Advances in ABA Regulation of Seed Development (Postprint)

Authors: Song Songquan, Tang Cuifang, Lei Huaping, Fei Sitian, Chen Haibo

Date: 2023-07-26T00:00:00+00:00

Abstract

Seed development is a complex biological process regulated by various genetic and environmental factors that significantly influences seed vigor, yield, and quality in crops, particularly cereal crops. Abscisic acid (ABA) is one of the most important plant hormones regulating seed development and germination, playing a pivotal role in controlling seed development—including embryogenesis and maturation—through its active levels, signal transduction, and the LAFL network. In recent years, substantial progress has been achieved in understanding ABA's role in regulating seed development. This review summarizes major research advances in this field, encompassing the regulation of seed development by ABA metabolism and signal transduction, interactions between ABA and seed maturation transcription factors (such as AFL-B3, FUS3, ABI3, and LEC2), and the underlying mechanisms of ABA action in seed development. Additionally, it identifies key scientific questions requiring further investigation, aiming to provide insights for deeper understanding of the molecular mechanisms governing seed development and to offer references for enhancing seed vigor and improving crop yield and quality.

Full Text

Preamble

Research Progress on Seed Development Regulated by ABA

SONG Songquan[1],[2]*, TANG Cuifang[1],[3], LEI Huaping[1], FEI Sitian[1], CHEN Haibo[1]

[1]Nanling Research Institute for Modern Seed Industry, Xiangnan University, Chenzhou 423099, Hunan, China
[2]Institute of Botany, Chinese Academy of Sciences, Beijing 100093, China
[3]College of Life Sciences, Hunan Normal University, Changsha 410081, China

Abstract: Seed development is a complex biological process controlled by various genetic and external factors, significantly affecting seed vigor, yield, and quality in crops, particularly cereals. Abscisic acid (ABA) is one of the most important phytohormones regulating seed development and germination, playing a key role in controlling seed development—including embryogenesis and maturation—through its active levels, signal transduction, and the LAFL network. In recent years, important progress has been made in understanding ABA's regulation of seed development. This review summarizes major achievements in this field, including the regulation of seed development by ABA metabolism and signal transduction, interactions between ABA and seed maturation transcription factors (AFL-B3, FUS3, ABI3, LEC2, etc.), and the mechanisms of ABA action in seed development. We also propose key scientific questions requiring further investigation, aiming to provide insights for understanding the molecular mechanisms of seed development and improving seed vigor, yield, and quality.

Key words: ABA metabolism, abscisic acid, transcription factor network, seed development, signal transduction

---

1.1 Changes in ABA Levels During Seed Development

In Arabidopsis, ABA levels in both siliques and seeds peak at mid-development (approximately 9 days after flowering) and subsequently decline. However, ABA levels in siliques begin increasing again at 12 days after flowering until late development (approximately 21 days after flowering) (Kanno et al., 2010; Kozaki & Aoyanagi, 2022). When zygotic tissues are deficient in ABA, ABA synthesized in maternal tissues can be transported to the embryo (Kanno et al., 2010). ABA synthesized in zygotic tissues primarily induces and/or maintains seed dormancy, while maternal ABA affects the thickness of the mucilage layer released upon imbibition in mature Arabidopsis seeds (Kanno et al., 2010).

In wheat (*Triticum aestivum*), ABA levels exhibit two peaks during seed development, with ABA synthesized during late development (approximately 35–40 days after pollination) correlating with seed dormancy levels (Tuan et al., 2018). In contrast, rice (*Oryza sativa*) and triticale show only a single ABA peak. In rice, ABA accumulation related to dormancy induction occurs during early and mid-seed development (10–20 days after pollination), earlier than in wheat (Gu et al., 2011; Liu et al., 2014). In triticale, the ABA accumulation peak occurs around 35 days after pollination, before substantial seed water loss (Fidler et al., 2016).

1.2 Regulation of Seed Development by ABA Metabolism

Active ABA is synthesized via an indirect pathway from xanthophylls (e.g., zeaxanthin, violaxanthin, and neoxanthin) (Marion-Poll & Leung, 2006). Three key

enzymes catalyze consecutive steps in ABA biosynthesis: zeaxanthin epoxidase (ZEP), 9-cis-epoxycarotenoid dioxygenase (NCED), and abscisic aldehyde oxidase (AAAO) (Dejonghe et al., 2018).

ZEP genes were first identified in Arabidopsis and *Nicotiana plumbaginifolia*. Their ABA-deficient mutants (*aba1/aba2*) are impaired in the oxidation of zeaxanthin to antheraxanthin and violaxanthin, considered the initial step of ABA biosynthesis (Sano & Marion-Poll, 2021). In rice, a *Tos17* viviparous mutant was identified as having precocious germination due to defective zeaxanthin oxidation during ABA synthesis (Ali et al., 2022). Additional ABA auxotrophic mutants identified in maize (*Zea mays*) through genetic screens (*vp2*, *vp5*, *vp7*, and *vp9*) exhibit defective ZEP activity, also blocking early steps in carotenoid biosynthesis (Ali et al., 2022). These results demonstrate that zeaxanthin oxidation is an important and conserved stage of ABA synthesis in plants. The conversion from all-trans-violaxanthin and all-trans-neoxanthin to 9-cis-violaxanthin and 9-cis-neoxanthin remains unclear. However, ABA4 was found to be responsible for converting all-trans-violaxanthin to all-trans-neoxanthin (North et al., 2007), providing clues for investigating these conversions.

The second key gene in ABA biosynthesis, NCED, was initially cloned from the maize viviparous mutant *VP14* (*VIVIPAROUS 14*). The *VP14* mutant exhibits oxidative defects in 9-cis-epoxycarotenoid cleavage during ABA biosynthesis and shows reduced ABA content in dry seeds (Tan et al., 1997). In Arabidopsis, NCED2, NCED3, NCED5, NCED6, and NCED9 are considered homologs of VP14 and participate in the rate-limiting step of ABA biosynthesis (Nambara & Marion-Poll, 2005). Additionally, PvNCED1 from soybean (*Glycine max*), LeNCED1 from tomato, and BdNCED1 from *Brachypodium distachyon* also play important roles in ABA biosynthesis and seed development (Barrero et al., 2012). These results indicate that oxidative cleavage of xanthophylls is a major step in ABA biosynthesis that regulates seed development.

The oxidation of abscisic aldehyde is the final step of ABA biosynthesis, converting abscisic aldehyde to ABA (Dejonghe et al., 2018). Initially, defective mutants in the oxidation of abscisic aldehyde to ABA were identified in tomato as *flacca* and *sitiens* (Taylor et al., 1988). Subsequently, abscisic aldehyde oxidase 3 (AAO3) identified in Arabidopsis functions in the final two steps of ABA biosynthesis in seeds, and its expression was observed in embryonic vascular tissue during mid-to-late seed maturation (Seo et al., 2004).

1.3 Regulation of Seed Development by ABA Signal Transduction

The core ABA signal transduction components include the ABA receptor PYR/PYL/RCAR (pyrabactin resistance 1/pyrabactin resistance 1-like/regulatory components of ABA receptor) family, group A type 2C protein phosphatases (PP2Cs), and sucrose non-fermenting-1-related protein kinase 2 (SnRK2) (Nonogaki, 2019a, b; Lim et al., 2022) (Figure 1 [Figure 1: see original

paper]).

