



Photoreceptor crosstalk in shade avoidance

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Plants integrate a variety of environmental signals to determine the threat of competitor shading and use this information to initiate escape responses, termed shade avoidance. Photoreceptor-mediated light signals are central to this process. Encroaching vegetation is sensed as a reduction in the ratio of red to far-red wavebands (R:FR) by phytochromes. Plants shaded within a canopy will also perceive reduced blue light signals and possibly enriched green light through cryptochromes. The detection of canopy gaps may be further facilitated by blue light sensing phototropins and the UV-B photoreceptor, UVR8. Once sunlight has been reached, phytochrome and UVR8 inhibit shade avoidance. Accumulating evidence suggests that multiple plant photoreceptors converge on a shared signalling network to regulate responses to shade.

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Introduction

Shading from neighbouring vegetation limits photosynthetic productivity and represents a major survival threat to plants grown in dense canopies. Some understory species have therefore evolved shade tolerance strategies which enable them to survive and reproduce at low light levels [1]. Other species compete for light, using an escape strategy termed shade avoidance [2]. Plants detect the presence of competing vegetation and use this information to trigger a suite of developmental responses termed the shade avoidance syndrome (SAS). Rapid SAS responses include leaf hyponasty and stem elongation, which raise leaves above the canopy or towards canopy gaps, facilitating light capture [2]. In *Arabidopsis thaliana*, light quality-mediated changes in gene expression have been detected within

15 minutes [3] and changes in hypocotyl length within 45 minutes [4]. Longer term SAS responses include reduced branching, altered leaf development and accelerated flowering [2]. SAS responses in 3 species (Barley (*Hordeum vulgare*), Coriander (*Coriandrum sativum*) and Arabidopsis) are shown in Figure 1.

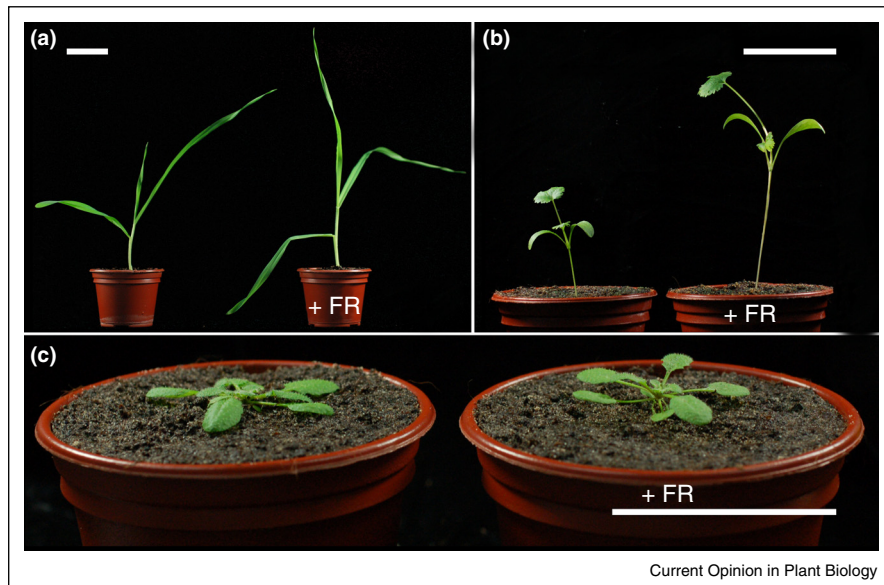
To detect neighbouring vegetation, plants use multiple aboveground cues including the physical touching of leaf tips, the sensing of plant-emitted volatile chemicals and alterations in light quantity and quality [5]. Light quality signals are of paramount importance. Sunlight reflected or transmitted through living vegetation is depleted in red, blue and UV-B wavebands which are absorbed by green tissue. Reflected/transmitted light is enriched in green and far-red wavebands, resulting in reduced ratios of red to far-red light (R:FR) and blue to green light (B:G). Plants sense these spectral differences using specialised photoreceptors. These include the red (R) and far-red light (FR)-absorbing phytochromes, the blue (B)/UV-A light sensing cryptochromes and phototropins and the UV-B photoreceptor protein, UVR8. Horizontally propagated FR signals from nearby plants can reduce R:FR ratio (R:FR), providing a pre-canopy closure warning of competition [6]. Once shaded, R:FR is lowered further, B:G ratios decrease and UV-B levels are severely depleted [2]. Recent studies have revealed that red/far-red, blue and UV-B photoreceptors converge on a shared signalling network to control shade avoidance. This review will summarise current understanding of this topic, focussing on elongation of the Arabidopsis hypocotyl.

