Phytochrome signaling mechanisms and the control of plant development

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As they emerge from the ground, seedlings adopt a photosynthetic lifestyle, which is accompanied by dramatic changes in morphology and global alterations in gene expression that optimizes the plant body plan for light capture. Phytochromes are red and far-red photoreceptors that play a major role during photomorphogenesis, a complex developmental program that seedlings initiate when they first encounter light. The earliest phytochrome signaling events after excitation by red light include their rapid translocation from the cytoplasm to subnuclear bodies (photobodies) that contain other proteins involved in photomorphogenesis, including a number of transcription factors and E3 ligases. In the light, phytochromes and negatively acting transcriptional regulators that interact directly with phytochromes are destabilized, whereas positively acting transcriptional regulators are stabilized. Here, we discuss recent advances in our knowledge of the mechanisms linking phytochrome photoactivation in the cytoplasm and transcriptional regulation in the nucleus.

Phytochrome signaling and seedling development

A major difference between plant and animal development is that most plant morphogenesis occurs post-embryonically, which confers plants with great developmental plasticity. Thus, even though they are rooted in the ground, plants can rapidly alter their growth and development in response to a wide spectrum of environmental cues. One of the most dramatic examples of this developmental plasticity occurs when seedlings emerge from soil [1]. Most seeds germinate in the ground, and seedlings must forage for light. These newly sprouted etiolated seedlings have a long primary stem and undeveloped embryonic leaves (called cotyledons), which are protected by an apical hook in the primary stem as the seedling pushes its way through the soil (Figure 1). In dicotyledonous plants, such as the reference plant Arabidopsis thaliana, a differential growth program is initiated when the seedling emerges from the soil. Stem (hypocotyl) growth rate is rapidly inhibited and cotyledons and leaves expand, turn green as chloroplasts develop and become photosynthetic, a process known as de-etiolation.

The morphological changes during de-etiolation are the result of a massive reprogramming of the transcriptome with 7–20% of the Arabidopsis genome differentially expressed between dark-grown and light-grown seedlings [2–6]. De-etiolation is first triggered by a suite of photoreceptors that perceive distinct colors of light [1]. The phytochrome (phy) family of receptors plays a major role in perceiving red (R) and far-red (FR) light, which carries information on the availability of photosynthetic energy and the proximity of neighboring plants.

Phys are bilin-containing proteins with two major domains: an N-terminal photosensory and signaling domain and a C-terminal dimerization and localization domain [7–10]. They exist in two relatively stable conformers: an R-light absorbing inactive Pr form (λmax = 660 nm) and an FR-absorbing active Pr form (λmax = 730 nm) [8]. In Arabidopsis, the phy family consists of five members, phyA through phyE, that form homo- and heterodimers [11]; phyA and phyB are the most prominent phys and regulate almost every facet of plant development and growth [12,13]. Due to differences in their photochemistry, stability, and rate of translocation to the nucleus, phyA and phyB are responsible for shared and distinct responses. PhyA proteins accumulate in the dark and in FR light and are rapidly degraded in R light [14]. PhyA plays a dominant role in FR light as well as during the dark-to-R transition. By contrast, phyB is relatively stable and plays a major role in R light. These different modes of action provide the molecular basis for distinct morphological responses of emerging etiolated seedlings to FR-rich shaded or R-rich open ambient light conditions.

Many phytochrome signaling components have been identified using forward-genetic studies, mainly in Arabidopsis [15]. The photomorphogenetic mutants can be divided into two general classes. The first class of mutants show de-etiolated or constitutively photomorphogenetic phenotypes, including short hypocotyls, expanded cotyledons, partial chloroplast differentiation and derepression of light-induced gene expression in the dark (Figure 1). This class of mutants includes the de-etiolated 1 (det1) mutants [16], the constitutively photomorphogenic 1 (cop1) mutants [17], the mutants in the components of the COP9 signalosome (CSN) [18], the quadruple mutant of the suppressor of phytochrome A-105 1-4 (spa1-4) [19], and the recently reported quadruple mutant phytochrome interacting factors 1,3,4,5 (pif1) [5,6,20]. Because mutations in these genes are recessive, these components most likely define negatively acting components in the light signaling pathways; they share similar phenotypes with a dominant constitutively active phyB allele, YHB [8,21].
suggesting that phy initiate photomorphogenesis by repressing these negative regulators.