PP2Cs are encoded by *ABI1* and *ABI2* genes. The key elements of DOG1 signaling are heme molecules and PP2Cs encoded by *AHG1* and *AHG3* genes. Triplex complexes of PYR/PYL/RCAR-ABA-PP2C and/or DOG1-HEME-PP2C block the binding of PP2C to SnRK2. Activated SnRK2 phosphorylates ABI3 and ABI5, which bind to promoters (Pro) of ABA-controlled genes. In seeds, parallel ABA and DOG1 signaling pathways activate synthesis of raffinose family oligosaccharides and expression of LEA and HSP genes, thereby regulating the onset of desiccation tolerance and transition to dormancy.

In Arabidopsis, 14 members of the PYR/PYL/RCAR protein family have been shown to be important in seeds. For example, the *pyr1/pyl1/pyl2/pyl4* quadruple mutant and *pyl* duodecuple mutant exhibit reduced seed dormancy and ABA insensitivity (Ma et al., 2009; Zhao et al., 2018). Additionally, the *ospyl* septuple mutant in rice is ABA-insensitive during seed germination (Miao et al., 2018).

In the absence of ABA, PYL proteins release PP2Cs and activate their phosphatase activity (Ma et al., 2009). PP2C proteins, including ABA-INSENSITIVE 1/2 (ABI1/2) and ABA-HYPERSENSITIVE GERMINATION 1/3 (AHG1/3), inhibit the activity of downstream ABA signaling proteins through protein dephosphorylation, thereby blocking downstream ABA signal transduction networks (Park et al., 2009). Consequently, PP2Cs act as negative regulators in the ABA signaling system, and their knockout mutants show ABA hypersensitivity and reduced seed dormancy (Yoshida et al., 2006). Studies have shown that ENHANCER OF ABA CO-RECEPTOR 1 (EAR1) can increase PP2C activity by acting together with PP2C proteins (ABI1/2, HAB1/2 (Hypersensitive to ABA 1/2), and AHG1/3) (Wang et al., 2018). Like EAR1, PR5 receptor-like kinase 2 (PR5K2) inhibits ABA signaling by increasing phosphorylation of ABI1/2 (Baek et al., 2019). Furthermore, DOG1 (DELAY OF GERMINATION 1) binds heme and interacts with AHG1 to block its phosphatase activity and increase seed dormancy (Nishimura et al., 2018). These studies demonstrate that PP2Cs can be regulated by PYL receptors or other proteins, though the interrelationships among PP2Cs, PYL proteins, and other regulatory factors (DOG1, PR5K2, and EAR1) during seed development remain unclear.

In the presence of ABA, PYR/PYL/RCAR proteins bind ABA and PP2C proteins to inhibit PP2C phosphatase activity, thereby releasing and activating SnRK2. Studies have shown that all Arabidopsis PYL family members can interact with PP2C family members and function in ABA-mediated responses (Zhao et al., 2013). In Arabidopsis, three SnRK2 proteins (SnRK2.2, SnRK2.3, and SnRK2.6) have been identified as positive regulators of the ABA signaling network, participating in many seed development processes such as de-greening, accumulation of seed storage products, acquisition of desiccation tolerance, and germination (Finkelstein et al., 2008). ABA signaling terminator (ABT), a WD40 protein, was found to effectively block ABA signaling and plays an im-

portant role in seed germination and seedling establishment. ABT is induced by ABA in a PYR1/PYL/RCAR-PP2C-dependent manner and interacts with PYR1/PYL/RCAR and PP2C proteins, thereby interfering with the interaction between PYR1/4 and ABI1/2 and blocking ABA signal transduction (Wang et al., 2020).

The primary targets of SnRK2 are ABFs (ABRE (ABA RESPONSIVE ELEMENT) binding factors). The ABF family consists of nine members, including ABF1, ABF2/AREB1 (ABRE BINDING PROTEIN 1), ABF3, ABF4/AREB2, AREB3, ABI5, bZIP15, bZIP67, and the bZIP subfamily EEL, which mainly participate in ABA-mediated transcriptional regulation (Nakashima et al., 2009). ABI5 transcription can be activated by SnRK2 through specific binding to ABRE cis-elements in the ABI5 promoter, thereby activating ABA-mediated transcriptional activity in late-maturation Arabidopsis seeds and imbibed seeds. Additionally, another key factor, ABI3, interacts with the ABI5 transcription factor and cooperates with ABI5 to promote transcription of downstream ABA-responsive genes. Both genes can be regulated by RAV1 (RELATED TO ABI3/VP1) through binding to their promoters (Ali et al., 2022). Interestingly, ABI5 also directly regulates transcription during germination by binding to promoters of *PYL11* and *PYL12* to modulate ABA responses. When ABI5 is mutated, the ABA-hypersensitive responses caused by overexpression of PYL11 and PYL12 are completely or partially impaired (Zhao et al., 2020).

2 ABA and Seed Maturation Transcription Factors

Genetic screening has revealed that LAFL genes play important roles in ABA-mediated seed development. The LAFL genes include AFL-B3 (AFL clade of B3 domain plant-specific transcription factor), FUS3 (FUSCA3), ABI3, LEC2 (LEAFY COTYLEDON 2), and the HAP3 subunit of CBF (CCAAT-binding transcription factor) or NF-Y (nuclear factor Y), LEC1, and L1L (LEC1-LIKE) (Smolikova et al., 2021; Kozaki & Aoyanagi, 2022) (Figure 2 [Figure 2: see original paper]). Mutations in LAFL genes affect many aspects of seed development, such as decreased storage reserves at maturity, reduced desiccation tolerance and ABA levels, and decreased dormancy (Holdsworth et al., 2008; Jia et al., 2014). In addition to seed development, the LAFL network also regulates genes involved in plant development, such as the zinc finger factor PEI1, AP2 (APETALA2) family factor BBM (BABY BOOM), NAC factor CUC1 (CUP-SHAPED COTYLEDON 1), and MADS box factor FLC (FLOWERING LOCUS C) (Jia et al., 2014).

Arrows and blunted lines indicate activation and repression, respectively. The black line between ABI3 and ABI5 indicates protein interaction. LEC1, LEC2, and FUS3 (surrounded by the thick black line) are involved in acquisition of desiccation tolerance, and all LAFL proteins participate in dormancy regulation. LEC1 is related to GA signaling, while ABI3 and ABI5 are related to ABA signaling. Chl, chlorophyll.

AFL factors activate target genes through RY cis-elements recognized by the B3-DNA binding domain (Braybrook et al., 2006). LEC1 and L1L, as subunits of the NF-Y complex, bind to CCAAT DNA motifs (Miller, 2016). Genome-wide analysis of LEC1 binding sites in upstream regions of Arabidopsis and soybean target genes revealed that, in addition to CCAAT motifs, G-box, ABRE (ABA-responsive promoter element)-like, RY, and BPC1 cis-elements are enriched in promoters of genes regulated during seed maturation, indicating that LEC1 regulates target genes by interacting with other transcription factors (Jo et al., 2019).

