA (Dave *et al.*, 2016); in addition, GA accumulation in seeds is unaffected by OPDA treatment, whereas ABA accumulation increases in wild-type Col and Landsberg *erecta* seeds. Furthermore, the increase in the ABA content is considerably inhibited in *rgl2-1* mutant seeds (Barros-Galvão *et al.*, 2019). These observations are consistent with earlier research that indicated that RGL2 indirectly promotes ABA biosynthesis (Lee *et al.*, 2010). Therefore, exogenously applied OPDA mainly triggers ABA accumulation by increasing *RGL2* and *ABA1* expression, ultimately resulting in inhibited seed germination (Fig. 2A).

Although crosstalk between the JA and GA signaling pathways has been reported for non-seed systems (Navarro *et al.*, 2008; Hou *et al.*, 2010; Wild *et al.*, 2012; Yang *et al.*, 2012; Qi *et al.*, 2014; Osadchuk *et al.*, 2019), their interaction during seed germination is unclear. In terms of the effects of GA signaling, GA-deficient *ga1-3* mutant seeds accumulate relatively little JA-Ile, but have significantly increased OPDA

levels. A significant decrease has been shown in the effect of the GA biosynthesis-inhibitor Paclobutrazol (PAC) and ABA on after-ripened *aos* mutant seeds, which are abnormal in an early step in the oxylipin pathway upstream of OPDA (Park *et al.*, 2002). Consistent with this finding, *opr3-1* seeds, which cannot convert OPDA to JA (Stintzi and Browse, 2000), are hypersensitive to ABA and PAC (Dave *et al.*, 2016). Accordingly, it is possible that GA and JA interact indirectly via the ABA signaling pathway. Thus, the GA-induced degradation of RGL2 indirectly promotes the accumulation of oxylipins (specifically JA) via stimulating ABA biosynthesis and increasing ABI5 levels (Piskurewicz *et al.*, 2008; Fig. 2). The inhibitory effects of PAC and ABA on germination are partially augmented by JA, reflecting the feedback from GA and ABA that regulates JA accumulation (Fig. 2A).

In addition to seed germination, GA promotes embryo development by releasing the inhibitory effect of DELLA on LEAFY COTYLEDON 1, with the resulting increase in auxin accumulation being conducive to embryo development (Hu *et al.*, 2018). Gibberellic acid also negatively affects the accumulation of seed oil and seed storage proteins (M. Chen *et al.*, 2012; Hu *et al.*, 2021). The DELLA protein RGA-LIKE3 serves as a co-activator of ABI3 to promote the biosynthesis of seed storage proteins during the seed maturation stage (Hu *et al.*, 2021). These findings suggest that the interaction between JA and GA during seed development might not depend on other phytohormones; however, the regulatory effects of GA and JA interactions on seed germination are mediated by ABA signaling.

JA, GA, and ABA

The roles of MOTHER OF FT AND TFL1 (MFT) on JA-mediated seed germination

In Arabidopsis, MFT belongs to the phosphatidyl ethanolamine-binding protein family. Although MFT, FLOWERING LOCUS T (FT), and TERMINAL-FLOWER1 (TFL1) are involved in the regulation of flowering time, MFT also negatively regulates ABA signaling, and *MFT* expression is promoted by RGL2 and ABI5 (Xi *et al.*, 2010; Fig. 2). A transcriptome analysis has revealed that *MFT* expression is higher in the *pxal-1* (*cts*) mutant than in wild-type seeds, and that *MFT* expression levels increase following the treatment of after-ripened wild-type seeds with OPDA (Dave *et al.*, 2016). These observations imply that endogenous or exogenous OPDA levels affect the expression of the dormancy-promoting *MFT* gene. Furthermore, *mft-2* mutant seeds are insensitive to OPDA (Dave *et al.*, 2016), suggesting that MFT is required for the OPDA-induced inhibition of seed germination. In addition, the ABA content and expression levels of *AOS* are lower in *mft-2* seeds than in control seeds. Hence, to trigger ABA biosynthesis, OPDA requires RGL2 as well as MFT (Fig. 2). Moreover, there might be MFT-to-OPDA feedback interactions. The DELLA proteins that repress GA signaling can promote ABA accumulation by inducing *XERICO* expression, but the mechanism by which XERICO enhances ABA accumulation is unknown (Ko *et al.*, 2006; Zentella *et al.*, 2007; Piskurewicz *et al.*, 2008). It has been shown that MFT influences ABA biosynthesis by promoting *ABA1* expression, while also affecting ABA sensitivity by negatively regulating *ABI5* and *RGL2* expression (Xi *et al.*, 2010; Fig. 2). Therefore, JA signaling through MFT might promote ABA biosynthesis and increase ABA sensitivity, with RGL2 and MFT positively affecting this process by inducing OPDA accumulation (Fig. 2A).

