# Photoreceptor Signaling Networks in Plant Responses to Shade

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## Keywords

phytochrome, cryptochrome, shade avoidance, PHYTOCHROME INTERACTING FACTOR (PIF), auxin

## Abstract

The dynamic light environment of vegetation canopies is perceived by phytochromes, cryptochromes, phototropins, and UV RESISTANCE LOCUS 8 (UVR8). These receptors control avoidance responses to preclude exposure to limiting or excessive light and acclimation responses to cope with conditions that cannot be avoided. The low red/far-red ratios of shade light reduce phytochrome B activity, which allows PHYTOCHROME INTERACTING FACTORS (PIFs) to directly activate the transcription of auxin-synthesis genes, leading to shade-avoidance responses. Direct PIF interaction with DELLA proteins links gibberellin and brassinosteroid signaling to shade avoidance. Shade avoidance also requires CONSTITUTIVE PHOTOMORPHOGENESIS 1 (COP1), a target of cryptochromes, phytochromes, and UVR8. Multiple regulatory loops and the input of the circadian clock create a complex network able to respond even to subtle threats of competition with neighbors while still compensating for major environmental fluctuations such as the day-night cycles.

## Contents



## 1. THE CANOPY LIGHT ENVIRONMENT

In crowded plant canopies, the upper leaves intercept the radiation that would otherwise reach lower leaf strata, and the plants become mutually shaded. Managing plant stands involves decisions about sowing density, defoliation, fertilization, cultivar architecture, etc., which affect the area and position of the foliage in relation to the angle of incoming radiation and therefore impact the degree of shade.

Shade involves the wavelength-selective attenuation of irradiance. Green leaves absorb strongly in the range of UV radiation (280–320 nm for UVB, 320–400 nm for UVA) and photosynthetically active radiation (PAR) (400–700 nm), particularly in the blue and red regions of the spectrum; they transmit and reflect more strongly in the far-red (700–800 nm) and infrared wavebands (**Figure 1*a***).

Several elements highlight the significance of the red (660 nm)/far-red (730 nm) photon flux ratio (R:FR) as a signal. As noted by Smith (128), R:FR (*a*) provides a highly reliable indication of shade because it is relatively unaffected by other environmental conditions,

**PAR:** photosynthetically active radiation (400–700 nm)

**R:FR:** red (660 nm)/far-red (730 nm) photon flux ratio

(*b*) relates to the activity of the phytochrome photoreceptor (see below), and (*c*) initiates responses to shade when experimentally altered to simulate a canopy condition. Changes in R:FR caused by reflected light were later shown to provide an early warning signal of the presence of neighbors even before mutual leaf shading is established in growing canopies (5). The reduced irradiance (7) and blue/green ratio (13, 118) of shade also provide signals, which do not share all the elegant features of R:FR but are important for plant responses to shade.

**Figure 1** describes key features of the light environment experienced by tomato plants grown at different densities. The remote sensor probe was placed either facing upward to characterize the light reaching the leaves (**Figure 1*c***) or facing neighbor plants to characterize the horizontally propagating light reaching the stem (**Figure 1*d***). Only the leaves of the plants grown at the highest density mutually shaded each other and received less PAR and a lower R:FR than isolated plants, but the stem received signals from neighbors even at densities lower than those required for mutual leaf shading. In the example presented here (**Figure 1*d***), compared with the controls grown at <20 plants $m^{-2}$, the R:FR reaching the stem was reduced at 90 plants $m^{-2}$ owing to selective light reflection on neighbors, which increased far-red light. At 120 plants $m^{-2}$, the lower stem R:FR was due to the projection of shade from neighbor leaves. The R:FR reaching the stem decreased gradually with increasing plant densities (**Figure 1*d***), and the plants responded to these signals (**Figure 1*e***).

## 2. THE PERCEPTION OF CANOPY LIGHT SIGNALS BY PHOTOSENSORY RECEPTORS

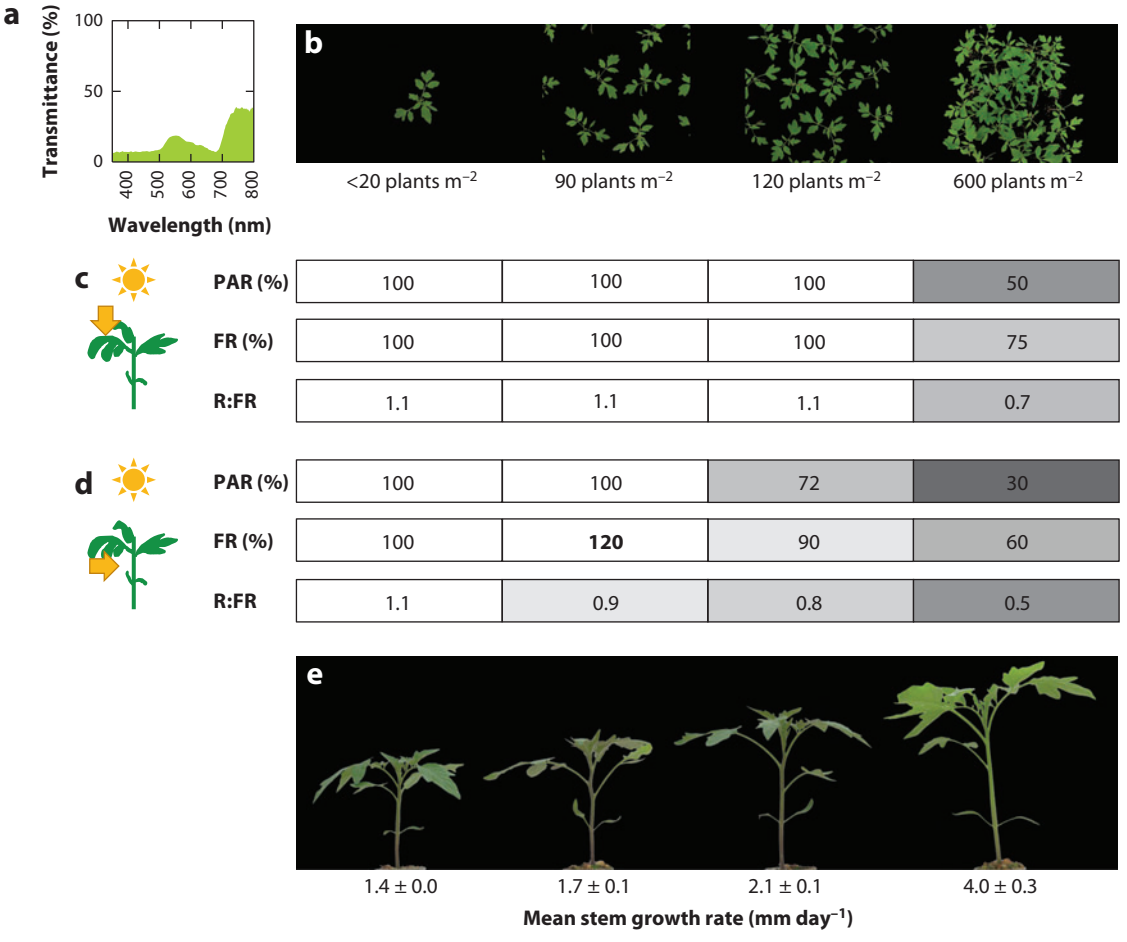
The degree of shade impacts several physiological processes. The lower irradiance in the shade limits photosynthesis and reduces the chance of damage from excess PAR and UVB. The changed spectral composition alters the balance between photosystems I and II. The associated changes in temperature can affect metabolic reactions and reduce water demand. The direct actions of these inputs via photosynthesis, photodamage, or temperature are beyond the scope of this review, which is focused on the action of specific photosensory receptors. Related reviews are helpful to obtain insight from different angles and gain a perspective on the evolution of this field of study (4, 18, 22, 47, 95, 127, 129).

