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Perception and Signaling of Ultraviolet-B Radiation in Plants

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Abstract

Ultraviolet-B (UV-B) radiation is an intrinsic fraction of sunlight that plants perceive through the UVR8 photoreceptor. UVR8 is a homodimer in its ground state that monomerizes upon UV-B photon absorption via distinct tryptophan residues. Monomeric UVR8 competitively binds to the substrate binding site of COP1, thus inhibiting its E3 ubiquitin ligase activity against target proteins, which include transcriptional regulators such as HY5. The UVR8–COP1 interaction also leads to the destabilization of PIF bHLH factor family members. Additionally, UVR8 directly interacts with and inhibits the DNA binding of a different set of transcription factors. Each of these UVR8 signaling mechanisms initiates nuclear gene expression changes leading to UV-B-induced photomorphogenesis and acclimation. The two WD40-repeat proteins RUP1 and RUP2 provide negative feedback regulation and inactivate UVR8 by facilitating redimerization. Here, we review the molecular mechanisms of the UVR8 pathway from UV-B perception and signal transduction to gene expression changes and physiological UV-B responses.

Contents

Photoreceptor: a photosensory protein that absorbs photons either directly or via a prosthetic chromophore and uses light information to modulate a specific signaling cascade, leading to specific light responses

Cryptochrome: a blue-light/UV-A photoreceptor that is evolutionarily derived from photolyases, bearing a flavin-based chromophore

UV RESISTANCE LOCUS 8 (UVR8): a photoreceptor that senses UV-B radiation via intrinsic tryptophan residues functioning as chromophores

Photoprotection: processes that prevent light-induced damage

1. INTRODUCTION

Sunlight fuels photosynthesis and provides crucial information about the environment, but it can also be an environmental stress factor for plants [e.g., high light, ultraviolet-B (UV-B) radiation] (27). Information from light is obtained through specific photoreceptors that affect plant metabolism, development, and viability throughout the life cycle (42, 70, 88, 176). In many cases, photoreceptor signaling optimizes photosynthesis and protects plants from potential light stress (27). A variety of photoreceptors have evolved that detect photons of specific wavelengths and convert these light signals into cellular signaling cascades. Photoreceptor-mediated signaling facilitates appropriate plant responses to changes in the light spectrum in terms of quality (color), quantity, direction, and duration (photoperiod). In flowering plants, these photoreceptors include the red/far-red light–perceiving phytochromes; the blue/ultraviolet-A (UV-A) light–perceiving cryptochromes, phototropins, and Zeitlupe family members; and the UV-B photoreceptor UV RESISTANCE LOCUS 8 (UVR8) (42). The red/far-red and blue/UV-A light photoreceptors absorb photons with bound chromophores that are derived from linear tetrapyrroles (bilins) and flavins, respectively (42). By contrast, the UVR8 perception mechanism is exceptional in that particular intrinsic tryptophan (Trp) residues absorb UV-B photons and initiate signaling (20, 125, 166). The UVR8 pathway is functionally conserved, from green algae to flowering plants, and plays key roles in photoprotection (2, 26, 36, 38, 47, 74, 143).

Ultraviolet (UV) radiation in the electromagnetic spectrum is conventionally defined as UV-A (315–400 nm), UV-B (280–315 nm), and UV-C (100–280 nm). Sunlight reaching Earth's surface includes UV-A and a part of UV-B radiation; however, UV-C and UV-B radiation below ~290 nm is absorbed by the stratospheric ozone layer. UV-B levels in the biosphere are highly dynamic and depend on large-scale variables such as stratospheric ozone, solar angle (latitude, season, time of day), altitude, tropospheric pollution, and cloud cover, in addition to small-scale variables such as surface reflectance and shading (114). However, UV-B is only a small fraction of the global irradiance, making up less than 0.1% of photons at Earth's surface (5). Nonetheless, the biological

effects of short-wavelength, high-energy UV-B photons are substantial because they are absorbed by numerous biologically relevant molecules, including DNA, to which UV-B radiation causes damage. UV-B therefore constitutes a potential abiotic stress factor for any organism exposed to sunlight, and particularly for sessile, photosynthetic plants. Yet, plants are capable of tolerating even the high-level UV-B of long, cloudless summer days, as can be appreciated on an outdoor hike through green fields.

Plant UV-B acclimation, in which the UVR8 photoreceptor–signaling pathway plays a key role, is essential to achieve UV-B tolerance. Generally, UV-B exposure regulates many aspects of plant metabolism, development, and morphology through the differential expression of numerous genes. Given the broad impact of UV-B on plants, it is crucial to understand the mechanisms of UV-B perception and signaling and its integration with other signaling pathways. This review focuses on our current understanding of the molecular mechanisms of UVR8 signaling and its outcomes, as well as UVR8 interactions with other plant signaling pathways and environmental responses. UV-B-stress signaling (i.e., when UV-B damage is perceived) and the ecological and agronomical aspects of UV-B radiation's impact on plants are not covered in any detail. For these aspects, we refer readers to the recent literature (11, 43, 44, 60, 66, 87, 101, 126, 139, 163, 165).

Acclimation: environmentally inducible process that leads to the physiological adjustment of an organism, allowing elevated tolerance to a potential environmental stress

E3 ubiquitin ligase: an enzyme that covalently links ubiquitin moieties to target proteins, often to induce their degradation through the proteasome

2. UVR8 ULTRAVIOLET-B PERCEPTION AND SIGNALING

UV-B-activated UVR8 signaling is mainly nuclear, affecting gene expression and thereby evoking many different physiological responses (12, 36, 72, 121, 156, 175). Currently, we know of two different mechanisms of UVR8 signaling. In the first, active UVR8 inhibits the E3 ubiquitin ligase CONSTITUTIVELY PHOTOMORPHOGENIC 1 (COP1), thereby stabilizing COP1 target transcription factors that in turn promote UV-B-induced gene expression changes (10, 36, 61, 77, 125). Related to this, the UVR8–COP1 interaction leads to the destabilization of a further set of transcription factors, resulting in gene repression under UV-B (50, 135, 140). The second UVR8 signaling mechanism involves the direct binding of UVR8 to specific transcription factors, a process that inhibits transcription factor binding to DNA and thus transcriptional activity (86, 88, 120, 172, 173). In contrast to the UVR8–COP1 pathway that is evolutionarily conserved from green algae to flowering plants (2, 47, 125, 136, 143), the functional conservation of UVR8 direct action on specific transcription factors remains an open question (**Table 1** provides an overview of UVR8-interacting proteins and their activities).

2.1. The UVR8 Photocycle

The core of the UVR8 photocycle comprises UVR8 forming homodimers in the inactive ground state, tryptophan-based UV-B absorption resulting in UVR8 monomerization, interaction of active UVR8 monomers with COP1, and, finally, redimerization of UVR8 to its homodimeric ground state that is facilitated by the action of REPRESSOR OF UV-B PHOTOMORPHOGENESIS 1 (RUP1) and RUP2.

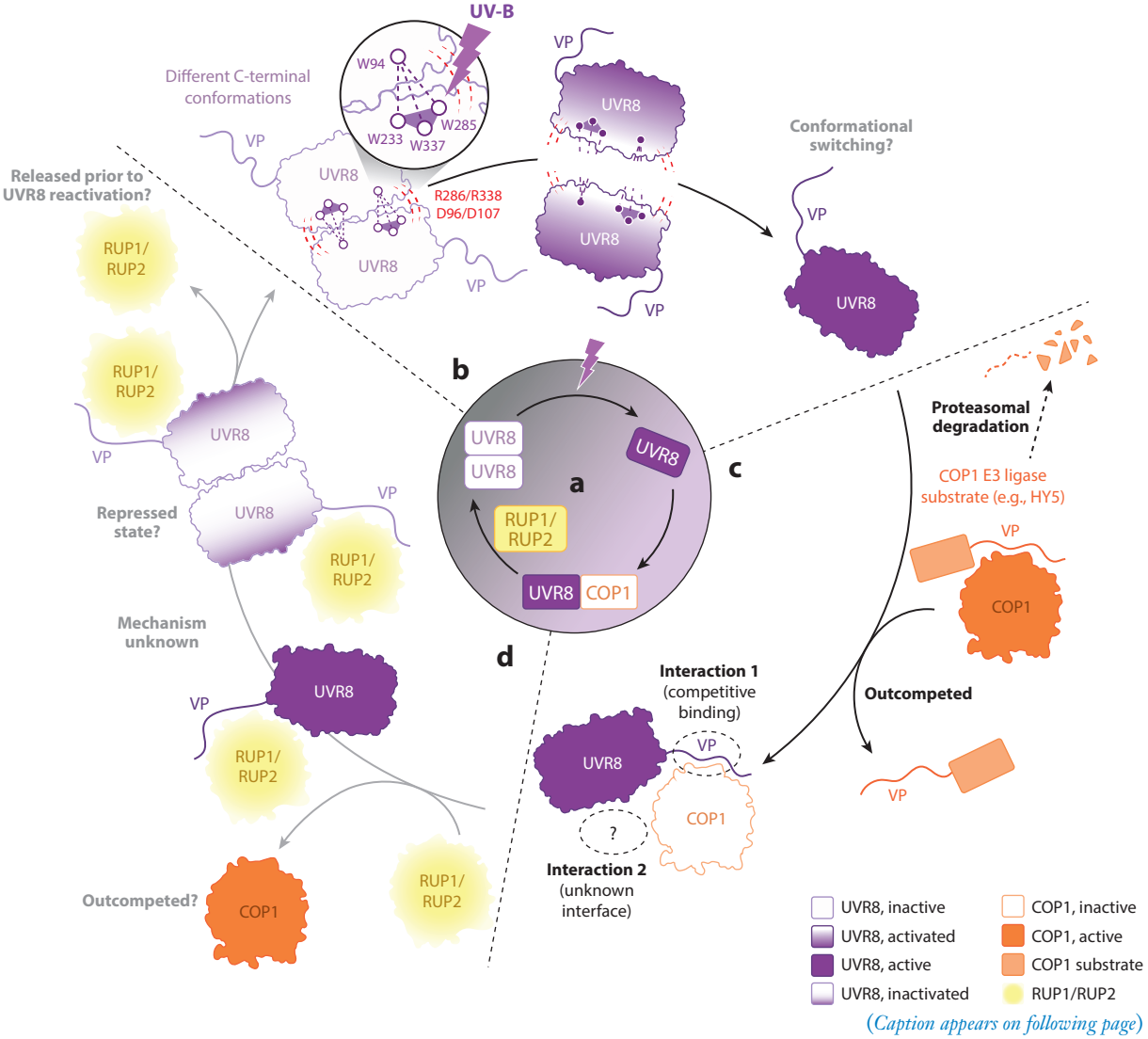
2.1.1. UVR8 forms a homodimer that monomerizes in response to ultraviolet-B. UVR8 is evolutionarily derived from Regulator of Chromatin Condensation 1 (RCC1), which functions as a guanine nucleotide exchange factor (GEF) for the Ran GTPase (74). However, UVR8 lacks Ran GEF activity (12) and has acquired specific UV-B photoreceptor activity (125) (**Figure 1**). The UVR8 core forms a doughnut-shaped, seven-bladed β-propeller fold (20, 166, 177). In its inactive ground state, UVR8 forms a homodimer arranged in a twofold symmetry configuration