Genetic analyses have revealed a complex interaction network among LAFL genes (Figure 2). LEC1 can activate expression of ABI3, FUS3, and LEC2, while ectopic expression of LEC2 can upregulate LEC1, ABI3, and FUS3 (To et al., 2006; Stone et al., 2008). ABI3 and FUS3 positively regulate each other and control their own expression (To et al., 2006; Mönke et al., 2012). Additionally, L1L is regulated by FUS3 (Yamamoto et al., 2010). Chromatin immunoprecipitation (ChIP) analysis showed that LEC1 regulates L1L (Junker et al., 2012), while FUS3 regulates LEC1, FUS3, and ABI3 (Wang & Perry, 2013).

In addition to LAFL genes, ABI5 and related bZIP transcription factors also bind to ABREs and participate in regulating seed maturation. ABI5 is a key player in ABA signal transduction (Collin et al., 2021). An important group of genes regulated by LAFL during seed maturation includes LATE EMBRYOGENESIS ABUNDANT (LEA) genes, whose promoters contain RY and ABRE motifs and are combinatorially regulated by ABI3 and ABI5-related bZIP transcription factors (Alonso et al., 2009). Thus, ABA signal transduction is integrated into the LAFL network through ABI5 and related bZIP factors that physically interact with the N-terminal COAR (co-activator/co-repressor) domain of ABI3 (Alonso et al., 2009). ABREs have also been found in promoters of other LAFL target genes, suggesting that other LAFL components may be co-regulated by ABA (Junker et al., 2012; Wang & Perry, 2013).

In Arabidopsis, exogenous ABA increases FUS3 expression (Kagaya et al., 2005), and FUS3 induces ABA accumulation (Gazzarrini et al., 2004). Therefore, FUS3 and ABA are mutually positive regulators (Braybrook & Harada, 2008). Additionally, FUS3 expression can be positively regulated by auxin (Gazzarrini et al., 2004).

3.1 Storage Reserve Accumulation

During seed maturation, accumulation of storage compounds such as seed storage proteins (SSPs), lipids, and carbohydrates is closely associated with ABA levels and signaling (Finkelstein, 2013) (Table 1). Mutations in ABA signaling components such as PYL and SnRK2 typically result in reduced seed storage reserves (Nakashima et al., 2009; Zheng et al., 2010; Zhao et al., 2018). Inactivation of SnRK2.6 leads to decreased seed oil content, while overexpression of SnRK2.6 increases overall seed weight (Zheng et al., 2010). The SnRK2 triple

mutant (*snrk2.2/3/6*) and *pyl* duodecuple mutant generally show reduced seed storage products, such as 12S globulins (Nakashima et al., 2009; Zhao et al., 2018). Starch biosynthesis in maize and rice is synergistically regulated by sucrose and ABA (Huang et al., 2016; Chen et al., 2019).

Table 1 Functions of ABA biosynthesis, signaling, and transcription factors involved in maturation in seed development

Genes/transcription factor	Function description	Reference
SnRK2.6	Inactivation reduces seed oil content, while overexpression increases overall seed weight	Zheng et al., 2010
PYL, SnRK2	Mutations often exhibit reduced seed storage products, such as 12S globulin	Zheng et al., 2010; Zhao et al., 2018
LEC1	Activates cruciferin C via direct interaction with bZIP67	Yamamoto et al., 2010
LEC2	Regulates oil and protein accumulation by activating expression of OLE1 (encoding oleosin) and genes encoding 2S and 12S storage proteins	Braybrook et al., 2006
FUS3	Regulates TTG1 and WRI1 expression negatively and positively, respectively	Mu et al., 2008; Chen et al., 2015
LEC1 and FUS3	Control ABI3 accumulation in an ABA-dependent manner and interact to regulate storage protein accumulation, anthocyanin synthesis, and chlorophyll and lipid accumulation	Mu et al., 2008; Zhang et al., 2016

Genes/transcription factor	Function description	Reference
FUS3 and LEC2	Induce WRI1 expression; WRI1 encodes an AP2 transcription factor that regulates sugar and oil content by increasing expression of fatty acid synthesis and sugar degradation genes	Yamamoto et al., 2010
TTG1	Encodes a transcription factor that suppresses SSP and oil accumulation; *ttg1* mutants show dramatic increases in storage reserves	Baud et al., 2008; Chen et al., 2015

LAFL genes are involved in regulating storage reserve accumulation. LEC1 and FUS3 control ABI3 accumulation during maturation in an ABA-dependent manner and interact to regulate accumulation of storage proteins (including Arabidopsis storage protein 3 (At2S3) and cruciferin C (CRC)), anthocyanin synthesis, and chlorophyll and lipid accumulation (Mu et al., 2008; Zhang et al., 2016). LEC1 also activates CRC through direct interaction with bZIP67 (Yamamoto et al., 2010).

FUS3 negatively regulates expression of TTG1 (TRANSPARENT TESTA GLABRA1), which encodes a transcription factor that suppresses SSP and oil accumulation in Arabidopsis (Chen et al., 2015). The *ttg1* mutant is characterized by dramatically increased storage reserves, including SSPs and oils (Baud et al., 2008). FUS3 likely promotes storage reserve accumulation by suppressing TTG1 (Chen et al., 2015). FUS3 also induces WRI1 (WRINKLED 1) expression in combination with LEC2. WRI1 encodes an AP2 transcription factor that regulates sugar and oil content in seeds by increasing expression of fatty acid synthesis and sugar degradation genes (Yamamoto et al., 2010). FUS3 promotes storage oil accumulation through both suppressing TTG1 expression and increasing WRI1 expression. This storage oil accumulation is also regulated by LEC1 and AFL genes through activation of WRI1 (Mu et al., 2008). Additionally, LEC2 regulates oil and protein accumulation by activating expression of the oleosin gene *OLE1* and genes encoding 2S and 12S storage proteins (Braybrook et al., 2006).

During seed maturation, factors other than LAFL genes also participate in storage reserve accumulation. bZIP67, together with L1L and NF-YC2 (NUCLEAR FACTOR-YC2), regulates FAD3 (FATTY ACID DESATURASE 3), which functions in ω-3 fatty acid storage during seed maturation (Mendes et al., 2013).

Expression of the DOGL4 (DOG1-LIKE4) gene, which is induced by ABA, regulates expression of some seed storage proteins during seed maturation, including CRC, albumins, and oleosins (Sall et al., 2019).

3.2 Acquisition of Desiccation Tolerance

Seed desiccation tolerance is an adaptive mechanism evolved in plants to ensure species survival and reproduction, playing a key role in crop seed preservation and long-term conservation of plant germplasm resources (Smolikova et al., 2021; Song et al., 2022). The desiccation tolerance mechanism is activated during late seed maturation and is associated with accumulation of LEA proteins, small heat shock proteins (sHSPs), non-reducing oligosaccharides, and antioxidants of various chemical properties (Smolikova et al., 2021; Song et al., 2022). The major regulators of maturation and desiccation tolerance are ABA and DOG1 protein, which control transcription factor networks including LEC1, LEC2, FUS3, ABI3, ABI5, AGL67, PLATZ1, and PLATZ2 (Smolikova et al., 2021) (Figure 2).

