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REVIEW

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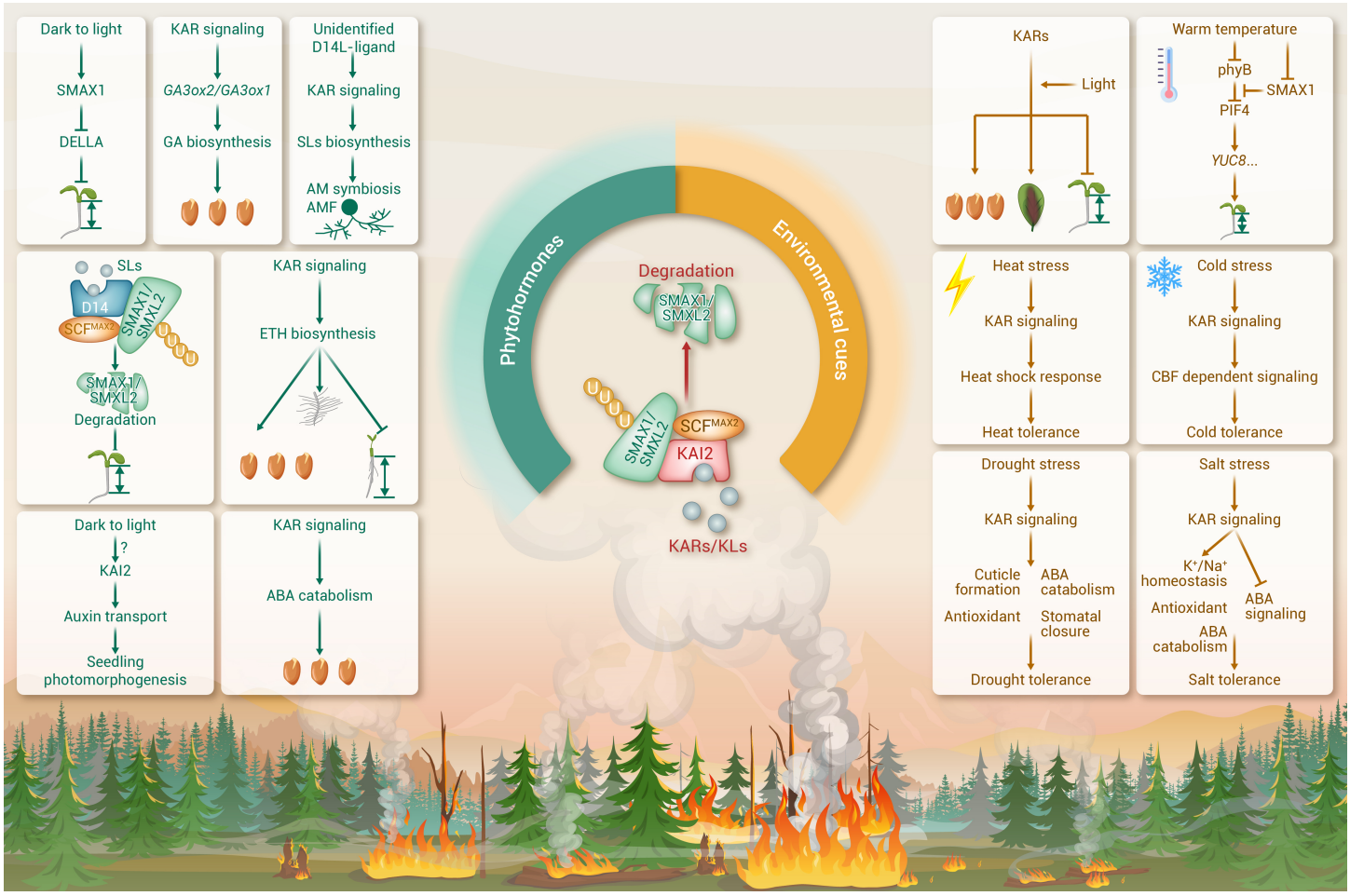
GRAPHICAL ABSTRACT



PUBLIC SUMMARY

- Karrikins (KARs) are a class of butenolide compounds derived from smoke following vegetation burning.
- KARs mimic an unknown phytohormone to regulate many aspects of development and environmental adaptation.
- KAR signaling is perceived and transduced to trigger transcriptional reprogramming by one pathway.
- KAR signaling interacts with many phytohormones and environmental signals.

REVIEW

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Interactions of the karrikin signaling pathway with phytohormones and environmental signals in plants

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Karrikins (KARs), a class of butenolide compounds discovered in the smoke of burned vegetation, can mimic an unidentified phytohormone to regulate plant development and responses to environmental cues. The KAR signaling pathway is mediated by the 26S proteasome. Upon perception of KARs, the receptor of KARs, KARRIKIN INSENSITIVE 2 (KAI2), recruits the F-box protein, MORE AXILLARY GROWTH 2 (MAX2), and the repressor proteins, SUPPRESSOR OF MAX2 1 (SMAX1) and SMAX1-LIKE 2 (SMXL2), leading to the degradation of SMAX1 and SMXL2, which then reprograms the transcription of the KAR-responsive genes. This review focuses on the latest research progress regarding KAR signaling transduction, the identification of KAI2 ligands, and the crosstalk between KAR signaling, plant hormones, and environmental cues, providing new insights for future studies and applications in agriculture.

INTRODUCTION

The discovery and research of phytohormones have substantially advanced our understanding of plant development and responses to environmental cues. Since auxin was discovered in 1928, more phytohormones have been identified, including gibberellin (GA), cytokinin (CK), ethylene (ETH), abscisic acid (ABA), brassinosteroid (BR), salicylic acid (SA), jasmonic acid (JA), strigolactones (SLs), and peptide hormones.[1] Recently, a class of butenolide compounds was identified in the smoke from burned vegetation and named karrikins (KARs) (Figure 1A), as they promote seed germination of fire-following plants after a wildfire.[2-4] Interestingly, increasing evidence has shown that KARs mimic a class of unknown endogenous signaling molecules, regulating development and responses to environmental cues in plants.[5-7] Furthermore, this kind of signaling molecule is probably an unidentified phytohormone.[5,8] Therefore, dissecting the mechanism of KAR signaling will provide new insights into plant development and environmental adaptation.

KARs have existed in nature for a long time, but they were discovered only in the last 20 years. In some Mediterranean climate areas, such as Australia, California, and South Africa, wildfires are prone to occur in summer.[9-12] In the mid-twentieth century, seeds of many native plant species in these areas were found to germinate quickly after wildfires.[9-12] Until 1977, burned wood was found to stimulate the germination of *Emmenanthe penduliflora*, a fire-following plant.[13] In 1990, de Lange and his colleagues reported that smoke from burning plants could promote the seed germination of *Audouinia capitata*, a fynbos species.[14] Next, numerous studies tried to isolate and identify the compounds in smoke that stimulate seed germination of fire-following plants. In 2004, one kind of butenolide, 3-methyl-2*H*-furo[2,3-*c*]pyran-2-one, was isolated and confirmed to be a major compound in smoke that promoted seed germination in a wide range of plant species.[15] To distinguish it from other butenolides, this compound was named "karrikinolide". Subsequently, karrikinolide and its analogues are also known as "karrikin". In "karrikinolide" or "karrikin", "karrik" means smoke. The first identified karrikinolide was abbreviated as KAR_1 (Figure 1B).[16,17] To date, five KAR_1 analogs have been discovered in smoke water and are named as KAR_2 to KAR_6 (Figure 1B).[3,17,18] Although the KARs all consist of a butenolide ring fused with a pyran ring, they differ in their methyl substitutions (Figure 1B). As a result, the effects of KARs vary from species to species. For instance, KAR_2 is the most active KARs in stimulating *Arabidopsis* seed germination, but it is less active than KAR_1 in other species.[17,19,20] Moreover, in rice, both KAR_1 and KAR_2 exhibit similar effects in inhibiting the elongation of the mesocotyls.[21] Besides stimulating seed germination, KARs also regulate photomorphogenesis, leaf morphology, root development, symbiosis with arbuscular mycorrhizal (AM) fungi, drought and salt tolerance, and other processes.[22-36]

In this review, we summarize the current progress on the KAR signaling pathway, the identification of the KAI2 ligands, and the crosstalk between KAR signaling, plant hormones, and environmental signals. We also provide an outline of prospects for future studies in this field. Furthermore, we will discuss the application potential of KARs in agriculture.

