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Review

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Signal transduction mediated by the plant UV-B photoreceptor UVR8

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Summary

Ultraviolet-B (UV-B) light is an intrinsic part of sunlight that has significant effects on plant development and acclimation responses. UVR8 (UV Resistance Locus 8) is the long sought-after UV-B photoreceptor that mediates UV-B light perception and signal transduction. UV-B irradiation induces the monomerization and nuclear accumulation of UVR8 in plant cells to activate the UV-B signaling pathway. The photoactivated UVR8 could transduce UV-B signal via multiple mechanisms to regulate transcription and plant growth. Here, we summarize current understanding of UVR8-mediated UV-B signal transduction pathways, including UVR8–COP1 (CONSTITUTIVELY PHOTOMORPHOGENIC 1) and UVR8–WRKY36 (WRKY DNA-BINDING PROTEIN 36), UVR8–BES1 (BRI1-EMS-SUPPRESSOR1) and BIM1 (BES1-INTERACTING MYC-LIKE 1).

I. Introduction

UV-B light (280–315 nm) is not only a potential stress but also an informational signal to regulate plant growth and development (Tilbrook *et al.*, 2013). UV-B irradiation serves as a signal to regulate plant photomorphogenesis, including inhibition of hypocotyl elongation, flavonoids and anthocyanins accumulation, and to induce the expression of UV-B-responsive genes. UVR8 is the UV-B photoreceptor that is responsible for UV-B photomorphogenesis and acclimation (Kliebenstein *et al.*, 2002; Brown *et al.*, 2005; Rizzini *et al.*, 2011; Christie *et al.*, 2012; Wu *et al.*, 2012; Jenkins, 2014). UV-B irradiation does not affect UVR8's protein abundance, but induces the monomerization and nuclear accumulation of UVR8 (Kaiserli & Jenkins, 2007; Rizzini *et al.*, 2011). In the absence of UV-B, UVR8 forms homodimer, while in the presence of UV-B, UVR8 absorbs UV-B by using its tryptophans as chromophore and further undergoes structural changes and immediately monomerizes into active monomers (Rizzini *et al.*, 2011; Christie *et al.*, 2012; Hofmann, 2012; Wu *et al.*, 2012; Zeng *et al.*, 2015). UVR8 is localized in both cytosol and nucleus without UV-B while it accumulates into nucleus in response to UV-B (Kaiserli & Jenkins, 2007; Qian *et al.*, 2016; Yin *et al.*, 2016). Both the monomerization and nuclear accumulation of UVR8 are critical for its function in controlling photomorphogenesis (Kaiserli & Jenkins, 2007; Rizzini *et al.*, 2011). The monomer and dimer status of UVR8 is dynamic and reversible. When plants are growing in diurnal photoperiods, UVR8 establishes a dimer/monomer photo-equilibrium (Findlay & Jenkins, 2016). UVR8 re-dimerization is promoted by WD40-repeat proteins, RUPs (REPRESSOR OF UV-B PHOTOMORPHOGENESIS) (Gruber *et al.*, 2010; Heijde & Ulm, 2013). The transcripts of *RUPs* are UV-B-induced and RUP proteins could physically interact with

UVR8 and mediate UVR8 re-dimerization, so as to negatively regulate UV-B signal transduction (Heijde & Ulm, 2013). Direct interaction between photoreceptors and their respective target proteins have been recognized as a fundamental mechanism underlying the signal transduction of those photoreceptors. In addition to RUPs, UVR8 physically interacts with COP1, WRKY36, BES1 and BIM1 to transduce UV-B light signal (Favory *et al.*, 2009; Liang *et al.*, 2018; Yang *et al.*, 2018).

