

Photobodies in Light Signaling¹

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Plant development and growth are extremely plastic in response to changes in ambient light conditions. Light is not only the ultimate energy source for photosynthesis; its physical parameters, such as quality, intensity, direction, and duration, also serve as key environmental and time cues (Chen et al., 2004). Therefore, it is vital for plants to closely monitor and precisely respond to changes in light properties in order to optimize growth under a wide range of ecological environments and to synchronize developmental transitions with diurnal and seasonal time. Plants have evolved to “see” the light spectrum between 280 and 750 nm, which spans UV-B, UV-A, and visible light, through five classes of photoreceptors. In the reference plant *Arabidopsis* (*Arabidopsis thaliana*), they include the newly determined UV-B receptor UV RESISTANCE LOCUS8 (UVR8; Kaiserli and Jenkins, 2007; Rizzini et al., 2011); three types of UV-A/blue light receptors, including cryptochromes (cry1 to 3; Kleine et al., 2003; Yu et al., 2010; Chaves et al., 2011), phototropins (phot1 and 2; Briggs and Christie, 2002), and the family of ZEITLUPE/FLAVIN-BINDING, KELCH, F-BOX1 (FKF1)/LOV KELCH PROTEIN2 (LKP2; Nelson et al., 2000; Somers et al., 2000; Schultz et al., 2001); and the red and far-red sensing phytochromes (phyA to E; Quail, 2010). Collectively, these photoreceptors regulate almost every facet of plant development and growth from seed germination to floral initiation (Franklin and Quail, 2010; Kami et al., 2010).

Many light responses are mediated by alterations in gene expression. Among the 15 photoreceptors discovered so far, 10 of them, UVR8 (Kaiserli and Jenkins, 2007; Favory et al., 2009), cry1 (Wu and Spalding, 2007), cry2 (Kleiner et al., 1999; Yu et al., 2009), FKF1 (Sawa et al., 2007; Fornara et al., 2009), LKP2 (Yasuhara et al., 2004), and phyA to E (Sakamoto and Nagatani, 1996; Yamaguchi et al., 1999; Kircher et al., 2002), have been shown to localize to the nucleus, where they regulate gene expression in a light-dependent manner (Jiao et al., 2007). An emerging common mechanism for such light-dependent gene expression is through regulating the stability of key transcriptional regula-

tors (discussed below; Yi and Deng, 2005; Brown et al., 2009; Chen and Chory, 2011; Leivar and Quail, 2011).

At the cellular level, photoactivation of photoreceptors triggers the rapid localization of a number of them, including phyA to E, cry2, and possibly cry1 and UVR8, to discrete subnuclear foci or nuclear bodies (Kleiner et al., 1999; Yamaguchi et al., 1999; Wang et al., 2001; Kircher et al., 2002; Favory et al., 2009; Yu et al., 2009; Gu et al., 2011; Lian et al., 2011; Liu et al., 2011). Nuclear bodies are morphologically distinct subnuclear domains that provide microenvironments for the regulation of protein dynamics, gene expression, and DNA replication and repair in both plant and animal cells (Shaw and Brown, 2004; Spector, 2006). The general principles of nuclear body function and assembly are still largely unknown. The photoreceptor-containing nuclear bodies, or “photobodies,” found in plants are a unique type of subnuclear domain whose size, number, and potentially function are directly regulated by external light cues (Chen and Chory, 2011). Observations of these speckle-shaped photobodies have raised many questions. How is the formation of photobodies regulated? What are the functions of the photobodies? What are the factors required for photobody formation? These are some of the key questions that are being actively investigated. Further understanding of photobody function and regulation will not only be important for understanding the subcellular organization of light signaling events in plants but also could potentially uncover general principles governing subnuclear domains in higher eukaryotes.

In this review, we will summarize recent developments related to phy-containing photobodies, touch briefly on photobodies containing crys, and discuss the potential functions of photobodies in relationship to protein degradation and gene expression. For more comprehensive coverage of light signaling in plants, the reader is referred to the following recent reviews (Henriques et al., 2009; Jenkins, 2009; Chory, 2010; Franklin and Quail, 2010; Kami et al., 2010; Lau and Deng, 2010; Möglich et al., 2010; Nagatani, 2010; Rockwell and Lagarias, 2010; Yu et al., 2010; Chaves et al., 2011; Chen and Chory, 2011; Leivar and Quail, 2011; Uljasz and Vierstra, 2011).