KAR SIGNALING PATHWAY

KARRIKIN INSENSITIVE 2 (KAI2), also known as HYPOSENSITIVE TO LIGHT (HTL),[37,38] has been identified as the receptor for KARs in *Arabidopsis*.[39,40] KAI2 can bind KAR_1 and $GR24^{ent\text{-}5DS}$, the enantiomer of the synthesized SLs analog $GR24^{5DS}$, exhibiting KAR activity, *in vitro*.[39-42] KAI2 belongs to the α/β hydrolase superfamily and exhibits weak hydrolase activity.[42,43] Its core domain consists of a seven-stranded β-sheet and seven α-helices that form a hydrophobic pocket. Two V-shaped α-helices comprise the cap of this pocket. Three conserved residues (S95, D217, and H246) form the catalytic triad and locate at the base of the pocket for substrate binding.[39,43] Mutating S95 to A95 could abolish the binding activity of KAI2.[42,44] Notably, KAI2 undergoes ligand-induced degradation, and this process is dependent on S95.[45] These data indicate that the S95 mediated binding and hydrolase activities are crucial for the ligand induced degradation of KAI2. Due to the importance of the catalytic triad, its nearby residues not only affect the binding and hydrolase activities but also determine KAI2's preference for substrates.[43,46] In addition, interior residues of the core domain usually have an impact on the stability of KAI2.[44]

The surface residues of KAI2 protein also play an important role in regulating its activity. In *Arabidopsis*, the null mutant *kai2-2* is insensitive to KAR_1, KAR_2, and $GR24^{ent\text{-}5DS}$ treatments and exhibits enhanced seed dormancy, elongated hypocotyl, and epinastic cotyledon phenotypes.[38,42,47-49] As another observation, *kai2-10*, which contains a KAI2 protein with a D184N mutation, shows a comparable phenotype to *kai2-2*,[44] indicating that this mutation disrupts the signaling activity of KAI2. D184 is situated at an extremity of the lid domain and is exposed to the solvent.[44] Interestingly, KAI2 D184N retains the substrate binding and hydrolysis activities, and is also induced for degradation by KARs treatment.[44] These observations can be interpreted that the substrate hydrolysis and degradation of KAI2 can be dissociated from its signaling activity.[44] However, considering that surface residues usually play a crucial role in recruiting interacting proteins, these observations are probably due to the fact that D184N affects the interactions between KAI2 and its partners, thereby blocking KAR signaling. Recently, a study of recombinant proteins from *Physcomitrium patens* highlights residues that are important or essential for KAI2 protein activity. More importantly, they have shown that some of the PpKAI2-like proteins can bind KAR_1 but are unable to complement the *Arabidopsis kai2* mutant.[50] This is probably due to PpKAI2-like proteins being unable to associate with other components of the KAR signaling pathway, thus failing to activate downstream signaling.

In *Arabidopsis*, *MORE AXILLARY GROWTH2* (*MAX2*), the orthologue of *DWARF 3* (*D3*) in rice,[51] encodes an F-box protein and positively regulates KAR signaling.[52,53] MAX2 interacts with S-PHASE KINASE-ASSOCIATED

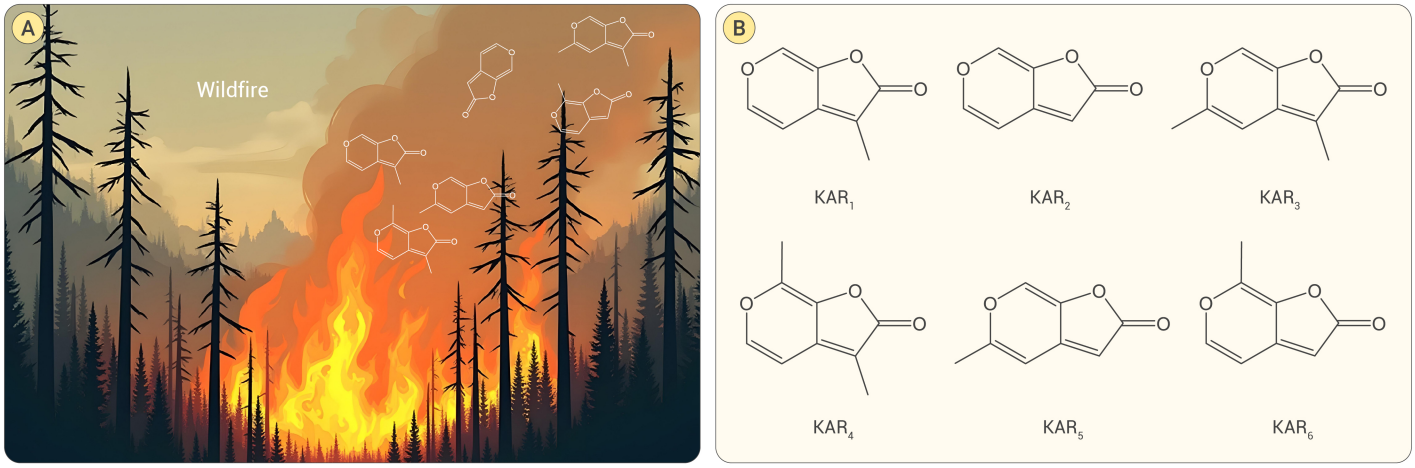

Figure 1. Structures of KARs (A) The schematic diagram of KARs production from vegetation burning during a wildfire.[2-4] (B) Structures of smoke-derived KARs. KAR_1-KAR_6 all consist of a butenolide ring fused with a pyran ring, differing in their methyl substitutions.[3,17,18]

PROTEIN 1 (SKP1) and Cullin proteins to form the E3 ligase complex, which recruits and ubiquitinates target proteins.[54-58] Similar to the *kai2* mutant, the *max2* mutant also shows KAR insensitivity, enhanced seed dormancy, elongated hypocotyl, and epinastic cotyledon phenotypes.[33,38,52,53,59-61] The crystal structure analysis has shown that the N-terminus of D3 contains a conserved F-box motif, which is the interface for the *Arabidopsis* SKP1-LIKE PROTEIN (ASK1). The C-terminus of D3 consists of 20 LEUCINE-RICH REPEATs (LRRs), and the C-TERMINAL α-HELIX (CTH), a part of the LRR 20, exhibits a dynamic topology and is able to switch between engaged and dislodged states.[62-64] Yoshimulactone Green (YLG), a fluorogenic ligand of KAI2, can be used to monitor the hydrolase activity of KAI2.[63] The YLG hydrolysis assay shows that the dislodged recombinant D3 protein inhibits the hydrolase activity of KAI2 *in vitro*. Meanwhile, in *Arabidopsis*, mutations in the CTH domain of MAX2 disrupt its function in regulating seed germination, hypocotyl elongation, and root hair development,[63,64] indicating that the CTH domain plays a role in KAR signaling by regulating the hydrolase activity of KAI2. Moreover, *max2* mutants exhibit an increased branching phenotype.[38,52-55,58,60] This is because MAX2 is also a core component in the SL signaling pathway.[38,52-55,58,60]

The KAR signaling repressor protein SUPPRESSOR OF MAX2 1 (SMAX1) was identified through a genetic screen for suppressors of the *max2* mutant.[33] In *Arabidopsis*, SMAX1 shows weak similarity to HEAT SHOCK PROTEIN 101 (HSP101). Moreover, it belongs to a protein family consisting of eight members: SMAX1 and SMAX1-LIKE 2 (SMXL2), 3, 4, 5, 6, 7, and 8.[33] Based on their function, they are classified into four subclades: SMAX1/SMXL2, SMXL3, SMXL4/5, and SMXL6/7/8.[65,66] SMXL2 is the closest paralog of SMAX1 and exhibits functional redundancy with SMAX1 in regulating hypocotyl elongation. *smax1 smxl2* double mutants effectively suppress hypocotyl elongation phenotype of *max2*.[33,61] SMAX1/SMXL2 are located in the nucleus and contain at least one conserved ethylene-responsive element binding factor-associated amphiphilic repression (EAR) motif, which is essential for the interaction with the transcriptional corepressor proteins TOPLESS (TPL) and TPL RELATED (TPR).[60,67] Therefore, SMAX1/SMXL2 display transcriptional repression activity and regulate the transcription of downstream genes.[47,60,68]

A recent study has found that non-hydrolysable ligands of KAI2, carba-dMGers, when bound to KAI2, fail to induce the interaction between KAI2 and SMAX1.[69] It is therefore speculated that KARs are metabolized into bioactive molecules, which are then hydrolyzed by KAI2, thereby activating the KAR signaling pathway. Meanwhile, KAI2 is targeted for degradation in response to induction by KARs (Figure 2);[44,45,70] this process may be crucial for shutting down the KAR signaling, rather than activating the downstream signaling. However, whether the hydrolysis of KARs is coupled with the degradation of KAI2 remains unclear. Next, KAI2 probably undergoes a conformation change and recruits MAX2 and SMAX1/SMXL2, leading to ubiquitination and degradation of SMAX1/SMXL2 (Figure 2).[7,39,57,67,70,71] In this process, the engaged MAX2 could accelerate the degradation of SMAX1/SMXL2.[63] Given that SMAX1 shows EAR motif-dependent transcriptional repression activity and binds to the promoters of downstream genes such as *SMAX1* and (*GIBBERELLIN 3-oxidase 2*) *GA3ox2* (Figure 2),[68] the degradation of SMAX1/SMXL2 will upregulate the expression of a considerable number of their downstream genes.[30,33,38,52,72] Notably, a recent study has revealed that SMAX1/SMXL2 interact with PHYTOCHROME INTERACTION FACTOR 4 (PIF4) and PIF5 (PIF4/5), which enhance the stability of PIF4/5 proteins by interrupting the interaction between phytochrome B (phyB) and PIF4/5. Through this mechanism, SMAX1/SMXL2 promote the expression of PIF4/5-targeted KAR-repressed responsive genes such as *INDOLE-3-ACETIC ACID INDUCIBLE1 29* (*IAA29*) and *SMALL AUXIN UPREGULATED RNA 57* (*SAUR57*) (Figure 2).[47] These observations indicate that the EAR motif independent action of SMAX1 plays a key role in regulating the transcription of downstream genes. Indeed, the expression of a large proportion of SMAX1 downstream genes, such as *KARRIKIN UP-REGULATED F-BOX 1* (*KUF1*), is regulated by the EAR motif-dependent action of SMAX1.[47] It is therefore speculated that SMAX1 recruits unknown transcription factors to regulate their expression (Figure 2). Collectively, although great progress has been made in understanding the mechanism of KAR signaling, the events downstream of SMAX1/SMXL2 are still largely unknown.

