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Updated role of ABA in seed maturation, dormancy, and germination

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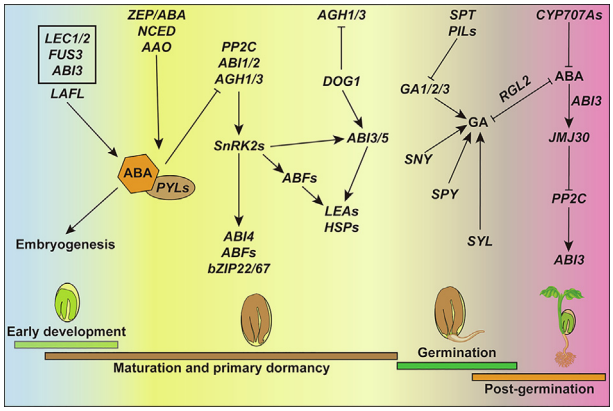
HIGHLIGHTS

- Functional ABA biosynthesis genes show specific roles for ABA accumulation at different stages of seed development and seedling establishment.
- *De novo* ABA biosynthesis during embryogenesis is required for late seed development, maturation, and induction of primary dormancy.
- ABA plays multiple roles with the key LAFL hub to regulate various downstream signaling genes in seed and seedling development.
- Key ABA signaling genes *ABI3*, *ABI4*, and *ABI5* play important multiple functions with various cofactors during seed development such as de-greening, desiccation tolerance, maturation, dormancy, and seed vigor.
- The crosstalk between ABA and other phytohormones are complicated and important for seed development and seedling establishment.

GRAPHICAL ABSTRACT



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ABSTRACT

Background: Seed is vital for plant survival and dispersion, however, its development and germination are influenced by various internal and external factors. Abscisic acid (ABA) is one of the most important phytohormones that influence seed development and germination. Until now, impressive progresses in ABA metabolism and signaling pathways during seed development and germination have been achieved. At the molecular level, ABA biosynthesis, degradation, and signaling genes were identified to play important roles in seed development and germination. Additionally, the crosstalk between ABA and other hormones such as gibberellins (GA), ethylene (ET), Brassinolide (BR), and auxin also play critical roles. Although these studies explored some actions and mechanisms by which ABA-related factors regulate seed morphogenesis, dormancy, and germination, the complete network of ABA in seed traits is still unclear.
Aim of review: Presently, seed faces challenges in survival and viability. Due to the vital positive roles in dormancy induction and maintenance, as well as a vibrant negative role in the seed germination of ABA, there is

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a need to understand the mechanisms of various ABA regulators that are involved in seed dormancy and germination with the updated knowledge and draw a better network for the underlying mechanisms of the ABA, which would advance the understanding and artificial modification of the seed vigor and longevity regulation.

Key scientific concept of review: Here, we review functions and mechanisms of ABA in different seed development stages and seed germination, discuss the current progresses especially on the crosstalk between ABA and other hormones and signaling molecules, address novel points and key challenges (e.g., exploring more regulators, more cofactors involved in the crosstalk between ABA and other phytohormones, and visualization of active ABA in the plant), and outline future perspectives for ABA regulating seed associated traits.

Introduction

The plant development starts with the seed, followed by the seedling, the vegetative phase, and end with the reproductive phase [1–3]. Seed production is important for the reproduction and diffusion of many plant species that contain a fully developed embryo and allows the embryo to stay alive during seed maturation and seedling establishment for next-generation initiation [4]. There are two important phases of seed development which include zygotic embryogenesis, seed maturation. Seed maturation occurs as a result of complex, overlapping developmental processes that start from the end of embryogenesis and end when seeds become physiologically independent of the parent plant. It includes a phase of seed storage reserve deposition and the less characterized phase of maturation drying. Furthermore, during maturation, seeds acquire a range of physiological traits i.e. dormancy, vigorous and homogenous germination, after these processes, a viable seedling is established in the field for the life cycle [1,5]. Seed dormancy and germination are critical phases in the higher plant life cycle and important traits for crop yield, however, both of them are influenced by developmental and environmental signals [6].

Seed dormancy is a key characteristic to prevent viable seed germinating during the harsh and tough growing season [4,7]. Low seed dormancy level or non-dormant seed increases the risk of seed death and directs the seed to germinate under unfavorable growth conditions, while a high seed dormancy level stops or reduces the seed germination under favorable growth conditions which ultimately reduces the length of the growing season or crop yield [6,8,9]. Thus, proper seed dormancy is an important component of plant fitness and provides adaptation to a wide variety of environmental conditions. Further, it is a genetic character influenced by inherence as well as environmental factors. Along with seed later development and maturation, seed dormancy starts to build and reaches a higher level in dry mature seeds known as primary dormancy [10]. In contrast, the induction of dormancy in non-dormant seed due to unfavorable environmental conditions for germination such as light and temperature is known as secondary dormancy [7].

Germination is important that occurs in the lifecycle of all higher plants and has the potential to influence the evolution of traits expressed throughout the life of plants [8]. Seed germination starts when the dormant seed uptakes water and accomplishes when a part of the embryo such as a radicle comes out from the seed coat. The emergence of a radicle by rupturing the seed coat is known as the completion of the germination, however, this procedure depends on the absorption of water by the embryo and activation of a series of physiological events [11]. Germination requires specific environmental conditions, the seed sensitivity to the environment changes continuously as a function to adapt to ambient conditions. Therefore, seed germination depends on endogenous hormonal as well as environmental signals such as temperature, water, and light that allow a dormant seed to germinate successfully [12].

Environmental factors (temperature, soil, nutrition, light, water, humidity, air, pollutants, etc.) influence seeds dormancy and germination as well as other developmental stages of seed [13]. Seed catch signals from the environment, then endogenous pathways involved by phytohormones, such as calcium ion, reactive oxygen species are activated and seed dormancy/germination and other developmental stages are designed accordingly [14,15]. Phytohormones play key distinct roles in the plant life cycle, from seed maturation, seed germination to the floral transition and abiotic/biotic stress responses [13,16]. For example auxin, ABA, ET, and GA have been found that have important roles during plant development and in seed dormancy and germination regulation [17–19]. Plants maintain the availability and level of hormones in different parts of the plant body at different developmental stages in an intricate and balanced manner [20]. ABA is derived from epoxycarotenoid cleavage and is obtained one of important plant-specific hormone among other, and performs various physiological functions in the plant such as in transpiration, improved resistance from temperature (low and high) during plant development, and in the regulation of dormancy and germination [23–25]. In dormancy and germination control, ABA is one of the key hormones that play a prominent role [10,21,22].

Similarly, it is hypothetical that ABA plays a vital role to maintain the dormant form of seeds in a severe environment [19,21,26]. Previously, it has been reported that ABA biosynthesis, signaling, and degradation genes play important functions in induction, stabilization, and release of dormancy. The mutation or overexpression of key ABA-related genes results in germination-associated phenotypes [27–30]. In this review, we focus and discuss the updated findings related to ABA biosynthesis, signaling and degradation, and its versatile functions associated with seed development and seedling establishment, raise some key questions for the future study of ABA function.

Role of ABA biosynthesis genes in seed development

Maternal ABA plays a significant role in embryo development and seed maturation in tobacco and Arabidopsis [31]. But, ABA is also *de novo* synthesized in embryo and testa during embryo development, as well as accumulates during seed maturation, facilitates late seed maturation processes, synthesis of storage proteins to prevent seed abortion, induces primary dormancy and allows successful germination as well as a successive seedling enterprise [32]. So, *de novo* synthesis of active ABA plays a more important role in seed development and later germination.

Active ABA is synthesized through an indirect pathway from xanthophylls (e.g., zeaxanthin, violaxanthin, and neoxanthin) [33,34]. Three types of genes are responsible for the successive