The second class includes mutants that share similar phenotypes with phyA and/or phyB loss-of-function mutants with long hypocotyls and under-developed cotyledons in the light. They are defective in either phyA or phyB signaling or both. For example, far-red elongated hypocotyl 1 (fhy1) [22,23], far-red elongated hypocotyl 3 (fhy3) [22,24], long after far-red light 1 (laf1) [25], far-red impaired response 1 (far1) [26], long hypocotyl in far-red 1 (hfr1) [27] are specific for phyA signaling, whereas the elongated hypocotyl 5 (hy5) mutant is defective in both phyA and phyB signaling [28,29]. The recently reported hemera (hmr) mutant defines a new subclass of phy signaling mutants, which are not only impaired in phyA and phyB signaling with long hypocotyls under both R FR light but also are defective in chloroplast development and die as albino seedlings (Figure 1) [30]. The albino phenotype in hmr is most likely due to defects in chloroplast differentiation, in addition to phy signaling, because the quintuple phy mutant still makes chlorophyll and is pale-green in the light [31]. HMR might be a dual function protein because it is localized in both chloroplasts and nuclei.

Many of the mutants from both classes define loci that encode transcriptional regulators of light-responsive genes, including the positively acting HY5, LAF1, HFR1, FHY3 and FAR1, as well as the negatively acting PIFs. A key mechanism by which phy regulate gene expression is modulation of the protein stability of these transcriptional regulators in the nucleus, where light directly regulates the affinity between phy and downstream signaling components. In addition, light regulates the translocation of phy to the nucleus into subnuclear foci called nuclear bodies, where phy and downstream signaling components are co-localized. This review focuses on the recent advances in our understanding of phy signaling mechanisms, with an emphasis on early events linking photoproduction of phy in the cytoplasm to transcriptional regulation in the nucleus.

Phytochrome nuclear accumulation is the earliest detectable light response

Akira Nagatani’s group reported 15 years ago that Arabidopsis phyB contains putative nuclear localization signals (NLSs) and could be localized to the nucleus [32]. Subsequent studies using phytochromes fused with a fluorescent protein tag confirmed these earlier results and further demonstrated that the nuclear accumulation of all five phys in Arabidopsis is light-dependent [33–36]. In dark-grown seedlings, phy accumulate in the Pr form and localize mainly to the cytoplasm. However, both phyA and phyB rapidly accumulate in the nucleus within minutes after exposure to R light [37]. Regulation of phy nuclear accumulation is a key regulatory mechanism for phy signaling [10].

PhyB nuclear localization is regulated by light-dependent unmasking of the NLS. In R light when phyB is in the Pr form, the N-terminal photosensory domain interacts less strongly with the C-terminal NLS domain than in the Pr conformer [38]. This led to a model in which photoproduction triggers a large movement of the two domains, exposing the NLS, and leading to nuclear accumulation.

The regulatory mechanism for phyA nuclear accumulation is more complex (Figure 2). No NLS has been identified in phyA. Rather, phyA nuclear localization requires binding to a pair of plant-specific proteins, FHY1 and its paralog FHL (FHy1-Like), both of which contain a conserved NLS and shuttle phyA to the nucleus [39–41]. FHY1 and FHL are required for both phyA nuclear accumulation and its subsequent signaling events [42,43]. Early reports suggested that FHY1 and FHL accumulate in the dark and interact more strongly with the Pr form of phyA in vitro [39,40]. More recently, it was shown that FHY1 and FHL interact preferentially with the Pr form of phyA in vivo [42,44,45].

PhyA nuclear accumulation is dependent on FHY1/FHL; as such, it might not be surprising that phyA nuclear accumulation is regulated by feed-back through the regulation of FHY1 and FHL levels. FHY1 and FHL transcripts accumulate in the dark and are reduced in the light

Figure 1. Two classes of photomorphogenetic mutants. The left panel shows 4-day-old wild type Col-0 and det1-1 seedlings grown in darkness; the right panel shows 4-day-old Col-0, phyB-9 and hmr-2 seedlings grown under 8 μmol m⁻² s⁻¹ of R light.