LIGHT-DEPENDENT DYNAMICS OF PHYTOCHROME PHOTOBODY FORMATION

Phys in higher plants are red (R) and far-red (FR) light receptors that use a linear tetrapyrrole, phyto-

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chromobilin, as their chromophore. Phys can interconvert between two relatively stable conformers: an R light-absorbing inactive Pr form ($\lambda_{\text{max}} = 660$) and a FR light-absorbing active Pfr form ($\lambda_{\text{max}} = 730$; Rockwell et al., 2006). Although it was initially thought that phys localized and functioned primarily in the cytoplasm, a series of studies performed over a decade ago using GUS-tagged and fluorescent protein-tagged phys convincingly demonstrated that photoactivation from the Pr to the Pfr form results in the rapid translocation of phys from the cytoplasm to the nucleus (Sakamoto and Nagatani, 1996; Kircher et al., 1999, 2002; Yamaguchi et al., 1999; Kim et al., 2000). This change in localization is one of the earliest phy responses to light; for both phyA and phyB, the most prominent phys in Arabidopsis, nuclear accumulation is required for most of their downstream responses (Huq et al., 2003; Genoud et al., 2008).

It was first reported by Akira Nagatani and colleagues that photoactivated phyB-GFP was not only localized to the nucleus but also further compartmentalized to subnuclear speckle-like photobodies (Yamaguchi et al., 1999). Parallel studies by Eberhard Schäfer, Ferenc Nagy, and colleagues demonstrated that all five Arabidopsis phys, phyA to E, localize to photobodies in the light and that phy photobody localization is conserved in both dicotyledonous and monocotyledonous plants (Kircher et al., 1999, 2002; Kim et al., 2000). Although most studies on phy photobodies were conducted using transgenic lines that overexpressed fluorescent protein-tagged phys, both native pea (*Pisum sativum*) phyA and Arabidopsis phyB photobodies have been observed using immunocytochemistry, suggesting that the formation of photobodies is not an artifact of phy overexpression (Hisada et al., 2000; Kircher et al., 2002).

The translocation of phys to photobodies happens very quickly during the dark-to-light transition; photobodies containing both phyA and phyB can be observed after 1 to 2 min of R light exposure (Bauer et al., 2004). PhyB photobody localization is triggered by R light (Yamaguchi et al., 1999; Kircher et al., 2002). In contrast, phyA photobody localization is triggered by R, FR, and blue light (Kim et al., 2000). These “early” photobodies are transient and disappear after 1 h of light exposure (Bauer et al., 2004). Phy photobodies reappear after 2 h in R light and remain present in the light (Yamaguchi et al., 1999; Bauer et al., 2004). These “late” photobodies contain mainly phyB, because phyA is rapidly degraded in R light (Kim et al., 2000; Kircher et al., 2002).

Joanne Chory and colleagues showed that the steady-state pattern (size and number) of phyB photobodies under continuous R light is determined by the percentage of phyB in the Pfr form at a given moment (Chen et al., 2003). Light conditions that shift the Pr/Pfr equilibrium to the Pfr side or stabilize the Pfr form will promote large phy photobody formation. Consistent with this notion, under high-intensity R light, which drives the equilibrium to the Pfr form, phyB

appears to be localized exclusively to a few large photobodies with diameters between 1 and 2 μm (Chen et al., 2003, 2010b). By contrast, under dim R light or light with a low R-to-FR ratio, where more phyB stays in the Pr form, phyB tends to localize to many smaller photobodies or localizes diffusely in the nucleoplasm (Fig. 1; Chen et al., 2003). The formation of large phyB photobodies correlates tightly with the light-dependent hypocotyl inhibition response. The fact that the steady-state pattern of phyB-GFP is predictable and can be precisely manipulated by external light quantity and quality makes it an excellent visible readout for genetic screens (discussed below; Chen, 2008). Although phyB photobodies appear to be morphologically stable, they are actually quite dynamic subnuclear domains; fluorescence recovery after photobleaching experiments on phyB-yellow fluorescent protein (YFP) photobodies showed that photobody-associated phyB-YFP is rapidly exchanged with nucleoplasmic phyB-YFP (Rausenberger et al., 2010).