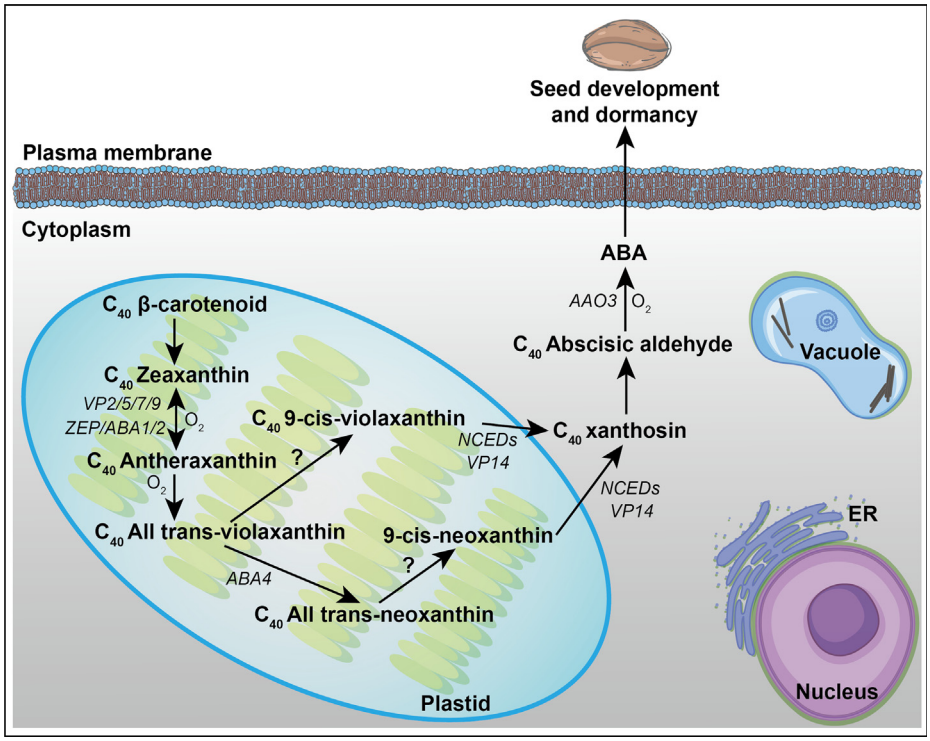

Fig. 1. Regulation of seed development and dormancy by ABA biosynthesis through the carotenoid pathway started from β-carotene (C40). The complete ABA synthesis process takess place in plastids and cytoplasm where ZEAXANTHIN EPOXIDASE (VPs, ZEP, ABA1/2) converts zeaxanthin into antheraxanthin and all *trans*-violaxanthin. ABA4 catalyzes the conversion from all-*trans*-violoxanthin to the all-*trans*-neoxanthin. The conversion of xanthoxin from 9′-*cis*-neoxanthin and 9′-*cis*-violaxanthin is exerted by VP14 and NCEDs (NINE-CIS-EPOXYCAROTENOID DIOXYGENASE), among which the NCEDs display different subcellular localization of plastid or cytoplasm. The oxidation of abscisic aldehyde by *AAO3* (*ABSCISIC ALDEHYDE OXIDASE3*) is responsible for the conversion from abscisic aldehyde into ABA, which in turn induces and maintains seed dormancy. But, it is yet unknown of the factors responsible for the conversion from all-*trans*-violoxanthin /all-*trans*-neoxanthin to 9-*cis*-violoxanthin/9-*cis*-neoxanthin.

steps of ABA biosynthesis such as *ZEAXANTHIN EPOXIDATION* (*ZEP*), *OXIDATIVE CLEAVAGE OF 9-CIS-EPOXYCAROTENOIDS* (*NCED*), and *ABSCISIC ALDEHYDE OXIDATION* (*AAO*) (Fig. 1) [35].

The *ZEP*/*ABA* gene was firstly identified in *Arabidopsis thaliana* and *Nicotiana plumbaginifolia* [36]. Their mutants (*aba1*/*aba2*) with deficient ABA were impaired in the oxidation of zeaxanthin into antheraxanthin and violaxanthin [37], which is thought as an initial step of ABA biosynthesis (Fig. 1). In rice, a *Tos17 viviparous* mutant was identified to have viviparous germination due to a defect in the oxidation of zeaxanthin during ABA synthesis [38]. Numerous other ABA auxotrophic mutants (*vp2*, *vp5*, *vp7*, and *vp9)* identified in maize by genetic screening have defects in zeaxanthin epoxidase activity and block the early steps of carotenoid biosynthesis too [39]. All these evidenced that the oxidation of zeaxanthin is an important and conservative phase in the ABA synthesis of the plant. It is always not very clear for the conversion from all-*trans*-violoxanthin and the all-*trans*-neoxanthin to 9-*cis*-violoxanthin and 9-*cis*-neoxanthin. However, ABA4 was found responsible for conversion from all-*trans*-violoxanthin to the all-*trans*-neoxanthin [40], providing some clues for the exploration of these transition.

The next pivotal gene in the subsequent stages of ABA biosynthesis was firstly cloned from maize viviparous mutant *vp14* as *NINE-CIS-EPOXYCAROTENOID DIOXYGENASE* (*NCED9*). The *vp14* mutant has a fault in the oxidation of 9-*cis*-epoxycarotenoid during the last few steps in ABA biosynthesis and exhibits reduced ABA content in the dry seed [41]. In *Arabidopsis*, *NCED2*, *NCED3*, *NCED5*, *NCED6*, and *NCED9* are known as the homologs of *VP14* participating in a rate-limiting step in ABA biosynthesis [24] (Fig. 1). Furthermore, the *PvNCED1*, *LeNCED1*, and *BdNCED1* identified from the bean, tomato, and *Brachypodium distachyon*, respectively also show the important roles in ABA biosynthesis and seed development [42,43]. All the above studies have delivered pieces of evidence that the oxidative cleavage of xanthophylls is the main step during ABA biosynthesis regulation for dormancy and development mediation in seeds [44].

Abscisic aldehyde oxidation is the last step of ABA biosynthesis, where abscisic aldehyde is oxidatively converted into ABA (Fig. 1) [45]. Firstly, identified mutants defective in the oxidation of abscisic aldehyde into ABA were *flacca* and *sitiens* in tomato [46]. Later on, abscisic aldehyde oxidase3 (AAO3) was identified in *Arabidopsis* which functions in the last two steps of ABA biosynthesis in seed and its expression was also observed in embryo vascular tissues during mid and late maturation phases [47,48]. The ABA synthetic pathway offers an active ABA pool during the whole plant development that is controlled by various homologous genes. The identification of cofactors of the enzymatic reactions in the ABA synthesis pathway would be helpful for the understanding of the complete network of ABA synthesis.

Role of ABA signaling components in different seed developmental stages

ABA works via a complex signaling network and initiates the cell response through activating downstream signaling genes to induce the response according to physiological effects [49,50]. In seed development and maturation, the role of ABA has been recognized by analyzing the mutants that were insensitive to ABA. The ABA insensitive mutants fail to promote ABA response due to defects in the ABA signaling pathway, which steadily affects seed maturation and several other important traits of the dormant seed [19].

Identification and mechanisms of the core components in ABA signaling pathway

The identification of PYL/RCAR family proteins verified that ABA receptor PYLs are essential ABA signaling components and predominantly function in seed [51]. In *Arabidopsis*, fourteen members of the PYR/PYL/RCAR protein family were documented that have vital roles in seed, such as *pyr1/prl1/prl2/prl4* quadruple and *pyl* duodecuple mutants show reduced seed dormancy and insensitivity to ABA [29,52]. Furthermore, an *ospyl* septuple mutant was identified in rice which is insensitive to ABA during seed germination [51].

In the absence of ABA, PYLs proteins release protein phosphatase type 2C (PP2C), another important component in ABA signaling, and activate their functions of phosphatase [29]. PP2Cs proteins including ABA-INSENSITIVE 1/2 (ABI1/2) and ABA-HYPERSENSITIVE GERMINATION3 (AHG3) suppress the activities of downstream ABA signaling proteins SUCROSE NONFERMENTING 1-RELATED PROTEIN KINASE 2s (SnRK2s) by protein phosphorylation, as a result, blocking the function of the downstream ABA signaling network (Fig. 2) [30]. So, PP2Cs function as negative regulators in the ABA signaling system and were identified through ABA insensitive mutants screening, whereas, their knockout mutants exhibited reduced seed dormancy and hypersensitivity to ABA [53]. Recently, it is demonstrated that ENHANCER OF ABA CO-RECEPTOR1 (EAR1) acts together with PP2C proteins (i.e. ABI1/2, HAB1/2, and AHG1/3), to increase the activities of PP2C [54]. Like EAR1, PR5 receptor-like kinase 2 (PR5K2) inhibits ABA-signaling via phosphorylation enhancement of ABI1/2 [55]. On the other hand, DELAY OF GERMINATION1 (DOG1) binds to heme and interacts with the AHG1 to stop its phosphatase function and enhance seed dormancy [56]. These studies concluded that PP2Cs can be regulated either by PYLs receptors or by other proteins, but the complete phenomenon and relationships between PP2Cs, PYLs, and other regulatory factors (DOG1, PR5K2, and EAR1) is unidentified in seed development.

In the presence of ABA, PYR/PYL/RCAR protein binds with both the ABA and the PP2C proteins to stop the phosphatase activity of the PP2Cs, which releases and enables the SnRK2s function. It is showed that all members of PYLs protein family from *Arabidopsis* can interact with PP2C family members and function in ABA mediating response [57]. Totally, three *SnRK2s* (*SnRK2.2*, *SnRK2.3*, and *SnRK2.6*) were found as positive regulators of the ABA signaling network and involved in various seed developmental processes such as the de-greening process, accumulation of seed storage products, seed maturation, desiccation-tolerant, and germination in *Arabidopsis* [19]. A recent report identified an ABA Signaling Terminator (ABT), a WD40 protein, which can efficiently shut down the ABA signaling and is vital for seed germination and seedling establishment. In a PYR1/PYL/RCAR-PP2C-dependent manner, ABT is induced by ABA and interacts with the PYR1/PYL/RCAR and PP2C proteins, which disturb the interaction between PYR1/4 and ABI1/2, thus cut off ABA signaling [58], which further enriches and illuminates the ABA signaling network.