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(Figure 2) [23,46]. The expression of FHY1 and FHL is regulated by the transposase-derived transcription factors FHY3 and FAR1 [46,47]. Light reduces FHY1/FHL transcript levels through the down-regulation of FHY3/FAR1 transcription [46,47]. In addition, light attenuates FHY3 and FAR1 function by promoting the accumulation of HY5 because HY5 binds to a cis-element next to a FHY3/FAR1 binding site in the FHY1/FHL promoters and negatively regulates the transcriptional activity of FHY3 or FAR1 [48].

In addition to regulation at the transcriptional level, phyA promotes the phosphorylation and proteasome-mediated degradation of FHY1 and FHL in R light [44,45]. Together with the rapid degradation of phyA (discussed below), these mechanisms work in concert to attenuate phyA signaling in the light.

**Are phytochrome-containing nuclear bodies (photobodies) the sites of phytochrome signaling?**

When they enter the nucleus, phys are further compartmentalized to subnuclear foci, called phytochrome speckles or phytochrome nuclear bodies [49]. Because these are unique plant subnuclear domains that are regulated by light, we name them photobodies. During the dark-to-light transition, photobodies containing both phyA and phyB are detected after 1–2 min of R light [37,50]. The localization of phyB to photobodies is triggered by R light. In contrast, phyA photobody localization is triggered by R and FR light. These early photobodies are transient and disappear after 1 h of light exposure [37]. In addition to phyA and phyB, FHY1, FHL, and the phytochrome interacting factors PIF3 and PIF7 have been localized to the early photobodies [37,51]. PhyB localization to the early photobodies is not dependent on phyA but dependent on PIF3 [37,52]. However, it seems that phyB photobody localization does not require its interaction with PIF3 or other PIFs, because phyB mutations defective in PIF binding could still be localized to photobodies [52,53]. By contrast, localization of PIF3 to early phy nuclear bodies requires its interaction with phys [50].

After 2 h in R light, phy photobodies reappear and remain present in the light [33,37]. These late phy photobodies contain mainly phyB because phyA is degraded rapidly during the dark-to-light transition (discussed below). Under continuous R light, the steady-state pattern (size and number) of photobodies is directly related to the amount of Pfr [54]. Under dim R light, phyB is evenly dispersed in the nucleoplasm, whereas under strong R light, phyB appears to be localized exclusively to a few large photobodies with diameters of 1–2 µm [30,54].

Several models have been proposed for the function of photobodies. Photobodies could be storage depots or reservoirs for activated phy. In this model, photobodies are sites where activated phyB is stabilized, but are not sites of phy signaling events. Consistent with the model, the Pfr form of phyB associated with photobodies is more stable in vivo [55]. In addition, the N-terminus of phyB fused to a dimerization domain and an NLS is active in mediating light responses without localization to photobodies [56,57].

A second model posits that the photobodies are sites of protein turnover. As will be elaborated below, Cullin-RING E3 ligases are master regulators of photomorphogenesis. COP1 and its target proteins LAF1, HY5, phyA and FAR1 colocalize in nuclear bodies, leading to the proposal that photobodies are sites for protein degradation [58–61]. Also, phyA, FHY1 and PIF3 are localized to early photobodies before their degradation [37,39,44,50]. Last year we reported a forward-genetic screen for mislocalization of a phyB::GFP fusion protein [30]. This screen identified a novel phy signaling component, HMR. In hmr mutants, phyB::GFP localized to smaller photobodies than predicted on the basis of the fluence rate of R light. In hmr mutants, phyA, PIF1 and PIF3 accumulated in the light, suggesting HMR either directly or indirectly controls the degradation of these proteins [30]. HMR is predicted to be structurally similar to RAD23, which is a multiubiquitin receptor that delivers mult ubiquitylated proteins to the proteasome for degradation. HMR’s localization to the periphery of photobodies, in combination with the other observations, supports a model in which photobodies are sites for the degradation of photolabile proteins [30]. Further investigation of the biochemical functions of HMR will likely elucidate the link between photobodies and protein degradation.