Table 1 List of UVR8-interacting proteins involved in the UVR8 UV-B signaling pathway in *Arabidopsis thaliana* and *Chlamydomonas reinhardtii*

Arabidopsis thaliana			
Gene identifier	**Name**	**Interactions[a]**	**Activities; role in UV-B signaling**
AT5G63860	UVR8	UVR8 (125) COP1 (36) RUP1/RUP2 (46) WRKY36 (172) MYB13 (120) MYB73/MYB77 (173) BES1/BIM1 (86)	UV-B photoreceptor (125); monomerization upon UV-B absorption (125); positive regulator of UV-B-induced photomorphogenesis and acclimation (12, 36, 74)
AT2G32950	COP1	HY5/HYH (36, 77) HFR1 (49, 141) PIF5 (135) RUP1/RUP2 (124) DDB1 (61)	E3 ubiquitin ligase (110); inactivated by UVR8 interaction (77)
AT5G52250	RUP1	UVR8 (46) HY5 (124) COP1 (124) DDB1 (124)	Negative regulator (46); facilitation of UVR8 redimerization (52); E3 ubiquitin ligase targeting HY5 (124)
AT5G23730	RUP2	UVR8 (46) HY5 (124) COP1 (124) DDB1 (124) BBX1/CO (6)	Negative regulator (46); facilitation of UVR8 redimerization (52); E3 ubiquitin ligase targeting HY5 (124); repressor of photoperiodic flowering (6)
AT1G69810	WRKY36	UVR8 (172)	Transcription factor; repressor of *HY5* transcription; DNA binding inhibited by UVR8 interaction (172)
AT1G06180	MYB13	UVR8 (120)	Transcription factor; positive regulator of UV-B-induced cotyledon expansion; DNA binding differentially modulated by UVR8 interaction, depending on the target genes (120)
AT4G37260	MYB73	UVR8 (173)	Transcription factor mediating auxin-responsive gene expression and lateral root growth; DNA binding inhibited by UVR8 interaction (173)
AT3G50060	MYB77	UVR8 (173)	Transcription factor mediating auxin-responsive gene expression and lateral root growth; DNA binding inhibited by UVR8 interaction (173)
AT1G19350	BES1	UVR8 (86)	Transcription factor (bHLH) mediating BR-regulated gene expression and growth; DNA binding inhibited by UVR8 interaction (86)
AT5G08130	BIM1	UVR8 (86)	Transcription factor (bHLH) mediating BR-regulated gene expression and growth; DNA binding inhibited by UVR8 interaction (86)
Chlamydomonas reinhardtii			
Gene identifier	**Name(s)**	**Interactions[a]**	**Activities; role in UV-B signaling**
Cre05.g230600	UVR8	UVR8 (143) COP1/LRS1/HIT1 (143)	UV-B photoreceptor (2); monomerization upon UV-B absorption (143); positive regulator of UV-B-mediated gene regulation and acclimation (143); induction of NPQ components (2)
Cre02.g085050	COP1/ LRS1/ HIT1	UVR8 (143) CO (145)	E3 ubiquitin ligase; targeting CO for degradation (145)

[a]Comprehensive list of UVR8 interactions; others are limited to those affected by UVR8 activity.
Abbreviations: bHLH, basic helix-loop-helix; BR, brassinosteroid; NPQ, nonphotochemical quenching; UV-B, ultraviolet-B.

(20, 125, 166). Cross-dimer interactions are mediated by a complex network of charged residues [arginine (Arg, R) and aspartic acid (Asp, D)] arranged in complementary positions. Of these, R286, R338, D96, and D107 are crucial for dimer stability (residue numbers are according to the *Arabidopsis thaliana* UVR8 sequence throughout this review) (20, 55, 63, 166). Unlike other plant photoreceptors (42), UVR8 does not require an extrinsic chromophore; the UV-B-absorbing property of intrinsic Trp residues provides chromophore function (20, 125, 166). UVR8 possesses 14 Trp residues: 6 residues are part of the β-propeller structure, 7 are located at the dimer interface, and 1 is in the C-terminal extension (20, 125, 166). Whereas the C-terminal Trp residue (W400) seems dispensable for function, three of the β-propeller Trp residues are important for the structural integrity of the UVR8 protein (107). The seven interface Trp residues can be separated further into two groups. One of these groups comprises W233, W285, and W337, which are part of a so-called Trp pyramid that interacts with W94 from the facing monomer (20). W285 and W233 play



(*Caption appears on following page*)

Figure 1 (*Figure appears on preceding page*)

Structural and mechanistic details of the core UVR8 photocycle. (*a*) In a simplified scheme of the UVR8 photocycle, the UVR8 homodimer is monomerized after UV-B absorption, leading to an active UVR8 monomer that interacts with COP1 to induce downstream signaling. UVR8 is reverted to its inactive state by redimerization through the action of RUP1 and RUP2 proteins. (*b*) In its inactive homodimeric state (*white*), UVR8 exists in different conformations whereby the C terminus may be restricted and bound to the UVR8 core β-propeller domain. In all cases, the VP motif–containing C terminus is shielded from interaction with COP1, preventing activity in noninductive conditions. The UVR8 homodimer is stabilized by a network of salt bridge interactions (*red dashed lines*), e.g., between specific Arg and Asp residues. UV-B photons are absorbed in the Trp pyramid (*close-up in circle*). The activated UVR8 (*violet*) is monomerized, and several conformations of the monomer exist, some of which may be primed for interaction with COP1. (*c*) UVR8 uses a cooperative binding mechanism to interact with COP1 (*dark orange*; only its structurally solved WD40 domain is shown) with high affinity. The UVR8 β-propeller core interacts with the WD40 domain of COP1 through an unknown interaction interface. Simultaneously, the UVR8 VP motif outcompetes COP1 substrates for binding to COP1. In this manner, COP1 substrates (*light orange*) are protected from polyubiquitination and proteasomal degradation and may induce photomorphogenic responses. (*d*) RUP1 and RUP2 (*yellow*; structure is not solved but contains a WD40 domain homologous to COP1) interact with the VP motif of UVR8, presumably in a manner homologous to COP1. Because RUP proteins and COP1 bind the same UVR8 VP domain, COP1 may be outcompeted, preventing its interaction with UVR8. The biochemical mechanism by which RUP proteins promote UVR8 redimerization is poorly understood. It is also unknown whether RUP proteins remain bound following UVR8 redimerization and whether this represents a repressed state whereby UVR8 reactivation is not possible. In the latter case, RUP proteins may need to be released from UVR8 to allow UVR8 reactivation. Solid arrows are part of the UVR8 photocycle, those in black are part of the cycle associated with UVR8 activation and activity, and those in grey are part of the cycle associated with UVR8 redimerization and inactivation. The dashed arrow in panel *c* is not part of the main UVR8 photocycle but indicates COP1 activity. Abbreviations: Arg, arginine; Asp, aspartic acid; D, aspartic acid; R, arginine; Trp, tryptophan; UV-B; ultraviolet-B; VP, Val-Pro; W, tryptophan.

major roles in the perception of UV-B photons (51, 63, 83, 100, 107, 125, 166, 168, 170). W285 plays a particularly crucial role, as W285 mutation to Phe ($UVR8^{W285F}$) abolishes UV-B perception (125). Interestingly, $UVR8^{W285F}$ exhibits a weak response to UV-C, which agrees with the spectral properties of Phe (20). The other four Trp residues at the dimer interface (W94, W198, W250, and W302) form a group that seems dispensable for UVR8 function in vivo (107). However, it has recently been suggested that these residues, together with the tryptophans buried in the β-propeller core, form a UV-B light-harvesting antenna (85). Indeed, 26 structural tryptophan residues per UVR8 homodimer (13 out of the 14 in each monomer) constitute an energy-transfer network, funneling all excitation energy to the Trp pyramid center, where the excitation-induced monomerization is initiated for signal propagation (85). The overall light-perception quantum efficiency of the pyramid Trps was thereby determined to be increased from 35% (direct excitation) to 73% (light harvesting) (84, 85).