In addition, the major targets of *SnRK2s* are *ABSCISIC ACID RESPONSIVE ELEMENT* (*ABRE*) binding factors (ABF). ABFs family consists of nine members *ABF1, ABF2/ABA–RESPONSIVE ELEMENT BINDING PROTEIN1 (AREB1), ABF3, ABF4/AREB2, AREB3, ABI5, bZIP15, bZIP67*, and *EEL* from *bZIP* subfamily, predominantly participates in the regulation of ABA-mediated transcription [59]. The transcription of *ABI5* can be activated by SnRK2s through specifically binding with *ABRE cis*-element in *ABI5* promoter, in turn, activate the ABA-mediated transcription activity in late seed maturation phase and imbibed seeds in *Arabidopsis* [60]. Moreover, another key factor-ABI3 interacts with the ABI5 transcription factor and functions collectively with ABI5 to promote transcription of downstream ABA-responsive genes [61], both of them can be regulated by RELATED TO ABI3/VP1 (RAV1) through binding to their promoters. Interestingly, ABI5 also modulates the ABA response through binding to *PYL11* and *PYL12* promoters to regulate the transcription directly during germination. ABA hypersensitivity resulting from *PYL11* and *PYL12* overexpression was totally or partially damaged when ABI5 was mutated. Above all these explore a feedback regulation in ABA signaling mediated by ABI5 [62].

By genetic screening, two *LEAFY COTYLEDON* (*LEC1/2*) genes and *FUSCA3* (*FUS3*) were identified as key roles in ABA-mediated seed development. ABI3 along with LEC1, LEC2, and FUS3 transcription factors play roles in seed development by mediating ABA biosynthesis in seed tissues. For the above four genes (i.e. *ABI3*, *FUS3*, *LEC1*, and *LEC2*), a defect in any one leads to abnormal seed development such as altered seed dormancy, failure to attain desiccation tolerance, and a low level of ABA contents [26]. All these supported that there is a positive correlation between these transcription factors and ABA content and signaling transduction [63]. Moreover, *ABI3/FUS3/LEC1/2* combine with *LEC1-LIKE* (*L1L*) to form a complex transcription control network known as *LAFL* that control the embryogenesis process, hormone signaling, metabolic pathways, and function upstream of several genes such as *PEI1*, *BABY BOOM* (*BBM*), *APETALA2* (*AP2*), *SEED STORAGE PROTEINS* (*SSP*), *FLOWERING LOCUS C* (*FLC*), including *2S ALBUMIN STORAGE CRUCIFERIN C* (*CRC*) and *PROTEIN 1* (*At2S1*) that involve seed development [64–66]. Many members of *LAFL* network are regulated by *BBM* during somatic embryogenesis [67], indicating the feedback regulation between BBM and LAFL components. Additionally, they are also regulated by VIVIPAROUS 8 (VP8), a B3 type transcription factor in maize, that show the pleiotropic roles during seed development [68]. In maize, a *defective kernel 33* (*dek33*) mutant was identified and the causal locus was cloned as a pyrimidine reductase in riboflavin biosynthesis. The genetic and molecular research indicated that DEK33 interacts with RGLG2 and SnRK1, influences the ABA synthesis positively to regulate seed development [69], which shed light on the regulation of ABA synthesis other than before in seed development.

Mechanisms of key genes associated with ABA in de-greening process of seed

In seed maturation, *SnRK2s* and *ABI3* genes were identified as an essential component for the de-greening process (Fig. 2) [70]. The *snrk2.2/snrk2.3/snrk2.6* triple mutant exhibits ABA insensitivity during seed development and produces green seeds [52,59]. Additionally, the targeted gene of *ABI3- stay-green (SGR1/2),* plays an important task in the process of de-greening of seed, whereas, *abi3-6* mutant exhibits pleiotropic effects during seed development including immature embryo growth, failure of embryo de-greening, and insensitivity to ABA as well as non-dormant and desiccation intolerant seeds [70], which indicated an important *SnRK2s -ABI3 -SGR1/2* pathway associated with ABA in seed de-greening and other traits determination.

Mechanisms of ABA in accumulation of seed storage products and desiccation tolerance achievement

Along with seed maturation, some reserve materials accumulate in the seed's later stages [71]. Six genes belonging to different transcription factors family (*ABI3/VP1*, *ABI4*, *ABI5*, *LEC1*, *LEC2*, and *FUS3*) have been identified that induces the expression of ABA-responsive and seed-specific factors such as *LEA* and storage protein genes [72]. ABI5, bZIP67 together with ABI3 and ABI4 control the expression of many genes that are involved in ABA-mediated seed storage processes [73,74]. *ABI4*, identified from the ABA-insensitive mutants, encoding an ERF/AP2 type transcription fac-

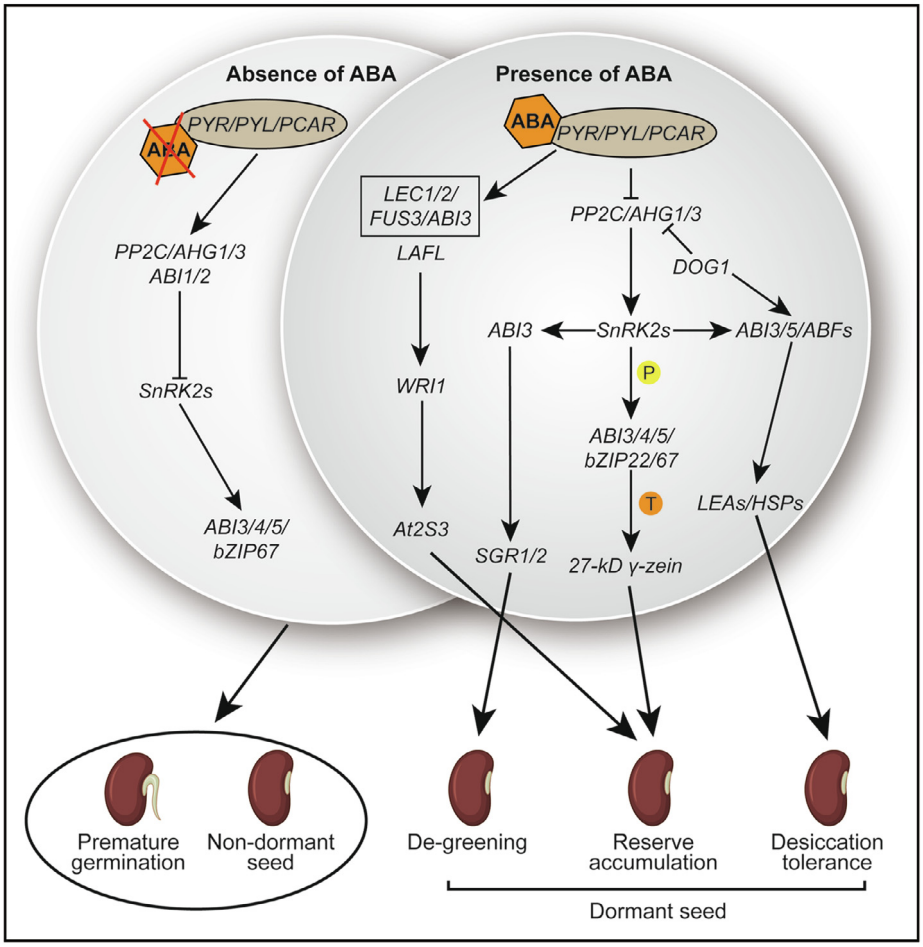

Fig. 2. The ABA signaling pathway is involved in seed development. **Left,** in the absence of ABA: Receptors PYLs release and activate protein phosphatase 2C (PP2C) such as ABI1/2 and AGH1/3. Downstream *SNF1-RELATED PROTEIN KINASE* subfamily (*SnRK2s*) genes are inactivated by active PP2C which leads to premature germination and the non-dormant seed through repression of lots of transcription factors such as ABI1/2/3/4/5 and bZIP67. **Right,** in the presence of ABA: Receptors PYR/PYL/RCAR bind ABA and PP2C together to inhibit the activity of PP2C, which release the activity of SnRK2s and downstream transcription factors such as ABI3 by protein phosphorylation, then regulate downstream genes *SGR1/2* function to mediate seed de-greening process. Additionally, the active *LAFL* (*ABI3, FUS3, LEC1,* and *LEC2*) network by ABA along with *WRI1* regulates the *At2S3* gene; an active *bZIP22* function downstream of *SnRK2s* to promote gene transcription of *27-kD γ-zein* for protein reserve accumulation in the seed. Along with seed de-greening and storage product accumulation, *SnRK2s* function upstream of *ABI3/5* and *ABFs* to regulate *LEAs* and *HSPs* that are pivotal for desiccation tolerance. In other branches, DOG1 also plays a role upstream of *ABI3/5/ABFs* as well as functions as a repressor of PP2Cs (AHG1/3) to involve seed desiccation tolerance acquirement. In combination, all key ABA signaling components (*SnRK2s, ABI3, ABI4, ABI5, ZmbZIP22, bZIP67,* and *ABFs*) are involved in storage product accumulation, de-greening, and desiccation tolerance with different function pathways to provide a mature and dormant seed. Letters "P" and "T" in the color circles indicate the two manners of protein phosphorylation and gene transcription regulation, respectively. Activated and repressive effects are shown by arrows and bars, respectively.