LEA gene expression is regulated by ABI3 and ABI5 (Bies-Ethève et al., 2008). ABI3 also regulates expression of the seed-specific heat shock factor HSFA9 (Kotak et al., 2007). Expression of LEA and HSP genes is increased by DOG1 through ABI5/ABI3, and their expression enhances storage of nitrogen-containing compounds in seeds, thereby promoting seed dormancy and improving seed vigor (Dekkers et al., 2016). Some studies have shown that DOG1 expression is negatively or positively controlled by bZIP67 and ERF12 (ETHYLENE RESPONSE FACTOR 12), respectively, during seed maturation (Bryant et al., 2019; Li et al., 2019). In *Medicago truncatula* and pea, ABI3, ABI4, and ABI5 are considered major hubs regulating acquisition of seed desiccation tolerance by controlling genes involved in raffinose family oligosaccharide (RFO) metabolism and LEA protein synthesis (Zinsmeister et al., 2016) (Table 1).

Mutations in LEC1, ABI3, or FUS3 significantly affect seed desiccation tolerance, indicating that these three transcription factors are all required for activating desiccation tolerance (Roscoe et al., 2015). LEC2 participates in establishing desiccation tolerance by affecting expression of LEA, EM1 (early methionine 1), and EM6 genes through inducing expression of the ENHANCED EM LEVEL (EEL) bZIP transcription factor (Bentsink et al., 2006). The EEL bZIP transcription factor is a negative regulator of EM proteins in Arabidopsis (Braybrook et al., 2006).

3.3 Induction and Maintenance of Primary Seed Dormancy

Dormancy is a temporary quiescent state that is an important characteristic of wild plant seeds to avoid germination under unfavorable environmental conditions and ensure offspring survival. For cultivated crops, seeds with rapid and uniform germination are selected to achieve high yield and quality. Conversely,

lack of seed dormancy, particularly harvest dormancy, is an undesirable agronomic trait as it can lead to pre-harvest sprouting (PHS), a serious problem in cereal cultivation, and non-dormant mutants may have reduced seed longevity (Finkelstein et al., 2008; Tuan et al., 2018). Seeds begin dehydration after storage reserve synthesis and at the end of maturation, storing newly synthesized ABA and entering dormancy. Evidence indicates that ABA is a key regulator of these processes (Finkelstein et al., 2008; Nambara et al., 2010). Mutations in ABA biosynthesis, perception, and signal transduction affect seed dormancy (Nakashima et al., 2009; Zhao et al., 2018) (Table 1).

Mature dry seeds of Arabidopsis *AtNCED6* and *AtNCED9* mutants show decreased ABA levels and dormancy (Lefebvre et al., 2006). Other ABA-deficient mutants, such as *aba1* and *aba2/3*, also display reduced dormancy levels (Kozaki & Aoyanagi, 2022). Arabidopsis ODR1 (suppressor of RDO5 (REDUCED DORMANCY 5)) acts together with bHLH57 and functions upstream of NCED6 and NCED9 to control ABA synthesis and seed dormancy (Liu et al., 2020). Ectopic expression and overexpression of soybean PvNCED1 in imbibed tobacco (*Nicotiana tabacum*) seeds increase ABA levels and delay seed germination. In tomato, LeNCED1 overexpression also increases dormancy by elevating ABA levels in seeds (Ali et al., 2022). In wheat, mutations in two *TaABA8'OH1* homologs (*TaABA8'OH1A* and *TaABA8'-OH1D*; homologs of *AtCYP707*) result in increased ABA content and dormancy (Chono et al., 2013). TsNCED1 is also associated with higher ABA content and increased PHS resistance (Fidler et al., 2016). Mutations in ABA signaling components, such as the rice *ospyl* septuple mutant and *snrk2.2/3/6* triple mutant, also cause precocious germination in rice and Arabidopsis seeds (Nakashima et al., 2009; Miao et al., 2018).

In Arabidopsis, AtMYB96 directly activates ABA synthesis genes (*NCED2*, *NCED5*, *NCED6*, and *NCED9*) and inactivates GA biosynthesis genes (*AtGA3ox1* and *AtGA20ox1*) to induce primary seed dormancy (Lee et al., 2015). AtABI4 increases seed dormancy through direct interaction with promoter regions of *AtNECD6* to enhance ABA biosynthesis and with promoter regions of the GA inactivation gene *AtGA2ox7* to inhibit GA accumulation (Shu et al., 2013, 2016).

LAFL gene members also participate in dormancy acquisition. Embryonic growth arrest in mature seeds is controlled by FUS3, LEC1, and LEC2, whose mutants cannot completely stop embryonic growth and exhibit precocious germination (Gubler et al., 2005). The maize *VP1* gene, a homolog of Arabidopsis *ABI3*, was among the first key components of ABA signaling to be identified and characterized. *VP1* mutation causes PHS and disrupts embryo maturation in maize. *VP1* genes in wheat, rice, and sorghum (*Sorghum bicolor*) are also associated with dormancy levels and sensitivity to ABA and PHS (Kozaki & Aoyanagi, 2022). In maize, LAFL gene members are regulated by VP8, which encodes a putative peptidase (Suzuki et al., 2008). Mutations in the rice VP8 homolog PLA3 (PLATOCHRON 3/GO (GOLIATH)) and Arabidopsis AMP1 (ALTERED MERISTEM PROGRAM 1) show reduced dormancy (Griffiths et

al., 2011). ABI5 is also important for dormancy induction during seed maturation in wheat and pea (Zinsmeister et al., 2016; Yamasaki et al., 2017; Utsugi et al., 2020). In sorghum, SbABI4 and SbABI1 enhance transcription of *SbGA2ox3* by directly binding to its promoter, thereby prolonging seed dormancy (Cantoro et al., 2013).

Two major dormancy genes, *DOG1* and *RDO5*, have been identified that appear to function independently of plant hormones including ABA (Bentsink et al., 2006; Xiang et al., 2014; Carrillo-Barral et al., 2020). RDO5 is a member of the PP2C protein phosphatase family but does not exhibit phosphatase activity (Xiang et al., 2014), whereas DOG1 is a protein of unknown function (Carrillo-Barral et al., 2020). Mutations in *DOG1* and *RDO5* completely abolish or reduce seed dormancy, respectively (Bentsink et al., 2006; Xiang et al., 2014). Genetic analyses indicate that both DOG1 and ABA are required for normal seed dormancy (Bentsink et al., 2006; Nakabayashi et al., 2012).

DOG1 interacts with four phosphatases, two of which belong to clade A type 2C protein phosphatases, AHG1 and AHG3 (Figure 1). The ABA and DOG1 pathways converge at the PP2C phosphatase level: DOG1 inhibits AHG1 and AHG3, while ABA inhibits other PP2C phosphatases and AHG3. By inhibiting PP2C phosphatases, both ABA and DOG1 promote and maintain seed dormancy (Antoni et al., 2012; Née et al., 2017). DOG1 is also required for many processes of seed maturation, partially by interfering with ABA signaling components (Dekkers et al., 2016).