II. The UVR8–COP1 pathway

The E3 ubiquitin ligase COP1, known as a repressor of photomorphogenesis, is involved in multiple light signaling pathways (Podolec & Ulm, 2018). COP1 interacts with SPA1 (SUPPRESSOR OF PHYA) and other components of E3 ubiquitin ligase complexes to promote the ubiquitination and degradation of transcription factor HY5 (ELONGATED HYPOCOTYL 5) and HYH (HY5 HOMOLOG) (Lau & Deng, 2012). However, in the UV-B signaling pathway, COP1 functions to promote HY5 expression and protein stabilization in response to UV-B (Oravecz *et al.*, 2006). The photoactivated monomeric UVR8 physically interacts with COP1, which is indispensable for the signal transduction of UVR8 (Favory *et al.*, 2009). UVR8 has two distinct domains: a seven-bladed β-propeller core domain and a C-terminal area of 27 amino acids, and both of these two domains could interact with the C-terminal WD40-repeat domain of COP1 (Cloix *et al.*, 2012; Yin *et al.*, 2015). A truncated UVR8 containing only the β-propeller domain interacts with COP1 in an UV-B-dependent manner, although the interaction is weaker than the wild-type UVR8 interaction with COP1. The truncated version of UVR8 containing only the C27 domain is able to interact constitutively with COP1 in both transgenic plants and yeast. However, the wild-type UVR8 containing C27 domain interacts with COP1 specifically after UV-B irradiation, so a hypothesis was raised that the C27 is masked in the absence of UV-B but unmasked in presence of UV-B (Yin *et al.*, 2015). The UV-B-triggered UVR8–COP1 interaction regulates COP1 activity, possibly induces functional disassociation of the COP1–SPA core complexes from the E3 ubiquitin ligase apparatus and formation of a unique complex containing UVR8–COP1–SPA1, so as to reduce the ubiquitination and degradation of HY5 (Huang *et al.*, 2013). HY5 and its homolog HYH could bind to the T/G-box in the *HY5* promoter and they act redundantly to induce *HY5* expression upon UV-B exposure (Binkert *et al.*, 2014). The accumulated HY5 could control multiple UV-B-responsive gene expression besides *HY5*, such as *RUPs*, *COP1* and flavonoid biosynthesis genes (Ulm *et al.*, 2004). In summary, UV-B irradiation triggers the interaction between UVR8 and COP1 to promote HY5 expression and protein stabilization, so as to induce UV-B-responsive gene expression and photomorphogenesis (Fig. 1).

In addition to signal transduction, UVR8–COP1 interaction is required for the nuclear accumulation of UVR8 (Qian *et al.*, 2016; Yin *et al.*, 2016). The photoactivated nuclear UVR8 is critical for *HY5* induction and photomorphogenesis, while cytoplasmic UVR8 is unable to induce *HY5* expression and photomorphogenesis (Kaiserli & Jenkins, 2007; Qian *et al.*, 2016; Yin *et al.*, 2016). The nuclear accumulation process of UVR8 is dependent on UVR8 monomerization and regulated by COP1 and RUPs. The UV-B-induced UVR8 nuclear accumulation is almost disrupted in the *cop1-4* mutant but it is enhanced in *rup1 rup2* mutant, indicating that UVR8 accumulation is promoted by COP1 but repressed by RUPs (Qian *et al.*, 2016; Yin *et al.*, 2016). How COP1 facilitates UVR8 nuclear accumulation remains elusive. Two models were raised: the first is the nuclear coimport model: UV-B triggers UVR8 monomerization and interacts with COP1 in the cytosol and the COP1-containing nuclear localization sequence mediates the nuclear coimport of UVR8. Another model is the nuclear retention model: UV-B promotes UVR8 monomerization and translocation into the nucleus by an unknown mechanism, while nuclear COP1 acts to inhibit UVR8 immediate nuclear export (Yin & Ulm, 2017). More efforts are needed to explore the detailed function of COP1 in mediating UVR8 nuclear accumulation.

III. The UVR8–WRKY36 pathway

Besides COP1 and RUPs, UVR8 could also interact with multiple transcription factors, such as WRKY36, BIM1 and BES1, to directly regulate gene expression. Among biological processes in response to UV-B irradiation, *HY5* induction is of great importance for downstream gene response and UV-B photomorphogenesis. The UV-B-stabilized HY5 protein could bind to its own promoter to activate the expression (Binkert *et al.*, 2014). Three cis-regulatory elements (an ACG-box (ACG), a T/G box, and an E-box) have been reported to mediate the transcription activation of *HY5*, the ACG-box that acts as a light-induced *HY5* repression element (Binkert *et al.*, 2014). It was proposed that *HY5*'s transcription was repressed by an unknown transcription factor which bound to this ACG-box in continuous visible light, while with UV-B light irradiation, the repressor might be degraded or competed by a positive regulator that had a higher affinity for the ACG motif (Binkert *et al.*, 2014). Very recently, transcription factor WRKY36 was found to be a repressing regulator of *HY5* transcription and UV-B photomorphogenesis, although WRKY36 is not the repressor which binds to the ACG-box (Yang *et al.*, 2018). WRKY36 was identified from a yeast two-hybrid screening using UVR8 as the bait. The mRNA expression of *WRKY36* is increased in response to UV-B within the first hour and then decreased, and this transcriptional change is not UVR8-dependent, indicating that there may be other new UV-B photoreceptors that are responsible for this transcriptional change. WRKY36 could interact with both dimeric and monomeric UVR8 *in vitro* and *in vivo*. The C-terminal DNA-binding domain of WRKY36 interacts with the C27 domain of UVR8. WRKY36 is localized in the nucleus and the nuclear accumulation of UVR8 is induced by UV-B light, so the accumulation of the UVR8–WRKY36 complex in the nucleus is promoted by UV-B irradiation. WRKY36 promotes hypocotyl elongation and is involved in UV-B responses. The *wrky36* mutant shows dramatic short hypocotyl phenotype under white light but has a similar short hypocotyl as the wild-type under UV-B light. WRKY36 acts downstream of UVR8 to regulate hypocotyl elongation as the long hypocotyl of *uvr8* mutant was partially