KAI2 ligands

Although KAI2 binds to KARs as shown by ligand-binding assays and crystal structure analyses *in vitro*,[39,40] considerable evidence clearly suggests that KAI2 recognizes an unknown endogenous signal, referred to as the KAI2 ligands (KLs). Firstly, in *Arabidopsis*, crude extracts from leaves can activate the expression of KAR-responsive gene, *D14-LIKE 2* (*DLK2*), in a KAI2-dependent manner.[6] Secondly, as mentioned above, KAI2 is an α/β hydrolase and contains a catalytic triad that is essential for the function of KAI2 in regulating KAR signaling,[42,45,67,73] suggesting that KAI2 retains the ability to hydrolyze substrates. Thirdly, *kai2* and *max2* mutants are insensitive to KARs treatment and show the phenotypes opposite to those observed with KARs treatment, indicating that KAI2 and MAX2 perceive an endogenous signal.[38,52] Fourthly, differential scanning fluorimetry (DSF) assays have shown that KAI2 specifically binds $GR24^{ent\text{-}5DS}$, but not KAR_1 or KAR_2.[42] Furthermore, compared with $GR24^{ent\text{-}5DS}$, KAI2 shows a preference to desmethyl-$GR24^{ent\text{-}5DS}$ ($dGR24^{ent\text{-}5DS}$) (Figure 3A), but DWARF 14 (D14), the homolog of KAI2 and the receptor of SLs,[38,62,64,74-76] does not show this preference for $GR24^{5DS}$ and $dGR24^{5DS}$ (Figure 3B).[70] These data suggest that KAI2 binds an endogenous substrate that has a different structure and biosynthetic pathway from SLs. Fifthly, $GR24^{ent\text{-}5DS}$ induces the degradation of SMXL2 faster than KAR_1,[57] implying that KAR_1 may be metabolized to KLs before being perceived by KAI2. Supporting this observation, KUF1 is speculated to regulate the metabolism of KAR_1, as its loss-of-function mutant, *kuf1*, exhibits a short hypocotyl phenotype and specific hypersensitivity to KAR_1 treatment.[72]

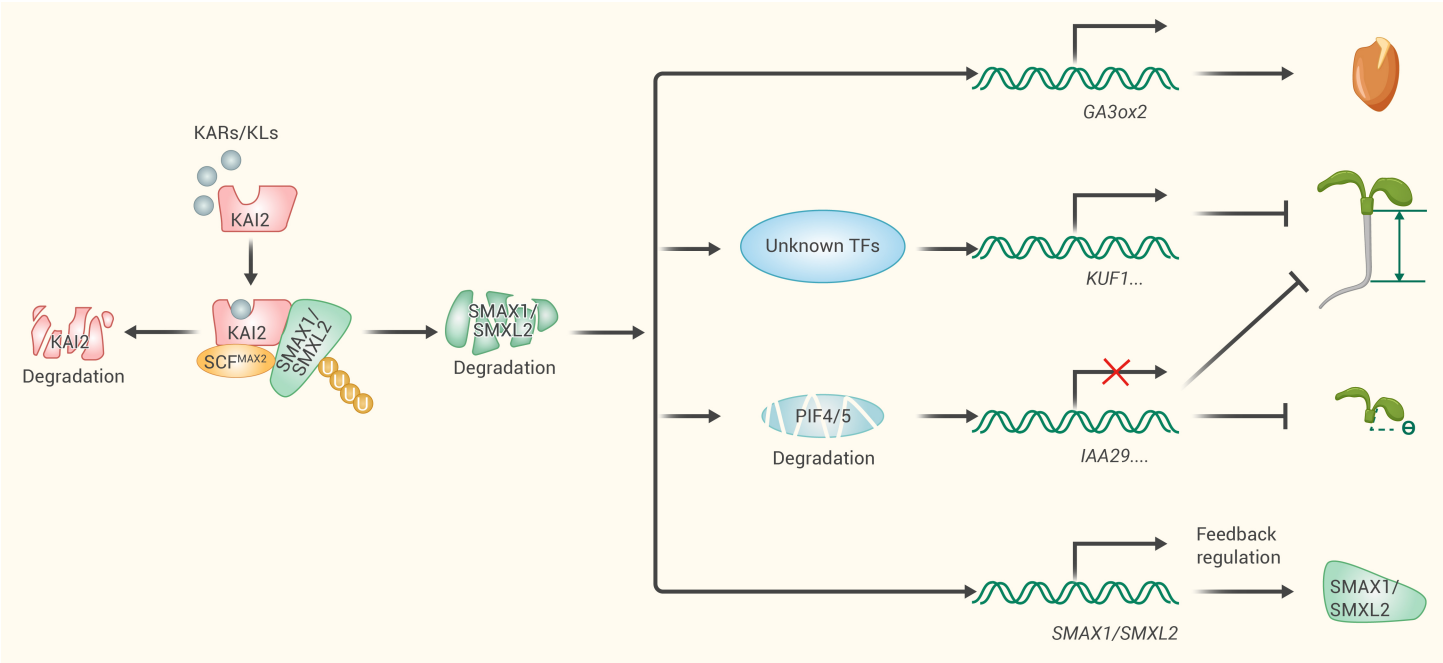

Figure 2. A proposed model of KAR signaling pathway in *Arabidopsis* In the presence of KARs/KLs, KARs/KLs are bound by KAI2, which promotes the degradation of KAI2 through an unknown mechanism.[44,45,70] On the other hand, KAI2 interacts with SCF^{MAX2} and SMAX1/SMXL2, thereby leading to the ubiquitination and degradation of SMAX1/SMXL2.[57,67] Along with the KARs/KLs induced-degradation of SMAX1/SMXL2, the expression of *GA3ox2* is enhanced by relieving the transcriptional repression of SMAX1 on its promoter, thereby promoting seed germination.[68] Meanwhile, the SMAX1/SMXL2-interacting transcription factors are released to upregulate the expression levels of target genes, such as *KUF1*, which inhibits hypocotyl elongation.[30,47] Furthermore, the degradation of PIF4/5 is enhanced by removing the repression of SMAX1/SMXL2 on the interactions between phyB and PIF4/5, thereby downregulating the expression levels of PIF4/5 target genes, such as *IAA29*, and inhibiting hypocotyl elongation and cotyledon epinasty.[47] Additionally, the expression level of *SMAX1*/*SMXL2* are upregulated to maintain the homeostasis of SMAX1/SMXL2 proteins by reducing the transcriptional repression exerted by SMAX1 on itself and SMXL2.[68] TFs, transcription factors; U, ubiquitin. Arrows represent positive regulation and bars indicate negative regulation.

Recently, an exciting advance was made in identifying candidate KLs. In *Petunia*, (−)-germacrene D, a kind of volatile organic compound that regulates stigma size (Figure 3C), is recognized by KAI2 to activate the KAR signaling in pistils.[77] However, (−)-germacrene D does not contain a butenolide moiety that is necessary for KARs activity (Figure 3C); thus, (−)-germacrene D may be further metabolized. Furthermore, whether (−)-germacrene D mimics the actions of KARs in regulating seed germination and plant development remains unknown. In addition, Sesquiterpene lactones (STLs) may be the candidates of KLs. Two sunflower STLs, 8-epixanthatin and tomentosin (Figure 3C), have been found to bind to sunflower KAI2 (HaKAI2s) with higher affinity than KAR_1 and KAR_2 in molecular docking assays.[78] Supporting this observation, upon perceiving the blue light, 8-epixanthatin and tomentosin briefly accumulate in the hypocotyl, implying that 8-epixanthatin and tomentosin regulate photo-inhibition of hypocotyl elongation in sunflower.[79] However, further experimental data should validate this result of the molecular docking assay, which is based on computational work. Therefore, more efforts are needed to identify KLs and their biosynthetic pathway. Given the potential functional redundancy of KL biosynthetic genes, developing a KAR-hypersensitive reporter is valuable for screening KLs biosynthesis mutants.

CROSSTALK BETWEEN KAR SIGNALING AND PHYTOHORMONES

KAR signaling and SLs

KARs/KLs and SLs regulate plant development and environmental responses through two closely related signaling pathways. As mentioned above, the receptor of SLs, D14, is a homolog of KAI2.[38] Upon binding to SLs, D14 hydrolyzes SLs into a covalently linked intermediate molecule (CLIM), undergoes a conformational change, then recruits MAX2 and SMXL6/7/8.[54,56,58,60,74-76,80-83] Therefore, MAX2 positively regulates KAR and SL signaling pathways through ubiquitinating SMXLs. Moreover, MAX2 and D3 mediate the SL-induced degradation of D14 in *Arabidopsis* and rice, respectively.[83-85] However, although KARs induce the degradation of KAI2, this process is independent of MAX2.[44,45,70] These observations indicate that MAX2 plays different roles in KAR and SL signaling pathways.

