Ceylon Journal of Science 45(1) 2016: 3-13 DOI: http://dx.doi.org/10.4038/cjs.v45i1.7359

REVIEW ARTICLE

Shedding light on plant development: light signalling in the model plant *Arabidopsis thaliana*

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Received: 19 April 2016; Accepted: 17 May 2016

Abstract: Light is one of the most important factors regulating plant growth and development. Depending on the availability of light, seedlings undergo two different developmental programs namely photomorphogenesis in the presence of light, and skotomorphogenesis in the absence of light. In the model plant *Arabidopsis thaliana* mutants in light signalling path ways have been identified that misregulate this response. The mechanisms behind light and dark growth have been studied extensively and recent studies have revealed how light signals are perceived and transmitted to downstream components. This review provides insight into light-perceiving photoreceptors and other positive and negative regulators of light signalling as well as interactions between these components. Genetic and biochemical evidence for the basis of light signalling mechanisms are discussed as well as the importance of light signalling in plant development.

Keywords:*Arabidopsis thaliana*, photomorphogenesis, photoreceptors, COP1, DET1, CUL4-DDB1 E3 ligases.

INTRODUCTION

Among all the external stimuli affecting plant development, light has an important role in photosynthesis, chloroplast biogenesis, germination, seedling development, floral induction, phototropism, and shade avoidance. Thus light acts not only as an energy source but, in addition, functions as a trigger for growth and development (Chory 1993, Deng 1994, Dong et al. 2015).

The effect of light on plant development can be clearly detected during seedling growth. Seedling growth in the dark (skotomorphogenesis) has a developmentally arrested etiolated phenotype with elongated hypocotyls (embryonic stems), small folded cotyledons (embryonic leaves), and undeveloped chloroplasts. In contrast, seedling growth in the light (photomorphogenesis) results in short hypocotyls, open cotyledons, and developed chloroplasts (Figure 1a) (Chory 1993). These distinct phenotypes have been used in genetic studies to examine light signal transduction pathways. Mutants have been identified in the model plant *Arabidopsis thaliana* that show the opposite phenotypes to those exhibited in wild type plants. These light signalling mutants are broadly divided into two classes, mutants showing light grown phenotypes in the dark and mutants showing dark grown phenotypes in the light (Figure 1b). These phenotypes are a consequence of defects in either positive or negative regulators of light signalling (Quail 1991, Chory 1993).

Arabidopsis thaliana (family Brassicaceae) is an excellent model plant to study the genetic basis of the effects of external environmental factors. It has a small genome (125Mb), the first plant genome to be fully sequenced. In addition, its small size, rapid growth, and extensive collections of mutants and other molecular resources make *Arabidopsis* an excellent model plant. Its ability to produce large numbers of progeny and ease of *Agrobacterium* mediated transformation has also contributed to making Arabidopsis a popular genetic model (Sivasubramanian et al. 2015).

Although the major positive and negative regulators of light signalling in *Arabidopsis* were discovered more than 20 years ago, direct biochemical interactions between these components were revealed only recently.

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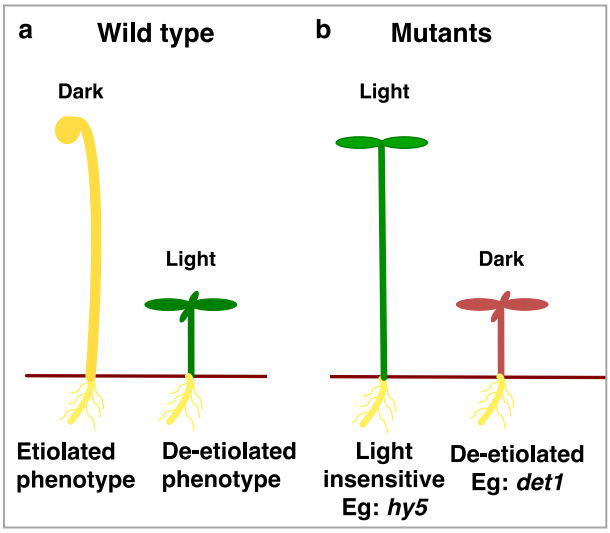

Figure 1: Dark and light grown wild type seedling phenotypes (a) and light signalling mutants (b).

