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Ultra-violet rays light up transcriptional networks regulating plant growth

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Abstract (46 words)

Light and hormones tightly regulate plant growth and development by both synergistic and antagonistic actions. In the current issue of *Developmental Cell*, Liang et al. (2018) uncover how the UV-B photoreceptor UVR8 mediates inhibition of plant growth via direct interactions with key transcriptional regulators of brassinosteroid signaling.

Ultra-violet B (UV-B) irradiation is known for its detrimental effects on biological macromolecules in living organisms. Unlike animals and phototactic microorganisms, plants are immobile and are therefore unable to escape excessive light exposure by sheltering under shaded environments. However, plants have acquired strategies that utilize UV-B light not only as a stimulus to induce photoprotection, but also as an information signal to regulate their development and growth. Seminal work on the model plant species *Arabidopsis thaliana* has led to the discovery of the first genetically encoded UV-B receptor, UV-RESISTANCE LOCUS 8 (UVR8) (Rizzini et al., 2011).

The molecular basis of UV-B perception and downstream signaling has recently been elucidated. Contrary to the blue and red/far-red light photoreceptor families, UVR8 absorbs UV-B in the absence of an exogenous chromophore. In vitro and in vivo studies have revealed the importance of a key triad of tryptophan residues in absorbing UV-B and in signalling (Christie et al., 2012; Wu et al., 2012). UV-B irradiation triggers the photo-conversion of UVR8 from an inactive homodimer to an active monomer allowing direct association with downstream signaling components (Christie et al., 2012; Rizzini et al., 2011; Wu et al., 2012). Photo-activated, monomeric UVR8 accumulates in the nucleus and induces the expression of photoprotective and photomorphogenic genes by interacting with the E3 ubiquitin ligase CONSTITUTIVE PHOTOMORPHOGENIC 1 (COP1) (Brown et al., 2005; Favory et al., 2009; Kaiserli and Jenkins, 2007; Yin et al., 2016) (Figure 1). Although UVR8 and COP1 are essential for inducing key UV-B responsive genes, such as the transcription factor (TF) ELONGATED HYPOCOTYL 5 (HY5), the exact molecular mechanism of how UVR8 regulates gene expression at the transcriptional level is still not fully understood. This is due in part to the fact that very few proteins have been reported to directly associate with UVR8.

To gain insight into UVR8 function, Liang et al. (2018) set out to identify interacting partners for UVR8. They employed a stringent yeast-two-hybrid screening approach and found that key transcriptional components of brassinosteroid (BR) signalling such as the TFs BES1 (BRI1-EMS-SUPPRESSOR 1) and BIM1 (BES1-INTERACTING MYC-LIKE 1) directly interact with UVR8. *In planta* interaction studies reveal that UV-B mediated nuclear accumulation of UVR8 is essential for stabilising the UVR8-BES1/BIM1 complex.

The physiological significance of the interaction between UVR8 and BR signaling was further investigated genetically by examining UV-B mediated hypocotyl

elongation phenotypes of UVR8 and BR signaling mutants. BR and UV-B have opposing effects on hypocotyl growth as BRs promote elongation and UV-B inhibits growth. The authors show that the BR signaling mutants *bes1* and *bim1,2,3* exhibit hypersensitivity to UV-B, whereas BES1 overexpression results in hyposensitivity to UV-B. Elegant pharmacological experiments complement the genetic studies indicating that UVR8 operates upstream of its interacting BR TFs.

To understand how UV-B affects BR signaling at the transcriptional level, Liang and co-workers (2018) took advantage of transcriptomic data reflecting the responses to UV-B and BR stimuli. These data confirmed that UV-B represses the expression of BR-induced genes. Indeed, gene ontology and quantitative RT-PCR analysis clearly show that the majority of cell-wall elongation genes induced by BES1 are negatively regulated by UV-B. More specifically, overexpression of UVR8 leads to a UV-B dependent downregulation of BES1-induced genes.

Finally and very importantly, the authors unveil the molecular mechanism integrating UV-B and BR signaling by demonstrating that photo-activated nuclear UVR8 associates and interferes with the DNA-binding ability of BES1 and BIM1, pausing the expression of BR-regulated growth-promoting genes in a UV-B-dependent manner (Figure 1B).

Overall, this study provides a mechanistic model for understanding the role of UV-B irradiation in fine-tuning plant growth via signal integration with the brassinosteroid pathway. In addition to dissecting the mechanism of UV-B mediated inhibition of hypocotyl elongation, the authors make two important discoveries. First, they show that UVR8 directly interacts with transcription factors to regulate gene expression in response to UV-B and second, they find that UVR8 promotes crosstalk between UV-B and the brassinosteroid signaling pathways to induce UV-B dependent inhibition of hypocotyl elongation.

UVR8 was recently shown to be involved in regulating thermomorphogenesis, the shade avoidance response, plant immunity and circadian clock entrainment, underlining the importance of signaling crosstalk among light, clock, hormone and defence pathways (Yin and Ulm, 2017). The excellent and elegantly performed study by Liang et al. (2018) now opens up avenues for future research in UV-B signaling crosstalk and transcriptional regulation through UVR8. A separate study just published (Yang et al., 2018) shows that nuclear UVR8 can directly interact and modulate the DNA binding activity of the WRKY36 transcription factor to allow the induction of *HY5* expression upon UV-B stimulation (Figure 1B). In both cases, the photo-activated UV-B receptor plays a pivotal role in positively or negatively regulating transcription by interfering with the action of key transcription factors that modulate growth in response to environmental and endogenous stimuli. These findings will allow researchers to understand not only how the UVR8 receptor operates in the broader context, but also how environmental and endogenous stimuli are integrated at the level of transcription to control development, with potential appplicability to many other eukaryotic systems.