Some phy photobodies might also contain the blue light receptor cry2. Crys are photolyase-like photoreceptors that use FAD as the chromophore. When phyB-GFP and cry2-red fluorescent protein were co-expressed in BY-2 protoplasts, not only did they colocalize on photobodies, they could also be coimmunoprecipitated (Más et al., 2000). Because cry2 is photolabile and rapidly degraded in blue light, it could be colocalized with phyB in early photobodies during the dark-to-light transition. Consistent with this hypothesis, cry2 photobody localization is also a rapid light response, as Arabidopsis cry2 is translocated to photobodies within 15 min after blue light

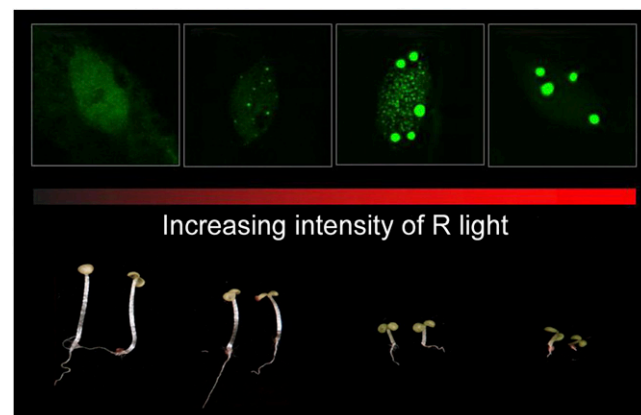


Figure 1. The morphology of phy photobodies is directly regulated by light. Confocal images of phyB-GFP localization patterns and corresponding PBC seedlings under increasing intensities of red light are shown. PhyB-GFP is evenly distributed under dim ($0.5 \mu\text{mol m}^{-2} \text{s}^{-1}$) R light. With increasing R light intensity ($1 \mu\text{mol m}^{-2} \text{s}^{-1}$ and $2 \mu\text{mol m}^{-2} \text{s}^{-1}$), phyB-GFP starts to form exclusively small, or both small and large photobodies, respectively. Under strong R light (above $8 \mu\text{mol m}^{-2} \text{s}^{-1}$), phyB-GFP localizes exclusively to large photobodies. The localization of phyB-GFP correlates with the degree of hypocotyl inhibition in the light.

exposure (Yu et al., 2009). Activated cry1 has also been suggested to localize to nuclear bodies (Wang et al., 2001; Gu et al., 2011; Lian et al., 2011; Liu et al., 2011). Because cry1 and cry2 were colocalized with COP1 and SPA1 on nuclear bodies (Gu et al., 2011; Lian et al., 2011; Liu et al., 2011; Zuo et al., 2011), it is likely that cry1, similar to cry2, could also localize to phy-containing photobodies in the light.

STRUCTURAL BASIS OF PHYTOCHROME PHOTOBODY LOCALIZATION

The intramolecular requirements for photobody localization have been most extensively studied using *Arabidopsis* phyB. Two general approaches have been taken to dissect the phy subdomains required for photobody localization: one approach is to examine the localization pattern of phy truncation fragments; the other approach is to characterize the localization pattern of loss-of-function or gain-of-function alleles of *phy*.

The domain structure of phys has been well defined. Phys can form either homodimers or heterodimers (Clack et al., 2009); each monomer is an approximately

125-kD polypeptide. The phy protein can be divided into two domains: an N-terminal photosensory and signaling domain and a C-terminal dimerization domain, with a hinge region connecting the two (Fig. 2; Nagatani, 2010). The N-terminal domain comprises four subdomains: an N-terminal extension (NTE); a PAS (for PER, ARNT, and SIM) domain; a GAF (for cGMP phosphodiesterase, adenylate cyclase, and FhlA) domain, which contains a conserved Cys residue forming a thioether linkage with the A ring of the chromophore phytychromobilin; and a PHY (for phytochrome-specific GAF-related) domain (Rockwell et al., 2006). The crystal structure of the PAS/GAF domain of the bacteriophytochrome DrBph1 from *Deinococcus radiodurans* revealed the presence of a light-sensing knot, which plays an important role in signaling. For example, amino acid residues located in the knot are involved in the interaction with phytochrome-interacting factors (PIFs; Oka et al., 2008; Kikis et al., 2009; Nagatani, 2010; Ulijasz and Vierstra, 2011). The C-terminal half of phys contains two subdomains, a PAS-related domain (PRD) containing two PAS domains (PAS-A and PAS-B) and a His kinase-related domain (HKRD; Rockwell et al., 2006).