**UVR8:** UV RESISTANCE LOCUS 8 (UVB photoreceptor)

The sensory photoreceptors involved in perceiving differences between full light and shade light include phytochromes, cryptochromes, phototropins, and UV RESISTANCE LOCUS 8 (UVR8) (**Figure 2*a***). The experimental evidence in favor of the roles of these photoreceptors is based on (*a*) the use of photoreceptor mutants, which show features of plants grown under shade when grown under natural or simulated sunlight conditions, and which are less different from the wild type when grown under natural or simulated shade light, and/or (*b*) the response to selected simulated features of the shade-light environment perceived by specific photoreceptors (e.g., the comparison of low and high R:FRs at equal PAR).

### 2.1. Phytochromes

Phytochromes are homodimeric photoreceptors of approximately 120-kDa monomers that bear a single linear tetrapyrrole chromophore (137). They have two forms. The biologically inactive Pr form, which has a maximum absorbance in red light (660 nm), is phototransformed into the biologically active Pfr form, which shows a different conformation and has a maximum absorbance in far-red light (730 nm). Excited Pfr relaxes into Pr. In addition, Pfr can be degraded in the proteasome after ubiquitination, and some pools can back-revert to Pr in a reaction that does not require light (thermal reversion or dark reversion). The phytochrome apoproteins are encoded by a small family of genes involving three main clades: *PHYA*, *PHYB*, and *PHYC* (93). In some species, the *PHYB* lineage includes different members (*PHYB*, *PHYD*, and *PHYE* in *Arabidopsis*; *PHYB*, *PHYB2*, and *PHYE* in tomato; etc.).



Phytochrome B (phyB) is clearly the most important photoreceptor in the vast majority of responses to shade, in some cases redundantly with other members of its clade (**Figure 1*f***). phyB Pfr migrates from the cytosol to the nucleus—a movement facilitated by its selective binding to transcription factors (106)—and exerts its biological activity there. The primary biochemical/biophysical action of phyB has not been established. phyB is able to perceive the low R:FR of shade light, which increases the magnitude of the Pfr-to-Pr reaction compared with the Pr-to-Pfr reaction, shifting the steady-state levels toward the Pr form (**Figure 2*b***). Plant phytochromes are well suited to operate in the canopy range of R:FR (128), particularly when compared with the microbial models (137).

In *Arabidopsis*, phyB shows dark reversion (133) (**Figure 2*b***), which makes it a good sensor of irradiance, at least during early stages of seedling development (23, 108). Under the low irradiances of shade, Pfr dark reversion may become relatively more important because phototransformation reactions are slow, shifting the steady-state levels of phyB to Pr. Under full sunlight, light reactions are much faster than dark reversion, and the proportion of Pfr is predicted to approach photoequilibrium. phyB Pfr destruction is relatively slow (81) and involves the E3 ligase CONSTITUTIVE PHOTOMORPHOGENESIS 1 (COP1) (61).

Phytochrome A (phyA) Pfr migrates to the nucleus after binding to FAR-RED ELONGATED HYPOCOTYL 1 (FHY1) and FHY1-LIKE (FHL) (52). Once in the nucleus, it must be transformed to Pr to be released from the inhibitory action of FHY1 and FHL and then back-transformed to Pfr for nuclear activity (109) (**Figure 2*c***). The destruction of phyA Pfr is fast and involves COP1 (122), and dark reversion depends on the genetic context (40). Some steps between inactive phyA Pr in the cytosol and active Pfr in the nucleus are favored by red light, but others are favored by far-red light (**Figure 2*c***). phyA is not a good R:FR sensor, as under mixtures of red and far-red light its activity is largely unaffected by R:FRs between 1.1 and 0.3 (118) and is actually increased by very low R:FRs (<0.3) (130). When the low R:FRs of shade are simulated by adding far-red light to a PAR background, the overall stronger irradiance increases phyA activity (64).

phyA is a good sensor of irradiance changes associated with shade (118), presumably owing to the multiple light reactions required to yield phyA Pfr in the nucleus (109) (**Figure 2*c***). Although often neglected, phyA can make a direct contribution to shade responses, which becomes obvious in the *phyB* mutant background (compare *phyA phyB1 phyB2* with *phyB1 phyB2* in **Figure 1*f***). In the presence of phyB, this contribution is obscured by the effects of phyA on phyB signaling (25).

**phyB:** phytochrome B (R:FR and red-light photoreceptor)

**COP1:** CONSTITUTIVE PHOTOMORPHOGENESIS 1 (E3 ligase)

**phyA:** phytochrome A (red- and far-red-light photoreceptor)



**Figure 1**

Light signals, photoreceptors, and shade avoidance for tomato plants in response to the density of neighbors. (*a*) Wavelength dependence of transmittance for a green tomato leaf, scanned with an Ocean Optics USB4000-UV-VIS spectrometer configured with a DET4-200-850 detector and QP600-2-SR optical fiber. (*b*) View of the canopies from above along with the plant density of each. (*c*) Photosynthetically active radiation (PAR), far-red light (FR), and red/far-red photon flux ratio (R:FR) of the incoming radiation, measured with the SKR 1850 four-channel sensor probe of a Skye Instruments SKL 904/I SpectroSense2 meter facing upward in the place of a plant removed from the canopy. (*d*) PAR, FR, and R:FR for the horizontally propagating radiation, measured with the sensor probe facing toward the neighbors within the canopy. In panels *c* and *d*, the intensity of the gray shading matches the light reduction compared with <20 plants $m^{-2}$. (*e*) Mean stem growth rates for wild-type plants grown at different densities (each data point is mean ± SE for 4 replicate plants). (*f*) Mean stem growth rates for wild-type and photoreceptor-mutant plants grown at the lowest and highest densities (each data point is mean ± SE for 12 replicate plants). The presence (+) or absence due to mutation (−) is indicated for phytochrome A (phyA), phyB1, and phyB2 in the left panel (GT background) and for cryptochrome 1 (cry1) in the right panel (Moneymaker background).