Upon UV-B absorption, the UVR8 homodimer converts instantaneously to monomers (20, 90, 100, 125, 166) (**Figure 1**). Several studies have attempted to characterize the UVR8 monomerization mechanism. Disruption of cation–π interactions between Trp and Arg residues at the dimer interface may lead to conformational changes surrounding R286 and R338, thus breaking key cross-dimer interactions (166). Other studies proposed that Trp UV-B absorption leads to the neutralization of salt bridge interactions by proton-coupled electron transfer from the pyramid Trp to proximal Arg residues (20, 94, 97, 169). A theoretical study suggested that W233 acts as a sink for excitation energy and that, after UV-B absorption, this energy causes a charge separation between W233 and W285 that destabilizes neighboring salt bridges (159). Dynamic crystallography comparing UVR8 crystals before and after UV-B exposure suggests that UV-B activation also involves a reorientation of the chromophores W285 and W233 due to charge separation resulting in the ejection of a water molecule, which combined weakens the salt bridge network, leading to monomerization (177). Spectroscopic analysis also supports a rearrangement of Trp and Arg/Asp residues after UV-B absorption (53).

2.1.2. Ultraviolet-B-activated UVR8 interacts with COP1. COP1 is a crucial component of the UVR8 signaling pathway (109). COP1 forms an E3 ubiquitin ligase complex together with the four SUPPRESSOR OF PHYA-105 proteins (SPA1–4), and this complex represses photomorphogenesis in the dark by directing photomorphogenesis-promoting factors to be degraded through the 26S proteasome system (57). UVR8 interacts via two distinct domains with the WD40-repeat domain of COP1 (23, 36, 125, 174) (**Figure 1**). Firstly, the β-propeller core domain of UVR8 interacts in a UV-B-specific manner with COP1 (77, 174). This interaction is likely triggered after monomerization by the exposure of a UVR8 interaction surface that is hidden by the dimeric state, as supported by the interaction of constitutively monomeric mutant versions of UVR8 with COP1 in the absence of UV-B (55, 63, 116). However, this interaction alone is unable to initiate UV-B responses in vivo (55, 174). Secondly, the C-terminal domain of UVR8 (C44, which includes a so-called C27 subregion) interacts with COP1 and is crucial for UVR8 signaling (23, 77, 174). The UVR8 C27 subregion contains a Val-Pro (VP) motif found in several COP1-interacting proteins (77, 167, 174). Mutation of the VP motif to alanine (Ala) residues abolishes the UVR8 C-terminal interaction with COP1 and, in turn, downstream UV-B physiological responses (174). In agreement is the finding that mutagenesis of the key VP-interacting residues in COP1 abolishes UV-B marker gene induction (77). The flexible UVR8 C-terminal extension was not included in the solved structures (20, 166, 177) but was shown to contribute to the adoption of multiple conformations of UVR8, some of which may represent the signaling active state (17). Recently, a C17 subregion of the UVR8 C-terminal domain (the last 17 amino acids, adjacent to the C27 subregion) was shown to interact with the UVR8 N-terminal domain, possibly contributing to these different C-terminal conformations (91). Deletion of this C17 subregion ultimately leads to enhanced UV-B photomorphogenesis by modulating the affinity of the UVR8–COP1 interaction (91).

Photomorphogenesis: photoreceptor-mediated regulation of plant growth, development, and differentiation in response to light

Together, the UVR8 N- and C-terminal interactions with COP1 lead to a strong association through cooperative binding (77). Upon UV-B-induced monomerization, the C27 domain appears to be released from structural constraints imposed by the UVR8 core (53, 77, 174, 176). This C27 release mechanism is maintained in UVR8 monomeric mutant versions for which UV-B exposure is required to initiate signaling, such as $UVR8^{D96N,D107N}$ and $UVR8^{G101S}$, thus indicating that C27 release is independent of UVR8 oligomeric state (55, 116). The UVR8 VP motif is similar in sequence to the VP motifs of COP1 degradation targets; however, using a cooperative binding mechanism, the affinity of UV-B-activated UVR8 for COP1 is stronger than that of COP1 targets (77). Underlining the importance of the cooperative binding and competition mechanism, chimeric UVR8 photoreceptors with VP motifs from other proteins are capable of inducing UV-B signaling, suggesting that there is no specificity per se in the UVR8 VP motif sequence (77).

In addition, the COP1/SPA complex functions as a substrate receptor of a larger E3 ligase complex containing a CULLIN 4 (CUL4)-DAMAGED DNA BINDING PROTEIN 1 (DDB1) scaffold (19), although there are reports suggesting an activity for COP1 on its own, at least in vitro (130, 134). Interaction with UVR8 disrupts the CUL4-DDB1–COP1/SPA complex and a unique UVR8–COP1/SPA complex is formed instead that interrupts target protein ubiquitination (61). Of note, although closely related in sequence to COP1, SPA proteins only interact with UVR8 indirectly through COP1 (51, 61).

Several mutant variants of UVR8 ($UVR8^{W285A}$, $UVR8^{G101S}$, $UVR8^{D96N,D107N}$, $UVR8^{R338A}$) interact with COP1 in a constitutive manner (55, 63, 107, 116, 125). $UVR8^{W285A}$–COP1 and even stronger $UVR8^{G101S,W285A}$–COP1 interactions lead to constitutive photomorphogenic phenotypes, presumably due to constant COP1 inactivation (51, 63, 116). On the other hand, $UVR8^{G101S}$, $UVR8^{D96N,D107N}$, and $UVR8^{R338A}$ enact UV-B-dependent responses while

interacting constitutively with COP1; in these cases, UV-B-induced release of the C terminus may be the critical activating mechanism (55, 63, 116). Interestingly, $UVR8^{W285A}$ interacts with COP1 via its C-terminal domain alone (174).

2.1.3. RUP1 and RUP2 facilitate UVR8 ground state reversion by redimerization. RUP1 and RUP2 are WD40-repeat proteins closely related to COP1 and SPA1–SPA4 (46). However, RUP proteins consist only of the WD40-repeat domain preceded by a short N-terminal extension of unknown function; they lack the RING and coiled-coil domains of COP1 and the kinase-like domain of SPA proteins (46). *RUP1* and *RUP2* transcripts are UV-B inducible in a UVR8- and COP1-dependent manner (46). In contrast to COP1, which shows much higher affinity for active monomeric UVR8 (36, 77, 125), RUP1 and RUP2 interact with both UVR8 homodimer and monomers, although they also have a somewhat stronger affinity for active UVR8 monomers (23, 46, 52, 89, 116, 174). In contrast to the UVR8–COP1 interaction, apparently only the UVR8 C-terminal VP motif is involved in the UVR8 interaction with RUP1 and RUP2 (23, 174). Thus, in the inactive state, the UVR8 VP motif seems accessible to RUP1 and RUP2 but much less so to COP1. By contrast, the strong UV-B-dependent UVR8–COP1 interaction is likely associated with the additional interaction of COP1 with the UVR8 core, and this interaction is lacking in the case of RUP1 and RUP2 (23, 174).

RUP1 and RUP2 facilitate efficient redimerization of UVR8 in vivo (**Figure 1**). In *Arabidopsis*, UVR8 redimerization is completed within about 2 h, whereas redimerization of recombinant UVR8 in vitro takes about 30 h (52, 54). In vivo, RUP1 and RUP2 play redundant roles in mediating UVR8 redimerization and thus adjusting the relative dimer/monomer photoequilibrium; there is slow UVR8 redimerization in *rup1 rup2* mutant plants and an exaggerated UV-B photomorphogenic phenotype (40, 46, 52). However, *rup2* mutant plants show a stronger phenotype than *rup1*, in line with respective RUP1 and RUP2 protein levels (46, 89). It is important to note that an absolute UVR8 dimer/monomer switch only occurs under experimental conditions with saturating UV-B. In more natural settings, UVR8 exists in a dimer/monomer photoequilibrium (40, 89, 103). Interestingly, temperature is an important parameter regulating this photoequilibrium, with an elevated redimerization rate observed at higher temperatures, which is abolished in a *rup1 rup2* mutant background (40). Additionally, UVR8 redimerization is promoted by blue light through cryptochrome-mediated transcriptional activation of *RUP1* and *RUP2* (144).

The exact mechanism through which RUP1 and RUP2 facilitate UVR8 redimerization remains somewhat unclear. Overexpression of RUP2 strongly inhibits UVR8 signaling and appears to render UVR8 constitutively dimeric (46, 52); however, it is unknown whether this is due to rapid redimerization or inhibition of monomerization. Whereas RUP1 and RUP2 facilitate UVR8 redimerization, it is not clear if the dissociation of RUP1 and RUP2 from UVR8 is necessary for subsequent UV-B-induced UVR8 monomerization. Nevertheless, the UVR8–COP1 interaction is also inhibited by RUP2 overexpression; however, it remains poorly understood whether RUP1 and RUP2 directly inhibit the interaction (through competitive binding to the UVR8 VP motif) or whether the disrupted interaction is merely because of UVR8 redimerization (52). COP1 does not play a role in regulating UVR8 redimerization because it occurs in the *cop1–4* mutant with kinetics comparable to that in the wild type (52, 54).