tor, expresses transcriptionally in all seed developmental stages [75]. Many studies reported that various transcription factors regulated *ABI4* transcription. Interestingly, ABI4 can also activate *ABI4* itself expression during the early stages of seedling growth [76]. The bZIP67 transcription factor together with L1L and NUCLEAR FACTOR-YC2 (NF-YC2) transcription factors form a complex to promote *FATTY ACID DESATURASE 3* (*FAD3*) in the seed which functions in the storage of omega-3 fatty acid during maturation [73]. Moreover, induced expression of maize *bZIP22* changes endosperm starch content and composition in maize and rice during seed storage and is required for the transcription of a *27-kD γ-zein* [77,78]. Many studies have reported that ABA insensitive mutants *snrk2.2/3/6* triple mutant and *pyl* duodecuple mutants exhibited less level of seed storage products due to defect in ABA signaling [52]. Moreover, the RNA-seq data analysis exhibited that the expression of 12S globulin storage protein was down-regulated in the *snrk2.2/3/6* triple mutant [79]. In addition, the induced expression of *SnRK2.6* showed increased seed production, on the contrary, *snrk2.6* mutant showed 7–25% reduced oil content of seed [80].

Besides above, the *lec1* and *fus3* mutant embryos exhibit reduced accumulation of storage proteins and lipids compared to wild-type during maturation [81]. Moreover, the LEC2 protein shows synergistic activity for the abundance of storage proteins with *ABI3, FUS3*, and *LEC1* during maturation [82]. FUS3 and LEC1 control the accumulation of ABI3 protein in seeds and function with each other in many physiological processes including lipids formation, anthocyanins synthesis, accumulation of chlorophyll, and storage proteins [81]. Comprehensive studies indicated that the expressions of several storage protein genes such as *Arabidopsis 2S storage protein 3* (*At2S3*) and *12S storage protein* gene rely on FUS3, ABI3, and WRINKLED1 (WRI1) transcription factors through an ABA-mediated manner [81,83,84] (Fig. 2). Moreover, *LEC1, LEC2*, and *GmDREBL* regulate *WRI1* to play roles in sugars and oil content storage in seed, as *wri1* mutant is revealed 80% less oil content and a higher level of soluble sugar in seed [85,86]. So, *LAFL* network regulates the expression of ABA signaling components including *PYR/PYL/RCARs, SnRK2s*, and *ABFs* that are involved to induce the expression of *LEAs* and *HSPs* genes at the time of seed maturation (Fig. 2) [52,59,87,88].

During the last phases of seed development, desiccation tolerance is acquired associated with the accumulation of antioxidants, sugar, and late embryogenesis abundant (LEA) proteins [89]. The genetic analysis of loss and gain of function mutants of *LEC1* indicated that *LEC1* is a major regulator during seed maturation and accumulation of storage products, desiccation tolerance as well as induction of dormancy [90]. *LEC1* functions together with *NF-YB, AREB3*, *bZIP67*, and *ABI3* to regulate genes required for seed maturation [91]. The *FUS3* gene is required for seed maturation and desiccation tolerance during seed development, thus, the *fus3* mutant showed premature embryo growth and seeds were desiccation intolerant [63,92]. The double mutant *aba/abi3* shows ABA insensitivity and produces seeds that are desiccation intolerant. ELONGATED HYPOCOTYL 5 (HY5) is an important light signaling component which binds with the promoter of *ABI5* to regulate the *LEAs* gene expression [93]. Further, *DOG1* increases the *LEA* and *HSP* genes expression through *ABI5/ABI3* and speeds up the storage of N-rich compounds in the seed which promotes the dormancy and viability of the seed [94]. In some studies, it is shown that *DOG1* expression is controlled by *bZIP67* and *ETHYLENE RESPONSE FACTOR12* (*ERF12*) during seed maturation negatively or positively [95,96]. Moreover, ABA regulates the expression of the *DOG1-LIKE 4* (*DOGL4*) gene and increases the expression of some seed storage proteins including CRUCIFERINs, ALBUMINs, and OLEOSINs during the seed maturation process [97]. From above, some specific factors (e.g., bZIP67, DOG1, NF-YC, AREB3) were identified to function associated with *LAFL* genes to mediate the seed storage protein accumulation and acquirement of desiccation tolerance related to maturation, which facilitates the elucidation of the regulation network of LAFL genes in the seed different developmental stages.

Mechanisms of ABA in primary seed dormancy induction

Dormancy is an imperative characteristic of wild plant species, prevents the seeds from adverse environmental conditions, and confirms the initiation of a next-generation [98,99]. Seed dormancy is achieved at the end of the seed maturation when molecular dependence from the mother plant disappears, storage products are synthesized, dehydration occurs, and *de novo* ABA is stored [24,100]. After dehydration, the seed enters into a state of dormancy physically and physiologically. The physical structures of the seed such as the testa and endosperm are responsible for the physical dormancy [101,102], from their ability to enhance seed impermeability or limit water uptake [103]. ABA is the major internal physiological factor inducing seed dormancy through affecting a lot of physiological pathways such as storage proteins and lipids in seed [79,82].

It is notable that *de novo* ABA biosynthesis occurs in the embryo and later is utilized during seed maturation and induction of primary dormancy indicating that the dormancy is a characteristic of an embryo and its related tissues [31,104]. Numerous ABA deficient (*aba* and *nceds*) and insensitive (*pyls*, *snrks*, and *abi3/4/5*) mutants show reduced seed dormancy and early germination indicating that ABA exerts a vital role in the induction of dormancy [59]. The AtNCED3 mainly expressed in the seed is perceived as the critical enzyme for ABA synthesis during early seed development, over-expression of which improved ABA contents in seeds and prolonged seed dormancy [44]. Similarly, *nced6* and *nced9* mutants show decreased ABA level and dormancy in mature dry seed [105]. Furthermore, a recent study speculated that *ODR1* (suppressor of *RDO5*) acted together with *bHLH57* and functioned upstream of *NCED6* and *NCED9* to control the ABA synthesis and seed dormancy in Arabidopsis [106]. Interestingly, the ectopic and over-expression of bean *PvNCED1* gene in imbibed seeds of tobacco elevated ABA levels and exhibited delayed seed germination. In tomato, the over-expression of *LeNCED1* also enhanced dormancy by enhancing the ABA level in seeds. In addition, MYB96 binds directly with ABA synthesis genes (*NCED2*, *NCED5*, NCED6, and *NCED9*) promoters and inactivates GA biosynthesis genes (*GA3ox1* and *GA20ox1*) to induced primary seed dormancy in *Arabidopsis* [107]. *ABI4* deepens seed dormancy in *Arabidopsis* through direct interaction with promoter regions of *NCED6* to increase ABA biosynthesis and with promoter regions of *GA2ox7*, a GA-inhibitor gene [108] to inhibit GA biosynthesis [109]. A study reported that peroxiredoxin PER1 improves the primary seed dormancy by inhibiting the ROS which in turn inactivates the ABA catabolism and GA biosynthesis genes in Arabidopsis [110]. In *Sorghum bicolor*, *ABI4* and *ABI5* (*SbABI4* and *SbABI5*) enhance the transcription of *SbGA2ox3* through directly binding to its promoter and consequently extend seed dormancy [111].

The loss of function mutant *lec1* shows premature germination during seed development indicating that *LEC1* is required for induction of primary seed dormancy [90]. During germination, the functions of *LAFL* network can be controlled or repressed by *VIVIPAROUS1/ABI3-LIKE1/2/3* (*VAL1/2/3*) [66]. Consistently, Members of *LAFL* genes are regulated by *VP8* in maize [68]. The mutations in *VP8* homolog gene *PLASTOCHRON3/GOLIATH* (*PLA3/GO*) in rice and *ALTEREDMERISTEM PROGRAM1* (*sAMP1*) in *Arabidopsis* show reduced dormancy phenotype [112]. Interestingly, VP8 and its homologs (PLA3/GO and AMP1) contained glutamate carboxypeptidase [68], indicating that this peptide might be important for seed maturation and dormancy, but its detailed biochemical mechanism is almost blank. Two individual studies demonstrated that the RAF-like MAPKKKs, RAF10/11 can phosphorylate SnRK2s and ABFs to influence the dormancy of seed [113,114]. The key factor DOG1 imposes primary seed dormancy by inhibiting AHG1 action to enhance ABA sensitivity [56]. Moreover, HISTONE DEACETYLASE 19 (HDA19) interacts with SIN3-Like 1 (SNL1) to modulate the ABA signaling pathway to promote seed dormancy [115], which contributes to further understanding between epigenetic modifications and ABA signal in seed development. In wheat, *TaABI5* transcripts accumulate in seed embryos. Over-expression of *TaABI5* in Arabidopsis displayed high sensitivity to ABA and increased dormancy, indicating that TaABI5 playing a positive role in dormancy maintenance as a functional ortholog to Arabidopsis ABI5 [116].