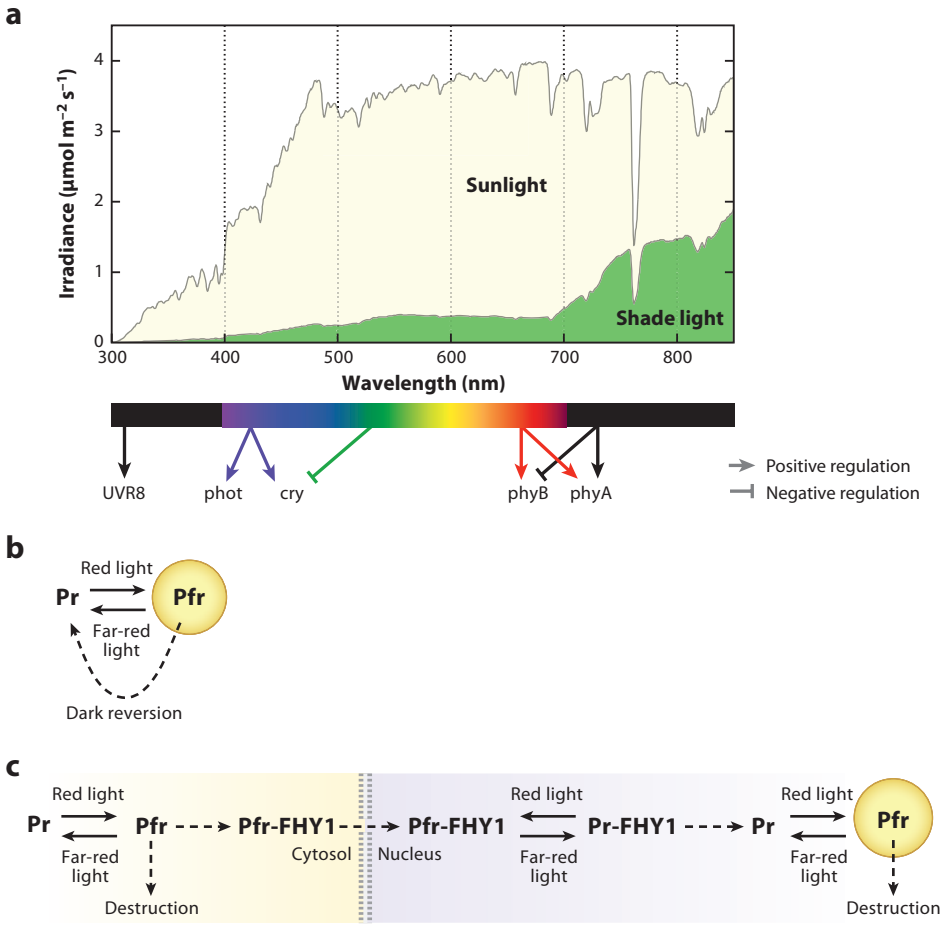



**Figure 2**

Perception of shade-light signals by photosensory receptors. (*a*) Spectral photon distribution of sunlight and shade light and the impact of different wavebands on the status of phytochromes, cryptochromes, phototropins, and UVR8. (*b*) Light (*solid lines*) and dark (*dashed lines*) reactions defining the abundance of active phyB Pfr (*yellow circle*) in *Arabidopsis*. (*c*) Light (*solid lines*) and dark (*dashed lines*) reactions defining the abundance of active phyA Pfr in the nucleus (*yellow circle*) in *Arabidopsis*.

In tomato plants grown for several weeks under sunlight, phyA makes little difference under shade (compare *phyA phyB1 phyB2* with *phyB1 phyB2* at the 600 plants $m^{-2}$ density in **Figure 1*f***). However, in young *Arabidopsis* seedlings, phyA is critical under shade to complete the transition between skotomorphogenesis (the developmental pattern in darkness) and photomorphogenesis (141), a transition also called de-etiolation.

**cry1 and cry2:** cryptochrome 1 and 2 (blue-light and UVA photoreceptors)

## 2.2. Blue-Light and/or UV Photoreceptors

Cryptochromes are photolyase-like blue-light/UVA receptors (**Figure 2*a***) that bind a flavin adenine dinucleotide chromophore (142). *Arabidopsis* cryptochrome 1 (cry1) is present in the nucleus and cytosol, and the amount and localization do not appear to be significantly affected by light. In addition to perceiving blue-light/UVA levels, cry1 would

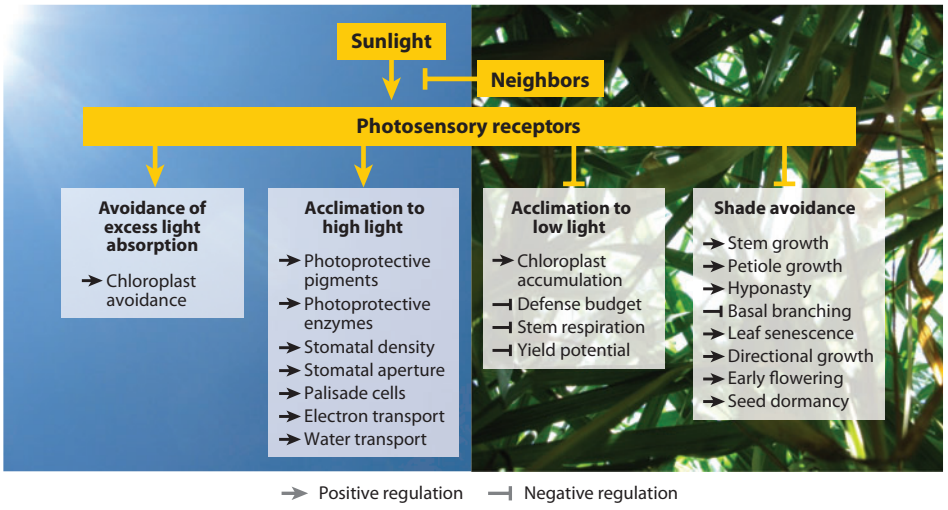



**Figure 3**

Plant responses to either sunlight (*left*) or shade light (*right*) perceived by photosensory receptors.



be involved in perceiving blue/green ratios (13, 118). Bouly et al. (13) have postulated that cry1 activation via blue light initiates the formation of a flavosemiquinone signaling state that can be converted by green light to an inactive form, but this idea has been challenged (86). *Arabidopsis* cry2 plays a key role in the response to day length and contributes to some shade responses.

Phototropins are serine/threonine protein kinases activated by blue-light/UVA radiation (29) (**Figure 2*a***). Phototropins bear two LOV domains that bind flavin mononucleotide chromophores. *Arabidopsis* has two phototropins (phot1 and phot2) with shared as well as specific functions. They are localized to the plasma membrane but dissociate from it upon activation. Phototropins play a key role in the perception of light gradients within plant tissues.

UVR8 is a β-propeller protein homodimer that monomerizes upon UVB irradiation absorbed by specific tryptophans proposed to act as a chromophore (55) (**Figure 2*a***). The UVR8 monomer migrates from the cytoplasm to the nucleus.

## 3. THE RESPONSES TO CANOPY LIGHT SIGNALS MEDIATED BY PHOTOSENSORY RECEPTORS

Dense and open canopies impose different challenges, ranging from a shortage of photoassimilates under limiting PAR levels to the damaging effects of excessive PAR and UV levels. Avoidance responses tend to minimize exposure to these sources of stress, and acclimation responses tend to reduce the adverse impact of extreme conditions that cannot be avoided (**Figure 3**). Canopy light signals perceived by photosensory receptors modulate the extent of these responses.

### 3.1. Shade Avoidance

The perception of signals of current or future shade by phytochromes (127–129) and cryptochromes (68, 118) triggers changes in plant body form that tend to reduce the degree of shade. These are the so-called shade-avoidance responses. Shade is more intense in the lowest strata of the canopy and close to neighbor plants and increases with the progression of

**phot1 and phot2:** phototropin 1 and 2 (blue-light and UVA photoreceptors)

**Shade-avoidance response:** shade-signal-induced change in plant architecture that reduces the chances of current or future shade

canopy growth; i.e., shade varies in the vertical, horizontal, and temporal dimensions. Shade-avoidance reactions involve one or more of these axes, and the dominant component of the syndrome depends on the species.

In vertical shade avoidance, the average position of the foliage is elevated by an increase in the height of the leaves and/or a decrease in the foliage placed at lower strata of the canopy. Consequently, the foliage is on average less shaded because the upper strata of the canopy are better lit. Increased stem growth (56, 97) (**Figure 1*e***), increased petiole growth, and hyponasty (upward bending of the leaves caused by their lower sides growing faster than their upper sides) (74, 117) elevate the position of leaf lamina. Reduced branching (45) and increased leaf senescence (114) downsize the foliage at the base of the canopy.

Increased stem extension growth is arguably the most well known shade response mediated by photosensory receptors (**Figure 1*e***). The selective exposure of the stem to low R:FR (97) or to low levels of blue light or red plus far-red light (7) is enough to promote the growth of this organ, but leaf perception of light signals contributes to the response (20). The growth promotion by low R:FR is already detectable on the order of minutes, and its magnitude inversely relates to R:FR (30, 97). Analysis of tomato photoreceptor mutants indicates that the stem response is mediated by phyB (phyB1 and phyB2), phyA, and cry1 (**Figure 1*f***). There is some degree of photoreceptor redundancy: The function of phyB1 and phyB2 is revealed by the *phyB1 phyB2* double mutant, and the function of phyA is revealed by the comparison of *phyA phyB1 phyB2* with *phyB1 phyB2*. These mutants show elongated stems when grown in isolation from neighbors but not when grown at high densities. There is significant genetic interspecific and intraspecific variability in shade avoidance, which might reflect adaptation to local conditions (12, 44, 98).