SMAX1/SMXL2 and SMXL6/7/8 act as the repressors in KAR and SL signaling pathways, respectively. Furthermore, KARs and SLs were considered to function through KAI2-MAX2-SMAX1/SMXL2 and D14-MAX2-SMXL6/7/8 complexes, respectively.[33,54,56,58,60,61,66] Interestingly, recent studies have revealed that $GR24^{4DO}$ and $GR24^{5DS}$, two SLs analogs, induce D14 interaction with MAX2 and SMAX1/SMXL2, leading to the ubiquitination and degradation of SMAX1/SMXL2, thereby inhibiting the hypocotyl elongation (Figure 4A).[57,86] Similarly, *rac*-GR24, a racemic mixture of GR24 that activates both SL signaling and KAR signaling, induces KAI2 interaction with SMXL7.[67] Although KAI2 and D14 are able to interact with SMAX1/SMXL2/6/7, KAI2 shows a preference for SMAX1/SMXL2, while D14 preferred SMXL2/6/7.[57,67,86] However, the molecular mechanism underlying KAI2 and D14 preference in recognizing SMXLs is unknown.

The crosstalk between KAR signaling and SLs biosynthesis has been found to coordinate the symbiosis between AM fungi and rice. During symbiosis, SLs have been identified as the inducers of AM hyphal branching, which is essential for the symbiosis between plants and AM fungi.[87] On the other hand, the orthologues of KAI2 in rice and *Brachypodium distachyon*, D14 LIKE (D14L) and BdKAI2, have been found to promote the colonization by AM fungi in the root.[26,29] Furthermore, SMAX1 functions downstream of D14L, negatively regulating colonization of AM in rice.[24] More importantly, SMAX1 decreases the SLs content in the rhizosphere by downregulating the expression of SLs biosynthesis genes, thereby inhibiting the symbiosis between rice and AM fungi (Figure 4B).[24] In consistency with this observation, the amount of SLs secreted by the *Ossmax1* mutant is higher than that secreted by the wild-type plant.[24] Interestingly, in *Arabidopsis*, a non-mycorrhizal plant, the KAI2 pathway does not affect the expression of SLs biosynthetic genes and the SLs content.[88] In addition, phosphorus starvation induces the symbiosis between plants and AM fungi by simultaneously elevating SLs biosynthesis and KAR signaling activity.[88-92]

KAR signaling and GA

KAR signaling interacts with GA to regulate seed germination and hypocotyl elongation. Physiological studies have shown that in *Arabidopsis*, KAR_1 is unable to stimulate seed germination in GA-deficient mutants such as the *ga requiring 1* (*ga1-3*) mutant and *ga3ox1 ga3ox2 double mutant*.[17] Furthermore, treatment with GA rescues the inhibited seed germination phenotype of *kai2* and *max2* mutants under weak light, while *smax1 smxl2* double mutants show higher GA content than wild-type plants and resis-

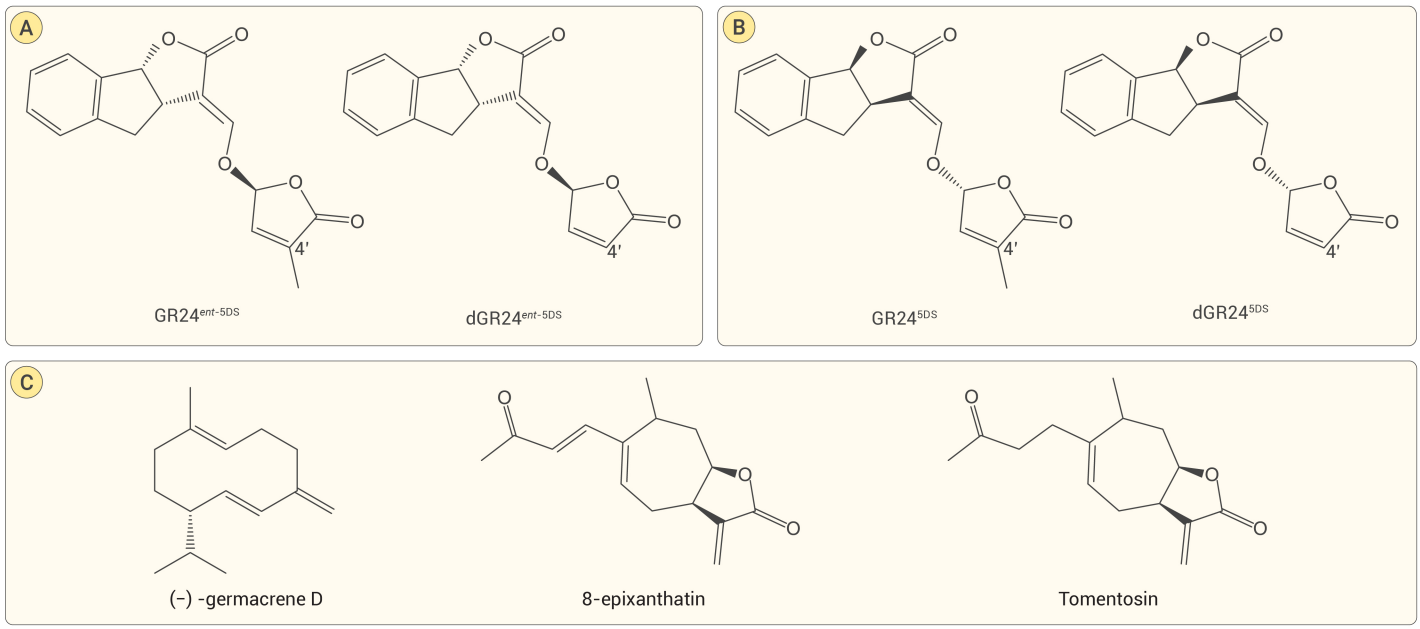

Figure 3. Structures of GR24 isomers and candidate KLs (A) Structures of the synthetic SL analogs GR24$^{ent\text{-}5DS}$ and dGR24$^{ent\text{-}5DS}$ showing KAR activity.[70] The butenolide ring of dGR24$^{ent\text{-}5DS}$ is demethylated at the C4′ position.[70] (B) Structures of the synthetic SL analogs GR24^{5DS} and dGR24^{5DS}.[70] GR24^{5DS} shows SL activity, while dGR24^{5DS}, a demethylated form of GR24^{5DS} at the C4′ position, does not exhibit SL activity.[70] (C) Structures of (−)-germacrene D, 8-epixanthatin and tomentosin.[77,78]

tance to GA biosynthesis inhibitor paclobutrazol (PAC) treatment during seed germination.[68] Transcriptional analysis has found that KAR_1 treatment upregulates the expression of *GA3ox1* and *GA3ox2* during seed imbibition (Figure 4C).[17] Correspondingly, in *pleiotropic long hypocotyl 2* (*ply2*), another KAI2 loss-of-function mutant, and in *max2* mutants, the expression level of *GA3ox1* is down-regulated.[41,93] Moreover, the expression level of the GA catabolic gene *GIBBERELLIN 2-OXIDASE 2* (*GA2ox2*) is up-regulated in *ply2* and *max2* mutants.[41,93] These observations indicate that KARs increase endogenous GA content by regulating the expression of GA biosynthesis and catabolism genes (Figure 4C & Figures 5A-B).

DELLA protein, which is the key negative regulator of GA signaling,[94] is another important node in the crosstalk between KAR and GA signaling. In *Arabidopsis*, DELLA proteins, including RGA-LIKE 1 (RGL1), RGL3, GAI, and RGA, interact with SMAX1 through their DELLA domains, forming a repressor complex that synergistically inhibits the expression of GA biosynthetic gene *GA3ox2*.[68] Moreover, DELLA proteins could enhance the transcriptional repression activity of SMAX1, thereby inhibiting seed germination under weak light (Figure 5B).[68] However, during the dark-to-light transition, the interactions between SMAX1 and DELLA proteins promote the degradation of DELLA proteins, thereby facilitating hypocotyl elongation (Figure 5C).[95] These data indicate that the effect of interactions between SMAX1 and DELLA proteins varies with the developmental stage transition, but the mechanism remains unknown.

KAR signaling and ABA

The interaction between KAR signaling and ABA has been noted in the regulation of seed germination. Firstly, in *Arabidopsis*, the ABA biosynthesis mutant *aba deficient 3* (*aba3*) exhibits a reduced response to KAR_1 compared with the wild-type during seed imbibition.[17] Meanwhile, in the wild-type, the seed germination response to KAR_1 was significantly decreased after treatment with 0.5 μM ABA, and the response was completely repressed after treatment with 1 μM ABA, indicating that exogenous ABA inhibits the KAR_1 response in a dose-dependent manner.[17] Moreover, in *Avena fatua*, KAR_1 treatment reduces the endogenous ABA content in embryos during seed germination.[96] Secondly, KAR_1 upregulates the expression of *CYTOCHROME P450 707A1* (*CYP707A1*) and *CYP707A3*, which encode ABA 8′-hydroxylases involved in ABA catabolism during seed imbibition (Figure 4C).[97,98] Correspondingly, in contrast to the wild-type, the expression of *CYP707A3* is downregulated in *htl-3*, another *kai2* mutant with elongated hypocotyls, and in *max2* mutants after 3 hours of seed imbibition, which probably leads to an increase in ABA content in these mutants.[98] However, after imbibition for 5 days, *kai2-2* shows a more sensitive phenotype to ABA treatment in *Arabidopsis*.[99] This observation may reflect the dynamic changes in ABA content during seed germination.