Mechanisms of ABA in seed germination and seedling establishment

Germination is a critical and initial process in the plant life cycle which starts with the uptake of water by mature seed at imbibition and shifts from maturation stage to germination stage via emerging the radicle [117]. During germination, the high levels of ABA in imbibed seeds of strongly dormant *A. thaliana* ecotype Cvi reduce clearly indicating that seed dormancy in *A. thaliana* Cvi accession seeds depends on the endogenous ABA level [118–120].

In many studies, it is proved that ABA catabolism is a crucial step to alter the dormancy state of seed to germination in *Hordeum vulagre*, *Pseudotsuga menziesii*, *Cupressus nootkatensis*, and yellow-cedar [121]. The ABA is degraded through consecutive hydroxylation and conjugation steps. The CYTOCHROME P450, FAMILY 707, SUBFAMILY A (CYP707As) provided with cytochrome P450 monooxygenase and ABA 8 prime-hydroxylase activity catalyzes the ABA to phaseic acid (PA). PA reductase (PAR) ABH2 and glycosyltransferase (GT) then catalyze PA to dihydrophaseic acid (DPA) and DPA-4-O-β-D-glucoside (DPAG), resulting in the ABA degradation (Fig. 3) [122]. The decreased level of ABA at the time of imbibition leads to a higher amount of PA and DPA accumulation in lettuce, *Arabidopsis*, and *H. vulgare* seed [27,123] suggesting the

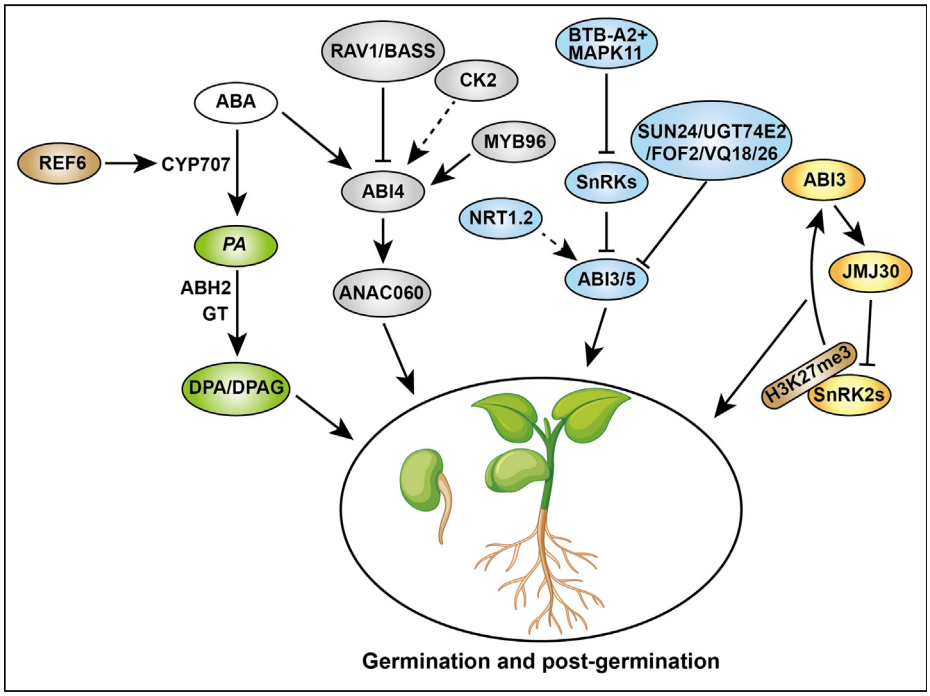

Fig. 3. The function of ABA in seed germination and seedling establishment. **Left,** Seed completes germination successfully through degradation of active ABA into PA (phaseic acid) and DPA (dihydrophaseic acid)/DPAG with CYP707As regulated by REF6 and phaseic acid reductase (ABH2 and GT) respectively. During germination and seedling establishment, the core ABA signaling component SnRK2s and downstream ABI3/4/5 are activated or repressed by many factors directly or indirectly to promote seed germination and seedling establishment. For example, RAV1 and BASS2 bind to the *ABI4* promoter to inhibit *ABI4* expression, while MYB96 promotes *ABI4* expression through binding to its promoter. A Casein Kinase 2 promotes *ABI4* expression indirectly. Furthermore, ANAC060 transcription is activated directly by ABI4 through binding to its promoter to enhance post-germination. Several BTB-A2 proteins can impair SnRK2.3 stability to act as negative regulators of ABA signaling. NRT1.2 is identified as an ABA transporter to regulate downstream factors ABI1-ABI5, RAB18, *etc.* positively to mediate germination and seedling development. Further, some negative factors such as SUN24, UGT74E2, FOF2, and VQs regulate seed germination and seedling development through repressing ABI3-, ABI5-mediated ABA pathway. Activated and repressive effects are shown by arrows and bars, respectively.

positive role of ABA 8-hydroxylation in germination [124]. Moreover, it was demonstrated that in non-dormant *H. vulgare* seeds, ABA catabolic enzyme *HvABA8′OH-1* expression was preferentially detected in coleorhiza which is an important tissue from where germination starts [125].

All four members of *CYP707As* (*CYP707A1-CYP707A4*) in *Arabidopsis* play regulatory functions to control the ABA level. The expression of *CYP707A1* is absent during zygotic embryogenesis [123] while is present in the embryo predominantly in the middle of seed maturation to inactivates ABA biosynthesis and decreases at maturity [125]. Whereas, CYP707A2 catabolism the ABA during late maturation, and *cyp707a2* mutant accumulates less ABA compared to *cyp707a1* mutant after imbibition [110]. The overexpression of *CYP707A2* decreased the ABA content in seed at maturity and reduced the storage time required to release the dormancy of the seeds whereas, *cyp707as* mutants required more storage time to reduce the dormancy compared to that of control [123,126]. In a recent study, it is demonstrated that Arabidopsis RELATIVE OF EARLY FLOWERING6 (AtREF6) directly binds and regulates the key ABA catabolism genes (*CYP707A1* and *CYP707A3*) to promote the catabolism of ABA and seed development [127].

Besides, the key components of ABA signaling also show indispensable roles in seed germination with various mechanisms. Recently, two members of the VQ family, VQ18 and VQ26, were proved to act as direct and negative interactors of the ABI5 to mediate the ABA signaling level and regulate seed germination and early seedling establishment [128]. Likewise, many studies proved that ABA signaling through the ABI4-mediated cascades such as miRNA 165/166, E3 ubiquitin ligase CER9 (ECERIFERUM 9), transcription factors OsAP2-39 and nuclear C2H2 zinc-finger protein ZFP3, and AtGLR3.5 (glutamate receptor homolog 3.5) [129–135] play important roles during seed germination and post-germination seedling growth, illustrating that ABI4 is a key factor with regard to ABA-mediated regulation of seed germination and early seedling establishment. Another gene *CK2* (*Casein Kinase 2*), positively enhances ABA signaling during seed germination and seedling establishment by enhancing *ABI4* expression partially and indirectly [136]. MYB96 increases *ABI4* expression during seed germination, while RAV1 and BILE ACID: SODIUM SYMPORTER FAMILY PROTEIN 2 (BASS2) repress *ABI4* expression during seedling development by binding to its promoter [76]. As the key terminator of ABA signaling, over-expression of ABT promotes seed germination and seedling greening in the presence of ABA, and knockout of ABT exhibits the contrary effect [58]. Three *BTB-A2* (*broad-complex*, *tramtrack*, *and bric-a-brac-A2*) domain family genes *BTB-A2.1, BTB-A2.2, and BTB-A2.3* act as negative regulators of ABA signaling by impacting *SnRK2.3* stability and subsequently weakening the expression of ABA-responsive genes, for example, *btb-a2.1/2/3* triple mutant showed a decrease in ABA-induced inhibition of seed germination by increasing ABA signaling [137]. In tomato, it is reported that MAPK11 also phosphorylates SnRK2s which affects ABA signaling by suppressing the transcription of *ABI5* and ultimately influences seed germination [138]; further, IQ67-Domain (IQD) protein SUN24 regulates seed germination by altering the expression of two key ABA signaling genes *Solanum lycopersicum ABA-insensitive 3/5* (*SlABI3* and *SlABI5*) in tomato germinating seeds [139]. UDP-glycosyltransferases (UGTs) transferring glucose to indole-3-butyric acid plays key roles in plant development. Overexpression of *OsUGT74E2* down-regulated the expression of *OsABI3* and *OsABI5*, and promoted seed germinating with a lower ABA level, indicating a regulation of seed germination involved by UGT74E2 function upstream of ABA signaling in rice

but not Arabidopsis [140]. In *Medicago truncatula*, a ATP-binding cassette (ABC) transporter, MtABCG20, functions as an ABA exporter in germinating seeds. In seeds of *mtabcg20*, due to the ABA translocation impairment, it showed more sensitivity to ABA in germination [141]. All these novel progresses in species other than *Arabidopsis* provide more rich knowledge for the mechanism of ABA in seed germination.

