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Review

Illuminating Progress in Phytochrome-Mediated Light Signaling Pathways

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Light signals regulate a plethora of plant responses throughout their life cycle, especially the red and far-red regions of the light spectrum perceived by the phytochrome family of photoreceptors. However, the mechanisms by which phytochromes regulate gene expression and downstream responses remain elusive. Several recent studies have unraveled the details on how phytochromes regulate photomorphogenesis. These include the identification of E3 ligases that degrade PHYTOCHROME INTERACTING FACTOR (PIF) proteins, key negative regulators, in response to light, a better view of how phytochromes inhibit another key negative regulator, CONSTITUTIVELY PHOTOMORPHOGENIC 1 (COP1), and an understanding of why plants evolved multiple negative regulators to repress photomorphogenesis in darkness. These advances will surely fuel future research on many unanswered questions that have intrigued plant photobiologists for decades.

Trends

Two classes of repressors called COP/DET/FUS complex and PIFs synergistically repress photomorphogenesis in darkness.

Light signals perceived by phytochromes inhibit these repressors to promote photomorphogenesis.

$CUL3^{LRB}$ induces polyubiquitylation and subsequent co-degradation of PIF3 and PHYB through the 26S proteasome pathway.

$CUL4^{COP1–SPA}$ E3 ligase promotes rapid light-induced degradation of PIF1 to promote photomorphogenesis.

Phytochromes directly interact with SPA1 and reorganize the COP1–SPA interaction to inhibit COP1 activity.

Phytochrome-Mediated Light Signaling Pathways

Light is an essential commodity for photosynthetic energy production as well as an environmental cue for increasing awareness and fitness to the surrounding conditions. Plants employ two contrasting developmental programs to succeed in ambient light conditions: skotomorphogenesis and photomorphogenesis (Figure 1). Skotomorphogenesis is characterized by elongated hypocotyl, closed cotyledon, and an apical hook to allow young seedlings to grow rapidly in darkness using the reserve energy present in the seed. By contrast, photomorphogenesis is the process where light signals inhibit the rapid elongation of hypocotyl, expand the cotyledons, and promote greening to allow the seedling body to adjust for optimal light-harvesting capacity and autotrophic growth. To promote photomorphogenesis and actively suppress skotomorphogenic development, plants have evolved multiple photoreceptors to track a wide spectrum of light wavelengths in a local environment. These include the UVB-RESISTANCE 8 (UVR8 for UV-B light), cryptochromes (CRY), phototropins, and ZEITLUPE/FLAVIN-BINDING, KELCH REPEAT, F BOX 1/LOV KELCH PROTEIN 2 family of photoreceptors (ZTL/FKF1/LKP2) (for UV-A/blue light) and phytochromes (phy for red/far-red light) [1]. This review will focus on the phytochrome family of photoreceptors that are encoded by five genes in *Arabidopsis thaliana* (*PHYA–PHYE*) [2]. Phytochromes perceive the ambient red (R) and far-red (FR) light signals in the environment and promote gradual progression to photomorphogenic development by orchestrating an elaborate signaling mechanisms [3,4]. These include allosteric conformation change of phytochromes to a biologically active Pfr form from an inactive Pr form followed by nuclear translocation to inhibit two classes of repressors of photomorphogenesis called CONSTITUTIVELY PHOTOMORPHOGENIC/DEETIOLATED/FUSCA (COP/DET/FUS) complex and Phytochrome Interacting Factors (PIFs) (Figure 1) [4,5]. In darkness, these dual repressors are actively promoting skotomorphogenic development by suppressing photomorphogenesis, and

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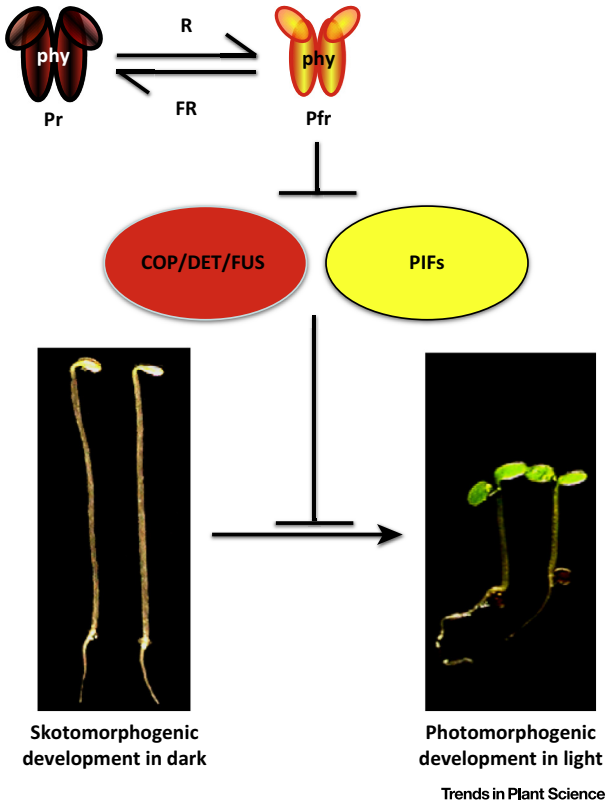

Figure 1. A Simplified View of the Phytochrome-Mediated Light Signaling Pathways. In the dark, phytochromes exist in the biologically inactive Pr form. The CONSTITUTIVELY PHOTOMORPHOGENIC/DEETIOLATED/FUSCA (COP/DET/FUS) complexes and PHYTOCHROME INTERACTING FACTOR (PIF) proteins function in the dark to repress photomorphogenic development. Seedlings grown in the dark show a long hypocotyl and closed cotyledons. Under light, phytochrome perceives red/far-red light signals and photoconverts from an inactive Pr form to an active Pfr form. The active Pfr form suppresses the functions of COP/DET/FUS complexes and PIFs. As a result, seedlings progress toward photomorphogenic development. Seedlings grown in light display short hypocotyl, open and expanded green cotyledons for optimal photosynthesis and autotrophic growth. Abbreviations: FR, far-red light; R, red light; Pfr, far-red light absorbing form of phytochrome; phy, phytochrome; Pr, red light absorbing form of phytochrome.

therefore inhibition of these repressors allows gradual progression to photomorphogenic development under light. This review will focus on recent progress on the mechanistic understanding of how phytochromes inhibit these repressors. For a detailed review on other aspects of photomorphogenesis, readers are directed to recent reports and reviews [6–13].

E3 Ligases for PIFs

PIFs belong to the basic helix-loop-helix (bHLH) family of transcription factors [14,15]. There are seven PIFs in *Arabidopsis* that function in a partially-differential to a largely-overlapping manner to regulate gene expression and ultimately photomorphogenesis [4,16–19]. All PIFs interact with the Pfr forms of phytochromes with differential affinities [4,20]. Phytochromes interact with PIFs through the APB (active phytochrome binding) or APA (active phytochrome A binding) domains present at the N termini of PIFs. Conversely, PIFs displayed higher affinity for the N terminus of phytochromes [21,22]. Direct physical interaction of PIFs with phytochromes leads to the light-induced phosphorylation followed by ubiquitylation and subsequent degradation of PIFs by the ubiquitin/26S proteasome system (UPS). In addition, light-induced phosphorylation is necessary for degradation of PIF3 [23]. The degradation kinetics of PIFs under different light qualities/quantities and early post-translational modifications have been extensively investigated [4]. A putative polyubiquitin binding factor called HEMERA is also necessary for degradation of PIF1