This review focuses on recent advances in our understanding of light signalling in *Arabidopsis thaliana*, with emphasis on interactions between key regulatory components of light signalling.

POSITIVE REGULATORS OF LIGHT SIGNALLING

When a component of the light signalling pathway that is involved in perception of the light signal or transduction of the light signal to downstream components is compromised, such mutants will exhibit a seedling phenotype that is insensitive, or exhibits reduced response, to light. That is, these seedlings will show an etiolated phenotype in the light, with elongated hypocotyls and reduced cotyledon expansion (Figure 1b). This group of mutants can be categorized into photoreceptor mutants and mutants in photomorphogenesis-promoting transcription factors.

Photoreceptors

Initiation of light signalling occurs via perception of light by photoreceptors. Plants have evolved several photoreceptors to sense and respond to a broad range of light frequencies in the environment. The major photoreceptors are the far-red and red light detecting phytochromes, blue/UV-A light sensing cryptochromes and phototropins, and UV-B detecting receptors, such as UVR8 (Galvão and Fankhauser 2015).

Arabidopsis has five phytochrome isoforms (phyA-E). Far red perception is mediated by phyA, while phyB-E initiate red light signalling, with phyB acting predominantly (Wang and Wang 2015). Phytochromes occur in a biologically active Pfr form and an inactive Pr form. Pfr and Pr are photoconvertible, where Pr is transformed into Pfr upon red light (R) absorption and Pfr transformed into Pr upon far-red (FR) light absorption. This conformational change in phytochromes is an important regulatory switch that mediates transduction of light signals to downstream components (Furuya 1993). For phyB, conversion to the Pfr form reveals a masked nuclear localization signal that results in nuclear import in the presence of light. PhyA nuclear localization requires FHY1 (FAR RED ELONGATED HYPOCOTYL 1) and FHL (FHY1 LIKE) (Wang and Wang 2015). Phys are homodimeric chromoprotein complexes that contain a phytochromobilin (PϕB) chromophore. Phys consist of chromophore binding, dimerization and kinase domains (Burgie et al. 2014). Pr to Pfr conversion occurs after light activates the bilin chromophore, which undergoes isomerization and thereby a confirmation change in hairpin and helical spine structure, which stabilizes the Pfr form (Burgie et al. 2016).

Cryptochromes are involved in blue light mediated regulation of seedling development and photoperiodic initiation of flowering. Analysis of cryptochrome 1 and 2 (*cry1 cry2)* mutants has shown that CRY1 and CRY2 have both unique and overlapping functions in these responses. CRY2 is always nuclear localized while CRY1 is

either nuclear or cytoplasmic. Upon blue light absorption, the main chromophore in CRYs is rapidly photo reduced, resulting in conformational changes that facilitate interactions with downstream signalling components (Galvão and Fankhauser 2015, Liu et al. 2016).

Photomorphogenesis-promoting transcription factors

LONG HYPOCOTYL 5 (HY5) was one of the first positive regulators of photomorphogenesis to be characterized. The *hy5* mutants were initially identified in a screen for insensitivity to light inhibition of hypocotyl elongation (Figure. 1b) (Koornneef et al. 1980). *hy5* mutants are deficient in red, far-red, and blue light responses and act downstream from the photoreceptors (Chory 1992, Ang and Deng 1994). In addition, *hy5* mutants have defects in chlorophyll accumulation and lateral root formation (Pepper and Chory 1997, Oyama et al. 1997).

HY5 encodes a nuclear localized basic leucine zipper transcription factor that promotes photomorphogenesis in a broad range of wavelengths (Oyama et al. 1997). Studies of photoreceptor mutants and overexpression lines have shown that both phytochromes and cryptochromes promote HY5 accumulation in the nucleus. The key photoreceptor responsible for HY5 accumulation in R light is phyB while phyA plays a more important role in FR light. CRY1 and CRY2 are involved in HY5 accumulation under blue light conditions (Osterlund et al. 2000).

Chromatin immuno-precipitation and whole genome expression analysis have shown that HY5 specifically binds to the promoters of a large number of genes of which 10% encode transcription factors. In addition, 24% of light regulated genes are HY5 targets, including both light induced and light repressed genes, indicating that HY5 has a dual role in transcriptional regulation of light signalling as an activator as well as a repressor (Lee et al. 2007).