Notably, as mentioned above, KAR signaling positively regulates GA content by increasing GA biosynthesis and decreasing GA catabolism. Consequently, KAR signaling intensively downregulates the ABA to GA ratio, which is crucial for modulating seed germination,[100,101] by decreasing ABA content and increasing GA content (Figure 4C). Supporting this view, the transcriptome analysis shows that the expression of *CYP707A3* and *GA3ox1/2* is upregulated while the expression of *GA2ox2* is downregulated in *KAI2*-overexpressing seeds treated with KAR_2 under dark conditions.[102] However, under certain conditions, KAR signaling has an opposite influence on the ABA-to-GA ratio. In soybean, under shade conditions, the expression level of the ABA biosynthesis gene *GmAAO* is significantly upregulated in KAR-treated seeds, while the expression levels of GA-biosynthesis genes, such as *GmGA3ox1* and *GmKAO*, are downregulated, thereby inhibiting seed germination.[103]

KAR signaling and ETH

Recently, the crosstalk between KAR signaling and ETH was found to regulate root development. *ACC SYNTHASE 7* (*ACS7*), the gene encoding the rate-limiting enzyme in ETH biosynthesis,[104] is an important node in the crosstalk between KAR signaling and ETH. KAR_1 significantly upregulates the expression of *ACS7* in a KAI2-dependent manner in *Lotus japonicus*.[23] Meanwhile, the expression of *ACS7* is upregulated in *Ljsmax1* mutants, leading to a higher level of ETH in these mutants, thereby inhibiting the primary root growth while promoting the root hair elongation (Figure 4D).[23] Supporting this observation, treatment with an ACC biosynthesis inhibitor AVG could rescue the shortened primary root and the elongated root hair phenotypes of *Ljsmax1* mutants.[23] These data demonstrate that KAR signaling modulates root development by regulating ETH biosynthesis (Figure 4D).

In another study, under phosphorus starvation conditions, KAR signaling is enhanced, thereby increasing the activity of the ETH signaling pathway by upregulating the expression of *ACS7* and *ETHYLENE INSENSITIVE 2* (*EIN2*), and promoting root hair elongation (Figure 4F).[36] Notably, under these conditions, the shootward auxin transport from the root tip is strengthened. It is likely due to the fact that the localized accumulation of AUXIN TRANSPORTER PROTEIN1 (AUX1) is enhanced in the epidermis of the lateral root cap (LRC), above the LRC, root apical meristem, and root stele. Moreover, the localized accumulation of PIN-FORMED 2 (PIN2) is also increased in the

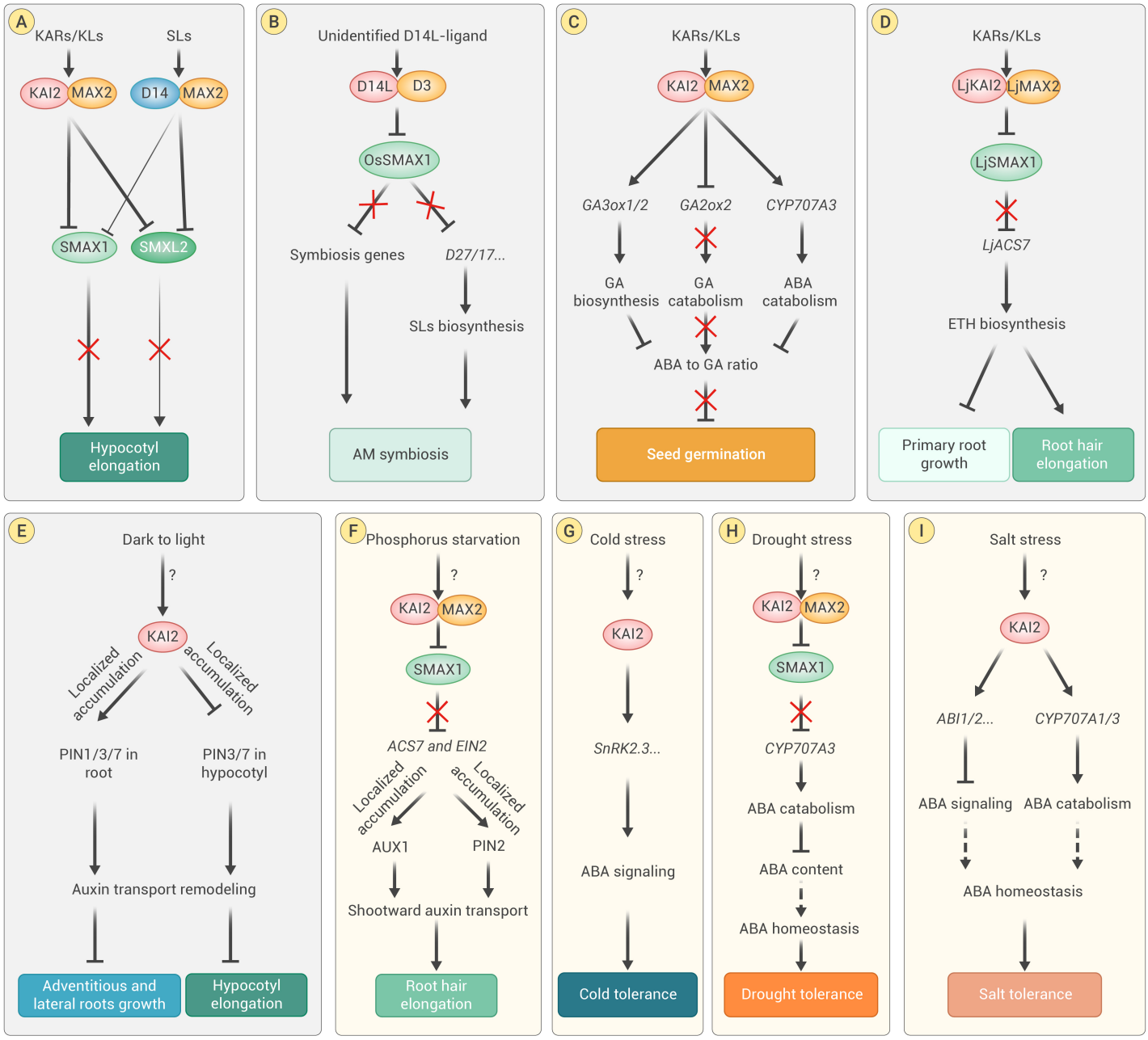

Figure 4. The interactions of the KAR signaling pathway with phytohormones (A) The crosstalk between KAR signaling and SLs in regulating hypocotyl elongation in *Arabidopsis*. In hypocotyl, KARs/KLs induce the association of KAI2 with MAX2 and SMAX1/SMXL2 to trigger the ubiquitination and degradation of SMAX1/SMXL2, thereby inhibiting hypocotyl elongation.[57] On the other hand, in the presence of SLs, D14 also associates with SMAX1and SMXL2, as well as with MAX2.[57,86] While the interaction between D14 and SMAX1 is weaker than that between D14 and SMXL2. These interactions also promote the ubiquitination and degradation of SMAX1/SMXL2, as well as the inhibition of hypocotyl elongation.[57,86] (B) The crosstalk between KAR signaling and SLs in regulating AM symbiosis in rice. During symbiosis between rice and AM fungi, an unidentified D14L-ligand induces the interaction of D14L with D3 and OsSMAX1, leading to the degradation of OsSMAX1. This process activates the expression of symbiosis genes and SLs synthesis genes, including *D27*, *D17*, and *D10*, thereby enhancing AM symbiosis.[24,88] (C) The interactions of KAR signaling with GA, as well as with ABA in regulating seed germination in *Arabidopsis*. KAR signaling enhances GA biosynthesis and ABA catabolism, and inhibits GA catabolism by respectively upregulating the expression of *GA3ox1/2* and *CYP707A3* and downregulating the expression of *GA2ox2*, thereby decreasing the ABA to GA ratio and promoting seed germination.[17,68,93,102] (D) The crosstalk between KAR signaling and ETH in regulating primary root growth and root hair elongation in *Lotus japonicus*. In roots, upon activation by KARs/KLs, LjKAI2 interacts with LjMAX2 and LjSMAX1, leading to the degradation of SMAX1. This process alleviates the transcriptional repression of *ACS7* expression, thereby increasing ETH levels, while inhibiting primary root growth and promoting root hair elongation.[23] (E) The crosstalk between KAR signaling and auxin in regulating adventitious and lateral roots growth and hypocotyl elongation in *Arabidopsis*. In hypocotyl and root, dark-to-light transition induces the expression of *KAI2* by an unknown mechanism. KAI2 orchestrates a rapid reorganization of the PIN-mediated auxin transport system. This involves a reduction in PIN3 and PIN7 levels in the hypocotyl, while increasing the abundance of PIN1, PIN3, and PIN7 in the root meristem, thereby remodeling auxin transport and inhibiting hypocotyl elongation, as well as adventitious and lateral root growth.[108] (F-I) The crosstalks between KAR signaling and abiotic stress involving plant hormones. (F) Phosphorus starvation elevates the expression of *KAI2* by an unknown mechanism. This upregulates the expression levels of *ACS7* and *EIN2*, resulting in enhanced ETH biosynthesis and signaling. The activated ETH signaling promotes shootward auxin transport in the root tip, thereby promoting root hair elongation.[36] (G) In Arabidopsis, drought stress activates the KAR signaling by an unknown mechanism, leading to decreased ABA content caused by upregulating the expression level of the catabolism gene *CYP707A3*, thereby enhancing drought tolerance.[27] (H) The expression of *KAI2* is induced by cold stress, which leads to enhanced activity of ABA signaling through upregulating the expression levels of ABA signaling genes, thereby enhancing cold tolerance.[129,131] (I) In Arabidopsis, salt stress induces the expression of *KAI2* by an unknown mechanism. Furthermore, the expression levels of ABA signaling negatively regulating genes *ABI1/2* and ABA catabolism genes *CYP707A1*/3 are upregulated, thereby inhibiting ABA signaling activity and increasing ABA catabolism, which enhances salt tolerance.[137] Arrows represent positive regulation and bars indicate negative regulation.

elongation and meristematic zones of the root (Figure 4F).[36] Consistent with these observations, treatment with 1-aminocyclopropane-1-carboxylic acid (ACC), an ethylene precursor, restores RH elongation in the *acs7* mutant, but not in the *aux1* and *pin2* mutants.[36] These data indicate that there is a hierarchical regulatory relationship among KAR signaling, ETH signaling, and auxin transport under phosphorus starvation conditions. However, the mechanism by which KAR signaling perceives the signal of phosphorus starvation and promotes the expression of *ASC7* and *EIN2* is still unclear.