In horizontal shade avoidance, plant organs grow away from neighbors. The heterogeneous nature of plant canopies leads to areas with less foliage on a horizontal plane (gaps). The projection of the foliage toward gaps by positive shoot phototropism enhances light interception, and the *Arabidopsis* phototropic-deficient *phot2* mutant shows impaired seedling survival under dense canopies (48). In maize, the leaves grow away from the sowing rows (where the R:FR is reduced as a result of nearby plants) toward the areas between rows (where PAR is not depleted) (90). In cucumber, the hypocotyl bends toward areas of high R:FR and high blue light (6). *Portulaca* seedlings do not develop in the direction of neighbors (100).

In temporal shade avoidance, the timing of developmental decisions is adjusted to minimize the impact of shade. Many seeds remain dormant on the soil surface when exposed to canopy shade (36, 136). The occurrence of gaps in the canopy increases the R:FR reaching the seeds and induces their germination. This delay in seed germination precludes the appearance of young seedlings in environments with severe PAR limitations. In growing canopies, early flowering in response to low R:FR (24, 41) can help to complete the cycle before canopy competition seriously limits PAR. In rosette plants, the transition to the reproductive stage also stimulates stem growth and vertical shade avoidance.

### 3.2. Avoidance of Excess Light Absorption

In plants exposed to high irradiance (typical of open places), the perception of blue light by phot2 causes the chloroplasts to move toward the anticlinal wall of palisade cells (63, 65, 115). This relocation decreases the amount of light absorbed by chloroplasts and reduces the damage of the photosynthetic apparatus (bleaching, necrosis) (67).

### 3.3. Acclimation to Limiting Photosynthetically Active Radiation

Under low irradiance, both phot1 and phot2 cause chloroplasts to accumulate at the periclinal wall of palisade cells (115), increasing efficient light capture (33). The responses that

help plants cope with the limited generation of photoassimilates under shade include a reduced investment in defense against biotic threats, a reduced stem respiration rate, and a reduced generation of yield potential.

Shade signals reduce the defense budget. Low R:FR and/or *phyB* mutations (either alone or combined with other *phy* mutations) enhance the growth of incompatible strains of *Pseudomonas syringae* in *Arabidopsis* (42, 51), the susceptibility of *Arabidopsis* to the necrotrophic fungus *Botrytis cinerea* (26), the susceptibility of rice to blast fungus (*Magnaporthe grisea*) (140), and the growth of caterpillars on *Arabidopsis* or tomato (60, 96). cry1 activity (139) and UVB (35) also enhance plant defenses. Reduced defense is not a consequence of resource investment in shade avoidance (19, 39, 76).

Shade enhances stem and/or petiole growth while reducing the level of photoassimilates to fuel growth. However, there are mechanisms that reduce the energetic cost of shade-avoidance reactions (15). In tomato, low R:FR causes a much stronger reduction in photosynthetic and photoprotective pigments (anthocyanin, flavonols) and photosynthetic capacity in the stem than it does in the leaves. Downsizing the stem photosynthetic apparatus reduces the cost of this organ under low R:FR, which is manifested in lower respiration rates (15).

Phytochrome perception of low R:FR can reduce grain yield. This reduction may be the consequence of shade-avoidance reactions (via, for instance, increased carbon allocation to the stem rather than to the grains, or reduced branching and a lower number of grain-bearing shoots), but there are more direct actions of low R:FR on yield. In wheat, low R:FR can simultaneously reduce the growth of both the ear and the stem, leading to reduced grain yield without stem shade-avoidance reactions (135). This pattern is not a remnant of wild wheat that breeding and selection for yield have failed to eliminate, because the analysis of cultivars released to the market at different times of the twentieth century revealed stronger responses in the latest genotypes of the series. The R:FR signal could set yield potential according to the perceived level of resources in crowded environments.

### 3.4. Acclimation to High Photosynthetically Active Radiation

The responses that help plants cope with the high irradiance of open places include increased stomatal conductance, photosynthetic capacity, stem water transport capacity, and protection against UV. phyB perception of low R:FRs (10) and low irradiances (23) reduces stomatal density in the leaves. The *cry1 cry2* mutations have a similar effect (9, 66). These responses involve a reduction in the number of guard cells per total number of epidermal cells (stomatal index). In addition, *cry1 cry2* mutants show reduced stomatal opening (9, 91). The classical induction of stomatal opening in response to blue light is mediated by phot1 and phot2 (71), and a full response requires the high irradiances typical of open places (9). The combined effects of phot1, phot2, phyB, cry1, and cry2 on stomatal density and/or opening increase stomatal conductance in open places, which implies a cost in terms of increased transpiration under conditions where atmospheric water demand is more intense. However, this cost would be overcompensated for by the additional influx of carbon dioxide, which allows a higher proportion of the energy derived from absorbed PAR to be diverted to photosynthesis rather than to the generation of damaging reactive oxygen species. These photoreceptors also have nonstomatic effects, such as enhanced light-saturated rates of electron transport per unit area (11), which promote photosynthesis at high PAR (9, 10). The development of leaf palisade parenchyma cells, a feature typical of sun leaves, depends on phot1 and phot2 (75). Both phyB and phyA enhance stem xylem development, favoring water transport to the leaves, which is particularly critical under the high water demands of open places (2, 19).

UVB causes DNA damage and generates reactive oxygen species. UVR8 perception of UVB stimulates the transcription of genes involved in UV-protective responses and induces the synthesis of flavonoids (14, 73).

**PIF:** PHYTOCHROME-INTERACTING FACTOR (bHLH transcription factor)

Because UVB levels are significantly attenuated under shade (**Figure 2*a***), UVB could act as a signal of foliage density (35). Under high PAR, cry1 also promotes the synthesis of flavonoids and the expression of genes involved in photooxidative stress tolerance, such as those encoding glutathione S-transferases and glutathione peroxidases (124).

## 4. PIFs CONTROL GROWTH DURING SHADE AVOIDANCE

Having provided a description of the different responses to sunlight and shade-light conditions mediated by photosensory receptors, the following sections will focus on the signaling network controlling stem growth in response to shade signals.

### 4.1. Phytochromes Negatively Regulate the Abundance of PIFs

PHYTOCHROME-INTERACTING FACTORS (PIFs) belong to a subfamily of the basic helix-loop-helix (bHLH) transcription factor superfamily. Seven members of this subfamily [PIF1/PIF3-LIKE 5 (PIL5), PIF3, PIF4, PIF5/PIL6, PIF6/PIL2, PIF7, and PIF8] have been shown to interact physically with phyB through the conserved N-terminal sequence, called the active phyB-binding motif; two members (PIF1/PIL5, PIF3) also interact with phyA via a separate motif (83). As a result of this direct interaction, PIF1 (102, 125), PIF3 (1, 8, 104), PIF4 (88), and PIF5 (88, 126) become phosphorylated and degraded via the ubiquitin-proteasome system, with degradation half-times in the range of 5–20 min (83). PIF7, in contrast, becomes phosphorylated but not strongly degraded (81, 85). There is a residual pool of PIF proteins even when prolonged light is perceived by the phytochromes. PIF3 (82, 84), PIF4 (88), and PIF5 protein levels increase rapidly when plants grown under high R:FRs become exposed to low R:FRs that reduce phyB activity (**Figure 4*b*,*c***).

### 4.2. Phytochrome Inhibits the Binding of PIFs to Their Target Promoters

Binding of PIF7 to its target gene promoters increases significantly 1 h after transfer from high to low R:FR (85). Because R:FR rapidly and significantly modifies PIF7 phosphorylation without concomitant changes in PIF7 protein levels, Pfr-induced phosphorylation has been proposed as the primary mechanism reducing PIF7 binding to its targets under high R:FR (85). phyB releases PIF1 and PIF3 from their DNA targets (105). A truncated version of phyB retaining the N-terminal domain is biologically active and releases PIF3 from its targets, but it is unable to induce PIF3 degradation, implying that the primary inactivation of PIF activity by phyB involves its negative regulation of promoter binding (105).