OsSDR4 (SEED DORMANCY 4) is considered a regulator associated with seed dormancy in rice with unknown function (Sugimoto et al., 2010). In Arabidopsis, AtSDR4L (SDR4-LIKE) regulates dormancy release and germination by modulating DOG1 and RGA-LIKE2 (encoding DELLA protein RGL2) in the GA pathway (Cao et al., 2019). Studies suggest that AtODR1 (reversal of rdo5), an ortholog of OsSDR4, acts together with bHLH57 and functions upstream of AtNCED6 and AtNCED9 to control ABA synthesis and seed dormancy in Arabidopsis (Liu et al., 2020).

3.4 Seed De-greening

During seed maturation, SnRK2 and ABI3 genes have been identified as important components of the de-greening process (Delmas et al., 2013). The *snrk2.2/snrk2.3/snrk2.6* triple mutant shows ABA insensitivity during seed development and produces green seeds (Nakashima et al., 2009; Zhao et al., 2018). The Arabidopsis *abi3–6* mutant exhibits a lack of de-greening, and ABI3 was found to control embryo de-greening by regulating expression of SGR (STAY GREEN, AtSGR1 and AtSGR) genes, which are homologs of the SGR gene encoded by Mendel's I locus (Armstead et al., 2007; Delmas et al., 2013). ABI5 also regulates de-greening and seed longevity in legume seeds (Verdier et al., 2013; Zinsmeister et al., 2016).

4 Concluding Remarks

Seed development is a complex process comprising embryogenesis and maturation stages, characterized primarily by storage reserve accumulation, acquisition of desiccation tolerance, growth arrest, and dormancy acquisition, which significantly affect seed vigor, yield, and quality (Kozaki & Aoyanagi, 2022). Plant hormone ABA regulates seed development mainly through ABA metabolism, signal transduction, and its LAFL network (Sano & Marion-Poll, 2021; Ali et al., 2022). Although important progress has been made in recent years regarding ABA regulation of seed development, several key scientific questions remain unclear. For example, endogenous ABA levels are regulated through carotenoid pathway synthesis and inactivated through 8'-hydroxylation. ABA glucosyltransferase can convert ABA into ABA-glucose ester (ABA-GE) as an ABA storage pool, and ABA-GE can be hydrolyzed by β-glucosidase into ABA and glucose (Sano & Marion-Poll, 2021). How these enzymes and their genes respond to developmental or environmental changes to maintain normal ABA levels required for seed development remains unclear.

The major regulators of seed maturation and desiccation tolerance are ABA and DOG1 protein, which control transcription factor networks such as LEC1, LEC2, FUS3, ABI3, ABI5, AGL67, PLATZ1, and PLATZ2 (Smolikova et al., 2021). The core ABA pathway and DOG1 pathway converge at PP2C (Figure 1). Notably, it remains unclear which pathway PP2C preferentially responds to when integrating developmental or environmental signals and how these two pathways coordinate. Although DOG1 is one of the main regulators of seed dormancy, its molecular function has not yet been determined (Nonogaki, 2019a; Sano & Marion-Poll, 2021).

During seed maturation, GA levels are downregulated. GA levels are regulated by FUS3 and LEC2, which inhibit enzymes related to bioactive GA synthesis (Kozaki & Aoyanagi, 2022). In Arabidopsis, GA signaling promotes embryo development by activating LEC1 (Figure 2) to increase late embryogenesis auxin accumulation. The GA signaling repressor DELLA interacts with LEC1 to promote YUCCA (YUC) gene expression and embryo development through increased auxin accumulation. GA triggers DELLA degradation to release its inhibition of LEC1, leading to activation of genes necessary for embryogenesis (Hu et al., 2018). However, the detailed function of GA in embryogenesis remains unclear (Kozaki & Aoyanagi, 2022). Auxin promotes ABI3 expression, which induces embryonic identity genes by activating ARF (AUXIN RESPONSE FACTOR) genes (Kozaki & Aoyanagi, 2022). Similarly, auxin stimulates ABA signaling to control seed dormancy by inducing ABI3 expression (Liu et al., 2013). Therefore, the regulation of seed development by other plant hormones and their interactions with ABA warrants further investigation.

Currently, many transcription factors involved in seed maturation have been identified and characterized at the molecular and genetic levels, but transcriptional regulation of early embryogenesis remains poorly studied. Moreover, tran-

scription factor activity is strictly controlled by genetic and epigenetic factors, yet our understanding of these factors is incomplete (Verma et al., 2022). In-depth investigation of these issues will help elucidate the molecular mechanisms of seed development, providing new knowledge and technologies for improving seed vigor and increasing yield and quality.

References

ALI F, QANMBER G, LI F, et al., 2022. Updated role of ABA in seed maturation, dormancy, and germination [J]. J Adv Res, 35: 199–214.

ALONSO R, OÑATR-SÁNCHEZ L, WELTMEIER F, et al., 2009. A pivotal role of the basic leucine zipper transcription factor bZIP53 in the regulation of Arabidopsis seed maturation gene expression based on heterodimerization and protein complex formation [J]. Plant Cell, 21(6): 1747–1761.

ANTONI R, GONZALEZ-GUZMAN M, RODRIGUEZ L, et al., 2012. Selective inhibition of clade A phosphatases type 2C by PYR/PYL/RCAR abscisic acid receptors [J]. Plant Physiol, 158(2): 970–980.

ARMSTEAD I, DONNISON I, AUBRY S, et al., 2007. Cross-species identification of Mendel's I locus [J]. Science, 315(5808): 73.

BAEK D, KIM MC, KUMAR D, et al., 2019. AtPR5K2, a PR5-like receptor kinase, modulates plant responses to drought stress by phosphorylating protein phosphatase 2Cs [J]. Front Plant Sci, 10: 1146.

BAROUX C, GROSSNIKLAUS U, 2019. Seeds – An evolutionary innovation underlying reproductive success in flowering plants [M]. Curr Top Dev Biol, 131: 605–642.

BARRERO JM, JACOBSEN JV, TALBOT MJ, et al., 2012. Grain dormancy and light quality effects on germination in the model grass *Brachypodium distachyon* [J]. New Phytol, 193(2): 705–716.

BATISTA RA, MORENO-ROMERO J, QIU Y, et al., 2019. The MADS-box transcription factor PHERES1 controls imprinting in the endosperm by binding to domesticated transposons [J]. eLife, 8: e50541.

BAUD S, DUBREUCQ B, MIQUEL M, et al., 2008. Storage reserve accumulation in Arabidopsis: Metabolic and developmental control of seed filling [J]. Arab Book Am Soc Plant Biol, 6: e0113.

BENTSINK L, JOWETT J, HANHART CJ, et al., 2006. Cloning of DOG1, a quantitative trait locus controlling seed dormancy in Arabidopsis [J]. Proc Natl Acad Sci USA, 103(45): 17042–17047.

BEWLEY JD, BRADFORD KJ, HILHORST HWM, et al., 2013. Seed: Physiology of Development, Germination and Dormancy [M]. 3rd edition. New York: Springer.

BIES-ETHÈVE N, GAUBIER-COMELLA P, DEBURES A, et al., 2008. Inventory, evolution and expression profiling diversity of the LEA (late embryogenesis abundant) protein gene family in *Arabidopsis thaliana* [J]. Plant Mol Biol, 67(1–2): 107–124.