KAR signaling also interacts with ETH to regulate seed germination. In *Avena fatua*, exogenous application of KAR_1 significantly upregulates the expression of ETH biosynthesis genes *ACC OXIDASE 1* (*AfACO1*) and *AfACO5* and ETH signaling pathway genes *ER-BOUND ETHYLENE RECEPTOR 3* (*ETR3*) and *ETR4*, thereby promoting seed germination.[105]

KAR signaling and auxin

Auxin is a key regulator of seedling morphogenesis in response to changes in the light conditions of the surrounding environment.[106] PIN-FORMED (PIN) proteins are the auxin efflux carriers and modulate directional auxin transport.[107] During the dark-to-light transition, auxin transport from the shoot apex toward the root is increased in *kai2* mutants in *Arabidopsis*.[108] The observations using GFP reporter lines have revealed that, in contrast to the wild-type, the distribution of PIN1, PIN3, and PIN7 in older tissues and root meristems is perturbed in the *kai2* mutant (Figure 4E).[108] Furthermore, treatments with the auxin transport inhibitor 1-N-naphthylphthalamic acid (NPA) or the auxin synthesis inhibitor L-kynurenine (L-Kyn) can rescue the abnormal development phenotype of *kai2* in hypocotyl, adventitious root, and lateral root.[108] Likewise, under shade conditions, treatment with L-Kyn also rescues the elongated phenotype of the *kai2* mutant.[109] Free IAA and DR5 reporter assays have shown that, compared to the wild-type, the auxin content in *kai2* mutant hypocotyls is increased.[109] These data indicate that KAR signaling affects auxin transport from the hypocotyl to the root via PINs.

To investigate how KAR signaling affects auxin transport, transcriptional analysis shows that the expression of *PIN3* and *PIN7* is significantly upregulated in *kai2* mutants.[109] However, *SMAX1* overexpression does not affect the expression levels of *PIN3* and *PIN7*.[109] This observation suggests that KAI2 represses the expression of *PIN3* and *PIN7* through SMXL2 or other unknown components. Notably, as mentioned above, under phosphorus starvation conditions, KAR signaling promotes the shootward auxin transport from the root tip by enhancing the localized accumulation of AUX1 and PIN2 through increasing ETH signaling activity (Figure 4F).[36] Therefore, the effect of ETH on auxin transport should be examined under shade and weak light conditions in KAR signaling mutants.

Besides auxin transport, KAR signaling regulates the expression of auxin-responsive genes such as *INDOLE-3-ACETIC ACID INDUCIBLE1* (*IAA1*), *IAA5*, *IAA29*, and *SAURs*.[30,33,38,47,48,52,57] As mentioned above, SMAX1 upregulates the expression of *IAA29* through an EAR motif-independent action.[47] However, the mechanism by which KAR signaling regulates *IAA1*, *IAA5*, and *SAURs* remains unclear.

CROSSTALK BETWEEN KAR SIGNALING AND ENVIRONMENTAL CUES

KAR signaling and light

Light is one of the most important environmental cues and modulates many aspects of plant development, such as seed germination and morphogenesis.[110] Numerous studies have noted the interaction between KAR and the light signaling pathways in regulating plant development. In *Arabidopsis*, light is required for KARs to promote seed germination and enhance seedling photomorphogenesis. Transcriptome analysis using KAR_1-treated seeds has revealed that many light-responsive genes were upregulated by KAR_1.[30] Furthermore, a subset of differentially expressed genes enriched in light-responsive genes has been identified in *ply2* mutants.[41,111]

Further investigation into the mechanism of crosstalk between KAR and light signaling has found that core components of the light signaling pathway participate in KAR signaling. PIF1 regulates seed germination partially by modulating the ratio of ABA to GA.[112-116] In the dark, the *pif1* mutant exhibits an increased seed germination phenotype and can remove the inhibition of *kai2* on seed germination.[102] Further analysis has shown that the expression of most of the PIF1-targeted genes is also regulated by KAR_2. More importantly, the expression patterns of these genes, such as phytochrome signaling genes and ABA-to-GA ratio-related genes, are largely identical between the *pif1* mutant and the KAR_2-treated *KAI2*-overexpressing lines.[102] These data indicate that KAR signaling regulates ABA-to-GA ratio through PIF1 to promote seed germination under dark conditions (Figure 5A).[102] Similarly, as mentioned above, under weak light conditions, KAR signaling promotes seed germination by upregulating the expression level of *GA3ox2*, thereby enhancing GA biosynthesis, which may affect the ABA-to-GA ratio (Figure 5B).[68]

LONG HYPOCOTYL 5 (*HY5*), which encodes a core transcription factor in the light signaling pathway, is a KAR-inducible gene.[30,117-119] In contrast to the wild-type, the inhibition of hypocotyl elongation by KARs treatment is reduced in the *hy5* mutant.[30] Moreover, the *kai2 hy5* and *max2 hy5* double mutants exhibit longer hypocotyls than any of the single mutants,[49] indicating that KAR signaling regulates hypocotyl elongation partially through HY5. Recently, HY5-B-BOX DOMAIN PROTEIN 20/21 (HY5-BBX20/21) module has been found to act downstream of SMAX1/SMXL2 to modulate KAR-mediated anthocyanin accumulation and hypocotyl elongation (Figure 5C).[22] However, the *bbx2021 hy5* triple mutant does not completely eliminate the inhibition of hypocotyl elongation in the *smax1 smxl2* double mutant.[22] This observation indicates that besides *HY5* and *BBX20/21*, other genes participate in regulating hypocotyl elongation downstream of *SMAX1/SMXL2*. Supporting this view, the phyB-PIF4/5 module is found to be located downstream of SMAX1/SMXL2 and regulates KAR-mediated hypocotyl elongation (Figure 5D).[47] Notably, as mentioned above, SMAX1/SMXL2 positively regulate the stability of PIF4/5.[47] On the contrary, SMAX1/SMXL2 probably have a negative effect on the stability of BBX20. Because the level of the BBX20-GFP fusion protein is significantly increased by KAR_2 treatment and is lower in the *kai2* mutant than in the wild-type plant.[22] Moreover, as another demonstration of crosstalk between light and KAR signaling, CONSTITUTIVELY PHOTOMORPHOGENIC1 (COP1), a key negative regulator of photomorphogenesis, inhibits the response to KAR_2 treatment under dark conditions in *Arabidopsis*.[111,120]

On the other hand, light could affect the activity of KAR signaling pathway in *Arabidopsis*. The expression of *KAI2* is upregulated by light.[37,49] This observation is consistent with the role of KAI2 in promoting photomorphogenesis. Furthermore, although light does not affect the expression of *SMAX1*, it induces the accumulation of the SMAX1 protein.[95] However, during seed germination, light can reduce the accumulation of the SMAX1.[102] This inconsistency is probably due to the differences in the light regimes, tissues, and developmental stages used in these studies.

KAR signaling and temperature

Temperature has a decisive influence on the distribution and behavior of plants.[121] Plants modulate their development and maintain physiological and biochemical homeostasis by reprogramming gene expression to adapt to changes in temperature.[122] Many studies have demonstrated that KAR signaling participates in this process.

Thermoinhibition refers to high temperature inhibiting seed germination, while germination proceeds immediately once the temperature drops below a certain threshold.[123] In *Arabidopsis*, seed germination is significantly inhibited at 27°C and above.[99] Interestingly, KAR_2 promotes germination at 20°C but mildly inhibits it at 27°C in *Arabidopsis*.[99] Compared with the wild-type, at 27°C, the germination rate is lower in the *kai2* mutant, and KAR_2 treatment does not inhibit the germination of the *kai2* mutant.[99] These observations indicate that the *kai2* mutant is hypersensitive to elevated temperature, and KAR_2 inhibits germination in a KAI2-dependent manner at 27°C.

Morphological changes in response to non-stress warm temperatures are collectively referred to as thermomorphogenesis.[121] phyB-PIF4 module functions as a thermosensor and plays a key role in regulating thermomorphogenesis.[124,125] Recent studies have shown that SMAX1 enhances the transcriptional activation activity of PIF4 through an unknown mechanism, thereby upregulating the expression of *YUCCA8* (*YUC8*) during thermomorphogenesis (Figure 5E).[126] Interestingly, SMAX1 is slowly degraded under warm temperatures,[126] but the mechanism remains unclear.