Truncation studies have revealed that the C-terminal half of phyB localizes to photobodies independently of light (Chen et al., 2003; Matsushita et al., 2003). Similarly, the photobody localization of phyA also requires its C-terminal half (Wolf et al., 2011). Within the C-terminal domain of phyB, the PRD is both required and sufficient for nuclear localization, suggesting that it either possesses a nuclear localization signal (NLS) or is able to bind to an unidentified shuttle protein containing a NLS (Matsushita et al., 2003; Chen et al., 2005). Both the PRD and HKRD are required for normal photobody localization (Chen et al., 2005). Consistent with this notion, several missense mutations that result in defective nuclear or photobody localization have been identified within the PRD (Fig. 2; Kircher et al., 2002; Chen et al., 2003; Matsushita et al., 2003). The role of the HKRD in photobody localization is still a mystery; although truncations lacking the entire HKRD do not localize to photobodies, a phyB truncation lacking a portion of the HKRD could still form smaller photobodies (Chen et al., 2005). Because nuclear and photobody localization are both light dependent, it raises the question of how the N-terminal photosensory domain regulates the C-terminal NLS/photobody localization signals. The current model is that, in the Pr form, C-terminal localization signals are masked by the N-terminal domain through an interaction between the GAF-PHY subdomains and the PRD, whereas both the putative NLS and photobody localization signals are exposed in the Pfr form as a result of light-dependent conformational changes (Chen, 2008; Fankhauser and Chen, 2008). The “open” conformation of the Pfr form could also expose domains required for interacting with other signaling components. Consistent with the notion that the conformation of Pfr is important for photobody localiza-

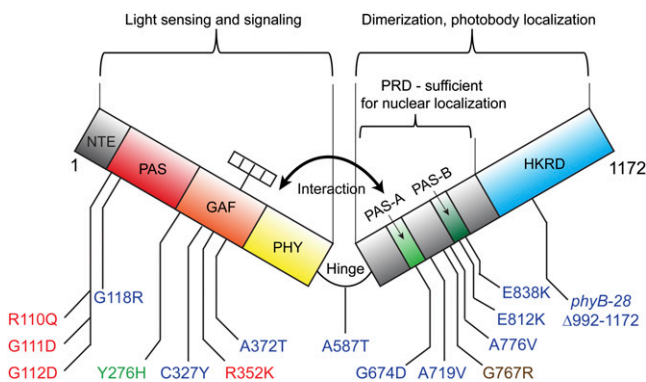


Figure 2. Structural basis for phyB localization and signaling. Phys can be divided into the N-terminal light-sensing and signaling domain and the C-terminal dimerization and localization domain (Nagatani, 2010). The GAF and PHY domains of phyB physically interact with the PRD to mask nuclear localization signals located in the PRD (Chen et al., 2005). Additionally, although the PRD alone is sufficient for nuclear import, the PRD and HKRD together are required for normal phyB photobody localization (Matsushita et al., 2003; Chen et al., 2005). Mutations in red are those that have apparently normal photobody localization but fail to complement a null *phyB* mutant, possibly due to reduced interaction with PIF proteins (Oka et al., 2008; Kikis et al., 2009). The Y276H mutation, in green, causes constitutive phyB localization to large photobodies and constitutive phytochrome signaling (Su and Lagarias, 2007). The G767R mutation, in brown, results in the inability of phyB to localize to the nucleus (Matsushita et al., 2003). All other mutations, represented in blue, show abnormal photobody localization as well as impaired light signaling (Kircher et al., 2002; Chen et al., 2003; Oka et al., 2008). Specifically, mutant G118R does not incorporate the chromophore; mutants C327Y and A587T show slightly faster dark reversion; and mutant A372T shows a slightly red-shifted spectrum in addition to highly accelerated dark reversion. The chromophore is represented by four consecutive squares.

tion, the NTE, which plays a role in stabilizing the Pfr form, is also required for phyB photobody formation (Chen et al., 2005).