CALMODULIN 7 (CAM7) has a critical role in transcriptional regulation of *HY5* during seedling development in a broad spectrum of light conditions. CAM7 directly interacts with the *HY5* promoter and upregulates *HY5* transcription. HY5 also activates its own gene expression. Therefore both HY5 and CAM7 positively regulate *HY5* transcription (Abbas et al. 2014).

Other positive regulators of photomorphogenesis include HY5 HOMOLOG (HYH), a G-box binding bZIP transcription factor which shows functional redundancy with HY5. Unlike *hy5*, *hyh* mutants exhibit resistance to inhibition of hypocotyl elongation only in blue light thus HYH acts as the main positive regulator of blue light signalling mediated by CRY1 and CRY2. HYH protein levels were significantly lower in *hy5* mutants indicating that HY5 is essential for HYH protein accumulation. *hyh* mutants flower earlier than wild type but *hy5 hyh* double mutants did not show an additive effect on flowering time phenotypes (Holm et al. 2002).

HFR1, a bHLH transcription factor, is another positive regulator of photomorphogenesis. The *hfr1* mutant has elongated hypocotyls in FR light. HFR1 is responsible for phyA mediated FR and CRY1 mediated blue light signalling (Jang et al. 2005, Yang et al. 2005, Casal et al. 2014).

LAF1, a Myb transcriptional activator, is involved in transmitting phyA signals to downstream signalling components. Interestingly HY5, HFR1, and LAF1 have the ability to bind with each other and decrease degradation of each other (Casal et al. 2014).

NEGATIVE REGULATORS OF LIGHT SIGNALLING

In *Arabidopsis* mutants have been identified that resemble light grown plants even when grown in the dark, that is, exhibit short hypocotyls, open cotyledons, and light regulated gene expression (Figure 1b) (Chory et al. 1989). These mutants are referred to as *constitutive photomorphogenic (cop), de-etiolated (det),* or *fusca (fus*). Subsequent cloning of the genes associated with these loci revealed the identity of these central repressors of photomorphogenesis. The COP/DET/FUS proteins are components of three distinct protein complexes: (i) the COP1-SPA complex, (ii) the COP9 signalosome (CSN) and (iii) the COP10-DET1-DDB1 (CDD) complex (Lau and Deng 2012).

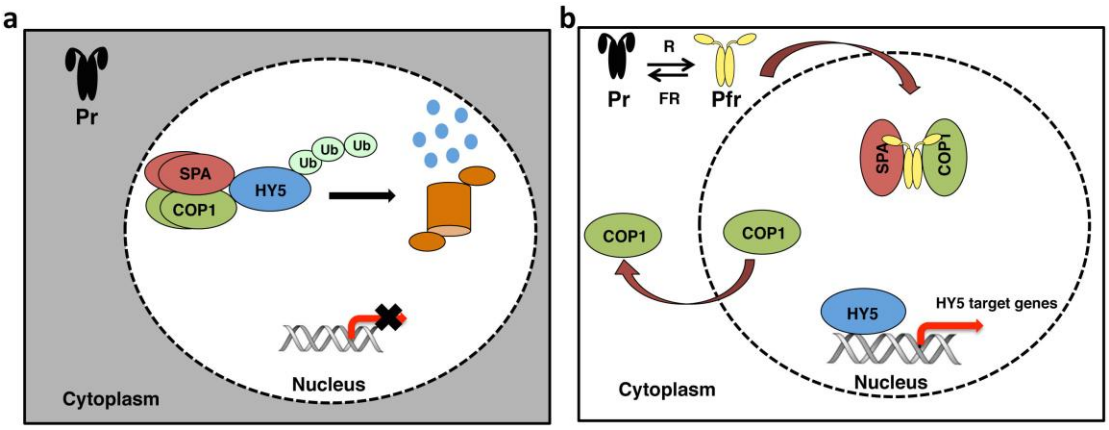

Figure 2: COP1 regulation of HY5 levels in (a) dark and (b) light. In the dark, nuclear localized COP1-SPA targets HY5 for degradation via the 26S proteasome and prevents photomorphogenesis. In the light, the active Pfr form of phytochrome enters the nucleus and inhibits COP1-SPA interaction. In addition, COP1 is slowly exported from the nucleus. This results in HY5 accumulation, expression of HY5 target genes and light growth.