Effects of light on hormonal crosstalk during seed germination

Evidence shows that ABI5, MFT, and RGL2 together with the JA pathway have inhibitory effects on seed germination (Dave *et al.*, 2016; Barros-Galvão *et al.*, 2019). Jasmonate also helps to regulate plant responses mediated by light (Yadav *et al.*, 2005; Ortigosa *et al.*, 2020), and light-dependent seed germination is induced and inhibited by GA and ABA, respectively (Barros-Galvão *et al.*, 2019). Excess red light (R) triggers GA accumulation and germination, whereas excess far-red light (FR; typical of shade conditions), triggers ABA accumulation and a block in germination (Seo *et al.*, 2006; Piskurewicz *et al.*, 2008). Of the five phytochromes phyA–E, phyB is the main promoter of germination under sunlight, while phyA is responsible for germination in shade conditions (Clack *et al.*, 1994; Shinomura *et al.*, 1994), and both are synthesized as inactive proteins that become active forms in a manner that is dependent on light quality. Upon activation, the transcription factor PHYTOCHROME INTERACTING FACTOR 1 (PIF1) is degraded by both phyA and phyB (Shen *et al.*, 2005; Park *et al.*, 2012), whereas under phytochrome inactivation, PIF1 accumulates and regulates the transcription of *SOMNUS* (*SOM*), leading to high ABA:GA ratios that repress germination (Oh *et al.*, 2004; Kim *et al.*, 2008, 2016; Park *et al.*, 2011a). Interestingly, PIF1 and SOM can promote *MFT* expression, and MFT has a vital role in repressing germination via regulating GA and ABA responses (Vaistij *et al.*, 2018). Moreover, PIF1 stimulates the expression of *RGA* and *GAI*, and the resulting two proteins act with another DELLA protein, RGL2, to repress seed germination specifically in the darkness (Cao *et al.*, 2005; Piskurewicz and Lopez-Molina, 2009). Under FR conditions, RGA, GAI, and RGL2 repress germination by stimulating ABA biosynthesis and further increasing the ABA:GA ratio (Lee *et al.*, 2012).

Seeds of the *aos* mutant and the ABA biosynthesis-deficient *aba2-1* mutant do not germinate under FR light conditions, which is in contrast to the high seed germination rate of the *aos aba2-1* double-mutant (Barros-Galvão *et al.*, 2019). This study found that OPDA plays a key role in the FR-triggered repression of germination, and alongside ABA it represses germination under shade conditions through an MFT-mediated process. There is a model in which the repression effect of OPDA and ABA on germination under shade conditions is

partially modulated by MFT (Fig. 2). Under FR light conditions phyB is deactivated, with the resulting accumulation of ABA or OPDA being sufficient to repress germination and increase the expression of *MFT* (encoding a germination repressor). In the absence of either ABA or OPDA, the decrease in the level of MFT is sufficient for preventing germination. When both OPDA and ABA are absent, the low *MFT* expression level is insufficient for repressing seed germination (Barros-Galvão *et al.*, 2019; Fig. 2).

JA and other phytohormones

Ethylene (ET) has diverse effects on plant development, one of which is to promote seed germination (Arc *et al.*, 2013; Corbineau *et al.*, 2014). In higher plants, ET is produced from methionine in the Yang cycle (Yang and Hoffman, 1984; Lin *et al.*, 2009). The enzyme catalysing the final rate-limiting step in the ET biosynthesis pathway (ACO) regulates seed germination (Kucera *et al.*, 2005; Matilla and Matilla-Vázquez, 2008; Linkies *et al.*, 2009). Seeds of the ET-insensitive mutant *etr1* germinate poorly (Beaudoin *et al.*, 2000; Chiwocha *et al.*, 2005). Pluskota *et al.* (2019) determined that the transcription levels of *SINP24* and the gene encoding its potential upstream regulator TERF1 (ethylene response factor) increase in response to MeJA in germinating tomato seeds. In addition, JA directly regulates *ERF1* expression during defense responses to pathogens (Solano *et al.*, 1998; Lorenzo *et al.*, 2003). Although ET and JA function synergistically or antagonistically during plant responses to developmental processes and stresses (Lorenzo and Solano, 2005), there has been relatively little research on their interaction during seed germination.