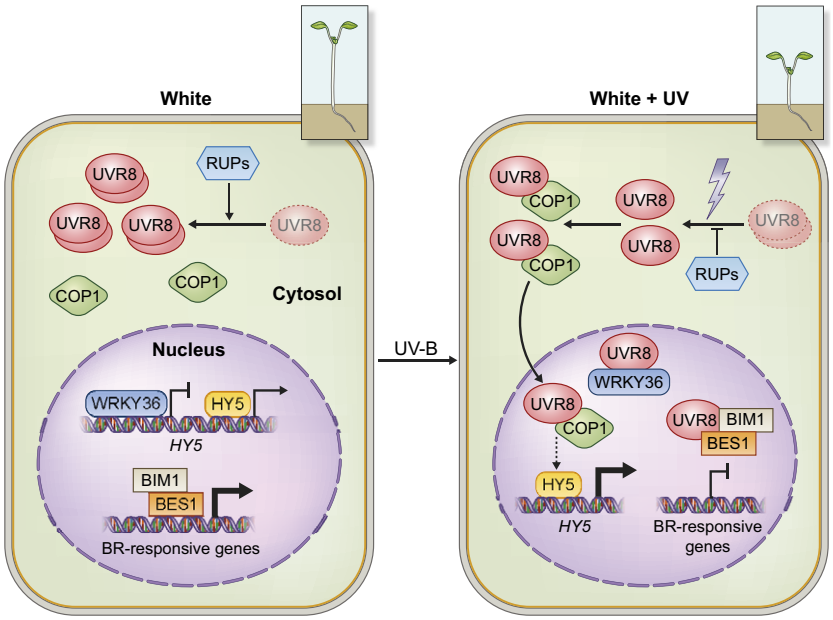

Fig. 1 Working model of UVR8-mediated signal transduction in Arabidopsis. When plants are grown in white light (left panel), the UV-B photoreceptor UVR8 is mainly localized in the cytosol as homodimer and COP1 is also localized in the cytosol. Transcription factors HY5, WRKY36, BIM1 and the functional BES1 are localized in the nucleus. HY5 binds to its own promoter to activate *HY5* transcription, and WRKY36 also binds to the promoter of *HY5* to inhibit its transcription. HY5 and WRKY36 function oppositely to regulate the expression of *HY5*. By contrast, BIM1 and BES1 act together to induce the expression of brassinosteroid (BR)-responsive genes. When plants are grown in white light plus UV-B light (right panel), UVR8 perceives UV-B light and monomerizes into active monomer. The active monomeric UVR8 interacts with COP1, and COP1 promotes UVR8 nuclear accumulation. The nuclear UVR8–COP1 complex facilitates HY5 protein stabilization and enhances the binding of HY5 to its own promoter, thus activating *HY5* transcription. Nuclear-localized UVR8 also interacts with WRKY36 to inhibit WRKY36 binding to the promoter of *HY5* so as to remove the inhibition of *HY5* expression. As a result, UVR8 employs at least two mechanisms to activate the transcription of *HY5*. In addition, the nuclear-localized UVR8 interacts with BIM1 and the functional dephosphorylated BES1 to inhibit their binding to the BR-induced genes involved in cell elongation, thus repressing the expression of BR-induced elongation genes and further repressing the BR-promoted plant growth. RUPs interact with UVR8 to facilitate UVR8 redimerization, probably in both the cytosol and the nucleus (only the cytosol part is shown for simplicity). See Box 1 for an explanation of the abbreviations.

dependent on WRKY36. WRKY36 regulates hypocotyl elongation via repressing *HY5* expression. *HY5* transcript is increased in the *wrky36* mutant compared with the wild-type specifically under white light but not in continuous UV-B light, indicating that WRKY36 inhibits *HY5* expression and UV-B suppresses WRKY36. WRKY36 could directly interact with the W-box in the *HY5* promoter and repress *HY5* expression while nuclear-localized UVR8 inhibits WRKY36's DNA-binding activity to suppress the inhibition of *HY5* expression. Taken together, WRKY36 works as an *HY5* transcription repressor to repress *HY5* transcription. UVR8 mainly localizes in the cytosol without UV-B, while WRKY36 localizes in the nucleus to inhibit *HY5* transcription and promote hypocotyl elongation. With UV-B treatment, UVR8 monomerizes and accumulates into the nucleus to interact with the WRKY36 transcription factor to inhibit its DNA-binding ability and thus to promote *HY5* transcription and inhibit hypocotyl elongation (Yang *et al.*, 2018) (Fig. 1).