In addition to the *COP/DET/FUS* genes, a group of phytochrome interacting basic helix-loop-helix (bHLH) transcription factors were later identified as another class of negative regulators of light signalling. These transcription factors are called PHYTOCHROME INTERACTING FACTORS (PIFs) (Leivar et al. 2008, Leivar and Monte 2014).

COP1

COP1 is a 76 kDa protein that targets positive regulators of light signalling for degradation. Ubiquitination is a mechanism whereby ubiquitin (Ub) tags are added to proteins, targeting them for subsequent proteolytic degradation via the 26S proteasome pathway. COP1 interacts with SUPPRESSOR OF PHYA-105 (SPA) proteins to form a RING E3 ubiquitin ligase which, in the dark, targets photomorphogenesis promoting transcription factors such as HY5, HYH, HFR1, and LAF1 for degradation via the 26S proteasome system, leading to skotomorphogenesis (Casal et al. 2014).

COP1 has 3 distinct domains that facilitate interaction with other proteins, namely a RING finger domain, a coiled–coil domain, and WD-40 repeat domain. COP1 interacts with most of its substrates via the WD-40 domain (Yi and Deng 2005). COP1 is capable of auto-ubiquitination and SPA1 has no specific role for this self-ubiquitination. However COP1 E3 ligase activity is strictly impaired in *spa1* mutants and thus, SPA1 is required for ubiquitination of other substrates (Seo *et al.*, 2003).

COP1 interacts with phyA, phyB, CRY1, and CRY2 in response to photoperception by the photoreceptors. Two mechanisms appear to contribute to repression of COP1 in the light. One is the slow translocation of COP1 from the nucleus to the cytosol and the other is rapid inhibition of COP1 by the photoreceptors (Casal et al. 2014, Lu et al. 2015). Recent studies have shown that both phyA and phyB co-localize with SPA1 in the nucleus. Photoactive phyA and phyB interact with SPA1 in a light dependent manner and prevent COP1-SPA interaction and thereby formation of the active COP1-SPA complex (Figure 2) (Lu et al. 2015, Sheerin et al. 2015). In addition, both CRY1 and CRY2 regulate COP1 activity in blue light by interacting with SPA1 and preventing COP1-SPA E3 ligase function (Lau and Deng 2012, Liu et al. 2016).

Another recently identified COP1 repressor, COP1 SUPPRESSOR 2 (CSU2), directly interacts with COP1 and inhibits its E3 ligase activity. *CSU2* loss of function suppresses the *cop1* mutant phenotype (Xu et al. 2015).

In contrast to its negative regulatory role in visible light signalling, COP1 acts as a positive regulator in UV-B signalling. Upon exposure to UV-B, the UV photoreceptor UVR8 undergoes a conformational change enabling interaction with COP1. As a result *HY5* expression is increased, which leads to activation of UV-B induced genes. Thus, COP1 has a role in plant UV-B tolerance (Lau and Deng 2012, Kong and Okajima 2016).

The COP9 signalosome

The COP9 signalosome (CSN) consists of eight subunits, six of which were identified as

cop/det/fus mutants. The CSN regulates the activity of CULLIN RING E3 ligases and thereby plays an important role in regulation of ubiquitin/proteasome mediated protein degradation. The CSN removes the ubiquitin-like modifier Nedd8 from CUL based E3 ligases (Lau and Deng 2012, Dong et al. 2015). Loss of function *csn* mutants shows a constitutive photomorphogenic phenotype because the CSN is essential for COP1 activity. Thus, a number of genes including light regulated genes are mis-regulated in *csn* mutants (Chamovitz 2009, Wang et al. 2009).

The COP10/DET1/DDB1 (CDD) complex

In the CDD complex, COP10 and DET1 form a complex with CUL4 via DAMAGED DNA BINDING protein 1 (DDB1).