Localization studies of missense *phyB* alleles have further demonstrated that the Pfr form of phyB is required for photobody localization. N-terminal missense loss-of-function *phyB* alleles that are defective in photobody localization also have less stable Pfr, and some have abnormal light absorption spectra (Fig. 2; Chen et al., 2003; Oka et al., 2008). By contrast, YHB, a constitutively active *phyB* mutant, localizes to photobodies regardless of light conditions (Su and Lagarias, 2007). Moreover, loss-of-function mutations that only affect signaling but not the absorption properties of phyB have normal photobody localization patterns (Oka et al., 2008; Kikis et al., 2009), which further suggests that the photobody localization depends on phyB being in the Pfr form and is not a consequence of phy signaling.

POSSIBLE FUNCTIONS OF PHOTOBODIES IN LIGHT SIGNALING

Ever since the initial observation of phy photobodies, there has been much speculation about their function. One hypothesis is that the photobodies are storage depots for active photoreceptors but are not required for light signaling (Fig. 3A). In this model, photobodies serve as a valve to regulate the amount of active phy in the nucleoplasm. Consistent with this model, the N terminus of phyB fused to a dimerization domain and a NLS is active in mediating light responses but does not localize to photobodies (Matsushita et al., 2003; Palágyi et al., 2010). However, accumulating evidence from localization and colocalization studies on light signaling components supports the idea that photobodies might be the sites of light signaling.

Most phy-mediated responses require global reprogramming of the transcriptome (Tepperman et al., 2006; Jiao et al., 2007; Hu et al., 2009; Leivar et al., 2009; Shin et al., 2009). Two emerging signaling mechanisms suggest that the key signaling events regulating gene expression work by modulating the stability of either positively or negatively acting transcription factors. The positively acting transcription factors include the basic leucine zipper (bZIP) transcription factor HY5 (for elongated hypocotyl 5; Koornneef et al., 1980; Oyama et al., 1997), the MYB factor LAF1 (for long after far-red light 1; Ballesteros et al., 2001), the helix-loop-helix (HLH) factor HFR1 (for long hypocotyl in far-red 1; Fairchild et al., 2000), and some members of the B-box zinc finger family (BBX; Kumagai et al., 2008; Khanna et al., 2009), including CONSTANS (CO)/BBX1 (Laubinger et al., 2006; Liu et al., 2008), COL3 (for CONSTANS-like 3)/BBX4 (Datta et al., 2006), LZFI (for light-regulated zinc finger protein 1)/STH3 (for salt tolerance homolog 3)/BBX22 (Chang et al., 2008, 2011; Datta et al., 2008), and BBX21/STH2 (Datta