### 4.3. PIFs Promote Shade-Avoidance Responses

Plants overexpressing PIF5 show enhanced stem and petiole growth even under high R:FR (88). Truncated PIF5, lacking the active phytochrome-binding domain that mediates PIF interaction with active phytochrome, shows impaired degradation but efficiently promotes stem and petiole growth (88). Full shade-avoidance responses require PIF3 (82, 84, 120), PIF4, PIF5 (82, 88, 120), and PIF7 (85) under low R:FR (82, 85, 88) or natural shade light (120) (**Figure 4*a***). These results are consistent with a model where the low R:FRs of shade reduce the levels of active phyB, increasing the activity of PIFs, which in turn induce growth responses to shade (**Figure 4*b***). PIFs control the expression of some cell wall–associated genes by binding their promoters (34, 58, 84). The latter genes include *XYLOGLUCAN ENDOTRANSGLYCOSYLASE 7* (*XTR7*)/*XYLOGLUCAN ENDOTRANSGLUCOSYLASE/HYDROLASE 15* (*XTH15*), which is required for the growth responses to shade (117) (**Figure 4*c***). However, many other growth-related genes are indirectly controlled by PIFs.

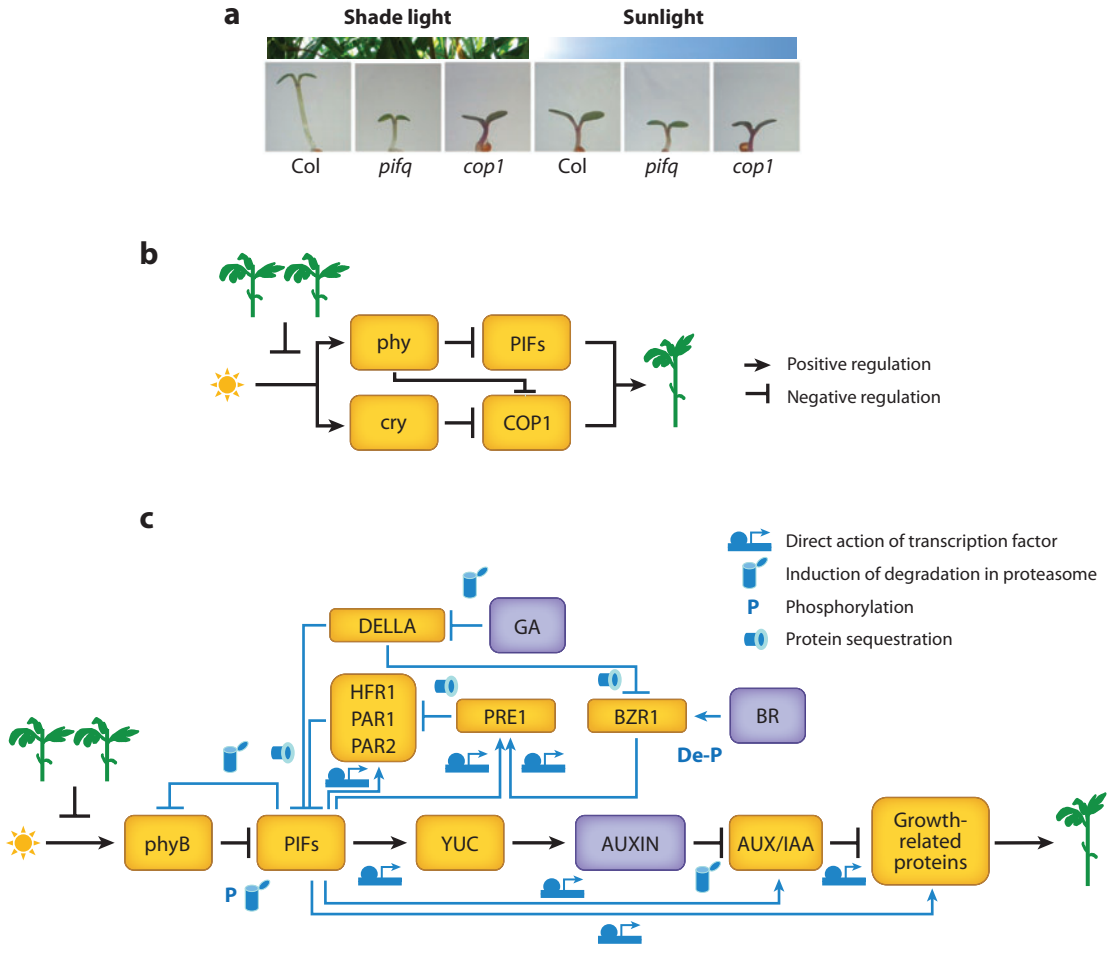



**Figure 4**

Signaling network in shade-avoidance responses. (*a*) *Arabidopsis* seedlings grown for one week under either a dense canopy (shade light) (*left*) or sunlight (*right*). Shade-avoidance responses require *PIF* and *COP1* genes. The wild type (Col) shows a long hypocotyl under shade compared with sunlight conditions, but this response is reduced in the quadruple *pif1 pif3 pif4 pif5* (*pifq*) and *cop1* mutants. (*b*) Schematic representation of the PIF module and the putative COP1 module. (*c*) Details of the PIF module. The main pathway (*black*) and the regulatory loops (*blue*) are indicated. Proteins are in orange; hormones are in purple. Abbreviations: BR, brassinosteroid; GA, gibberellins.

## 4.4. PIFs Enhance Auxin Synthesis to Promote Stem Growth

Plant responses to R:FR involve changes in the status of hormone signaling (**Table 1**). Several features of the shade-avoidance syndrome, such as increased stem growth, leaf hyponasty, and apical dominance, are characteristic of high auxin levels. After 1–2 h of low R:FR, *Arabidopsis* plants can exhibit elevated auxin levels (58, 85, 134). Auxin-related genes are overrepresented among those showing promoted expression in response to low R:FR (37, 74, 123). The expression of the *DR5-GUS* reporter, indicative of auxin signaling, is promoted in the cotyledons and hypocotyl by low R:FRs given to the whole seedling, but the hypocotyl response is blocked by the addition of the auxin transport inhibitors, suggesting that auxin produced in the

**Table 1 Hormone levels and associated processes in response to the red/far-red photon flux ratio (R:FR)**

| Hormone | Levels under low R:FR | Process[a] |
|---|---|---|
| Auxin | ↑ *Arabidopsis* seedlings (58, 85, 134), sunflower stems and leaves (77)<br>— *Arabidopsis* petioles (74), tomato stems and leaves (15) | Shade avoidance (growth, branching) (45, 69, 85, 134) |
| Gibberellins | ↑ Cowpea stems (92), sunflower stems and leaves (77) | Shade avoidance (growth) (38) |
| Brassinosteroids | — Sunflower stems (78) | Shade avoidance (growth) (74, 89) |
| Ethylene | ↑ Sorghum (46), tobacco (107), tomato (15) | Shade avoidance (growth) (107) |
| Cytokinins | ↑ Sunflower leaves (77) | Shade avoidance (growth) (16) |
| Strigolactones | ? | Shade avoidance (branching) |
| Jasmonic acid | ↓ Tomato stems (15)<br>—Tomato leaves (15) | Defense (26, 96), growth, pigments (15, 111) |
| Salicylic acid | ↑ Sunflower stems (79)<br>—*Arabidopsis* seedlings (51)[b] | Defense (51) |
| Abscisic acid | ↑ *Arabidopsis* leaves (53),[b] sunflower leaves (77), tomato leaves (15) | Water relations (53) |

↑, increased; ↓, decreased; —, not affected.
[a]Mutants or inhibitors impairing hormone synthesis or signaling affect the indicated responses to R:FR.
[b]*phyB* or *phyA phyB* mutant seedlings compared with the wild type under high R:FR.

cotyledons is transported to promote hypocotyl growth in response to shade (134). Mutations at genes involved in auxin synthesis (85, 134), perception, or transport (69) impair shade-avoidance responses. Increased free auxin levels in the hypocotyl require the presence of the auxin efflux regulator PIN-FORMED 3 (PIN3), which in response to low R:FR shows increased abundance and predominantly lateral cellular localization in the endodermis cells, potentially redirecting the auxin flow to the growth-limiting epidermal cells (69). Changes in auxin levels are not observed under every condition where auxin signaling plays a significant role in shade-avoidance responses (74).