COP10

COP10 is an ubiquitin conjugating enzyme (E2) variant (Suzuki et al. 2002). COP10 is also required for COP1 mediated degradation of HY5 (Osterlund et al., 2000). COP10 directly interacts with COP1, the CSN, and proteasome subunits and forms a stable complex with DDB1 and DET1 (CDD complex). The CDD complex promotes ubiquitin chain formation and enhances E2 activity. COP10 itself has no E2 activity but can enhance the activity of other E2s in the presence or absence of the CDD complex (Yanagawa et al. 2004). The CDD complex interacts with CUL4 and shows E3 ligase activity, however the target proteins were unknown until recently. The only known direct target of the CDD complex is HFR1 (Chen et al. 2006, Shi et al. 2015).

DET1

De-etiolation refers to inhibition of hypocotyl elongation and induction of leaf expansion and differentiation. The de-etiolated mutants in Arabidopsis, such as *det1*, resemble light grown plants when grown in complete darkness. Hence det1 mutants exhibit short hypocotyls, expanded cotyledons with noticeable leaf primordia and initiation of chloroplast development in the dark. In addition, det1 mutants express light regulated genes in the dark, such as photosynthesis related genes. Thus, DET1 acts as a negative regulator of seedling de-etiolation response. *det1* mutants can continue to grow for extended periods in the dark, developing leaves and flowers. Light grown adult *det1* plants are small with increased number of inflorescence stems as well as reduced fertility (Chory et al. 1989, Pepper et al. 1994). DET1 is also involved in spatial patterning of light regulated gene expression and chloroplast development (Chory and Peto 1990). In addition, pleiotropic defects in *det1* mutants, including morphological defects and abnormal gene expression in the dark and light, can be restored by increased peroxisome function. TED3 is the Arabidopsis homologue of the yeast and mammalian peroxisomal protein PEX2 and *ted3* gain of function mutants can rescue *det1* phenotypes. This indicates that both DET1 and peroxisomes play important roles in photomorphogenesis (Hu et al. 2002).

hy5 mutants suppress *det1* dark grown seedling phenotypes as well as *det1* light grown adult phenotypes such as size, flowering, apical dominance, and fertility phenotypes. This suggests that *HY5* acts downstream from *DET1*, consistent with the lack of HY5 degradation in *det1* mutants (Chory 1992, Pepper and Chory 1997, Osterlund et al. 2000, Fernando and Schroeder 2015). DET1 represses *CHLOROPHYLL A/B BINDING PROTEIN 2* (*CAB2*) gene expression in the dark but activates it in the light. DET1 regulation of *CAB2* expression was found to be via HY5 and the circadian regulator CIRCADIAN CLOCK ASSOCIATED 1 (CCA1) (Maxwell et al. 2003).

DET1 is nuclear localized and interacts directly or indirectly with a number of other proteins. DET1 interacts physically and genetically with DAMAGED DNA BINDING PROTEIN 1 A/B (DDB1A/B) and COP10 to form the CDD complex (Pepper et al. 1994, Schroeder et al. 2002, Yanagawa et al. 2004, Ganpudi and Schroeder 2013). The CDD complex in turn interacts with CUL4 to form an active E3 Ub ligase. However, a direct target of the complex was not known until the recent discovery that HFR1 degradation mediated by the CUL4-CDD complex (Chen et al. 2006, Shi et al. 2015). In addition, COP1 nuclear retention and HY5 degradation require the activity of the CDD and CSN complexes (von Arnim et al. 1997, Osterlund et al. 2000, Wang et al. 2009). Thus, *det1* mutants have increased levels of HY5 protein but DET1 does not appear to directly interact with HY5 (Osterlund et al. 2000, Lau et al. 2011). Also there is no evidence of direct interaction between COP1 and DET1, therefore

the basis of this mechanism is still not clear (Chen et al. 2010).

In contrast, DET1 was found to directly interact with the SINAT5 E3 ligase and block degradation of LATE ELONGATED HYPOCOTYL (LHY), a component of the Arabidopsis circadian clock. SINAT5 interacts with both LHY1 and DET1 but ubiquitinates only LHY. Thus, DET1 may influence flowering time in Arabidopsis by affecting protein abundance of LHY via inhibition of the SINAT5 E3 ligase (Song and Carré 2005, Park et al. 2010).