BRAYBROOK SA, HARADA JJ, 2008. LECs go crazy in embryo development [J]. Trends Plant Sci, 13(12): 624–630.

BRAYBROOK SA, STONE SL, PARK S, et al., 2006. Genes directly regulated by Leafy Cotyledon 2 provide insight into the control of embryo maturation and somatic embryogenesis [J]. Proc Natl Acad Sci USA, 103(9): 3468–3473.

BRYANT FM, HUGHES D, HASSANI-PAK K, et al., 2019. Basic LEUCINE ZIPPER TRANSCRIPTION FACTOR 67 transactivates DELAY OF GERMINATION1 to establish primary seed dormancy in Arabidopsis [J]. Plant Cell, 31(6): 1276–1288.

CANTORO R, CROCCO CD, BENECH-ARNOLD RL, et al., 2013. In vitro binding of *Sorghum bicolor* transcription factors ABI4 and ABI5 to a conserved region of a GA 2-OXIDASE promoter: Possible role of this interaction in the expression of seed dormancy [J]. J Exp Bot, 64(18): 5721–5735.

CAO H, HAN Y, LI J, et al., 2019. *Arabidopsis thaliana* SEED DORMANCY 4-LIKE regulates dormancy and germination by mediating the gibberellin pathway [J]. J Exp Bot, 71(3): 884–896.

CARRILLO-BARRAL N, DEL CARMEN RODRÍGUEZ-GACIO M, MATILLA AJ, 2020. Delay of germination 1 (DOG1): A key to understanding seed dormancy [J]. Plants, 9(4): 475.

CHEN T, LI G, ISLAM MR, et al., 2019. Abscisic acid synergizes with sucrose to enhance grain yield and quality of rice by improving the source-sink relationship [J]. BMC Plant Biol, 19(1): 388.

CHEN S, WANG X, ZHANG L, et al., 2016. Identification and characterization of tomato gibberellin 2-oxidases (GA2oxs) and effects of fruit-specific SlGA2ox1 overexpression on fruit and seed growth and development [J]. Hortic Res, 3: 16059.

CHEN M, ZHANG B, LI C, et al., 2015. TRANSPARENT TESTA GLABRA 1 regulates the accumulation of seed storage reserves in Arabidopsis [J]. Plant Physiol, 169(1): 391–402.

CHONO M, MATSUNAKA H, SEKI M, et al., 2013. Isolation of a wheat (*Triticum aestivum* L.) mutant in ABA 8'-hydroxylase gene: Effect of reduced ABA catabolism on germination inhibition under field condition [J]. Breed Sci, 63(1): 104–115.

COLLIN A, DASZKOWSKA-GOLEC A, SZAREJKO I, 2021. Updates on the role of ABSCISIC ACID INSENSITIVE 5 (ABI5) and ABSCISIC ACID-RESPONSIVE ELEMENT BINDING FACTORs (ABFs) in ABA signaling in

different developmental stages in plants [J]. Cells, 10(8): 1996.

DEJONGHE W, OKAMOTO M, CUTLER SR, 2018. Small molecule probes of ABA biosynthesis and signaling [J]. Plant Cell Physiol, 59(8): 1490–1499.

DEKKERS BJ, HE H, HANSON J, et al., 2016. The Arabidopsis DELAY OF GERMINATION 1 gene affects ABSCISIC ACID INSENSITIVE 5 (ABI5) expression and genetically interacts with ABI3 during Arabidopsis seed development [J]. Plant J, 85(4): 451–465.

DELMAS F, SANKARANARAYANAN S, DEB S, et al., 2013. ABI3 controls embryo degreening through Mendel's I locus [J]. Proc Natl Acad Sci USA, 110(40): E3888–E3894.

FIDLER J, ZDUNEK-ZASTOCKA E, PRABUCKA B, et al., 2016. Abscisic acid content and the expression of genes related to its metabolism during maturation of triticale grains of cultivars differing in pre-harvest sprouting susceptibility [J]. J Plant Physiol, 207: 1–9.

FINKELSTEIN R, 2013. Abscisic acid synthesis and response [J]. Arab Book, 11: e0166.

FINKELSTEIN R, REEVES W, ARIIZUMI T, et al., 2008. Molecular aspects of seed dormancy [J]. Annu Rev Plant Biol, 59: 387–415.

GAZZARRINI S, TSUCHIYA Y, LUMBA S, et al., 2004. The transcription factor FUSCA3 controls developmental timing in Arabidopsis through the hormones gibberellin and abscisic acid [J]. Dev Cell, 7(3): 373–385.

GRIFFITHS J, BARRERO JM, TAYLOR J, et al., 2011. ALTERED MERISTEM PROGRAM 1 is involved in development of seed dormancy in Arabidopsis [J]. PLoS ONE, 6: e20408.

GU XY, FOLEY ME, HORVATH DP, et al, 2011. Association between seed dormancy and pericarp color is controlled by a pleiotropic gene that regulates abscisic acid and flavonoid synthesis in weedy red rice [J]. Genetics, 189(4): 1515–1524.

GUBLER F, MILLAR AA, JACOBSEN JV, 2005. Dormancy release, ABA and pre-harvest sprouting [J]. Curr Opin Plant Biol, 8(2): 183–187.

HOLDSWORTH MJ, BENTSINK L, SOPPE WJ, 2008. Molecular networks regulating Arabidopsis seed maturation, after-ripening, dormancy and germination [J]. New Phytol, 179(1): 33–54.

HU Y, ZHOU L, HUANG M, et al., 2018. Gibberellins play an essential role in late embryogenesis of Arabidopsis [J]. Nat Plants, 4(5): 289–298.

HUANG H, XIE S, XIAO Q, et al., 2016. Sucrose and ABA regulate starch biosynthesis in maize through a novel transcription factor, ZmEREB156 [J]. Sci Rep, 6: 27590.

JIA H, SUZUKI M, McCARTY DR, 2014. Regulation of the seed to seedling developmental phase transition by the LAFL and VAL transcription factor networks [J]. Wiley Interdisciplinary Reviews Develop Biol, 3(1): 135–145.

JO L, PELLETIER JM, HARADA JJ, 2019. Central role of the LEAFY COTYLEDON 1 transcription factor in seed development [J]. J Integr Plant Biol, 61(5): 564–580.

JUNKER A, MÖNKE G, RUTTEN T, et al., 2012. Elongation-related functions of LEAFY COTYLEDON 1 during the development of *Arabidopsis thaliana* [J]. Plant J, 71(3): 441–455.

KAGAYA Y, OKUDA R, BAN A, et al., 2005. Indirect ABA-dependent regulation of seed storage protein genes by FUSCA3 transcription factor in Arabidopsis [J]. Plant Cell Physiol, 46(2): 300–311.

KANNO Y, JIKUMARU Y, HANADA A, et al., 2010. Comprehensive hormone profiling in developing Arabidopsis seeds: Examination of the site of ABA biosynthesis, ABA transport and hormone interactions [J]. Plant Cell Physiol, 51(12): 1988–2001.

KOTAK S, VIERLING E, BAUMLEIN H, et al., 2007. A novel transcriptional cascade regulating expression of heat stress proteins during seed development of Arabidopsis [J]. Plant Cell, 19(1): 182–195.