KAR signaling has been found to regulate the tolerance response to high-temperature stress. The survival rate of the *kai2* mutant seedlings is lower than that of the wild-type seedlings after being treated at 40°C for 5 days.[127]

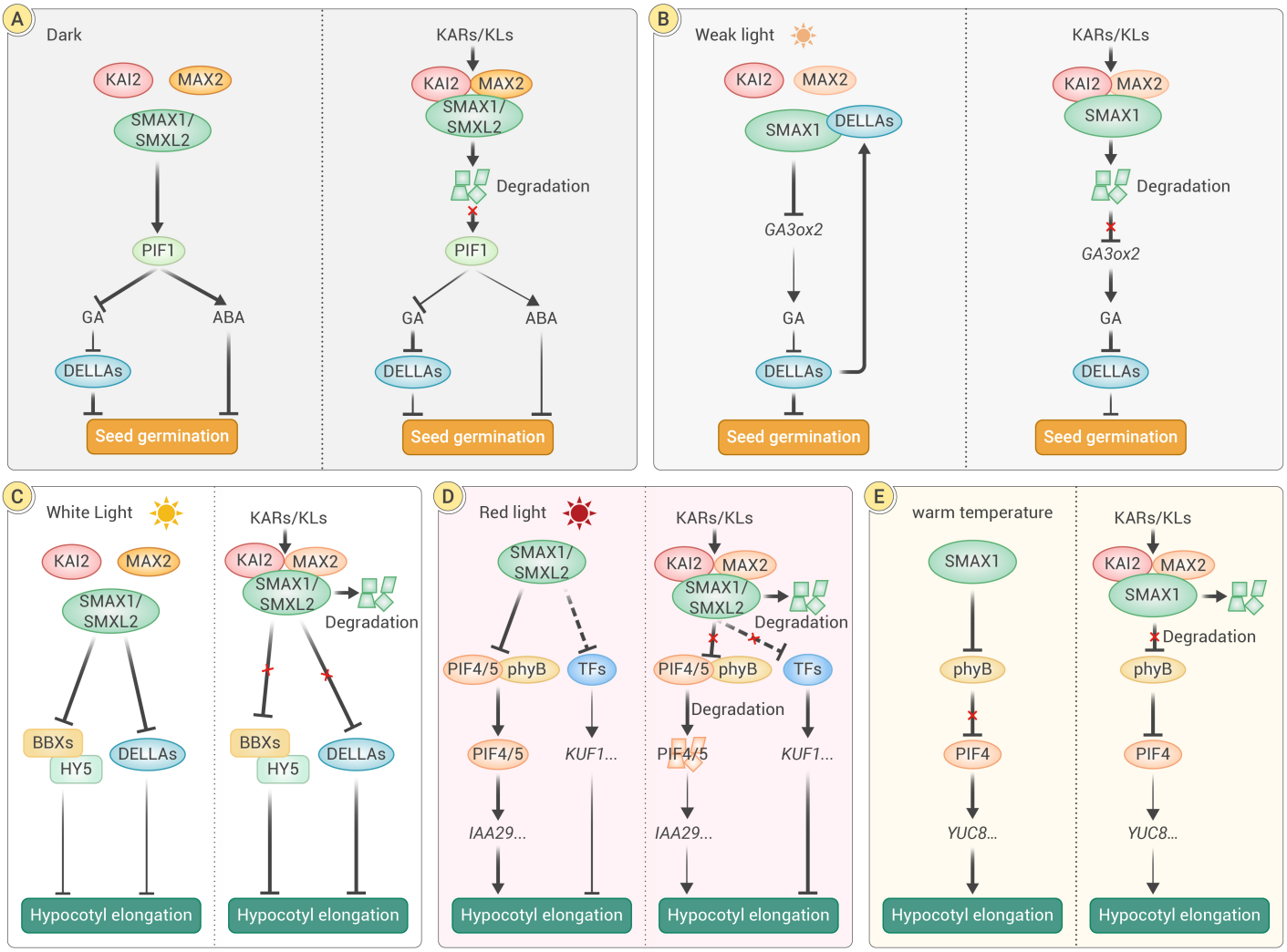

Figure 5. The interactions of KAR signaling pathway with light and warm temperature (A) KAR signaling substitutes for light to promote seed germination. In the dark, SMAX1 enhances the activity of PIF1 to increase the ratio of ABA to GA, thereby repressing seed germination. In the presence of KARs/KLs, SMAX1 is degraded to reduce the activity of PIF1. This process decreases the ratio of ABA to GA, thereby promoting seed germination.[102] (B) KAR signaling integrates GA to regulate seed germination under weak light conditions. Under weak light conditions, SMAX1 interacts with DELLA proteins, which enhances the transcriptional repression of SMAX1 on *GA3ox2*, leading to decreased GA levels and suppressed seed germination. Upon the KARs/KLs perception, SMAX1 is degraded, and the transcriptional repression of *GA3ox2* is relieved, thereby promoting seed germination.[68] (C) KAR signaling integrates white light to inhibit hypocotyl elongation. Under white light conditions, SMAX1/SMXL2 repress the activity of the HY5-BBXs module by downregulating the expression levels of *BBXs*, thereby promoting hypocotyl elongation.[22] On the other hand, under these conditions, SMAX1 promotes hypocotyl elongation by interacting with DELLA proteins and enhancing their degradation.[95] Upon the KARs/KLs-induced degradation of SMAX1/SMXL2, the expression levels of *BBXs* are upregulated, and DELLA proteins accumulates, thereby inhibiting the hypocotyl elongation.[22,95] (D) KAR signaling integrates red light to inhibit hypocotyl elongation. Under red light conditions, SMAX1/SMXL2 promote the accumulation of PIF4/5 by inhibiting the interactions between PIF4/5 and phyB, thereby upregulating *IAA29* expression and promoting hypocotyl elongation. Additionally, SMAX1/SMXL2 interact with unknown transcription factors to repress *KUF1* expression, thereby contributing to hypocotyl elongation.[47] In the presence of KARs/KLs, SMAX1/SMXL2 are degraded, leading to the degradation of PIF4/5 and downregulation of *IAA29* expression. Meanwhile, due to the removal of repression on unknown transcription factors, *KUF1* expression is enhanced. Finally, the hypocotyl elongation is inhibited by altering the expression levels of *IAA29* and *KUF1*.[47] (E) Under warm non-stress temperature conditions, SMAX1 enhances the transcriptional activation activity of PIF4 by suppressing the effect of phyB on PIF4, thereby upregulating the expression of *YUC8* and promoting hypocotyl elongation.[126] KARs/KLs suppress the PIF4 transcriptional activation activity by inducing the degradation of SMAX1, thereby inhibiting hypocotyl elongation under warm temperature conditions.[126] TFs, transcription factors. Arrows represent positive regulation and bars indicate negative regulation.

Further transcriptome analysis has shown that the expression of genes related to heat stress tolerance, such as *HEAT SHOCK PROTEINs* (*HSPs*) and *HEAT SHOCK TRANSCRIPTION FACTORs* (*HSFs*), was downregulated in *kai2* mutants (Figure 6).[127]

KAR signaling also participates in regulating the response to cold stress in plants. In *Arabidopsis*, cold stress could induce the expression of *KAI2*.[128] Consistent with this observation, in *Sapium sebiferum*, the expression of *SsKAI2* is also upregulated by cold stress, and overexpressing *SsKAI2* in *Arabidopsis* significantly enhances cold tolerance.[129] Further analysis has shown that the content of proline, total soluble sugars, and total soluble proteins increased, and the expression of *C-REPEAT BINDING FACTORs* (*CBFs*) is upregulated in *SsKAI2* overexpressing lines under cold stress (Figure 6).[129] Notably, the cold-responsive gene *SNF1-RELATED PROTEIN KINASE 2.3* (*SnRK2.3*), which encodes a positive regulator of ABA signaling,[130] is significantly induced by cold stress in *SsKAI2* overexpressing lines in *Arabidopsis* (Figure 4G),[129] indicating that ABA signaling contributes to KAI2-mediated cold tolerance. Similarly, in tomato, *SnRK2.5* is the homolog of *SnRK2.3*, and its expression is upregulated by KARs treatment and is significantly lower in *KAI2* knockdown plants than in wild-type plants under cold stress.[131] Furthermore, knocking down the expression of *SnRK2.5* impairs KAR-induced cold tolerance in tomato.[131]

KAR signaling and drought stress

KAR signaling regulates plant responses to drought stress. In *Arabidopsis*, in contrast to the wild-type, *kai2* and *max2* mutants are hypersensitive to drought stress, while *smax1 smxl2* double mutants exhibit hyposensitivity to drought stress.[25,27,28,132,133] The phenotype analysis shows that *kai2* and *max2* mutants exhibit thinner cuticles, while *smax1 smxl2* double mutants have thicker cuticles, which is consistent with the expression levels of cuticle formation genes *ECERIFERUM 1* (*CER1*) and *CER4*.[25,27,132] Furthermore, the