In addition to its role in E3 ligase complexes, DET1 has been shown to be involved in transcriptional regulation (Lau et al. 2011, Huang et al. 2014). DET1 acts as a transcriptional co-repressor of the Arabidopsis circadian clock. CIRCADIAN CLOCK ASSOCIATED 1 (CCA1) and LHY1 are MYB transcription factors with partially overlapping functions that act as transcriptional repressors in the morning phase of the central loop of circadian clock. DET1 directly interacts with CCA1 and LHY1 to repress CCA1/LHY1 target genes. DET1 is essential for CCA1 transcriptional repression activity and for functioning of the plant circadian clock (Lau et al. 2011). Moreover, DET1 has a possible role in chromatin remodelling via binding to non-acetylated histone 2B (H2B) tails in nucleosomes (Benvenuto et al. 2002).

DET1 not only represses photomorphogenesis but also represses flowering by altering the photoperiod and autonomous pathways. DET1 delays flowering particularly under short day conditions. Flowering is controlled by multiple signalling components including GIGANTIA (GI) and FLOWERING LOCUS T (FT). GI functions as a flowering inducer and activates *FT* transcription. DET1 directly interacts with GI and delays flowering by inhibiting the interaction between GI and the *FT* promoter. Thus, DET1 does not affect GI protein stability but functions as a repressor of *FT* transcription (Kang et al. 2015). In addition, DET1 binds directly to MULTICOPY SUPPRESSOR OF IRA1 4 (MSI4), which is part of a CUL4-DDB1 complex that alters the expression of *FLOWERING LOCUS C (FLC).* FLC inhibits floral transition by suppression of floral inducers like FT. The CUL4-DDB1-MSI4 E3 ligase associates with POLYCOMB REPRESSIVE COMPLEX 2 containing histone methyltransferase CLF (CLF-PCR2) and represses *FLC* expression. *det1* exhibits altered *FLC* promoter methylation and expression (Pazhouhandeh et al. 2011, Kang et al. 2015).

A recent study showed that DET1 suppresses accumulation of DELLA proteins, which are negative regulators of Gibberellic acid (GA) signalling. Light and GA antagonistically regulate seedling growth and DELLAs play a role in promoting skotomorphogenesis in the dark. Therefore, DET1's role in repression of photomorphogenesis may be partly through negative regulation of DELLA protein levels in the dark (Li et al. 2015). DELLAs inhibit another negative regulator of photomorphogenesis, PIFs (discussed below) (Davière and Achard 2016).

Other roles of DET1 include the recent discovery that DET1 is involved in stabilization of PHYTOCHROME INTERACTING FACTORS (PIFs), which will be discussed later in this review, and mediating degradation of HFR1 (Dong et al. 2014, Shi et al. 2015). DET1 directly regulates both positive (HFR1) and negative (PIF1) regulators of seed germination. In the dark, DET1 degrades HFR1 and stabilizes PIF1, repressing seed germination. In the light DET1 is somehow inactivated, resulting in increased HFR1 and decreased PIF1, inducing seed germination (Shi et al. 2015). Therefore, DET1 is not only a central repressor of photomorphogenesis but also a central repressor of seed germination and flowering time.

CUL4 / DDB1A/B E3 ligase complexes in light signaling

Cullin proteins are the scaffolding subunits of E3 ligase complexes, where the N-terminus of the cullin binds to an adaptor protein and the C-terminus binds to the RING finger protein RBX1. The adaptor protein functions to connect the cullin to specific substrate receptors that enable interaction with the substrate to be targeted for ubiquitination. For instance, in CUL4 E3 ligases, DDB1 acts as the adaptor and interacts with a number of different substrate receptors. These substrate receptors commonly have roughly seven WD40 domains thus are called DWD (DDB1 binding WD40) proteins or DDB1 CUL4 ASSOCIATED FACTORS (DCAFs). There are certain proteins, such as DET1 and COP10, which lack a WD40 domain

but still interact with DDB1. CUL4-DDB1-DCAF complexes are involved in a wide array of functions in plants including repression of photomorphogenesis, facilitating damaged DNA repair, and response to abiotic stress (Biedermann and Hellmann 2011).