RUP1 and RUP2, similar to COP1 and SPA1–4, contain WDxR motifs that are present in DDB1-interacting proteins, suggesting that RUP1/RUP2 function as substrate receptors in CUL4–DDB1-based E3 ubiquitin ligase complexes as well (19, 124, 179). Although WDxR motifs are not directly involved in the interaction with DDB1 (133, 149), mutating these motifs abolishes the interaction (19, 124, 179). Mutating the WDxR motifs in RUP2 abolishes its function as a repressor of UV-B photomorphogenesis (124). Additionally, it was reported that the

CUL4–DDB1 substrate adaptor DWD HYPERSENSITIVE TO UV-B 1 (DHU1) negatively regulates UVR8-mediated responses by directly interacting with COP1 and RUP1, although the mechanism remains unclear (73).

2.2. Subcellular Localization and Activity of UVR8

In its ground state, UVR8 localizes predominantly to the cytosol in *Arabidopsis* seedlings, but a fraction localizes to the nucleus (12, 72). Upon activation by UV-B, UVR8 monomers accumulate in the nucleus, although UVR8 contains no obvious nuclear localization signal (NLS) (72). UVR8 nuclear localization is indeed required for UV-B signaling; however, the functional role of the additional UV-B-induced UVR8 nuclear accumulation is not completely understood (121, 175). Of note, even under UV-B, a significant part of UVR8 remains localized to the cytosol (72, 121, 175), leaving open the possibility that there is a presently unknown function of cytosolic UVR8. Similar to *Arabidopsis*, UV-B-induced UVR8 nuclear accumulation has been demonstrated in the liverwort *Marchantia polymorpha*, the moss *Physcomitrella patens*, and the alga *Chlamydomonas reinhardtii* (75, 136, 145).

Intriguingly, COP1 is required for UVR8 nuclear accumulation in response to UV-B (121, 175). COP1 includes both an NLS and a nuclear export signal and exhibits light-regulated nucleocytosolic partitioning (78, 112, 113, 137). Moreover, COP1 shows specific UV-B-mediated nuclear accumulation, likely associated with the posttranslational stabilization of COP1 under UV-B and, as detailed above, interacts with UVR8 in a UV-B-dependent manner (36, 77, 109, 125). Thus, COP1 may facilitate a piggyback style of activated UVR8 coimport into the nucleus. Alternatively, UVR8 may enter the nucleus in a COP1-independent manner, through either diffusion of UVR8 monomers or their interaction with presently unknown NLS-containing proteins. For the latter, nuclear COP1 would be required to keep activated UVR8 in the nucleus (176). Importantly, however, artificial enhancement of nuclear UVR8 levels does not suppress the *cop1* UV-B phenotype (175). Therefore, irrespective of the molecular mechanism underlying UVR8 nuclear accumulation under UV-B, COP1 plays a dual role in UVR8 nuclear accumulation and downstream signaling.

2.3. UVR8-Mediated Gene Regulation

Although it is well established that activation of UVR8 leads to UV-B-dependent changes in the expression of a broad set of genes (12, 36, 74), the underlying mechanisms are poorly understood. This process involves changes in the activity of different transcription factors, including the stabilization of COP1 substrates such as the basic leucine zipper transcription factors ELONGATED HYPOCOTYL 5 (HY5) and HY5-HOMOLOG (HYH) (10, 14, 36, 61, 77, 138); degradation of the basic helix-loop-helix (bHLH) transcription factors PHYTOCHROME INTERACTING FACTOR 4 (PIF4) and PIF5 (50, 135, 140); and UVR8 interaction–induced interference in the DNA binding of specific transcription factors, including BRI1-EMS-SUPPRESSOR 1 (BES1), BES1-INTERACTING MYC-LIKE 1 (BIM1), MYB DOMAIN PROTEIN 13 (MYB13), MYB73, MYB77, and WRKY DNA-BINDING PROTEIN 36 (WRKY36) (86, 88, 120, 172, 173) (**Figure 2**). These three mechanisms are described in more detail below.

Furthermore, chromatin immunoprecipitation (ChIP) experiments have suggested that UVR8 binds directly to chromatin via histone H2B (12, 22, 23, 36, 72). The idea that UVR8 chromatin association at UV-B-regulated target genes may play a role in UVR8 signaling was motivated by sequence similarity between UVR8 and chromatin-associated RCC1 proteins (12, 74). It was proposed that chromatin-associated UVR8 recruits and/or activates chromatin-modifying enzymes

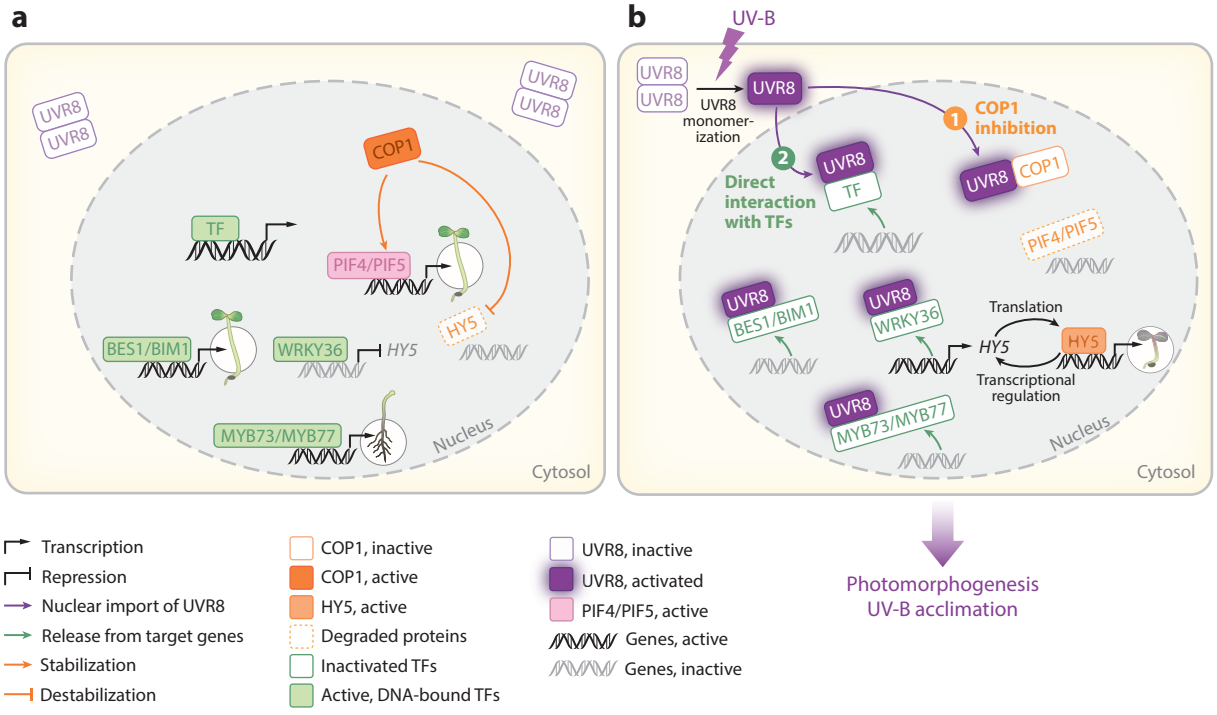


Figure 2

Mechanisms of UVR8-mediated gene regulation. (*a*) UVR8 exists as an inactive homodimer in the cytosol and also to a lesser extent in the nucleus (not depicted). The E3 ubiquitin ligase COP1 destabilizes the TF HY5 (*dashed lines* represent proteasomal degradation), whereas COP1 promotes the stability of TFs PIF4 and PIF5 that regulate genes associated with elongation growth. In addition, brassinosteroid-responsive TFs BES1 and BIM1 regulate genes associated with elongation growth, WRKY36 represses *HY5* expression, and auxin-responsive TFs MYB73 and MYB77 regulate the expression of genes associated with lateral root formation. (*b*) UVR8 activation by UV-B triggers its monomerization and concomitant accumulation in the nucleus (in a COP1-dependent manner; not depicted). Nuclear UVR8 reprograms gene expression through two major molecular mechanisms: (❶) Activated UVR8 interacts and represses COP1 activity, which leads to stabilization of COP1 substrates, in particular HY5, which is a key positive regulator of photomorphogenesis. In addition, COP1 inactivation leads to destabilization of PIFs through an unknown mechanism. (❷) UVR8 interacts directly with and represses TFs by interfering with their DNA-binding capacity. Inhibition of WRKY36 alleviates its repression of *HY5* expression. Inhibition of BES1/BIM1 and MYB73/MYB77 represses brassinosteroid- and auxin-related gene expression, thus repressing hypocotyl elongation and lateral root formation, respectively. Note that MYB13, which plays a role in UVR8-mediated cotyledon expansion, has been omitted for simplicity. Abbreviations: PIF, phytochrome interacting factor; TF, transcription factor; UV-B, ultraviolet-B.

and/or transcription factors (22). Although UVR8 is indeed required for UV-B-induced histone H3 lysine K9 and/or K14 acetylation at UVR8-regulated gene loci, no mechanistic link between UVR8 and chromatin modification could be established (158). It is of note that the proposed UVR8 chromatin association was apparently unaffected by the presence or absence of UV-B in gel-based ChIP analyses, which, however, may not be satisfactorily quantitative (12, 22, 158). On the other hand, a more quantitative ChIP analysis by real-time polymerase chain reaction (PCR) did not support any chromatin association of UVR8 (9). Additionally, critical histone- and DNA-interaction residues known from the *Drosophila* DmRCC1 crystal structure are not conserved in the UVR8 structure, and recombinant UVR8 fails to bind nucleosomes in vitro (9). Thus, whether UVR8 signaling includes direct chromatin-binding activity of the receptor itself remains controversial, and, if true, its functional significance awaits further clarification.

