

IN BRIEF

How ELONGATED HYPOCOTYL5 Helps Protect Plants from UV-B Rays

Most of us do what we can to avoid the DNA-damaging effects of UV-B rays. While plants can't slap on sunscreen, put on a nice hat, or move to a shady spot, they too have ways to protect themselves from (and acclimate to) damaging UV-B radiation. For example, plants undergo UV-B-dependent photomorphogenic changes (see figure), including reduced hypocotyl and leaf expansion, increased stomatal closure, and the production of UV-absorbing compounds such as flavonoids. In *Arabidopsis thaliana*, UV-B-induced responses are mediated by the photoreceptor UV RESISTANCE LOCUS8 (UVR8) (Rizzini et al., 2011), which lacks the usual prosthetic light-sensing chromophore, instead relying on specific intrinsic tryptophans for light detection (Jenkins, 2014). Upon sensing UV-B light, the homodimeric UVR8 rapidly switches to its active, monomeric form, which interacts with the E3 ubiquitin ligase CONSTITUTIVELY PHOTOMORPHOGENIC1 (COP1), a well-known repressor of photomorphogenesis. Downstream of UVR8 and COP1, the bZIP transcription factor ELONGATED HYPOCOTYL5 (HY5) and its homolog HYH mediate most UV-B-induced changes in gene expression. While uncomplexed COP1 targets HY5 for ubiquitination, HY5 induces COP1 expression by binding to a specific UV-B-responsive element in its promoter, and UVR8-COP1 somehow stabilizes HY5 (Huang et al., 2012). However, it is not known how HY5 expression is induced by UV-B exposure. In addition, while HY5 potentially interacts with thousands of target genes, it is unclear how HY5 associates with chromatin in response to UV-B light exposure.

A recent study by Binkert et al. (2014) has brought us much closer to elucidating the role of HY5 in the UV-B response. First, chromatin immunoprecipitation (ChIP) experiments using anti-HY5 antibodies demonstrated that UV-B treatment affects the association of HY5 with its target genes. Next, HY5 ChIP analysis of a *uvr8* null mutant



UV-B-induced photomorphogenesis. Wild-type (WT) and *uvr8* mutant *Arabidopsis* seedlings grown for 4 d in the absence or presence of UV-B light. (Figure courtesy of M. Binkert.)

revealed that the UV-B-enhanced association of HY5 with its target promoters is regulated by UVR8. Conversely, the authors observed a reduced UV-B-specific response in plants overexpressing REPRESSOR OF UV-B PHOTOMORPHOGENESIS2, a negative regulator of UVR8. They also found that HY5 associates with the promoters of various UV-B-regulated genes but not with that of the constitutively expressed UVR8. Importantly, one of these UV-B-regulated genes is HY5 itself; ChIP analysis revealed that both HY5 and HYH specifically bind to the HY5 promoter. In fact, transgenic seedlings expressing the *ProHY5:LUCIFERASE (LUC)* reporter construct exhibited increased luminescence in response to UV-B irradiation in the *hy5*, *hyh*, and wild-type backgrounds, while *ProHY5:LUC* induction was nearly absent in the *hy5 hyh* double mutant background and completely absent in *uvr8* knockout mutants, demonstrating that HY5 and HYH act redundantly to regulate UV-B-induced transcription of HY5. A similar assay using

truncated and mutated HY5 promoters uncovered a T/G-box in the HY5 promoter that's required for its UV-B responsiveness. The binding of this promoter region by HY5 and HYH was confirmed by electrophoretic mobility shift assays. Therefore, HY5 and HYH bind to the T/G-box of the HY5 promoter, thus playing a crucial role in the UV-B-induced regulation of HY5, which in turn mediates UV-B responses.

It remains to be determined how UV-B light enhances the binding of HY5 and HYH to the T/G-boxes in the promoters of their many target genes. Moreover, additional transcription factors that regulate UV-B-induced HY5 expression remain to be identified. Nonetheless, this pivotal study sheds light on the role played by HY5 to help plants cope with excess UV-B rays—sunscreen not required.

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