In addition to ABA, GA, and ET, almost all other phytohormones are likely to be involved in the regulation of seed germination, including auxin, brassinosteroids (BRs), cytokinins (CTKs), strigolactones (SLs), and salicylic acid (SA). Application of exogenous auxin suppresses seed germination under highly saline conditions; however, auxin alone is not considered a key regulator of seed germination, and it participates in crosstalk with ABA during germination (Park *et al.*, 2011b; L. Wang *et al.*, 2011). Unlike ABA, BR promotes seed germination partly through the MFT-mediated pathway, which forms a negative feedback loop that regulates ABA signaling (Xi and Yu, 2010; Xi *et al.*, 2010). A key repressor of BR signaling, BRASSINOSTEROID INSENSITIVE 2, phosphorylates and stabilizes ABI5 to mediate ABA signaling during seed germination (Hu and Yu, 2014). Furthermore, CTKs promote seed germination by limiting the effects of ABA and inducing ABI5 degradation (Y. Wang *et al.*, 2011; Guan *et al.*, 2014). In addition, SA inhibits germination by repressing the expression of GA-induced α-amylase genes under normal growth conditions (Xie *et al.*, 2007). Some key components of the SL signaling pathway also affect seed germination, including Suppressor of More Axillary Growth2 1 and OsD53 (Stanga *et al.*, 2013; Jiang *et al.*, 2013; Zhou *et al.*, 2013), which may initiate germination by decreasing the ABA:GA ratio (Toh *et al.*, 2012).

Several phytohormones most likely mediate seed germination by regulating ABA and/or GA biosynthesis and signaling, especially ABA signaling, and these include JA, ET, BRs, CTKs, SA, and SLs. However, whether other phytohormones interact with JA to mediate germination by modulating ABA biosynthesis and/or signaling remains to be investigated.

In summary, the existing evidence shows that JA-mediated germination is mainly modulated by ABA and GA, and that light signals also interact with the JA signaling through GA and ABA to affect germination. The biosynthesis of phytohormones is precisely regulated during seed germination. Light signals induce GA accumulation and inhibit ABA synthesis in the process of germination; GA effects the content of JA through the DELLA member RGL2; JA also regulates the expression of *ABA1* to enhance ABA accumulation; and in addition, ABA modulates JA synthesis through the bZIP72–AOC pathway (Fig. 2A). The light and hormone-mediated seed sensitivity is regulated by receptors (PHY, GID1, PYL, and COI1), signaling-repressor proteins (DELLA and JAZs), kinases (SnRKs), and transcription factors (PIF1, SOM, MFT, ABI5/3, and MYC2). JA-inhibited germination in seeds is regulated by alterations of ABA and GA synthesis and/or signaling. The synergistic action between JA and ABA in regulating seed germination is that JA activates ABA signaling via degrading JAZs, which act as a negative regulator of JA signaling and a repressor of ABI3 and ABI5, thereby delaying germination (Fig. 2B). JA also interacts with GA to affect ABA signaling mainly through the RGL2–ABI5–MFT pathway; light involves JA-mediated germination via the PIF1–SOM–MFT complex, influencing the ABA:GA ratio during seed germination.

Perspectives and conclusions

The contributions of JAs to plant defenses have been thoroughly characterized, but their effects on plant development are only gradually being clarified (Qi *et al.*, 2011, 2015; Jiang *et al.*, 2014; Wang *et al.*, 2017; Han *et al.*, 2018, 2020). Studies in recent decades have generated evidence of the regulatory effects of JA on seed germination (e.g. Dave *et al.*, 2011; Ju *et al.*, 2019; Pan *et al.*, 2020). However, the molecular basis of JA-mediated seed development and germination remains unclear. Although several studies have demonstrated that the interaction between JAs and ABA delays germination in Arabidopsis, wheat, and rice (Dave *et al.*, 2011; Ju *et al.*, 2019; Pan *et al.*, 2020), the precise mechanisms underlying the crosstalk between JA and ABA are still largely unknown. In addition, some questions regarding JA-regulated seed germination remain to be answered.