In yet another model, photobodies are sites for phytochrome signaling. This model is supported by a close correlation between the localization of phyB to large nuclear bodies under continuous R light and phyB-mediated responses [54]. The constitutively active phyB mutation, YHB, is localized to large phy nuclear bodies even in darkness [21]. The three models are not necessarily mutually exclusive because phy signaling involves the light-regulated proteolysis of PIF transcription factors (see below).

**Down-regulation of phyA and phyB in the light**

Both phyA and phyB levels are repressed in the light to desensitize or regulate phy signaling [61]. During the dark-to-R-light transition, phyA levels are rapidly reduced by 50–100-fold, whereas phyB levels are reduced gradually by five four-fold [62]. PhyA accumulation is negatively regulated both at the transcriptional and post-translational levels. PHYA transcripts are strongly reduced by both R and FR light [63]. This is correlated with histone modification changes at the PHYA locus. Histone marks for gene
activation, such as H3K9/14ac, H3K27ac and H3K4me3 are enhanced during phyA activation in darkness; in contrast, histone marks for gene silencing, such as H3K27me3, are enriched at the PHYA locus in the light [64].

The Pfr form of phyA is subject to ubiquitin–proteasome-dependent protein degradation [14,61,65,66]. PhyA degradation is mediated by the E3 ubiquitin ligase cullin 4 (CUL4)-damaged DNA-binding protein 1 (DDB1)–COP1–SPA complex, where the RING protein COP1 is associated with members of the SPA protein family to form the substrate receptor for the E3 ubiquitin ligase [42,61,67,68]. Recognition of phyA by a COP1–SPA complex is enhanced by phyA phosphorylation in the light, because unphosphorylated phyA preferentially interacts with signaling components FHY1/FHY3, preventing phyA from binding to COP1–SPA [42]. Independent of the CUL4–DDB1–COP1–SPA complex, other E3 ubiquitin ligases might be involved in phyA degradation [42]. One possible candidate is a Cullin 1 (CUL1)-based E3 ubiquitin ligase [69], although no substrate receptor F-box protein has been identified. PhyA degradation also requires HMR, which is structurally similar to the multiubiquitin receptor RAD23 and might act like RAD23 in delivering phyA to the proteasome [30]. PhyA degradation occurs in both the cytoplasm and the nucleus, with faster degradation kinetics in the nucleus [70–73]. Within the nucleus, phyA degradation likely occurs on photobodies, because phyA and COP1 were co-localized to photobodies [37,61]. This notion was further supported by the identification of the hmr mutant, where defects in photobodies led to phyA accumulation in the light [30].

Down regulation of phyB occurs mainly at the post-translational level, because PHYB transcripts in Arabidopsis are relatively unaffected by light [74]. Degradation of phyB is mediated by COP1 [65], most likely through the CUL4–DDB1–COP1–SPA complex. In addition, the level of phyB protein in the light is negatively regulated by PIFs [51,65,75], which could promote COP1-mediated phyB ubiquitylation, possibly by enhancing the interaction between phyB and COP1 [65]. This serves as one of the mechanisms for PIFs to regulate phyB signaling [76].

CUL4 is part of the multisubunit Cullin-RING ubiquitin ligase superfamily [83]. The Cullin-RING ubiquitin ligases share a similar modular design. The C-terminus of CUL binds a catalytic RING subunit, such as Rbx1, and Rbx1-associated E2 ubiquitin conjugating enzyme; the N-terminus of CUL associates with an adaptor protein and a substrate receptor that recognizes target proteins. Cullin-RING ubiquitin ligases facilitate the transfer of ubiquitin from E2 to target proteins. CUL4-based E3 ubiquitin ligases utilize DDB1 as adaptor proteins and DDB1-binding WD40 proteins as substrate receptors [84]. Recent biochemical analysis suggested that the master repressors for photomorphogenesis form two distinct E3 ubiquitin ligases; the CUL4–DDB1–DET1–COP10 complex and the CUL4–DDB1–COP1–SPA complex [67,82]. Cullin-based E3s are regulated by the cyclic attachment and removal of the ubiquitin-related protein RUB or NEDD8. The CSN is a highly conserved protein complex, which resembles the 19S lid of the 26S proteasome and catalyzes the deneddylation reaction for Cullins. Taken together, all three complexes are involved in ubiquitin-proteasome-mediated protein degradation.