2.3.1. COP1-targeted transcriptional regulators are stabilized in response to ultraviolet-B. UV-B activated UVR8 has increased affinity to COP1, outcompeting COP1-interacting transcription factors, allowing their stabilization, accumulation, and activities.

2.3.1.1. The basic leucine zipper transcription factors HY5 and HYH. HY5 plays a central role in UV-B signaling (10, 12, 14, 62, 138, 150). HY5 and its homolog HYH form homo- and heterodimers and are positive regulators of photomorphogenesis (58). HY5 was shown to directly bind DNA and regulate gene expression at numerous loci (15, 79). HY5 and HYH show partially overlapping function in signaling pathways downstream of the phytochrome, cryptochrome, and UVR8 photoreceptors; however, HY5 plays the major role under all light conditions (14, 37, 58, 138). HY5 is a substrate for the COP1/SPA E3 ubiquitin ligase complex and is stabilized in response to light, including UV-B (36, 58, 61, 78, 110). Due to the UVR8–COP1 interaction, HY5 is rapidly posttranscriptionally stabilized under UV-B (36, 61, 77, 111). This is balanced by the accumulation of RUP1 and RUP2 under UV-B, which were proposed to function as part of a CUL4–DDB1 ubiquitin ligase complex targeting HY5 (124). HY5 accumulation in a *rup2* mutant is thought to be because of (*a*) sustained activity of UVR8 and thus COP1 inactivation (36, 52) and (*b*) decreased ubiquitination through CUL4–DDB1–RUP2 (124). RUP2 also interacts directly with COP1, thereby inhibiting the COP1–HY5 interaction, and COP1 negatively regulates the levels of RUP2 (124). Together, these mechanisms are thought to contribute to the regulation of HY5 levels under UV-B (124).

In addition to posttranslational stabilization, UV-B exposure results in rapid and transient induction of *HY5* and *HYH* expression in a UVR8- and COP1-dependent manner (12, 13, 29, 36, 109, 150). It has recently been shown that WRKY36 inhibits *HY5* transcription and that the UVR8–WRKY36 interaction represses WRKY36 activity, thus promoting *HY5* transcription (172). Moreover, it is of note that both HY5 and HYH are required for UVR8-mediated transcriptional activation of *HY5* (10). Indeed, HY5 and HYH bind a T/G-box in the *HY5* promoter, which is a *cis*-regulatory element required for *HY5* expression induction in response to UV-B (1, 10).

It is assumed that HY5 and HYH regulate most UVR8-mediated gene induction (69, 142). In addition to its own promoter, HY5 associates with promoter regions of its UV-B-induced target genes, including *CHS*, *RUP1*, *RUP2*, *MYB12*, and *COP1*; UV-B promotes these associations in a UVR8-dependent manner (10, 138). For *COP1*, *MYB12*, and *CHS*, it was shown that HY5 binds via the ACGT-containing elements (ACEs) present in their promoters (4, 62, 138). How exactly HY5 activates the expression of its target genes in response to UV-B is an open question, as HY5 lacks transcriptional activation activity (4, 138); however, a recently identified group of B-box-containing transcriptional regulators that work in concert with HY5 in response to visible light may be involved (16).

2.3.1.2. Zinc-finger proteins of the BBX family. The B-box (BBX) proteins are a large family (32 members in *Arabidopsis*) of zinc-finger transcription factors, many of which have been implicated in photomorphogenic responses to visible light, often in concert with COP1 and HY5 (151).

The flowering regulator CONSTANS (CO/BBX1) is a crucial component that induces flowering in response to photoperiod (152). COP1-mediated proteolysis plays an important role in the posttranscriptional control of CO levels (67, 92). In short days under UV-B, RUP2 was shown to repress CO-induced flowering by repressing CO chromatin binding and transcriptional activity (6). It should be noted that no evidence for CO stabilization under UV-B associated with flowering time regulation has been found in CO-overexpression lines (6).

BBX24 is a negative regulator of photomorphogenesis (64), including UV-B photomorphogenesis (71). *BBX24* expression is induced and the BBX24 protein accumulates under UV-B. BBX24 then interacts with COP1 and HY5 and represses HY5 accumulation and transcriptional activity under UV-B (71). BBX31 is also a negative regulator of photomorphogenesis under white light, acting in part independently of HY5 even though *BBX31* itself is transcriptionally repressed by HY5 (56). *BBX31* is induced after exposure to UV-B, and BBX31 is a positive regulator of UV-B signaling (171).

The single-celled, green alga *C. reinhardtii* (herein referred to as *Chlamydomonas*) contains a single CO-like BBX protein (CrCO), which is stabilized under UV-B and transcriptionally induces genes involved in algal photoprotection (145). CrCO is targeted for degradation by the *Chlamydomonas* COP1/SPA E3 ligase complex, and UV-activated UVR8 abrogates this mechanism through interaction with CrCOP1/LRS1, leading to CrCO stabilization (41, 145).

2.3.1.3. The atypical bHLH protein HFR1. LONG HYPOCOTYL IN FAR-RED 1 (HFR1) is a protein related to the PIF family of bHLH regulators; however, it lacks a basic domain, which prevents binding to DNA (33, 59). HFR1 heterodimerizes with PIF proteins, inhibiting their binding to chromatin (59). HFR1 contains a VP motif, and its stability is regulated by COP1 (31, 77). Under UV-B, UVR8 outcompetes HFR1 for COP1 binding, and HFR1 is strongly stabilized (49, 77, 141).

2.3.2. PIF4 and PIF5 are degraded in response to ultraviolet-B. UVR8-mediated repression of several hypocotyl elongation–related genes is independent of HY5 and HYH but largely associated with UVR8-dependent PIF4 and PIF5 degradation, a process that consequently diminishes PIF4/5 target promoter occupancy (140). UV-B stabilization of the COP1 target and PIF repressor HFR1 also contributes to altered PIF4/PIF5 activity (49, 141).

UVR8 promotes rapid PIF4 and PIF5 degradation via the ubiquitin-proteasome system (50, 135, 140). Interestingly, PIF5 interacts with COP1; however, this interaction counterintuitively promotes PIF5 stabilization in light-grown plants (135). Binding of UV-B-activated UVR8 to COP1 disrupts PIF5 stabilization, underlying the mechanism by which PIF5 levels are promptly reduced in full sunlight (135). Whether PIF4 stability is similarly regulated through direct interaction with COP1 is unknown. The E3 ubiquitin ligase mediating PIF4 and PIF5 polyubiquitination and proteasomal degradation in response to UVR8 activation remains to be determined.

2.3.3. Transcription factors are regulated through direct interaction with UVR8. There are documented instances of direct physical interaction between UV-B-activated nuclear UVR8 and plant transcription factors. WRKY36 binds to the W-box motif in the *HY5* promoter and inhibits its expression (172). Nuclear UVR8 directly interacts with WRKY36, which inhibits WRKY36-DNA binding, thereby promoting *HY5* expression (172). It seems that both homodimeric and monomeric forms of UVR8 interact with WRKY36, but the UV-B-specificity is proposed to be associated with UV-B-mediated nuclear accumulation of UVR8, which results in stronger WRKY36 inhibition (172). Nuclear UVR8 also interacts with BIM1 and the dephosphorylated active form of BES1, which inhibits their DNA binding (86). Finally, UVR8 in the nucleus also interacts with MYB73 and MYB77, inhibiting their DNA-binding activities and thereby repressing target gene transcription (173). A further UVR8-interacting MYB transcription factor, MYB13, is UV-B inducible and mainly expressed in cotyledons. Surprisingly, interaction with UVR8 promotes the in vitro and in vivo binding of MYB13 to the promoters of *CHS* and *CHALCONE ISOMERASE* (*CHI*), yet inhibits MYB13 binding to *SMALL AUXIN UP RNA 27/28* (*SAUR27/28*) and *SAUR66* promoters in vitro, although this is apparently counteracted by MYB13

accumulation under UV-B in vivo (120). Thus, through direct interaction, UV-B-activated UVR8 differentially modulates MYB13 affinity with its target genes; however, the underlying mechanism for these different outcomes remains to be determined (120).

The discovery of the UVR8–WRKY36, UVR8–BES1/BIM1, and UVR8–MYB protein interactions indicates that a further UVR8 signaling pathway exists in addition to COP1 inactivation. This pathway is based on UV-B-dependent nuclear accumulation of UVR8 and inhibition of the DNA-binding activities of specific transcription factors through their direct interaction with UVR8 (88). However, it remains to be shown why artificial UVR8 nuclear localization in *cop1* mutant backgrounds does not result in UV-B-responsive gene expression (175), which suggests that COP1 is also required for this alternate mode of UVR8 signaling.

2.3.4. FHY3 contributes to the ultraviolet-B-induced expression of *COP1*. HY5 binds an ACE in the *COP1* promoter and promotes *COP1* gene expression in response to UV-B (62). Moreover, a FAR-RED ELONGATED HYPOCOTYL 3 (FHY3) binding site (FBS) in the *COP1* promoter functions as a further UV-B-responsive *cis*-element (62). In addition to *COP1* regulation, FHY3 also contributes to the UV-B-induced expression of *HY5* and other genes, leading to increased tolerance to UV-B exposure (62). Interestingly, UV-B diminishes physical interaction between HY5 and FHY3, thus allowing their independent binding to distinct ACE and FBS *cis*-elements and consequential *COP1* expression regulation in a combinatorial, noncompetitive manner (62). How UVR8 signaling negatively affects the HY5–FHY3 interaction remains to be clarified.