Seeds of peroxisomal ABC transporter mutants and β-oxidation mutants exhibit impaired seed germination (Table 1) and show strong correlations between germination

frequency and elevated levels of JA, JA-Ile, and OPDA; however, the underlying mechanisms are still unknown. Under different light conditions, the accumulation of OPDA or ABA is different according to phytochrome inactivation or activation (especially of phyA and phyB; Fig. 2), but it is not clear why two phytohormone-based repression pathways have evolved to control seed germination in light or darkness. And further studies are need to establish how oxylipin biosynthesis is regulated in seeds and how endogenous JA (especially OPDA) in particular acts as a signaling molecule to influence germination.

The current mechanistic model indicates that JA and ABA signals are integrated to regulate seed germination by the synergistic association between SCF^{COI1}/JAZ and ABI3/ABI5 (Varshney and Majee, 2021; Fig. 2). In the JA signaling pathway, MYC2 activity is repressed by the direct binding of JAZ proteins. Examination of the *myc2* mutant, which exhibits decreased ABA sensitivity, confirms that MYC2 interacts with PYL6 (Abe *et al.*, 2003; Aleman *et al.*, 2016; Fig. 2). Moreover, JA up-regulates the expression of the ABA receptor genes *PYL4* and *PYL5*, with knockout mutants of *pyl4* and *pyl5* being hypersensitive to JA (Lackman *et al.*, 2011). Therefore, it needs to be determined whether JAZ–MYC2–PYL controls seed germination. The JAZ proteins are generally ubiquitinated and degraded by SCF^{COI1} E3 ligase, and it has been shown that the E3 RING ligase KEG interacts with JAZ12 and regulates its stability (Pauwels *et al.*, 2015). The ABI5 transcription factor is a known target of KEG, and hence whether ABI5–KEG–JAZ helps regulate germination should be investigated.

There is considerable evidence that JA-mediated seed germination involves the ABA signaling pathway (Ju *et al.*, 2019; Pan *et al.*, 2020; H. Wang *et al.*, 2020; Varshney and Majee, 2021); however, the mechanisms underlying this interaction and crosstalk with other hormones have not been characterized. Diverse phytohormones, including GA, auxin, ET, and BRs, reportedly influence ABA signaling. For example, GA-signaling repressors (DELLA proteins) can modulate JA signaling via interactions with JAZ proteins (Hou *et al.*, 2010). Hence, the potential crosstalk between DELLA and ABI3/ABI5 might suppress seed germination (Lim *et al.*, 2013). The mechanisms by which other hormones regulate JA or by which JA mediates the biosynthesis and signaling pathways of other hormones during seed germination needs to be elucidated.

Research has confirmed that phytohormones are directly or indirectly associated with multiple plant processes, including those related to biotic and abiotic stress responses as well as plant physiology and development. The effects of plant hormones, especially JAs, on pathways associated with biotic and abiotic stress responses are linked by secondary signals, including Ca^{2+} and reactive oxygen species (ROS; Xia *et al.*, 2015; Jamra *et al.*, 2021; Jiménez *et al.*, 2021), and Ca^{2+} and ROS signaling also affect germination (Kwak *et al.*, 2003; Singh *et al.*, 2017; Pan *et al.*, 2021). Hence, the connections between JA and secondary signals needs to be determined in future studies.

Under FR light conditions, phyB is deactivated whilst OPDA and ABA accumulate, leading to repressed germination (Barros-Galvão *et al.*, 2019). Similarly, phyB-mediated light signaling is related to JA signaling in defense responses. The light-enhanced degradation of JAZ9 requires the receptor COI1 (Xiang *et al.*, 2021). In addition, under white light, the contents of OPDA and JA-Ile increase. Therefore, phyB might modulate the stability of JAZ proteins to regulate seed germination. However, the mechanism mediating the phyB-regulated synthesis of OPDA or JA-Ile during germination remains unknown.

Future breakthroughs regarding these regulatory mechanisms will further clarify the mediation of seed germination by JA and other phytohormones, and this will have the potential to lead to the development of methods for controlling the seed germination of agriculturally important crops.

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Author contributions

JP drafted and corrected the manuscript; all the authors edited and improved the manuscript, and contributed to specific sections.

Conflict of interest

The authors have no conflicts to declare in relation to this work.

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Data availability

All data supporting the findings of this study are available within the paper and within its supplementary materials published online.

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