The COP1/SPA1 E3 mediates the degradation of positively acting factors for photomorphogenesis [67]. Besides phyA, its substrates include three transcriptional regulators, the bZIP protein HY5 [85], the MYB protein LAF1 [60] and the helix-loop-helix protein HFR1 [58,86,87]. The selective degradation of these transcriptional regulators in darkness is a key mechanism to repress photomorphogenesis. Consistent with this notion, expressing truncations of HY5 or HFR1 without their corresponding COP1 binding motifs stabilizes HY5 or HFR1, leading to enhanced photomorphogenetic phenotypes [58,59,87,88]. COP1/SPA-mediated protein degradation could occur on photobodies because COP1 colocalizes with phyA, HY5, LAF1 and HFR1 in photobodies and all SPA proteins are localized to nuclear bodies [58,60,61,87,89]. Although DET1 and COP1/SPA form distinct E3 ubiquitin ligases, genetic studies suggest that DET1 works in concert with COP1 in plant development [90]. One possible mechanism is that DET1 E3 is involved in the regulation of components of the COP1/SPA E3, such as COP1 [82]. It has been proposed that phyB inhibit the function of the DET/COP master repressor proteins in the light; however, the mechanism is not totally clear. Some possibilities include: (i) light regulation of the nuclear/cytoplasmic partitioning of COP1 [91]. COP1 accumulates in the nucleus in darkness to repress photomorphogenesis and is localized mainly to the cytoplasm in the light. The nuclear accumulation of COP1 is dependent on DET1, COP10 and direct interaction with the CSN1 subunit of CSN [92–94]. Therefore, phyB could either directly regulate COP1 localization or indirectly regulate its localization through DET1 or CSN. However, it should be mentioned that the kinetics of this regulation is slow during the dark-to-light transition and there are always COP1 proteins remaining in the nucleus in the light [65]. (ii) PhyB could negatively regulate the amount of COP1/SPA complex by down-regulating both SPA1 and SPA2 protein levels [95]. (iii) PhyB could regulate COP1/SPA activity because COP1/SPA interactions are weakened in R, FR and blue light [89]. Recently, it was

Repression of photomorphogenesis by Cullin4-RING ubiquitin ligases

Our understanding of how photomorphogenesis is repressed in dark-grown seedlings began from genetic screens for Arabidopsis mutants exhibiting de-etiolated (DET) or constitutive photomorphogenic (COP) phenotypes in the absence of light. These genetic screens identified a group of master repressors for photomorphogenesis, including DET1 [77], COP1 [78], COP10 [79], SPA1-4 [19,80], and subunits of the COP9 signalosome (CSN) [18]. Characterization of the DET1 protein complex identified the WD-repeat protein DDB1, a key component for regulating photomorphogenesis [81]. Recent biochemical analysis revealed that these master repressor proteins form three protein complexes involved in ubiquitin–proteasome-mediated protein degradation, including two CUL4-based E3 ubiquitin ligases and the COP9 signalosome, which modulates the activity of Cullin-based E3 ubiquitin ligases [18,67,82].
shown that the blue light photoreceptors cryptochromes directly regulate either the formation of the substrate receptor COP1/SPA1 complex or the interaction between the substrate receptor COP1/SPA complex and its target proteins [96–98]. Phys could utilize a similar mechanism to regulate the activity of COP1.