2.4. UVR8 Signaling in Diverse Plant Species

An inspection of plant genome databases reveals numerous putative UVR8 homologs within the green lineage, with conservation of the UVR8 key functional amino acids from green algae to flowering plants (38, 47, 125, 142). Two main features of the UVR8 sequence are diagnostic: first, the presence of critical Trp residues, particularly a triad corresponding to *Arabidopsis* W233, W285, and W337 within conserved GWRHT motifs, which underlies the capacity to perceive and transduce UV-B signals, and, second, the presence of a C-terminal domain containing the VP motif, required for interaction with core components of the downstream signaling pathway (23, 38, 77, 125, 142). Experimentally, complementation assays in *Arabidopsis* confirmed that the UVR8 molecular mechanism of action is conserved among UVR8 orthologs from numerous plant species (e.g., 95, 96, 136, 143, 180), as did knockout/knockdown of the UVR8 candidates that led to impaired UV-B signaling (2, 75, 93, 145). In agreement is the finding that core players of UVR8 signaling (COP1, RUP proteins, HY5) are also largely conserved across Viridiplantae (47, 76, 132, 143, 145, 148). The high degree of sequence conservation and early appearance of UVR8 in plant evolution underscore its functional importance. It is likely that the evolution of the UVR8 photoreceptor from its ancestral eukaryotic RCC1-encoding gene (74) provided these early photosynthetic organisms with tolerance to the high-level UV-B that existed before the establishment of a mature stratospheric ozone layer (68, 128).

In *Arabidopsis* and many other plants, *UVR8* exists as a single-copy gene (74, 142). Nonetheless, in some species (e.g., *Physcomitrella patens*), UVR8 is encoded by multiple gene copies; however, currently there is no information available about whether differential expression or subfunctionalization among different UVR8 proteins has evolved in any single species, similar to other photoreceptor families, or whether the paralogs are fully redundant in their functions. On the other hand, the seagrass *Zostera marina*, an angiosperm adapted to a marine lifestyle, possesses the only known plant genome that apparently lacks a UVR8 photoreceptor–encoding gene (108).

Table 2 Ultraviolet-B responses in plants and their UVR8 dependence

UV-B response[a]	Plant	UVR8[b]	Reference(s)
Hypocotyl growth inhibition	At, Sl	+	36, 86, 93, 140, 172
Cotyledon expansion	At	+	8, 120
Phototropism (hypocotyl)	At	+[c]	153, 154
Phototropism (inflorescence stem)	At	+	157
Leaf development (including rosette growth inhibition)	At	+	8, 39, 164
Downward leaf curling	At	+	39
Stomatal opening	At	+	147
Flowering	At	+[d]	6
Accumulation of UV-B-absorbing metabolites (e.g., flavonols)	At, Mp	+	36, 74, 75
Accumulation of anthocyanins	At, Sl	+	36, 93
Acclimation and UV-B tolerance	At, Cr, Mp, Sl	+	36, 74, 93, 122, 143
Photoprotection (photosynthetic efficiency)	At, Cr	+	2, 26, 143, 145, 146
Defense responses	At	+	28
Root microbiome	Na	+	131
Entrainment of circadian clock	At	+	37
Salt and osmotic stress tolerance	At	+	35
Antagonizing shade avoidance	At	+	50, 98, 141
Antagonizing auxin responses	At	+	50, 153, 173
Antagonizing thermomorphogenesis	At	+	49
UV-B stress tolerance and MAP kinase activation (unacclimated)	At	–	43

[a]Note that *uvr8* and wild type are indistinguishable in the absence of UV-B.
[b]UV-B responses not yet known to be or not UVR8 regulated are omitted.
[c]Phenotype is detectable only in *phot1 phot2* double-mutant background.
[d]Phenotype is detectable only in *rup2* mutant background.
Abbreviations: At, *Arabidopsis thaliana*; Cr, *Chlamydomonas reinhardtii*; MAP, mitogen-activated protein; Mp, *Marchantia polymorpha*; Na, *Nicotiana attenuata*; Sl, *Solanum lycopersicum*; UV-B, ultraviolet-B.

3. UVR8 PHOTORECEPTOR–MEDIATED ULTRAVIOLET-B RESPONSES

UV-B has many effects on plants, but it remains unknown whether all the responses characterized are UVR8 photoreceptor dependent (**Table 2** lists those responses for which UVR8 implication was tested). Currently, there are *uvr8* null mutants available in *Arabidopsis* (12, 36, 74), tomato (*Solanum lycopersicum*) (93), *Chlamydomonas* (2, 45, 146), and *M. polymorpha* (75), as well as RNAi-silenced lines in tomato (81), and there is a possibility of creating further null mutants in other species with the advent of CRISPR/Cas9 and related technology (119). Given these mutants and technologies, it should be straightforward in the future to clarify the link between a specific UV-B response and UVR8 signaling, even without being limited to a particular current model system. For UVR8-independent responses, it will be of great interest to identify the UV-B perception mechanism, which may be either direct through an as-yet-unknown photoreceptor (photomorphogenic signaling, as for UVR8) or indirect through recognition of damage-associated stress (nonspecific signaling) (43, 70, 82, 106, 129, 150).

Arabidopsis has been instrumental in identifying UVR8 and revealing the physiological responses to UV-B that are mediated by the UVR8 photoreceptor. Experiments to study UVR8 activity are usually performed under experimentally defined, nondamaging supplemental UV-B (i.e., photomorphogenic conditions), thus preventing interference by stress signaling (36, 43).

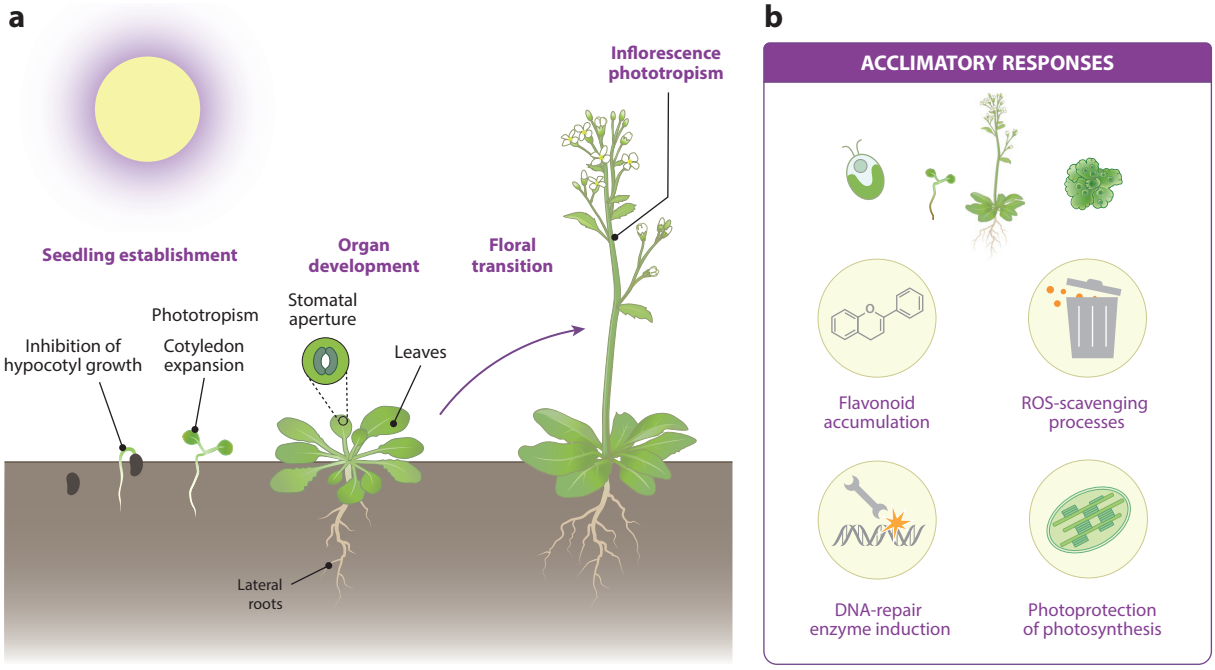


Figure 3

UVR8-mediated UV-B signaling affects growth, development, and survival throughout the plant life cycle. (*a*) Effects of UVR8 signaling on growth and development in *Arabidopsis*, from early seedling establishment to flowering. Note that UVR8-mediated hypocotyl phototropism is only observed in the absence of phototropin photoreceptors, and potent UVR8-mediated flowering in noninductive photoperiods is repressed by RUP2 in wild type. (*b*) UVR8-mediated acclimatory responses leading to elevated UV-B tolerance. UVR8-mediated UV-B acclimation has been shown in the green alga *Chlamydomonas reinhardtii*, flowering plants (*Arabidopsis* and tomato), and the liverwort *Marchantia polymorpha*. Although acclimatory responses have been studied mainly in *Arabidopsis*, flavonoid production was also associated with UV-B acclimation in *M. polymorpha*. UVR8-mediated photoprotection of photosynthesis is particularly well-documented for *Chlamydomonas*. UVR8-dependent induction of DNA-repair enzymes, in particular photolyases, and ROS-scavenging processes is mainly based on gene expression analyses in these three organisms. Further details and references can be found, e.g., in **Table 2**. Abbreviations: ROS, reactive oxygen species; UV-B, ultraviolet-B.