IV. The UVR8–BES1/BIM1 pathway

UVR8 also physically interacts with brassinosteroid (BR) signaling transcription factors to control transcription. BR signaling components BES1 and BIM1 could also interact with UVR8 (Liang *et al.*, 2018). BRs are steroidal hormones critical in regulating plant growth and development, such as skotomorphogenesis, photomorphogenesis, and in mediating biotic and abiotic stresses (Clouse, 2011). BRs are perceived by the surface receptor kinase BRASSINOSTEROID INSENSITIVE 1 (BRI1), which initiates a signaling cascade to activate downstream transcription factor BES1 and BZR1 (BRASSINAZOLE RESISTANT 1) (Li & Chory, 1997; He *et al.*, 2002; Nam & Li, 2002). In the absence of BR, the GSK3-like kinase BIN2 (BR-INSENSITIVE 2) phosphorylates and inhibits the function of BES1 and BZR1 through multiple mechanisms (Li & Jin, 2007); in the presence of BR, BIN2 activity is inhibited, leading to the accumulation of dephosphorylated BES1 and BZR1 in the nucleus (Belkhadir & Jaillais, 2015), which promote expression of BR target genes (Wang *et al.*, 2002; Yin *et al.*, 2002). BIM1 could interact with BES1 and act together to coordinately regulate BR-induced gene expression and hypocotyl elongation (Yin *et al.*, 2005). BIM1 was identified as a UVR8-interacting protein from a yeast two-hybrid screening using UVR8 as the bait (Liang *et al.*, 2018). The UVR8 photoreceptor physically interacts with BIM1 and BES1, independently of UV-B treatment. Furthermore, UVR8 prefers to interact with the dephosphorylated BES1, which is the active form in regulating transcription and BR signaling; dephosphorylated BES1 has the DNA-binding activity and is induced by BR treatment (Vert & Chory, 2006). UVR8 also interacts with BES1 homolog BZR1 and the long isoform of BES1 (BES1-L), which contains an additional N-terminal nuclear localization signal (Jiang *et al.*, 2015). UVR8 has a strong

interaction with the BIN2 phosphorylation domain of BES1 and the C-terminus of BIM1, and has a weak interaction with the basic helix–loop–helix (bHLH) domain of BIM1 itself, while it has a strong interaction with the bHLH domain combined with either the N-terminus or C-terminus of BIM1. As transcription factors, BIM1 and the functional dephosphorylated BES1 are localized in the nucleus, while UVR8 accumulates into the nucleus in response to UV-B, so the nuclear accumulation of UVR8 and also the UVR8–BES1 protein complex formation in the nucleus are promoted by UV-B light. As positive regulators of BR signaling, BIM1 and BES1 promote hypocotyl elongation and they are involved in UV-B-controlled photomorphogenesis. UV-B-repressed hypocotyl elongation is mediated by inhibition of BR signaling. UV-B and nuclear-localized UVR8 inhibit BR responses to inhibit hypocotyl elongation and plant growth. Transcriptomic analysis reveals that BR induces, but UV-B represses, the expression of growth-related genes. Quantitative reverse transcription polymerase chain reaction show that UV-B and UVR8 repress the expression of BES1-targeted growth-related genes but they do not regulate BR-biosynthesis genes expression, which is consistent with the idea that UV-B represses BR responses to inhibit plant growth. Nuclear-localized photoactivated UVR8 associates with dephosphorylated BES1 to prevent it from binding to the promoters of growth-related genes so as to repress the expression of those genes. UVR8–BIM1/BES1 interaction represents an early photoreceptor signaling transduction pathway and UV-B integrates with BR signal to fine-tune plant growth (Liang *et al.*, 2018) (Fig. 1).