Besides the seed development, ABA synthesis and signaling genes play direct and crucial roles in the establishment of post-germination development but not in the phase conversion from germination to the seedling establishment [142]. In post-germination, ABA plays the role with multiple other participants such as JUMONJI-C domain-containing protein 30 (JMJ30), a histone demethylase activated by ABI3, which inhibits the enrichment of H3K27me3 at the promoter of *SnRK2.8*, activates the SnRK2.8′s kinase activity and ABI3 function to encourage post-germination growth (Fig. 3) [143]. This study revealed a forward regulatory loop associated with ABI3 in post-germination. F-BOX OF FLOWERING 2 (FOF2), a key factor in flowering, plays an important negative role in ABA-mediated seed germination and early seedling development, partially by repressing the expression of *ABI3* and *ABI5* [144]. During post-germination, ABI4 enhancec ANAC060 transcription to start post-germination growth by directly interacting with its promoter to reduce ABA sensitivity and glucose-mediated ABA accumulation [145]. Interestingly, another NAC family factor, NAC103 was up-regulated and enhanced in transcription and protein stabilization by ABA treatment respectively. Moreover, NAC103 over-expression plants showed more sensitivity to ABA during seed germination and young seedling growth, which was acquired by regulating some downstream genes such as *MYB78*, *PLP3*, and *RGL2* in Arabidopsis [146].

Pyrenophoric acid (P-Acid) is one kind of phytotoxic sesquiterpenoids produced by the Pyrenophora semeniperda, an effective mycoherbicide in crop cultivation. An intensive study found that it inhibites seedling establishment through activating the ABA signaling pathway; further, P-Acid B exerts the ABA biosynthesis pathway but not interacts with PYR/PYL receptors to involve the ABA pathway, which also explores the underlying mechanism associated with ABA of parasites in seed [147]. NITRATE TRANSPORTER 1.2 (NRT1.2) is identified as a nitrate transporter and an ABA transporter in Arabidopsis. Some ABA-responsive genes, *ABI1-ABI5*, *RAB18*, *RD29A*, and *PHOSPHOLIPASE Dα1* (*PLDα1*) were up-regulated by over-expression of *NRT1.2* as well as exogenous ABA. Consequently, NRT1.2 interacts with PLDα1 at the plasma membrane and positively involves the ABA pathway to mediate germination and seedling development [148]. From above, ABA interacts with different molecules or metabolites to involve seed germination and seedling development antagonistically/synergistically.

Crosstalk between ABA and other phytohormones and signaling molecules in seed germination

As endogenous organic substances, phytohormones play distinct roles in the plant life cycle from seed maturation, seed germination to the floral transition, and abiotic/biotic stress responses [13,149]. Numerous elegant studies have demonstrated that different phytohormones interact antagonistically and/or synergistically with one another and form complicated networks in seed germination regulation [108,150–152]. ABA and gibberellins (GA) are one pair of classic phytohormones, which antagonistically mediate several plant developmental processes and regulate the decision between dormancy and germination [6,19,153]. Therefore, the balance between catabolism and synthesis of ABA/GA by regulating signaling pathways stabilizes the balance between germination and dormancy (Fig. 3). Genetic and mutational analyses of the ABA and GA metabolism and signaling genes suggested that some genes are evidenced the importance in the regulation of seed germination and seedling growth [154,155]. For example, *FUS3* plays an important role to maintain ABA:GA balance by inhibiting GA biosynthesis and activating ABA biosynthesis during *Arabidopsis* seed development [63,92], and a reduced amount of ABA and increase in GA content in *fus3* mutant was detected during seed maturation indicating that FUS3 protein functions as a hub in GA and ABA synthesis in the seed [92,156]. ABI4 is another central factor mediating the antagonism between ABA and GA by regulating the biosynthesis of both phytohormones, resulting in the precise control of the degree of seed dormancy and post-germination seedling growth [16,157]. Alteration in *Arabidopsis ABI4* accumulates the GA content and reduces ABA content, which retrieves the dormant phenotype of *ga1-1* mutant, indicating that *ABI4* is also important to maintain the balance between ABA:GA ratio during seed development similar to *FUS3* (Fig. 4) [108,109]. However, the underlying molecular mechanisms between FUS3 and ABI4 in regulating the ABA/GA simultaneously are still a mystery.

Some generic nuclear factors are also involved in the crosstalk between ABA and GA. A study illustrated that *GERMINATION DEFECTIVE 1* (*GD1*) encoding a B3 domain TF suppresses *LEC2* and *FUS3* like gene (*OsLFL1*) and modulates GA biosynthesis genes (*OsGA3ox*, *OsGA20ox*, and *OsGA2ox*) expression to regulate germination in rice [158]. Another transcription factor containing AP2 domain CHOTTO1 (CHO1) enhances seed germination by regulating ABA-related genes to suppress GA biosynthesis genes in *Arabidopsis* [159]. Interestingly, three *NUCLEAR FACTOR-Y C (NF-YC)* homologs genes in *Arabidopsis NF-YC3/4/9* are involved in the regulation of GA-ABA crosstalk during seed which are regulated by GA to suppress ABA signaling [160]. Further research explored that *NF-YC9* promotes ABA responses in early seedling growth by binding to *ABI5* to increase ABA sensitivity [161] elucidating that the NF-YCs-ABI5 module integrates the antagonistic GA and ABA signaling in seed germination and post-germination stages (Fig. 4). These provide novel information to explore the underlying mechanisms associated with ABI5 of ABA and GA antagonism.

Further, the well-known negative GA signaling components such as DELLA proteins (i.e. GA INSENSITIVE (GAI), REPRESSOR OF GA1-3 (RGA), RGA-LIKE1 (RGL1), RGL2, and RGL3 influence seed dormancy and germination [162,163] through stimulating the ABA biosynthesis and *ABI5* activity, in which *NF-YC* and *RGL2* together promote the expression of *ABI5* and enhance the ABA-mediated repression of seed germination [160]. As a result, the *rgl2* mutant exhibited a reduced ABA concentration during imbibition, terminated dormancy, and accelerated germination [164]. In addition, in *Arabidopsis* RGL2 forms a complex with DOF6 transcription factor which positively activates GATA12 transcription to control seed germination [165]. The RGL2 can also be degraded by the COP9 Signalosome 1 (CSN1) which may inhibit *ABI5* activity and promote seed germination [166]. However, the mutation in GA signaling gene *SLEEPY1* (*SLY1*), exhibites higher germination and mRNA level of *RGL2*, indicating that *SLY1* functions independently of *RGL2* in seed germination [167]. Interestingly, ABA can enhance *RGL2* expression, this feedback loop modifies ABA and GA paths in the seed germination process [164]. Epigenetically, it is stated that E3 SUMO ligase SIZ1 sustained ABA: GA level by SUMOylating ABI5 to negatively regulate ABA signaling and SLY1, as well as to positively regulate GA signaling during germination in *Arabidopsis* [168–170]. Furthermore, some other genes involved in GA signaling were also identified including *SPINDLY* (*SPY*) and *SNEEZY* (*SNE*) belonging to F-box proteins involved in seed germination regulation (Fig. 4) [171,172]. All above these indicate that some factors in the GA pathway participate in the interplay of ABA and GA in seed germination through diverse function patterns.