Plant photoreceptors

Phytochromes absorb light using a linear tetrapyrrole chromophore, phytychromobilin, and display R/FR photoreversibility. Synthesised in the biologically inactive Pr form, phytochromes acquire biological activity following photo-conversion to the biologically active Pfr form. Conversion of Pr to Pfr is optimised at R wavelengths (600–700 nm), while Pfr to Pr conversion is optimised at FR wavelengths (700–750 nm). The R:FR of ambient light therefore controls Pr:Pfr equilibrium and hence phytochrome signalling in de-etiolated plants [7]. In Arabidopsis, there are 5 phytochromes (phyA–E), which perform distinct and overlapping functions throughout plant development. phyA displays greater light lability than phytochromes B–E and signals during rapid photo-cycling between its Pr and Pfr forms in FR [8].

The sensing of blue/UV-A wavelengths (300–500 nm) in plants involves three classes of flavoproteins; cryptochromes, phototropins and the Zeitlupe family [9]. Of

Figure 1



Shade avoidance phenotypes. Plants were grown in white light ($90 \mu\text{mol m}^{-2} \text{s}^{-1}$) of high (>6), or low (0.05) R:FR (+FR) to simulate, respectively, the absence or threat of shade in 12 h light/12 h dark photoperiods at 20°C . (a) Barley (*Hordeum vulgare*), (b) Coriander (*Coriandrum sativum*) and (c) *Arabidopsis thaliana*. Scale bar = 50 mm.

these, only cryptochromes have an established role in shade avoidance. In *Arabidopsis*, there are two cryptochrome photoreceptors (*cry1* and *cry2*) which display significant homology to DNA photolyases and perform overlapping and unique roles in plant growth and flowering control. Unlike *cry1*, *cry2* displays blue light-mediated ubiquitination and degradation within the nucleus [10] and is thought to enhance plant sensitivity to low photon irradiances [11]. Phototropins are serine/threonine kinases which undergo blue light-mediated autophosphorylation. Blue/UV-A sensing involves two specialised Light Oxygen Voltage (LOV) domains at the N terminus of the protein. Two phototropins exist in *Arabidopsis*, *phot1* and *phot2*, which regulate phototropism, stomatal opening, chloroplast movement and leaf flattening [9]. A role for phototropism has been suggested in light foraging within dense canopies, but experimental evidence for this is currently lacking [5]. For further information on photoreceptor nomenclature, see Table 1.

In *Arabidopsis*, UV-B signals are perceived via the 7-bladed β -propeller protein, UVR8 [12]. UVR8 has a dimeric resting state that on absorbance of UV-B monomerises and initiates signalling. The early molecular events associated with UV-B absorbance and UVR8 monomerisation are well characterised [12,13,14,15,16] but the details of how UVR8 monomerisation leads to altered gene expression remain less clear.

Phytochromes and shade avoidance

phyB performs a dominant role in SAS inhibition, with redundant roles identified for phyD and phyE in *Arabidopsis* [8]. High R:FR establishes a high proportion of active phyB Pfr [7], which is translocated to the nucleus where it binds to the PHYTOCHROME INTERACTING FACTOR (PIF) family of basic Helix-Loop-Helix (bHLH) transcription factors via a conserved Active Phytochrome Binding (APB) domain (Figure 2). Pfr binding triggers PIF phosphorylation, ubiquitination and degradation by the 26s proteasome [17**]. PIF degradation in high R:FR is accompanied by a concomitant reduction in phyB involving Light-Response Bric-a-Brack/Tramtrack/Broad (LRB) E3 ubiquitin ligases [18]. phyB signalling has also recently been shown to be negatively regulated by SUMOylation [19], suggesting additional mechanisms of phy signal attenuation. Low R:FR drives Pfr to Pr conversion, releasing PIF suppression to allow their stabilisation, accumulation and promotion of stem elongation by binding to G-box motifs in a broad range of target genes [17**,20].