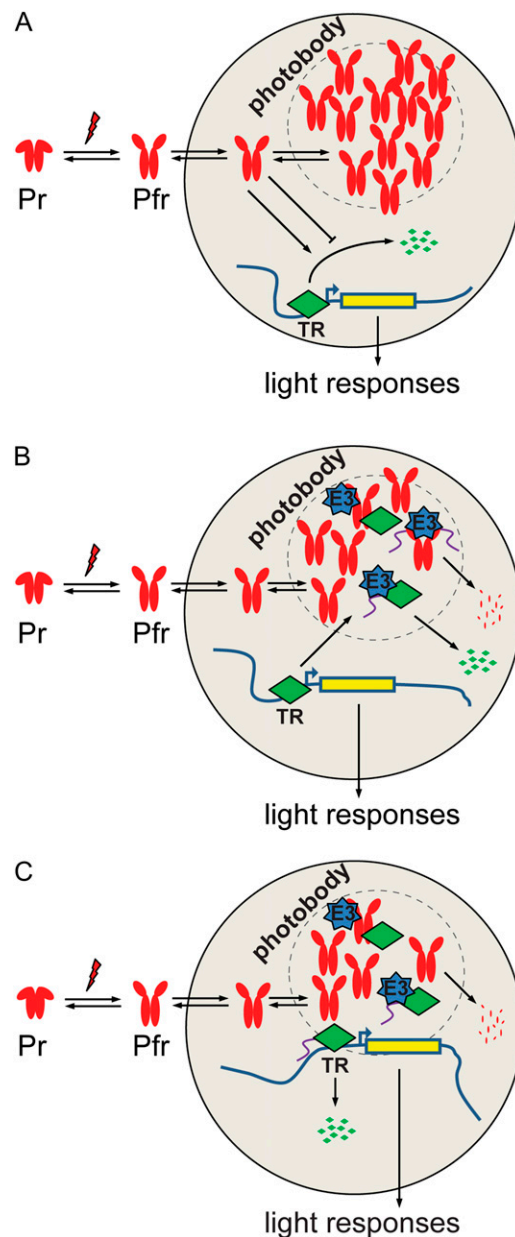


Figure 3. Alternative models of photobody function. A, The storage depot model. In this model, the amount of photoactivated nucleoplasmic phys is regulated by sequestering them within photobodies. These photobodies serve as storage depots that later release the phys into the nucleoplasm to carry out their signaling functions, resulting in the regulation of light-responsive genes. B, The degradation model. In this model, the photobodies are sites for the ubiquitination and degradation of key transcriptional regulators. C, The transcription model. Transcriptional regulators localize to photobodies, bringing their target DNA with them. The expression of the target genes is regulated within or in the vicinity of the photobodies. E3, E3 ubiquitin ligase; TR, transcriptional regulator.

et al., 2007). These proteins are degraded in the dark by the E3 ubiquitin ligase COP1 (for constitutively photomorphogenic 1) and/or the cullin4-DDB1 (for

damaged DNA-binding protein 1)-COP1-SPA (for suppressor of *phytochrome A-105*) E3 ubiquitin ligase complex, where COP1 and members of the SPA family of proteins form the substrate recognition complex (Osterlund et al., 2000; Seo et al., 2003; Duek et al., 2004; Jang et al., 2005; Yang et al., 2005; Datta et al., 2006, 2007; Laubinger et al., 2006; Liu et al., 2008; Chen et al., 2010a; Chang et al., 2011). COP1 is also involved in the turnover of both phyA and phyB, partly as a mechanism to attenuate phy signaling in the light (Seo et al., 2004; Jang et al., 2010). The current model is that phys promote the stability of this group of positively acting transcription regulators by repressing E3 ubiquitin ligases (Chen and Chory, 2011). Although the molecular mechanism of how phys repress COP1 and/or the COP1 E3 complex is still unclear, recently it has been shown that crys 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 (Lian et al., 2011; Liu et al., 2011; Zuo et al., 2011). It is quite possible that phys could utilize a similar mechanism to regulate the activity of COP1.

Besides positively acting transcriptional regulators, there are also transcriptional regulators that are antagonistic to phy signaling. Some of the well-studied members of this group are the bHLH transcription factor called PIFs (Ni et al., 1998; Oh et al., 2007; Shen et al., 2008; Lorrain et al., 2009; Shin et al., 2009; Leivar and Quail, 2011). Phys bind directly to PIFs and trigger their phosphorylation and subsequent degradation in the light (Al-Sady et al., 2006; Leivar and Quail, 2011). The rapid turnover of PIFs in the light is a key mechanism to turn on phy-mediated responses (Leivar et al., 2009; Shin et al., 2009).

One widely proposed hypothesis is that photobodies are sites for protein degradation (Fig. 3B). This model is supported by the fact that many signaling components are localized to photobodies prior to their degradation (Table I). For example, during the dark-to-light transition in seedling development, both phyA and PIF3 colocalize to early phy photobodies before their degradation (Al-Sady et al., 2006). In addition, the positively acting transcriptional regulators, including HY5, LAF1, HFR1, and some BBX proteins, also colocalize with COP1 on nuclear bodies (Table I).

Table I. *Photobody constituents*

A list of light signaling components that have been shown to localize to photobodies or photobody-like subnuclear domains. These include not only photoreceptors but a number of transcriptional regulators and their E3 ubiquitin ligases, suggesting that photobodies are involved in light-regulated protein degradation and/or transcription.