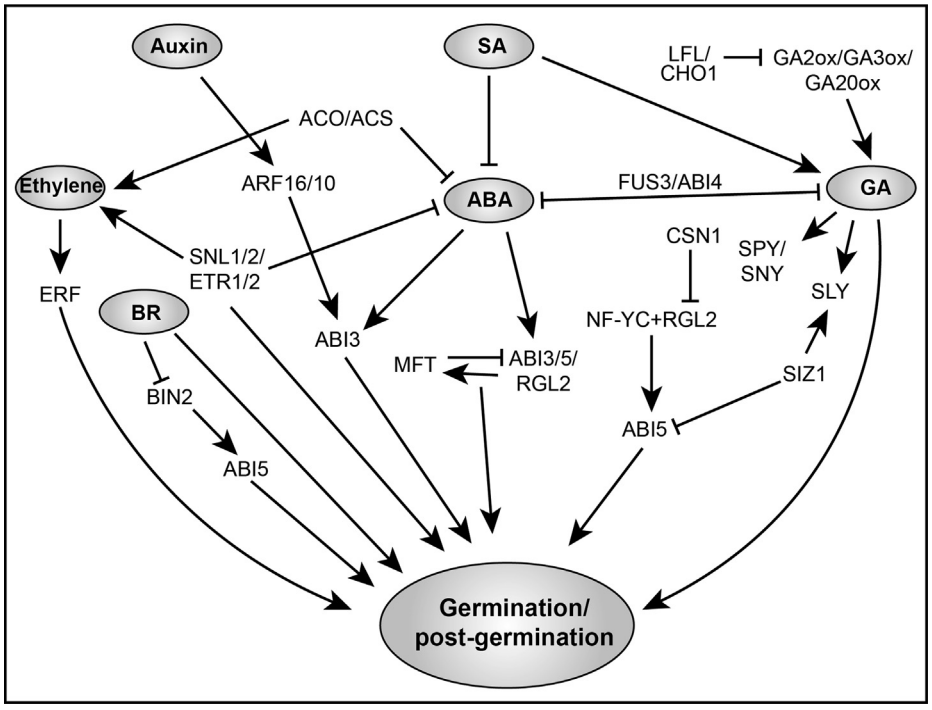

Fig. 4. The interplay of ABA and other phytohormones (GA, ET, SA, BR, and Auxin) signaling in the regulation of seed germination and post-germination growth. ABA crosstalks with other phytohormones either by affecting their biosynthesis or by interfering with their signaling pathways during germination and post-germination growth. Among these, the interaction between ABA and GA is most studied and important. FUS3 and ABI4 play more vital roles to mediate the antagonism between ABA and GA. SA was found to regulate the content of ABA negatively and GA positively, respectively in seed germination. Both two factors LFL and CHO1 were showed inhibition to GA synthesis to involve seed germination regulation. After that, some downstream factors of GA such as SPY, SNY, SLY, SIZ, RGL2, *etc.* display different functions in the crosstalk between GA and ABA. Both SIZ and RGL2 can repress and promote ABI5, respectively. Besides, RGL2 can be degraded by CSN1 and regulate ABI5 together with an NF-CY factor. ABA-mediating *ABI3, ABI5,* and *RGL2* regulate *MFT* by establishing a negative feedback loop to modulate the ABA and GA antagonism, in which, MFT also inhibits ABI5. A study indicated that auxin stimulates *ABI3* expression through ARF10 and ARF16 indirectly, which connects the ABA and auxin in seed germination regulation. In some ways, ABA inhibits ACO and ACS to influence ethylene synthesis negatively. Meantime, ETR1/2 and histone deacetylation cofactors SNL1/2 mediate the antagonism between the ABA and ethylene to involve seed germination, in which some ERF factors are involved. BR promotes seed germination as well as inhibits BIN2, which interacts with ABI5 and positively regulates ABA responses during seed germination and post-germination. Activated and repressive effects are shown by arrows and bars, respectively.

MOTHER OF FT AND TFL1 (*MFT*) genes, encoding phosphatidylethanolamine-binding proteins regulate germination in many species by the ABA-mediated pathway. *ABI3, ABI5,* and *RGL2* regulate *MFT* by establishing a negative feedback loop in the ABA signaling pathway to modulate the ABA and GA signaling and to stimulate embryo growth during germination in Arabidopsis (Fig. 4) [173]. *ABI4* reduces *MFT* gene expression through its effect on ABA, which promotes *MFT* itself expression, all these indicate the feedback between MFT and ABA signaling [173]. However, later studies revealed that *AtMFT* inhibits the germination in freshly mature seeds, while reduces the dormancy in after-ripened seeds [174]. In wheat, *TaMFT* acts as a repressor for seed germination, and a high level of *TaMFT* expression is correlated with a low germination index. In rice, the *OsMFT2* gene plays a function in the regulation of seed germination through interacting with OsbZIP23/66/72 and the ABA-mediated pathway [175]. The above results indicate that MFT may regulate different seed developmental stages with diverse mechanisms and through participating in the antagonism between ABA and GA.

The phytochrome A (PHYA) and PHYB mediated photo-signal are important for seed dormancy and germination regulation, in which PHYTOCHROM-INTERACTING FACTOR1 (PIF1) plays a downstream and vital role. Previous work has shown that *ABI3* expression is induced under PHYB and, in turn, ABI3 controls expression of ABA-response related genes including ABI5 [176]. Whereas under light conditions it activated by PHYA, the pattern of expression of *ABI4* is opposite to that of *ABI3* and *ABI5* in both Arabidopsis seed dormancy [177,178] and *Aethionema arabicum* light-dependent seed germination [179]. Further study indicated that ABI4 promotes PHYA-dependent germination and inhibits ABA accumulation and *MFT* gene expression in *Arabidopsis* [180]. Interestingly, PIF1 is shown to regulate GIBBERELLIN 3-OXIDASE2 (GA3OX2) and GA content. Reciprocally, PIF1 inhibits the transcriptional activity and DNA-binding ability of REVEILLE1 (RVE1), while, RVE1 stimulates the PIF1 DNA binding capability to modify ABI3 expression. As a result, PIF1 and RVE1 coordinately work as a feedback loop to regulate seed germination [181], which is also achieved dependent on the antagonism between ABA and GA. The PHYTOCHROME INTERACTING FACTOR 3-LIKE 5 (PIL5), a basic helix loop helix, exhibits a significant function in germination through phytochrome [182]. In *pil5* mutant, the expression of ABA and GA metabolism genes was disturbed meanwhile a defect in GA signaling was also detected. Moreover, the PIL5 acts as an RNA binding protein, activated through phytochrome, to influence the ABA and GA metabolism by directly activating the *SOMNUS* (*SOM*) gene transcription [183,184]. Both bHLH transcription factors, SPATULA (SPT) and PIL5 inhibit GA synthesis genes such as *GA3OX1/2* and directly activate the GA catabolism gene (*GA2ox2*), thereby preventing germination [174,185,186]. Furthermore, SPT controls the germination by repressing the expression of *ABI4* and *RGA* and promoting the expression of *ABI5* and *RGL3* [174]. However, PIL5 deactivates the ABA catabolism gene (*CYP707A2*) and positively regulates ABA biosynthesis genes (*ABA1*, *NCED6/9*) [19] to inhibit the germination, which suggests that PIL5 and SPT functions as a crosslink and fundamental hub during the antagonism of ABA and GA in the context of light condition.

Additionally, DOG1 functions downstream of PIL5 and increased expression of it inhibit the GA biosynthesis and activate

ABI3 and ABI5 to control seed dormancy and germination [187]. A study demonstrated that a Dof-type transcription factor DOF AFFECTING GERMINATION 1 (DAG1) functions downstream of PIL5 to inhibit the synthesis of GA by binding with the promoter of *GA3OX1*, meanwhile, the *dag1* mutant exhibited the up-regulated expression of ABA catabolism gene and down-regulated expression of ABA biosynthesis genes [186]. The above results indicate that the antagonistic role of ABA and GA during seed germination is also regulated by some factors activated by light signals such as PIF1 and PIL5 exemplifying that seed germination is also regulated partially by light-mediated pathway.

Besides GA, ABA also interacts with ET by the regulation of important ET biosynthesis and signaling genes such as *1-AMINOCYCLOPROPANE-1-CARBOXYLIC OXIDASE* (*ACO*) and *1-AMINOCYCLOPROPANE-1-CARBOXYLIC ACIDSYNTHASE* (*ACS*), and *ETHYLENE RESPONSE FACTOR 11* (*ERF11*) to regulate the ABA-ET mediated seed ripening [132,188–191]. Mutation in *ERA3* (*enhanced response to ABA3*) belonging to *ETHYLENE INSENSITIVE2* locus showed increased sensitivity to ABA, which illustrated that ET is a negative regulator of ABA [192,193]. In tomato, the ethylene response factor (ERF) Pti4 is involved in the regulation of seed germination by mediating ABA synthesis and signaling positively [194]. In addition, *ETR1/2* and *SNL1/2* regulate the ABA-ET crosstalk between dormancy and germination [115,131]. Thus, the crosstalk between ABA and ET is also important in maintaining the hormonal level of each other for finalizing decisions on dormancy and germination (Fig. 4) [195,196].

Glucose-6-phosphate dehydrogenase (G6PDH) plays a key role in reactive oxygen species (ROS) scavenging as the supply of NADPH. A study found that a null mutant *g6pd5* is more sensitive to ABA during seed germination, whereas over-expression of G6PD5 showed hyposensitive to ABA compared to WT. Furthermore, it is found that G6PD5 restrain the expression of ABI5 to repress the ABA signaling in seed germination [197]. GLUTATHIONE S-TRANSFERASE (GST) plays pivotal roles in redox associated processes, metabolism, and detoxification in plants. AtGSTU7, a member of GST, whose null mutant (*atgstu7*) showed hyposensitivity to ABA in germinating seeds dependent on ABI3 [140]. These indicate a potential correlation between ROS and ABA in seed germination regulation. Moreover, a study reported that phytohormone salicylic acid (SA) together with hydrogen peroxide (H_2O_2) up-regulated transcription of both the GA biosynthesis gene *ZmGA20ox1* and the ABA catabolism gene *ZmCYP707A2*, while down-regulated the expression of the GA catabolism gene *ZmGA2ox1* [198], indicating the interplay among SA, ROS, ABA and GA.