Hypocotyl elongation in low R:FR involves rapid tryptophan-dependent auxin biosynthesis requiring TRYPTOPHAN AMINOTRANSFERASE OF ARABIDOPSIS1 (TAA1) [21]. PIF4, PIF5 and PIF7 have a central role in this process; PIF4 and PIF5 display low R:FR-mediated protein stabilisation, whereas PIF7 increases activity following low R:FR-mediated dephosphorylation [22,23]. All drive cell elongation, in part, by up-regulating the

Table 1

Photoreceptor nomenclature, based on [68]

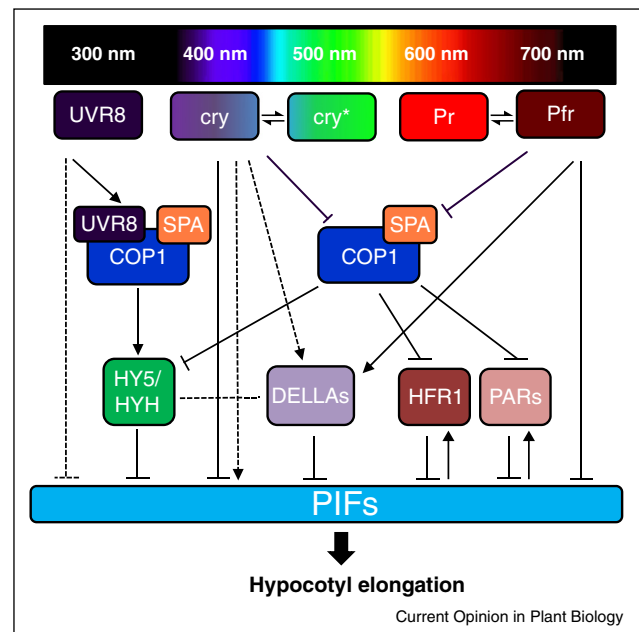
Nomenclature	Description
Pr	Red-light absorbing, biologically inactive form of phytochrome. Pr to Pfr conversion is optimised by red wavelengths (660–670 nm).
Pfr	Far-Red light absorbing, biologically active form of phytochrome. Pfr to Pr conversion is optimised by far-red light wavelengths (725–735 nm).
<i>PHY, CRY</i> e.g. <i>PHYA, CRY2</i>	Wild-type photoreceptor gene.
<i>PHY, CRY</i> e.g. <i>PHYB, CRY2</i>	Photoreceptor apoprotein- the polypeptide encoded by the <i>PHY</i> or <i>CRY</i> gene.
<i>phy, cry</i> e.g. <i>phyA, cry2</i>	Photoreceptor holoprotein. Apoprotein with covalently bound chromophores, representing the fully-assembled photoreceptor.

transcription of *YUCCA* enzymes controlling the rate limiting step of a major auxin biosynthesis pathway [20,23]. In seedlings, low R:FR-induced auxin biosynthesis has been shown to drive increased expression and relocalisation of the auxin efflux regulator PIN-FORMED 3 (PIN3) to direct auxin to the hypocotyl epidermis [24]. Under low levels of photosynthetically active radiation (PAR), PIF4 and PIF5 increase the sensitivity of plants to auxin [20,25]. Modelling has shown that increased auxin production via the TAA1 pathway is primarily responsible for shade-avoidance at high PAR. At low PAR, however, PIF4 and PIF5 mediate increases in auxin sensitivity to counter-balance a relative reduction in auxin production [26**]. The authors suggest that in low PAR conditions, where resources are limited, increasing sensitivity to basal auxin levels is more efficient than *de novo* synthesis [26**].

PIFs are generally regarded as positive regulators of SAS. They do, however, promote transcript abundance of the bHLH protein, LONG HYPOCOTYL IN FAR RED (HFR1) and the HLH proteins PHYTOCHROME RAPIDLY REGULATED 1 (PAR1) and PAR2, which are thought to prevent excess elongation by forming competitive complexes with PIFs to negatively regulate their activity (Figure 2) [27,28,29,30]. Further limitation of SAS responses is provided by low R:FR-mediated stimulation of *phyA* signalling in deep shade [2,31]. The bZIP transcription factor, ELONGATED HYPOCOTYL 5 (HY5), has been implicated in the *phyA*-mediated down-regulation of early-induced shade avoidance genes in prolonged low R:FR [32] and, similarly to HFR1, forms non-functional complexes with PIFs as well as occupying PIF-target promoters [33,34**,35**].

In addition to the direct regulation of PIFs, photoactivated *phyB* has also been observed to bind to SUPPRESSOR OF *PHYA*-105 (SPA) proteins and inhibit their interaction with CONSTITUTIVELY PHOTOMORPHOGENIC 1 (COP1) [36]. The COP1/SPA1 E3 ubiquitin ligase

Figure 2



Photoreceptor-PIF signalling pathways in shade avoidance.