The identification of genome-wide PIF5 binding sites in seedlings harvested 2 h after transfer from high to low R:FR has revealed that 96% of the 200-base-pair sequences centered to a binding peak summit contain an E-box (5′-CANNTG-3′), the majority of which are a G-box DNA motif (5′-CACGTG-3′) (58), which is known to be bound by PIF5 (59) and other PIFs (83). Many of these genes are involved in the response to hormone stimulus, especially the response to auxin stimulus. Genes bound by both PIF4 and PIF5 that exhibit reduced expression in the *pif4 pif5* double mutant include *YUCCA 8* (*YUC8*), which encodes a rate-limiting enzyme in auxin synthesis (58). Similarly, the *pif7* mutant shows reduced promotion of *YUC2*, *YUC5*, *YUC8*, and *YUC9* genes in response to low R:FR, and at least the *YUC8* and *YUC9* promoters bind PIF7 (85). It is interesting that a genome-wide association study has identified variants at the *YUC5* and *YUC9* genes as potentially underlying variations in shade avoidance (44). Both *pif4 pif5* and *pif7* mutants show impaired auxin-level increase in response to low R:FR (58, 85). The backbone of the shade-avoidance signaling network involves the reduction of active phyB by low R:FR, the subsequent increase in PIF binding, and the activation of auxin-synthesis genes,

which lead to increased auxin levels. PIF7 is also required for low R:FR to promote the expression of the auxin transporter genes *PIN3* and *PIN4* (85). Low R:FRs shift the location of PIN3 transporters from the basal to the lateral side of the membrane of the endodermal cells, but this could be an indirect consequence of the increased auxin levels (69).

## 4.5. PIFs Reduce the Abundance of phyB

The residual pool of PIFs present under high R:FR promotes the polyubiquitination of active phyB by COP1, which leads to the degradation of phyB in the proteasome (61, 81) (**Figure 4*c***). Under high R:FR, young *Arabidopsis pif1 pif3 pif4 pif5* seedlings contain approximately three times the level of PHYB observed in the wild type (82). When seedlings grown under high R:FR are transferred to low R:FR, the PHYB levels increase and become largely independent of PIFs 12 h later (82). Although PIFs are more abundant under low R:FR, under this condition they have little effect on phyB levels, likely owing to their reduced interaction with the Pr form of phyB, which predominates under low R:FR. Therefore, PIFs do not enhance shade-avoidance responses by further reducing the active phyB levels; rather, keeping the phyB level under the control of PIFs helps to sustain basal stem growth under high R:FR.

## 4.6. Negative Feedback Loop Involving PIFs and HLH

PIFs have multiple connections with other players in shade avoidance (**Figure 4*c***). LONG HYPOCOTYL IN FAR-RED 1 (HFR1), PHYTOCHROME RAPIDLY REGULATED 1 (PAR1), and PAR2 are HLH proteins that lack the typical basic domain necessary for binding to gene promoters (50). HFR1 forms non-DNA-binding heterodimers with PIF4 and PIF5 (59), and PAR1 and PAR2 do so with PIF4 (54). This interaction reduces shade-avoidance responses (54, 59, 113, 123). PIF5 binds the promoter of *HFR1*, *PAR1*, and *PAR2* (58), and PIFs promote the expression of at least *HFR1* and *PAR1* (58, 84, 88). This defines a negative feedback loop where, via PIFs, low R:FRs promote the expression of genes involved in the repression of shade-avoidance responses (**Figure 4*c***).

## 4.7. DELLA Proteins Link PIFs to Signaling by Gibberellins

The promotion of stem and petiole growth is also a typical response to the application of gibberellins, and low R:FR can increase the levels of gibberellin $A_1$ (**Table 1**) (77, 92), in some cases by reducing its inactivation (92). Gibberellins cause the degradation of DELLA proteins (132). Canopy shade light, low R:FR, and low blue irradiance reduce the stability of DELLAs, likely as a consequence of increased gibberellin levels (38). Canopy-light-induced DELLA degradation appears to be a prerequisite for shade-avoidance responses, because mutants bearing stable DELLA versions (i.e., mutated at the DELLA domain required for degradation) show reduced responses to low R:FR or low blue-light signals, and mutants combining loss-of-function alleles at multiple DELLA loci show elongated stems even in the absence of shade signals (38). DELLAs bind PIF3, PIF4, and likely other PIFs (34, 43). The first conserved heptad leucine repeat of DELLA and the PIF DNA-recognition domain mediate this interaction, which prevents PIFs from binding to their target gene promoters and regulating gene expression (34, 43). It is noteworthy that PIF5 directly binds the promoters of the *GIBBERELLIC ACID INSENSITIVE* (*GAI*) DELLA gene and the *GIBBERELLIN 2-OXIDASE 6* gene, the latter of which is involved in a major gibberellin inactivation pathway (110). PIFs promote the expression of these genes under low R:FR (84), apparently generating a negative regulatory loop.

## 4.8. DELLA Proteins Link PIFs to Signaling by Brassinosteroids

The promotion of stem growth by far-red light reflected from neighbors requires

brassinosteroid synthesis (89). This is also the case for petiole growth through the lowering of phytochrome (74) or cryptochrome (68, 70) activity. The transcription factor BRASSINAZOLE-RESISTANT 1 (BZR1), which is involved in brassinosteroid responses, and PIF4 physically interact and synergistically control the expression of common target genes, including those encoding PACLOBUTRAZOL RESISTANCE (PRE) HLH proteins, which promote stem growth (103) (**Figure 4*c***). DELLAs also negatively regulate brassinosteroid signaling by binding BZR1 and impeding its binding to brassinosteroid-responsive genes (3, 49). DELLAs, BZR1/2, and PIFs could form the central command system in the control of growth processes (3), potentially including those connected to shade avoidance. *PRE1*, *PRE5*, and *PRE6* are direct targets of both BZR1 and PIF4 that synergistically promote their expression (3). PRE1 binds PAR1 and apparently prevents PAR1 from interacting with PIF4 (54).

### 4.9. PIF Feed-Forward Loops

AUXIN/INDOLE-3-ACETIC ACID INDUCIBLE (AUX/IAA) proteins are transcription factors that repress the expression of auxin-responsive genes, including those related to growth (28). TRANSPORT INHIBITOR RESPONSE 1 (TIR1) is an auxin receptor with E3 ligase activity, which upon activation by auxin targets AUX/IAA proteins to degradation, thus releasing the expression of auxin-responsive genes. PIFs promote auxin synthesis under low R:FR, and therefore they are predicted to induce the degradation of AUX/IAAs as they promote stem growth (**Figure 4*c***). Mutants bearing stable forms of AUX/IAA show impaired shade-avoidance responses (117).

Auxin promotes the expression of *AUX/IAA* genes. One of the most distinctive responses to low R:FR is the promotion of *AUX/IAA* gene expression (37, 74, 123); not surprisingly, this promotion shows robust dependency on PIFs (58, 84, 85, 88). Interestingly, PIFs directly bind the promoter of many of these genes, indicating that to a large extent the control is direct and not just mediated by altered auxin levels. This generates a negative feed-forward loop, where PIFs promote the expression of downstream repressors of growth both directly and via changes in auxin levels (**Figure 4*c***).