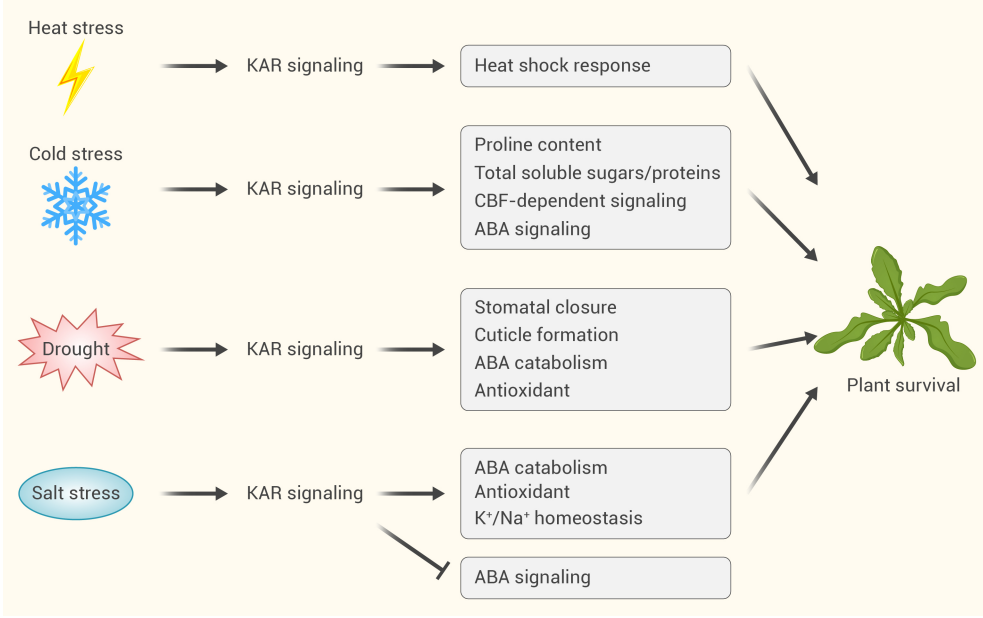

Figure 6. The interaction of KAR signaling pathway with abiotic stress KAR signaling positively regulates heat stress tolerance by activating the heat shock response.[127] During the response to cold stress, KAR signaling increases the levels of proline, total soluble sugars, total soluble proteins, and antioxidants, and activates CBF-dependent signaling and ABA signaling.[129,131] Under drought stress, KAR signaling enhances stomatal closure, cuticle formation, ABA catabolism and antioxidant defense, thereby improving drought tolerance.[25,27] Under salt stress, KAR signaling increases ABA catabolism and inhibits ABA signaling activity, enhances antioxidant defense and maintains K^+/Na^+ homeostasis to enhance salt tolerance.[31,32,137] Arrows represent positive regulation and bar indicates negative regulation.

kai2 mutant shows a larger stomatal aperture phenotype, and its stomatal closure is hyposensitive to ABA treatment, while the *smax1 smxl2* double mutant exhibits opposite phenotypes.[25,27] Consistent with these observations, the ABA content is higher in *kai2* mutants but lower in *smax1 smxl2* double mutants under drought conditions.[25,27] Transcriptional analysis shows that the expression of the ABA catabolism gene *CYP707A3* is significantly inhibited in *kai2* mutants but enhanced in *smax1 smxl2* double mutants under drought conditions.[25,27] These data indicate that KAR signaling negatively regulates the ABA content by increasing the expression of *CYP707A3* under drought conditions (Figure 4H). However, ABA biosynthesis and/or signaling activity is usually enhanced under drought conditions and plays a positive role in regulating drought tolerance,[134-136] suggesting that KAR signaling fine-tunes ABA homeostasis under drought conditions. In addition, reactive oxygen species (ROS) are involved in KAR signaling related drought responses. The expression levels of H_2O_2 scavenger-encoding genes, *GLUTATHIONE PEROXIDASE 3* (*GPX3*) and *GPX7*, are upregulated in the *smax1 smxl2* double mutant under drought conditions (Figure 6).[25]

KAR signaling and salt stress

Salt exists naturally in the surroundings of plants, but excessive salt can cause serious inhibition of growth and development. KAR signaling has been reported to be involved in the response to salt stress in plants. In *Arabidopsis*, KAR_2 inhibits seed germination under salt stress conditions.[99] However, KAR_1 promotes seed germination and seedling growth in *Sapium sebiferum* and wheat under salt stress conditions.[31,32] Moreover, in contrast to the wild-type, *kai2* mutants exhibit delayed seed germination, inhibited growth, and a decreased survival rate under salt stress conditions.[137]

ABA is involved in KAR signaling mediated salt stress response. In *Sapium sebiferum*, KAR_1 induced the expression of genes encoding ABA signaling positive regulators, including *SnRK2.3*, *SnRK2.6*, *ABSCISIC ACID INSENSITIVE 3* (*ABI3*), and *ABI5*, under salt stress conditions, which probably enhances the activity of ABA signaling.[32] However, the interaction between KAR signaling and ABA under salt stress conditions shows an obvious difference in other plants. In *Arabidopsis*, salt significantly induces the expression of *KAI2*.[137] More importantly, the expression levels of ABA signaling negatively regulated genes, *ABSCISIC ACID INSENSITIVE1* (*ABI1*), *ABI2*, and *HYPER-SENSITIVE TO ABA1* (*HAB1*), as well as ABA catabolism-related genes, *CYP707A1* and *CYP707A3*, are downregulated in the *kai2* mutant under salt stress conditions (Figure 4I).[97,137-139] These observations indicate that KAR signaling represses the activity of ABA signaling under salt stress conditions in *Arabidopsis*. This is similar to the fact that ABA content is negatively regulated by KAR signaling under drought conditions,[25,27] suggesting that the fine-tuned ABA signaling homeostasis by KAR signaling is important for salt tolerance.

Besides fine-tuning ABA signaling, KAR signaling could reduce the ROS level under salt stress. In *Arabidopsis*, the *kai2* mutant exhibits higher H_2O_2, malondialdehyde (MDA), and electrolyte leakage (EL) levels than the wild-type under salt stress (Figure 6).[137] Similarly, KAR_1 treatment could reduce the ROS level under salt stress by enhancing the antioxidant activity in *Sapium sebiferum* and wheat.[31,32] In addition, in wheat, KAR_1 treatment upregulates the expression of *SALT OVERLY SENSITIVE1* (*SOS1*, an important Na^+/H^+ antiporter encoding gene) and *HIGH-AFFINITY POTASSIUM TRANSPORTER1* (*HKT1*), thereby reducing Na^+ accumulation, increasing the K^+/Na^+ ratio, and alleviating seedling growth inhibition under salt stress (Figure 6).[31]

CONCLUSION AND PROSPECTS

Although many exciting advances have been made in the study of KAR signaling, several gaps still need to be addressed. Firstly, what kind of substance is KLs? To date, although many studies indicate that plants can produce KLs and that KARs can be metabolized into KLs by plants, direct evidence is still lacking. It is necessary to identify the key enzymes in the KLs biosynthesis and KARs metabolism pathways. Given that KUF1 potentially regulates the protein stability of enzymes involved in KAR_1 metabolism,[72] identifying its target proteins is valuable for this research. Meanwhile, developing a reporter that sensitively reflects the fluctuation of endogenous KLs content will provide vital assistance in cloning KLs biosynthesis genes. Furthermore, the role of (−)-germacrene D in regulating KAR-related development, such as germination and hypocotyl elongation, should be further analyzed. Secondly, as mentioned above, KAI2-MAX2-SMAX1/SMXL2 modules regulate the expression of downstream genes, which mediate the interactions of KAR signaling with phytohormones and environmental cues. However, in the KAR signaling pathway, the action of SMAX1/SMXL2 downstream is still unclear. SMAX1/SMXL2 exhibit transcriptional repression activities,[47,68] indicating that they are similar to repressors in the IAA, SL, and JA signaling pathways and could interact with transcription factors and repress their transcriptional activity, thereby regulating downstream gene expression.[140] Therefore, the transcription factors, which interact with SMAX1/SMXL2 and whose transcriptional activity is repressed by SMAX1/SMXL2, still need to be identified, although several proteins have been found to associate with SMAX1/SMXL2.[47,68,95,126,141-143] Thirdly, KAR_1 and KAR_2 induce the degradation of KAI2,[45,70] and light and temperature affect the accumulation of SMAX1;[95,102,126] however, the mechanisms remain unclear. Amino acid saturation mutation analysis in KAI2 and SMAX1/SMXL2 will contribute to understanding the mechanisms regulating their protein stability.

KARs could improve drought and salt tolerance in plants and regulate many aspects of development through their signaling pathway, indicating that they have considerable potential for application in agriculture. However, before being used in agriculture, less expensive and more efficient KARs/KLs should be produced. Actually, identifying the KL biosynthesis pathway will contribute to using synthetic biology approaches for producing KARs/KLs. On the other hand, studies on the function of KARs/KLs in crops are valuable for crop genetic improvement. The studies of SLs provide an encouraging and

useful case. In rice, *D17* encodes a key enzyme involved in SLs biosynthesis.[144] During the Green Revolution, an elite haplotype of *D17* was utilized and co-selected with *SEMIDWARF1* (*SD1*), leading to a reduction in plant height while increasing tiller number.[145] Notably, a recent study has identified an elite haplotype of D14, which significantly enhances nitrogen use efficiency in rice.[83] Furthermore, this study uncovered a new mechanism of SL signaling termination in rice whereby the N-terminal disordered region (NTD) of D14 mediates the interaction between D14 and the 26S proteasome, thereby promoting SL-induced degradation of D14.[83] Collectively, an in-depth study on KAR signaling in crops in the future will provide important gene resources for crop improvement and new insights into the mechanism of KAR signaling regulating plant development and adaptation.

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AUTHOR CONTRIBUTIONS

L.W. conceived the project. S.C. and X.L. wrote the manuscript. L.W. supervised and edited the manuscript. All authors contributed to the manuscript and approved the final version.

DECLARATION OF INTERESTS

The authors declare no competing interests.

ETHICAL STATEMENT AND PATIENT CONSENT

Not applicable.

DATA AND CODE AVAILABILITY

Not applicable.

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