UVR8 contributes to the general photomorphogenesis program and controls a set of responses related to acclimation and photoprotection, enabling the prevention, limitation, or repair of UV-B-induced damage in the plant (**Figure 3**). Recent research on other UVR8-containing photosynthetic organisms confirmed the evolutionary conservation of many of these responses and also expanded the roles identified for UVR8 signaling (2, 75, 81, 93, 145, 146). The main focus of this section is on work in *Arabidopsis*, mainly due to the wealth of research performed in this model organism; however, we mention work in other species, with a particular emphasis on *Chlamydomonas*, which recently emerged as an excellent model for the role of UVR8 in photoprotection of the photosynthetic machinery. It is of note that, according to current understanding, all known UV-B responses mediated by UVR8 are linked to changes in nuclear gene expression.

3.1. Plant Growth and Development (Photomorphogenesis)

Various growth and developmental responses have been linked to UVR8 photoreceptor activity, from seedling establishment over organ development to inflorescence phototropism.

3.1.1. Seedling de-etiolation. Seedling de-etiolation involves inhibition of hypocotyl elongation, opening of the apical hook, and cotyledon expansion. De-etiolation is mainly induced by phytochrome, cryptochrome, and UVR8 photoreceptors in response to red/far-red, blue, and UV-B light qualities, respectively (42, 70, 117, 176). Usually, low levels of photosynthetically active radiation (corresponding to visible light) are used to reduce the impact of phytochrome- and cryptochrome-mediated seedling de-etiolation to distinguish UVR8 activities. Indeed, UVR8-mediated hypocotyl growth inhibition has been extensively documented in the literature (23, 36, 50, 55, 62, 63, 86, 140, 172, 174). In contrast to the unresponsiveness of *uvr8* null mutants, the inhibition of hypocotyl elongation under UV-B is strongly enhanced in UVR8 overexpressors or mutants lacking negative regulation of UVR8 (36, 46, 52, 116). Hypocotyl growth inhibition is associated with UVR8-mediated *HY5*/*HYH* induction and HY5/HYH stabilization (36, 62, 109, 140, 172). Moreover, the UVR8 interaction with the transcription factors BES1 and BIM1, as mentioned above, represses growth-associated genes that are under the control of brassinosteroids, contributing to the inhibition of hypocotyl elongation under UV-B (86). Degradation of PIF4 and PIF5 has a similar effect on hypocotyl growth elongation, in this case mainly through repression of genes encoding proteins involved in auxin biosynthesis and auxin signaling (140). Similar effects are seen for cotyledon expansion under UV-B (8, 35, 120). As mentioned above, UVR8 interacts directly with MYB13, a positive regulator of UV-B-induced cotyledon expansion and stress acclimation (120). Moreover, UVR8-induced, BBX24-mediated negative regulation of UV-B photomorphogenesis is supported by *bbx24* mutant plants that exhibit exaggerated UV-B photomorphogenesis at the seedling stage (71).

3.1.2. Leaf development. UVR8-mediated growth regulation extends beyond the hypocotyl and cotyledons. Overexpression of UVR8 leads to growth inhibition of different organs by inhibiting cell expansion (35). Indeed, UVR8 hyperactivity results in dwarfed plants that resemble *cop1* mutants (36, 46, 51, 116). UVR8 is also necessary for endocycle progression under UV-B, reflecting the requirement of UVR8 for normal cell growth (164). Moreover, UVR8 stimulates downward leaf curling in *Arabidopsis* leaf blades (i.e., epinasty) (39).

3.1.3. Root development. UVR8 was shown to inhibit *Arabidopsis* lateral root growth through apparent tissue-autonomous inhibition of auxin-responsive gene expression via regulation of the MYB73 and MYB77 transcription factors, as mentioned above (173). Thus, shoot and root development may be coordinately but tissue-autonomously regulated by UVR8.

3.1.4. Stomatal density and aperture. Stomata development and stomatal aperture are also controlled by UV-B in a UVR8-dependent manner (147, 155, 164). UV-B increases stomatal density in wild type but decreases it in a *uvr8* mutant (164). In another report, however, stomatal density was found to decrease in wild type under UV-B levels that were detrimental to *uvr8* (155). Thus, the effect of UV-B on stomata differentiation and the mechanism of how UVR8 affects this process remain to be clarified.

UV-B triggers stomatal closure through UVR8-dependent and -independent pathways, via hydrogen peroxide–induced nitric oxide (NO) production in guard cells (80, 147). UV-B thus seems to antagonize blue light– and phototropin-induced stomatal opening through NO (65).

3.1.5. Phototropism. Phototropins are the major photoreceptors for the directional growth towards light, termed phototropism (34). UVR8 was shown to mediate directional growth of *Arabidopsis* hypocotyls toward UV-B in *phot1 phot2* mutants (153, 154). This response is much

slower than phototropin-mediated phototropism toward blue light, but it also relies on asymmetric auxin redistribution that could be a consequence of asymmetric regulation of HY5 stability in the hypocotyl (153, 154). It remains to be determined if UVR8 contributes to phototropism in wild-type seedlings in the presence of functional phototropins.

Interestingly, a prominent role for UVR8 in inflorescence stem phototropism has been reported (157). The underlying mechanism involves rapid differential growth through unilateral growth inhibition on the lit side, associated with increased HY5 accumulation, reduced gibberellic acid levels, and reduced auxin signaling. UVR8-mediated inflorescence stem phototropism was hypothesized to function in pollinator attraction, since it changes flower positioning and optimizes UV-B perception, triggering the production of volatiles and the flavonoid compounds that make flowers colorful, altogether increasing flower visibility (157).

3.1.6. Flowering time. UV-B exposure of *Arabidopsis* plants grown in long days resulted in a delay of flowering, mostly through an effect on the age pathway (30). The flowering-time delay of UV-B-exposed plants is abolished in a *uvr8* null mutant background; however, the interpretation of this is complicated because *uvr8* mutants flowered constitutively late under the experimental growth conditions used (30).

A repressive role for RUP2 in the regulation of photoperiodic flowering under UV-B has been described (6). *Arabidopsis* is a facultative long-day plant, which means that it flowers early in long days but also eventually flowers in short days. Interestingly, *rup2* mutants show a daylength-neutral phenotype under UV-B and flower early in a UVR8-dependent manner under noninductive, short-day conditions (6). RUP2 physically interacts with the key flowering inducer CO and represses the association of CO at the promoter of the flowering hormone florigen FT (6). Through this mechanism, the potential early flowering induced by UVR8 is counterbalanced in plants containing RUP2 so that under short-day conditions these plants flower late, providing a crucial mechanism of photoperiodic flowering control (6). Currently, it is a matter of speculation why wild-type *Arabidopsis* has a potent pathway that enables flowering in response to UV-B but that is apparently disarmed. It may be that the *Arabidopsis rup2* mutant exemplifies a UVR8 flowering pathway that is indeed active in some plants, such as in day-neutral species. Alternatively, RUP2 may integrate other environmental factors into flowering regulation in the field. For example, RUP2 degradation may be a potent inducer of flowering in noninductive photoperiods, a possibility that remains to be tested.

3.2. Acclimatory Responses

uvr8 null mutants are impaired in UV-B acclimation and show compromised growth under elevated UV-B in laboratory conditions and sun simulators (8, 36, 74). In addition to UVR8, cryptochromes and phytochromes contribute to the establishment of UV-B tolerance (144), and UVR8 and cryptochromes are indeed required for UV-B tolerance and survival in the field (122). The requirement of UVR8 for UV-B tolerance is further supported by genetic studies in organisms other than *Arabidopsis*, such as tomato, *M. polymorpha*, and *Chlamydomonas* (75, 81, 93, 143).

3.2.1. UVR8 induces biosynthesis of sunscreen metabolites. UVR8 controls the production of phenylpropanoid derivatives, including anthocyanins and flavonols, through regulation of genes encoding key biosynthetic enzymes (8, 36, 74, 102, 122, 138). The primary function of this response has been attributed to the sunscreen quality of these compounds, which serves to shield DNA and potentially other macromolecules from UV-B damage (99, 127). In the liverwort *M. polymorpha*, *Mpuvr8* knockout and *MpUVR8* overexpressor plants support an important role of

UVR8 in UV-B acclimation and tolerance (21, 75). UV-B tolerance involves activation of *CHALCONE ISOMERASE-LIKE* (CHIL) via MpMYB14 and consequent flavonoid production (21).

Photoinhibition: photosynthetic yield decrease due to light-induced damage to the photosynthetic machinery, often in response to excessive or fluctuating sunlight

Our current understanding of UVR8 signaling and responses in *Arabidopsis* is principally based on experiments performed in lab conditions, but field studies have also provided precious information regarding UVR8 function in the complex environment of natural conditions (24, 102, 103). Outdoor experiments where UV-A and UV-B were modulated confirmed the ability of UVR8 to activate photoprotective mechanisms, such as phenylpropanoid and antioxidant production, but did not capture UVR8 function in growth regulation (24, 102, 122, 123). However, the analysis of transcriptional response and UVR8 monomerization under short-wavelength UV-A photons (<340 nm) indicates that UVR8 photoreceptor responsiveness extends into this region of the solar spectrum (123). UVR8-mediated hypocotyl growth inhibition and flavonoid accumulation were observed upon sunfleck perception in a natural canopy environment (103).

3.2.2. UVR8 maintains photosynthetic performance. UV-B radiation is harmful for the photosynthetic machinery because it directly damages photosystem II (PSII), enhancing the production of reactive oxygen species (ROS) and thus challenging the PSII repair cycle (27, 139). *Arabidopsis* plants lacking functional UVR8 are hypersensitive to UV-B-induced PSII damage (24, 26). Together, this suggests a role for UVR8 in photoprotection of the photosynthetic machinery under UV-B, although the molecular mechanisms remain unknown.