DDB1 was first identified in mammals as part of the DDB1-DDB2 complex that binds to UV damaged DNA and is involved in nucleotide excision repair of damaged DNA. DDB1 is a highly conserved protein in eukaryotes. In Arabidopsis DDB1 exists as two homologues, DDB1A and DDB1B, which exhibit 91% amino acid identity with each other. Although the two proteins are not biochemically different, DDB1A and DDB1B show distinct functions in the light and dark and the double mutant is embryonic lethal (Schroeder et al. 2002, Bernhardt et al. 2010, Ganpudi and Schroeder 2013).

Distinct DDB1 complexes appear to interact with each other genetically and biochemically. DDB2 is mainly involved in the global genomic repair pathway as a substrate receptor for CUL4-DDB1 (Ganpudi and Schroeder 2011). In *Arabidopsis* however *DDB2* also genetically interacts with *DDB1A* and *DET1*. *DDB2* interactions with *DET1* were shown to be *DDB1A* independent for some adult phenotypes while in some dark grown seedling phenotypes the interactions were *DDB1A* dependent (Al Khateeb and Schroeder 2007). In addition, DET1 is essential for the degradation of DDB2 via the CUL4-DDB1 E3 ligase during UV damage repair. Removal of DDB2 after damaged DNA lesion recognition is an important step, allowing the repair machinery to access the damaged lesions. Thus DET1 and DDB2 work together in UV damaged DNA repair (Castells et al. 2011).

Phytochrome Interacting Factors (PIFS)

The bHLH type transcription factors PHYTOCHROME INTERACTING FACTORs (PIFs) act directly downstream of phytochromes to negatively regulate photomorphogenes is and promote skotomorphogenesis. PIFs (PIF1/PIF3-LIKE 5, PIF3, PIF4, PIF5/PIL6, PIF6/PIL2, PIF7, and PIF8) accumulate in the dark to promote dark growth. In the presence of light, activated phytochromes, in the nuclear Pfr form, interact directly with PIFs. Phytochromes then phosphorylate the PIFs, targeting them for ubiquitination and degradation via the proteasome (Figure 3). While there is redundancy among the PIFs, *pif3* mutants have short hypocotyls in red light while PIF4 and PIF5 are also involved in negative regulation of light signalling. PIF1 regulates seed germination and hypocotyl elongation. Furthermore, *pifq* (which lacks PIF1, PIF3, PIF4 and PIF5) shows a constitutive photomorphogenic phenotype (Leivar and Monte 2014).

Microarray expression profile analysis indicated that DET1 represses photomorphogenesis by regulating a number of transcription factors including PIFs. Thus, lack of PIF3 enhances the *det1* de-etiolated phenotype while overexpression can partially restore seedling de-etiolation in the dark. In addition, DET1 and other components of the CDD complex affect the protein stability of PIFs at the post-transcriptional level. DET1 positively regulates only *PIF3* at the gene expression level but positively regulate all the PIFs at the post-translational level. Moreover, both *det1* and *cop1* mutants have significantly reduced PIF3 protein levels, suggesting that the *DET/COP/FUS* group of genes repress photomorphogenesis in part by mediating protein stability of PIFs and upregulating the function of PIF3 in the dark (Lau and Deng 2012, Dong et al. 2014, Dong et al. 2015, Shi et al. 2015).

Interactions between the two main classes of repressors of photomorphogenesis, COP/DET/FUS and PIFs, are just beginning to unravel. COP1 and PIFs have additive roles in the dark. PIFs enhance the substrate recruitment and ubiquitination functions of COP1 and PIF1 interacts with COP1, SPA1 and HY5. Basically PIF1 functions in COP1 mediated HY5 degradation by enhancing COP1 affinity to HY5, promoting auto-ubiquitination of COP1 then facilitating transubiquitination of HY5 by COP1. Thus, negative regulation of photomorphogenesis by PIFs is not an independent mechanism but acts by affecting the protein stability of HY5 via regulation of COP1-SPA E3 ligase activity. Therefore, PIF1 and COP act as cofactors and synergistically repress light growth in the dark (Xu et al. 2014).