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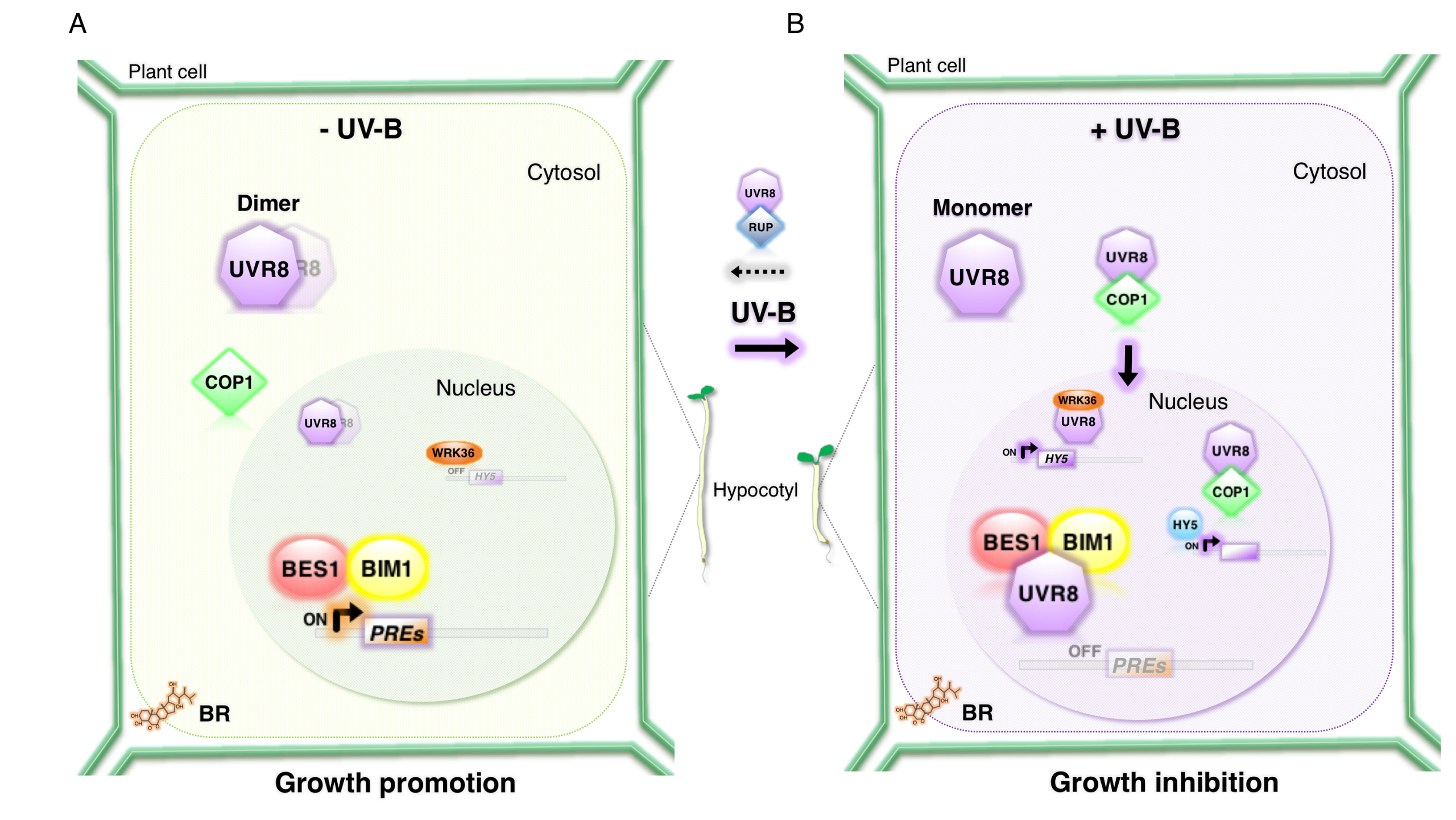
Figure 1. UVR8 integrates UV-B and brasssinosteroid signaling to regulate hypocotyl growth. Schematic representation of UVR8 action. (A) In the absence of UV-B, UVR8 exists primarily as a cytosolic homodimer, which allows nuclear BES1 and BIM1 to cooperatively upregulate

the expression of brassinosteroid-induced growth-promoting genes. (B) Upon UV-B irradiation, photo-activated UVR8 monomerizes, associates with COP1 and accumulates in the nucleus. Monomeric UVR8 interacts with BES1 and BIM transcription factors and inhibits their DNA-binding activity. As a result of the BIM1-UVR8-BES1 interaction, BR-induced growth-promoting genes are not expressed in the presence of UV-B light. In contrast, UVR8 interference with the DNA-binding activity of the transcriptional repressor WRK36 allows *HY5* expression. Collectively, UVR8 induces inhibition of hypocotyl elongation by directly inhibiting transcription factor action leading to the downregulation of growth-promoting genes.


A
B
Plant cell
- UV-B
Cytosol
Dimer
UVR8
COP1
Nucleus
WRK36
OFF
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BES1
BIM1
ON
PREs
BR
Growth promotion
UVR8
RUP
UV-B
Hypocotyl
Plant cell
+ UV-B
Cytosol
Monomer
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WRK36
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