Photobody Constituent	Function	Reference
Photoreceptors		
phyA to E	R and FR receptors	Yamaguchi et al. (1999); Kim et al. (2000); Kircher et al. (2002); Chen et al. (2003); Matsushita et al. (2003); Bauer et al. (2004)
cry1 and 2	UV-A/blue receptors	Wang et al. (2001); Yu et al. (2009); Gu et al. (2011); Lian et al. (2011); Liu et al. (2011); Zuo et al. (2011)
UVR8	UV-B receptor	Favory et al. (2009)
Related to protein degradation		
COP1	E3 ubiquitin ligase	Stacey and von Arnim (1999); Wang et al. (2001); Seo et al. (2004); Subramanian et al. (2004)
SPA1 to 4	E3 ubiquitin ligase	Seo et al. (2003); Laubinger et al. (2006); Zhu et al. (2008); Lian et al. (2011); Liu et al. (2011); Zuo et al. (2011)
HMR	Structurally similar to RAD23	Chen et al. (2010b)
Transcriptional regulators		
PIFs (PIF3 and PIF7)	bHLH transcription factor	Bauer et al. (2004); Al-Sady et al. (2006); Leivar et al. (2008); Kidokoro et al. (2009)
HFR1	HLH transcriptional regulator	Yang et al. (2005); Jang et al. (2007)
HY5/HYH	bZIP transcription factor	Ang et al. (1998); Holm et al. (2002)
LAF1	MYB transcription factor	Ballesteros et al. (2001); Seo et al. (2003); Jang et al. (2007)
BBXs (CO, COL3, LZFI/STH3/BBX22, STH2/BBX21, STO/BBX24, DBB1a/BBX18, and DBB1b/BBX19)	Transcriptional regulators	Datta et al. (2006, 2007, 2008); Laubinger et al. (2006); Indorf et al. (2007); Jang et al. (2008); Liu et al. (2008); Yan et al. (2011)
ELF3	phyB signaling/clock associated	Yu et al. (2008)
GI	phyB signaling/clock associated	Yu et al. (2008)
Other proteins		
FHY1/FHL	phyA signaling component	Hiltbrunner et al. (2005, 2006)
PAPP5	Phosphatase	Ryu et al. (2005)

Moreover, members of the SPA protein family have also been colocalized with COP1 on nuclear bodies (Seo et al., 2003; Zhu et al., 2008). These results suggest that these transcriptional regulators could at least be ubiquitinated on photobodies. In addition, cry2-containing photobodies may be associated with cry2 degradation (Yu et al., 2009).

In mammalian cells, components of the ubiquitin-proteasome pathway have been shown to localize to subnuclear foci called clastosomes (Lafarga et al., 2002). However, it has not been demonstrated whether the proteasome colocalizes with photobodies in plants. In fact, the *Arabidopsis* proteasome components AtS6A and AtS9 were instead shown to localize to the nucleoplasm (Kwok et al., 1999). CUL4 and DDB1, the other two key components of the CUL4-DDB1-COP1-SPA complex, also localize to the nucleoplasm (Zhang et al., 2008). In addition, COP9, which is the key component of the COP9 signalosome that regulates the activity of cullin-based E3 ubiquitin ligases, localizes to the nucleoplasm in both light- and dark-grown *Arabidopsis* cotyledon and hypocotyl protoplasts (Chamovitz et al., 1996; Staub et al., 1996). Taken together, these results suggest the possibility that the protein substrates are modified on photobodies and are subsequently degraded in the nucleoplasm. Future investigation into the constituents of photobodies will further clarify this model.

Although photobodies are likely involved in protein degradation, it is quite clear that not all of their constituents are subject to protein degradation. For example, PIF7 is localized to phy photobodies but is stable in the light (Leivar et al., 2008). What other functions could photobodies have besides protein degradation? One possibility is that photobodies are involved in transcriptional regulation (Fig. 3C). This is supported by the fact that many of the photobody constituents are transcriptional regulators, which could bring their targeted genes to the vicinity of photobodies. The link between nuclear bodies and transcriptional regulation is well documented (Zhao et al., 2009). For example, Promyelocytic Leukemia (PML) protein bodies have been shown to organize the higher order chromatin structures of the Major Histocompatibility Complex (MHC) class I locus, and they regulate the expression of MHC class I genes in mammalian cell lines by direct interactions between PML and Special AT-Rich Sequence Binding Protein1 (Kumar et al., 2007). Likewise, phy photobodies could serve as organization centers involved in the regulation of light-responsive genes (Fig. 3C). This model and the degradation model in Figure 3B are not mutually exclusive, as the degradation of some transcriptional activators has been shown to be coupled with their transcriptional activity (Lipford et al., 2005; Collins and Tansey, 2006). Therefore, it is also possible that photobodies are sites for both the degradation of transcriptional regulators and the regulation of transcription.