In tomato, UVR8 induces the expression of the transcription factor GOLDEN2-LIKE2 (SlGLK2), which controls chloroplast development and proliferation, as well as chlorophyll levels in the fruit (81). However, it remains unknown whether *SlGLK2* induction contributes to UV-B tolerance (81, 93).

Analogous to *Arabidopsis*, *Chlamydomonas* UV-B acclimation has been demonstrated through colony-survival and photosynthetic-efficiency assays (143). UV-B leads to broad changes in the *Chlamydomonas* transcriptome, including expression changes of genes related to photosynthesis (2, 143). UV-B acclimation was found to preserve the PSII core proteins D1 and D2 under UV-B stress, mitigating UV-B-induced photoinhibition (143). Thus, UVR8 signaling evolved early in the green lineage, allowing UV-B acclimation and protection.

3.2.3. Photoprotection against high light stress through nonphotochemical quenching in green algae. In *Chlamydomonas*, UVR8 signaling results in protection of the photosynthetic apparatus from high light through induction of nonphotochemical quenching, which allows dissipation of harmful excess light energy as heat (qE) and prevents photodamage (2). Indeed, UVR8 signaling enhances the capacity of qE in *Chlamydomonas* through transcriptional induction of light-inducible photoprotective proteins LHCSR1 and PSBS, and to a much lesser extent LHCSR3, representing an anterograde link between UV-B photoreceptor–mediated signaling in the nucleocytosolic compartment and photoprotective regulation of photosynthetic activity in the chloroplast (2, 145, 146). Interestingly, the molecular effectors that are induced by UVR8 signaling and that underlie UVR8-mediated photoprotection are partially different from those induced by high light, which are regulated by the blue-light photoreceptor phototropin (2, 3, 41, 76, 115, 145). Active UVR8 interacts with CrCOP1, resulting in the stabilization of transcription factors CrCO and NUCLEAR FACTOR Y (NF-Y), which form a complex that governs light-dependent photoprotective responses in *Chlamydomonas* (145).

4. INTEGRATION OF UVR8 SIGNALING WITH OTHER PATHWAYS

Plants exist in complex environments and must respond appropriately to diverse endogenous signals and environmental cues, both biotic and abiotic. For example, plants grow in a polychromatic

environment and integrate information from different light qualities. UVR8 triggers a large variety of responses that may modulate and/or be modulated by other pathways and responses. We provide here a few examples; however, many more remain to be discovered or require further investigation.

4.1. Cryptochrome Signaling Represses UVR8 Photoreceptor Activity

It was recently reported that cryptochrome-mediated blue-light signaling modulates UVR8 photoreceptor activity by activating expression of the *RUP1* and *RUP2* repressors of UVR8 signaling (46, 144). Indeed, UVR8 signaling is hyperactivated in *cry1 cry2* mutants, as reflected by both the degree of UVR8 monomerization and the transcriptional profile (123, 144). Reciprocally, UVR8-mediated UV-B signaling activates the expression of *BLUE-LIGHT INHIBITOR OF CRYPTOCHROMES 1* (*BIC1*) and *BIC2*, which encode repressors of cryptochrome activity and may thus modulate blue-light signaling (36, 144, 161, 162). Such photoreceptor pathway cross-regulation allows a balanced and appropriate response to light information in a polychromatic environment, particularly as cryptochromes and UVR8 largely overlap in their signaling mechanisms and responses (77, 117, 118, 160). This photoreceptor cross-regulation may extend to phytochromes and UVR8, as it was already shown to exist between phytochromes and cryptochromes (32). A UVR8–cryptochrome–phytochrome network is likely relevant considering the partially redundant contributions of these three photoreceptors for UV-B tolerance (122, 123, 144). Such an intricate interplay between photoreceptors for both visible light and UV-B radiation allows the regulation of tolerance to UV-B and potentially to high light through the integration of diverse sunlight signals.

4.2. UVR8 Antagonizes Shade-Avoidance Responses

Shade-intolerant plants perceive neighboring plants and canopy shade though phytochrome B and cryptochrome inactivation and initiate stem elongation, thus outgrowing competitors and ensuring access of their photosynthetic organs to light. Under strong shade conditions, low-level UV-B represses the shade-avoidance response in a UVR8-dependent manner by antagonizing the activity of PIF proteins and the effects of the phytohormones auxin and gibberellin (50, 98, 135, 141). UVR8 also perceives and responds to sunflecks that transiently interrupt shade (103).

4.3. UVR8 Antagonizes Thermomorphogenesis

The effect of elevated temperature on growth, termed thermomorphogenesis, is similar to the shade-avoidance response. In this context, stem/hypocotyl and petiole elongation combined with leaf elevation are thought to promote leaf cooling (49). UVR8 attenuates thermomorphogenesis responses by repressing *PIF4* expression and PIF4 DNA-binding activity through HFR1 stabilization under UV-B and PIF4–HFR1 heterodimer formation (49). Thus, when plants experience both high temperature and high UV-B levels, as occur toward midday, UVR8 may function antagonistically to thermomorphogenesis, thereby balancing the growth response.

4.4. UVR8 Contributes to the Entrainment of the Circadian Clock

Circadian clocks allow synchronization of physiological responses with the environment, allowing their timing to correspond with the most appropriate part of a 24-h diurnal cycle. Light perception is critical for the entrainment and sustainment of circadian rhythms. Alongside

phytochromes, cryptochromes, and Zeitlupe family members, UVR8 is one of the photoreceptors found to provide an entraining input for the clock, likely through transcriptional activation (37, 105). Reciprocally, UV-B induction of gene expression under diurnal conditions was found to be gated by the clock (37). Interestingly, transcriptomic analysis of the circadian clock suggests that it orchestrates the production of photoprotective pigments, including UV-B-absorbing phenylpropanoids, early in the day in anticipation of the rising sun (48).

4.5. Plant Defense and Biotic Interactions

In their natural environment, plants face biotic stresses such as pathogens or herbivory. As shown for other light qualities, the presence of UV-B promotes plant defense (7, 155, 165). Among the secondary metabolites synthesized upon UV-B perception, UVR8 induction of sinapates (but not flavonoids) plays a major role in enhanced plant defense against the fungal pathogen *Botrytis cinerea* (28). Moreover, a study of the *Nicotiana attenuata* microbiome demonstrated that root colonization by *Deinococcus* bacteria increased following UVR8 activation through a currently unknown mechanism (131).

5. CONCLUDING POINTS AND PERSPECTIVES

Our understanding of the molecular players and their early mechanisms and functions in the UV-B perception and signaling pathway has considerably advanced over recent years. UV-B perception by UVR8 followed by the UVR8–COP1 interaction and COP1 inactivation through competitive binding constitute the primary mechanism of the UV-B response crucial for UV-B acclimation and tolerance. Evidence for direct interaction of UVR8 with several transcription factors emerged recently, which is apparently dependent on the UV-B-dependent nuclear accumulation of UVR8 and seems associated mostly with inhibition of transcription factor DNA-binding capacity. However, numerous questions remain regarding the photochemistry, signal transduction, and regulatory mechanisms of UVR8, as well as the physiological roles and cross-regulations of UVR8 with other signaling pathways. The answers to these and related questions will enhance our understanding of plant UV-B responses, which is essential to fully comprehend the effects that sunlight has on plant growth and development.

SUMMARY POINTS

1. UVR8 is a homodimeric photoreceptor that perceives ultraviolet-B photons based on intrinsic tryptophan residues, which results in its monomerization.
2. Active UVR8 inhibits the E3 ubiquitin ligase COP1 by competitive binding via a C-terminal Val-Pro motif that mimics the interaction motifs of COP1 substrates, such as those in several transcription factors that in turn become stabilized.
3. UVR8 induces PIF4 and PIF5 degradation and represses PIF-regulated processes such as hypocotyl elongation, shade avoidance, and thermomorphogenesis.
4. UVR8 interacts with a diverse set of transcription factors, inducing their separation from target DNA and thus inactivating their transcriptional activities.
5. UVR8 signaling is conserved from single-celled green algae to flowering plants.

6. UVR8 signaling is closely integrated with other photoreceptor pathways to regulate plant growth, development, and photoprotection.
7. UVR8 alongside cry1 is required for survival under natural sunlight.

FUTURE ISSUES

1. Do additional UV-B photoreceptors exist in plants?
2. How is UVR8-mediated photoprotection established in flowering plants?
3. What is the mode of action of RUP1 and RUP2 in facilitating UVR8 redimerization?
4. How is UVR8 nucleocytoplasmic partitioning regulated, and what is the relevance of UVR8 nuclear accumulation for signaling?
5. How does COP1 function in the direct effect of UVR8 on transcription factors?
6. How are multichromatic light signals integrated (including integration on COP1 regulation), and how are they integrated with other environmental cues?
7. Is the potent UVR8 pathway affecting photoperiodic flowering relevant for induction of flowering in any plant species?
8. How may the UVR8–COP1 interaction be used for optogenetics in plants under controlled conditions (see the sidebar titled UVR8-Based Optogenetics and Materials)?

UVR8-BASED OPTOGENETICS AND MATERIALS

Given the specificity and sensitivity of UVR8 for ultraviolet-B (UV-B), which allows UV-B to exert regulation in a visible-light background, UVR8 has been included in the growing optogenetic toolkit to engineer UV-B-specific responses in nonplant systems (18, 25, 104). Indeed, the first implementations of UVR8 in novel optogenetic systems were reported whereby UV-B is used to control nuclear retention, chromatin association, protein secretion, and gene expression in mammalian cells (18, 25, 104). In addition, UVR8 has been used in chemical biology to generate photoresponsive hydrogels (178).

DISCLOSURE STATEMENT

The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

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