KOZAKI A, AOYANAGI T, 2022. Molecular aspects of seed development controlled by gibberellins and abscisic acids [J]. Int J Mol Sci, 23(3): 1876.

LEE HG, LEE K, SEO PJ, 2015. The Arabidopsis MYB96 transcription factor plays a role in seed dormancy [J]. Plant Mol Biol, 87(4–5): 371–381.

LEFEBVRE V, NORTH H, FREY A, et al., 2006. Functional analysis of Arabidopsis NCED6 and NCED9 genes indicates that ABA synthesized in the endosperm is involved in the induction of seed dormancy [J]. Plant J, 45(3): 309–319.

LI J, BERGER F, 2012. Endosperm: Food for humankind and fodder for scientific discoveries [J]. New Phytol, 195(2): 290–305.

LI X, CHEEN T, LI Y, et al., 2019. ETR1/RDO3 regulates seed dormancy by relieving the inhibitory effect of the ERF12-TPL complex on DELAY OF GERMINATION 1 expression [J]. Plant Cell, 31(4): 832–847.

LIM J, LIM CW, LEE SC, 2022. Core components of abscisic acid signaling and their post-translation modification [J]. Front Plant Sci, 13: 895698.

LIU Y, FANG J, XU F, et al., 2014. Expression patterns of ABA and GA metabolism genes and hormone levels during rice seed development and imbibition: A comparison of dormant and non-dormant rice cultivars [J]. J Genet Genom, 41(6): 327–338.

LIU F, ZHANG H, DING L, et al., 2020. REVERSAL OF RDO5 1, a homolog of rice seed dormancy 4, interacts with bHLH57 and controls ABA biosynthesis and seed dormancy in Arabidopsis [J]. Plant Cell, 32(6): 1933–1948.

LIU X, ZHANG H, ZHAO Y et al., 2013. Auxin controls seed dormancy through stimulation of abscisic acid signaling by inducing ARF-mediated ABI3 activation in Arabidopsis [J]. Proc Natl Acad Sci USA, 110(38): 15485–15490.

MA Y, SZOSTKIEWICZ I, KORTE A, et al., 2009. Regulators of PP2C phosphatase activity function as abscisic acid sensors [J]. Science, 324(5930): 1064–1068.

MARION-POLL A, LEUNG J, 2006. Abscisic acid synthesis, metabolism and signal transduction [M]. In: HEDDEN P, THOMAS SG (EDS), Plant Hormone Signalling. Ann Plant Rev, 24: 138–165.

MENDES A, KELLY AA, VAN ERP H, et al., 2013. bZIP67 regulates the omega-3 fatty acid content of Arabidopsis seed oil by activating fatty acid desaturase 3 [J]. Plant Cell, 25(8): 3035–3048.

MIAO C, XIAO L, HUA K, et al., 2018. Mutations in a subfamily of abscisic acid receptor genes promote rice growth and productivity [J]. Proc Natl Acad Sci USA, 115(23): 6058–6063.

MILLER M, 2016. Interactions of CCAAT/enhancer-binding protein β with transcriptional co-regulators [J]. Postepy Biochem, 62: 343–348.

MÖNKE G, SEIFERT M, KEILWAGEN J, et al., 2012. Toward the identification and regulation of the *Arabidopsis thaliana* ABI3 regulon [J]. Nucleic Acids Res, 40(17): 8240–8254.

MU J, TAN H, ZHENG Q, et al., 2008. LEAFY COTYLEDON 1 is a key regulator of fatty acid biosynthesis in Arabidopsis [J]. Plant Physiol, 148(2): 1042–1054.

NAKABAYASHI K, BARTSCH M, XIANG Y, et al., 2012. The time required for dormancy release in Arabidopsis is determined by DELAY OF GERMINATION 1 protein levels in freshly harvested seeds [J]. Plant Cell, 24(7): 2826–2838.

NAKASHIMA K, FUJITA Y, KANAMORI N, et al., 2009. Three Arabidopsis SnRK2 protein kinases, SRK2D/SnRK2.2, SRK2E/SnRK2.6/OST1 and SRK2I/SnRK2.3, involved in ABA signaling are essential for the control of seed development and dormancy [J]. Plant Cell Physiol, 50(7): 1345–1363.

NAMBARA E, MARION-POLL A, 2005. Abscisic acid biosynthesis and catabolism [J]. Annu Rev Plant Biol, 56: 165–185.

NAMBARA E, OKAMOTO M, TATEMATSU K, et al., 2010. Abscisic acid and the control of seed dormancy and germination [J]. Seed Sci Res, 20(2): 55–67.

NÉE G, KRAMER K, NAKABAYASHI K, et al., 2017. DELAY OF GERMINATION 1 requires PP2C phosphatases of the ABA signalling pathway to control seed dormancy [J]. Nat Commun, 8: 72.

NELSON SK, STEBER CM, 2016. Gibberellin hormone signal perception: Down-regulating DELLA repressors of plant growth and development [M]. In: HEDDEN P, THOMAS SG (EDS). The Gibberellins. Ann Plant Rev, 49: 153–187.

NISHIMURA N, TSUCHIYA W, MORESCO JJ, et al., 2018. Control of seed dormancy and germination by DOG1-AHG1 PP2C phosphatase complex via binding to heme [J]. Nat Commun, 9: 2132.

NONOGAKI H, 2019a. Seed germination and dormancy: The classic story, new puzzles, and evolution [J]. J Integr Plant Biol, 61(5): 541–563.

NONOGAKI H, 2019b. ABA responses during seed development and germination [J]. Adv Bot Res, 92: 171–217.

NORTH HM, DE ALMEIDA A, BOUTIN JP, et al., 2007. The Arabidopsis ABA-deficient mutant aba4 demonstrates that the major route for stress-induced ABA accumulation is via neoxanthin isomers [J]. Plant J Cell Mol Biol, 50(5): 810–824.

PARK SY, FUNG P, NISHIMURA N, et al, 2009. Abscisic acid inhibits type 2C protein phosphatases via the PYR/PYL family of START proteins [J]. Science, 324(5930): 1068–1071.

ROSCOE TT, GUILLEMIINOT J, BESSOULE JJ, et al., 2015. Complementation of seed maturation phenotypes by ectopic expression of ABSCISIC ACID INSENSITIVE 3, FUSCA3 and LEAFY COTYLEDON 2 in Arabidopsis [J]. Plant Cell Physiol, 56(6): 1212–1225.

SALL K, DEKKERS BJW, NONOGAKI M, et al., 2019. DELAY OF GERMINATION 1-LIKE 4 acts as an inducer of seed reserve accumulation [J]. Plant J, 100(1): 7–19.

SANO N, MARION-POLL, 2021. ABA metabolism and homeostasis in seed dormancy and germination [J]. Int J Mol Sci, 22(10): 5069.

SEO M, AOKI H, KOIWAI H, et al., 2004. Comparative studies on the Arabidopsis aldehyde oxidase (AAO) gene family revealed a major role of AAO3 in ABA biosynthesis in seeds [J]. Plant Cell Physiol, 45(11): 1694–1703.

SHU K, CHEN Q, WU Y, et al., 2016. ABI4 mediates antagonistic effects of abscisic acid and gibberellins at transcript and protein levels [J]. Plant J, 85(3): 348–361.