The UVR8 photoreceptor is activated by UV-B [12]. *crys* are activated by B/UV-A light and converted to an inactive, fully-reduced state by G (*cry**). Reversion to an oxidised form occurs in the dark [40]. Conversion of *phy* to the active Pfr form is optimised in R and reversed by FR [7]. In shade conditions; low R:FR ratio promotes *phy* conversion to the Pr form, while low B promotes *cry* accumulation. Sun flecks from canopy gaps moderate the shade avoidance response by driving Pr to Pfr conversion and the monomerisation of UVR8 [54*,65]. UV-B triggers PIF degradation, likely via UVR8 [54*]. *phys* physically bind to PIFs in R and promote their degradation [17**]. *crys* bind to PIF4 and PIF5 in LBL, forming a regulatory complex which may promote or repress shade avoidance, depending on cell type [42*]. UVR8 binds to COP1/SPA and promotes the accumulation of HY5 and HYH [49,50,51,52]. HY5 inhibits PIF4 transcript abundance and limits PIF activity via direct interaction and competitive promoter binding [34**,35**,66*]. In UV-B, HY5 increases expression of *GA2ox1* which likely stabilises DELLAs [54*]. DELLAs inhibit PIF function [58,59*] and are degraded in LBL and low R:FR, suggesting *cry* and *phy*-mediated stabilisation [56,57,58]. *phyB* and *crys* disrupt the COP1/SPA complex [36,37] which targets HY5, HFR1 and PARs for degradation [30,33]. HFR1 and PARs are positively regulated by PIFs [67]. HFR1 and PARs bind to PIFs to form non-functional complexes [27,28,29,30]. In low R:FR, PIFs primarily promote auxin biosynthesis. In LBL, PIFs primarily promote the accumulation of cell wall modifying enzymes. Both drive elongation growth in shade avoidance [23,38,42**]. Solid lines represent mechanisms shown to regulate at least one of the key PIFs controlling shade avoidance- PIF4, PIF5 or PIF7. Dotted lines represent hypothesised regulatory mechanisms.

complex degrades negative regulators of PIF activity such as HY5 and HFR1 (Figure 2). Mutant analyses in low R:FR suggest that the COP1/SPA1 complex is responsible for positive regulation of shade avoidance [37]. It is therefore possible that in low R:FR, reduced levels of *phyB* Pfr indirectly enhance PIF activity via increased COP1/SPA1-mediated degradation of negative regulators.

Cryptochromes and shade avoidance

Alongside decreases in R:FR ratio, plants shaded within a canopy also perceive a reduction of ultraviolet-A (UV-A) and blue light (B) and an enrichment of green light [2]. The attenuation of blue light is perceived by cry1 and cry2 in seedlings, with cry1 adopting a dominant role in adult plants [38,39]. Green light has been shown to partially inactivate cryptochrome signalling [40] (Figure 2). Decreased B:G ratios within canopies may therefore further exacerbate low B (LBL)-mediated shade avoidance responses [41]. cry1 and cry2 increase following blue light depletion [42**]. It has recently been shown that both physically interact with PIF4 and PIF5 [42**,43**], confirming a previously identified role for these transcription factors in LBL responses [38,39]. LBL promotes the accumulation of PIF5, but does not seem to effect PIF4 abundance [42**]. Furthermore, cry2 has been shown by chromatin immunoprecipitation-sequencing experiments to bind to PIF4 and PIF5-regulated gene promoters [42**]. Despite crys binding outside the APB motif, this region of PIF4 and PIF5 was required for full LBL response to occur [42**]. The binding of phyB and cry to distinct PIF4/5 regions suggest that PIFs provide a node of cross-talk between low R:FR and LBL light signalling in shade avoidance (Figure 2).

The exaggerated response to LBL observed in *cry* mutants suggests that crys act to suppress shade avoidance [38,42**,44]. The accumulation of cry2 in LBL conditions may therefore act as a negative regulator of PIF activity, preventing excessive elongation. Similar to phys, crys have been shown to bind SPA1 so may also inhibit COP1-E3 ligase activity to antagonise shade avoidance in LBL [45]. Pedmale and colleagues speculate that cry/phyB/PIF may form a multimeric regulatory complex driving shade avoidance in a conditional and cell type-specific manner [42**]. Analysis of cry-PIF-phy signalling in vegetational shade, where R and B are simultaneously depleted would therefore be of interest.

Comparative transcriptomic analysis of low R:FR- and LBL-treated seedlings has revealed clear differences in transcriptome profile, suggesting that low R:FR and LBL-mediated shade avoidance responses operate, at least in part, through separate mechanisms [42**]. Although conclusions differ between studies, it is clear that auxin synthesis is a more dominant component of shade avoidance signalling in low R:FR than LBL [38,39,42**]. In the latter, an additional requirement for brassinosteroid synthesis exists, with hypocotyl elongation responses involving changes in the abundance of cell wall modifying proteins [38,42**].