Box 1 Glossary

UVR8: UV-B photoreceptor in plants
UV-B photomorphogenesis: UV-B light-mediated development, including inhibition of hypocotyl elongation, flavonoid and anthocyanin accumulation, and induction of UV-B responsive genes
COP1: CONSTITUTIVELY PHOTOMORPHOGENIC 1, E3 ubiquitin ligase, interact with UVR8 and several other photoreceptors
RUPs: REPRESSOR OF UV-B PHOTOMORPHOGENESIS, WD40 repeat protein, interact with UVR8, promote UVR8 redimerization
HY5: ELONGATED HYPOCOTYL 5, master transcription factor of light signaling
WRKY36: WRKY DNA-BINDING PROTEIN 36, transcription factor
BRs: brassinosteroids, growth-promoting steroidal hormone in plants
BES1: BRI1-EMS-SUPPRESSOR1, BR signaling master transcription factor
BIM1: BES1-INTERACTING MYC-LIKE 1, transcription factor, acts together with BES1 to regulate the expression of BR-induced genes
PIF4: PHYTOCHROME INTERACTING FACTOR4, key transcription factor of light signaling, promotes cell elongation
DELLAs: master regulators of gibberellin signaling pathway, growth-repressing proteins
HFR1: LONG HYPOCOTYL IN FAR RED, transcription factor of light signaling
Shade avoidance: responses of plants to shade, including elongation of stem to compete for light
Thermomorphogenesis: responses of plants to ambient temperature change

V. Other pathways

UVR8 could also control PIF4 (PHYTOCHROME INTERACTING FACTOR 4) in differing degrees to regulate shade avoidance responses and thermomorphogenesis (Hayes *et al.*, 2014, 2017). When grown in close proximity to neighboring vegetation, shade-intolerant plants detect the presence of competing vegetation and trigger shade avoidance responses, including enhanced growth of the stem to compete for light (Fraser *et al.*, 2016). The UV-B-activated UVR8 inhibits shade-promoting hypocotyl elongation by controlling the protein stability and function of PIF4 and PIF5, which play a central role in shade-avoidance responses (Lorrain *et al.*, 2008). DELLAs are growth-repressing proteins and act to inactivate PIF function (Feng *et al.*, 2008). UV-B could promote PIF degradation and stabilize DELLAs to inhibit PIF function (de Lucas *et al.*, 2008). This dual mechanism prevents PIF from activating the expression of auxin biosynthesis genes, and thus inhibits shade-avoidance responses (Hayes *et al.*, 2014). Similar to shade-avoidance responses, increases in ambient temperature elicit plant morphological changes, including hypocotyl and petiole elongation. PIF4 is a critical positive regulator in thermomorphogenesis, and UV-B could inhibit PIF4 transcriptional expression and suppress PIF4 function to inhibit thermomorphogenesis (Koini *et al.*, 2009; Hayes *et al.*, 2017). The UVR8–COP1 module inhibits PIF4 transcription at both cooler and warmer temperatures. UV-B could also stabilize HFR1 (LONG HYPOCOTYL IN FAR RED) which forms a competitive complex with PIFs to inhibit their -DNA binding activity (Hayes *et al.*, 2017; Yin, 2017). Collectively, UV-B activated UVR8 controls PIF4 activity through multiple mechanisms to suppress shade- and high temperature-promoted plant growth.

VI. Conclusion and perspectives

As a UV-B photoreceptor, UVR8 is essential for photomorphogenic responses and UV-B acclimation. Significant progress has been made in understanding mechanisms of UVR8 signal transduction in recent years. The UV-B-dependent interaction between UVR8 and COP1 is not only a key mechanism for UV-B signaling but is also essential for the nuclear accumulation of UVR8 in response to UV-B. UVR8 could also interact with transcription factors WRKY36, BIM1 and BES1 to directly regulate transcription and hypocotyl elongation. UVR8 could inhibit plant growth by repressing BR-promoted plant growth through the repression of BIM1 and BES1, and also by promoting *HY5* transcription via repressing WRKY36, which is a transcription repressor of *HY5* (Fig. 1). The interaction between UVR8 and transcription factors may be one of the biologically significant factors of the nuclear accumulation of UVR8 under UV-B light. However, many aspects of mechanistic action and signal transduction of UVR8 still remain to be investigated, such as the detailed mechanism of the nuclear accumulation of UVR8, and how COP1 facilitates this process. The function of COP1 in the UVR8 signaling pathway remains unclear. Why is COP1 indispensable for the stabilization and expression HY5? Are there additional UVR8-interacting proteins

involved in early UV-B signal process? Are there transcription factors interacting with UVR8 in a UV-B dependent manner? Furthermore, direct links between UVR8 and hormone signalings besides brassinosteroids remain unclear. Further investigations of these questions are required to fully understand the action mechanism of UVR8.

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