GENETIC DISSECTION OF PHOTOBODY FUNCTION

Although nuclear bodies have been extensively studied, particularly in mammalian systems, and key constituents of a few nuclear bodies have been successfully identified by cell biology and proteomic approaches (Gall, 2000; Bernardi and Pandolfi, 2007), the precise function and regulation of nuclear bodies are still poorly understood. *Arabidopsis* represents an ideal organism to dissect the function of nuclear bodies by the combination of molecular genetic and cell biological approaches (Shaw and Brown, 2004; Collier et al., 2006; Fang and Spector, 2010). Because their steady-state pattern can be precisely manipulated by external light quality and quantity, phyB-containing photobodies provide an excellent model system to investigate the function and regulatory mechanisms of nuclear bodies in relation to signaling events in the nucleus (Chen, 2008).

We recently reported a forward genetic screen aimed at isolating mutants defective in phyB-GFP photobody localization in the light (Chen et al., 2010b). This screen identified a novel phy signaling component, HEMERA (HMR), which itself is localized to the periphery of phyB photobodies. The *hmr* mutant represents a new class of light signaling mutants that are albino and tall in R and FR light. In addition, phyB-GFP localizes to smaller photobodies in *hmr* (Fig. 4). More interestingly, in *hmr* mutants, phyA, PIF1, and PIF3 accumulate in the light (Chen et al., 2010b). HMR is predicted to be structurally similar to RAD23, which is a multiubiquitin receptor that delivers multiubiquitinated proteins to the proteasome for degradation, suggesting that HMR could play a similar role in phyA, PIF1, and PIF3 degradation in the light (Chen et al., 2010b). Taken together, these results provide genetic evidence supporting the model in which photobodies are sites for protein degradation (Fig. 3B; Chen et al., 2010b). Further investigation of the biochemical functions of HMR as well as the identification of other genes from the same genetic screen will

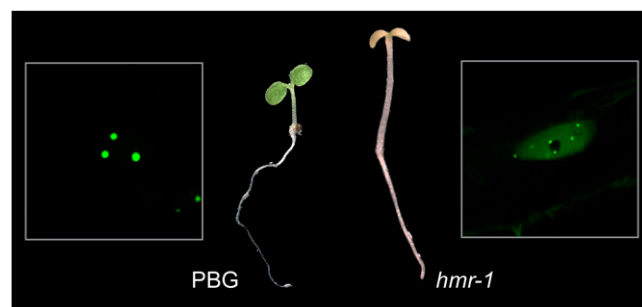


Figure 4. The *hmr* mutant. Images of 4-d-old PBG (the parental type of *hmr-1*) and *hmr-1* mutant seedlings grown under $8 \mu\text{mol m}^{-2} \text{s}^{-1}$ light. The *hmr* seedling has both long-hypocotyl and albino phenotypes. Confocal images show that phyB-GFP is localized to large photobodies in PBG seedlings. By contrast, phyB-GFP fails to form large photobodies and is instead localized to smaller photobodies in *hmr-1*.

likely provide greater insight into the link between photobodies and protein degradation.

PERSPECTIVE

Photoreceptor-containing photobodies in plants are unique and fascinating subnuclear domains whose assembly and function are directly regulated by light. The localization of photoreceptors, including *phys* and *crys*, to photobodies is triggered by a light-induced conformational switch to their active states. It is likely that the exposure of certain domains in the active state, such as the C-terminal domain of *phyB*, facilitates new protein-protein interactions and the “formation” or recruitment of photoreceptors and other signaling molecules to photobodies. The detailed mechanism of photobody assembly is still elusive. Localization studies of light signaling components and recent genetic evidence support the model that photobodies are sites for light signaling events, such as light-dependent turnover of key transcriptional regulators. However, we have only begun to understand the regulatory mechanisms and functions of photobodies. Many key questions remain to be answered. It is still not clear whether photobody-localized transcriptional regulators are degraded or only modified on photobodies. We still do not know whether photobodies are directly involved in transcriptional regulation and whether they are associated with chromatin. Accumulating evidence shows a convergence between the light signaling pathway and other signaling pathways, including those of temperature, hormones, and the circadian clock, on shared downstream signaling molecules (Kami et al., 2010; Lau and Deng, 2010; Leivar and Quail, 2011). Could photobodies serve as a hub for the interaction between these signaling pathways? We anticipate that by using a combination of molecular genetic, cell biological, proteomic, and genomic approaches, studies over the next few years promise to answer some of these questions and uncover new mechanisms of function and regulatory mechanisms for photobodies in light signaling.

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