Phototropins and shade avoidance

Another B signal which may have a role in shade-avoidance under deep shade is the directionality of light filtering through the canopy. Phototropins re-orient leaves and stems towards B/UV-A and reposition chloroplasts to the

surface of the leaf. Lateral B gradients in patchy canopies can induce phototropic curvature of stems [46]. This may be important to maximise energy capture in energy limiting conditions [5]. A role for PIF4 and PIF5 in B-mediated phototropism has been proposed involving repression of auxin biosynthesis. This contrasts with observations in light-grown plants, where PIF4 and PIF5 promote auxin biosynthesis to drive hypocotyl elongation [20,47]. It has recently been suggested that UV-B can enhance B-driven phototropism [48] which may further optimise light foraging in deep shade-conditions.

UVR8 and shade avoidance

UV-B is strongly filtered by canopies [2] so could provide information on the levels of competition faced by a plant. Contrasting with its role in R and B signalling, COP1 acts as a positive regulator of UV-B signalling [49]. UVR8 monomers bind directly to COP1, promoting the expression of HY5 and its close relative HY5 HOMOLOGUE (HYH) [12,50,51,52]. HY5 and HYH are required for the regulation of a large proportion of UVR8-regulated genes [50,51,52]. Downstream of UVR8 monomerization, UV-B inhibits auxin signalling [53,54*] and promotes the inhibition of hypocotyl elongation, petiole elongation and rosette expansion [53,54*,55]. Many of the phenotypic effects of UV-B are opposite to those induced by shade. Indeed, recent studies have indicated that there is direct antagonism of low R:FR and LBL-mediated shade responses by UV-B [54*,56].

UV-B inhibits low R:FR-induced hypocotyl elongation by a dual mechanism (Figure 2). Gibberellic acid (GA) levels increase in low R:FR [57] and promote degradation of growth-repressing DELLA proteins [58]. UVR8 increases the expression of a GA catabolism gene, *GA2ox1*, in a HY5/HYH-dependent manner, which likely contributes to UVB-mediated stabilisation of the DELLA protein RGA [54*]. As degradation is an essential pre-requisite for low R:FR-mediated shade-avoidance to occur [59,60], increased DELLA stability in UV-B would contribute to shade avoidance inhibition, in part through the formation of inactive DELLA:PIF complexes [61,62]. DELLAs can also modify cell elongation independently of PIFs, through regulating microtubule organisation [63]. UV-B appears to directly affect the stability of PIF4 and PIF5 [54*], although the mechanistic understanding of this process is currently lacking. Additional regulatory components of UV-B-mediated shade avoidance inhibition may include direct inhibition of PIFs by HY5 [34**,35**] or increased HFR1 stability via a UVR8-mediated sequestration of COP1, reducing its E3 ligase activity [64].

Conclusions

Recent advances in photomorphogenesis research have revealed a complex shade avoidance signalling network involving multiple photoreceptors (summarised in Figure 2). PIFs 4, 5 and 7 perform a dominant role as

integrators of multiple light cues, driving hormone signalling, the expression of cell wall modifying enzymes and stem elongation in an environment-dependent and possibly cell type-specific manner [23,42**]. The ability to control auxin sensitivity and biosynthesis enables plants to differentially modulate SAS responses to encroaching vegetation and deep canopy shade, where resources are limiting [26**]. Interesting differences have been observed in photoreceptor/PIF interaction. Phytochromes physically bind PIFs, leading to their ubiquitination and degradation [17**]. Cryptochromes also bind PIFs but form regulatory complexes to control gene expression, possibly in combination with phys [42**,43**]. Contrasting with phys and crys, UVR8 appears to regulate PIF function and/or abundance without direct physical interaction [54*]. Opposing roles for COP1 have additionally been observed between phy/cry and UVR8 signalling suggesting fundamental differences in mechanism of action between these photoreceptors [33,64]. Negative regulators of PIF function (HFR1, PARs, HY5, DELLAs) perform a key role in attenuating PIF activity and constraining SAS responses [27,28,29,30,32,34**, 35**,59*,61,62]. Mechanistic detail of how SAS responses are inhibited once the canopy has been overtopped or penetrated by sunflecks is more limited but appears to involve phyA/phyB/UVR8-mediated induction of HY5 and UVR8-mediated suppression of auxin biosynthesis [54*,65]. The majority of photomorphogenesis research to date has focussed on individual photoreceptor signalling pathways. Future analysis of an integrated photoreceptor signalling network in fluctuating natural canopies will be central to developing a full understanding of plant shade avoidance.

Acknowledgements

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