Furthermore, ABA works antagonistically with auxin to regulate developmental processes and to contribute to the survival of seeds [199,200]. For instance, the core ABA signaling gene ABI3 is an auxin-regulated, ABRE-based transcription factor that plays important role in seed dormancy [152]. An intensive study showed that through recruiting auxin responsive factors 10 (ARF10) and ARF16 with ABI3, auxin regulates the seed dormancy in synergy with ABA [201]. Brassinosteroids (BRs) play a critical antagonistic function in the seed germination inhibition of ABA [202,203]. The advanced study showed that Glycogen Synthase Kinase 3-like kinase BRASSINOSTEROID INSENSITIVE2 (BIN2), a critical repressor of BR signaling, interacts with ABI5, and functions upstream of ABI5 to positively regulate ABA responses during seed germination and post - germinative growth. Accordingly, BRs repress the BIN2-ABI5 cascade to antagonize ABA-inhibited seed germination and seedling establishment (Fig. 4) [204]. All these progresses uncover some mask of the interaction of ABA and other hormones (e.g., SA, auxin, BR) in the seed development.

Concluding remarks and future prospects

ABA is the most important hormone and shows versatile roles in seed development as well as the seed germination. Plants synthesize their ABA through indirect pathways in embryo and endosperm during seed development which accumulates continuously in seed late maturation. For many years, the functions of ABA have been studied comprehensively, in which metabolism and signaling pathways were focused to understand the regulation of different traits in seed development. Some important proteins involved in the different stages of ABA metabolism were identified (e.g., ZEP, ABAs, NCEDs, AAOs, and CYP707As), most of which usually display specific roles in special developmental stages, but how they are regulated differentially and specifically is unknown. Moreover, lots of downstream signaling-related genes were identified, in which some PYR/PYLs, PP2Cs, SnRK2s, and components in LAFL hub show vital roles in multiple developmental stages of seed. Firstly, PYR/PYLs are responsible to accept the ABA signal redundantly. After that, the PP2C activity is inhibited by dephosphorylation to activate downstream SnRK2s proteins and several other transcription factors including ABIs and bZIPs that play crucial roles in many processes during seed development such as accumulation of seed storage products, seed maturation, seed de-greening, desiccation-tolerant acquirement, maintenance and induction of primary dormancy and germination (Table 1). The finding of the new ABA signaling terminator – ABT further enriched the understanding of the ABA signaling pathway. So, both ABA synthesis and signaling all show very complicated networks in plant development. Here, we systematically summarize the updated progresses in ABA synthesis and ABA signaling regulation, as well as their interaction in seed, which still needs much work to explore the detailed regulators and intrinsic mechanisms.

Although ABA shows versatile roles in plant development, we focus on the biological roles and underlying mechanisms of ABA in seed-related straits. In seed development, dormancy is a decisive factor influencing seed vigor and plant propagation. Alteration in the state of dormancy (dormant to non-dormant) is an active process that involves variations in the expression of genes in after-ripening dry seeds, in this period, ABA-associated ways play crucial roles. The level of dormancy is severely poor in ABA biosynthesis and signaling mutants, indicating the direct and important function of ABA in seed dormancy maintenance and induction. Both ABA catabolism and stability between ABA-GA crosstalk both put an impact on the level of seed dormancy. So, the exploration of the detailed interaction between ABA and GA could also facilitate the illumination of mechanisms of ABA in seed development including dormancy. From numerous studies, it is proved that ABA activates some key proteins (e.g., ABI3, LEC1, LEC2, and FUS3) comprising a LAFL hub that plays roles in ABA metabolism, showing the reciprocal effect between ABA signaling and syntheses, but, the complete work model of these proteins is not clear yet. More comprehensive biochemical and genetic analysis for the key genes would be helpful for the elucidation of the detailed interplay of ABA synthesis and signaling in seed development.

The crosstalks between ABA and other phytohormones such as GA, ET, and auxin are also important for finalizing decisions of the seed dormancy, germination, or seedling establishment [133]. Studies about light signal-related factors (e.g., HY5 and PIF1) have provided some clues for interpretation of the interaction between ABA and exogenous signal in seed development. Here, we provide a comprehensive and updated network for crosstalks between ABA and other phytohormones in seed development, which indicates the bona fide case that it is common of pleiotropism and the reciprocal regulation between different factors or signals. But, how the

Table 1
Associated ABA metabolism (synthesis and catabolism) and response genes in this paper.

Gene Name	Protein/Enzyme	Mutant	Mutant phenotype	Specie	Reference
ABA Biosynthesis					
ABA1	Zeaxanthin epoxidase (ZEP)	*aba1, vp2/5/7/9*	Reduced dormancy	*Arabidopsis*, Maize, Tobacco	[104]
ABA2	Short-chain dehydrogenase reductase (AB-SDR)	*aba2*	Reduced dormancy	*Arabidopsis*	[31,37]
ABA2	Zeaxanthin epoxidase (ZEP)	*aba2*	Reduced dormancy	Tobacco	[37]
VP14	9-cis Epoxycarotenoid dioxygenase	*vp14*	Reduced dormancy	Maize	[35,205]
NCED	9-cis Epoxycarotenoid dioxygenase	*nced1-9*	Reduced dormancy	*Arabidopsis*, Bean, *Brachypodium distachyon*	[44]
AAO3	Aldehyde oxidase 3	*aao3-1*	Slightly reduced dormancy	*Arabidopsis*	[47]
ABA Catabolism					
CYP707A2	ABA 8′-hydroxylase	*cyp707a2-1/2*	Enhanced dormancy	*Arabidopsis*	[27]
CYP707A1	ABA 8′-hydroxylase	*cyp707a1*	Enhanced dormancy	*Arabidopsis*	[28,206]
ABA signaling components					
PYR, PYL/ RCAR	ABA receptors	*Pyls*	Reduced dormancy and ABA sensitivity	*Arabidopsis* , Rice	[29,30]
ABI1	Protein phosphatase 2C	*abi1-1*	Reduced dormancy and ABA sensitivity	*Arabidopsis*	[207–209]
ABI2	Protein phosphatase 2C	*abi2-1*	Reduced dormancy and ABA sensitivity	*Arabidopsis*	[209–211]
AHG1	Protein phosphatase 2C	*ahg1-1/2/3/4/5*	Enhanced dormancy and ABA sensitivity	*Arabidopsis*	[54,55]
AHG3	Protein phosphatase 2C	*Ahg3-1/2*	Enhanced dormancy and ABA sensitivity	*Arabidopsis*	[212,213]
SnRK2s	Protein kinase	*snrk2.2, 2.3, 2.6 triple mutant*	Green seed, Reduced dormancy and ABA sensitivity	*Arabidopsis*	[214,215]
Transcription factors					
ABI3/VP1	B3 domain	*abi3-1 to 17, vp1*	Green seed, Reduced dormancy and ABA sensitivity	*Arabidopsis*, Rice, Maize	[60,216,217]
ABI4	ERF/APETALA domain	*abi4-1*	ABA insensitive	*Arabidopsis*	[75,218]
ABI5	ABF, bZIP	*abi5-1/7/8*	ABA insensitive	*Arabidopsis*	[60,219]
FUS3	B3 domain	*fus3-3/8*	Reduced dormancy	*Arabidopsis*	
LEC1	B3 domain	*lec1-1/2*	Reduced dormancy	*Arabidopsis*	[68,220]
LEC2	B3 domain	*lec2-1/3*	Reduced dormancy	*Arabidopsis*	[26,220]

temperature, humidity and other exogenous factors (e.g., oxygen, calcium, and pollutants) influence the ABA/GA balance or the interaction between ABA and other hormones is still in a blank.

During post-germination growth, ABI3 activated by ABA activates JMJ30 and accelerates SnRK2.8 expression through H3K27me3 demethylation, promoting downstream *ABI3* expression to enhance post-germination growth as a feedback loop, which provides some hints to reveal the relationship of ABA and epigenetic modification machinery to improve the network of ABA in seed straits as well as shed light for the regulation of ABA pathway in genomic level. Due to the technology limitation of ABA visualization, the regulation of ABA metabolism has not been well studied, especially at cellular and tissue levels. Along with the advance of technology, uncovering the profiles of GA and ABA in different tissues during seed development would provide direct evidence for the antagonism between them. Compared to fresh harvest seeds, the seeds after-ripening or stratification display decreased dormancy and increased seed germination, how activation of GA biosynthesis impels the break of dormancy is still an open question during this period. Expectedly, the low temperature and high humidity play some roles in GA activation, but the underlying factors and mechanisms are still unclear.

Collectively, although great progress of roles of ABA in understanding the regulation of seed development has been done, some open questions remain unanswered. For example, it is still unclear how ABA regulates downstream genes such as components of LAFL, and the function of ABA signaling factors in response to ABA against dormancy. Many genes influenced dormancy, but, it is unknown how these genes interact with each other in detail. In the future, studies should be focused on the questions discussed above to improve the understandings of the mechanisms by which ABA and genetic factors regulate and maintain seed dormancy and germination, which would be conducive to a better presentation of the system of ABA function and continuous agricultural productivity.

Compliance with Ethics Requirements

This article does not contain any studies with human or animal subjects.

Declaration of Competing Interest

All the authors in the manuscript have no conflicts of interest.

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Author's contribution

Z.W. and F.L. conceived the manuscript; F.A, G.Q, and Z.W. drafted the manuscript and prepared the figures. All authors revised and approved the final manuscript.

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