Initiation of photomorphogenesis by removal of phytochrome interacting factors
Phytochrome interacting factors (PIFs) are bHLH transcription factors that negatively regulate photomorphogenesis [76]. Since the founding member, PIF3, was identified by Peter Quail’s laboratory in 1998 [99], great advances have been made by several laboratories toward understanding the roles of PIFs in photomorphogenesis. The PIFs belong to a 15-member subfamily of bHLH transcription factors. All of the characterized PIFs, including PIF3, PIF1/PIL5, PIF4, PIF5/PIL6 and PIF7, have been shown to bind to the G-box motif (CACGTG) in light-regulated genes, where they act as either transcriptional activators or repressors [5,51,100–105]. PIFs are involved in shared and distinct phy-mediated responses. During de-etiolation, PIF1, PIF3, PIF4, PIF5 and PIF7 are all involved in hypocotyl growth inhibition [75,100,101,106–109]. PIF1, PIF3 and PIF5 are also repressors for chloroplast development, down regulating the expression of genes encoding key chlorophyll biosynthetic enzymes [5,6,101,103,110]. Moreover, PIF1 down regulates carotenoid biosynthesis by repressing the expression of the key carotenoid biosynthetic enzyme phytoene synthase [111]. Therefore, PIFs link phy signaling to downstream light-mediated morphological responses.

All of the PIF proteins contain phytochrome-interacting domains in the N-terminal region and bHLH DNA-binding and dimerization domains at their C-terminus. PIFs preferentially bind to the active Pfr form of phy [76]. PIF1 and PIF3 are capable of interacting with both photo-activated phyA and phyB; in contrast, PIF4, PIF5, PIF6 and PIF7 bind only to phyB [51,99,101,108]. Upon light activation, accumulation of phys in the nucleus triggers phosphorylation of PIFs and rapid degradation of PIFs by the ubiquitin-proteasome system [50]. It is not completely clear which kinase is responsible for PIF phosphorylation. It was recently suggested that PIF1 is phosphorylated by CK2 and possibly other unidentified kinases [112]. The E3 ubiquitin ligase for PIF degradation is unknown. With the exception of PIF7, most PIFs have a half-life of 5–20 min in the light [113–117]. The significance of PIF degradation was demonstrated by the characterization of the pif1pif3pif4pif5 quadruple (pifq) mutant [5,6]. The pifq mutant showed de-etiolated phenotypes in darkness similar to those of a constitutively active phyB YHB allele and the det/cop mutants [3,5]. Thus, PIFs are required to promote etiolation (or stem growth) and phys might regulate photomorphogenesis by promoting PIF degradation [5,6].

A key question now is to understand the relationship between PIFs and DET/COP proteins (Figure 3). COP1/SPA does not appear to be the E3 for PIF degradation but rather is required for at least PIF3 accumulation [20,37,65]. However, the mechanism linking COP1/SPA and PIF3 stability is unknown (Figure 3). COP1/SPA could regulate the function of PIFs indirectly through the HLH protein HFR1, which forms heterodimers with PIFs and prevents PIF binding to DNA [102].

At the cellular level, because PIF3 is localized to early photobodies before its turnover, it has been proposed that photobodies are sites for PIF degradation [37]. The characterization of the hmr mutants that are defective in the late photobodies supported this hypothesis, as both PIF1 and PIF3 accumulate to higher levels in the hmr mutants in the light [30]. This suggests that photobodies are sites for PIF degradation. Because HMR is also required for phyA degradation in the light, HMR could be involved in PIF degradation directly or indirectly through the regulation of phy [30]. Further characterization of HMR function will distinguish between these possibilities.

Concluding remarks
Numerous recent studies have established the main framework of phy signaling during seedling emergence. First, phy trigger the rapid degradation of the PIF family of bHLH transcription factors that promote the dark program. Second, phy negatively regulate two CUL4-based E3 ubiquitin ligases, CUL4–DDB1–COP1–SPA and CUL4–DDB1–DET1–COP10, to stabilize a group of transcriptional regulators, including HY5, LAFl and HFR1, that promote photomorphogenesis. Both phyA and phyB are turned-over by the same mechanism to desensitize phy signaling in the light. Third, at the cellular level, phys rapidly accumulate in the nucleus and are compartmentalized to photobodies in the light. The translocation of phy is regulated by conformational changes of phys as well as by other signaling components. Photobodies are likely sites for light-regulated proteolytic events. Despite these great advances, many questions remain unanswered. It is not clear how phys inhibit the activity of
DET1, COP1/SPA and CSN activities in the light. The mechanism of PIF degradation is unknown. Finally, how photobodies are assembled and regulated, as well as their precise function in phy signaling will be an active area of future investigation.

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