Additional direct PIF targets include the homeodomain–leucine zipper transcription factor ARABIDOPSIS THALIANA HOMEOBOX PROTEIN 2 (ATHB2) (58, 76) and PIL1 (58), which are promoted by low R:FR at least partially via PIFs (84, 85, 88). ATHB2 apparently promotes stem growth by modifying the responsiveness to auxin (131). PIL1 can be a positive (116) or negative (112) regulator of shade-avoidance responses to low R:FR.

## 5. THE COP1 PATHWAY IN SHADE AVOIDANCE

Full shade-avoidance responses require both COP1 and PIF (**Figure 4*a***). A low-resolution image of the signaling network leading to shade-avoidance responses could show two major pathways involving PIFs and COP1, respectively (**Figure 4*b***). However, whereas the major events linking PIFs to shade avoidance have been established, crucial questions concerning COP1 remain unanswered.

During the transition between full darkness and light that seedlings experience upon emergence from the soil, the activity of COP1 is reduced by cry1, phyA, and phyB (80). Compared with full darkness, light perceived by cry1, phyA, and/or phyB induces the slow migration of COP1 from the nucleus to the cytosol, but more rapid effects have been documented. In response to blue light, cry1 disrupts the SUPPRESSOR OF PHYA-105 1 (SPA1)/COP1 complex and hence COP1 activity (87). In contrast to cryptochromes and phytochromes, UVR8 increases the activity of COP1 (55). Because shade reduces phyA, phyB, cry1, and UVR8 activity, it is not clear whether shade should increase or decrease COP1 activity.

During skotomorphogenesis, COP1 ubiquitinates and targets to degradation

transcription factors like ELONGATED HYPOCOTYL 5 (HY5), which are required for photomorphogenesis (80). However, the putative pathway by which COP1 could induce responses to shade is not established. The *cop1* mutant does respond to shade in the *b-box domain protein 21* (*bbx21*) *bbx22* double-mutant background, lacking two B-box-containing zinc-finger transcription factors (BBX) (31). BBX21 and BBX22 are negative regulators of shade-avoidance responses. COP1 recruits BBX22 into nuclear speckles in onion epidermal cells and is able to ubiquitinate BBX22 in vitro, but these proteins show no direct physical interaction (32). BBX22 is degraded in the proteasome; this reaction is faster in darkness than under light and requires COP1 (27). It is tempting to speculate that shade allows COP1 to recover activity and that, in turn, COP1 targets BBX22 and other negative regulators of shade avoidance to degradation. COP1 is also required (likely indirectly) for the accumulation of PIF3 in the dark (8), suggesting that it could be required for the accumulation of PIFs in response to shade.

## 6. SHADE-AVOIDANCE RESPONSES IN FLUCTUATING LIGHT ENVIRONMENTS

### 6.1. Owing to the Evening Complex and Light-Derived Signals, For Plants, Night Is Not the Same as Shade

Some features of the light environment that provide signals of canopy status also respond to unrelated atmospheric factors. Irradiance is reduced by shade but also changes with cloudiness, time of day, season, and latitude. R:FR is stable for most of the day but drops at the extremes of the photoperiod (57). Plants have mechanisms to discriminate between canopy signals and at least some of the latter sources of noise.

Plants do not take the night for shade. Particularly during the first part of the night and in plants well beyond the process of de-etiolation, stem growth rate does not increase compared with the growth rate during the day, and it may actually decrease (21). In *Arabidopsis*, nights of up to 9–12 h make little difference compared with continuous white light (99). Although nights beyond that duration do yield longer stems (resembling a shade-avoidance response), in nature these long nights are normally associated with lower temperatures that would minimize their shade-like effect. phyA and cry1 are predicted to be largely inactive during the night. Active phyB is certainly more stable, as demonstrated by classical experiments where stem growth is promoted by a brief pulse of far-red light that transforms Pfr into Pr several hours into the night (39). phyB is necessary to maintain low growth rates during the night (99). Therefore, phyB stability is a first component of the mechanisms that help to extend the daytime no-shade signal into the night (**Figure 5**).

The arrest of stem growth during the night is established by the synergistic interaction between phyB and cryptochromes (119). Blue light perceived by cryptochromes during the day enhances the expression of *SPA1*, *SPA4*, *HY5*, and *HY5 HOMOLOG* (*HYH*) independently of phyB. In turn, SPA1, SPA4, HY5, and HYH enhance phyB-mediated signaling independently of cryptochromes. This creates a hysteretic switch that extends light signaling beyond the presence of light (119).

Although phyB stability is required to restrain the induction of shade avoidance by night, phyB is not fully stable, and Pfr-to-Pr dark reversion can cause physiologically relevant reductions of Pfr levels during the night (**Figure 5*a***). Furthermore, the R:FR declines at the extremes of the photoperiod to levels that would induce shade avoidance if they had occurred during the day. Therefore, to preclude taking the night for shade, plants require a second layer of control, which involves reduced sensitivity to decreased phyB activity during the night. In plants grown under high R:FR, transfer to low R:FR is much more effective at promoting stem growth during the night if this

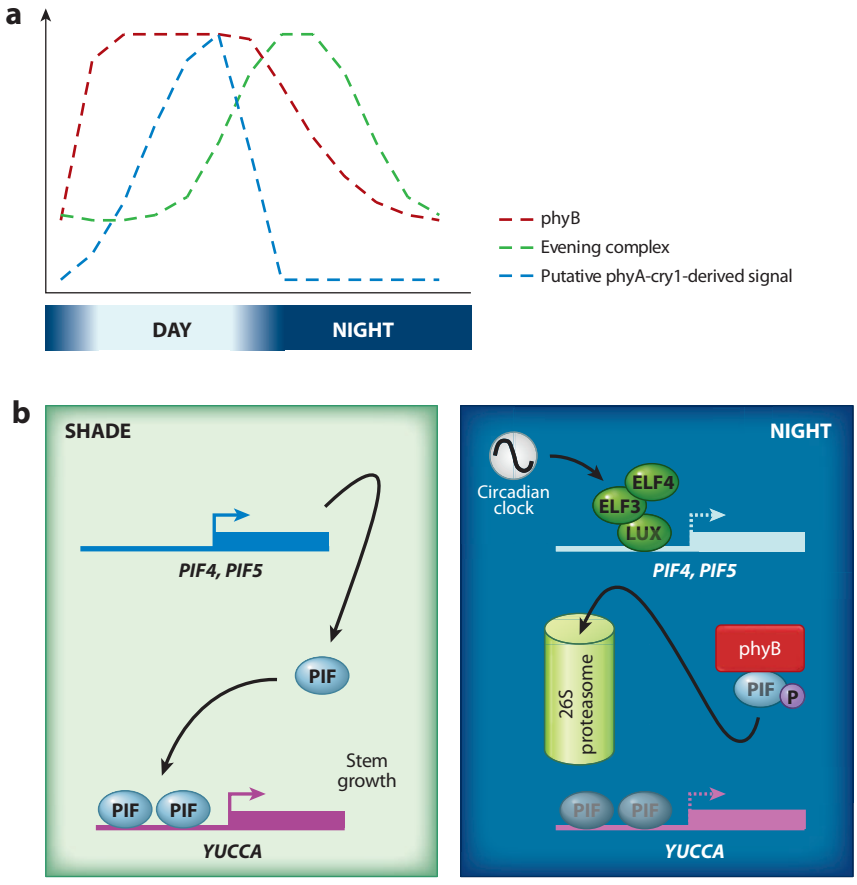



**Figure 5**

Owing to the evening complex and light-derived signals, for plants, night is not the same as shade. (*a*) Diurnal variation of the predicted levels of the three elements—active phyB, the evening complex (ELF4-ELF3-LUX), and a putative signal derived from phyA and cry1 activity—that inhibit the occurrence of shade avoidance in response to the night. (*b*) Differences between shade and night. During the day, shade light allows the accumulation of active PIFs that promote the expression of auxin-synthesis genes. During the night, there is residual active phyB, which reduces the activity of PIFs. In addition, clock-controlled expression of *ELF3*, *ELF4*, and *LUX* sets the formation of the evening complex to the first hours of the night, repressing the expression of *PIF4* and *PIF5*.

transfer occurs during the day rather than immediately before the night (21). Similarly, a low R:FR pulse gradually loses effectiveness during the night (17, 39). These are true effects on sensitivity and not the result of reduced growth capacity during the night, because the stem grows rapidly during the night if the signals are provided early. This control of sensitivity to low R:FR involves clock- and light-derived signals.