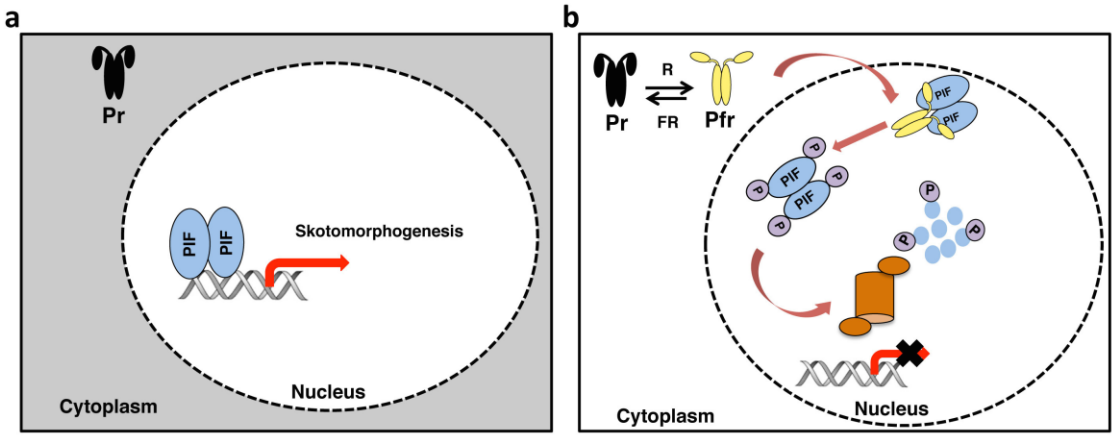

Figure 3: Interaction between light and PIFs in light signalling (a) In the dark phytochromes are in the biologically inactive Pr form and are localized in the cytosol. Homo and heterodimers of PIFs bind to light regulated genes, preventing their expression and repressing photomorphogenesis. (b) In the light, the active Pfr form of phytochromes move to the nucleus to bind and rapidly phosphorylates PIFs. The phosphorylated PIFs are degraded via the 26S proteasome. As a result photomorphogenesis occurs.

LIGHT SIGNALLING OVERVIEW

In summary, different wavelengths of light are perceived by photoreceptors, with phyA mediating FR perception, phyB red light, and CRY1 and CRY2 blue (Figure. 4). In general light absorption results in changes in photoreceptor conformation and/or localization, which facilitates interactions between the photoreceptors and downstream negative regulators. The photoreceptors then inhibit the negative regulators, resulting in disruption of COP1 activity and degradation of the PIFs. In the absence of light the negative regulators positively reinforce each other, with DET1 stabilizing the PIFs and the PIFs promoting COP1 degradation of HY5 and other photomorphogenesis promoting transcription factors. Thus in the dark the negative regulators promote skotomorphogenesis and inhibit photomorphogenesis, while the light inactivation of the negative regulators by the photoreceptors allows light development to proceed (Huang et al., 2014; Dong et al., 2015). Light influences nearly all aspects of growth and development in plants and our understanding of this critical process is finally becoming illuminated. The knowledge gained through genetic and biochemical studies in the model plant Arabidopsis can be transferred to agriculturally more important crops, allowing us to optimize light use during crop production.

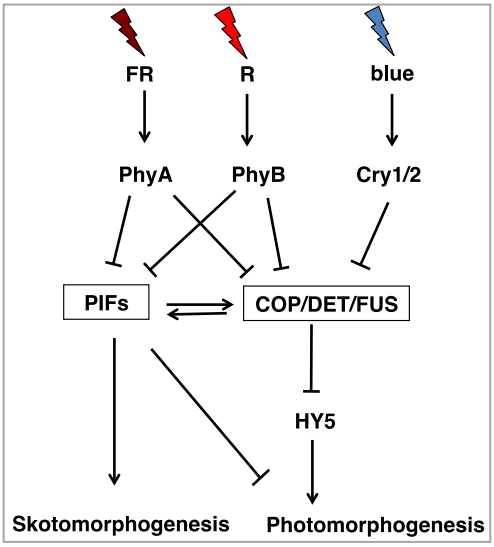

Figure 4: Light perception and signalling pathways (arrows indicate positive regulation and T-bars indicate negative regulation).

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