SHU K, ZHANG H, WANG S, et al., 2013. ABI4 regulates primary seed dormancy by regulating the biogenesis of abscisic acid and gibberellins in Arabidopsis [J]. PLoS Genet, 9(6): e1003577.

SINGH DP, FILARDO FF, STOREY R, et al., 2010. Overexpression of a gibberellin inactivation gene alters seed development, KNOX gene expression, and plant development in Arabidopsis [J]. Physiol Plant, 138(1): 74–90.

SMOLIKOVA G, LEONOVA T, VASHURINA N, et al., 2021. Desiccation tolerance as the basis of long-term seed viability [J]. Int J Mol Sci, 22(1): 101.

SOHN SI, PANDIAN S, KUMAR TS, et al., 2021. Seed dormancy and pre-harvest sprouting in rice—An updated overview [J]. Int J Mol Sci, 22(21): 11804.

SONG SQ, LIU J, HUANG H, et al., 2020. Gibberellin metabolism and signaling and its molecular mechanism in regulating seed germination and dormancy [J]. Sci Sin Vitae, 50(6): 599–615.

SONG SQ, LIU J, TANG CF, et al., 2022. Research progress on the physiology and its molecular mechanism of seed desiccation tolerance [J]. Sci Agric Sin, 55(6): 1047–1063.

STONE SL, BRAYBROOK SA, PAULA SL, et al., 2008. Arabidopsis LEAFY COTYLEDON 2 induces maturation traits and auxin activity: Implications for somatic embryogenesis [J]. Proc Natl Acad Sci USA, 105(8): 3151–3156.

SUGIMOTO K, TAKEUCHI Y, EBANA K, et al., 2010. Molecular cloning of Sdr4, a regulator involved in seed dormancy and domestication of rice [J]. Proc Natl Acad Sci USA, 107(13): 5792–5797.

SUZUKI M, LATSHAW S, SATO Y, et al., 2008. The maize Viviparous 8 locus, encoding a putative ALTERED MERISTEM PROGRAM1-like peptidase, regulates abscisic acid accumulation and coordinates embryo and endosperm development [J]. Plant Physiol, 146(3): 1193–1207.

SWAIN SM, REID JB, KAMIYA Y, 1997. Gibberellins are required for embryo growth and seed development in pea [J]. Plant J, 12(6): 1329–1338.

TAN BC, SCHWARTZ SH, ZEEVAART JA, et al., 1997. Genetic control of abscisic acid biosynthesis in maize [J]. Proc Natl Acad Sci USA, 94(22): 12235–12240.

TAYLOR IB, LINFORTH RST, AL-NAIEB RJ, et al., 2010. The wilty tomato mutants flacca and sitiens are impaired in the oxidation of ABA aldehyde to ABA [J]. Plant Cell Environ, 11(8): 739–745.

TO A, VALON C, SAVION G, et al., 2006. A network of local and redundant gene regulation governs Arabidopsis seed maturation [J]. Plant Cell, 18(7): 1642–1651.

TUAN PA, KUMAR R, REHAL PK, et al., 2018. Molecular mechanisms underlying abscisic acid/gibberellin balance in the control of seed dormancy and germination in cereals [J]. Front Plant Sci, 9: 668.

UTSUGI S, ASHIKAWA I, NAKAMURA S, et al., 2020. TaABI5, a wheat homolog of *Arabidopsis thaliana* ABA insensitive 5, controls seed germination

[J]. J Plant Res, 133(2): 297–307.

VERDIER J, LALANNE D, PELLETIER S, et al., 2013. A regulatory network-based approach dissects late maturation processes related to the acquisition of desiccation tolerance and longevity of *Medicago truncatula* seeds [J]. Plant Physiol, 163(2): 757–774.

VERMA S, ATTULURI VPS, ROBERT HS, 2021. An essential function for auxin in embryo development [J]. Cold Spring Harbor Perspect Biol, 13(4): a039966.

VERMA S, ATTULURI VPS, ROBERT HS, 2022. Transcriptional control of Arabidopsis seed development [J]. Planta, 255(4): 90.

WANG K, HE J, ZHAO Y, et al., 2018. EAR1 negatively regulates ABA signaling by enhancing 2C protein phosphatase activity [J]. Plant Cell, 30(4): 815–834.

WANG F, PERRY SE, 2013. Identification of direct targets of FUSCA3, a key regulator of Arabidopsis seed development [J]. Plant Physiol, 161(3): 1251–1264.

WANG Z, REN Z, CHENG C, et al., 2020. Counteraction of ABA mediated inhibition of seed germination and seedling establishment by ABA signaling terminator in Arabidopsis [J]. Mol Plant, 13(9): 1284–1297.

XIANG Y, NAKABAYASHI K, DING J, et al., 2014. REDUCED DORMANCY 5 encodes a protein phosphatase 2C that is required for seed dormancy in Arabidopsis [J]. Plant Cell, 26(11): 4362–4375.

YAMAMOTO A, KAGAYA Y, USUI H, et al., 2010. Diverse roles and mechanisms of gene regulation by the Arabidopsis seed maturation master regulator FUS3 revealed by microarray analysis [J]. Plant Cell Physiol, 51(12): 2031–2046.

YAMASAKI Y, GAO F, JORDAN MC, et al., 2017. Seed maturation associated transcriptional programs and regulatory networks underlying genotypic difference in seed dormancy and size/weight in wheat (*Triticum aestivum* L.) [J]. BMC Plant Biol, 17: 154.

YOSHIDA T, NISHIMURA N, KITAHATA N, et al., 2006. ABA hypersensitive germination 3 encodes a protein phosphatase 2C (AtPP2CA) that strongly regulates abscisic acid signaling during germination among Arabidopsis protein phosphatase 2Cs [J]. Plant Physiol, 140(1): 115–126.

ZHANG YQ, LU X, ZHAO FY, et al., 2016. Soybean GmDREBL increases lipid content in seeds of transgenic Arabidopsis [J]. Sci Rep, 6: 34307.

ZHAO Y, CHAN Z, XING L, et al., 2013. The unique mode of action of a divergent member of the ABA-receptor protein family in ABA and stress signaling [J]. Cell Res, 23(12): 1380–1395.

ZHAO H, NIE K, ZHOU H, et al., 2020. ABI5 modulates seed germination via feedback regulation of the expression of the PYR/PYL/RCAR ABA receptor genes [J]. New Phytol, 228(2): 596–608.

ZHAO Y, ZHANG Z, GAO J, et al., 2018. Arabidopsis duodecuple mutant of PYL ABA receptors reveals PYL repression of ABA-independent SnRK2 activity [J]. Cell Rep, 23(11): 3360–3371.

ZHENG Z, XIE X, CROSLEY RA, et al., 2010. The protein kinase SnRK2.6 mediates the regulation of sucrose metabolism and plant growth in Arabidopsis [J]. Plant Physiol, 153(1): 99–109.

ZINSMEISTER J, LALANNE D, TERRASSON E, et al., 2016. ABI5 is a regulator of seed maturation and longevity in Legumes [J]. Plant Cell, 28(11): 2735–2754.

Note: Figure translations are in progress. See original paper for figures.