Night occurs at a time predicted by the circadian clock, and repression of stem growth in darkness requires the correct function of the circadian clock. In plants with severe clock dysfunction like the *pseudo-response regulator 9* (*prr9*) *prr7 prr5* mutant or *CIRCADIAN CLOCK ASSOCIATED 1* (*CCA1*) overexpressor, the stem is long, particularly when grown at nights of 9–12 h (which in the wild type causes little difference compared with

continuous light), and the expression of *PIF4* and *PIF5* is high throughout the night (99). The clock controls the coordinate expression of *EARLY FLOWERING 3* (*ELF3*), *ELF4*, and the transcription-factor-encoding gene *LUX ARRHYTHMO* (*LUX*), which under short days show a peak of high expression close to the end of the photoperiod (101). Acting as an adaptor protein, ELF3 bridges an interaction between ELF4 and LUX to form a tripartite complex, the so-called evening complex, which accumulates during the early part of the night (**Figure 5*a*,*b***). The LUX transcription factor recruits the complex to the full consensus LUX-binding sites present in the 5′ untranslated region of both *PIF4* and *PIF5* and reduces the expression of these genes (101). *PIF4* and *PIF5* mRNA levels are elevated in *elf3*, *elf4*, and *lux* mutants compared with the wild type, particularly during the early evening, and this causes the long-hypocotyl phenotype of these mutants (101). The *lux* mutant, at least, shows elevated expression of hormone-related, growth-correlated genes during the night (94), and some of these genes are targets of PIF4 and PIF5 (58). Because full expression of shade-avoidance reactions requires PIFs (88), the evening complex would repress early-night-induced shade-avoidance responses. Following this rationale, the *elf3*, *elf4*, and *lux* mutants would have a constitutive shade-avoidance phenotype under day-night cycles because they take the night for shade.

In addition to the action of the clock, the sensitivity to low R:FR signals is positively affected by light perceived by phyA and cryptochromes (17) (**Figure 5*a***). In a photoperiod of 10 h, the most effective time to promote stem growth with a shade event is the afternoon (120). However, shade signals given at this time are not effective if phyA or cryptochromes are not active during the preceding hours. The mechanisms involved in this light sensitization to shade signals are unknown, but it is interesting that the promotion of stem growth by auxin shows the same pattern of light sensitization (120).

### 6.2. HY5 Stops Shade-Induced Signaling in Response to Sunflecks

Depending on the angle of incidence, sunflecks can penetrate through canopy gaps without much interference. Therefore, a plant can be shaded at a given time and become exposed to sunlight (high R:FR, high irradiance) at a different time. In *Arabidopsis* plants grown under shade, a transient (2-h) exposure to sunlight to simulate a sunfleck is perceived primarily by phyB and secondarily by phyA. Sunflecks can severely reduce the hypocotyl shade-avoidance response; promote the expression of genes involved in fatty-acid metabolism, pigment metabolism, and responses to red and far-red light; and reduce the expression of genes involved in hormone-related functions such as auxin stimulus response, ethylene stimulus response, brassinosteroid stimulus response, and the jasmonic acid–mediated signaling pathway (121). Sunflecks increase the expression of the basic leucine zipper transcription factors *HY5* and *HYH* (121). The inhibition of hypocotyl growth by sunflecks is reduced in *hy5* and absent in *hy5 hyh*. Under sunfleck conditions, the *hy5* mutant shows elevated expression of several genes repressed by sunflecks, such as auxin-related genes and *PHYTOCHROME KINASE SUBSTRATE 4* (*PKS4*), some of which are direct targets of HY5.

Compared with uninterrupted shade during the photoperiod, sunflecks are effective at enhancing *HY5* expression and inhibiting shade-avoidance reactions if they occur in the afternoon but not if they occur in the morning (121). In the morning, *HY5* expression increases in response to the transition between night and day, but this response is similar if the seedlings begin the day under sunlight or under shade. In the afternoon, *HY5* expression is again low in both seedlings exposed to sunlight and those exposed to shade during the preceding hours of the photoperiod, suggesting the occurrence of desensitization, but transfer from shade light to sunlight (i.e., sunfleck conditions) promotes *HY5* expression. In other words, sunflecks inhibit hypocotyl growth compared with shade

conditions only when they are able to promote *HY5* expression compared with the expression level under shade. *HY5* expression responds more to the fact that a change in light conditions has occurred (night to day, shade to sunlight) than to the magnitude of that change. Correct circadian clock function is necessary for the growth response to afternoon sunflecks, apparently through the establishment of a permissive, low-auxin signaling state in the afternoon.

## 7. CONCLUDING REMARKS

Plastic plant responses to shade involve phytochromes, cryptochromes, phototropins, and UVR8. These sensory receptors partially differ in photoperceptive function and target processes. All of them can perceive changes in irradiance, but phyB and other members of its clade perceive R:FR. phyB dominates in growth and developmental shade-avoidance and shade-acclimation responses, phot2 in rapid and reversible positional adjustments of chloroplasts and leaves, cry1 in photoprotective mechanisms, and UVR8 in UVB screens.

In response to the degree of shade, photoreceptor signaling targets master transcription factors to regulate diverse processes. For instance, in response to R:FR, phyB directly regulates the abundance/activity of the transcription factors PIF3, PIF4, PIF5, and PIF7 to control stem growth (83) and PIL5 to control seed germination (102). phyB also regulates the morning abundance of CONSTANS (62) to control flowering (138) and the expression of BRANCHED 1 (BRC1) and BRC2 to control branching (45). phyB opposes PIF4 activity in growth responses, but phyB and PIF4 act in the same direction in stomatal-index responses (23). Under high light levels, cry1 uses HY5 to induce anthocyanin synthesis (72) and ZINC-FINGER PROTEIN EXPRESSED IN INFLORESCENCE MERISTEM–LIKE 1 (ZML1) and ZML2 to enhance the expression of antioxidative enzymes (124). Some of these transcription factors are shared by different receptors and/or different responses. For instance, cry1 (72) and UVR8 (55) use HY5 for anthocyanin synthesis, whereas phyB and phyA use HY5 for growth responses to sunflecks (121).

A very simple pathway links shade signals to target shade-avoidance genes. The low R:FR caused by neighbors reduces phyB Pfr. PIFs are then released from phyB binding and phyB-induced phosphorylation, allowing them to bind and activate auxin-synthesis genes to promote stem growth. Wired to this simple pathway are a complex set of regulatory loops that includes links to gibberellins, brassinosteroids, the circadian clock, and light-derived signals (signals perceived by photosensory receptors that sensitize the response to shade). We can intuitively guess that this complexity relates to the complex environment that plants face.

### FUTURE ISSUES

1. The primary light-driven action of phytochromes leading to the phosphorylation of PIFs and the release from their DNA targets remains to be investigated.
2. The primary targets and downstream players in cryptochrome action during shade avoidance have not been fully elucidated.
3. The molecular mechanisms involved in the action of COP1 in shade-avoidance responses require further investigation.
4. The mechanisms by which photoreceptors control the levels and/or signaling status of ethylene, gibberellins, cytokinins, jasmonic acid, salicylic acid, strigolactones, and abscisic acid have not been established.

5. The functional consequences in complex light environments of the regulatory loops involved in the shade-avoidance network will require detailed analysis.
6. The molecular mechanisms involved in the control of sensitivity to shade signals by light-derived signals remain largely unknown.

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**Errata**

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